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THE SENSITIVITY OF SOUTH AFRICAN PLANTS TO ACUTE DOSAGES OF SULPHUR DIOXIDE

by

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ABSTRACT

Relative susceptibility to SO_2 has been determined for a range of South African plants. Young trees, shrubs and herbaceous species were used for experiments in which the phytotoxic effects of SO_2 were studied at different concentrations and exposure times. The plants were fumigated in an exposure chamber at ambient conditions of temperature, light intensity and relative humidity.

Considerable variations in sensitivity were noted for the 63 species investigated and, among the families well represented in the South African flora, Ericaceae were resistant while Proteaceae were sensitive to SO_2. The injury symptoms developed have been described and foliar necrosis evaluated in terms of an injury index.

The results of this study form the first documented account of the susceptibility of South African plants to SO_2, and include a pictorial record of acute injury symptoms. A literature survey of current knowledge concerning visible and subtle phytotoxic effects of SO_2 is also presented.

TABLE OF CONTENTS

CHAPTER 1	INTRODUCTION	1
CHAPTER 2	PHYTOTOXIC AIR POLLUTANTS	9
2.1	TYPES AND SOURCES OF PHYTOTOXICANTS	10
2.2	PHYTOTOXICITY AND TYPES OF INJURY	12
2.3	VISIBLE INJURY SYMPTOMS	15
	2.3.1 Vascular Plants	15
	2.3.2 Lower Plants	19
2.4	DIAGNOSIS OF INJURY	20
2.5	BIOLOGICAL MONITORING TECHNIQUES	22
2.6	TIME-CONCENTRATION-INJURY RELATIONS	24
2.7	POLLUTANT UPTAKE	27
2.8	ECONOMIC CONSIDERATIONS	27
2.9	ECOLOGICAL IMPLICATIONS	29
CHAPTER 3	SULPHUR DIOXIDE AS AN AIR POLLUTANT	33
3.1	SULPHUR DIOXIDE IN THE ENVIRONMENT	33
	3.1.1 Gaseous Sulphur Pollutants	34
	3.1.2 Reactions of SO_2 in the Atmosphere	37
	3.1.3 Ambient Levels of SO_2	40
	3.1.4 Effects of Atmospheric SO_2	42
3.2	SULPHUR DIOXIDE AND PLANT RESPONSE	44
	3.2.1 Visible Symptoms of SO_2 Injury	45
	3.2.2 Effects of SO_2 on Plant Metabolism	47
	3.2.3 Factors Affecting the Response of Plants to SO_2	55

CHAPTER 4	EXPERIMENTAL	68
4.1	EQUIPMENT	68
	4.1.1 Greenhouse	68
	4.1.2 Exposure Chamber	70
	4.1.3 Air System	70
	4.1.4 SO_2 Supply	71
	4.1.5 Monitoring Instruments	71
4.2	PROCEDURE	72
	4.2.1 Fumigations	73
	4.2.2 Plants	74
	4.2.3 Injury Assessment	76
CHAPTER 5	RESULTS AND DISCUSSION	79
5.1	EVALUATION OF INJURY	79
5.2	FOLIAR INJURY DESCRIPTIONS	86
5.3	DOSE-RESPONSE REPRESENTATIONS	99
CHAPTER 6	CONCLUSIONS	104
REFERENCES		109
APPENDIX		
ACKNOWLEDGEMENTS		

CHAPTER 1

INTRODUCTION

Air pollution problems are usually related to some activity of man and, since the 13th century, contaminated air has been of social concern. Smoke and soot, produced by the combustion of fuels, were the first pollutants to attract attention, and by about 1600, sulphur dioxide was recognised as the major irritant in coal smoke. With the development and growth of the chemical industry and of manufacturing processes, increasing types and quantities of gases, particulates and aerosols were released into the atmosphere and, in addition, the internal combustion engine contributed quantities of hydrocarbons, nitrogen oxides and carbon monoxide. In the presence of sunlight, reactions between pollutants emitted to the atmosphere can result in the formation of secondary toxic products, and in the early 1950's some components of the photochemical smog of Los Angeles were identified. Comprising a number of reactive compounds, photochemical air pollution is prevalent nowadays in many urban areas of the world. Thus, over the years, the character of air pollution problems has changed: previously simple in composition, reducing in chemical nature and localised in occurrence, today pollutants are complex and variable, mainly oxidising in type, and widespread in effect.

Urban atmospheres usually contain SO_x, NO_x, HC, and other reactive organic compounds and particulates. These pollutants may accumulate during conditions of low ventilation potential to reach concentrations which are deleterious to health, reduce visibility, damage materials, and injure plants. Issued in America by the Environmental Protection Agency, air quality criteria are descriptive expressions concerning the relationship between various concentrations of pollutants and their adverse effects on man and his environment. Air quality standards based

on these criteria are prescriptive and indicate pollutant exposures which should not be exceeded in order to protect public health (primary standards), and public welfare (secondary standards). The latter standards are specifically intended to protect property, aesthetics and vegetation, and have, to date, been promulgated for SO_2, CO, NO_x, HC, photochemical oxidants, and particulates.

For over a century, air pollution has been known to cause injury to plants. The early investigations, carried out mainly in Germany and America, centered on problems concerning smoke and oxides of sulphur. The history of these studies has been surveyed, and comment made on the long period of conflict between agrarian and industrial interests [1,2]. The first of many reviews dealing with pollution injury to plants was published in 1951 [3], and today a large volume of literature covers the many aspects of plant injury.

Pollutants toxic to plants may be present in the atmosphere as gases, aerosols or particulates, though gaseous pollutants account for the most widespread cause of injury to vegetation. In general, vegetation injury induced by air pollutants has become a significant factor in evaluating the impact of man's activities on the environment. Plants growing in the vicinity of an emission source can be affected directly, and adverse effects may also be observed at considerable distances from a source as a result of secondary reactions of pollutants. In cases of severe injury to vegetation occurring around an emission source, a zonal pattern of differing degrees of destruction is observed, and is related to the atmospheric diffusion and transport of emissions. Data on the extent of pollutant injury to vegetation can also be provided by remote sensing techniques, which permit the mapping and monitoring of large areas and offer a means of detecting plant injury from pollutants before being visually identifiable.

Numerous compounds produce adverse effects in plants, the concentration levels in ambient air influencing the extent of the plant response. The resulting effects are related to the production and reactions of pollutants, and the dispersive processes of the atmosphere, in addition to the plant species involved. Air pollutants may be cumulative or non-cumulative in plants, and the effects produced can be classed as acute, chronic, or long-term. Differences in dose rate (concentration x time) can cause either acute or chronic injury, while an accumulation of heavy metals in vegetation and soil is considered a long term effect, as are changes in the composition of plant communities.

The visible effects of air pollutants on plants are primarily noted on the leaves which, being the site of gaseous exchange, are most vulnerable to injury. Localised tissue destruction, necrosis, characterises acute injury to leaves, and occurs when the rate of absorption of gas exceeds the capacity of the tissues to assimilate the pollutant. Chronic injury occurs when the absorption rate is slow and the leaf tissues are able to metabolise the gas; chlorophyll appears bleached, chlorosis usually appearing without the death of cells. At sublethal concentrations the growth pattern of the plant may be affected and alterations can occur in photosynthesis, respiration, stomatal resistance, enzyme activity and cell wall permeability. Such physiological effects include both visible and subtle effects [4], and result from disturbances of physiological processes at the molecular level. The exact mechanisms of injury to plant tissue are, however, largely undetermined.

Injury symptoms are characteristic for a phytotoxicant and the colour, shape and location of necrotic lesions on the leaf are of importance in the identification of specific pollutant effects, and in addition, the structure of different plant

leaves affects the development of symptoms. Descriptions and illustrations of visible injury symptoms for a range of air phytotoxicants are to be found in the literature, and are reviewed in Chapters 2 and 3.

Plants vary in susceptibility to air pollutants, sensitivity differing for different pollutants and between plant types. Additionally, numerous factors influence or modify plant response; Figure 1.1 illustrates some of the interactions involved. The effects of environmental parameters have been generally determined by laboratory exposure studies, and include

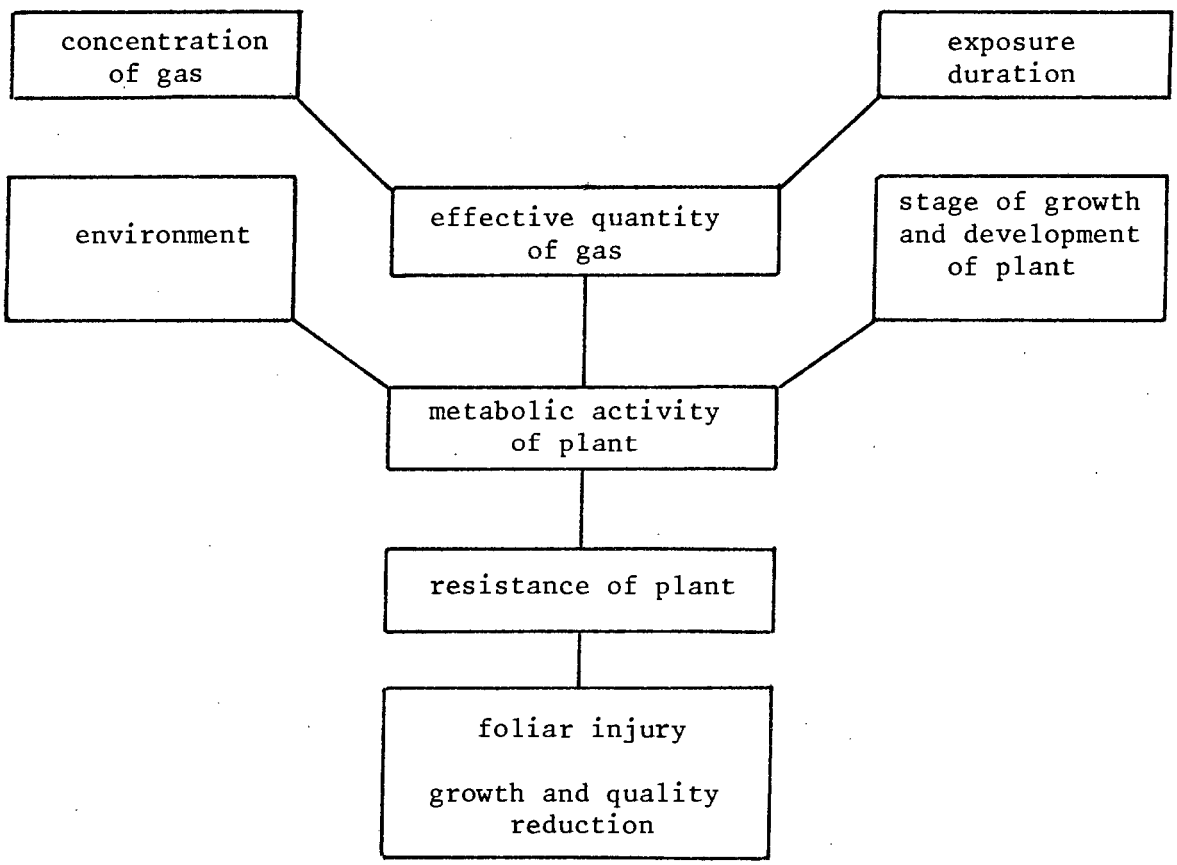


Figure 1.1: Influence of various factors on plant response to air pollutants

variations in light, temperature, humidity, and water supply. Dosages for some individual pollutants have been suggested for acceptance as criteria for plant injury; however, knowledge of conditions under which combinations of air pollutants produce adverse effects in plants is very limited. The subtle consequences of exposures of plants for long periods to low levels of toxicants are of great importance and a better understanding of these responses is needed. Little is known too, of the genetic factors conferring resistance or sensitivity in different plant species or varieties, though genetic variability can be employed in plant breeding techniques for the selection of varieties showing increased resistance to air pollutants. This aspect is of significance in agricultural and forestry practices in areas subject to air pollution.

Symptoms of leaf injury are often the first signs of an air pollution problem, and the recognition and assessment of injury patterns are fundamental to an understanding of the phytotoxic effects of air pollutants. Plants are particularly suitable for air pollutant studies as they are available in large numbers, and can be subjected to controlled conditions, the resultant effects being readily observed. Field surveys of the vegetation in areas of air pollution concern, provide a means of determining the geographical distribution of pollutants and of evaluating the efficiency of air pollution control legislation. Characteristics of source emissions, meteorology and topography require consideration, in addition to the plant species. Only a few prepollution studies have been made to describe baseline situations of pre-existing ecosystems, although the environmental impact statement dictated by certain environmental legislation requirements, may, in future, provide adequate background information. Vegetation sampling also provides a method of monitoring the accumulation in crops of pollutants which may be harmful to animals or man. Plants can be used in bioassay techniques to identify and assess concentrations of

toxicants in the field, and to study the reactions and effects of individual pollutants or combinations of pollutants in controlled exposures.

Any identifiable and measurable adverse effect on the intended or desired use of a plant or derived plant product is defined as damage [5] (injury being any identifiable and measurable response of a plant to air pollution), and therefore involves the yield and aesthetic value of plants. Costs incurred due to air pollution are difficult to estimate, especially as evaluation of the impact of pollutants in the absence of visible injury should be incorporated in the assessment.

The contribution of many variables (such as pollutant properties, exposure parameters, and receptor characteristics) must be considered in evaluating plant response for the establishment of "risk limits" for air pollutant effects, especially on important crop plants, trees, and indigenous vegetation. The interrelations of phytotoxicant concentration and exposure time (essential elements of air quality criteria) have been expressed by several mathematical equations, which can be used to predict the degree of acute injury sustained by sensitive plants at various dosages. Data on the dose relationships of chronic injury or of pollutant mixtures is, however, fragmentary. Interdisciplinary research in such subjects as plant physiology, ecology, meteorology, and chemistry is needed for the development of criteria applicable for a variety of environmental conditions and species of plants. As stated by McCune [6], to summarise and synthesise the toxicology of air pollutants in plants "is to sort out the segments of information, join them by inference, and fill in the gaps with speculation".

In South Africa, measurements of air pollutants have been made, since 1955, at sites in the major cities. Data obtained, mostly concerning smoke, SO_2 [7] and vehicle exhaust fumes [8], compare favourably with the values adopted as air quality

standards in other parts of the world. High individual concentrations have been recorded but these persist only for short periods, and although the levels for specific pollutants are below those at which harmful effects are normally observed, it is possible for combinations of toxicants, known to react synergistically, to produce adverse effects. Accurate measurements of the emissions from all major pollution sources are not available, though a survey [9] of the major types of industries reveals that over 2 million metric tons of air pollutants were emitted during the period mid 1969 to mid 1970. The need for air pollution control is evident, and since 1970 concern for air pollution problems in the Republic has become an important national issue.

Numerous research projects and programmes have been initiated relating to various aspects of air pollution [10]; these include, diffusion studies, atmospheric monitoring, ventilation potential of the atmosphere, emission control techniques, urban climates, health hazards, and corrosion of materials. Consideration is also being given to a study of the transport and effects of SO_2 [11]. Although injury to vegetation caused by fluorine compounds released in phosphate extraction operations has been noted [10], and HF injury to sugar cane has been studied at the ultrastructure level [12], the acute effects of air pollutants on South African plants have not been detailed.

In view of the importance of SO_2 as an air phytotoxicant in major industrial areas of the world, an investigation into the effects on South African vegetation of exposures to SO_2 was undertaken. For this purpose, 63 species of indigenous plants were selected, comprising 21 families and 36 genera, and representing different growth forms. Plants were exposed to various concentrations of SO_2 for differing periods of time in a fumigation chamber maintained under prevailing environmental

conditions. The main objectives of this study were to establish the procedure for controlled fumigation of plants, to determine the SO_2 dosages that cause foliar injury, to record the characteristic injury symptoms, and to ascertain the relative sucepti-bility to SO_2 of different species.

CHAPTER 2

PHYTOTOXIC AIR POLLUTANTS

Air pollution effects on vegetation have been recognised for more than a century, and have often resulted in large economic losses. Initially the destruction of vegetation around smelter areas attracted most attention and was associated with emissions of SO_2, although smoke and fumes from combustion and industrial processes also caused concern as important pathogens. More recently, emissions from electricity generating operations and photochemical reactions involving automobile exhaust fumes have accounted for much of the adverse effect on vegetation.

One of the first reviews of plant injury caused by air pollutants was published in 1951 [3], and today a comprehensive literature provides information on an increasing range of phytotoxicants [13-20]. Several publications (described as pictorial atlases) present colour photographs of the visible effects of air pollutants on plants [21-23]. It has become evident that air pollution affects many different vegetation types, and instances of plant injury due to air pollutants have been reported from many parts of the world. The nature and quantity of the phytotoxicants varies due to differences in emission sources, topography, and meteorological conditions; for instance, Los Angeles smog mainly comprises photochemical reaction products, while London smog essentially consists of smoke and SO_x, and in the industrial areas of America and Europe, fluorides and sulphur oxides cause extensive injury to forests, crops and ornamental plants.

Air pollution injury to vegetation is of importance not only with regard to economic losses, but also because plants can serve as monitors or indicators of the build-up of noxious substances in the air. In considering the impact of air pollution on vegetation, it is necessary to take into account the

types of compounds adversely affecting plants, sources emitting phytotoxicants, as well as symptoms of plant injury and plant species affected, and the mode of action of pollutants. These aspects, together with techniques for biological monitoring, and economic and ecological consequences, are discussed in the following sections.

2.1 TYPES AND SOURCES OF PHYTOTOXICANTS

Air pollutants pathogenic to plants arise from a variety of natural and man-related phenomena. Substances emitted directly from sources are termed primary pollutants, while those resulting from reactions in the atmosphere are termed secondary pollutants. The latter are more widespread and may occur over extensive areas in phytotoxic concentrations. Components of the photochemical smog complex such as ozone (O_3) and peroxyacetyl nitrate (PAN), are secondary pollutants and are considered among the more important phytotoxicants and, with sulphur dioxide and fluorides, pose serious threats to vegetation. In addition to PAN, other members of the homologous series of photochemical products which are toxic to plants are peroxypropionyl nitrate (PPN) and peroxybutyryl nitrate (PBN). Other gaseous pollutants known to injure plants include ethylene, chlorine and nitrogen dioxide; in addition, particulate matter and acid aerosols may also adversely affect plant life. Interactions between pollutants can result in effects which are greater, or less than, or equal to those caused by individual phytotoxicants; such synergistic, antagonistic or additive effects can be very complex and require careful evaluation.

In a paper presented to the American Phytopathological Society in 1967, Wood [24] outlined the types of the more significant plant pathogenic air pollutants and their sources. The relative importance of the major sources of phytotoxicants is shown in Table 2.1, indicating the contributions of industry,

TABLE 2.1 MAJOR SOURCES OF PLANT PATHOGENIC AIR POLLUTANTS [24]

Pollutant	Source (10^6 tons/year)				
	Trans-port	Industry	Elect. gener.	Space heating	Refuse disposal
Sulphur oxides	1	9	12	3	<1
Hydrocarbons	12	4	<1	1	1
Nitrogen oxides	6	2	3	1	<1
Fluorides		<1			
Particulates	1	6	3	1	1
Miscellaneous	<1	2	<1	<1	<1
Total	<21	<24	<20	<7	<5
%	28	30	26	9	7

transportation and electricity generation. Injury to vegetation may result from localised emissions of phytotoxicants (as F, NO_2, SO_2), accidental spillage (as Cl_2, NH_3), or from more general distribution over large distances (as O_3 and PAN). Residence time in the atmosphere also determines the injury potential of phytotoxicants.

Fluorides are emitted as effluents from a number of industrial processes, as in the production of phosphate fertilizers, glass, ceramics and steel, and in the reduction of aluminium. The combustion of coal, oil and S-containing ores, and the manufacture of sulphuric acid results in the production of sulphur oxides, the most important single source of SO_2 being coal-burning power stations. Many compounds are released by the burning of fuel in motor vehicles, in particular, nitrogen oxides and hydrocarbons, and the solar irradiation of these exhaust emissions results in the production of oxidants such as O_3 and PAN, and other inorganic and organic peroxides

considered to comprise the photochemical smog complex. Oxides of nitrogen are also produced in petroleum refining and other combustion and manufacturing processes. Ethylene, a contaminant of artificial illuminating gas previously used in greenhouses, is also emitted in vehicle exhausts and as a by-product in the manufacture of polyethylene. Particulates originate from a variety of sources, among the more important of which, in terms of plant injury, are deposits of soot and ash from combustion processes, dusts from cement and lime operations and agricultural practices, as well as heavy metals from foundry stack discharges.

2.2 PHYTOTOXICITY AND TYPES OF INJURY

A number of factors influence phytotoxicity, which is related to absorbability of gases by plant tissue and the toxic nature and reactivity of the element or compound itself, and the plant species involved. The concentration of an air pollutant and the duration of exposure are important parameters determining phytotoxicity, the product of concentration and time being defined as the dosage received by the plant. The importance of concentration and duration of exposure was noted by O'Gara [25], who in the early 1920's, formulated a law of gas action on the plant cell, expressed by an equation. Later investigators extended this work to cover dose-response relationships for many different species of plants. Such mathematical equations and models, developed for the expression of the degree of injury sustained under known conditions, are discussed in section 2.6.

Depending on the extent of injury developed, plants can be broadly categorised as sensitive, intermediate, or resistant to air pollutants. Sensitivity ratings for different species appear in many publications [16,21,22,26,27].

Pollutant effects on plants result from biochemical disturbances which may give rise to visible symptoms, or to measurable growth or physiological changes in the plant. Acute injury to plants results from short exposures (measured in hours) to relatively high concentrations of an air pollutant and is identifiable by the development of necrotic lesions. Symptoms of acute injury often appear on leaves within 24 hours of exposure. Chronic injury results from exposures to low concentrations of the gas over long periods; chlorotic leaf patterns develop, and leaf drop or early senescence may also occur. In addition to symptoms due to acute and chronic injury, plant growth alterations and reductions in yields may be evident, with or without attendant visible symptoms of injury.

