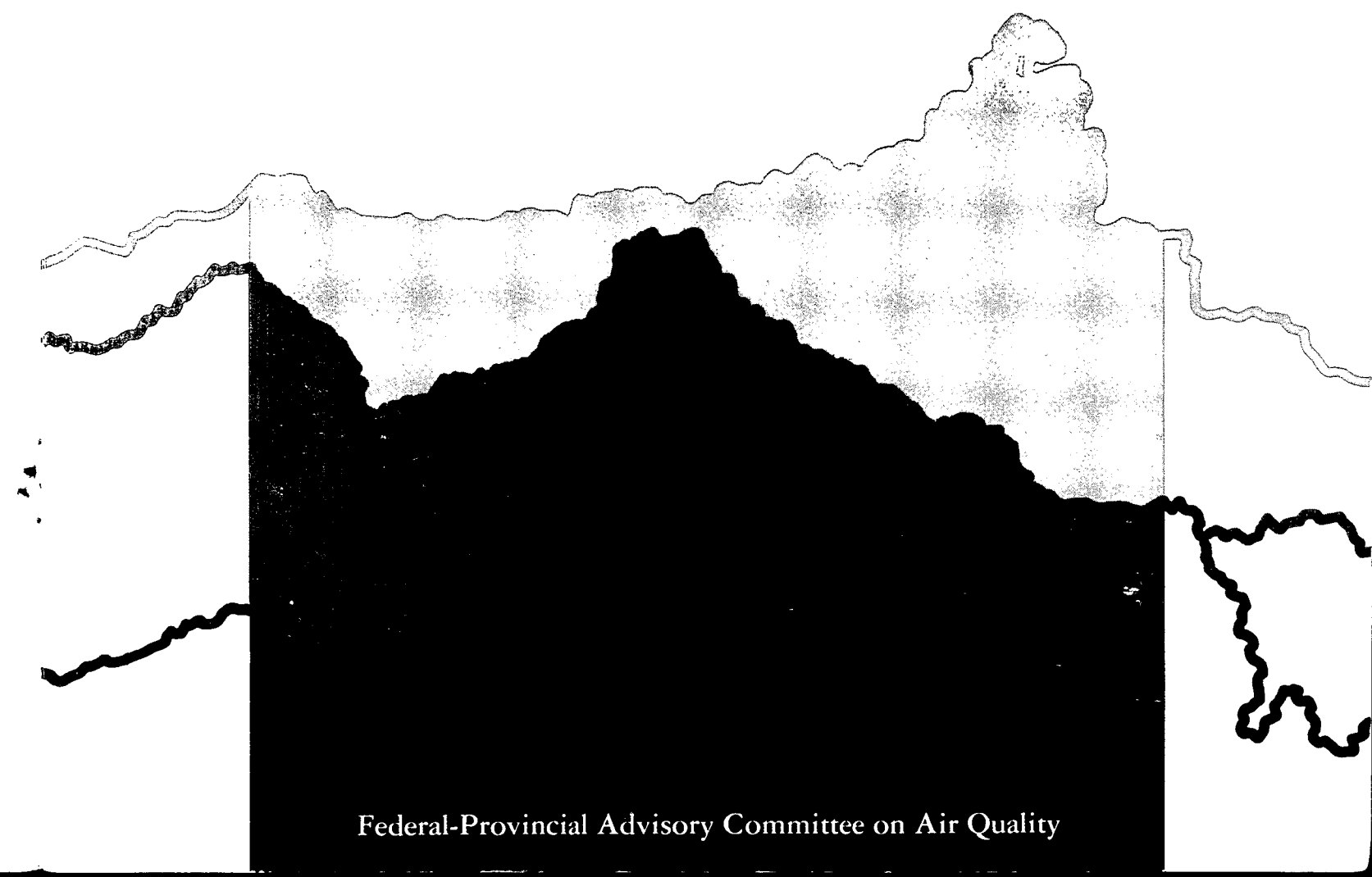


Review of National Ambient
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4.3 Epidemiological Studies

4.3.1 Effects of Short-term Exposure on Mortality. Increases in daily mortality rates have been reported in association with such episodes of acute air pollution as occurred in the Meuse Valley in 1930, Donora in 1948, and London in 1952. People primarily affected were those with existing cardiorespiratory disease. Unfavourable weather conditions resulted in stagnation and the buildup of pollutants, which, under normal conditions would have been dispersed. Air concentrations of SO₂ were reported as high as 3380 µg/m³ (1.3 ppm), 48-hour average, and concentrations of particulate matter were also high. In view of the fact that all pollutant concentrations were most likely elevated because of the stagnation, no single pollutant could be incriminated as being responsible for the observed effect.

Several studies indicate that less severe episodes of air pollution affect the daily mortality rates (Table 5). There is, however, no agreement among these studies on the number of deaths which can be related to air pollution or on the threshold concentrations above which the daily mortality rate is significantly increased above that expected. The relationship between air pollution and mortality is extremely complex and is influenced by several other factors such as temperature extremes, influenza epidemics, day of the week, season of the year. Sulphur dioxide may simply be an index variable for other unmeasured extraneous variables and not in itself the cause of the health effects described in Table 5. Therefore it is difficult to draw meaningful conclusions on the basis of the available data.

4.3.2 Effects of Long-term Exposure on Mortality. Several studies show a relationship between residence in areas of heavy pollution and mortality from all causes combined and mortality from respiratory diseases. It is virtually impossible, however, to derive a dose-response function from these studies because of the many other variables involved and the lack of definitive exposure data.

4.3.3 Effects of Short-term Exposure on Morbidity. Most of the observational studies on health effects associated with short-term exposure to air pollution have been cross-sectional in design; that is, the prevalence of disease in different groups at a certain time has been compared with surmised exposures. Although such studies provide important preliminary information (Table 6), it is difficult to establish with any certainty cause-effect relationships on the basis of such results since current exposure may be irrelevant to current disease. In addition, results observed in cross-sectional studies of

TABLE 5 EFFECTS OF SHORT-TERM EXPOSURE ON MORTALITY*

SO ₂ µg/m ³ (ppm)	(24-hour average) Particulate (µg/m ³)	Protocol	Results	Comments	Reference
1100 (0.42)	2000		increased mortality		Gore and Shaddick (1958)
300 (0.12)	600(TSP)	examination of daily mortality in London during the winter of 1958-59	increases in daily total mortality above the 15-day moving average detected at 300 µg/m ³ SO ₂	correlation with temperature, humidity and visibility also examined	Martin and Bradley (1960)
400 (0.15)	600(TSP)	examination of daily mortality in London during the winter of 1959-60	increases in daily total mortality above the 15-day moving average detected at 400 µg/m ³ SO ₂	correlation with meteorological factors also examined	Martin (1964)
500 (0.19), 786 (0.30)		examination of daily nonepisodic variations in mortality rates in New York City	increase in residual mortality	association probably explained by joint temperature and other variables; 1 monitoring station	Buechly et al. (1973); Glasser and Greenburg (1971)
170 to 625 (0.065 to 0.24)	2.07 to 2.13 (CoH)	time series analysis of daily mortality in New York City, 1963-72	percentage of excess deaths attributable to air pollution not associated with SO ₂ levels	authors conclude SO ₂ not a cause of adverse health effects at these levels; results dependent upon choice of model; 1 monitoring station	Schimmel and Greenburg (1972), Schimmel and Murawski (1975)
900 (0.35)	500 to 800(BS)	examination of mortality in London during an air pollution episode in 1975	200 excess deaths	doctors' strike prior to episode and decrease in temperature; elevated levels of H ₂ SO ₄ , hydrocarbons and NO _x	Ellison et al. (1978)
65 to 123 (0.025 to 0.047)	0.581 to 1.273 (CoH)	time series analysis of daily mortality for three areas in Pittsburgh, 1972-77	no association between SO ₂ and mortality	meteorological factors considered	Mazumdar and Sussman (1983)

