External Review Draft No. 1
April 1980

Draft
Do Not Quote or Cite

Air Quality Criteria for Particulate Matter and Sulfur Oxides

Volume I
Summary and Conclusions

NOTICE

This document is a preliminary draft. It has not been formally released by EPA and should not at this stage be construed to represent Agency policy. It is being circulated for comment on its technical accuracy and policy implications.

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PREFACE

AIR QUALITY CRITERIA FOR PARTICULATE MATTER AND SULFUR OXIDES

This criteria document has been prepared in response to Sections 108 and 109 of the 1977 Clean Air Act Amendments. The 1977 Amendments modified Section 109 of the 1970 Clean Air Act by adding the following subsection:

> "Not later than December 31, 1980, and at five-year intervals thereafter, the Administrator shall complete a thorough review of the criteria published under section 108 and the national ambient air quality standards promulgated under this section and shall make such revisions in such criteria and standards and promulgate such new standards as may be appropriate in accordance with section 108 and subsection (b) of this section...."

The goal of the Clean Air Act is to protect public health and welfare and enhance the quality of the nation's air. Under the Clean Air Act, the Environmental Protection Agency is responsible for reviewing air quality criteria and establishing, on a nationwide basis, ambient air quality standards protective of health (national primary ambient air quality standards) and welfare (national secondary ambient air quality standards). To meet the national ambient air quality standards, the States and Territories are responsible for developing pollutant emission limiting regulations and strategies for controlling particular sources.

The first step in carrying out the nation's air quality management program is to identify specific pollutants which, in the words of the Clean Air Act, "may reasonably be anticipated to endanger the public health or welfare." Particulate matter and sulfur oxides have been identified as such pollutants since 1971. Once a pollutant is "listed"

under Section 108(a) of the Clean Air Act, EPA publishes an air quality criteria document which forms the scientific basis for the national ambient air quality standard. The Act requires the criteria document to contain the "latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare."

Separate criteria documents for sulfur oxides and particulate matter were published in 1969. These documents, Air Quality Criteria for Sulfur Oxides (AP-50) and Air Quality Criteria for Particulate Matter (AP-49), were the bases for the national ambient air quality standards for sulfur oxides (SO_x) and total suspended particulate matter (TSP) promulgated in 1971. In this present review of criteria, EPA has combined information on sulfur oxides and particulate matter into a single document. Sulfur oxides and particulate matter have many common sources, such as combustion of fossil fuels, and they often exert a combined action which adversely affects man and his environment.

Manmade emissions of sulfur oxides come primarily from burning oil and coal, while airborne particulate matter emanates from virtually all of man's industrial activities. Sulfur oxides exist in the atmosphere as gaseous sulfur dioxide (SO_2) and as other sulfur compounds. Much of the sulfur from the combustion of fossil fuels enters the atmosphere as SO_2. About two-thirds of these emissions are deposited in the region of their origin. Of the SO_2 which enters the atmosphere, it has been suggested that approximately one-third of it is converted to sulfates.

The primary objective of this air quality criteria document is to identify the effects of particulate matter and sulfur oxides on the public health and welfare and provide a sound scientific basis for the

consideration of National Ambient Air Quality Standards for these pollutants. In addition to updating and reviewing the scientific evidence for health and welfare effects, the document discusses sources, emissions and exposures for these pollutants.

This document is presented in four volumes. Volume I, "Summary and Conclusions" provides an overview of the document and its major findings. Volume II focuses on measurement methods; sources and emissions; concentrations and exposures, and transport, transformation, and removal mechanisms. Volume III examines the impact on the public welfare, covering effects on ecosystems and vegetation, acidic precipitation, visibility impairment, climatological effects, and effects in materials. This information is intended to identify criteria for development of National Secondary Ambient Air Quality Standards. Volume IV presents the effects on human health by examing deposition, animal and human experimental studies and epidemiological studies. This volume provides EPA's Administrator with information to propose and promulgate National Primary Ambient Air Quality Standards.

EPA gratefully acknowledges the efforts and contributions of all persons and groups who have participated in the preparation of this document. The Environmental Protection Agency assumes full responsibility for its content.

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1. INTRODUCTION, EXECUTIVE SUMMARY, AND CONCLUSIONS

1.1 INTRODUCTION

Section 109(d) of the Clean Air Act as amended in 1977 requires the Environmental Protection Agency (EPA) to review the air quality criteria for sulfur oxides (SO_x) and particulate matter (PM) and to revise related standards, as appropriate, by December 31, 1980, and at 5-year intervals thereafter. In addition, the Administrator may from time to time review and, where appropriate, modify pertinent criteria under the authority of Section 108(c) of the Clean Air Act. Pursuant to the above provisions of the Clean Air Act, the present document represents an important step in the preparation of the revised criteria documents for SO_x and PM.

The decision to issue a single document for these two pollutants was based on several considerations. These include primarily the recognition that: (1) both SO_x and PM originate from many common emission sources such as the burning of fossil fuels; (2) SO_x and PM levels frequently covary in ambient air; (3) SO_x and PM appear to likely act together to adversely affect both man and his environment; (4), in community health studies and other types of studies where combined exposure to SO_x and PM is a factor, it is difficult to isolate the effects of one pollutant from those of the other; and (5) the combination of sulfur oxides and particulate matter has been linked with acidic precipitation, which damages aquatic and terrestrial ecosystems.