Histologically the leaf comprises 3 types of tissue systems: epidermis, mesophyll and vascular tissue; Figure 2.1 illustrates general leaf structure. The epidermis covers the leaf surface and is composed of different types of cells which protect the underlying tissues; gases, vapours (and small particles) can

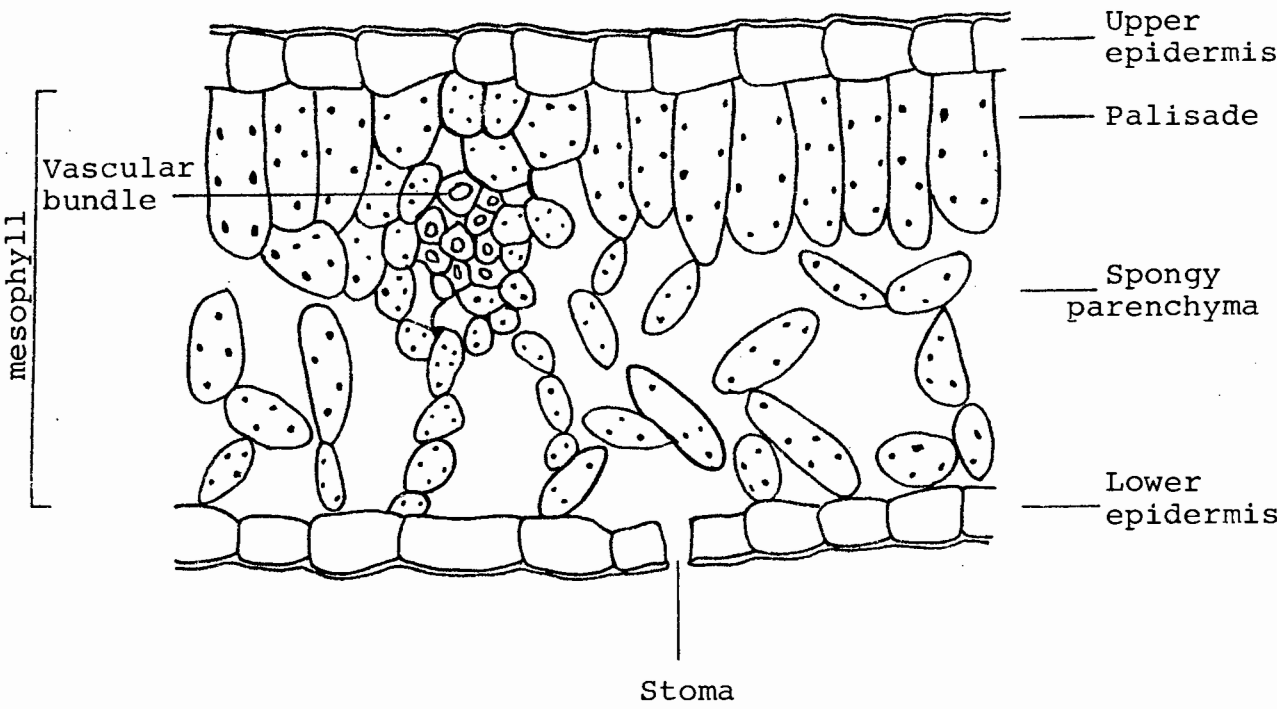


Figure 2.1 Internal structure of the leaf (dicot)

pass into and out of air spaces of mesophyll tissue through stomatal openings in the epidermal layer. The mesophyll, containing chloroplasts, is differentiated into spongy parenchyma and elongated palisade cells. Vascular bundles provide for the transport of water and food substances throughout the leaf area. The major functions of the leaf are photosynthesis, respiration and transpiration, and consequently injury to leaves will affect the vitality of the whole plant.

As the leaf is the primary indicator of injurious effects of air pollutants, foliar symptoms are often the only evidence of pollutant assault. Injury symptoms can be grouped into 5 broad categories [28]: general leaf necrosis, glazing and silvering of leaf surface cells, flecking or stippling, chlorosis, and suppression of growth. Necrotic lesions, indicative of acute injury due to absorption of gas in amounts sufficiently high to kill tissue, range in colour from pale yellow or white to dark brown, and characteristically appear first at leaf margins or leaf tips. Collapse of tissue on the underside of leaves results in a glazed, silvered, or bronzed appearance, while scattered necrotic areas produce flecked or stippled effects. A chronic symptom of injury, chlorosis, is manifest as a bleached condition, and in some instances occurs in conjunction with necrotic areas. Tissues which have accumulated an excessive but non-lethal amount of toxicant may develop chlorosis due to loss of chlorophyll. Growth is largely influenced by photosynthesis and respiration, and alterations in these two fundamental processes affect growth responses. Studies have shown that reductions in yield, and changes in growth and reproductive capacity occur at pollutant dosages which may or may not cause visible injury [29].

2.3 VISIBLE INJURY SYMPTOMS

2.3.1 Vascular Plants

Leaves generally first display markings indicative of the effects of certain air pollutants, although flowers and fruits may also be affected. Foliar symptoms are characteristic of a pollutant, species and leaf type, but are not necessarily definitive of the cause of injury. Numerous environmental stresses and interactions elicit injury responses similar to those caused by air pollutants; such mimicking symptoms result from adverse soil and nutrient conditions, drought, frost, sun-scorch, insects, disease, and other physiological disorders. Responses of plants to air pollutants are affected by various external and internal factors: environmental parameters (such as light, temperature, humidity) influencing the vigour of the plant, also affect sensitivity; different species, and leaves and plants of different ages, show considerable variation in relative susceptibility to toxicants. Thus, the recognition and identification of symptoms is a complex procedure, but necessary for the reliable diagnosis of pollutant impact on vegetation.

Numbers of crop species are known to be injured or damaged by air pollutants; these include potatoes, tomatoes, beans, tobacco, alfalfa, lettuce, grapes and oranges. Ornamental plants affected include petunia, gladiolus, tulip and rose. Many of the more devastating effects of air pollution on plant life concern trees; such adverse situations occur both around localised sources and at considerable distances downwind. Forest stands comprise many different species and thus a wide variety of response to phytotoxicants results. Conifers, many species of which are evergreen, are notably affected by air pollutants. Miller and McBride [30] present a detailed historical account of important episodes of injury to forests in the United States and Europe, together with descriptions of symptoms observed due to sulphur dioxide, fluoride, and oxidant injury.

TABLE 2.2

POLLUTANT EFFECTS ON VEGETATION [27]

Pollutant	Symptoms	Maturity of leaf affected	Part of leaf affected	Injury threshold		
				ppm (vol)	$\mu g/m^3$	Sustained exposure
Sulfur dioxide	Bleached spots, bleached areas between veins, chlorosis; insect injury, winter and drought conditions may show similar markings	Middle-aged most sensitive; oldest least sensitive	Mesophyll cells	0.3	785	8 hours
Ozone	Fleck, stipple, bleached spotting, pigmentation; conifer needle tips become brown and necrotic	Oldest most sensitive; youngest least sensitive	Palisade or spongy parenchyma in leaves with no palisade	0.03	59	4 hours
Peroxyacetyl-nitrate (PAN)	Glazing, silvering, or bronzing on lower surface of leaves	Youngest most sensitive	Spongy cells	0.01	50	6 hours
Nitrogen dioxide	Irregular, white or brown collapsed lesions on intercostal tissue and near leaf margin	Middle-aged leaves most sensitive	Mesophyll cells	2.5	4700	4 hours
Hydrogen fluoride	Tip and margin burn, dwarfing, leaf abscission; narrow brown–red band separates necrotic from green tissue; fungal disease, cold and high temperatures, drought, and wind may show similar markings; suture red spot on peach fruit	Youngest leaves most sensitive	Epidermis and mesophyll cells	0.1 (ppb)	0.08	5 weeks
Ethylene	Sepal withering, leaf abnormalities; flower dropping, and failure of leaf to open properly; abscission; water stress may produce similar markings	Young leaves recover; older leaves do not recover fully	All	0.05	58	6 hours
Chlorine	Bleaching between veins, tip and margin burn, leaf abscission; marking often similar to that of ozone	Mature leaves most sensitive	Epidermis and mesophyll cells	0.10	290	2 hours
Ammonia	"Cooked" green appearance becoming brown or green on drying; over-all blackening on some species	Mature leaves most sensitive	Complete tissue	~ 20	~ 14,000	4 hours
Hydrogen chloride	Acid-type necrotic lesion; tipburn on fir needles; leaf margin necrosis on broad leaves	Oldest leaves most sensitive	Epidermis and mesophyll cells	~ 5–10	~ 11,200	2 hours
Mercury	Chlorosis and abscission; brown spotting; yellowing of veins	Oldest leaves most sensitive	Epidermis and mesophyll cells	< 1	< 8,200	1–2 days
Hydrogen sulfide	Basal and marginal scorching	Youngest leaves most affected		20	28,000	5 hours
2,4-Dichloro-phenoxyacetic acid (2–4D)	Scalloped margins, swollen stems, yellow-green mottling or stippling, suture red spot (2,4,5-T); epinasty	Youngest leaves most affected	Epidermis	< 1	< 9,050	2 hours
Sulfuric acid	Necrotic spots on upper surface similar to caustic or acidic compounds; high humidity needed	All	All	—	—	—

Foliage leaves exhibit great variations in anatomical and morphological structure and the type of lesion caused by air pollution effects often differs in relation to the structure of the leaf affected. Broad leaves typically show injury as marginal or interveinal blotches; irregular necrotic streaking, either side of the mid-vein, characterises injury to grasses and other parallel-veined leaves; needle-like leaves are usually injured at the tips. Table 2.2 summarises these characteristics and indicates injury thresholds for a selection of phytotoxicants; Table 2.3 shows species typically sensitive to phytotoxicants. Brief descriptions of visible injury effects due to fluorides, sulphur dioxide, photochemical oxidants, and others, are given below, while details of SO_2 injury development and metabolic effects of SO_2 are reviewed in section 3.2.

TABLE 2.3 SELECTED SPECIES SENSITIVE TO PHYTOTOXICANTS

Phytotoxicant	Sensitive Species
F	apricot, gladiolus, white pine
SO_2	alfalfa, lupin, Douglas fir
O_3	tobacco, grape, ponderosa pine
PAN	pinto bean, tomato, petunia
NO_2	lettuce, orange, azalea

Fluorides. Direct injury may be caused to sensitive plants and fluorides may also act as cumulative poisons. Both gaseous fluorides (as HF, SiF_4) and fluoride particulates affect plants, being rapidly absorbed and translocated to leaf tips (particularly in monocots) and leaf margins (especially on broad-leafed plants) where necrotic areas develop. Lesions may be light green in colour becoming reddish brown; chlorotic patterns may

also appear, sometimes without necrotic areas. In cases of severe injury, premature leaf abscission occurs. Some fruits may be more sensitive than leaves, resulting in dropping of fruit or premature ripening (as "suture red spot" of peach). Plants have been shown to accumulate F in tissues with or without visible injury [29], thus diagnosis of F injury may additionally involve analysis of leaf tissue.

Sulphur dioxide. Acute and chronic injury are caused by SO_2, and both these symptoms may appear on the same plant. Fully expanded and middle aged leaves are usually most sensitive, and enlarging leaves more resistant. Acute injury to broad-leafed plants is characterised by irregular marginal and interveinal necrotic areas, bleached (straw to ivory) or red brown in colour, and becoming dry; major veins remain green. Necrotic streaking is shown by parallel-veined leaves and is usually more severe near the tips. Conifers too display tip necrosis and often a brown banded appearance. In cases of severe injury, leaves may be shed. Chlorotic symptoms develop as a result of chronic exposures, and leaves may show an increase in sulphur content.

Photochemical oxidants. 3 major compounds (O_3, PAN, NO_2) result from photochemical reactions involving hydrocarbons and nitrogen oxides. A wide range of foliar symptoms is shown depending on the phytotoxic components present in the smog complex. Some attempts have been made to associate PAN injury with higher $HC:NO_x$ ratios, and O_3 injury with lower ratios; observations indicate that on the West coast of America, PAN injury is more common, while O_3 injury affects many plants on the East coast [31]. Patterns of O_3 injury vary considerably between species and varieties, and is characterised by the appearance of flecks or stipple-like lesions, white or brownish in colour, especially on the upper surfaces of leaves. Where lesions are large, bifacial necrosis occurs. Palisade tissue

is affected first, though cell collapse is uniform in grasses and cereal plants. Conifers show brown necrosis of the needle tips. While the phytotoxic effect of O_3 is most pronounced on older and fully expanded leaves, PAN generally affects young expanding tissue. Silvering, bronzing and glazing of the undersurface of leaves distinguishes PAN injury. Irregular banding, bleached yellow to tan, sometimes occurs, more especially in leaves of monocotyledons. Chlorosis and leaf abscission may be caused by both PAN and O_3. Closely resembling symptoms of SO_2 injury, NO_2 produces irregular white or brown lesions between veins and near leaf margins.

Other phytotoxic air pollutants. Ethylene can interfere with the activities of plant hormones and in phytotoxic concentrations may cause growth abnormalities, epinasty, abscission of buds, fruit and leaves, necrosis and chlorosis. Injury symptoms of chlorine pollution are primarily of an acute nature and are similar to those of SO_2. Ammonia causes tissue collapse with or without subsequent loss of chlorophyll. Foliar necrosis caused by emissions of boron has recently been described [32], and airborne herbicides, especially weedkillers of the 2,4-D type, can induce growth malformations. Particulate deposits (especially when heavy) on leaves are of importance as regards reduction in light intensity and possible retardation of growth [33]. Accumulations of certain heavy metals are known to adversely affect vegetation [34], as particulates absorbed into the leaf may disrupt various metabolic pathways. In addition, deposits of dusts containing heavy metals may cause direct "burn" injury to plants [35], and acid aerosols and caustic emissions penetrating the leaf cuticle may cause pitting or a shot-hole type of injury.

2.3.2 Lower Plants

Over 100 years ago, botanists observed a decline in the numbers and luxuriance of epiphytic lichens in the vicinity of

towns, a phenomenon later ascribed to air pollution, and to SO_2 in particular. Lichens and bryophytes, especially mosses, being efficient absorbers and accumulators of substances from the environment, are very susceptible to atmospheric pollutants, which may either affect sensitive species directly or indirectly by rendering substrates unsuitable for propagule establishment. In addition to SO_2, fluorides and fertilizer dust have also been identified as exerting major adverse effects. No distinction is generally made between acute and chronic injury, and in polluted areas, abundance and fertility of epiphytic species may be reduced and smaller plant bodies produced; annual average concentrations of over 0,03 ppm SO_2 result in a marked decline in lichen flora [36]. SO_2 primarily affects the algal component of lichens, inducing the conversion of chlorophyll to phaeophytin [37]; injury symptoms are manifested by a whitening, browning or violet tinging of marginal lobes of the thallus [38], and at high SO_2 concentrations death results.

2.4 DIAGNOSIS OF INJURY

A disorder can be defined as any plant irritation, whether chronic or acute, whether visible symptoms are present or only metabolic effects are suspected [16]; the diagnosis of such a disorder is a scientific determination based on careful examination. Symptoms characteristic of a pollutant, provide a major basis for the identification of air pollution effects. In the field, the diagnosis of injury to vegetation due to air pollutants is complex and many factors need to be considered in determining the causal agent or agents. The plant is a product of its environment and thus each environmental parameter produces a response in the plant. Injury may be modified by such factors as seasonal variations, environmental conditions, genetic make-up, and senescence; additionally, symptoms may develop due to other causes which are difficult to distinguish from those produced by air pollutants. Also, air pollutants themselves can mimic each other.

Environmental pathogens (physiopaths) which cause symptoms similar to those produced by air phytotoxicants include temperature stresses, unfavourable light conditions, climatic extremes, adverse water relations, mineral deficiencies or excesses, soil O_2-CO_2 balance, and chemical formulations used as pesticides. Biogenic pathogens causing disorders in plants may be insects, fungi, bacteria or viruses. Growth suppression and yield reductions can result from air phytotoxicants and other pathogens: viruses and cultural conditions are especially significant factors influencing production in the absence of visible symptoms [28]; the subtle effects of air pollutants are, however, not generally used as diagnostic aids. Leaf tip burn is of wide occurrence in many plants; chlorosis is a chronic symptom of injury produced by several phytotoxicants, and is a condition particularly difficult to appraise in the field. Genetic abnormalities, induced by mutations or recessive factors, are likewise difficult to identify.

Injury symptoms of some diseases, the etiology of which was previously unknown, have been attributed to air pollutants, notably ozone. During the late 1950's, observations of O_3 levels in urban atmospheres and in experimental fumigations, revealed the range of symptoms produced and indicated the widespread extent of O_3 injury. "Grape stipple" was the first disease recognized [39] to be caused by O_3, and in 1959 Heggestad and Middleton [40] reported that "weather fleck", a serious disease of tobacco, was caused by O_3. Various diseases affecting forest trees were also found to be pollutant induced: "chlorotic dwarf", which stunts the growth of eastern white pine, has been ascribed to low levels of O_3 and SO_2, probably acting synergistically [41]; semi-mature tissue needle blight (SNB) and emergence tipburn, which affect certain individuals of eastern white pine, are thought [42,43] to be symptoms of injury due to SO_2 and/or O_3; "X-disease" of ponderosa pine bears the additional name, "ozone needle mottle" [44],

indicative of the causal agent. In some cities, a pollutant induced decline of urban trees, "bus stop disease", is apparently associated with photochemical reactions involving vehicle exhaust fumes.

Colour illustrations of characteristic injury symptoms caused by air phytotoxicants and other pathogens are presented in some publications [22,23,26], and provide guidance for the identification of plant disorders. Laboratory and field chamber studies are essential for the recognition and identification of injury symptoms produced by air phytotoxicants. Considerations necessary for correct field diagnosis have been summarised [26, 28], and indicate the complexities involved and the necessity for competent observers in evaluating vegetation injury as a criterion in air pollution situations.

2.5 BIOLOGICAL MONITORING TECHNIQUES

Information regarding the symptoms of injury due to air pollutants is derived largely through controlled fumigations of plants, which together with field surveys and ambient air monitoring, permit an evaluation of the impact of pollutants on vegetation.

The exposure of plants to phytotoxicants, under greenhouse or laboratory conditions, may be carried out in a variety of experimental chambers (closed or open-top), which range from constructions of simple design to complex equipment incorporating the fine control of environmental variables. This exposure method supplies data on injury symptoms, dosages causing injury, and relative susceptibility of different plant species and varieties. In addition, controlled fumigations may be employed in the determination of significant culture and environmental conditions, and aid in the elucidation of the mechanisms by which plants are injured by air pollutants.

Generally, experimental exposures do not directly simulate ambient field conditions where numerous interactions cause varied responses in plants; nevertheless, this technique is essential for the development of qualitive and quantitive models of the consequences of air pollution effects [4].

Field surveys, although constituting after-the-fact studies, are a valuable means of determining the extent and severity of injury, and in some cases provide the ultimate measure of the significance of air pollution conditions. Inspection and evaluation of plants is considered [28] to provide a net index of injury which is useful regardless of environmental or cultural factors that might affect the degree of injury. Where visible injury occurs and yield is affected, damage can be assessed quantitatively. Comprehensive surveys of vegetation have been made in numerous areas and surveillance programmes initiated; however, considerable caution must be exercised in the field in attributing non-specific signs of poor growth and general abnormality to air pollution [15] (section 2.4). Expertise in diagnosis is of great important in such surveys, which require an appreciation of all the possible interactions involved in the production of injury patterns.

Chemical analyses of plant tissue can provide an indication of pollutant load. These analytical methods are particularly suitable for the monitoring of plants for cumulative pollutants, such as fluoride, lead, beryllium, nuclides and pesticides, though are less useful with regard to sulphur, chlorine, O_3 and PAN. Considerations concerning the recognition and monitoring of air pollutants by means of bioassay techniques, both in the laboratory and in the field, have been outlined by a number of workers [15,45-48], and indicate the usefulness of plants as indicators of air pollution.

The uptake and accumulation of pollutants from the air by mosses and lichens has been employed for pollutant detection and monitoring studies. An extensive literature is available [20,37,49-51] regarding the use of lichens as indicators of pollution, and certain species have been used in establishing qualitative [36,52] and quantitative [53] relationships of SO_2 levels and changes in lichen flora. Schönbeck [54] has adopted a technique [55] utilising transplanted lichen discs, which can be exposed in polluted areas to record the effects of ambient air pollutants.

Differences in susceptibility to air pollutants between species of higher plants also permit the selection of sensitive plants as indicators for use in air pollution monitoring programmes. Plants grown under known conditions in the greenhouse, can be exposed to ambient air in special chambers or containers maintained in field plots. The development of injury signs occurring over a period of time can therefore be observed and evaluated. Various inexpensive portable systems for biological monitoring of air pollutants have been developed [56-58], and are especially useful in areas which cannot be easily monitored by instrumental methods. Many plants have been successfully used as indicators of the presence of different pollutants; these include, annual bluegrass and certain varieties of petunia for PAN and oxidants; pinto bean and tobacco (Bel W_3) for O_3, species of gladiolus and grass for fluorides, and alfalfa and cotton for SO_2.

2.6 TIME-CONCENTRATION-INJURY RELATIONS

A knowledge of the interrelations of pollutant concentration and exposure time is necessary for an understanding of pollutant effects on vegetation. These two variables in particular affect the degree of plant injury sustained, and attempts have been made to quantify plant response through mathematical modeling.

O'Gara [25] formulated a law of gas action on the plant cell, expressed by the equation:

$$t(L - \ell) = K$$

where t is the time through which the gas acts; L is the concentration of the gas; ℓ is that concentration of gas which will not injure the plant even after prolonged exposure; K is a constant. This equation incorporates the concept of a threshold concentration for acute injury, and the expression fits experimental data obtained over short periods. O'Gara's factors of relative sensitivity to SO_2 (determined by fumigation experiments), were published [59] for numbers of plants. Thomas and Hill [60] modified the O'Gara equation to include gas absorption rate. Similar models have been developed to express dose (concentration x time) response relationships and Guderian et al. [5] have suggested an exponential relationship, applicable over a wider range of time periods. This equation, which includes a biological complex factor, b, and the vegetation life time, K, is expressed as:

$$t = Ke^{-b(c-a)}$$

where t is exposure time; c is pollutant concentration when above threshold; a is injury threshold. Such equations permit the assumption that, provided a certain threshold concentration is not exceeded for a given time period, no acute injury will occur [17].

Heck and Tingey [61] proposed a time-concentration model to predict acute foliar injury, which treats concentration as the dependent variable, and both injury and time as independent variables. Their equation is given as:

$$c = A_0 + A_1 I + A_2/t$$

where c is concentration; I is per cent injury; t is time; A_0, A_1, A_2 are constants (partial regression coefficients). A large number of data points is needed in order to use this type of equation; however, in common with some of the more recently proposed expressions, the cumbersome approach of determining a threshold concentration before the equation can be solved, is avoided. Larsen and Heck [62], in adopting this approach, have expressed the degree of plant injury as a function of air pollutant concentration and exposure duration. Their equation has the form:

$$c = ms^z t^\rho$$

where c is pollutant concentration; t is exposure time; m,s,z,ρ are statistical parameters (m includes an injury factor).

Three-dimensional surface response curves have been constructed which show graphically the non-linear relation of dose and injury [61,63,64]. Response surfaces of this type illustrate the interaction between concentration and time on the production of acute injury. Observed effects of time-concentration variations have been used to compile tables [17] which indicate exposure periods and concentrations likely to cause acute injury to plants in different susceptibility groups. While there is considerable information on the effect of acute injury, knowledge regarding the effects of time and concentration on the reduction of growth, yield or quality of plant material, or on the development of chronic injury, is extremely limited.

2.7 POLLUTANT UPTAKE

Many air pollutants are removed from the atmosphere fairly rapidly by natural processes of precipitation, deposition, absorption and adsorption. The importance of vegetation in the uptake of pollutants is well known, and use has been made of vegetation "greenbelts" as a sink for air pollutants around certain urban and industrial complexes. Recent studies [65,66] have indicated that plant canopies can efficiently remove such gaseous pollutants as SO_2, NO_2, O_3, HF and Cl_2, thereby providing an important cleansing action of the atmosphere. The complexities of the gas transfer process involved in the transport of pollutants to the plant, are outlined in a review of micrometeorological methods [67].

Investigations have concerned the foliar sorption of pollutants by plants [68-72], and a few attempts have been made to determine the flux of pollutants [73]. The actual effective dosage received by the plant is more directly related to the flux (rate of transfer) than to the concentration of the pollutant [74], and investigations have shown that the interaction of a number of physical, chemical and biological factors affect pollutant absorption and penetration of leaf tissues. Such factors, which include leaf surface characteristics, diffusion resistance of stomata, pollutant solubility and concentration, have been incorporated into a model for simulating pollutant exchange by leaves [75], from which absorbed pollutant dosages can be obtained.