* Modified from Health and Welfare Canada (1984)

CoH = Coefficient of haze
BS = Black Smoke

TSP = Total suspended particulate

TABLE 6 EFFECTS OF SHORT-TERM EXPOSURE ON MORBIDITY*

SO ₂ µg/m ³ (ppm)	(24-hour average) Particulate (µg/m ³)	Study Type	Health Effects	Comment	References
400 (0.15)	600(TSP)	cross-sectional	increase in hospital admissions (cardiac or respiratory illness) at 400 µg/m ³		Martin (1964)
300 to 500 (0.11 to 0.19)	low	cross-sectional	increase in hospital admissions		Brasser (1967)
500 (0.19)	350(TSP)	cohort	worsening of health status in persons with chronic respiratory disease at 500 µg/m ³	peak levels much higher than 24-h averages	Lawther (1958), Lawther et al. (1970)
200 (0.077)	150(TSP)	cohort	increased prevalence of symptoms in bronchitics at 200 µg/m ³	no information on confounders such as cold, allergens	Cohen et al. (1972)
193 (0.07)	-	cohort	no adverse effect on bronchitics	small study group (18)	Emmerson (1973)
310 to 430 (0.12 to 0.17) (pollution episodes)	145 to 240(TSP)	cohort	increased prevalence of respiratory symptoms during pollution episodes in three areas of New York	variation in SES between two of the communities may be partially responsible	Cohen et al. (1974)
350 (0.135)	770(TSP)	cohort	decrease in FEV _{0.75} and FVC in children during and after pollution episode	no pre-alert lung function measurements for comparison	Stebbins et al. (1976)
350 (0.135)	770(TSP)	case-control	reduced lung function in children	analysis based on unvalidated methodology	Stebbins et al. (1979)
300 (0.12)	160(BS)	cross-sectional (1969)	increase in FEV _{1.0} and VC in urban community residents between 1969 and 1972; decrease in rural areas	suggested that improved lung function in urban residents related to improvement in air quality; however, results not confirmed in 1972 study	Van der Lende et al. (1975)
100 (0.04)	40(BS)	cross-sectional (1972)			
30 to 70 (0.012 to 0.027)	35 to 100(TSP)	cohort	frequency of respiratory symptoms in healthy subjects increased at 40 µg/m ³	poor response rate, poor monitoring data and over-interpretation of results	Stebbins and Hayes (1979)
74 to 179 (0.029 to 0.07)	-	cross-sectional	no association between prevalence of asthma attacks and pollution		Goldstein and Dulberg (1981)
0 to 183 (0 to 0.07)	-	cross-sectional	association between acute respiratory illness and pollution index and components SO ₂ and CoH	data from only one monitoring station; no control for SES or smoking	Levy et al. (1977)
4 to 369 (0.0015 to 0.142)	-	cross-sectional	weak correlation between SO ₂ and TSP and emergency room visits for respiratory diseases	authors conclude "Causality should be inferred with caution, given the potential limitations of the pollution data and analytical methods".	Samet et al. (1981)
14 to 141 (0.005 to 0.054)	-	case-control	no association between exposure and peak flow in asymptomatic women or women with chronic respiratory symptoms	cases and controls matched for residence and smoking	Schenker et al. (1983)
169 to 455 (0.065 to 0.175)	159 to 422(TSP)	cohort	decrease in pulmonary function with increasing TSP and SO ₂		Dockery et al. (1982)

* Modified from Health and Welfare Canada (1984)
TSP = Total Suspended Particulate
BS = Black Smoke
CoH = Coefficient of Haze

SES = Socioeconomic Status
FVC = Forced Vital Capacity
FEV_{0.75} = Forced Expiratory Volume in 0.75 seconds

TABLE 4 CLINICAL STUDIES*

SO ₂ Concentration µg/m ³ (ppm)	Exposure Duration (minutes)	Health Effects	Comments	Reference
780 to 2600 (0.3 to 1.0)	-	taste and odor threshold	immediate perception	Patty (1963)
1308 (0.5)	5	SRaw significantly increased in 6/6 subjects	exercising (5 min.) asthmatic subjects mouthpiece or facemask	Kirkpatrick et al. (1982)
1963 (0.75)	120	reversible changes in SRaw	exercising (15 min.) healthy males (1/2 with positive allergen skin tests)	Stacy et al. (1978)
523 to 1570 (0.20 to 0.60)	5	reversible changes in SRaw at 1047 µg/m ³	exercising (5 min.) asthmatic subjects	Linn et al. (1983)
1308 (0.5)	3	reversible changes in SRaw	asthmatic subjects; mouthpiece	Sheppard et al. (1983)
654 to 1308 (0.25 to 0.5)	60	no effect on pulmonary function	exercising (30 min.) asthmatic subjects	Linn et al. (1982)
654 to 2617 (0.25 to 1)	40	transient bronchoconstriction above 1963 µg/m ³	exercising (10 min.) asthmatic subjects	Schachter et al. (1984)
654 to 2617 (0.25 to 1)	40	mild respiratory symptoms related to lower airway dysfunction	exercising asthmatic subjects	Witek et al. (1984)
2617 to 13 085 (1 to 5)	10	significant increase in SRaw in asthmatics above 2617 µg/m ³ ; significant increase in SRaw at 13 085 µg/m ³ in normal subjects	mouthpiece	Boushey et al. (1984)
654 to 1308 (0.25 to 0.5)	10	significant increase in SRaw at 654 µg/m ³	exercising asthmatic subjects; mouthpiece	Boushey et al. (1984)

* Modified from Health and Welfare Canada (1984)
SRaw = Specific Airway Resistance

The results of available clinical studies of populations exposed to sulphur dioxide conducted prior to 1981 have been reviewed by Ericsson and Camner (1983). The results of more recent investigations are presented in Table 4.

In the clinical studies conducted to date, clear effects have not been observed in healthy subjects exposed for brief periods to concentrations of sulphur dioxide less than $2600 \mu\text{g}/\text{m}^3$ (1 ppm) (Ericsson and Camner, 1983; Lipfert, 1980; Kleinman, 1984). At levels greater than $2600 \mu\text{g}/\text{m}^3$ (1 ppm), increases in respiratory rate and airway and nasal airflow resistance and decreases in tidal volume, forced expiratory flow and the nasal mucociliary flow rate have been reported. At much higher levels ($>14000 \mu\text{g}/\text{m}^3$; >5.38 ppm) an increase in tracheobronchial clearance has been reported.