This document critically reviews scientific information bearing on health and welfare criteria that will form the bases for National Ambient Air Quality Standards (NAAQS) for SO_x and PM.

The information reviewed concerns: (1) pertinent air quality information; (2) welfare effects associated with SO_x and PM; and (3) health effects associated with exposure to those air pollutants. The present volume (Volume I) includes the general introduction, executive summary and conclusions for the entire document. The second volume (Volume II) on air quality aspects, includes chapters discussing: physical properties of SO_x and PM and air quality measurement techniques for each; critical appraisal of practical applications of such measurement methods; sources of SO_x and PM emissions; related atmsopheric transport and transformation processes; and ambient air concentrations and exposure levels. Volume III, concerning welfare effects, contains document chapters on: SO_x and PM effects on vegetation and natural ecosystems; acidic precipitation formation and effects; effects on visibility and climate; and materials damage effects. The last volume (Volume IV), dealing with health effects of SO_x and PM contains chapters on: the uptake, deposition, and absorption of SO_x and PM health effects; human clinical (experimental) studies of SO_x and PM health effects; and pertinent community health (epidemiology) studies.

In the present volume, there is first provided a brief historical review of important events which have (1) contributed to concern about the health and welfare effects of SO_x and PM; (2) stimulated extensive and highly varied related research efforts; and (3) helped to shape social/ legal actions taken to eliminate or ameliorate such effects as major public health and welfare problems. It is hoped that such a historical review will provide background material useful in assisting readers, especially those not well familiar with the present subject matter, to develop a better informed vantage point or perspective from which to view the information subsequently presented in this document. The major points addressed by such information and important

conclusions regarding them, as discussed in each of the remaining three volumes of the document, are then summarized following the historical perspective discussion presented below.

Before proceeding with the historical review, it should be mentioned that a number of other important reviews and commentaries concerning the health and welfare effects of SO_x and PM have appeared during the past decade. In that regard, the reader is referred to certain review articles and documents appearing in the mid-to late 1970s for additional information on the present subject matter. Such materials include critical reviews and commentaries written by Rall (1974), Higgins et al. (1974), Goldsmith and Friberg (1977), Ferris (1978), and Waller (1978). They also include the following evaluative documents appearing in 1978: an American Thoraic Society (ATS) review of Health Effects of Air Pollution (1978); a National Research Council/National Academy of Science (NRS/NAS) document on Airborne Particles (1978); and an NRC/NAS document on Sulfur Oxides (1978). More recent such reviews and commentary appearing in 1979 include: the 1979 World Health Organization (WHO) document, Environmental Health (8): Sulfur Oxides and Suspended Particulate Matter; a report by Holland et al. (1979) written for the American Iron and Steel Institute and appearing in the American Journal of Epidemiology; and a reply to that report in the same journal by Shy (1979).

1.2 HISTORICAL PERSPECTIVE

There is little question that severe air pollution generated by anthropogenic activities has long exerted significant, even lethal, adverse effects on the health and welfare of many industrial societies. It has only been

within the past 50 years or so, however, that sufficient recognition of such effects as major public problems has stimulated both (1) extensive research to better understand such problems and (2) strong governmental and private sector actions to control or eliminate them.

It is not surprising that the air pollution incidents most often cited as signal events precipitating great concern about air pollution problems and strong actions in response to them occurred among the most heavily industrialized societies then extant, i.e., in Western Europe, Great Britain and the United States. It is also easy to understand the urgency associated with taking strong actions to control such problems in light of the magnitude and seriousness of the effects experienced during those incidents.

For example, in describing the effects of a thick fog that covered the industrial Meuse Valley in Belgium during early December 1930, Firket (1931) noted that several hundred people were afflicted by suddenly appearing acute respiratory symptoms, complicated in many instances by serious cardiovascular failure. Firket (1931) further noted: "More than sixty died on the 4th and 5th of December after only a few hours of sickness. A sizeable number of livestock had to be slaughtered." Also, taking into account that mortality rates were more than 10 times normal, Firket projected that over 3,000 deaths would occur if a similar fog were to occur in a city the size of London.

Twenty-two years later, such an event did occur in London and more than 4,000 deaths appeared to be attributable to the four-day London Fog of December 1952, according to Logan (1953). Logan further noted: "The incident was a catastrophe of the first magnitude in which, for a few days, death rates attained a level that has been exceeded only rarely during the past hundred

years - for example, at the height of the cholera epidemic of 1854 and of the influenza epidemic of 1918-19." Indeed, the death rate rivaled or exceeded that on many of the worst days of other catastrophic events afflicting London in its more recent past, e.g., the Battle of Britain during World War II.