2.8 ECONOMIC CONSIDERATIONS

The relative importance of a number of phytotoxicants has been rated, and in order of significance those cited [76] are ozone, sulphur dioxide, peroxyacyl nitrates, ethylene and other hydrocarbons, fluoride, pesticides, chlorine and hydrogen chloride, nitrogen dioxide, heavy metals, particulates. In general, O_3, PAN, SO_2 and F are considered to be the most

important plant pathogenic air pollutants [17,22,24,77], and while on a world-wide basis SO_2 apears to be more injurious than O_3, in North America O_3 appears to be the most significant phytotoxicant. Air pollution effects which result in economic loss are evidenced by both visible symptoms and reductions in growth, yield and quality of forest trees, agronomic and horticultural crops, and indigenous vegetation. Several attempts have been made to estimate such economic losses; the 1975 report of the Council on Environmental Quality [78] summarises selected studies on vegetation damage costs, and Ross [79] has also reviewed reported losses. Exact assessments are difficult to obtain due to variations in growth, delayed maturity and recovery of the plant, possible "hidden injury", yield and marketability of the product; and additionally, the expertise of the investigator affects the reliability of a survey. In assessing loss, it should be noted that a distinction is often made between the terms injury and damage: injury refers to any identifiable and measurable response of the plant to air pollution; damage infers a reduction in value or use of the plant (aesthetic or economic loss) [21]. Thus, a plant may be injured without damage if aesthetic value or yield is not affected.

In afforestation operations the economic loss due to diminished annual wood volume is of prime concern, and data on the decrease in volume growth form the basis for monetary estimates [80]. Linzon has calculated a drop in income of more than $1 million over a 10 year period in the Sudbury area of Ontario [81]. Estimates of annual losses for agronomic species in California have been quoted by Brandt and Heck [15]. A statewide field survey was undertaken in 1969 to determine the nature and extent of depletion of food, fibre and ornamental crops in Pennsylvania; visible injury only was assessed, direct economic losses being estimated in excess of $3,5 million and total indirect losses accounting for a further $8 million [82]. The Stanford Research Institute has initiated a programme for

estimating crop losses due to direct effects of O_3, PAN, NO_2, SO_2 and F; emission data from fuel consumption sources is utilised in deriving formulae for evaluation of losses incurred; annual losses in excess of $71,5 million were suggested (though based on 1964 data only) for 68 crops studied in over 500 American counties [31]. The annual cost to the United States as a whole has been estimated [83] to amount to $500 million, though Heggestad and Heck [17] believe that gross estimates of between $500 million and $1 000 million annually may be too low.

Reduction in such air pollution losses and increases in agricultural production may be achieved by the application of chemical protectants to plants and by the implementation of plant breeding techniques. The use of antioxidants has shown potential and certain systemic fungicides and herbicides have rendered species less susceptible to air pollutant attack. Several programmes have been undertaken [84], directed towards the selection and breeding of certain plants for the development of varieties relatively resistant to air pollutants; notable success has been achieved in producing O_3-resistant varieties of tobacco and potato. It appears likely that further research will reveal many more species of plants whose varieties display resistance to phytotoxicants.

2.9 ECOLOGICAL IMPLICATIONS

Woodwell [85] has indicated the predictability of changes in natural ecosystems as caused by many different types of disturbance. The expected effects can be summarised as changes in structure, resulting in elimination of sensitive species, and involving primary production and nutrient resources. Treshow [16] notes too, that population shifts can occur through modifications of growth and reproductive potential of species which reduce the competitive ability of sensitive species. Air pollutants may modify the composition of plant communities due to

differential sensitivity among species, even though only a few species may be affected. Ecological consequences of air pollution can be considered as reductions of structure of plant communities, from forest towards shrub-dominated communities, and under severe conditions, towards lower growing plants. Figure 2.2 presents diagrammatically the possible effects of air pollutants on plant communities.

Adverse effects of air pollution on forest areas were observed many years ago, and on occasions, entire plant communities have been devastated. Naegele [19] mentions the subtle selective manner in which mountainsides of vegetation are being affected by photochemical oxidants. Community changes have been observed in the San Bernadino National Forest where destruction of many acres of pines has occurred due to chronic photochemical oxidant air pollution [80]. From the Ruhr Valley area of Germany, severe losses to pine plantations due to SO_2 are reported by Knabe [87], and attempts are being made to replace sensitive species with hardwoods or more resistant pines [88]. Population changes have also been observed in forage crops, more tolerant rye grass replacing the more sensitive clover species [21]. Ecological studies have shown that the distribution pattern of lichens, especially around industrial areas, is related to atmospheric SO_2 levels. Although changes in incidence and severity of various plant disorders and diseases may be associated with air pollutants [16,83], relatively little is known of how pollutants affect fungal, bacterial and viral plant pathogens [89,90]; interactions may be more toxic either to the host or to the biotic agent. A secondary impact on the composition of populations may result from disturbances of host-parasite relationships [91], as sensitive species become eliminated from the leaf surface microhabitat [92].

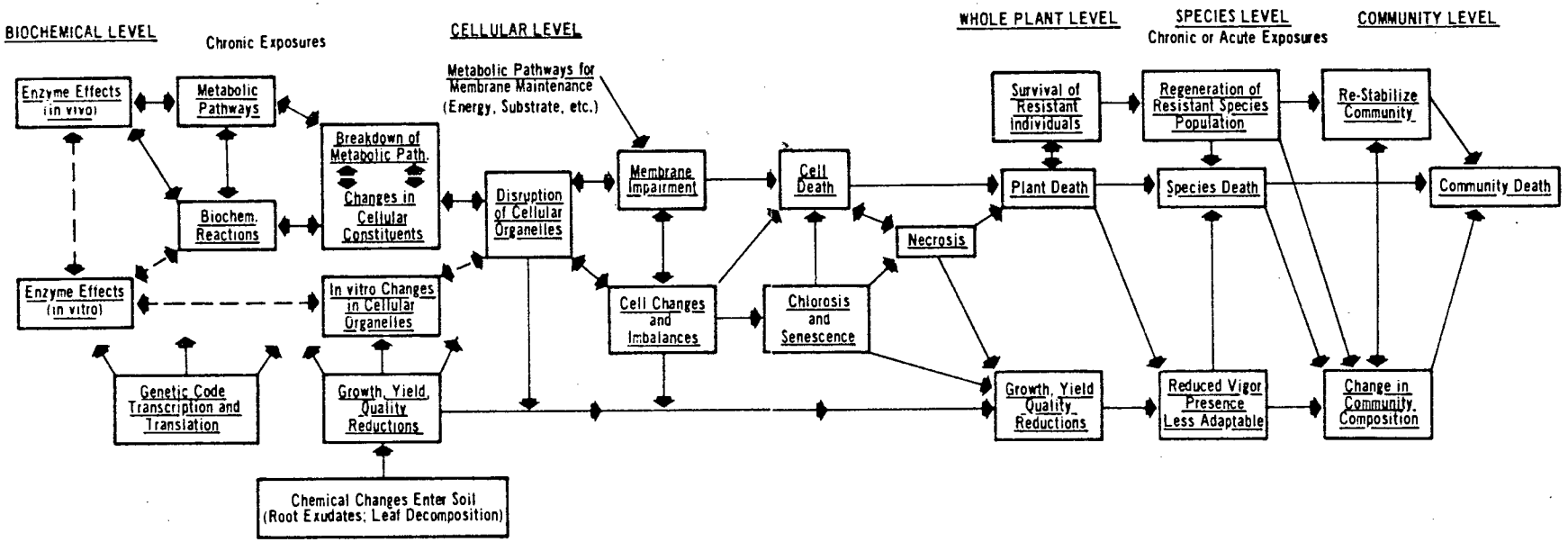


Figure 2.2 Effects of air pollutants on plant communities [86]

In general, an accurate assessment of the overall impact of air pollution on vegetation, necessitates additional specific and detailed information concerning the different responses of species and varieties of plants, and the likely effects of pollutant interactions in the environment. Predictions on the future contributions of the major sources of phytotoxicants have been made [24], and in view of the increasing demands of industrialisation and urban development, it is probable that the air pollution problem involving vegetation will continue, in spite of the control measures currently operative.

CHAPTER 3

SULPHUR DIOXIDE AS AN AIR POLLUTANT

Since earliest recorded history, sulphur dioxide fumes have been known and utilised, due to the wide occurrence and peculiar properties of sulphur and sulphides. The first gas to be widely recognised as an air pollutant, SO_2 is produced primarily in combustion processes, and causes many adverse effects on man and the environment. Sources, reactions, ambient levels, and general effects of SO_2 are discussed in this chapter, with particular reference to effects of SO_2 on plant life.

3.1 SULPHUR DIOXIDE IN THE ENVIRONMENT

Certain chemical elements are cycled in the biosphere, and between 30 and 40 of the elements occurring in nature are required by living organisms. Sulphur, as one of these essential elements, moves in definite biogeochemical cycles, and occurs in a number of forms, as elemental sulphur (in brimstone and pyrites), in gaseous form (as hydrogen sulphide and sulphur dioxide), and as sulphates. The major annual contributors of sulphur to the atmosphere [93] are H_2S produced by biological decay (98×10^6 tons S), sea-spray sulphate (44×10^6 tons S) and SO_2 from pollutant sources (70×10^6 tons S). The circulation of sulphur through the environment in various forms is shown schematically in Figure 3.1, and is based on estimates of emissions and depositions, though some of the data must remain speculative. Total world-wide sulphur emissions are calculated [94] as 220×10^6 tons per year.

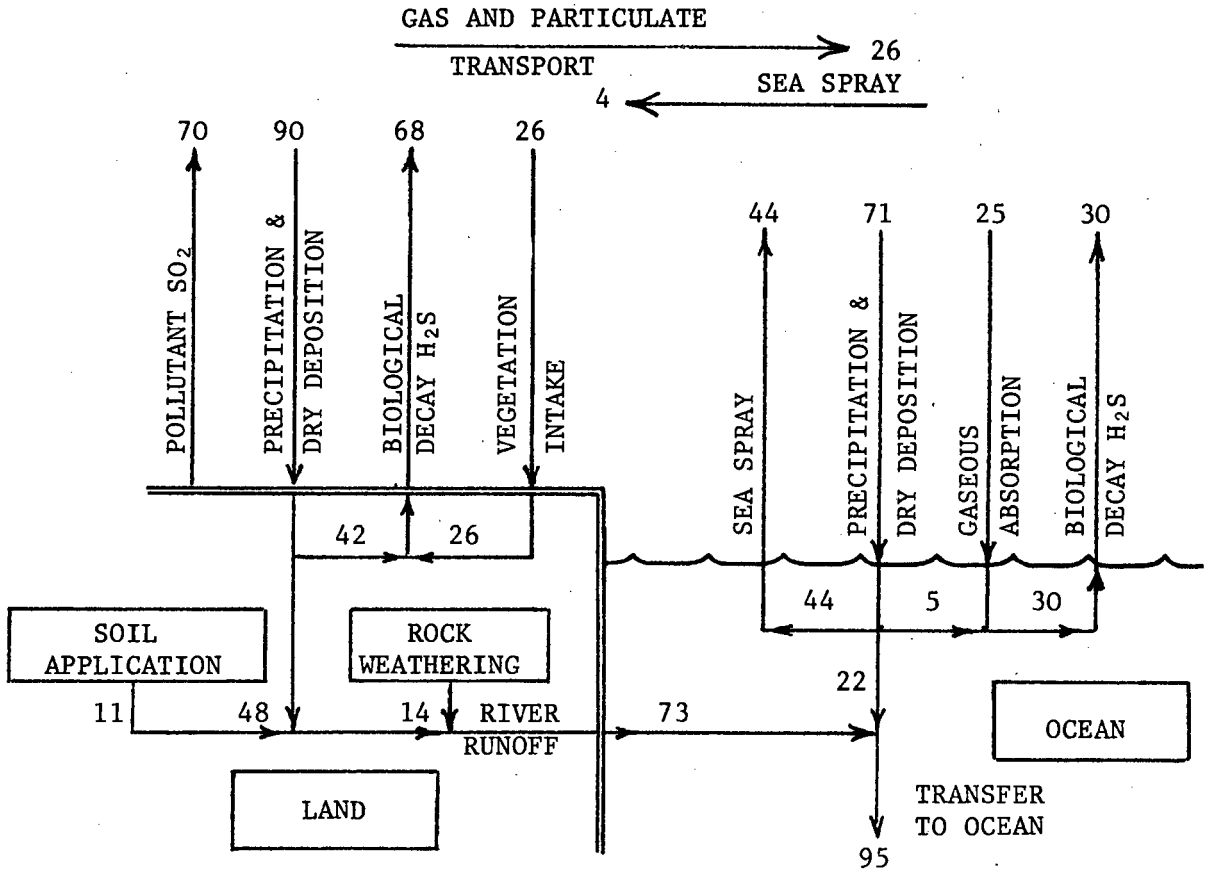


Figure 3.1 Environmental Sulphur Circulation [93]
(10^6 tons/year)

Pollution of the atmosphere by natural and anthropogenic sources is well known and in many air pollution disasters the sulphur pollutants have been implicated. Of the sulphur oxides, SO_2 is the most infamous in this regard.

3.1.1 Gaseous Sulphur Pollutants

Natural emissions of H_2S result from the decay of organic matter and from volcanic activity. Some industrial operations such as petroleum refining and viscose rayon processing, release quantities of H_2S. In considering natural emissions of sulphur, as H_2S, it is estimated [93] that these are about 30% greater than industrial emissions of H_2S and SO_2. H_2S can be

oxidised readily to SO_2 and has a residence time in the atmosphere of between 2 hours and 2 days.

Atmospheric SO_2 is derived mostly from sources of pollution, mainly the product of domestic and industrial activities. 136×10^6 tons of the total SO_2 emissions (146×10^6 tons) is derived from the northern hemisphere, thus the southern hemisphere contributes only 7% to the total SO_2 pollution [93,94]. The combustion of fuels, especially petroleum and coal, and smelting operations involving sulphide ores release quantities of sulphur oxides to the air. Of the total annual world-wide SO_2 pollutant emissions, 70% is estimated to be derived from coal combustion, and 16% from combustion of petroleum products (Figure 3.2).

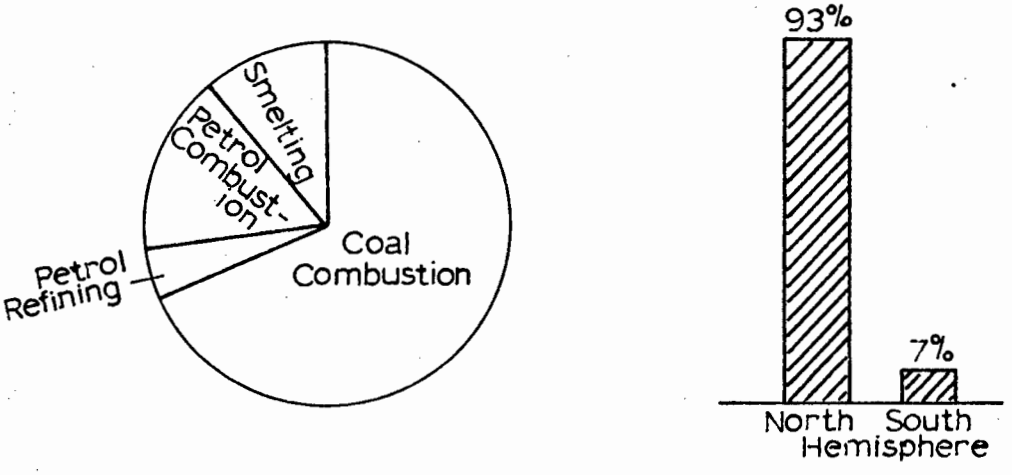


Figure 3.2 SO_2 Pollutant Sources [94]

Estimated SO_2 emissions from fuel combustion sources in the United Kingdom during 1974 are given in Table 3.1, and are grouped according to emission levels above the ground; the most important sources are power stations and industries [95].

During the last 30 years global emissions of SO_2 have doubled [96] and seem likely to continue to rise as a result of increased consumption of coal and petroleum for power

TABLE 3.1 Estimated Sulphur Dioxide Emissions from Fuel Combustion in the United Kingdom, for 1974, for Various Heights of Emitter [95] (10^6 metric tons)

High Level		
Power stations	coal	1,81
	gas oil	0,01
	fuel oil	1,00
	total	2,82
Refineries	fuel oil/gases	0,30
Total high level		3,12
Medium level		
Other industry	coal	0,33
	solid smokeless fuel	0,05
	gas oil	0,08
	fuel oil	1,01
	coke oven gas	0,12
Total medium level		1,59
Low level		
Domestic	coal	0,24
	solid smokeless fuel	0,09
	gas oil	0,01
	fuel oil	0,00
	total	0,35
Agriculture	gas/diesel oil	0,01
	fuel oil	0,02
	total	0,03
Commercial/public services	coal	0,06
	solid smokeless fuel	0,02
	gas oil	0,05
	fuel oil	0,14
	total	0,27
Rail transport	gas oil	0,01
	fuel oil	0,00
	total	0,02
Road transport	motor spirit	0,01
	diesel fuel	0,04
	total	0,05
Total low level		0,72
Grand total		5,43

production and heating. In some areas nevertheless, SO_2 levels are showing a decreasing trend; the report for 1975 of the Council on Environmental Quality [78], indicates a decrease in SO_2 concentrations in major metropolitan areas of the United States, and a nationwide decline of 8% between 1970 and 1974. In London since the implementation of the Clean Air Act of 1956, the amount of smoke in the atmosphere has been reduced, and although emissions of SO_2 have changed little, more efficient dispersion has been achieved, resulting in lower ground level SO_2 concentrations [97]. Intensive studies are being made to control SO_2 emissions both from stationary and mobile sources, and to determine the reactions involving SO_2 in polluted atmospheres.

3.1.2 Reactions of SO_2 in the Atmosphere

A colourless gas, soluble in water, SO_2 has a characteristic pungent odour. Under atmospheric conditions SO_2 can act either as an oxidising or as a reducing agent. The extent of oxidation depends on such factors as, atmospheric moisture, intensity and duration of sunlight, presence of sorptive materials and catalysts.

Photochemical reactions lead to the formation of sulphur trioxide (SO_3) which rapidly combines with water vapour yielding sulphuric acid aerosols. Typical photochemical reactions of SO_2, quoted by Urone [98], are given in Table 3.2 The photo-oxidation of dilute SO_2 in air follows a first order mechanism, and while the rate of photo-oxidation of SO_2 in clean air is slow, in the presence of hydrocarbons and nitrogen oxides this rate increases markedly; in polluted air and in the presence of mist or fog the photochemical reaction rate is very rapid [98,99].

TABLE 3.2

PHOTOCHEMICAL REACTIONS OF SULPHUR DIOXIDE [98]

Primary reactions	Secondary reactions
1. $SO_2 + h\nu \rightarrow SO_2^-$	5. $SO_4 + SO_2 \rightarrow 2SO_3$
2. $SO_2^- \rightarrow SO_2^= (+M \rightarrow SO_2)$	6. $SO_4 + O_2 \rightarrow SO_3 + O_3$
3. $SO_2^- + M \rightarrow SO_2 + M$	7. $SO_3 + H_2O \rightarrow H_2SO_4$
4. $SO_2^- + O_2 \rightarrow SO_4$	
(where M = N_2, O_2 or SO_2)	

Oxidation of SO_2 to H_2SO_4 by atmospheric oxygen is catalysed by oxides or salts of Fe or Mn [100]. With ammonia, SO_2 forms solid particulates of ammonium sulphate which are removed from the atmosphere by precipitation or gravitational settling. The mechanisms by which SO_2 is converted to sulphates or H_2SO_4 aerosols are outlined in Table 3.3.

TABLE 3.3

CONVERSION OF SULPHUR DIOXIDE TO SULPHATES OR SULPHURIC ACID AEROSOLS [78]

Mechanism	Overall reaction	Factors on which sulphate formation primarily depends (in addition to sulphur dioxide concentration)
Direct photo-oxidation	$SO_2 \xrightarrow[\text{water}]{\text{light, oxygen}} H_2SO_4$	Sunlight intensity
Indirect photo-oxidation	$SO_2 \xrightarrow[\text{org.oxidants, } OH]{\text{smog,water,}NO_x} H_2SO_4$	Organic oxidant concentration, OH,NO_x
Air oxidation in liquid droplets	$SO_2 \xrightarrow{\text{liquid water}} H_2SO_3$ $NH_3 + H_2SO_3 \xrightarrow{\text{oxygen}} NH_4^+ + SO_4^=$	Ammonia concentration
Catalyzed oxidation in liquid droplets	$SO_2 \xrightarrow[\text{heavy metal ions}]{\text{oxygen, liquid water}} SO_4^=$	Concentration of heavy metal (Fe,Mn) ions
Catalyzed oxidation on dry particles	$SO_2 \xrightarrow[\text{carbon,water}]{\text{oxygen, particulate}} H_2SO_4$	Carbon particle concentration (surface area)

The formation of H_2SO_4 aerosols and other sulphates resulting from the oxidation of atmospheric SO_2 account for between 5 and 20% of the total suspended particulate matter in urban air [101]. Formed mostly through secondary chemical reactions of atmospheric sulphur compounds, suspended sulphate aerosols are believed to be among the air pollutants most damaging to human health (Table 3.4).

TABLE 3.4

ESTIMATES OF ADVERSE HEALTH EFFECTS OF AEROSOL ACID SULPHATES [78]

Effect	Threshold concentration (micrograms per cubic meter)	Duration of exposure
Increased daily mortality (four studies)	25	24 hours or longer
Aggravation of heart and lung disease in elderly (two studies)	25	24 hours or longer
Aggravation of asthma (four studies)	6-10	24 hours or longer
Increased acute respiratory diseases in children (four studies)	13	Several years
Increased risk of chronic bronchitis		
Cigarette smokers	15	Up to 10 years
Nonsmokers	10	Up to 10 years

The average lifetime of the SO_2 molecule in the atmosphere is about 4 days, and a number of mechanisms operate in aiding reaction, dispersal and removal of the gas. Land and water surfaces absorb SO_2 and, as indicated previously (Figure 3.1), removal of the gas from the atmosphere can be achieved by wet precipitation processes (washout and rainout from clouds), and by dry deposition (wind impaction and settling on vegetation). The latter process probably accounts for the removal of up to 80% of atmospheric SO_2 by surfaces of materials, waters, soils and vegetation.

Many questions remain unanswered regarding the reactions and role of SO_x in the environment. "Air Quality Criteria for Sulphur Oxides" [101], issued in accordance with the American Clean Air Act amendments of 1967, summarises current scientific knowledge for SO_x, and also indicates deficiencies in this knowledge and the needs for future research in this regard. The criteria are compiled as "useful statements of the effects that can be predicated when sulphur oxides are present in the atmosphere; they are derived from a careful evaluation of what has so far been reported".

3.1.3 Ambient Levels of SO_2

Natural background concentrations of SO_2 range between 0,0001 and 0,001 ppm. Ambient SO_2 measurements in urban atmospheres may vary over a range of 3 orders of magnitude [102], depending on parameters such as proximity to SO_x sources, or wind direction. Short period averages are sometimes considerably greater than annual and 24 hour averages, and as some adverse effects of SO_2 are associated with peak concentrations, distribution plots, showing the percentage of the time that SO_2 concentrations are above certain levels, give a more complete picture of the SO_2 concentration patterns at a given site.