Exercising individuals and asthmatics are more sensitive to sulphur dioxide than healthy subjects at rest. However the levels at which sulphur dioxide causes adverse effects in these groups are less clear. There are variations in the reported results obtained to date that may be attributable to variations in the sensitivity of the study populations (which usually number less than 20); in experimental conditions (e.g., patterns of exposure and exercise, temperature and relative humidity); in routes of exposure (e.g., mouthpiece vs. natural breathing); or in the time course of manifestation of the effect. However, taking these factors into consideration, the following conclusions can be drawn based on the results of the clinical studies conducted to date:

1. There is only limited and as yet unconfirmed evidence that minor and transient effects in healthy exercising individuals occur at levels of SO_2 less than $2600 \mu\text{g}/\text{m}^3$ (1 ppm) (Stacy et al., 1978).
2. Natural breathing of sulphur dioxide can cause some asthmatics to experience symptoms and increased airway resistance at concentrations as low as $1000 \mu\text{g}/\text{m}^3$ (0.4 ppm), with sufficiently heavy exercise (Hackney et al., 1984). Increases in airway resistance have also been observed in exercising asthmatics exposed to low levels administered through a mouthpiece. These results, however, were obtained by unnatural means of exposure and are not considered to be relevant to the establishment of guidelines for air quality. In most subjects, the effects disappear in less than an hour with rest, even if sulphur dioxide exposure continues. There is also evidence that tolerance develops following repeated exposure of asthmatics to sulphur dioxide.
3. The health effects of exposure to sulphur dioxide have been enhanced by combined exposure with either water vapour, sulphuric acid aerosols (Kagawa, 1984) or "inert dust". In some studies, concomitant exposure to ozone has exacerbated effects of sulphur dioxide on pulmonary function; however, there is no clear convincing evidence of synergistic effects following exposure to mixtures of sulphur dioxide, ozone, and nitrogen dioxide (Ericsson and Camner, 1983).
4. The taste and odour threshold for SO_2 ranges between 780 and $2600 \mu\text{g}/\text{m}^3$ (0.30 and 1.0 ppm) (Patty, 1963).

populations exposed to air pollutants have not been confirmed in more recent and sensitive cohort studies (Van der Lende et al., 1981). There has also been inadequate control for confounding variables in several of the available studies, and exposure data for all of the investigations were limited and, possibly, unrepresentative (Table 6). However, based on examination of the acceptable epidemiological evidence for health effects associated with exposure to sulphur dioxide concentrations less than $1000 \mu\text{g}/\text{m}^3$ (0.39 ppm) (Lawther, 1958; Lawther et al., 1970; Martin, 1964; Samet et al., 1981) some tentative conclusions can be drawn. Increases in hospital admissions for cardiac or respiratory illness have been associated with 24-hour mean sulphur dioxide levels greater than $400 \mu\text{g}/\text{m}^3$ (0.15 ppm) (Martin, 1964), and weak associations have been observed between emergency room visits and levels of sulphur dioxide up to $369 \mu\text{g}/\text{m}^3$ (0.142 ppm) (24-hour mean) (Samet et al., 1981). Worsening of health status in bronchitics has been associated with exposure to sulphur dioxide levels greater than $500 \mu\text{g}/\text{m}^3$ (0.192 ppm) (24-hour average) (Lawther, 1958; Lawther et al., 1970). In some studies, there has been evidence of an association between adverse effects and exposure for short periods (24 hours) to lower levels of sulphur dioxide (Table 6). In all of these investigations, however, there has been inadequate control for confounding variables or other unacceptable limitations of study design. It should also be noted that in all of these studies in which adverse effects have been observed, levels of pollutants other than sulphur dioxide have also been elevated.

4.3.4 Effects of Long-term Exposure on Morbidity. In 1981, Ware et al. reviewed the results of observational studies of the effects of long-term exposure to sulphur oxides and particulate matter. Based on evaluation of the design of studies conducted up until that time, the authors concluded that six provided reliable evidence of the health effects of long-term exposure (Table 7). On the basis of these results, Ware et al. (1981) concluded that upper and lower respiratory symptoms were associated with total suspended particulate concentrations greater than $180 \mu\text{g}/\text{m}^3$. These studies, however, provided little evidence to assess the health effects associated with elevated sulphur dioxide concentrations in the presence of moderate levels of particulate matter.

It should also be noted that five of the six studies chosen by Ware et al. (1981) as a basis for assessing the effects of long-term exposure to sulphur dioxide and particulate were cross-sectional in design. The limitations of such investigations have been discussed with respect to short-term exposure; however, these shortcomings are more serious when considering effects of chronic exposure. Exposure in such studies is

TABLE 7 EFFECTS OF LONG-TERM EXPOSURE ON MORBIDITY*

SO ₂ µg/m ³ (ppm)	Annual Average Particulate (µg/m ³)	Study Type	Health Effect	Comment	Reference
225 (0.086)	360	cross-sectional (four areas)	increased frequency of respiratory symptoms; decreased lung function in five-year-olds		Ferris (1978); Lunn et al. (1970)
100 (0.038)	200	cross-sectional across Britain	increased prevalence of respiratory symptoms		Lambert and Reid (1970)
125 (0.048)	270	cross-sectional (two areas)	more chronic bronchitis and asthmatic disease in smokers; reduced FEV	judged by Ware et al. (1981) to provide reliable evidence of health effects of long-term exposure	Sawicki and Lawrence (1977)
125 (0.048)	285	cross-sectional (four areas)	increased history and symptoms of respiratory illness		Rudnik (1977)
55 (0.060)	180	longitudinal and cross-sectional	higher rate of respiratory symptoms; and decreased lung function		Ferris and Anderson (1962)
<25 (0.01)	135	cross-sectional (two areas)	increased frequency of acute lower respiratory disease		Hammer (1976)
70 to 80 (0.027 to 0.031)		cohort	increase in ARI and reduced FEV in children	several confounders (including parental smoking) taken into account; indoor levels measured; small cohort	Saric et al. (1981)
77.5 (0.03)		cohort	increase in respiratory symptoms and reduced airway flows in children	inadequate control for confounders; inadequate statistical analysis	Mostardi et al. (1981)
48 (0.018)	28	cohort	increased prevalence of cough in children	small cohort; low response rate (70%) and significant loss to follow-up (35%)	Dodge (1983)
56 to 104 (0.02 to 0.04)	53 to 83	cross-sectional	SO ₂ significant risk factor for serious chest illness before age 2 and for chest illness >3 days in lifetime residents	several confounders taken into account	Schenker et al. (1982)
12 to 114 (0.0046 to 0.044)	8 to 51	cross-sectional and longitudinal	no association between SO ₂ and respiratory illness in children	several confounders taken into account; some data on indoor exposure	Melia et al. (1981a); Melia et al. (1981b)
10 to 120 (0.004 to 0.046)		longitudinal (four areas)	no association of ARI in children with SO ₂	several confounders (including parental smoking) taken into account	Love et al. (1982)
21.6 to 51.4 (0.008 to 0.020)		longitudinal (two areas)	ARI incidence significantly higher in children in com- munities with higher levels of SO ₂	particulate levels also high in "high exposure" communities; indoor exposure considered	Love et al. (1981)
62 to 99 (0.024 to 0.038)		cross-sectional	no increase in prevalence of respiratory symptoms in adult women at concentrations up to 100 µg/m ³ with the exception of wheeze	several confounders including SES and smoking taken into account	Schenker et al. (1983)
<5 to >15 (0.0019 to 0.0058)		cross-sectional	no evidence that spontaneous abortions are associated with SO ₂	confounding factors such as previous reproductive history not considered; no exposure data for gest- ation period	Hemminki and Niemi (1982)
139 to 229 (0.053 to 0.088)		longitudinal (two areas)	significantly greater decline in VC and FEV with increasing age	confounding factors such as smoking taken into account; however, SO ₂ and smoke sometimes accompanied by high oxidant levels	Van der Lende et al. (1981)
133.6 (0.05)		cross-sectional (four areas)	significantly higher percentage of persons with an FVC or FEV ₂₅ to 75% below 50% of expected	SES not considered in analysis	Detels et al. (1981)

* Modified from Health and Welfare Canada (1984)
FEV = Forced Expiratory Volume
ARI = Acute Respiratory Illness

SES = Socioeconomic Status
VC = Vital Capacity

4 EFFECTS OF SULPHUR DIOXIDE ON HUMAN HEALTH

4.1 Absorption, Distribution and Excretion

Sulphur dioxide is a very soluble gas which reacts with water to form sulphurous acid which readily dissociates into hydrogen ions, sulphite ions, and bisulphite ions. Sulphites and bisulphites formed following absorption of SO₂ are converted to inorganic sulphates which are readily excreted in the urine.