Similar catastrophic air pollution incidents also occurred around the same time in the United States. Almost half of the residents of Donora, Pennsylvania, for example, were afflicted with respiratory symptoms as the result of a "smog" covering the coke- and steel-producing Monongahela River Valley during October, 1948 (Schrenk et al., 1949). Twenty people in the small town of about 10,000 population died during the final week of the "Donora Smog Episode," in comparison to the 2 or 3 deaths normally expected for the same period. Over the next few years, catastrophic air pollution incidents also affected other United States communities. One such case was a dramatic increase in infant mortality in Detroit, Mich., attributed to a "pollution incident" in September 1952 (Int. Joint Commission, 1960). This was followed by marked increases in respiratory distress cases and fatalities attributable (Greenburg et al., 1962) to air pollution occuring on an even larger scale during a "Thanksgiving Day" pollution episode in New York City in November, 1953--an experience later to be repeated several more times in New York City during the early 1960s.

It was clear from the above incidents and others occurring elsewhere in the industrialized world that air pollution, especially under certain weather conditions leading to stagnant masses of pollutant-laden "fog" or "smog", was capable of causing incidents rivaling natural disease epidemics or man-made

wartime disasters that constituted national emergencies for the affected societies. Equally clear was the urgent need to take immediate action to avert or reduce in severity future air pollution disasters. Among the first to act were two of the most severely affected countries, Great Britain and the United States.

Many parallel historical threads can be discerned regarding the paths taken by the two nations in trying to cope with air pollution problems, including a number of similar mistakes as well as successes. In each country, for example, there occurred extensive expansion of epidemiological and toxicological research aimed at identifying components of the killer smogs or fogs responsible for the observed lethal effects and increased morbidity. Also, in each country, although occurring within different specific time frames and at different specific paces or rates, there ensued the upgrading and expansion of air quality monitoring networks. This included the expansion of monitoring networks capable of indexing high levels of various industrial pollutants implicated by the historical data as being associated with increased incidences of both morbidity and mortality. The latter invariably included oxides of sulfur and particulate matter present in the killer fogs or smogs of the 1940s and 1950s, although the relative contributions of each could not be precisely linked to observed health effects.

It is important to note in regard to the air quality data that very precise accuracy and specificity were not then demanded; rather, "benchmark" ranges of estimates of air pollutant concentrations were acceptable, especially for indexing the rather high levels of pollution associated with severe

morbidity or mortality effects. It mattered little if the exact amounts of SO_2, for example, were 143 or 781 or 1408 $\mu g/m^3$ plus or minus 5 or 10 percent, when one was concerned that people would begin to experience severe morbidity or even die when SO_2 or particulate matter exceeded several hundred $\mu g/m^3$. Being able to establish that increased mortality or severe morbidity occurred at, say, a range of 550 to 600 or even 500 to 1000 $\mu g/m^3$ of SO_2 or particulate matter in comparison to the incidence of such effects observed at, say, 70 to 100 or even 50 to 200 $\mu g/m^3$ of the same pollutants was, understandably, sufficient scientific evidence to support political and social measures needed to avert the worst air pollution episodes.

Nor was it particularly important whether or not SO_2, specifically, or particulate matter of whatever specific size-range or chemical composition could be precisely implicated as the "culprit" causing one or another very specific health effect. Rather, it was sufficient to recognize that those substances might not be any more than representative indicators of the total mix or some other potentially lethal subfraction of pollutants typically present during the dangerous past pollution episodes. Basically, the main objective, regardless of specific fine details associated with various pollution situations, was to obtain sufficient information: (1) to allow for reasonable conclusions to be drawn regarding ranges of air levels of various pollutants (or indices) empirically linked to the occurrence of severe health effects and (2) to help serve as a guide in directing pollution control efforts toward sources emitting such pollutants (or mixes containing them).

In addition to the above developments, certain regulatory actions were taken in response to the severe air pollution situations in Britain and the

United States in the 1950s. The British acted strongly on a national scale by passing the Clean Air Act of 1956 which forced implementation of very stringent controls on emissions from coal-fired combustion sources, essentially eliminating the use of coal for home-heating and many industrial purposes in heavily industrialized and congested urban areas. The effectiveness of those measures was reflected in the resulting declines seen by the early 1960s in both atmospheric sulfur oxides and particulate matter and associated mortality effects (WSL, 1967).

As for the United States, action initially tended to be taken mostly on more restricted geographic bases and consisted mainly of local or state air pollution control ordinances being passed, often with the cooperation of local industrial leaders, to reduce air pollution. Among the more notable examples were early actions taken to control air pollution in one of the most heavily industrialized regions of the country, the Pittsburgh area. There, and in other areas of Pennsylvania, extensive steel-making and coking operations and the burning of locally produced high-sulfur coal contributed to widespread elevations in both particuate matter and sulfur dioxide air concentrations. Notable improvements in air quality in various American regions were attained as the result of such initial actions. However, simultaneous deterioration in air quality in many other communities or states lacking effective pollution control laws, and the growing recognition of air pollution as a multi-state, regional or national problem eventually led to the passage in the United States of the Clean Air Act of 1970 and consequent promulgation of National Ambient Air Quality Standards for sulfur oxides, particulate matter, and other air pollutants of widespread concern.