Measurement of SO_2 is usually considered synonymous with total gaseous sulphur pollution [103], and a range of analytical techniques and sampling methods has been developed. Networks of air quality monitoring sites supply data for evaluation of emission trends and ambient concentrations of pollutants, and in the United States monitoring programmes are carried out by the National Air Surveillance Network (NASN), the Continuous Air Monitoring Program (CAMP), and other organisations. NASN covers over 200 sites on a 24 hour sampling basis, CAMP records 5 minute averages in 6 major cities. NASN data show annual average concentrations ranging from 0,002 to 0,17 ppm; the highest 24 hour average concentration, 0,38 ppm being found in

New York City. CAMP records over 6 years show annual average concentrations of 0,01 ppm in San Francisco, and 0,18 ppm in Chicago [101]. In the United Kingdom, the Warren Spring Laboratory carries out 24 hour measurements of atmospheric SO_2; in polluted areas the concentration ranges from 0,02 to 1,1 ppm (50-3 000 $\mu g/m^3$), though as reported by the National Society for Clean Air [104], concentrations greater than about 0,4 ppm are now rarely encountered.

In South Africa research on air pollution and its causes was initiated in 1955, and 10 years later, the Atmospheric Pollution Prevention Act (No.45 of 1965) was promulgated. This act provides for the control of four kinds of air pollutants: noxious or offensive gases, smoke, dust, and fumes from vehicles. Monitoring of air pollutants is carried out in the major cities, and statistics on smoke and SO_2 pollution are published from time to time by the Council for Scientific and Industrial Research [105]. SO_2 monthly averages and maximum concentrations each month are obtained at a number of sites, and observations show that maximum concentrations may rise to about 0,1 ppm (250 $\mu g/m^3$) for a 2-day period [7]. Table 3.5 indicates average concentrations for the period October 1975 to September 1976, as measured in Cape Town, Port Elizabeth, Bloemfontein, Pretoria and Durban. With the exception of Cape Town, average winter values are greater than those for summer. Highest monthly concentrations of over 0,035 ppm (100 $\mu g/m^3$) recorded during this period, occurred in Cape Town and Durban.

TABLE 3.5

SO_2 AVERAGE CONCENTRATIONS ($\mu g/m^3$)
FOR 5 SOUTH AFRICAN CITIES [106]

	Cape Town	Port Elizabeth	Bloemfontein	Pretoria	Durban
summer	28	18	11	12	16
winter	21	19	29	29	38
year	24	18	20	21	27

SO_x criteria [101] serve as a base for the ambient air quality standards for SO_2 in the United States. Standards for SO_2, published by the Environmental Protection Agency in 1971, are detailed in Table 3.6: primary standards are set to protect public health, secondary standards are necessary to protect public welfare from any known or anticipated adverse effects. Questions have been raised concerning the specific values chosen for these standards and these values have become a subject of great controversy in the United States [107]. The annual report for 1974 of the Council on Environmental Quality [108] however, notes the conclusion of the Department of Health, Education, and Welfare, that data on health effects indicate "no basis for relaxation of the present standards for sulphur oxides at this time".

TABLE 3.6

U.S. AMBIENT AIR QUALITY STANDARDS FOR SO_2

Averaging Time	Primary Standards	Secondary Standards
Annual Arithmetic Mean	0,03 ppm (80 $\mu g/m^3$)	0,02 ppm (60 $\mu g/m^3$)
24 hours*	0,14 ppm (365 $\mu g/m^3$)	0,10 ppm (260 $\mu g/m^3$)
3 hours*	—	0,5 ppm (1300 $\mu g/m^3$)

* not to be exceeded more than once per year.

3.1.4 Effects of Atmospheric SO_2

Visual range in the atmosphere is reduced by the scatter and absorption of light by particles in the size range 0,1 to 1μ radius, and, as about 80% by weight of the particulate sulphates found in urban air have radii less than 1μ, these suspended sulphates can make a significant contribution to reduction in visibility. Metal corrosion rates show increases in atmospheres

of relatively high levels of SO_2 (0,12 ppm); particulates, high humidity and temperature are also important factors in these reactions. Deterioration of various building materials results from the presence of atmospheric SO_x; the tensile strength of fibres may be weakened, and the fading of dyes is caused in some fabrics.

Annual average concentrations of above 0,03 ppm SO_2 may cause injury to sensitive plants. Phytotoxic concentrations affect many species of plants, including conifers and other forest trees, ornamental and agronomic crops, lichens, and fungal pathogens. Two general categories of response to SO_2 can be distinguished: visible effects and subtle effects [4]. In addition, the interaction of SO_2 with other pollutants may cause foliar and growth effects which are greater than the effects produced by SO_2 alone. These considerations, and others pertaining to types of injury, factors affecting plant response, and the possible mechanism of the toxic action of SO_2 are discussed in section 3.2.

Epidemiological and clinical studies have correlated respiratory diseases and air pollution, notably SO_2 dosages, and in several air pollution episodes SO_2 has been suspect. Sensitive individuals show detectable changes in pulmonary function at SO_2 concentrations of 1 ppm, however, most people will experience mild chronic respiratory irritation at concentrations above 5 ppm, and eye irritation at 20 ppm [109]. Threshold limit value (representing the highest concentration to which workers can be exposed repeatedly without adverse effect [79], is generally accepted as 5 ppm in air. Nevertheless, the levels of a pollutant at which effects are detectable are not necessarily those concentrations at which effects on health are evident. Adverse health effects (such as broncho-constriction) of varying severity, even death, are observed in associations of SO_2 and particulate matter. Conclusions drawn in the SO_x

criteria document [101], regarding effects on health, reveal that an increased daily death rate may occur at concentrations of about 0,25 ppm SO_2 (24 hour mean), accompanied by smoke concentrations of 750 $\mu g/m^3$, and that at concentrations of about 0,05 ppm SO_2 (annual mean) accompanied by smoke concentrations of about 100 $\mu g/m^3$, increased frequency and severity of respiratory diseases in school children may occur. These synergistic reactions increase the adverse effects of SO_2 on human health and underline the necessity for limiting emissions of atmospheric pollutants.

3.2 SULPHUR DIOXIDE AND PLANT RESPONSE

The toxic nature of SO_2 was established in the latter years of the 19th century. Investigators in Germany were responsible for much of the earliest work on the oxides of sulphur, and in America, during the first decades of this century, the deleterious effects of SO_2 on vegetation were noted especially around smelter sites. Litigation over damage caused by smelter fumes took place, investigations continuing over some years. Fundamental knowledge of relative susceptibility, and the symptoms and physiological effects of SO_2 on different plants was acquired during this period. Advances in analytical methods and instrumentation were made; a continuous and automatic recorder for SO_2 analysis was designed, a method of controlled fumigation of plants developed, and work initiated on field plots of vegetation. That both duration of exposure and the concentration of SO_2 were important in determining the extent of injury to plant species had become apparent from these studies, and environmental factors affecting sensitivity were also noted. The theory of "invisible" injury (which proposed that growth rate and assimilation of CO_2 could be reduced by atmospheric SO_2 without the presence of any visible foliar lesions), became the subject of a long-standing controversy, and led to extensive fumigation experiments being carried out.

In 1973, the E.P.A. published a revised chapter of the Air Quality Criteria for SO_x, dealing with the effects on vegetation [4]. Two general categories are distinguished, visible effects and subtle effects, both being considered as physiological disturbances. Subtle effects, however, are not visibly identifiable and result in measurable growth or physiological changes in the plant. The problem of the influence of low concentrations of SO_2 on plant life, often manifest as subtle effects, is of great importance, as significant economic loss occurs annually in crop yields and in injury to ornamental plants and forest trees.

The following sections of this chapter provide a review of the current knowledge of SO_2 effects on plants. Visible symptoms of injury, metabolic changes, and factors affecting susceptibility are discussed.

3.2.1 Visible Symptoms of SO_2 Injury

Leaves provide the primary sites for gaseous exchange in plants, and are vulnerable to attack by atmospheric SO_2. Injury patterns develop on sensitive plants which are characteristic, but not specific for SO_2. Symptoms vary for different species of plant with regard to location, shape and colour of injured tissue. In addition, similar markings may be produced by other agents; the problems associated with accurate diagnosis in the field have been mentioned previously (section 2.4). Nevertheless, leaf injury symptoms are the principal basis for the identification of the adverse effects of SO_2 on plants, and an extensive literature [15,16,20,110-113], including pictorial atlases [21-23], provides descriptions and illustrations of injury to numbers of different types of plants. Young leaves, undergoing expansion, are rarely affected, while fully expanded (middle aged) leaves are most sensitive. The stage of growth of the plant and various environmental factors described later (section 3.2.3), also influence the nature and degree of injury produced.

Visible effects of SO_2 on leaves can be classed as those due to acute or chronic injury, associated with the toxicity of different dosages. The rapid absorption of toxic concentrations of SO_2 results in acute injury, while low concentrations absorbed over long periods cause chronic injury. Biochemical reactions and toxic action of SO_2 in leaf tissue are discussed in section 3.2.2. Acute injury effects typically appear as bifacial lesions between the veins and at the margins of broad-leafed and parallel-veined plants, and on the tips of needle-like leaves. The colour of necrotic tissue varies from ivory or light tan to red brown, and may cover fairly extensive areas of the leaf. The separation of healthy tissue from necrotic areas is usually distinct. Chlorosis, a yellowing of the leaves, appears sometimes from lower to upper surfaces of broad leaves, and at the tips of needle-like leaves; chlorotic or reddish brown flecks are also indicative of chronic injury. Both necrotic and chlorotic signs may occur on the same plant, or on the same leaf.

Microscopic examination of acutely injured tissue shows the initial stages of injury occurring in the spongy mesophyll cells closest to the lower epidermis [114]. The diffusion of chlorophyll into the cytoplasm results in the appearance of flaccid areas of dull grey green colour, followed by plasmolysis of cells, and subsequent desiccation and bleaching [115]. Chlorotic symptoms are evidenced by chloroplast destruction, and death of single cells in the mesophyll sometimes occurs [21]; leaves remain turgid.

Continual exposure to concentrations up to 0,2 ppm was previously accepted as a critical limit below which most higher plants would not be acutely injured, however, recent studies [43] have shown foliar injury occurring at dosages of 0,05 ppm for 1 hour. Instances of synergistic effects have also been reported (section 3.2.3). The E.P.A. criteria document for SO_2

has indicated [4] that sensitive species may be injured by short term exposures of 0,05 to 0,5 ppm (131 to 1 316 $\mu g/m^3$) over periods of 8 hours, or 1,0 to 4,0 ppm (2 620 to 10 480 $\mu g/m^3$) over periods of ½ hour. Controversy persists as to whether more importance should be attached to high concentration exposures over short periods, or to average levels over longer time periods, though short term concentrations are generally regarded as being of greater significance.

3.2.2 Effects of SO_2 on Plant Metabolism

Both acute and chronic exposures to SO_2 can alter plant function, and in addition, recurring sublethal exposures of short duration may modify plant response to succeeding episodes. Stunting of growth or lack of vigorous growth, early senescence, and reduced yield are some of the indications of physiological disturbances caused by SO_2. Comparative studies under controlled conditions have provided information on reduced yield and growth of plants in polluted atmospheres, and the earliest SO_2 fumigation studies showed that a decrease in yield was correlated with a definite amount of leaf destruction [116,117]. No significant reduction in yield was found if the leaf area of visible markings was less than about 5%. Extensive photosynthetic experiments were begun in 1937 [118], and indicated that carbon assimilation was not reduced by a significant amount in the absence of visible injury, though a temporary lowering of photosynthesis was noted when a threshold concentration was exceeded [119]. Such effects, however, were reversible when SO_2 fumigations were discontinued. These experiments were undertaken primarily to establish whether "invisible injury" existed.

As quoted by Thomas [119], the concept of invisible injury, developed by Stoklasa in 1923, was considered to be associated with a reduction in photosynthetic activity, buildup of sulphates in leaves, early senescence, overall unthrifty appearance without actual leaf lesions, reduced growth and yield, and increased

susceptibility to disease and invasion of pests. The disturbance of biochemical processes, occurring before external symptoms are identifiable, has been variously referred to as "invisible", "hidden", or "physiological" injury. No standardised definition has been adopted and consequently a range of interpretations is found in the literature. As stated in the criteria document for SO_x, physiological effects include both visible and subtle effects, originating at the molecular level [4]. In causing disturbances in physiological processes, SO_2 interferes with the permeability of cellular membranes and with enzyme activity in cells; however, the precise mechanism by which plants are injured remains poorly understood. Possible biochemical reactions involving SO_2, the toxic action of SO_2 in plant cells, and observations which have been made on the effects of SO_2 on general growth patterns, photosynthesis, respiration and other metabolic processes are outlined below.

Sulphur is an essential constituent of most plant proteins and their amino acid derivatives, and may enter plants through the leaves as SO_2 or through the roots as sulphate [111,120]. Sulphur derived from SO_2 may supply up to 40% of the requirement of the plant [13], and can be rapidly translocated throughout the plant; however, chlorotic signs appear as the S content of the leaf approaches 2% [118]. The presence of a high S content in leaves [110] can be associated with injury resulting from chronic exposure to SO_2. Beneficial effects of low concentrations of SO_2 have also been noted in some instances for S-deficient crops [121]. In considering nutritional and damaging effects of SO_2, Pahlich [122] has suggested that specific reponses to pollution should be interpreted as varying modes of transport and deposition of accumulated ions, rather than as metabolic adaption.

SO_2 is easily absorbed by plant leaves and, entering through stomata, the gas dissolves on the surfaces of the mesophyll cells.

In aqueous solution SO_2 dissociates according to the equations,

$$SO_2 + H_2O \rightleftarrows H^+ + HSO_3^- \rightleftarrows 2H^+ + SO_3^{=}.$$

The distribution of sulphite, bisulphite and sulphurous acid is determined by the pK values; at low pH (<3) H_2SO_3 is found, and the ions predominating at intermediate pH are bisulphite, and at high pH (>8), sulphite. As toxicity of an aqueous solution of SO_2 is determined by the amount of gas absorbed and the pH of the solution, the buffering capacity of cells is important. More acid can be buffered at low concentration fumigations with SO_2 for an extended time than at high sudden concentrations [123].

The toxic agent in plant injury appears to be sulphite, and mechanisms resulting in a rapid removal of sulphite aid in the reduction of toxicity. Sulphite can be oxidised to less toxic sulphate ($SO_3^{=}$ is estimated [120] to be 30 times more toxic than $SO_4^{=}$), and this oxidative ability of plant systems may be correlated with resistance [124]. Injurious effects are caused when SO_2 is taken up in excess of the capacity of plant tissue to incorporate sulphur into normal metabolic processes. (Both $SO_3^{=}$ and $SO_4^{=}$ are toxic to plant cells if present in excess.) Thus, toxicity of a given dose of SO_2 depends on the rate of absorption of the gas; acute injury results from the rapid absorption of SO_2 at toxic concentrations, and chronic injury occurs when $SO_3^{=}$ is converted to $SO_4^{=}$ at about the same rate at which SO_2 is absorbed.

Injury by SO_2 is local, no systemic effects having been observed [13], though the chemical composition of plants may be changed by exposure to SO_2. Different levels of free amino acids, sulphur, potassium and silicic acid have been reported [4], and total S-content of lichen species growing in polluted areas has been assessed and a quantitative method developed relating accumulated sulphur to mean SO_2 levels [53].

Size and shape of plants especially trees [21,112], can be affected by SO_2. Reductions in tree growth have been observed in experiments conducted to determine the effects of smelter fumes on vegetation [110,125], and results of long-term studies in the mining area of Sudbury, Ontario, described by Linzon [81,126], have shown increased mortality rates for white pine and reduced radial and volume growth. Decreases in forest tree growth and retardation of pine cone production have also been reported [30,127,128]. Recent experiments (for radish [129] and ryegrass [130,131]) have demonstrated growth and yield reductions in the absence of visible injury. Such effects are reported in the revised criteria for SO_x and reflect the best growth data available [4]. In discussing the cumulative effects of chronic exposure to low pollutant levels, Feder [29] has criticised the almost total reliance on acute injury data in the setting of pollutant standards, since depressed growth and yield may occur with or without visible injury symptoms.

Plant growth is extremely complex and depends on a number of interrelated metabolic processes. Photosynthesis and growth are closely connected. Apparent photosynthetic rate decreases at SO_2 concentrations of about 0,2 ppm (as determined for alfalfa [119]) and higher concentrations appreciably inhibit assimilation; ability to regain full photosynthetic activity depending on the length and intensity of the fumigation [118]. Bennett and Hill [132] discuss six major phytotoxic air pollutants which have been shown to reversibly inhibit apparent photosynthetic rate. Figure 3.3 illustrates experimental results obtained for barley and oat canopies after 2 hour exposures to 6 different pollutants.

Exposure to weak acids causes the loss of Mg^{++} from chlorophyll, phaeophytin being formed. This degradation of chlorophyll was shown to occur in lichen thalli after exposure to 5 ppm SO_2 for 24 hours; permanent plasmolysis and bleaching occurred, and chloroplast damage was also observed [37]. Irreversible

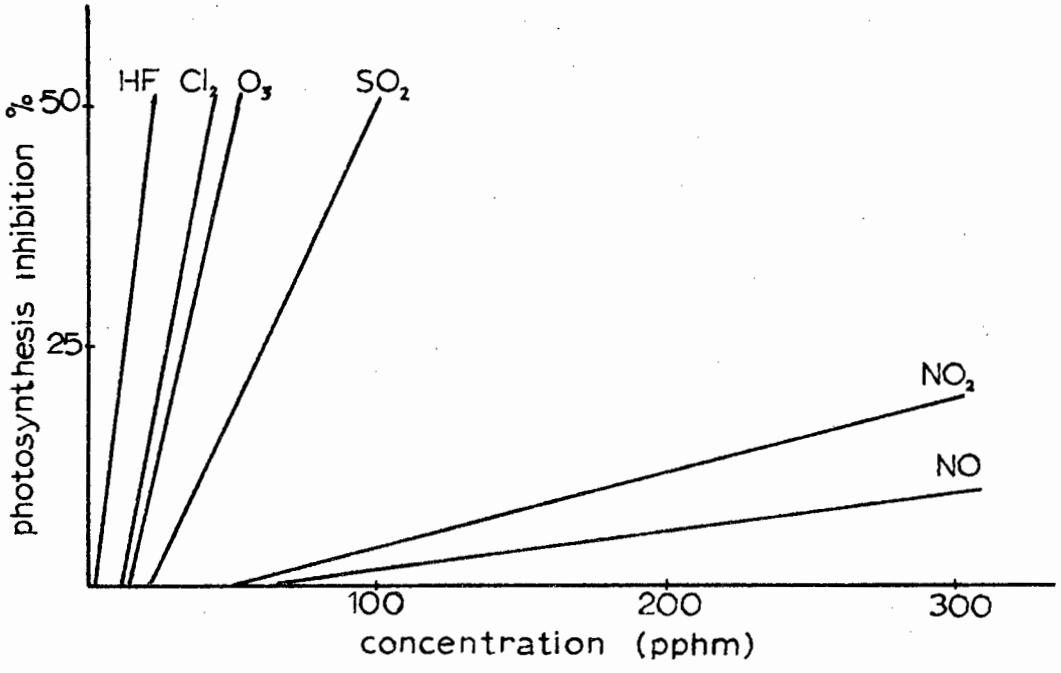


Figure 3.3 Inhibition of apparent photosynthetic rate by phytotoxicants [132]

destruction of chlorophyll (at low pH) is considered instrumental in reducing the rate of net carbon fixation [133]. The development of chlorophyll in plant tissue has been related to SO_2 sensitivity, resistance being noted in plant organisms not containing chlorophyll [110]. Laboratory studies have indicated that stages of increasing interference with photosynthesis by SO_2 can be summarised as, temporary inhibition with subsequent recovery, permanent reduction without chlorophyll breakdown, and permanent reduction associated with chlorophyll breakdown [134].

Under conditions of acute injury internal leaf structure shows marked changes as the cell contents shrink and leaf pigments are destroyed. Microscopically, injury is characterised by plasmolysis of the chlorophyll-containing palisade cells and the spongy parenchyma, and drying of the plasma, leading to a

collapse of mesophyll tissue [15]. Cytological studies, using a light microscope, were reported by Solberg and Adams [114], and recently, Welburn et al. [135], employing electron microscopy, recorded the effects of SO_2 on the ultrastructure of broad bean chloroplasts; subcellular effects occurring in pine needles have also been documented [136]. Swelling of the stroma thylakoids of chloroplasts was observed at low concentrations, increased swelling of the granum thylakoids at higher and prolonged concentrations, and severe chloroplast disruption resulted from exposures to 1 ppm SO_2 for 2 hours [135]. Chloroplast structural injury is more pronounced in old tissues as compared to more metabolically active tissues; young, actively growing tissue retains active chloroplasts at high SO_2 concentrations, indicating the capability of tissue with a high rate of metabolic activity to incorporate more SO_2 into its metabolism than older, less active tissue [136]. These changes in structure of chloroplasts imply an inhibition of the photosynthetic process and it is suggested that low concentrations of aqueous SO_2 (which do not produce visual symptoms) will cause injury at the molecular level, after prolonged exposure by affecting enzyme systems such as chlorophyllase [137].

Studies of enzyme systems have suggested that competition between CO_2 and $SO_3^{=}$ for the bicarbonate site on ribulose 1,5-diphosphate carboxylase results in inhibition of photosynthetic fixation [123]; sulphonation of sulphydryl groups of RUDP also causes reversible reduction of photosynthesis [138]. Conformation changes of enzyme systems, and differential effects of SO_2 on the cytoplasmic and mitochondrial forms of glutamate-oxalacetate-transaminase have been observed [139]. DNA and RNA may also be inactivated by reactions involving sulphite and free radicals [137]. The action of sulphite on membranes requires further interpretation, though studies have indicated that α-hydroxysulphonates may interfere with inner or outer

chloroplast membranes or transport systems associated with chloroplast membranes [140]. Disulphide bonds of proteins can be cleaved by sulphite, and additional compounds are formed in reactions of bisulphite and pyrimidines. The biological significance of these reactions, however, as related to SO_2 pollution effects on plants requires further investigation.

Unspecific alterations of enzyme and membrane activities caused by reaction of SO_2 and its derivatives with metabolically important molecules result in physiological changes in the plant, and various mechanisms have been proposed to determine the injurious action of SO_2. A concept involving an imbalance in equilibrium between sulphydryl groups and oxidised sulphur compounds, attributed to the reducing properties of SO_2, is considered [16] to be the most plausible.

Few studies of the effects of SO_2 on respiration have been reported, though a transitory increase, followed by a decreased rate was observed in lichen studies [141]. Respiration in higher plants is generally not affected before visible injury occurs. Increased transpiration, followed by a decline, has been recorded and reflects stomatal behaviour [123].

Gaseous exchange takes place through stomata of higher plants and under daylight conditions these pores are normally open. Changes in turgor between the guard cells and subsidiary cells are responsible for stomatal movements, and while knowledge of stomatal regulation is fairly precise, interpretations on the mechanisms of operation remain largely hypothetical. Factors influencing stomatal movement (such as light, temperature, CO_2, water content of the leaf) likewise affect the response of plants to SO_2.