Exposure to SO₂ stimulates the irritant receptors in the nose, nasopharynx and bronchi, if the gas reaches the bronchi, with resultant naso-constriction, broncho-constriction and increased mucus secretion. These reflex reactions enable the gas to be localized in the upper respiratory passages, especially the nose, where it is absorbed; increased mucus secretion facilitates the absorption of the highly soluble gas, thereby decreasing its irritant effects.

Animal studies indicate that at exposure concentrations of 20 ppm or more, 80 to 90% of the nasally breathed SO₂ is absorbed in the nose. If the nose is bypassed, these concentrations reach the trachea and major bronchi. If concentrations are low (e.g., 1 ppm or less) stimulation of the receptors in the upper respiratory tract is not as great and a larger percentage of the inhaled gas reaches the bronchi where receptors are stimulated and reflex broncho-constriction follows. This decreases the opportunity for the gas to descend further into the respiratory passages.

Studies on human subjects have shown that sulphur dioxide is almost completely absorbed between the nose and the pharynx. However, due to a variety of gas-to-particle conversions (e.g., to sulphates) or gas-particle chemical reactions, sulphur dioxide may also be carried deeper into the respiratory tract. Absorption is significantly greater during nose breathing than mouth breathing (95% vs. 70%) especially with increased ventilation, since the moist surfaces of the nasal cavity scrub sulphur dioxide effectively.

4.2 Clinical Studies

The results of controlled, clinical studies in human populations are the most reliable for derivation of exposure-response relationships as a basis for air quality standards. However, such studies are restricted to examination of mild, temporary effects of short-term exposures in a limited number of subjects.

TABLE 3 POLLUTANT COMBINATIONS WHICH CAUSED SYNERGISTIC RESPONSES IN VEGETATION

Pollutant Combination (ppm)	Exposure Period (h)	Plant Injured	Reference
0.02 to 0.03 SO ₂ and 0.008 to 0.01 O ₃	4 to 5	peanut	Applegate and Durrant (1969)
0.025 SO ₂ and 0.05 O ₃	6	eastern white pine	Houston (1974)
0.10 SO ₂ and 0.03 O ₃	4	tobacco	Heck (1968)
0.10 SO ₂ and 0.10 O ₃	4	horticultural crops	Tingey et al. (1973)
0.10 SO ₂ and 0.10 NO ₂	4	horticultural crops	Tingey et al. (1971)
0.20 SO ₂ and 0.05 O ₃	3	trembling aspen	Karnosky (1976)
0.24 SO ₂ and 0.027 O ₃	2	tobacco	Menser and Heggstad (1966)

the ambient air; therefore, it is possible that plant effects observed in the field which have been attributed to individual pollutants may have been caused by gaseous mixtures.

commonly represented by arithmetic average concentrations during the study period, which may be misleading when concentrations of pollutants have changed substantially during the years preceding the study.

In the past several years there have been improvements in the design of studies to examine the effects of long-term exposure to air pollutants such as sulphur dioxide. Nevertheless, each of the studies reviewed in Table 7 has one or more limitations, and none is ideal for assessment of the effects of long-term exposure to sulphur dioxide. However, two studies (Van der Lende et al., 1981; Saric et al., 1981) are considered to be most reliable for evaluation based primarily on the following criteria:

1. longitudinal study design;
2. adequate control of appropriate confounding factors; and
3. some attempt to take individual variations in exposure into account (e.g., area of residence, gas stove use).

The SO₂ levels at which effects were observed in these studies are shown in Table 8.

TABLE 8 LEVELS OF SULPHUR DIOXIDE AT WHICH EFFECTS WERE OBSERVED

SO ₂ Level (annual average) µg/m ³ (ppm)	Effect	Comment	Reference
70 to 80 (0.027 to 0.031)	increase in acute respiratory illness (ARI) and effects on pulmonary function in children	small cohort (N=117)	Saric et al. (1981)
139 to 339 (0.053 to 0.088)	significantly greater decrease in VC and FEV ₁ with increasing age	cohort size N=1500 to 2000	Van der Lende et al. (1981)

It is not possible to attribute the effects observed in these studies to sulphur dioxide alone, since in all cases, levels of particulate matter and other pollutants were elevated. Based on review of the available data from observational studies, therefore, it was concluded that effects on pulmonary function and increases in respiratory disease in children and adults have been observed in populations exposed to mixtures of pollutants, where the concentration of sulphur dioxide exceeds approximately 100 µg/m³ (annual average).

4.4 Sulphur Dioxide and Cancer

It has been postulated that SO₂ plays a role in the development of cancer, particularly pulmonary cancer. There is, however, no clear evidence from studies conducted to date in human and animal populations that sulphur dioxide is in itself carcinogenic.

In Celna, Czechoslovakia, Materna et al. (1969) reported moderate chronic injury to foliage of spruce trees under the influence of an average SO₂ concentration of 50 µg/m³ (0.019 ppm) which occurred during 1966 and 1967. The authors stressed the importance of both long-term concentrations of SO₂ and other environmental factors in producing injury to plants.

Epiphytic lichens, which are perennial and evergreen, are extremely sensitive to SO₂ because they are continually exposed to the gases in a polluted environment. In studies conducted on the occurrence of lichens at Sudbury, (Leblanc et al., 1972) the number of epiphytes found growing on *Populus balsamifera* trees was drastically reduced in zones where the growing season mean levels of SO₂ were over 52 µg/m³ (0.02 ppm), and slightly reduced in zones where the mean levels of SO₂ were over 26 µg/m³ (0.01 ppm). Similarly, in Sweden (Skye, 1964) it was found that the survival of lichens was less in areas with an annual SO₂ concentration of approximately 39 µg/m³ (0.015 ppm) and in the Tyne Valley, England (Gilbert, 1969) species diversity of lichens was reduced when the annual average concentration of SO₂ was above 42 µg/m³ (0.016 ppm).

3.3 Effects of Sulphur Dioxide in Combination with Other Air Pollutants

Rarely is plant life in nature exposed to the influence of only one air pollutant. The bulk of an emission from a particular industrial source may comprise only one pollutant, but other pollutants may also be present. It is important to know the concentrations of various phytotoxic pollutants in the atmosphere and the effects on plant life from gas mixtures. Some controlled environment research has been conducted in which plants have been subjected to combinations of SO₂ and ozone (O₃), SO₂ and nitrogen dioxide (NO₂), and SO₂ and hydrogen fluoride (HF). Results obtained have been classified as additive (equal to the sum of the effects of the individual pollutants); synergistic (greater than the additive effects); or antagonistic (less than the additive effects).