Subsequent to many of the above actions taken in the 1950s and early 1960s, questions began to be raised regarding what less severe, but important,

effects on human health and welfare might be caused by lower air levels of oxides of sulfur, particulate matter, and other air pollutants, especially under conditions of prolonged periods (months or years) of exposure. This included increased recognition of and research on such problems as SO_x and PM effects on vegetation, visibility, and climate, as well as related materials damage effects and acidic precipitation formation and effects. Again, fairly similar paths were followed in both Great Britain and the United States in trying to deal with such questions.

In both countries, epidemiologists began to design studies to either retrospectively or prospectively define in more precise terms both qualitative and quantitative relationships between elevations of various pollutants in the ambient air and specific sublethal health effects, such as acute or chronic bronchitis, respiratory infections, temporary decrements in pulmonary function, and asthma attacks. Air quality monitoring networks set up or expanded earlier to provide at least representative, but not necessarily thorough, coverage of geographic areas having varying pollution levels were often looked to by the epidemiologists to provide requisite, albeit less than perfect, quantitative estimates of air quality to help define quantitative air pollution-effect relationships. Only in relatively rare circumstances were sufficient funds or other resources available to allow for more thorough monitoring coverage to be arranged specifically for collection of community air quality data to be coupled with health endpoint measurements. Similarly, expanded demands were placed on air monitoring capabilities in terms of their being needed to provide requisite air quality data for use in evaluating various welfare effects and related atmospheric transport and transformation phenomena.

At the same time as the above expansion of research efforts, more precise estimates of air pollutant levels were being demanded or expected from existing or expanding air monitoring networks to help index progress in reductions of air pollutants in Britain and the United States. Bearing on this point, the following was later noted in Her Majesty's Report on the Investigation of Atmospheric Pollution 1958-1966 (Warren Spring Laboratory, 1967):

> The industrial provisions of the Clean Air Act 1956 had come into force by June 1958, and the first smoke control areas were declared under the domestic provisions in April 1958. This timetable added urgency to the view that the existing survey of air pollution throughout the United Kingdom was too blunt an instrument either to assess the benefits accruing from the Act or to guide its future application, and that a scientifically planned National Survey was necessary. Such a survey was designed and the co-operation of the local authorities concerned was obtained where measurements were required in addition to those already being made. Observations were started in the winter of 1962-63. Not only has the whole pattern of co-operative observations been transformed in this way, but so also has the basis of the co-operation: the local authorities and other organizations are now making measurements as required to conform to an overall statistical plan.

A growing need was also felt for establishing or maintaining monitoring systems using sufficiently uniform and reliable measurement approaches to allow for comparability of air quality data from disparate geographic sites. This need became perhaps most acutely felt in the United States in the late 1960s in light of the growing prospect of having to attain national ambient air quality standards at lower air concentrations than were earlier envisaged. Pressure to meet such needs and demonstrate the benefits of air pollution control in the United States intensified greatly with the passage of the Clean Air Act of 1970.

Both the above needs and the anticipated use of air quality data in future epidemiology and welfare effects studies increased the necessity for

better air quality data in terms of their specificity, sensitivity, accuracy, precision, and reliability. Thus, it became increasingly more important to be able to distinguish with confidence between much closer absolute levels of air pollutants even at relatively low ambient air concentrations (e.g., at levels around or lower than 100 to 200 $\mu g/m^3$ of either SO_2 or particulate matter). In other words, the emerging demands required practical applications of available or newly developed measurement techniques to meet previously unheard-of levels of accuracy. At times expectations may have exceeded what could realistically be achieved with field applications of available technology, especially in comparison to theoretical limits of what could be achieved with particular measurement techniques under ideal laboratory conditions.

In responding to the above demands, several similar historical parallels can again be discerned between the British and American experiences, as well as some quite significant differences. In Britain, steps were taken to assure that a high degree of uniformity in measurements of pollutants was maintained or further enhanced; this included the establishment of the National Air Pollution Survey alluded to above. As part of this effort, the British Smoke (BS) filter method, widely used in Britain since the early 1900s for the measurement of black suspended particulate matter, was officially adopted to monitor air quality across the United Kingdom. The daily smoke filter has since been the standard air particulate matter measurement instrument used in the United Kingdom, and responsibility for quality assurance for the use of the instrument throughout the entire United Kingdom was assigned to the Warren Spring Laboratory (WSL) of the Department of Industry, where central coordination and evaluation of uniformity, accuracy, and reliability of all National Survey air quality measurement have been carried out since the late

1950s. Based largely on WSL evaluations, the daily smoke filter was later adopted as a standard also by the British Standards Institute and became one of OECD's recommended procedures for measuring suspended particulate matter.

The WSL was also instrumental in selecting SO_2 measurement methods for the British National Survey air monitoring program. Under WSL guidance, the hydrogen peroxide method was adopted in the early 1960s as the standard approach for measuring SO_2. This replaced the lead dioxide gauge still in use at many sites in Britain (in 1962 there were about 1200 gauges in operation). The decision to adopt the hydrogen peroxide method was based on recommendations of a WSL-organized "Work Party" that considered both the deliberations of an OECD Working Party and detailed comparisons of the two methods under field conditions by WSL.