Stomatal opening can be either stimulated by SO_2 [142], or closure can result, depending on a range of external conditions. Studies on stomatal diffusive resistance have suggested

that ambient SO_2 levels of about 0,03 ppm (72 $\mu g/m^3$) may increase stomatal opening [143,144]. Consequences of such responses are potentially very damaging as open stomata allow ready access of phytotoxicants to the mesophyll and also result in a rapid rate of water loss. Relative humidity affects stomatal reaction to SO_2; Mansfield and Majernik [145] found that a closing reaction is induced by SO_2 (in the range 0,25 to 1 ppm) at a R.H. of less than 40% at 18°C, and an opening of stomata when R.H. is greater than 40%. Stomatal sensitivity to CO_2 is well documented [146,147], closure being initiated when the concentration rises above the normal level of about 320 ppm. In the presence of SO_2, this stomatal response still operates [148,149]. Differential mechanisms for the reactions to CO_2 and SO_2 have been suggested as SO_2 may be preferentially absorbed by the subsidiary cells causing a decrease in turgor and thereby resulting in an increase in stomatal apperture [144]. Although stomata provide the pathway for entry of pollutants, and the response of plants to SO_2 is mediated through stomatal control, a variety of reactions (both internal and external) contribute to cellular response.

Effects on plant development produced by combinations of SO_2 with other pollutants have been summarised [132,150], though current knowledge is often fragmentary. Various growth disorders of pine trees have been ascribed to SO_2 and O_3 (section 2.4), and additive effects have been observed in growth reductions of foliage and roots of crop species [151], resulting from fumigations of SO_2 and O_3. Synergistic inhibition of photosynthesis of alfalfa by SO_2 and NO_2 was reported recently and was found to be reversible provided tissue injury did not occur [152]. Additive decreases in growth were noted for citrus [153] after exposure to SO_2 and HF. The longterm consequences of combinations of air pollutants affecting plant communities have not received attention.

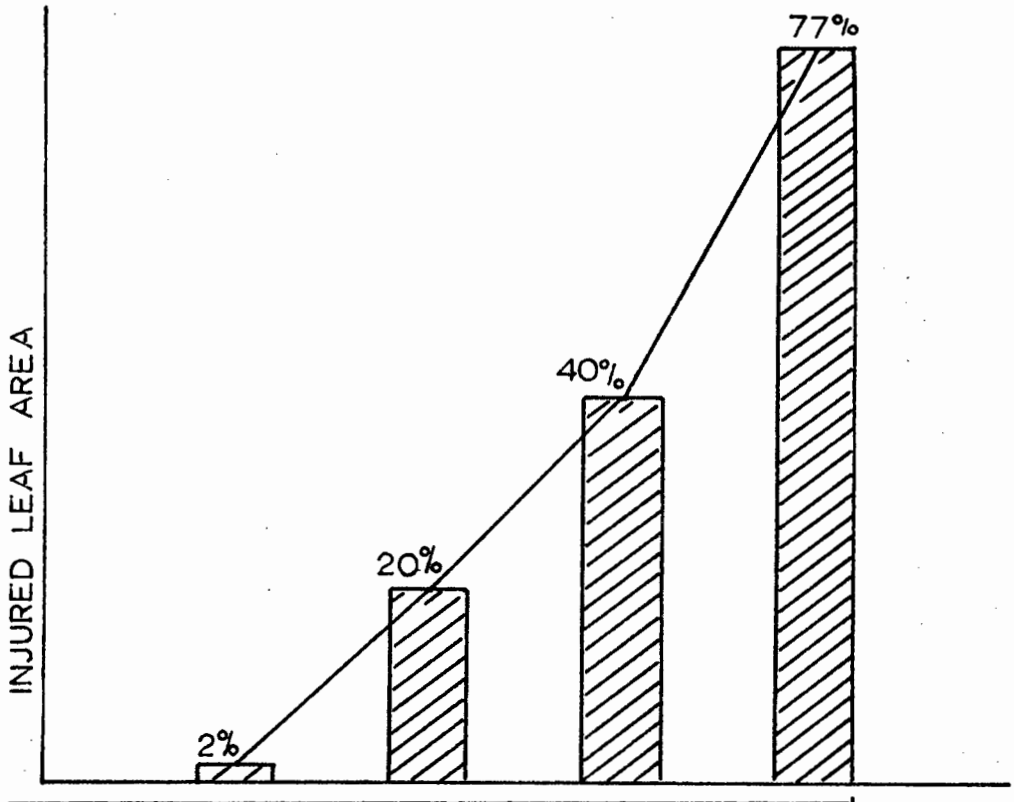
It is evident from the foregoing discussion that, although numerous studies have been undertaken, the extent of impairment of growth and other metabolic processes by sublethal concentrations of SO_2, remain largely unresolved, nor is the mechanism understood by which SO_2 is converted to sulphate in plants.

3.2.3 Factors Affecting the Response of Plants to SO_2

Different families, genera and species of plants vary widely in their susceptibility to SO_2. This susceptibility to injury is influenced by numerous factors, and among the more significant are dosage of SO_2 received by the plant, certain characteristics inherent in the plant itself, environmental parameters, pollutant interactions, and edaphic conditions. Discussion of these and other factors which impinge upon plants during growth and exposure periods is given below. Factors affecting the response of lower organisms are also reviewed.

In considering plant response, a distinction is sometimes made between the terms, susceptible and sensitive. Ting et al. [154] define the age of maximum injury potential as the susceptible age, and describe sensitivity in terms of variation in the degree of leaf injury (related primarily to environmental conditions). As this terminology has not been generally adopted, no differentiation between the two terms is made in this study.

Dosage. SO_2 is not a cumulative air pollutant, and phytotoxic action is related to concentration and the duration of exposure (dosage). Acute and chronic effects are expressions of the dosage received by plants, the rate of absorption of SO_2 by the leaf being of importance. High concentrations over a short period are more injurious than low concentrations over longer periods. Figure 3.4 illustrates degree of acute injury, which does not linearly follow the product of concentration and time, but increases to a greater degree with higher concentration



mg/m^3 (c)	3	6	9	12
hours (t)	12	6	4	3
c x t	36	36	36	36

Figure 3.4 Increase in leaf injury with concentration (c x t constant) [21]

The degree of leaf destruction is more directly dependent on the amount of gas absorbed than on the SO_2 concentration in the air (although these are related), and, with regard to injury sustained under field conditions, the chronological sequence of peak SO_2 concentrations is of importance, as the closer the peaks are in time, the greater the effect. It is generally agreed that sensitive species will be injured when SO_2 levels exceed 0,2 to 0,3 ppm over an 8 hour period; other potentially injurious concentrations which have been suggested are 0,35 ppm for 4 hours, 0,55 ppm for 2 hours, and 0,93 ppm for 1 hour [115]. Phytotoxic effects also become evident when mean annual concentrations exceed about 0,03 ppm.

The concept of an injury threshold is contained in the equation of O'Gara [25]. Data on the relative sensitivity of numbers of plants were obtained over 50 years ago, and were determined by fumigations of plants for one hour with different concentrations of SO_2 to establish the concentration which caused only traces of injury [59].

Plant factors. Differences in response to SO_2 are evident between species, though susceptibility to one air pollutant does not necessarily result in susceptibility to another. Within species, variations are also noted, and species previously considered susceptible have been shown to possess resistant varieties. Plant sensitivity can be viewed as a function of morphological and biochemical characteristics controlled by the genetic plasticity of plants within a population [4].

Genetic composition:

Numerous investigations carried out in the field and in exposure chambers, have resulted in the compilation of lists of plants, classified broadly into 3 categories with respect to susceptibility to SO_2 (resistant, intermediate or sensitive), and based on the severity of foliar injury occurring under specific conditions [15,21,22]. Lichens are among the most sensitive plants [51] and some coniferous trees [43] are more sensitive than other vascular plants.

Genetic variability is a major factor determining plant tolerance, though the sites of action of SO_2 and the reactions involved are not well understood. Many metabolic processes are influenced by SO_2 (section 3.2.2), and the nature and degree of the response are affected by inherent, physiological characteristics of the plant. It is necessary to distinguish between resistant species and resistance within species, as major varietal differences have been noted [84] for some species. According to Taylor [155], the "differential in susceptibility

within a single variety or cultivar may be as great as that between species". Relatively resistant varieties have been found in conifers, notably pine [43] and fir [21], and a range of tolerance observed in tulip [156], petunia [157], poinsettia [158], gladiolus and rose [13]. The genetic make-up of the plant is thus of importance in conferring resistance to injury. Little is known, however, of the physical basis of resistance to SO_2, though a single dominant gene pair has been shown [159] in onion varieties to control sensitivity of the guard cells to ozone. Breeding programmes have been initiated for the selection of resistant strains, and to date have mainly concerned trees and agricultural crops.

Stage of development:

Susceptibility to injury is influenced by the growth stage or the developmental phase of plants when exposed to SO_2. Species shown to be affected at different stages of growth include: conifers, susceptible in the seedling stage [110]; cereal crops (annuals), more resistant in the seedling stage [160]; root crops, susceptible in the early stages of root development and in the seedling stage; beans and tomatoes, susceptible during reproductive growth [21]. Resulting from such variations a "critical development stage" has been determined for some species [21], during which period a high probability exists that leaf injury will result in yield reductions.

Seasonal changes in sensitivity parallel the changes in physiological activity of the leaves. Conifers are more sensitive in summer and spring though, in winter if water is available, needles may remain sensitive. Deciduous trees, after the shedding of leaves, can tolerate strong fumigations, and consequently may be resistant at certain times of the year and sensitive when in leaf. Thus the length of time of leaf retention should be taken into consideration in determining the overall susceptibility of plants exposed to field conditions.

Leaf characteristics:

The age of the leaf is of major importance in determining foliar injury sustained, and studies have shown that leaf buds and young leaves are able to resist fumigations that cause death of older leaves. Recently, Craker and Starbuck [161] observed a differential movement of labelled SO_2 into tobacco leaves of different ages: uptake is high in leaves of intermediate age, and low in older and younger leaves on the same plant. It appears therefore, that most leaves tend to be more resistant either when developing or fully mature, and that newly expanded leaves are most sensitive. Among coniferous trees, however, some variation is evident, and needles of pine and larch become sensitive at early ages.

The leaf is a most variable plant organ, and morphological and anatomical features of leaves need consideration in evaluating the effects of SO_2 on different plant species. Physical properties of leaves are influenced by surface smoothness or roughness, which, in turn result from different venation systems (parallel or reticulate), surface characters of epidermal cells and cuticle, surface wax, exudates, and appendages (hairs and glands). Plant cuticles, of complex chemical composition, vary in thickness, wettability and perviousness, and within the leaf, the area of intercellular air space available for absorption of gas is also variable. Structual characteristics of epidermis and mesophyll of different plants are commonly associated with adaptations to habitat conditions, and affect gaseous exchanges.

The efficiency of stomatal openings as pathways for gaseous diffusion into the leaf is affected by the distribution and size of stomata. Variations in numbers of stomata per unit area are found between species and within any one species (due to the influence of environmental factors during growth), and, where they are more numerous, stomata tend to be smaller [146]. No correlation has been found, however, between susceptibility to

SO_2 and the number of stomata per unit area, though injury is greater when stomata are open. Response of stomata has been shown, measured as decreased stomatal resistance, to occur at low concentrations (about 70 $\mu g/m^3$); increased stomatal resistance has been correlated with leaf age (in experiments on *Vicia faba* plants) [144].

The magnitude of the role of stomata in affecting sensitivity of plants is controversial, and while surface characteristics and internal leaf structure influence the rate of uptake of SO_2, the great variation that exists in susceptibility of different plants cannot be accounted for by these factors alone.

Environmental factors. Plant response may be altered by environmental conditions prior to, during, and (to a lesser extent) after exposure to air pollutants [17]. External conditions which favour stomatal opening may also enhance the possibility of injury by SO_2; such factors include high light intensity, temperatures above 5°C, high relative humidity, and an adequate moisture supply.

Light:

The intensity, quality and duration of light received are of importance to plant activity. As reported by Heck et al. [162] (in porometer studies with the pinto bean), stomata open rapidly in the light at 20 klx and close rapidly in the dark. Provided that the plant is not under moisture stress during exposure to SO_2, susceptibility to injury increases with light intensity, up to full sunlight [163], and conversely, injury is lessened at night. Van Haut and Stratmann [21] consider that plants are up to 4 times more resistant to fumigation in the dark as in the light, but note that exposure to SO_2 during darkness, followed by daylight exposure, may intensify the resultant injury. It is therefore apparent that the effect of light on plant sensitivity to SO_2 involves more than a direct

influence on stomatal movement. Diurnal changes also affect stomatal movements and the greatest sensitivity of alfalfa has been noted [110] between midmorning and early afternoon. An accumulation of carbohydrates in the leaves may contribute to the decrease in sensitivity in the later hours of the day [59], and consequently may provide a basis for the biochemical control of sensitivity.

Temperature:

Plants grown at low temperatures, before exposure to SO_2, appear to be less sensitive than those grown at higher temperatures [162], though variations in temperature between 18^{o} and $40^{o}C$ do not markedly affect susceptibility. Studies of conifer trees in winter have shown a correlation of increased resistance with a lowering of physiological activity of the needles [110]. The effects of temperature and light are difficult to separate and further research is needed in this regard. In general, however, plants are resistant to SO_2 at temperatures below $5^{o}C$.

Humidity:

Conditions which cause moisture stresses in plants also affect stomatal movements. High relative humidity favours the opening of stomata if light and soil moisture are not limiting and results in a high rate of gas absorption. During growth, a positive correlation of humidity with injury can be considered as a response of cell membranes, while stomatal movement due to changes in leaf water potential influences sensitivity during exposure periods [17].

Relative humidity below 70% was thought by Swain [1] to be one of the most important external factors affecting susceptibility of plants to SO_2. Zimmerman [163] reported, however, that above 40% R.H. differences of 20% R.H. had no appreciable effect on sensitivity. A loss in sensitivity of 90% was shown to occur when R.H. was reduced from 100-0% at the time of exposure [59]. In general, it appears that although variations

do exist, sensitivity to SO_2 increases with increased relative humidity.

In the presence of dew, mist or light rain, plant injury can be caused by lower SO_2 levels than during a dry period. A spotted type of leaf injury has been demonstrated in experiments with H_2SO_4 aerosols when leaf surfaces were wetted [164, 165], though no injury was produced by acid aerosols on the surfaces of dry leaves.

Edaphic factors. Any soil factor which adversely affects plant-water relations and reduces uptake of water may, through stomatal effects, limit pollutant absorption. Minor variations in soil moisture have little influence on the sensitivity of plants to SO_2; however, plants grown under conditions of drought are generally less susceptible, and plants at or near the wilting point show increased resistance [110]. According to van Haut and Stratmann [21], this resistance to SO_2 of water stressed plants prevails even under conditions of high relative humidity.

Soil texture and temperature also affect water uptake in plants though correlation with plant sensitivity requires further research. Soil nutrient levels, while influencing plant growth, may further mediate plant response to SO_2; however, studies concerning nutrition and sensitivity have yielded varying results. A reduction in needle injury of white pine was noted [166] after fertilisation with N,P,K; Leone and Brennan [167] found that an excess of S increased sensitivity and an excess of N decreased sensitivity of tobacco and tomato plants. (Nitrogen probably contributes to greater resistance through its action of increasing the capacity for synthesis of S-containing proteins [123].) As a general observation, Heck et al. [162] have suggested that plants grown under low fertility are more sensitive than those grown at high fertility levels.

Pollutant interaction. A wide array of phytotoxicants may occur in ambient air and differing combinations of pollutants can cause effects which may be additive, antagonistic or synergistic. The first studies to consider the possible phytotoxic interactions were initiated in the early 1950's, and the first positive synergistic reaction was reported [168] for Bel W_3 tobacco, injured by exposure to SO_2 and O_3. Further research has extended knowledge and aided in explanations of some of the inconsistencies in results obtained from field and laboratory studies of SO_2 injury responses [129,169].

Factors affecting the response of plants to single pollutants can also be expected to affect pollutant combinations, in addition to which three further variables must be considered: the ratio of the concentration of each gas to the other, the concentration of each gas with respect to the injury thresholds of the individual pollutants, and the method of exposure to the pollutants (i.e. simultaneous, sequential, and/or intermittent) [150].

Foliar lesions resulting from exposures to combinations of SO_2 and O_3 (below the individual injury thresholds) resemble those caused by O_3, similarly, SO_2 and NO_2 combinations produce mimicking of O_3 symptoms [150]. Synergistic effects of SO_2 and NO_2 have been reported by Tingey et al. [170]; the greatly enhanced sensitivities observed, however, could not be confirmed by other workers, though phytotoxicity of several crop species to combinations of SO_2 has been noted at relatively high concentrations of these gases [171]. Some effects of SO_2 and HF on foliar injury were reported [153,172], but little is known of the potential effects of combinations of SO_2 and the photochemical complex.

Research on pollutant interactions has mainly concerned combinations of two phytotoxicants, and although leaf injury symptoms were studied initially, more recently the effects on plant development and productivity have been reported. This aspect was mentioned in section 3.2.2.

Other factors affecting susceptibility. Laboratory and greenhouse investigations have yielded a wealth of information regarding factors which influence pollutant injury, nevertheless a direct relationship to field conditions is desirable for the full interpretation of these findings. Meteorological parameters (turbulence, wind speed, temperature inversions), as well as climatic and seasonal variations significantly influence the dispersion of air pollutants and thereby affect the concentration at ground level. Plants can therefore be exposed to fluctuations in SO_2 concentrations, and injury to certain "spring flowers" and vegetable seedlings in New Jersey has been noted [173], particularly during May, when meteorological conditions are conducive to the accumulation of SO_2 in the lower atmosphere.

Ketellapper [147] drew attention to the concentration of CO_2 within the leaf as a possible factor controlling stomatal action. Raised CO_2 levels suppress stomatal opening of many plant species in the light, and Majernik and Mansfield [148] have shown that this response still occurs in the presence of SO_2. In view of greenhouse growth practices, a need for further investigation of this variable is suggested [17], with respect to the potential protection of plants from SO_2 injury.

While the usefulness of SO_2 as a fumigant and preservative to limit growth of micro-organisms is well established, little attention has been given to the role plant diseases may play in weakening plants and thereby predisposing them to further injury, or conversely, in acting as protectants. Plant sensitivity may consequently be modified by the presence of pathogens which, by inducing changes in the physiology of the host, could promote synergistic or antagonistic reponses. More usually, however, SO_2 is more toxic to the pathogen than to its host, and in this regard reductions in the incidence of diseases as mildew and rust have been observed both in the field and in culture experiments.

Various chemical compounds have shown potential in protecting plants from injury by air pollutants, and the application of certain organic systemic fungicides has reduced susceptibility, most notably in lessening injury from oxidants [174]. Deposits of road dusts on the upper surface of leaves have been reported [175] to afford some protection from SO_2, due to adsorptive properties of particulates, though direct and indirect adverse effects caused by dusts have also been noted [33,34].

Factors affecting the susceptibility of lower organisms to SO_2. Bryophytes and lichens are especially sensitive to air pollution, and the absence of lichens in cities and industrial areas has been correlated with the presence of air pollutants ("pollution hypothesis"). Supporters of a "drought hypothesis", however, considered that this absence of lichens was essentially due to conditions of desiccation prevalent in urban areas. Evidence today indicates that the level of atmospheric SO_2 is the significant factor in determining lichen distribution patterns.

Lichens are combinations of fungi and algae existing in partnership, and absorption of moisture, nutrients, and gases takes place over the whole surface of the plant body; mosses too, lack specialised vascular systems and absorption occurs directly through stems and leaves. Lichens and bryophytes show similar responses to SO_2 pollution, though considerable differences exist in susceptibility of species to injury. Few species are able to tolerate heavily polluted atmospheres, though some species which do survive in such areas can be considered as either toxiphilous or toxitolerant. Sensitivity of certain species provides the basis for techniques utilising lichens as indicators of atmospheric SO_2 pollution (section 2.5).

The interaction of environmental parameters, dosage of SO_2, and the morphology and physiology of individual species modifies sensitivity. As many surveys have revealed [49-54, 176], sensitive epiphytic species are generally affected by annual average SO_2 concentrations of 0,005 to 0,01 ppm (<30 $\mu g/m^3$), while resistant species can tolerate concentrations of about 0,06 ppm (>170 $\mu g/m^3$). The physical state of the pollutant is of importance in affecting the physiological response of these organisms, and according to Saunders [177], SO_2 concentrations of 0,035 ppm (100 $\mu g/m^3$) in air can be considered as equivalent to 35 ppm in aqueous solution.

In studies concerning distribution patterns of lichens the importance of the substrate has been noted. Lichens growing on acid substrates are more sensitive to SO_2 than those growing on basic substrates [49,178]. The order of increasing sensitivity generally is from terricolous to saxicolous to corticolous species, due probably to the acid-buffering capacity of the substrates. (SO_2 is less toxic when buffered at high pH, as in solution at pH < 5 the more toxic bisulphite and sulphurous acid prevail.)

Within the plant body, high pH also aids in reducing SO_2 toxicity and consequently the species most sensitive to SO_2 are those having low buffering capacity for acidic substances [50]. In addition, the moisture content of the lichen thallus affects susceptibility, dry lichens being more resistant [179]; the non-wetting properties of the crustose thallus of *Lecanora conizaeoides* may be a major factor contributing to its remarkable resistance to SO_2.

Thus, a number of factors influence the response of plants to SO_2 and no one factor may be considered independently. Species differ markedly in susceptibility to injury, and although this characteristic is apparently genetically based, cultural and environmental conditions under which plants are grown or exposed to SO_2, and time-concentration relationships, significantly affect sensitivity. For any species, a spectrum of genotypes may exist representing varying susceptibility [30], however, the influence of environmental factors on injury to sensitive genotypes may be less pronounced if specific biochemical or physiological mechanisms involved in susceptibility have weak interactions with the environment.

CHAPTER 4

EXPERIMENTAL

In order to obtain information on the symptoms of acute injury caused to plants by SO_2 and to determine the susceptibility of South African species, a selection of plants was exposed to dosages of SO_2 in a specially constructed fumigation chamber. 63 species of indigenous plants, representing 21 families and 36 genera were chosen for these experiments. The discussion which follows describes the equipment used (specifications are contained in the Appendix), and the procedure adopted for the fumigation of plants.

4.1 EQUIPMENT

The design of chambers for studies of the effects of air pollutants on plants varies widely, from simple arrangements of plastic sheets and air systems [42] to modified plant growth chambers [65]; however, certain basic principles of design must be considered. These features, which have been detailed by Heck et al. [180], provided the guidelines for the design of the exposure chamber used in these studies. Figure 4.1 illustrates the system lay-out. As described below, the chamber is designed for positive pressure and a single-pass flow system, and is controllable with respect to air velocity and toxicant concentration. It is contained in a greenhouse which is naturally illuminated, and operates under ambient conditions of temperature and relative humidity.

4.1.1 Greenhouse

Exposure chamber studies were conducted in a greenhouse, located in the nursery area of the National Botanic Gardens at Kirstenbosch, on a site receiving full sunlight. Measuring 3,1 m x 2,3 m x 2,2 m, the greenhouse is constructed of wood to a height of one metre, and the remaining sections, of fibreglass sheeting, give uniform light transmission and diffusion.