Very low concentrations of SO₂ for periods of a few hours have been found to cause foliar injury to sensitive plants when combined with another air pollutant. The same concentrations of SO₂ alone, however, were either not toxic or caused minimal injury. Some of the low concentration pollutant combinations for short time periods which caused synergistic effects on exposed plants are shown in Table 3 (Linzon, 1978). These experiments indicate that a low concentration of 0.10 ppm SO₂ in combination with either O₃ or NO₂ for periods of four hours can injure a wide variety of plants. The pollutant levels and time periods utilized in these experiments occur quite frequently in

5 DISCUSSION

In formulating air quality standards for SO₂ for a particular jurisdiction, many factors must be taken into consideration. Foremost are the scientific criteria which describe the effects of SO₂ for given concentrations and time periods on exposed receptors. These criteria are generally assembled from information published in scientific and technical reports. The published data may be based on effects in the field caused by ambient atmospheric concentrations of SO₂ or from experiments conducted under controlled environmental conditions.

Generally, the effects of SO₂ observed in the field, under natural conditions, provide the best basis for interpretation. Difficulties are encountered in attempting to extrapolate the results of experimental artificial fumigations to natural ecosystems. The reasons for the difficulties are that the varying environmental conditions which occur out-of-doors are almost impossible to duplicate in experimental fumigations. The temperature, relative humidity, light quantity and quality, wind, soil moisture, receptor responsiveness, SO₂ concentrations, and presence of other air pollutants are continuously fluctuating in the field at the site of the receptor-environment interface.

Despite these difficulties, published data on artificial fumigation work indicated that effects on plant life were caused by some extremely low SO₂ doses. Conversely, the literature showed that some plants were notably resistant and could tolerate very high concentrations of SO₂. Examination of Table 2 showed injury occurring on extremely sensitive strains of eastern white pine at a dose as low as 78 µg/m³ (0.03 ppm) SO₂ for one hour, whereas 7800 µg/m³ (3.0 ppm) for one hour was required to cause minimal injury to *Acacia pruinosa*. Many jurisdictions have an air quality standard for SO₂ close to 7800 µg/m³ (0.30 ppm) for one hour which is ten times higher than one of the lowest dose-responses reported and ten times lower than one of the highest dose-responses reported.

In evaluating published dose-response data, the reviewer must use judgement in determining the quality and significance of the reported work. A number of questions must be answered: can results of the experiment be repeated; were the plants established for a sufficient period of time to overcome the shock of transplanting or grafting; and, was the SO₂ monitoring instrument reliable and calibrated?

Other factors to be considered by administrators in establishing air quality standards for SO₂ are somewhat beyond the scientific criteria stage. Regional, economic, and social factors may be important considerations. What degree of effects is a

TABLE 2 DEGREE OF FOREST EFFECTS AT VARIOUS DISTANCES FROM SUDBURY SMELTERS¹

Forest Sampling Station ¹ (distance and direction from Sudbury)	Trees with One-Year-Old Foliage Injured			Trees with Two-Year-Old (1961) Foliage		Net Annual Average Gain or Loss in Total Volume 1953 to 1963 (%)	Annual Average Mortality 1953 to 1963 (%)	Degree of SO ₂ Damage	Average SO ₂ Concentration for Total Measurement Period 1954 to 1963 ² µg/m ³ (ppm)
	Trees with Current Year's Foliage Injured in August 1963 (%)	June 1963 (%)	Aug. 1963 (%)	Injured in June 1963 (%)	Lacking in Aug. 1963 (%)				
30 km NE (West Bay of Lake Wanapitei)	2.0	38.0	77.9	96.0	20.6	-1.3	2.6	acute and chronic injury	117 (0.045)
40 km NE (Portage Bay of Lake Wanapitei)	1.1	21.5	55.6	77.0	15.2	-0.5	2.5	mostly chronic and little acute injury	44 (0.017)
64 to 69 km NE (Grassy Lake to Emerald Lake)	0.4	2.5	16.7	37.5	9.1	+1.8	1.4	no acute injury; very slight chronic injury	21 (0.008)
150 km W (Lake Matinenda)	0.6	0.3	2.1	10.1	3.9	+2.1	0.5	control no SO ₂ injury	2.6 (0.001) ³ (Sturgeon Falls)
Correlation Coefficient (r)	0.96*	0.96*	0.93**	0.90**	0.94**	0.90**	0.81		

¹ Linzon (1971)
² Dreisinger (1965)
³ Data for 5-month growing season - 1971

*p < 0.05
**p < 0.10

jurisdiction willing to accept? If a jurisdiction is willing to accept some adverse effects, then the air quality standards can be set above threshold levels. If a jurisdiction desires to safeguard the environment and other receptors against any effects, then the air quality standard will have to be set below threshold levels.

Air quality standards for sulphur dioxide of various agencies around the world are listed in Table 9. Most of the agencies have a one-hour, short-term standard with a median value of approximately $900 \mu\text{g}/\text{m}^3$ (0.34 ppm). Several investigators have reported acute injury to vegetation in this range or at somewhat higher values.

The majority of the agencies also have a long-term standard for one year with a median value of about $60 \mu\text{g}/\text{m}^3$ (0.02 ppm).

In addition, a few agencies have a standard for a twenty-four hour period, the median value being about $300 \mu\text{g}/\text{m}^3$ (0.11 ppm).

alter the delicate balance of the ecological system. This alteration may be beneficial or deleterious depending on the magnitude of the addition and the state of the receptor.

Smith (1974) distinguished three major categories of forest ecosystem effects arising from exposure to various concentrations of atmospheric pollutants. In Class I, under conditions of low dosage, the vegetation and soils of forest ecosystems may act as a sink for contaminants with no detectable effects occurring except an increase in nutrient levels, accompanied possibly by a stimulatory (fertilizing) effect. In Class II, under conditions of intermediate dosage, individual trees may be adversely or subtly affected by nutrient stress, reduced photosynthetic rate, and predisposition to entomological or microbial organisms. In Class III, under conditions of high dosage, acute morbidity or mortality of specific trees occurs which may seriously alter the structure and function of the forest ecosystem.

By the 1950s, in the Sudbury area of Ontario, three large nickel and copper smelters were discharging approximately 5443 t/d of SO_2 into the surrounding atmosphere. Forest effects in the area were excessive with severe injury on trees occurring up to 40 km northeast of Sudbury. Based on studies of over 6000 white pine trees on 42 sample plots during a 10-year period, the Sudbury area of Ontario was segregated into three fume zones: inner, intermediate, and outer (Linzon, 1966). In the inner fume zone, an area of about 1840 km^2 , white pine trees displayed acute and chronic foliar injuries which resulted in reduced radial and volume growth and excessive tree mortality. In the intermediate fume zone, an area of about 4100 km^2 , some chronic SO_2 injury was present, while in the outer fume zone, atmospheric contamination was too dilute to cause visible injuries. The inner, intermediate, and outer fume zones may be compared to the three categories of forest ecosystems effects distinguished by Smith (1974): Class III (high dosage); Class II (intermediate dosage); and Class I (low dosage).

Table 2 shows the results of forest studies conducted over a 10-year period (1953 to 1963) in the area near Sudbury affected by sulphur-fumes (Linzon, 1971). Very little acute injury to the current year's needles occurred in the forests northeast of Sudbury. The greatest increase in foliar injuries during the growing season occurred on one-year-old needles. The continuous development of injuries on the older needles was reflected in early abscission of the oldest foliage, reduced radial and volume growth, and premature death of the trees. It is apparent that chronic effects on forest growth were prominent where SO_2 air concentrations averaged $44 \mu\text{g}/\text{m}^3$ (0.017 ppm), the arithmetic mean for the total 10-year measurement period, and chronic effects were slight where SO_2 annual concentrations averaged $21 \mu\text{g}/\text{m}^3$ (0.008 ppm).

cases the minimum found by the investigators to cause injury. Higher concentrations of SO₂, or longer periods of time, usually caused more severe effects. In some studies, however, the concentrations and the exposure periods were arbitrarily selected for the purposes of the experiment, and although injury occurred, the SO₂ doses reported were not necessarily threshold levels for the plant species tested.