Turning to concomitant developments in the United States during the past twenty years, one can discern a lage in the development of a standardized nationwide approach to air pollution monitoring and control. During the 1950s and early 1960s numerous air monitoring systems established by different governmental units sprang up around the country, often to meet needs associated with enforcement of newly enacted air pollution control ordinances. It was not until the mid-to-late 1960s that effective procedures were implemented to feed data obtained from the multiplicity of air monitoring sites operated by city, county, state, and federal agencies into a central data bank as part of a National Air Survey Network (NASN). Even then, those data were still often derived from different measurement methods used by various agencies for monitoring of a given pollutant. Generally, the high-volume TSP measurement method developed in the United States in the 1950s was used for assessing airborne particulate levels; but, at times, other methods such as coefficient of haze (CoH) measurements were also employed to assess particulate levels.

Similarly, several approaches, e.g., the West-Gaeke or sulfation methods, were used to measure SO_2.

Considerably greater uniformity in monitoring approaches has, however, been achieved over the past decade or so through the publication of "Federal Reference Methods." Additional efforts were undertaken in the late 1960s and early 1970s by EPA and its predecessor Federal agencies to establish a new nationwide air monitoring network using uniform measurement methods. That network was established as part of what became known as EPA's Community Health and Environment Surveillance System (CHESS) Program. The "CHESS" monitoring network, set up in addition to other Federal air sampling stations used for monitoring compliance with air regulations, included monitoring sites dispersed in widespread urban and semi-rural areas of the United States to provide air quality data representative of pollutant exposures experienced by surrounding population groups. Various health endpoints were evaluated for those population groups as part of CHESS Program epidemiology studies. Thus, the CHESS monitoring network, including sampling sites often situated near or along side local or state monitoring sites, was designed to provide air quality data from a nationwide network using uniform measuring methods that supplemented other data entered into the NASN data bank. The hi-volume TSP sampling procedure was usually employed in the CHESS Program to monitor atmospheric particulate matter levels and the West-Gaeke method was generally employed for SO_2 measurements, along with additional procedures for estimating suspended sulfates discussed later. The series of major air pollution/health effects studies carried out between 1969 and 1975 as part of the CHESS Program, and coupling such aerometric measurements with community health surveys, have been considered by many experts as the most comprehensive of their kind.

It should be noted, however, that a number of methodological problems were engendered by attempts to rapidly deploy air monitoring stations at widespread sites across the United States and to bring them up to full operational status in time to collect air quality data to be coupled with health surveys as part of the CHESS Program. Of particular concern are problems which were detected regarding the collection of health endpoint data and certain errors in air quality data generated from CHESS network sampling sites--several types of errors which were either not detected at all during the CHESS health endpoint data collection period (1969-75) or were only detected and corrected through improved quality control procedures implemented and applied in the last few years of the Program (i.e. 1972 or 1973 onward).* Improvements in the air quality data obtained during the last few years of the CHESS Program and, also, in other EPA air monitoring efforts, were accomplished via a substantially expanded in-house EPA quality assurance program. That program, conducted by EPA's Environmental Monitoring Systems Laboratory in Research Triangle Park, N.C., has since provided quality assurance backup for all of EPA's research and nationwide enforcement air monitoring activities.

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*As discussed in more detail later in Chapters 3 and 14, the matter of errors in air quality data collected as part of the CHESS Program (together with other concerns regarding the collection of health endpoint data in CHESS studies) contributed to considerable controversy regarding the validity and accuracy of results of early CHESS studies, as interpreted and reported in a 1974 EPA monograph entitled "Health Consequences of Sulfur Oxides: A Report from CHESS" 1970-71, U.S.EPA Document No. EPA-650/1-74-004 (May 1974). The controversy eventually led to the 1974 "CHESS Monograph" becoming the subject of U.S. Congressional oversight hearings in 1976. Subcommittees of the U.S. House of Representatives Committee on Science and Technology produced a report on the Monograph, other aspects of the CHESS Program, and EPA's air pollution research programs generally--a report entitled "The Environmental Protection Agency's Research Program with Primary Emphasis on the Community Health and Surveillance System (CHESS): An Investigative Report." Of primary importance for the present discussion, that report, widely referred to either as the

From the foregoing, it can be seen that similar needs and demands as those felt by the British in conjunction with the passage of their Clean Air Act of 1956 were later experienced by the Americans with the passage of their Clean Air Act of 1970. Remarkably similar paths were also followed in both countries in responding to those needs; that is, in both cases intensive efforts were carried out to rapidly expand and improve air monitoring capabilities, including introduction of more uniformity in measurement approaches across geographic regions along with increased quality control efforts to help assure the validity of the aerometry data collected. Also, in each case it was contemplated that such improved and uniformly obtained aerometry data from throughout the two nations could serve as useful data pools to be coupled with welfare effects studies and community health epidemiology studies aimed at both (1) improving knowledge of quantitative air pollution/effects relationships and (2) demonstrating the benefits to

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*(continued)

"Brown Committee Report" or the "Investigative Report" (IR), contained various comments regarding sources of error in CHESS Program air quality and health data and
quality control problems associated with the data collection and analysis. The I.R. also contained various recommendations to be implemented by the Administrator of EPA pursuant to Section 10 of the Environmental Research, Development, and Demonstration Authorization Act of 1978 ("ERDDAA," P.L. 95-155, 91 Stat. 1257, November 8, 1977). ERDDAA also requires that EPA and the Agency's Science Advisory Board report to Congress on the implementation of the IR recommendations.