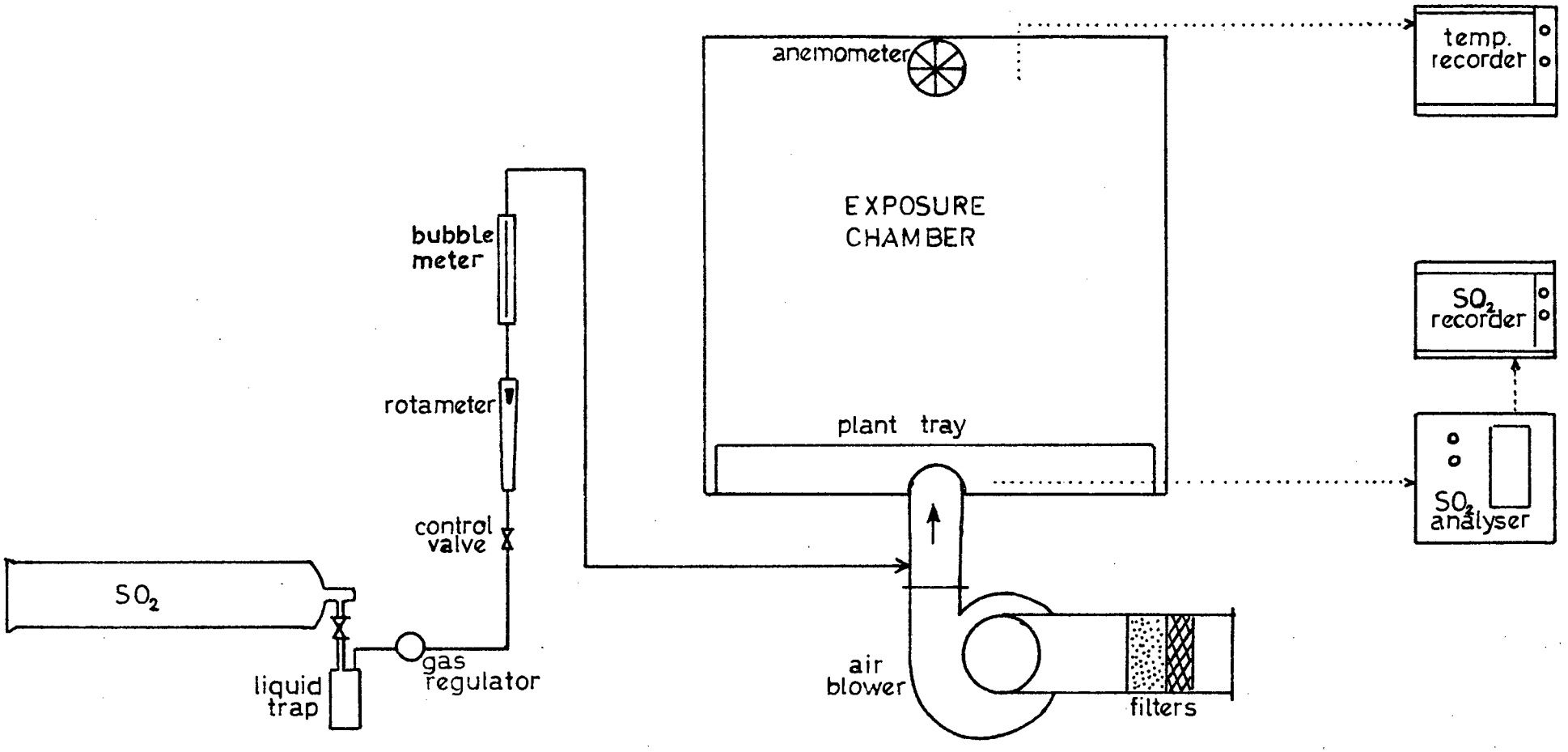


FIG 4.1 EQUIPMENT LAY-OUT

A skylight, 3 windows and 2 doors provide adequate ventilation, and a wooden-slat workbench supports the exposure chamber at a height of 0,7 metres.

4.1.2 Exposure Chamber

The dimensions of the chamber are 940 x 920 x 1 250 mm high, giving an internal volume of approximately 1 m^3. A welded steel angle-iron frame, coated with white epoxy paint, holds the six faces of the chamber, each constructed of perspex sheet having low SO_2 adsorption characteristics and high light transmission. One side is removable, forming the door of the chamber, and is held in place by several brass studs which together with a door gasket, ensure a leak-proof system. At the base of the chamber runners support a 0,85 m^2 expanded metal tray at a height of 20 mm, on which the plants are placed for fumigation.

4.1.3 Air System

The positioning of the chamber inlet and outlet ducts (of 100 mm diameter) provides the single-pass flow system. The centrifugal blower, which supplies air to the chamber, is situated beneath the workbench and draws air from outside the greenhouse. A poly-urethane filter and an activated carbon bed are positioned in the air inlet to retain dust and particulates, and to adsorb gaseous phytotoxicants. Air flow rate is controlled by a butterfly valve which is set to give a rate of approximately 2 m^3/minute. Equivalent to 2 air changes per minute, this supply is sufficient to maintain temperature, R.H. and CO_2 at ambient levels in the chamber. The system is designed to operate at a slight positive pressure. A perspex plate with large holes, placed across the base of the chamber, serves as a distributor for the airflow. (Smoke tests confirmed the uniformity of air distribution in the chamber.) The air outlet duct holds a vane-anemometer to measure the air flow rate. Monitoring probes can be inserted through special openings in the chamber walls.

4.1.4 SO_2 Supply

The gas cylinder, containing liquid SO_2 (>99,9% pure) under pressure, is located outside the greenhouse. The cylinder, operating at boil-off pressure, was placed in the shade to minimise temperature and hence pressure variations. When placed in an upright position, liquid droplets of SO_2 carry over from the cylinder and cause malfunctioning of the gas regulator; it was found essential to mount the cylinder horizontally as the internal gas take-off is then above the level of the liquid SO_2. As a further precaution, a liquid trap was installed between the cylinder and regulator. High pressure flexible hose connects the cylinder to a stainless steel control needle valve adjacent to the chamber, and SO_2 flow rate is measured through a rotameter and a bubble meter. These two separate flow measuring devices are used to facilitate the measuring of the very low flow rates required to give SO_2 concentrations in the 1 to 4 ppm range. SO_2 joins the air supply line through a perforated delivery tube, and good mixing is effected before entry into the chamber.

4.1.5 Monitoring Instruments

SO_2 levels are measured using a Beckman SO_2 analyser linked to a chart recorder and a Casella SO_2 sampler. In those concentration ranges where both instruments operate together (up to 2 ppm), satisfactory agreement is obtained, and compares closely with readings recorded on the rotameter and bubble meter. SO_2 concentration can be monitored at different points in the chamber, and in the inlet and outlet ducts. Due to the low flow rates of SO_2, it is essential that the SO_2 levels in the chamber be checked throughout the fumigation run, so that adjustments to the supply can be made as necessary. (During fumigations, concentrations were maintained within 0,2 ppm of the desired level.)

A copper-constantan thermocouple was constructed and is inserted through the top of the chamber. This thermocouple

measures temperatures in the chamber with reference to a cold junction maintained in ice, and the millivolt output is taken through an amplifier to a chart recorder.

Humidity readings are made using a wet-and-dry bulb thermometer at the chamber outlet; Dräger tubes measuring water vapour can be inserted into the chamber. Dräger tubes for CO_2 are also employed to check chamber levels. Light intensity is measured using a solarimeter and a calibrated photometer.

4.2 PROCEDURE

Controlled fumigations of plants in the laboratory or greenhouse provide information on the resultant type of injury, symptom expression, and the relative susceptibility of different species, and also enable determinations to be made of the pollutant dosages causing injury to plants. Studies involving acute injury utilise varying concentrations in the range 0,25 to 8 ppm over time periods of ½ to 8 hours [62].

This study of the effects of SO_2 on plants indigenous to South Africa, involved fumigations carried out under greenhouse conditions, using dosages of 4 ppm for 1, 3 and 4 hours; 3 ppm for 1 hour; 2 ppm for 1 and 3 hours; 1 ppm for 1 and 3 hours. Initial screening of all plants was made at 4 ppm for 4 hours, and dependent upon the response observed, the subsequent fumigation programme was determined.

As outlined previously (section 3.2.3), susceptibility to SO_2 is influenced by many factors, both genetic and environmental, and in fumigation studies some control of these variables is desirable. Since all fumigations could not be carried out under identical natural conditions, the effect on sensitivity of variations in environmental parameters was minimised by exposing plants to SO_2 only on days when conditions were conducive to producing injury [180]; such situations included high light intensity, high relative humidity, and moderately high

temperatures. Under bright sunlight, conditions within the chamber corresponded closely to the prevailing ambient situation, and measurements made at several points in the chamber were consistent. Typically, light intensity varied between 60 and 90 klx (full sunlight), temperature ranged from 22° to 30°C and R.H. 58-70%. The air flow rate in the chamber provided 2 air changes per minute thereby producing an adequate CO_2 supply over the plants. Conditions in the chamber therefore ensured that stomata were open to allow for gaseous exchange to take place in the leaf tissues [146], and experiments were conducted during the mornings and early afternoon periods when photosynthetic activity was maximal. Soil moisture stress, causing stomatal response, was avoided as plants were well watered prior to fumigation periods.

4.2.1 Fumigations

At the start of a fumigation run, the chamber door was sealed and the air supply introduced into the system. Temperature and relative humidity in the chamber were recorded and, when conditions were stable, SO_2 was added to the system. An equilibration period of between ½ to ¾ hour was found to be necessary to allow for uptake of SO_2 by the chamber materials and during this period, SO_2 concentrations of 3-4 ppm were employed for the purpose of more rapid attainment of steady state conditions. To determine possible uptake of SO_2 by soil and the plastic containers, initial runs were performed using soil-filled plastic containers without plants. After the equilibration period, however, no further uptake of SO_2 by the containers could be detected.

After loading the chamber with plants, the time was noted and the SO_2 addition adjusted to the required level. SO_2 concentration was continuously monitored at the inlet, outlet or at other points within the chamber. Temperature was also recorded continuously, and measurements were made of relative

humidity, air flow rates and CO_2 levels in the chamber. Daylight was used for the illumination of the chamber, and light intensity was recorded throughout the fumigation period both in the greenhouse and in the chamber.

4.2.2 Plants

The relative susceptibility of 63 species was investigated. To some extent the range of plants studied was limited by their availability during the period of the investigation, though consideration was given to the selection of species with regard to different families and genera, life forms, habitat, length of life cycle, and leaf characteristics. Indigenous plants studied included annuals, perennials, succulents, monocotyledonous geophytes, shrubs, and trees; pteridophytes, aquatic plants and grasses were not studied. A wide range of leaf types was selected and included leaves simple, compound, broad, needle-like, glabrous, pubescent, fleshy, succulent, leathery, and those having entire, dentate or rolled margins.

Plants were grown from seed supplied mainly by the National Botanic Gardens, and seedlings were potted in a standard soil mixture (3 sand : 2 leaf mould : 1 soil) in black plastic containers of approximate size 8 x 14 cms. To ensure uniformity of growing conditions for all species, the young plants were located in the nursery at Kirstenbosch in an open area, subjected to prevailing weather influences, and watered and tended regularly. About 30 individuals of a species were made available by the National Botanic Gardens, though fewer numbers, especially of trees and ericaceous plants, could be obtained from other sources. No pot-bound material was used and all plants were in a healthy condition. When used for fumigations, plants were actively growing but were not undergoing reproductive growth. Trees were generally over 1½ years in age, and between 20 and 25 cms in height; ericas were 12 to 20 cms in height and about one year old; succulents were small, up to

8 cms, and about 10 months old; all other plants ranged in size from 10 to 18 cms, and in age from between 5 and 10 months.

The day before each fumigation, sets of plants, together with duplicates and controls, were chosen, well watered, and labelled with details of the proposed experimental run. Records were made, before fumigation, of the overall appearance and leaf characteristics of each plant.

After equilibration of the exposure chamber, plants were placed on the tray at the base of the chamber, the door was sealed and the fumigation period begun. Up to 30 plants could be accommodated in the chamber for each fumigation run, and another complete set of plants was placed in the greenhouse alongside the chamber to act as controls. During fumigation the general condition of the exposed plants was observed and compared with the controls; at this stage, no stress signs were noted. Duplicate plants were used in the exposures, and although obtained from selected, pure seed, and grown and maintained under uniform conditions, some variation in sensitivity occurred between individuals of the same species. Where differences in percentage necrosis were observed, however, the relative susceptibilities determined for individual plants were similar. Experimental runs for any one species were made sufficiently close together so that differences in plant age or growth stage would be minimised and therefore should not influence the response of the species. Control runs, using filtered air only, were also carried out on sets of plants placed in the chamber. Environmental conditions similar to those maintained under SO_2 fumigations prevailed, and in these cases no injury or indications of plant stress were observed. Fumigations were repeated on occasions as checks on the reproducibility of the chamber operation.

Controlled fumigation studies provide information on the visible symptoms of SO_2 injury and enable comparative susceptibilities of different plants to be determined. The technique, however, cannot simulate exactly the response of plants under field conditions, where pollutant interactions, fluctuations in concentrations and other environmental stresses may be operative; data on the sensitivity of species must be considered in relation to the particular situations under which the observations are made.

4.2.3 Injury Assessment

At the termination of the fumigation period, plants were removed from the chamber and replaced in the nursery, together with the control set, and the normal schedule of watering resumed on the day following the fumigation. Visual inspections of the leaves were made at regular intervals, daily for the first week and later, every 3 to 4 days for a period of up to 6 weeks. A magnifying lens was used to examine the lesions more closely, and the development of injury was recorded photographically. Certain injury signs were visible on sensitive leaves within 24 hours of exposure to SO_2 (especially after high dosages), though full development of symptoms required 3 to 4 days. After 5 days the injury assessment, on which susceptibility categorisations were based, was made. Subsequent assessments showed no further increase in foliar lesions, and after about 4 weeks an overall evaluation of injury to the plant became unreliable due to increases in vegetative growth.

Details were noted of the type, colour and location of necrotic markings on the leaves, age of leaves injured, and the percentage of the leaves of the whole plant affected. Necrosis of the 3 most severely injured leaves was estimated, and the average percentage necrosis of these 3 leaves was used as a basis for rating injury [61] and for the compilation of an injury index. According to the observed percentage foliar

necrosis, five degrees of injury were distinguished on a scale of 0 to 4 (Table 4.1). If only one or two leaves of the entire plant were injured the injury index still reflected a 3 leaf average as zero scoring was included.

TABLE 4.1 INJURY INDEX

Ave. % Foliar Necrosis	Injury Index
75 - 100	4
50 - 75	3
25 - 50	2
1 - 25	1
0	0

Different dosages (SO_2 concentration x exposure time) resulted in foliar injury of varying severity and, based on the injury occurring at certain dosages, plants were categorised as sensitive, intermediate, or resistant to SO_2 [17]. Table 4.2 indicates the requirements for these 3 categories. In order to allocate the susceptibility category of a species, compliance with at least three of the five injury indices given in each row of the table was required.

TABLE 4.2 DOSAGE/INJURY INDEX TO DETERMINE SUSCEPTIBILITY CATEGORY OF SPECIES

ppm hours	2 1	3 1	4 1	2 3	4 3	Category
Injury Index	⩾1	⩾2	⩾3	⩾3	4	Sensitive
	0	1	2	1-2	2-3	Intermediate
	0	0	0-1	0	0-1	Resistant

Dose-injury relationships may also be expressed in terms of mathematical equations. These equations, whose limitations have been described in section 2.6, require the testing of large numbers of plants of the same species for the evaluation of data; in this investigation such an approach could not be made.

CHAPTER 5

RESULTS AND DISCUSSION

Over 60 species of indigenous plants have been studied and the results of the SO_2 fumigations are presented, together with appropriate susceptibility classifications of the species. A wide range in sensitivity was observed, and plants have been broadly classified as resistant (R), intermediate (I), or sensitive (S) to SO_2 according to the extent of foliar necrosis sustained. Symptoms of acute injury are described and 21 species are illustrated by colour photographs. Additional representation of dose-response data in 3-dimensional form is given for selected species.

Sensitivity to SO_2 is primarily a function of the inhibition of metabolism, initial reaction taking place in the mesophyll cells of the leaf, and is moderated by the morphology and physiology of the plant species. Differences in leaf anatomy affect gaseous exchange in plants and also influence the foliar sorption of air pollutants, although many additional factors are involved [66,75]. Plants which are adapted to arid conditions are often less sensitive to SO_2[86,182], and rough, pubescent leaves have been shown to adsorb greater amounts of particulates and aerosols than smooth leaves [183]. The presence of various types of protuberances and exudates on leaf surfaces also influences the uptake of gaseous pollutants from the atmosphere [65]. In this investigation therefore, attention was also given to the anatomical features of leaves in relation to the susceptibility of plants to SO_2 injury.

5.1 EVALUATION OF INJURY

At present no standard procedure exists for assessing plant injury caused by exposure to SO_2. Various methods of evaluation are in use ranging from simple descriptive terms of presence or absence of visible injury (as severe, moderate, slight, trace,

zero), to numerical scales of injury based on degrees of foliar necrosis. Attempts to relate growth effects to pollutant injury consider several growth phenomena: photosynthetic and respiration rates, dry and fresh weight, transpiration rate, growth in length and leaf area, and relative chlorophyll content; statistical methods are often used in evaluating such effects, though no uniformity has been achieved in defining hidden injury. The E.P.A. criteria document [4], in summarising the responses of vegetation to various SO_2 dosages, notes that the effects observed "are reported differently" by investigators. Comparison of results is therefore difficult, nor has an adequate quantitative method been designed which combines utility with accuracy.

Acute foliar injury is characterised by necrotic lesions and assessments of this injury may consider percentage of total leaf surface affected, percentage of leaf length showing necrosis, percentage of leaves injured, or percentage of a given sensitive area injured. The assessment is largely subjective despite efforts at quantification. Estimates of percentage foliar necrosis are often employed in constructing an injury rating system, in which various levels of injury are distinguished, ranging from those based on a 0 to 100 scale, to a broad subdivision of 0%, <50%, >50%. Standard terminology needs to be developed and a uniform index system applied. In some major studies, techniques for measuring exposed leaf area and necrotic area, employing grids or mechanical planimeters and photoelectric methods, have been developed or adapted. Heck [48], however, comments that the workload in obtaining an accurate estimation of leaf area injury is out of proportion to the slightly more accurate data obtained.

In these experiments, injury was assessed by estimating percentage leaf necrosis of the 3 most severely injured leaves of each plant, and an injury index compiled as described in section 4.2.3. The injury index is therefore a measure of the severity of injury caused to susceptible leaves, and provides

a convenient method for the evaluation of injury, and is of practical use in field surveys. Injury indices for the 63 species studied are given in Table 5.1 and are derived from average values of percentage necrosis for duplicate plants. In addition, percentage of the whole plant affected was estimated and this figure was used in the calculation of the overall degree of plant injury for each dosage (presented in section 5.3). The system of Engler [181] has been followed in placing the plant families in sequence in Table 5.1, an arrangement widely adopted in South African herbaria.

TABLE 5.1

SO_2 FUMIGATION RESULTS FOR SOUTH AFRICAN SPECIES:

DOSE-RESPONSE EXPRESSED AS INJURY INDEX

Species	Injury Index				hrs	Susceptibility Category
	4 ppm	3 ppm	2 ppm	1 ppm		
Podocarpaceae						
Podocarpus falcatus	0		0		3	R
(Thunb.) R.Br. ex Mirb.	0	0	0		1	
Cupressaceae						
Widdringtonia nodiflora	1		0		3	R
(L.) Powrie	0	0	0		1	
Ulmaceae						
Celtis africana	4		0		3	R
Burm.f.	1	0	0		1	

Species	Injury Index 4 ppm	3 ppm	2 ppm	1 ppm	hrs	Susceptibility Category
Proteaceae						
Aulax cancellata	4		4	0	3	S
(L.) Druce	4	2	0		1	
Aulax umbellata	4		3	0	3	S
(Thunb.) R.Br.	3	2	0		1	
Leucadendron conicum	4		4	1	3	S
(Lam.) Williams	4	2	1		1	
L. coniferum	4		4	0	3	S
(L.) Meisn.	4	3	0		1	
L. laureolum	4		4	0	3	S
(Lam.) Fourcade	4	3	0		1	
L. meridianum	4		3	1	3	S
Williams	3	2	1		1	
L. microcephalum	4		4	1	3	S
(Gandoger) Gandoger & Schinz.	4	2	0		1	
L. procerum (Salisb. ex Knight)	4		2	0	3	I
Williams	2	1	0		1	
L. rourkei	4		2	0	3	I
Williams	2	1	0		1	
L. rubrum	4		1	0	3	I
Burm.f.	2	1	0		1	
L. spissifolium (Salisb. ex Knight)	4		3	1	3	S
Williams ssp. *fragrans* Williams	2	2	0		1	
L. uliginosum R.Br.	4		4	0	3	S
ssp. *uliginosum*	3	2	1		1	
L. xanthoconus	4		3	0	3	S
O. Ktze. K. Schum.	4	3	0		1	
Protea acuminata	4		4	2	3	S
Sims.	4	2	1		1	
P. obtusifolia	4		2	0	3	I
Buek. ex Meisn.	2	1	0		1	
P. laurifolia	4		2	1	3	I
Thunb.	2	1	0		1	
P. repens	4		4	2	3	S
(L.) L.	3	2	0		1	

Species	Injury Index 4 ppm	3 ppm	2 ppm	1 ppm	hrs	Susceptibility Category
Aizoaceae						
Astridia hallii	0				3	R
L. Bolus	0	0	0		1	
Lampranthus amoenus	0				3	R
(Salm.) N.E.Br.	0	0	0		1	
Ochnaceae						
Ochna serrulata	4		0		3	R
(Hochst.) Walp.	2	0	0		1	
Leguminosae						
Indigofera filifolia	3		2		3	I
Thunb.	2	1	0		1	
Podalyria sericea	1				3	R
R.Br.	0	0			1	
Schotia brachypetala	4		0	0	3	R
Sond.	1	0			1	
Virgilia divaricata	4		4	2	3	S
Adamson	4	4	2		1	
Geraniaceae						
Pelargonium angulosum	3		2	1	3	I
Ait.	2	2	1	0	1	
P. betulinum	4		3	2	3	S
(L.) Ait.	3	3	2	0	1	
P. capitatum	3		2	1	3	I
(L.) Ait.	2	2	1	0	1	
P. graveolens	3		2	1	3	I
Ait.	2	2	1	0	1	
P. peltatum	4		3	1	3	S
(L.) Ait.	4	2	2	0	1	
Meliaceae						
Ekebergia capensis	0		0		3	R
Sparrm.	0	0	0		1	
Polygalaceae						
Polygala myrtifolia	1		1		3	R
L.	1	0	0		1	
Anacardiaceae						
Rhus lancea	4		4	1	3	S
L.f.	4	3	3		1	
R. lucida	4		2	0	3	I
L.	2	1	0		1	

Species	Injury Index 4 ppm	3 ppm	2 ppm	1 ppm	hrs	Susceptibility Category
Flacourtiaceae						
Dovyalis caffra (Hook.f. & Harv.) Hook.f.	0 0	0	0 0		3 1	R
Umbelliferae						
Heteromorpha arborescens Ch. & Schel.	2 2	2	2 0		3 1	I
Ericaceae						
Erica baccans L.	2 1	0	1 0		3 1	R
E. glandulosa Thunb.	4 1	1	3 0	0	3 1	I
E. glauca Andr. var. *glauca*	3 1	0	2 0		3 1	R
E. mammosa L.	3 1	0	1 0		3 1	R
E. patersonia Andr.	2 0	0	1 0		3 1	R
E. sessilifolia L.f.	3 0	0	2 0		3 1	R
Gentianaceae						
Chironia baccifera L.	2 2	1	1 0	0	3 1	I
Orphium frutescens (L.) E.Mey	0 0	0	0 0		3 1	R
Boraginaceae						
Lobostemon fruticosus (L.) Buek.	4 3	3 3	3 1	1	3 1	S
Labiatae						
Leonotis leonurus R.Br.	3 3	1	2 1	1	3 1	I
Plectranthus ecklonii Benth.	3 3	1	2 0	2	3 1	I
Salvia aurea L.	3 2	2	2 1	0	3 1	I

Species	Injury Index				hrs	Susceptibility Category
	4 ppm	3 ppm	2 ppm	1 ppm		
Selaginaceae						
Selago serrata	4	3	2	1	3	I
Berg.	2	1	1		1	
S. thunbergii	4	3	3	1	3	S
Choisy	4	2	1		1	
Campanulaceae						
Lobelia pinifolia	3		2	0	3	I
L.	2	1			1	
Compositae						
Cotula turbinata	4	4	4	4	3	S
L.	4	4	4	2	1	
Dymondia margaretae	0		0	0	3	R
Compton	0	0	0		1	
Euryops pectinatus	4		3	1	3	S
(L.) Cass.	4	2	0		1	
Gazania linearis	2		1	0	3	I
(Thunb.) Druce	1	1	0		1	
Helichrysum maritimum	3		1	0	3	I
(L.) D.Don ex G.Don	2	1	0		1	
Leyssera gnaphaloides	4	4	4	4	3	S
L.	4	4	4	1	1	
Osteospermum oppositifolium	4	4	4	4	3	S
(Ait.) Norl.	4	4	4	1	1	
Iridaceae						
Watsonia bulbillifera	4		3	1	3	S
Math. & L. Bolus	4	1	1		1	
W. fergusonae	4		3	0	3	S
L. Bolus	3	2	0		1	
W. fourcadei	4		3	0	3	S
Math. & L. Bolus	3	2	0		1	

5.2 FOLIAR INJURY DESCRIPTIONS

The range of South African plants studied showed considerable differences in susceptibility to SO_2; some species were entirely resistant, although fumigations were performed under environmental conditions likely to render plants sensitive to injury, while other species were injured even at low dosages. (Three susceptibility categories have been detailed in section 4.2.3.) The development of acute injury followed the pattern typical of plants exposed to short term, high SO_2 concentrations, and in the following section, types of foliar injury are described for the individual species studied. These descriptions are grouped broadly as monocotyledonous plants; gymnosperms; tree, shrub, and herbaceous species; and, in some instances, as plant families. Colour photographs (Plates 1 to 24) are presented for a series of indigenous plants, and in addition a collection of 150 colour slides showing acute injury symptoms, as determined by this study, is housed in the library of the National Botanic Gardens at Kirstenbosch.