3.2 Chronic Effects

Chronic injury appears as a yellowing or chlorosis of broad leaves. In perennial conifers, chronic injury affects older needles and appears as a yellowish-green colour, then changes to reddish-brown, starting at the tips and developing towards the base (Linzon, 1969). The rate of metabolism is reduced in levels displaying chronic injury.

Chronic injuries develop slowly on coniferous perennial foliage. The greatest increase in injuries has occurred on the one-year old needles of eastern white pine trees in the Sudbury area from the effects of sulphur-fumes. Continued chronic injury to perennial foliage of coniferous trees results in premature needle abscission, reduced radial and volume growth, and early death (Linzon, 1971).

Long-term chronic effects on trees are related to a variety of SO₂ exposures, which include: short-term high concentrations; short-term and long-term sublethal concentrations; and SO₂-free periods in which time the plant life can recuperate by translocating and assimilating accumulated sulphur. To correlate the chronic effects on forests with atmospheric SO₂ levels, it is preferable to use the average concentration for the total period of exposure, rather than the average of SO₂ fumigation periods only. The frequency of intermittent fumigation at any particular location is unpredictable. Chronic effects develop slowly, and the response of the receptor is influenced by other environmental factors in addition to SO₂. The vegetation affected is exposed throughout the entire growing season to the vagaries of the environment (sun, rain, wind, and drought) all of which have an influence on plant response to SO₂.

Generally, the effects of SO₂ observed in the field, under natural conditions, provide the best basis for interpretation of dose-response criteria, since difficulties are encountered in attempting to extrapolate the results from experimental artificial fumigations to natural ecosystems.

Atmospheric SO₂ may affect a forest ecosystem in various ways. Forest communities have evolved and become established through the selective, environmental pressures. The addition of a new pressure, such as increased atmospheric sulphur, can

TABLE 9 AIR QUALITY CRITERIA, OBJECTIVES, AND STANDARDS FOR SULPHUR DIOXIDE

Agency		SO ₂ Concentration (µg/m ³) (ppm)		Averaging Time	
1. Canada	National	Acceptable	900 (0.34) 300 (0.11) 60 (0.02)	1-hour 24-hour 1-year	
		Desirable	450 (0.17) 150 (0.06) 30 (0.01)	1-hour 24-hour 1-year	
			Tolerable	800 (0.30)	24-hour
	Alberta		525 (0.19) 450 (0.17) 150 (0.06) 30 (0.01)	30-min. 1-hour 24-hour 1-year	
		British Columbia	- Level "A"	450 (0.17) 375 (0.14) 160 (0.06) 25 (0.01)	1-hour 3-hour 24-hour 1-year
			- Level "B"	900 (0.34) 665 (0.25) 260 (0.10) 75 (0.03)	1-hour 24-hour 3-hour 1-year
				Ontario	690 (0.25) 275 (0.10) 55 (0.02)
	Quebec				1310 (0.50) 228 (0.09) 52 (0.02)
		(N.B. All other Provinces follow Canada's air quality objectives.)			
		2. U.S.A.	National	Secondary	1300 (0.50)
	Primary			365 (0.13) 80 (0.03)	24-hour 1-year
				California	1300 (0.50)
	Florida		260 (0.10)	1-hour	
	Missouri		665 (0.25)	1-hour	
	Montana		665 (0.25) 60 (0.02)	1-hour 1-year	
			New York	665 (0.25)	1-hour
	Vermont		260 (0.10) 60 (0.02)	1-hour 1-year	
			3. Argentina	75 (0.03)	30-days
4. Belgium	150 (0.06)		1-year		
5. Columbia	75 (0.03)		1-year		
6. Denmark	750 (0.30)	30-min.			
7. Finland	750 (0.30) 75 (0.03)	30-min. 1-year			
	8. Italy	750 (0.30)	30-min.		
9. Japan	260 (0.10) 100 (0.04)	1-hour 24-hour			
	10. Switzerland	30 (0.01)	Annual		
11. West Germany	750 (0.30)	30-min.			
12. W.H.O.	- Acceptable (98% of 1-hour averages <200 µg/m ³)	60 (0.02)	1-year		
	- Desirable (98% of 1-hour averages <120 µg/m ³)	40 (0.015)	1-year		
13. Commission of European Communities:					
Levels of total suspended particulate (TSP):					
	TSP >40 µg/m ³	80 (0.03)	1-year		
	TSP ≤40 µg/m ³	120 (0.04)	1-year		
	TSP >60 µg/m ³	130 (0.05)	Winter (Oct. 1-Mar. 31)		
	TSP ≤60 µg/m ³	180 (0.06)	Winter (Oct. 1-Mar. 31)		
	TSP >150 µg/m ³	250 (0.10)	24-hour		

6 RATIONALE AND RECOMMENDATIONS

Establishing a satisfactory annual average of SO₂ is important for the protection of all receptors, including man, animals, vegetation, water, and fish. An adequate annual average air quality standard for SO₂, combined with an appropriate short-term standard, will help to prevent:

- 1) chronic forest damage which includes accumulation of excess sulphur in perennial foliage on trees, progressively increasing injuries to older foliage, early abscission of older foliage, reduced radial and volume of growth of trees, and premature tree mortality;
- 2) killing of lichens and changes in the species of lichen populations in sulphur-polluted areas (lichens provide fodder for reindeer and caribou); and,
- 3) adverse effects on lake quality and fish populations caused by long range transport of SO₂ which contributes to acid precipitation.

The evidence to date indicates that acute injury to native vegetation does not occur below 1820 µg/m³ (0.70 ppm) of SO₂ for one hour or 468 µg/m³ (0.18 ppm) of SO₂ for exposures of eight hours. However, acute injury may occur if SO₂ persists for several hours at concentrations over 650 µg/m³ (0.25 ppm). Prominent chronic injury or slight chronic injury to natural forests may occur from average concentrations of SO₂ as low as 44 µg/m³ (0.017 ppm) or 21 µg/m³ (0.008 ppm), respectively, over entire growing seasons in which the SO₂ fumigations are of variable intensities (Linzon, 1978).

The American Industrial Hygiene Association (AIHA) (1970) cited a paper (Patty, 1963) which found that SO₂ can be detected by taste and odour at a range from 780 to 2600 µg/m³ (0.30 to 1.0 ppm). Thus the threshold for very sensitive individuals is 780 µg/m³.

The available studies indicate that relatively high concentrations of SO₂ can be tolerated by humans under experimental conditions. In reliable clinical studies, the lowest level at which effects have been observed is 1000 µg/m³ (0.38 ppm). Exposure to this concentration for one hour caused reversible effects on pulmonary function in exercising asthmatics.