One recommendation of the IR was that an addendum to the sulfur oxides monograph be published, to be used in part to qualify the usefulness of the CHESS studies, and to apprise the public of the controversy surrounding CHESS. An addendum has been published, and is available from EPA, as announced in the Federal Register of April 2, 1980, 45 F.R. 21702. The addendum is incorporated by reference in this document in partial qualification of the CHESS studies cited herein, and is part of the public file (or docket) established for revision of this criteria document. The addendum contains the full text of the IR, reports to Congress by EPA on its implementation of the IR recommendations, and a report to Congress by EPA's Science Advisory Board on the same subject. See also Appendix A of of Chapter 14 of the present draft criteria document.

be accrued from implementation of the respective Clean Air Acts. In addition, as will become apparent below, remarkably similar problems were encountered and responded to with roughly comparable degrees of success in the course of practical applications of air pollution measurement techniques undertaken to achieve the above objectives.

In addition to the above developments in the United Kingdom and the United States, many analogous steps have been taken by numerous other industrialized countries over the past 30-40 years to cope with air pollution problems. In that regard, again many parallels (and dissimilarities) in the historical evolution of their air monitoring programs, epidemiologic research efforts, and political/legal regulatory control activities could be noted in comparison to developments in Britain and the United States. However, a historical review of the evolution of such activities or analysis below of results obtained with practical applications of sulfur oxides or particulate matter measurement approaches or with community health studies outside Britain and the United States is beyond the scope of present purposes.

1.3 SO_x AND PM AIR QUALITY ASPECTS

Turning now to the major points addressed in the remainder of this document, much of the key information contained in Volume II, on air quality aspects, is summarized below under Subsections 1.3.1 to 1.3.7. Particular emphasis is placed here on the critical assessment of practical applications of air quality measurement techniques of crucial later importance in evaluating community health studies. Of additional special interest below are the discussions on transport and transformation phenomena and ambient air concentration and human exposure data.

1.3.1 Chemistry and Analytical Methods

The chemical and physical properties of SO_x and PM determine their behavior in the atmosphere and biologic toxicologic activity. Increasing sophistication of analytical instrumentation and methodology has enhanced the understanding of the relationship between atmospheric processes and sulfur oxides and particulate matter.

Sulfur dioxide dissolves readily in water, such as atmospheric moisture, to form sulfurous acid which oxidizes to sulfuric acid and subsequently to a variety of particulate sulfates. Metallic oxides such as manganese or iron catalyze the reaction. Particles may vary in size, shape, molecular composition, and optical properties. Thus, analytical methods for particulate matter vary according to the specific parameters measured.

Methods used to measure sulfur dioxide can be classified as integrated or continuous. Most of the methods are based on techniques involving absorption and stabilization on a substrate. The analysis of the collected sample is commonly based on colorimetric, titrimetric, turbidimetric, gravimetric, and ion chromatographic measurement principles. The most widely used integrated method to determine atmospheric sulfur dioxide is an improved version of the colorimetric method developed by West and Gaeke and adopted as the EPA reference method in 1971. Sulfation methods, based on the reaction of SO_x with lead peroxide paste to form lead sulfate, have commonly been used to estimate ambient SO_x concentration over extended time periods. Continuous methods for the measurement of ambient levels of sulfur dioxide have gained widespread use in the air monitoring community. Continuous sulfur dioxide analyzers using the techniques of flame photometric detection, fluorescence, and second

derivative spectrometry have been developed over the past 10 years and are commercially available.

Methods for determining soluble sulfates, total sulfates, and specific sulfate species involve the collection of particulate matter and its subsequent analysis by direct or indirect methods. For trace soluble sulfate determinations, a commercial method based on ion exchange chromatography is specific, exceptionally accurate, and sensitive. Methods of analysis for total sulfur include x-ray fluorescence, electron spectroscopy, and flame photometry. X-ray fluorescence methods are nondestructive and applicable to large numbers of ambient aerosol samples. Procedures for determining specific sulfate species include thermal volatilization and solvent extraction techniques, gas phase ammonia titration, infrared and visible spectrometry, flame photometry, and electron microscopy. Sulfate species may be estimated quantitatively by gas phase ammonia titration methods and infrared spectroscopy.