Three species of the monocotyledonous genus, *Watsonia*, were studied: *W. bulbillifera*, found in mountain regions of the south west Cape; *W. fourcadei*, from the Knysna area and coastal regions; *W. fergusonae*, an evergreen from the southern Cape. Plants were mostly at the 4 to 5 leaf stage, the leaves being erect and fairly rigid. Fumigations especially affected the middle aged leaves, although older leaves were also susceptible; the youngest leaves were rarely injured, except where dosages were high. Injury appeared initially as necrosis of leaf tips, tan to ivory in colour, and extended longitudinally down the blade in stripes towards the basal area. The edges of the blade were also affected, though the central portion of the blade often remained green and, on occasions, reddish bands also developed. Severe injury resulted from exposures to 4 ppm for 1 and 3 hours and 2 ppm for 3 hours, extensive ivory coloured necrotic areas being evident (Plates 1 and 2). The emergence

of new blades from the bulb was not retarded and recovery took place in all plants. These 3 species are classed as sensitive.

Indigenous gymnosperms were represented by 2 evergreen tree species, over one year in age: *Podocarpus falcatus* (Outeniqua Yellowwood), and *Widdringtonia nodiflora* (Mountain Cypress). The leaves of *P. falcatus* are leathery, glabrous and have a polished appearance; this species was not visibly affected by fumigations. *W. nodiflora* has dimorphic leaves, which are small and needle-like in the juvenile phase, and become reduced and adpressed on the mature plant. Some variation in injury response of this species was observed, though generally, the species appeared resistant even at high dosages (as 4 ppm for 3 hours). The needle tips of some individuals, however, were injured by concentration of 4 ppm for 1 hour, becoming reddish brown in colour and displaying necrosis typical of coniferous species.

An alien conifer of economic importance in the western Cape, *Pinus radiata*, was also studied and showed injury symptoms characteristic of needle leaves. Tip necrosis, initially light brown in colour and becoming reddish brown or tan, occurred at concentrations between 3 and 4 ppm, and extended from edge to edge towards the leaf base; needles were shed (usually the oldest leaves) in severe cases of injury. Variations in sensitivity were observed between individuals of this species.

Other tree species classed as resistant are *Ekebergia capensis*, an evergreen tree having dark green, glossy leaves; *Dovyalis caffra*, a hardy tree of the eastern Cape coastal regions *Schotia brachypetala*, a deciduous tree of Natal, Transvaal and Transkei; *Celtis africana*, found in many forest areas (and used as a shade tree in urban plantings), deciduous and having roughly hairy leaves. The first and second mentioned species

were not affected by exposures to SO_2, while the other two species showed acute symptoms of injury only after dosages of 4 ppm for 3 hours. *S. brachypetala* showed tip and marginal necrosis (reddish brown), the leaf midribs remaining green; leaflets severely affected were shed subsequently (Plate 3). Leaves of *C. africana* were irregularly blotched and brown in colour, margins and tips were also injured; a distinct dark border line surrounded necrotic tissue (Plate 4).

Injury to *Ochna serrulata*, an evergreen tree with dark green, leathery leaves of high gloss, (classed as resistant) was evident only at concentrations of 4 ppm. Dark brown necrotic streaks extended on either side of the midrib, and parallel to lateral veins; young leaves were not affected (Plate 5).

Rhus lancea, an evergreen with smooth leathery trifoliate leaves, is classed as sensitive, being injured by low dosages (2 ppm for 1 hour and 1 ppm for 3 hours). At higher concentrations, loss of injured leaves occurred within 4 days of exposure. In general, injury was evident as necrotic stripes between veins and along the edges of the blade (Plate 6). *R. lucida*, a very shiny leafed shrub, and classed as intermediate in susceptibility, was severely affected by 4 ppm for 3 hours. Red tan areas extended over the blade from tips and margins towards the leaf base at high dosages, while tan spots or flecks were typical of tissue injury caused by 2 ppm for 3 hours and 3 ppm for 1 hour.

Virgilia divaricata, an evergreen tree of the Leguminosae family, has soft, pinnate leaves, covered with silver grey hairs. Low dosages (1 ppm for 3 hours) affected this species, which is considered sensitive. Bleached areas (marginal and surrounding the tips) developed fairly extensively over the blades (Plate 7),

and leaflets were shed after exposure to concentrations of 3 and 4 ppm. Chlorosis was also evident (Plate 8).

Another member of the Leguminosae, *Podalyria sericea* appears resistant; the plant has hard leaves and is densely covered with silky hairs. *Indigofera filifolia*, having glabrous leaflets, is classed as intermediate; a browning of leaf tips was noted at concentrations between 3 and 4 ppm, and blades curled inwards from the margins. Chlorosis was also observed on older leaves.

An aromatic herbaceous shrub of the Umbelliferae, *Heteromorpha arborescens* has trifoliate, glabrous leaves. Injury occurred at most dosages, however, necrosis (light brown in colour) was not extensive and was usually confined to the leaf margins. Ivory to light tan flecks also appeared irregularly over the leaf surface.

Resistance is shown by *Polygala myrtifolia* and *Orphium frutescens*, both species having leaves which are tough in texture. An evergreen shrub, *P. myrtifolia* was injured to some extent by 4 ppm for 1 hour; ivory spots were scattered irregularly on leaves of middle age. A perennial, growing in sandy, brackish soils along the Cape coastal areas, and having thick leathery leaves, *O. frutescens* showed no symptoms of injury.

Leonotis leonurus, *Plectranthus ecklonii* and *Salvia aurea*, all members of the Labiatae family, are aromatic perennials or undershrubs having soft-textured, hairy leaves. Severe injury was not produced (species are classed as intermediate), though necrotic lesions appeared as dark brown blotches or streaks, especially near the leaf margins.

Lobelia pinifolia, of the south western Cape mountain areas, was not extensively injured by SO_2 dosages (intermediate

in susceptibility). Necrotic symptoms appeared as a darkening of the tips of the narrow, hard leaves, extending towards the leaf base, and some brown banding also occurred. *Chironia baccifera*, a species tolerant of maritime conditions, was susceptible to injury at concentrations of 4 ppm (intermediate classification); the leaf tips mainly were affected, becoming ivory.

Perennial herbaceous plants of the Selaginaceae (a family allied to the Scrophulariaceae), *Selago serrata* and *S. thunbergii* are classed as intermediate and sensitive respectively. The leaves of *S. serrata* are tough, leathery and glabrous, and the margins toothed; *S. thunbergii* has narrow, short needle-like leaves, in clusters. Both species were affected by dosages of 1 ppm for 3 hours and 2 ppm for 1 hour, and concentrations above 2 ppm produced brown necrotic lesions, located typically at the leaf tips and edges. Concentrations of 4 ppm also resulted in chlorosis of the oldest and middle aged leaves of *S. serrata* while leaf abscission occurred in *S. thunbergii*.

A member of the daisy family and found near the sea (as the specific name suggests), *Helichrysum maritimum* is a perennial having both leaf surfaces and stems covered with woolly hairs. At dosages which caused moderate injury to this species (4 ppm for 1 hour, 4 ppm for 3 hours), dark brown flecked areas were visible, scattered irregularly over the blades of the middle aged and older leaves. These lesions became blackened in time, but no leaf loss was observed. The species is classed as intermediate. *Dymondia margaretae* is a species cushion-like in habit and normally tolerant of periods of desiccation and inundation; an indumentum of white hairs clothes the undersurfaces of the leaves. No injury was observed, the species being resistant to SO_2 at all dosages. The undersides of the leaves of *Gazania linearis*, another composite, are also densely

woolly, and while extensive necrosis did not occur on exposure to SO_2, dark brown marginal necrosis (as produced by 4 ppm for 3 hours) caused deep pitting of the upper leaf surface, the undersurface remaining uninjured (Plate 9). Some chlorotic signs were evident, especially at high concentrations.

A resinous shrub, *Euryops pectinatus* has distinctive silver grey foliage, leaves being pinnately lobed and densely woolly. Necrosis was initially evident as a yellowing of the terminal lobes of the leaves, extending to other lobes nearer the petiole; these areas became dark grey or brown. After about 5 days, and in cases of more severe injury (4 ppm for 1 hour, 4 ppm for 3 hours and 2 ppm for 3 hours), the lobes were blackened and later dried out. The youngest leaves were never affected (Plate 10), though the oldest leaves showed some signs of chlorosis at lower dosages. The upward curling of the lobes of injured leaves was a distinctive feature in the development of symptoms.

Classed as sensitive, *Lobostemon fruticosus* has coriaceous leaves, covered with rough, bristly hairs. Bruising causes a rapid discolouration of the leaves and is typical for the genus. Injury to this species was characterised by black necrotic areas, especially at the leaf tips and margins. At dosages of 3 and 4 ppm for 1 and 3 hours, necrosis extended over the blade surface, though the prominent midrib usually remained unaffected (Plate 11). Necrotic leaves tended to curl upwards and were shed fairly rapidly, regrowth taking place from axillary buds. Young leaves were not affected.

The Aizoaceae is the largest family of plant succulents in South Africa, and displays many morphological and physiological adaptations for tolerating hot, dry condition. *Astridia hallii* (from the arid southern Namib area), a fleshy, wax coated

species, showed no symptoms of injury. Similarly, *Lampranthus amoenus*, a shrubby species is classed as resistant.

Erica comprises the largest genus in the flora of South Africa, and of the total number of species of *Erica* occurring in southern Africa, 95% are found in the south western and southern Cape. Morphologically, the genus is characterised by small, narrow leaves (often referred to as ericoid leaves) which are wax covered and the margins rolled inwards on the undersurface. These features, which limit gaseous exchange, are adaptations to a dry climate. The species studied, *E. baccans*, *E. glandulosa*, *E. glauca* var. *glauca*, *E. mammosa*, *E. patersonia*, and *E. sessilifolia*, were mainly affected by dosages of 4 ppm for 3 hours, and, with the exception of *E. glandulosa*, can be considered as resistant to SO_2. The branches and leaves of *E. glandulosa* are covered with gland tipped hairs, and after exposure for 3 hours at 2 and 4 ppm, tan to dark brown necrosis of leaf tips occurred (Plate 12). Leaf loss was noted after some days where entire leaf surfaces were injured.

The Geraniaceae is well represented in South Africa by the genus *Pelargonium*, which comprises about 250 species; plants may be herbs or shrubs. Five species of pelargoniums were studied, although some differences in susceptibility were shown, no species is considered as resistant to SO_2. *P. peltatum* (from the eastern Cape), having smooth fleshy leaves, was found to be sensitive. At concentrations between 2 and 4 ppm marginal necrosis, tan to ivory in colour, was evident; after prolonged exposures, the leaf edges curled upwards and injured areas became torn. This species also showed signs of chlorosis. Injury to *P. betulinum* (a species typical of stabilised dune systems of the south western and southern Cape) followed a pattern similar to *P. peltatum*, though those leaves having distinct red toothed edges were more readily affected by fumigations.

The leaves of *P. capitatum* are soft and covered with glandular hairs; typically, necrotic streaking was shown near the leaf margins, and blotches or flecks between the veins. These markings ranged in colour from brown to ivory, and were often bordered by a distinct reddish line separating necrotic and healthy tissue. *P. angulosum*, which has large, firm and hairy leaves, was similarly affected (Plate 13). *P. graveolens* (from the eastern Cape) has divided leaves, softly pubescent; injury appeared as tan to light brown lesions, located along the indented margins and surrounded by yellowish areas (Plate 14). The lobes tended to curl over when necrotic and after exposure to concentrations of 4 ppm; the older and middle aged leaves also became chlorotic. The three last mentioned species are classed as intermediate in susceptibility.

Three members of the Compositae family, all found to be extremely sensitive, were *Cotula turbinata*, *Leyssera gnaphaloides* and *Osteospermum oppositifolium*. These species were extensively injured by dosages above 1 ppm for 1 hour. *C. turbinata*, a small annual, cushion-like in habit and widely distributed, has soft leaves of a feathery appearance. Necrosis, tan brown in colour, extended over the pinnately divided lobes and noticeably affected old and middle aged leaves; the youngest leaves were never injured. Tissues of the whole leaf were usually killed, though leaves were rarely shed. The leaves of *L. gnaphaloides* are very narrow, stiff, covered with glandular hairs, giving a greyish green colour to the plant. Injury to the sessile, needle-like leaves was evident as tip necrosis, tan brown to blackish in colour, and covering the entire leaf in severe cases of necrosis; leaf abscission occurred at high dosages. Leaves are dimorphic in this species, and the longer leaves rather than the short tufted leaves, were typically affected (Plate 15). Both *L. gnaphaloides* and *O. oppositifolium* are aromatic shrubby plants, the latter possessing the distinctive

odour of the Calendulae tribe. The oblong-shaped leaves of *O. oppositifolium* initially showed necrosis of the margins and tips, the progression of injury, however, was rapid and covered the entire blade; leaves dried to a grey black colour within 2 to 3 days (Plate 16). Very young leaves, and those at the ends of side branches, were generally not injured even at high dosages, though the death of some plants was recorded after exposures of 4 ppm for 3 hours. Necrotic leaves were shed fairly rapidly but regrowth from axillary buds was not observed for some weeks.

The Proteaceae is typically a family of the southern hemisphere; about 400 species are found in South Africa, the majority in the south western Cape. The responses of 17 species of Proteaceae were studied, represented by the genera *Protea* (4 species), *Leucadendron* (11 species), and *Aulax* (2 species).

The leaves of *Protea repens* are thin and glabrous; response to fumigations was rapid and leaves became dried from the tips towards the base. Little marginal necrosis occurred (possibly due to the narrowness of leaves), but rather, the entire blade from edge to edge became light brown to tan in colour (Plate 17). Dosages above 2 ppm for 3 hours resulted in injury to both middle aged and older leaves. *P. acuminata* (formerly named *cedromontana*), also having thin and glabrous leaves, was similarly sensitive; leaves became blackened after exposures, especially at concentrations between 2 and 4 ppm. The leaves of *P. laurifolia* are hard and coriaceous in texture, hair covered, the margins thickened and red; injury occurred as dark brown areas, marginally and at the leaf tips (Plate 18). Necrosis did not extend over the whole surface of the leaf, except at high dosages. This species is classed as intermediate in susceptibility, as is *P. obtusifolia* (one of the few proteas which tolerates alkaline soil conditions). Severe injury to *P. obtusifolia* was not evident at dosages other than 4 ppm for 3 hours; necrosis occurred

at the tips, along the margins and as sharply defined reddish brown blotches on the leaf blades (Plate 19).

Of the species of *Protea* studied, none can be classed as resistant, and recovery from injury caused by high dosages progressed slowly, though a few individuals were killed by concentrations of 4 ppm sustained for 3 hours.

Leaves of *Leucadendron rourkei* and *L. rubrum* are pubescent, and those of *L. procerum*, glabrous but cutinised. These species are classed as intermediate in susceptibility; injury was not severe, except for exposures of 3 hours at 4 ppm. At most dosages, necrosis was restricted to scattered areas on the leaf blades, buff to light brown in colour and tending to become ivory at concentrations of 4 ppm.

Other leucadendrons studied were *L. conicum*, *L. coniferum*, *L. laureolum*, *L. meridianum*, *L. microcephalum*, *L. spissifolium* ssp. *fragrans*, *L.xanthoconus*, *L. uliginosum* ssp. *uliginosum*. All these species are classed as sensitive. Having leaves soft in texture and usually covered with silky hairs, plants were noticeably affected by concentrations of 2 ppm for 3 hours. Necrosis of tips and margins was initially light brown, becoming darker with time, and often covered the surface of narrow leaves from edge to edge (Plates 20 and 21). Generally only middle aged leaves were injured, though at high concentrations older leaves were also affected (Plate 22). Severely injured leaves usually curled upwards on drying out, but abscission rarely occurred and growth continued from apical buds.

The genus *Aulax* is allied to *Leucadendron*, differing in the arrangement of the male flowers. 2 species were studied, both having glabrous, leathery leaves; both are classed as sensitive. Necrosis appeared as tan brown lesions along margins and tips of leaves of *A. umbellata*, and generally injured areas were distinctly separate from the green tissue (Plate 23).

Symptoms of injury to *A. cancellata* (formerly named *pinifolia*) were characteristic of needle-shaped leaves; reddish brown necrosis of tips, extending longitudinally, often resulted in a bonded appearance of the needles (Plate 24). Dosages of 2 ppm for 3 hours caused severe injury to both species, and after exposures, growth of plants appeared to progress slowly; complete destruction of leaves, however, did not occur.

Foliar Injury caused by SO_2

Plates 1 - 12

Photographs in black and white shown on the following pages are presented in colour in the original copies of the thesis.

Key to photographs

Plate	Species	Dosage (ppm:hrs)	Magnification	Day*
1.	*Watsonia bulbillifera*	4:1	x 0,75	3
2.	*Watsonia bulbillifera*	2:3	x 1,25	3
3.	*Schotia brachypetala*	4:3	x 0,75	5
4.	*Celtis africana*	4:3	x 1	5
5.	*Ochna serrulata*	4:1	x 1,25	3
6.	*Rhus lancea*	2:1	x 1,5	5
7	*Virgilia divaricata*	3:1	x 0,5	5
8.	*Virgilia divaricata*	2:3	x 1	5
9.	*Gazania linearis*	4:3	x 1,5	5
10.	*Euryops pectinatus*	4:1	x 0,5	14
11.	*Lobostemon fruticosus*	3:1	x 1	5
12.	*Erica glandulosa*	4:3	x 1	5

* number of days after fumigation


1


2


3


4


5


6


7


8


9


10


11


12

Foliar Injury caused by SO_2

Plates 13 - 24

Key to photographs

Plate	Species	Dosage (ppm:hrs)	Magnification	Day*
13.	*Pelargonium angulosum*	4:1	x 0,5	14
14.	*Pelargonium graveolens*	3:1	x 0,5	5
15.	*Leyssera gnaphaloides*	2:1	x 2	2
16.	*Osteospermum oppositifolium*	2:1	x 0,75	2
17.	*Protea repens*	2:3	x 1	5
18.	*Protea laurifolia*	3:1	x 1	5
19.	*Protea obtusifolia*	2:3	x 1	5
20.	*Leucadendron conicum*	2:3	x 0,75	5
21.	*Leucadendron coniferum*	3:1	x 1	5
22.	*Leucadendron conicum*	4:1	x 0,5	10
23.	*Aulax umbellata*	4:1	x 1	5
24.	*Aulax cancellata*	2:3	x 1	5

* number of days after fumigation

13

14

15

16

17

18

19

20

21

22

23

24

5.3 DOSE-RESPONSE REPRESENTATIONS

The extent of injury can be related to exposure time and concentration by various mathematical equations (as outlined in section 2.6). Among these are the equation of O'Gara [25], which is a mathematical expression of a relationship between injury, time and concentration, applicable over relatively short periods, and an exponential relationship (developed by Guderian et al. [5]) which considers parameters varying with species and degree of injury. Two-dimensional representations developed from these equations do not adequately describe the complex interactions which affect plant response. Expressions of the degree of injury produced with variations in time and concentration can be better represented by three-dimensional surfaces which illustrate the non-linear relationship of injury and dosage [63].