Increased mortality rates have been reported in association with 24-hour SO₂ concentrations of 1000 µg/m³ or higher. The contribution made to daily mortality rates by lower concentrations of SO₂ is less well defined. Some studies suggest an increasing contribution with increasing SO₂ concentrations in excess of 500 µg/m³; however, a threshold for this effect has not been adequately defined. Increased morbidity in

TABLE 1 DOSE-RESPONSE TO SULPHUR DIOXIDE — ACUTE EFFECTS

SO ₂ Concentration µg/m ³ (ppm)*	Exposure Period (h)	Plant Response	Reference
A - Response to Low SO₂ Doses			
78 (0.03)	1	injury to sensitive eastern white pine	Costonis (1971)
65 (0.025)	6	injury to sensitive eastern white pine	Houston (1974)
130 to 312 (0.05 to 0.12)	4 to 8	injury to peanut	Applegate and Durrant (1969)
B - Response to Medium SO₂ Doses			
650 (0.25)	1	injury to begonia	Metcalfe (1941)
650 (0.25)	1	injury to eastern white pine	Berry (1967)
520 (0.20)	2	injury to several Kentucky bluegrass cultivars	Murray et al. (1975)
650 (0.25)	2	injury to eastern white, red, and jack pines	Berry (1971)
650 (0.25)	2	injury to virginia, short-leaf, slash, and loblolly pines	Berry (1974)
650 (0.25)	4	injury to broccoli	Tingey et al. (1973)
910 (0.35)	3	injury to trembling aspen	Karnosky (1976)
1404 (0.54)	3	injury to mountain ash	Spierings (1967)
1716 (0.66)	1	injury to buckwheat	Zimmerman and Crocker (1934)
2470 (0.95)	1	injury to foliage of forest trees**	Dreisinger (1965)
1430 (0.55)	2		
910 (0.35)	4		
650 (0.25)	8		
1820 (0.70)	1	injury to forest trees under sensitive environmental conditions**	Dreisinger and McGovern (1970)
1040 (0.40)	2		
676 (0.26)	4		
468 (0.18)	8		
780 (0.30)	8	injury to western larch	Katz and McCallum (1939)
364 (0.14)	12	injury to Douglas fir	
C - Response to High SO₂ Doses			
5200 (2.0)	2	injury to 10 of 87 native desert species	Hill et al. (1973)
5200 (2.0)	2	injury to Chinese elm	Temple (1972)
5200 (2.0)	4	injury to 10 weed species	Benedict and Breen (1955)
6500 (2.5)	3	injury to 7 of 16 chrysanthemum var.	Brennan and Leone (1972)
7800 (3.0)	1	injury to <i>Acacia pruinosa</i>	O'Connor, Parbery and Strauss (1974)

* 1 ppm = 2600 µg/m³ at 25°C and 101 kPa.

** field observations, other references report plant responses observed in artificial fumigation experiments.

Note: Not all the literature on SO₂ dose/response is cited in this table, but pertinent references have been included.

Acute injury to forests has been attributed to the following SO₂ doses: 2470 µg/m³ (0.95 ppm) for one hour, 1430 µg/m³ (0.55 ppm) for two hours, 910 µg/m³ (0.35 ppm) for four hours, and 650 µg/m³ (0.25 ppm) for eight hours (Dreisinger, 1965). For acute injury to occur, other environmental and plant factors are important including: sunlight, moderate temperature, high relative humidity, adequate soil moisture, and plant genotype and stage of growth. If these factors are not conducive to injury, the plants will escape harm even in the presence of doses two or three times higher than those previously mentioned. Conversely, if the predisposing factors are especially conducive to plant injury, the SO₂ doses could be reduced by about 25% to 1820 µg/m³ (0.70 ppm) for one hour, 1040 µg/m³ (0.40 ppm) for two hours, 676 µg/m³ (0.26 ppm) for four hours, and 468 µg/m³ (0.18 ppm) for eight hours (Dreisinger and McGovern, 1970).

In a one-hour experimental exposure, Zimmerman and Crocker (1934) reported that 1716 µg/m³ (0.66 ppm) SO₂ injured buckwheat (*Fagopyrum esculentum*). Karnosky (1976) produced acute injury on foliage of trembling aspen (*Populus tremuloides*) in artificial fumigations with 910 µg/m³ (0.35 ppm) SO₂ for a period of three hours. Metcalfe (1941) reported damaging begonia varieties in fumigations of 650 µg/m³ (0.25 ppm) SO₂ for one hour under very humid conditions. Berry (1967) reported injuring foliage of eastern white pine (*Pinus strobus*) at a concentration of 650 µg/m³ (0.25 ppm) SO₂ in two hour exposure periods. Murray et al. (1975) induced moderate to severe injury on several Kentucky bluegrass cultivars (*Poa pratensis*) in artificial fumigations of 520 µg/m³ (0.20 ppm) SO₂ for two hours.

These acute effects may be considered responses to medium doses of SO₂. The literature reports plant responses to much lower SO₂ doses and to substantially higher SO₂ doses. For example, in artificial fumigation experiments, SO₂ doses as low as 78 µg/m³ (0.03 ppm) for one hour (Costonis, 1971) and 65 µg/m³ (0.025 ppm) for six hours (Houston, 1974) have been reported to injure extremely sensitive strains of eastern white pine. Doses as high as 7800 µg/m³ (3.0 ppm) for one hour (O'Connor et al., 1974), 5200 µg/m³ (2.0 ppm) for two hours (Hill et al., 1973) and 6500 µg/m³ (2.5 ppm) for three hours (Brennan and Leone, 1972) have been reported to cause injury to tolerant plant species such as *Acacia pruinosa*, several native desert plant species, and several chrysanthemum varieties, respectively. Interpretation of results using very low or very high levels of SO₂ in short-term artificial fumigations, requires careful consideration of the experimental conditions.

Sulphur dioxide concentrations that caused acute injury to vegetation during short exposure periods are listed in Table 1 (Linzon, 1978). These doses were in many

bronchitics occurs when the SO₂ concentration exceeds 500 µg/m³ and hospital admissions increase when the concentration exceeds 300 to 500 µg/m³.

The available studies on the long-term chronic effects of exposure to air pollution indicate an increased incidence of morbidity in children when the annual average SO₂ concentrations exceed 100 µg/m³. The recommended maximum acceptable objectives for SO₂, based on this information, are (Table 10):

- 1) a 900 µg/m³ (0.34 ppm) average for one hour will help prevent taste and odour discomfort to sensitive individuals. In conjunction with the lower, recommended limit for 24 hours, it will also prevent the persistence of concentrations of SO₂ for several hours that have the potential to injure vegetation;
- 2) a 300 µg/m³ (0.11 ppm) average for 24 hours in conjunction with low suspended particulate matter in air will help prevent health effects; and
- 3) a 60 µg/m³ (0.02 ppm) average for one year will minimize the occurrence of chronic effects in natural forests.

Maximum desirable limits for SO₂ are shown in Table 11.