The majority of atmospheric particle samples are collected by either filtration or impaction devices. The collected samples are usually analyzed gravimetrically to provide a direct measure of mass concentration. The most sidely used method for gravimetric analysis of mass concentrations is the high volume sampler, the current EPA reference method for particles. Other methods include the British Smoke Shade and AISI tape samplers, multi-stage cascade impactors, cyclone samplers, and the dichotomous sampler. Chemical analyses consist of manual wet-chemistry, atomic absorption, x-ray fluorescence, optical emission spectrometry, spark source mass spectrometry, neutron activation analysis, and thin-layer chromatography with fluorescence detection. Methods for continuous or <u>in situ</u> monitoring or atmospheric particles are also available. They are not as closely related to mass concentration as are the integrated

methods, but they provide useful information for studying particulate sources, transport, episodes, and effects such as visibility. The integrating nephelometer measures light scattering by atmospheric particles and is commonly used as an indicator of visibility.

It should be noted that the most widely used particulate matter measurement device in the United States, the high-volume sampler, usually has a relatively high cut point, so that not only fine particles ($\leq$2.5 um MMD) but also coarse mode (2.5 to 15.0 um) and large particles (mainly up to 30 μm MMD) are sampled. In contrast, the other most frequently used method, the British smoke (BS) instrument tends to sample a much more restricted range of particles sizes, these typically being less than 5.0 ug/m (mainly <3.0 um MMD) and chiefly in the fine mode particle range.

1.3.2 Air Quality Measurement Applications

The critical assessment contained in Chapter 3 regarding practical applications of measurement approaches employed in Great Britain and the United States for determinations of air concentrations of suflur oxides (SO_x) and particulate matter (PM) is concisely summarized here. Information presented concerns published evaluations of the relative specificity, sensitivity, accuracy, precision, and reliability of the methods when used under optimum conditions in the hands of technically-expert analysts. Much more emphasis, however, is placed on evaluation of results obtained with practical applications of the measurement methods, often by less technically-skilled personnel. That evaluation draws mainly upon published commentary on quality control assessments for the different applications. Also, the major focus here is on British and American air measurement approaches most widely used in acquiring SO_2 and particulate matter data utilized in quantitative community health studies discussed later.

1.3.2.1 British Approaches

British SO_2 Measurements--As noted earlier, the lead dioxide gauge was used extensively in Britain during the years prior to 1960. However, use of the hydrogen peroxide method was gradually interspersed with the lead dioxide gauge during the course of the 1950s, often being coupled in tandem, as it were, with the apparatus for smoke measurements. Much of the early (1950s) British epidemiology data discussed later has been related to SO_2 measurements obtained either by the hydrogen peroxide method, especially where 24-hr SO_2 values are used, or the lead dioxide sulfation rate method in some cases where long-term (days to a month) data were acquired.

In 1962, as part of the establishment of the British National Air Pollution Survey, a working party was set up to compare the lead dioxide gauge with the hydrogen peroxide method, which was then chosen as the standard method for use in the Survey. As quoted in Atmospheric Pollution, 1958-1966 (WSL, 1967):

> The hydrogen peroxide method is subject to the limitation that its reaction is not confined to sulphur compounds; the lead dioxide method has the limitation that the extent of the reaction can be substantially influenced by weather conditions. Despite limitations, both methods estimate pollution by sulphur compounds; the hydrogen peroxide method is somewhat more complicated, but has the outstanding advantage that it can measure concentrations of pollution over short periods; the lead dioxide method is simple in operation, but it is incapable of measuring concentrations over short periods.
>
> Even so, it was considered desirable to compare the results from the two types of instrument under controlled conditions. A statistical analysis was made by Warren Spring Laboratory of results from a group of 20 sites at which both lead dioxide and hydrogen peroxide instruments had been operated over a period of 48 months. The 20 sites selected were those with a reasonably complete set of results from March 1957 to February 1961 at which the two instruments were not more than 100 feet apart.
>
> The correlation between 829 pairs of results from the 20 sites over a period of four years was highly significant, showing that both instruments were predominantly affected by the same pollutant, sulphur dioxide.

The WSL (1967) report presented a plot of the comparison data showing that the ± 2σ confidence limits correspond to ± 1.8 mg SO_3/100 cm^2/day for a given hydrogen peroxide reading and ± 0.18 mg SO_2/m^3 for a given lead dioxide reading.

In other words, estimates of SO_2 levels derived from lead dioxide sulfation rate measurements, especially 24-hr estimates, can only be roughly compared with SO_2 estimates obtained by the hydrogen peroxide method at other geographic sites or at later times at the same location(s). Also, comparisons between sulfation rate readings may only be meaningful when such readings differ by the equivalent of about 180 μg/m^3 of SO_2. Some of the types and magnitudes of errors encountered in the British application of lead dioxide gauges to measure SO_2 levels are summarized in Table 1-1. As shown in Table 1-1, several problems (e.g., humidity and temperature effects) result in the lead dioxide method being essentially useless for 24-hr. measurements and in their otherwise having a rather large (±180 μg/m^3 2σ) error band associated with them.

Based on some of the above problems, when the National Survey began in 1961 it was recognized that the lead dioxide method could not provide the 24-hour SO_2 measurements necessary for correlation with mortality and morbidity effects investigated by epidemiology studies. The hydrogen peroxide method for SO_2 was, therefore, adopted as being more valid than the old lead dioxide gauge sulfation method. Because many of the staff making the measurements would be the same people who had been servicing particle deposit gauges and the lead candles without detailed technical knowledge of the analyses, however, an Instruction Manual (IM) issued by WSL in 1966 had to be quite detailed and clearly readable by people with no training in analytical techniques.