Such mathematical surfaces are presented (Figures 5.1 to 5.4) for a selection of species, typifying the response of plants which are considered to be either sensitive, intermediate, or resistant to SO_2. The degree of overall foliar injury for the whole plant, plotted as the vertical plane in these dose-response surfaces, is derived from the product of the percentage of the plant injured and the injury index assessed for each dosage. This product gives a more complete picture of the extent of injury to the whole plant than the index alone, which applies only to injury sustained by the three most susceptible leaves of the plant. Each horizontal line on the vertical plane indicates an increase of 50 units; the maximum value possible on this scale is 400, representing total plant injury. The four figures presented show the development of injury as concentration and time vary, and indicate that, in general, a change in concentration has a greater effect than that of time. Estimations of injury thresholds can be made from such dose-response surfaces without

DOSE - INJURY RESPONSE

FIG. 5.1

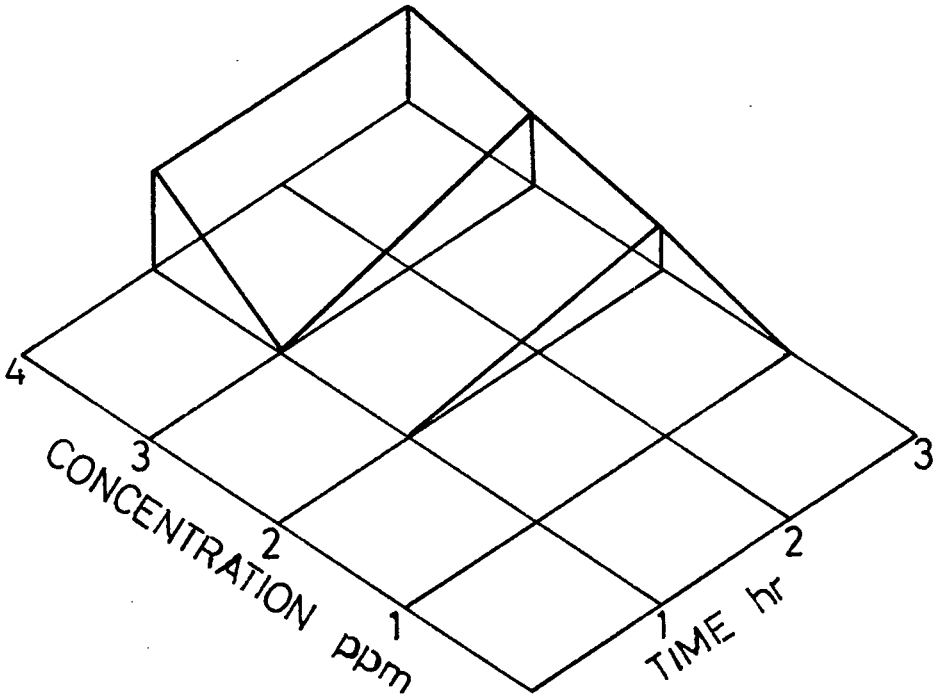
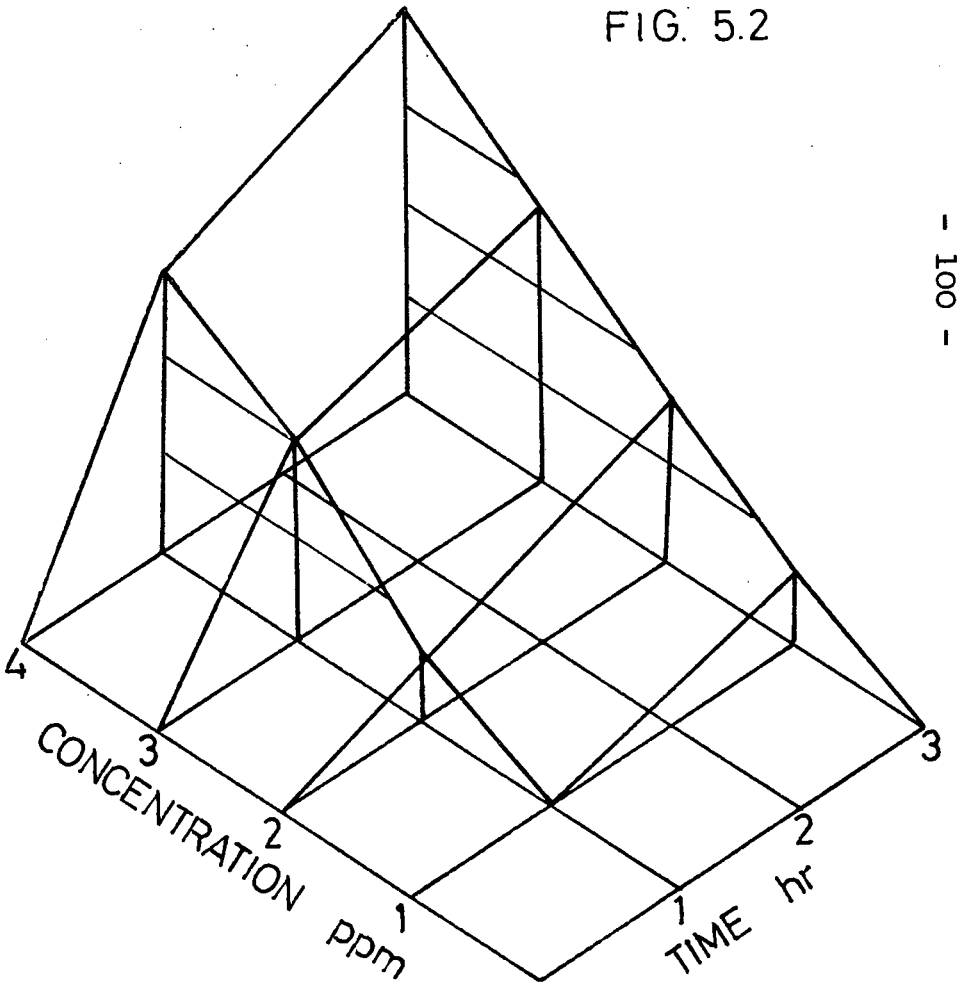


FIG. 5.2



DOSE – INJURY RESPONSE

FIG 5.3

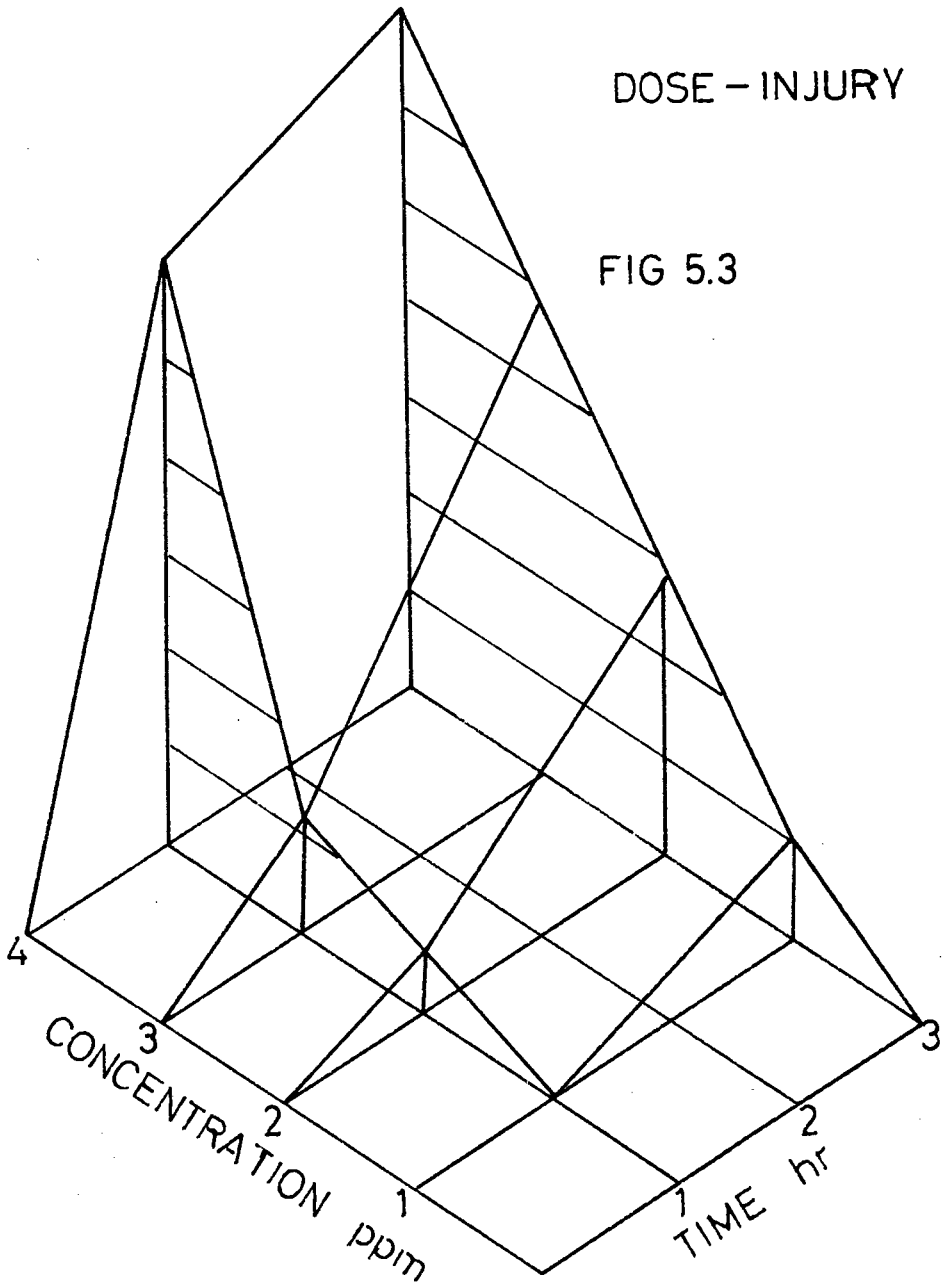
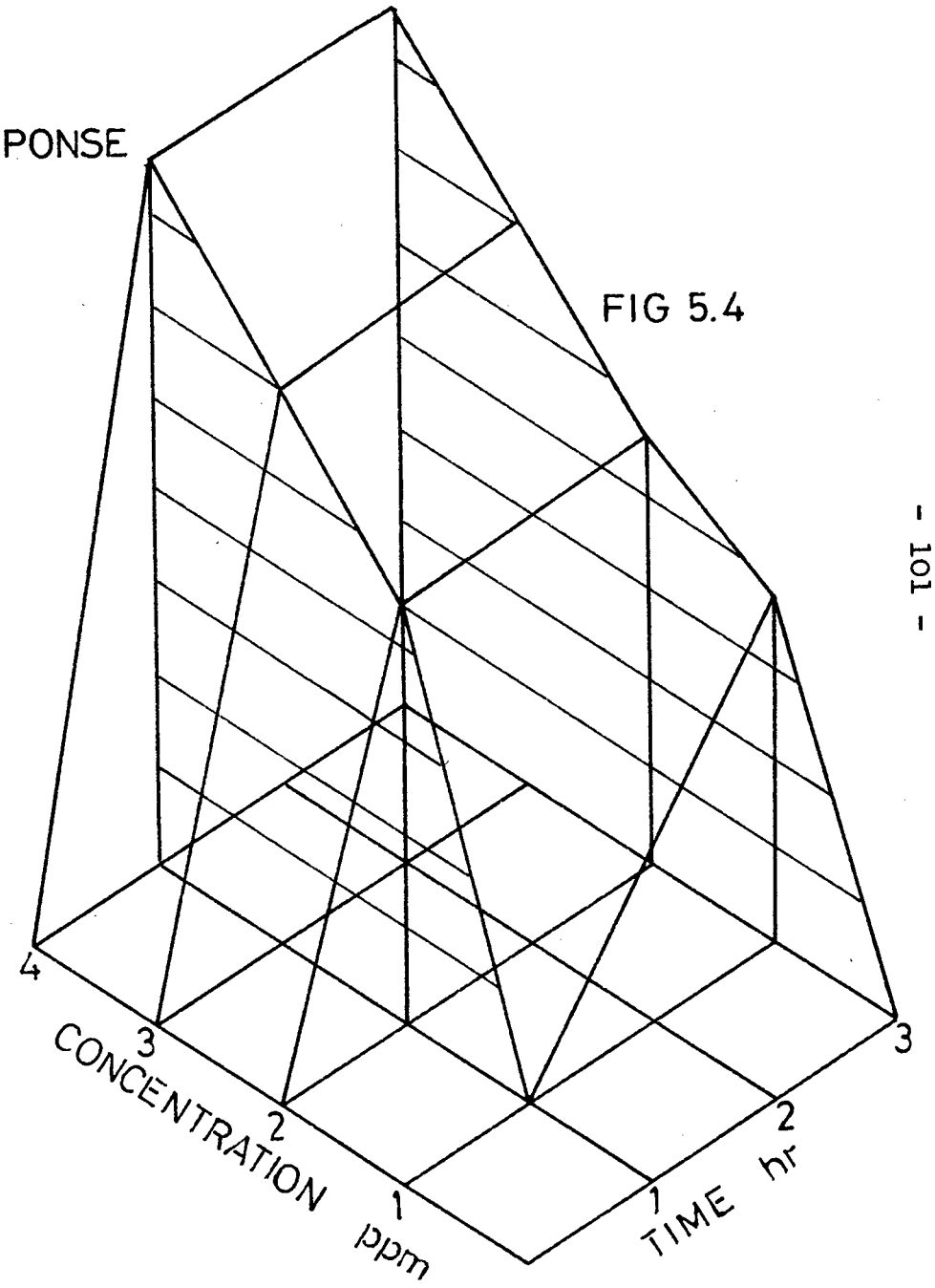


FIG 5.4



calculation from equations. Figures 5.1 to 5.3 are representative of the 3 susceptibility categories previously described: sensitive, intermediate, and resistant; Figure 5.4 indicates an extreme sensitivity shown by a few species.

Figure 5.1 illustrates the response of a resistant species, only traces of injury (interpreted as an injury index of 1) developing at some dosages. The shape of the response surface shown in Figure 5.2 is roughly symmetrical, suggesting that in this case SO_2 concentration and duration of exposure are of similar importance in the production of injury. Symptoms of injury appear after dosages of 1 ppm for 3 hours and after 2 ppm for 1 hour, while severe injury (corresponding to an index of 4) does not occur at any of the dosages employed. Figure 5.3 shows the occurrence of trace injury at 2 ppm for 1 hour and at about 0,5 ppm for 3 hours, and severe injury at 4 ppm for 1 and 3 hours. The steepness of the slope in the concentration plane indicates the rapid increase of injury with respect to concentration and the gradual increase with respect to time, and thus, in this instance, concentration is of greater importance in producing injury than the period of exposure to SO_2. The response surface of Figure 5.4 is characterised by steep rises of the slopes in both the concentration and time planes. Trace injury is shown at 1 ppm for 1 hour, and at about 0,5 ppm for 3 hours; at all dosages above 1 ppm for 1 hour, severe injury is recorded.

The dose-response surface for *Polygala myrtifolia* (Figure 5.1) is typical of resistant species. Similar responses are shown by: *Podocarpus latifolius*, *Widdringtonia nodiflora*, *Celtis africana*, *Astridia hallii*, *Lampranthus amoenus*, *Ochna serrulata*, *Podalyria sericea*, *Schotia brachypetala*, *Ekebergia capensis*, *Dovyalis caffra*, *Erica baccans*, *E. glauca* var. *glauca*, *E. mammosa*, *E. patersonia*, *E. sessilifolia*, *Orphium frutescens*, *Dymondia margaretae*.

Figure 5.2 represents the dose-response surface for *Pelargonium angulosum*, and is typical of species intermediate in susceptibility. Similar responses are shown by: *Leucadendron procerum*, *L. rourkei*, *L. rubrum*, *Protea laurifolia*, *P. obtusifolia*, *Indigofera filifolia*, *Pelargonium capitatum*, *P. graveolens*, *Rhus lucida*, *Heteromorpha arborescens*, *Erica glandulosa*, *Chironia baccifera*, *Leonotis leonurus*, *Plectranthus ecklonii*, *Salvia aurea*, *Selago serrata*, *Lobelia pinifolia*, *Gazania linearis*, *Helichrysum maritimum*.

Figure 5.3 represents the dose-response surface for *Watsonia bulbillifera*, and is typical of species classed as sensitive. Similar responses are shown by: *Aulax cancellata*, *A. umbellata*, *Leucadendron conicum*, *L. coniferum*, *L. laureolum*, *L. meridianum*, *L. microcephalum*, *L. spissifolium* ssp. *fragrans*, *L. uliginosum* ssp. *uliginosum*, *L. xanthoconus*, *Protea acuminata*, *P. repens*, *Virgilia divaricata*, *Pelargonium betulinum*, *P. peltatum*, *Rhus lancea*, *Lobostemon fruticosus*, *Selago thunbergii*, *Euryops pectinatus*, *Watsonia fergusonae*, *W. fourcadei*.

Figure 5.4 represents the dose-response surface for *Osteospermum oppositifolium*, which together with *Cotula turbinata* and *Leyssera gnaphaloides*, is considered extremely sensitive in response to SO_2 exposures.

CHAPTER 6

CONCLUSIONS

The objectives of this study have been realised in that the construction and operation of a suitable fumigation chamber has enabled observations to be made on the relative susceptibility to SO_2 of a range of South African plants. In addition, concentrations of SO_2 and duration of exposures which cause foliar injury have been determined, and the characteristic injury symptoms have been recorded and photographed. Several general conclusions can be drawn from the results of this investigation:

Plants studied showed considerable variations in response to SO_2 dosages under similar environmental conditions. Many plants were resistant to SO_2 even at high dosages, while others were extremely sensitive. Differences in susceptibility were noted between species, and between genera of plant families, although genetic variability was not investigated at the subspecies level. Three broad categories of susceptibility were designated - resistant, intermediate, and sensitive. For this classification, an injury index, based on the average percentage necrosis of the 3 most severely injured leaves, was determined and found to be a convenient method of acute injury evaluation.

SO_2 concentrations of 2 ppm were required to cause injury to many of the plants studied, although some resistant species were not affected by concentrations as high as 4 ppm. The most sensitive plants were injured by concentrations of 1 ppm and this may be compared with the injury threshold value for alfalfa, determined by O'Gara, of 1,25 ppm for 1 hour [111].

Differences in concentration and exposure time affect the degree of injury, and in general, changes in concentration were found to be more important than changes in time. These variations are made apparent in 3-dimensional graphs which reflect the interaction of dosage and injury, and show that this relationship is non-linear. Within each of the susceptibility classes distinguished, similar dose response trends are evident.

The Proteaceae and Ericaceae are important families in the flora of South Africa, both being well represented in the western Cape. Of the proteaceous plants studied, none could be considered as resistant to SO_2, while in contrast, ericas tended to be resistant. Many of the tree species, including Yellowwood and other representatives of forest areas, appeared to be tolerant of SO_2. (The fact that these plants were more mature than other species tested, however, could have influenced this observation.) Succulent species, and plants normally occupying specialised habitats were, in general, also found to be resistant. Monocotyledonous plants tested were classed as sensitive, and a range of SO_2 response was shown by shrubs and herbaceous plants, a few species being extremely sensitive.

The acute effects on South African plants of exposures to SO_2 were observed to follow descriptions typical of SO_2 injury as reported from other countries for various plant types. Injury generally appeared as bifacial lesions on the leaves of affected plants within a few days of exposure, though at high dosages some sensitive species showed signs of plasmolysis immediately following fumigation. Symptoms of injury were usually fully developed 3 to 4 days after fumigation. Visual evaluation could be reliably made at this stage as no injury occurred subsequently. Although basic cellular responses to SO_2 are the same for all susceptible

species, differences in leaf structure cause variations in the expression of acute injury symptoms. On broad leaves, necrotic tissue developed intercostally and marginally, though small, narrow leaves were often injured over the entire surface. Tip necrosis developed on needle-shaped leaves, and on parallel-veined leaves injury appeared as streaked areas. The colour of the necrotic tissue varied from tan to dark brown, or buff to ivory, depending on the species. Chlorotic signs were also observed on some species. Young leaves were rarely affected, middle aged and older leaves being most susceptible to SO_2 injury.

Leaves of certain plants tended to curl upwards and abscission occurred in cases of severe injury. Regrowth of injured plants took place mainly from apical shoots, and from axillary buds where leaf abscission had been caused. The death of a few sensitive plants was observed after exposures to high SO_2 dosages, especially when extensive leaf loss occurred.

This study concerned plants having a wide range of leaf types, and as a general trend those species having thin, soft, glabrous leaves appeared sensitive to SO_2 (being especially sensitive at high dosages), while those having tough, leathery or densely hairy leaves were more resistant to injury. Aromatic or resinous plants with numerous oil glands, sunken or sessile on the leaf surface, were usually readily affected. Although variations in plant susceptibility to SO_2 cannot be explained by differences in leaf structure alone, the above observations indicate that surface uptake of SO_2, as influenced by external leaf characteristics, may be an important factor in determining the extent of injury.

Certain sensitive plants can be used as biological indicators of pollution, and consideration could be given to the

use of the more sensitive species, as determined by this study, for the monitoring of SO_2 in field surveys. Of these, *Cotula turbinata*, an annual plant, has a short life cycle, and for this reason may be less suitable as an indicator than the perennials, *Leyssera gnaphaloides* and *Osteospermum oppositifolium*.

A survey of the literature concerning effects of phytotoxicants, together with the experimental results otained in this study, lead to the following general observations:

Acute injury symptoms provide the basis for diagnosis of injury in the field, and colour photographs of such injury patterns, as determined by experimental fumigations, are an additional aid in the recognition and identification of air pollution effects. No single procedure has been adopted, however, for the quantification of pollution effects on vegetation. Acute injury can be expressed in a number of ways, ranging from purely descriptive terms to numerical scales based on average percentage foliar necrosis, and some uniformity in the method of injury assessment is required for the comparison of responses as observed by different workers. Mathematical expressions, which can be used to predict the combinations of SO_2 concentrations and exposure times causing injury to plants, are available, though such models are limited in application.

A relationship exists between the response of plants to short or to long term exposures, and the relative susceptibilities of plants are similar whether expressed as acute injury or as chronic injury [29,184]. Knowledge of the response of plants to high concentration, short term exposures is therefore useful in providing an indication of the probable effects of prolonged, low levels of SO_2; however, predictions of changes in yield or growth cannot be made based solely on the extent of visible injury symptoms.

Phytotoxic effects have been noted at growing season averages of 0,017 to 0,025 ppm, associated with maximum ½ hour concentrations of 0,8 to 1,9 ppm SO_2, and instances of foliar injury under field conditions have been reported at concentrations of 0,41 to 0,63 ppm for 1 hour [4]. Although phytotoxic SO_2 concentrations do not widely prevail in South African urban areas, the occurrence of high peak values and the interaction of SO_2 with other air pollutants, could cause adverse effects on plants at present SO_2 levels, especially in localities of industrial processes and power generating operations. While fumigation experiments carried out in the laboratory or in the field cannot directly simulate ambient conditions experienced by growing plants, such studies do contribute valuable information on the impact of air pollutants on vegetation. Data so obtained can form the basis of recommendations for the acceptance of air pollutant levels necessary to protect plant life.

Exposure chamber studies, such as this investigation dealing with South African plants, can thus be used in the evaluation of injury caused to different species by single and/or multiple pollutants, and for the assessment of genetic factors and cultural practices in relation to the resistance of plants to air pollutants.

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APPENDIX

Equipment Specifications

Fibreglass Sheeting: 'Filon' (Pty) Ltd.
Light transmission 88%. Solar energy transmission 79%.

SO_2 Cylinder: AECI, 72 kg. >99,9% pure, liquid under pressure.

Centrifugal Blower: Air-Steel (Pty) Ltd., 45cm diam., 2850 rpm, 0,56 kW motor.

Metering Control Valve: Nupro "MG" series, 0,055 in. orifice diam., stainless steel.

Rotameter: Fisher-Porter Triflat, Tube No. 08F 1/16-08-4/35, ruby float.

Thermocouple: Copper-Constantan, wire supplied by C.S. Gordon Co., Illinois.

Vane Anemometer: Thies, Göttingen, range 0,3-20 ms^{-1}.

Photometer: Metrix. Model MX602A, with filter, 0-200,000 lx.

Solarimeter: Kipp thermopile instrument.

Multi Gas Detector: Drägerwerk-Ag-Lubeck. Mod.21/31.

SO_2 Analyser: Beckman Model 906 A, range 0,02-2 ppm.

SO_2 Sampler: Casella Model T1400, range 0,005-500 ppm.

Camera: Asahi Pentax Spotmatic F.

Lens: Macro-Takumar 1:4/100

Film: Agfacolor CT18. ASA50.

Colour Plates: Cibachrome-A Print System.

Conversion Factors for SO_2

At 25°C, 760 mm Hg:

ppm (vol) to $\mu g/m^3$: multiply by 2620

$\mu g/m^3$ to ppm (vol): multiply by $0{,}38 \times 10^{-3}$

(At N.T.P.: 1 ppm (vol) = 2860 $\mu g/m^3$)

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