TABLE 10 MAXIMUM ACCEPTABLE LIMITS FOR SULPHUR DIOXIDE (SO₂)

Maximum Acceptable Limits:	1-hour avg. 900 µg/m ³ (0.34 ppm) 24-hour avg. 300 µg/m ³ (0.11 ppm) 1-year avg. 60 µg/m ³ (0.02 ppm)
Criteria:	
650 µg/m ³ (0.25 ppm) average for one hour artificially injured begonia	Metcalfe (1941)
520 µg/m ³ (0.20 ppm) average for two hours artificially injured several bluegrasses	Murray et al. (1975)
910 µg/m ³ (0.35 ppm) average for three hours artificially injured trembling aspen	Karnosky (1976)
650 µg/m ³ (0.25 ppm) average for four hours artificially injured broccoli	Tingey et al. (1973)
Injury to natural forest trees by:	
1820 µg/m ³ (0.70 ppm) average for one hour 1040 µg/m ³ (0.40 ppm) average for two hours 676 µg/m ³ (0.26 ppm) average for four hours 468 µg/m ³ (0.18 ppm) average for eight hours	Dreisinger and McGovern (1970)
780 µg/m ³ (0.30 ppm) threshold of taste and odour for SO ₂ in sensitive individuals	AIHA (1970); Patty (1963)
300 µg/m ³ (0.11 ppm) average for 24 h in combination with suspended particulate matter increased hospitalization	Brasser et al. (1967)
44 µg/m ³ (0.017 ppm) average for growing season resulted in prominent chronic effects to natural forests	Linzon (1978)

TABLE 11 MAXIMUM DESIRABLE LIMITS FOR SULPHUR DIOXIDE (SO₂)

Maximum Desirable Limits:	1-hour avg. 450 µg/m ³ (0.17 ppm) 24-hour avg. 150 µg/m ³ (0.06 ppm) 1-year avg. 30 µg/m ³ (0.01 ppm)
Criteria:	
	nil background values
	at or below threshold levels of health effects
	slight chronic effects to natural forests at 21 µg/m ³ (0.008 ppm) average for growing season (Linzon, 1978)

3 EFFECTS OF SULPHUR DIOXIDE ON VEGETATION

The first visible evidence of SO₂ injury to plants is discernible in the foliage; the stems, buds, and reproductive parts of plants are visibly more resistant. It is important to stress the difference between the acute, chronic, and subtle effects of SO₂ on plant life. Sulphur dioxide enters leaves mainly through the stomata and is toxic to the metabolic processes taking place in the mesophyll cells (Linzon, 1972). Acute injury is caused by a rapid accumulation of bisulphite and sulphite. When the oxidation product, sulphate, accumulates beyond a threshold value that the plant cells can tolerate, chronic injury occurs. It is estimated that sulphate is about 30 times less toxic than sulphite (Thomas, 1951).

Definitions vary for acute and chronic injury and for subtle effects caused by air pollutants. To avoid different interpretations of these terms with respect to SO₂, the following definitions apply (Linzon, 1978):

Acute injury - is macroscopic necrotic injury to plant tissue visible within hours or days following exposure to short-term (less than 24 hours) high concentrations of SO₂;

Chronic injury - is macroscopic chlorotic injury (sometimes changing to necrotic injury) to plant tissues usually developing over a long period (from over one day to one or more years) of exposure to varying concentrations of SO₂; and

Subtle effects - are measured physiological or biochemical changes, and/or reductions in plant growth or yield in the absence of macroscopic injury.

3.1 Acute Injury

Acute injury to broad leaves appears as lesions on both surfaces, usually occurring in a local area between veins, and often more prominent towards the petiole. The metabolic processes are completely disrupted in the dead or necrotic areas, with the surrounding green tissue remaining functional. The tissue immediately adjacent to the veins is extremely resistant. In some cases, injury can occur on leaf margins. Young leaves rarely display necrotic markings, whereas fully expanded leaves are most sensitive to acute SO₂ injury. The oldest leaves are moderately sensitive. In monocotyledonous leaves the injury can occur at the tips and in lengthwise areas between the main veins. In conifers, acute injury usually appears as a bright orange-red tip necrosis on current-year needles, often with a sharp line of demarcation between the injured tips and the normally green bases. Occasionally the injury may occur as bands at the tip, middle, or base of the needles (Linzon, 1972).

2 PROPERTIES AND OCCURRENCE OF SULPHUR DIOXIDE

Sulphur is an essential element for plants. It is a constituent of the amino acids cystine, cysteine, and methionine, which are some of the building blocks of plant proteins. It is also a constituent of the plant vitamins thiamine and biotin, and of other biochemical constituents such as: glutathione, coenzyme A, and cytochrome C. Normally, sulphur is taken up by plants from soil in the sulphate form and assimilated into various compounds (usually after being chemically reduced). Sulphur dioxide absorbed from the air has been shown to rapidly undergo oxidation to sulphates inside plant tissues. It is apparent that sulphur in its various forms is necessary in the intermediary metabolism of plants.

Background levels of sulphur dioxide in the atmosphere are $4 \mu\text{g}/\text{m}^3$ or less and there is no evidence that these levels can cause harm to the environment (Georgii, 1970). Sulphur in excessive amounts can have a deleterious effect on plant and human life; however, current information demonstrates that vegetation is generally more sensitive than human health.

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1 AIR QUALITY OBJECTIVES FOR SULPHUR DIOXIDE

The recommended maximum desirable levels and maximum acceptable levels for sulphur dioxide (SO₂) in ambient air in Canada are listed in the following.

	Average Concentration (µg/m ³ (ppm))* Over a Continuous Period		
	1 hour	24 hours	1 year
Maximum Desirable Level	450 (0.17)	150 (0.06)	30 (0.01)
Maximum Acceptable Level	900 (0.34)	300 (0.11)	60 (0.02)

The factor used to convert µg/m³ units (at 25°C and 101 kPa) to ppm was 1 µg SO₂/m³ = 3.82 x 10⁻⁴ ppm SO₂.

These levels are the same as the current published objectives for periods of one hour, 24 hours and one year. The decision to retain these objectives is based on a review of the scientific literature.

*µg/m³ = micrograms per cubic metre
ppm = parts per million

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PREFACE

Under the Clean Air Act, national ambient air quality objectives can be prescribed reflecting three ranges of air quality: desirable, acceptable, and tolerable. Air quality objectives for the desirable and acceptable ranges were published in the Canada Gazette for carbon monoxide, suspended particulate matter, sulphur dioxide, and oxidants (ozone) (1974); and nitrogen dioxide (1975). These ranges were recommended by the Federal-Provincial Committee on Air Pollution (FPCAP), now called the Federal-Provincial Advisory Committee on Air Quality (FPACAQ), as outlined in a 1976 publication entitled "Criteria for National Air Quality Objectives". In 1978, on the recommendation of the FPCAP, the tolerable ranges for these air contaminants were published.

The following definitions are used in setting numerical values for the highest concentration levels in the desirable and acceptable ranges:

The Maximum Acceptable Level is intended to provide adequate protection against effects on soil, water, vegetation, materials, animals, visibility, personal comfort, and well-being.

The Maximum Desirable Level is the long-term goal for air quality and provides a basis for an antidegradation policy for unpolluted parts of the country, and for the continuing development of control technology.

The maximum acceptable level will be of most concern to control agencies in their day-to-day operations. It is intended to provide adequate protection to receptors and guidance for long-term planning. When this level is exceeded, control action is indicated. At levels below the maximum desirable level, the pollutant would have little or no effect. It should be noted that the effects considered in the development of ambient air quality objectives are those of the pollutants themselves; effects of secondary or transformation products are not considered in depth.

The FPACAQ periodically reviews the national ambient air quality objectives in the light of new information. This report is a review of the desirable and acceptable ranges for sulphur dioxide. Reviews of the air quality objectives for other air contaminants will be published separately. The maximum tolerable levels also will be reviewed separately.

Pollutant levels in ambient air in Canada are monitored through the National Air Pollution Surveillance (NAPS) Network. Data summaries are available from Environmental Protection Publications, Environment Canada.

REVIEW OF NATIONAL AMBIENT AIR QUALITY OBJECTIVES FOR SULPHUR DIOXIDE
(Desirable and Acceptable Levels)

A Report by the
Federal-Provincial Advisory Committee on Air Quality

April 1987