TABLE 1-1. SUMMARY OF EVALUATION OF SOURCES, MAGNITUDES, AND DIRECTIONAL BIASES OF ERRORS ASSOCIATED WITH BRITISH SO_2 MEASUREMENTS

Time period	Measurement method	Reported source of error	Direction and magnitude of reported error	Likely general impact on British SO_2 data
Pre-1961	Lead Dioxide	Humidity (RH)	Reaction rate increases with RH.	Variable positive bias, especially in summer.
		Temperature (T)	Reaction rate increases 2% per 5° rise.	Variable positive bias, especially in summer.
		Wind speed (WS)	Reaction rate increases with WS.	Variable positive bias, under high wind cond.
		(Overall errors)		Can be up to ± 180 $\mu g/m^3$ (2σ).
1961-1980 (British National Air Pol. Survey)	Hydrogen Peroxide	Siting of Sample Line Intake:		
		a. too near boiler chimneys	50 - 100 $\mu g/m^3$ overestimation.	Occasional (prob. rare) positive bias.
		b. too near vegetation	50 - 70 percent underestimation.	Occasional (prob. rare) negative bias.
		Sample Line Adsorption:		
		a. Good care & cleaning	10 $\mu g/m^3$ underestimation.	Possible general 10 $\mu g/m^3$ negative bias.
		b. Average care	20-25 $\mu g/m^3$ low from 50 $\mu g/m^3$.	Occasional 40-50% negative bias.
		c. Poor care (insects, dirt)	Probable greater underestimation.	Likely rare 50-90% negative bias.
		Flow Meter Problems:		
		a. Daily normal conditions	± 3 percent variation.	Negligible impact. Presumed ±3% precision of data.
		b. 8-port unit with only one weekly flow reading	± 5 percent variation.	-5% negative bias on high SO_2-BS days. +5% positive bias on low SO_2-BS days.
		Allowable Filter Clamp Leakage	1-2 percent underestimation.	Negligible impact. Presumed ±2% precision of data.
		Poor Clamp Care & Technique	5-10 percent underestimation.	Likely occasional 5-10% negative bias.
		Grade B Glassware Usage	2-5 $\mu g/m^3$ underestimation.	Negligible impact.
		Improper Alkalinity Buffering	5-10 $\mu g/m^3$ underestimation.	Occasional 5-10 $\mu g/m^3$ negative bias.
		CO_2 in Demineralized in H_2O	40 $\mu g/m^3$ low from 50 $\mu g/m^3$ monthly mean.	Occasional negative bias of up to 80%.[b]
		Atmospheric Ammonia	25 $\mu g/m^3$ underestimation on 10% of summer samples in urban areas. ≤80 $\mu g/m^3$ low on ind. days & 40 $\mu g/m^3$ low monthly summer mean in country areas.	≥25 $\mu g/m^3$ neg. bias on 10% of summer samples in urban areas. Occasional neg. bias in country areas- up to 80 $\mu g/m^3$ daily data & up to 100% monthly mean in summer.
		Titration Error:		
		a. Normal-sharp color change of indicator at pH 4.5	±5 $\mu g/m^3$ error in determination.	Presumed ± 5 $\mu g/m^3$ precision of data.
		b. Gradual color change of indicator at pH 4.5	±10 $\mu g/m^3$ error in determination.	Actual ± 10 $\mu g/m^3$ precision level.[c]
		c. Rounding off to 0.1 ml of alkali volume added.	±5 $\mu g/m^3$ error in determination.	Added ± 5 $\mu g/m^3$ precision error.[c]
		Evaporation of reagent:	≤15 $\mu g/m^3$ overestimation, especially in summer months.	15-100% pos. bias for SO_2 data <100 $\mu g/m^3$. 7.5-15% pos. bias for SO_2 of 100-200 $\mu g/m^3$. 3.25-7.5% pos. bias for SO_2 of 200-400 $\mu g/m^3$. ≤3.25% pos. bias for SO_2 data >400 $\mu g/m^3$.
		Temperature and Pressure:		
		a. Corrections - normal	5% underestimation.	General 5% neg. bias in SO_2 data.
		b. Large ΔP at filter	10% underestimation.	Occasional ~ ±10% negative bias in SO_2 data.

[a]Data from 1965-1968 most clearly impacted.

[b]Data from 1966-1967 most clearly impacted.

[c]At ≤50 $\mu g/m^3$ uncertainty due to these two errors is ~ 7 $\mu g/m^3$ or 14%. That is, 68% of the data are within 14% and 5% are >28% in error.

As mentioned above, the lead peroxide method was selected because its sensitivity, reliability and precision were demonstrated to be much better than that obtained in comparison to the lead dioxide method. More specifically, the British Standard for sulfur dioxide determination by the hydrogen peroxide method states that replicate determinations can be expected to be within ±20 $\mu g/m^3$ for concentrations up to 500 mg/m^3 and within ±4 percent for concentrations above 500 $\mu g/m^3$; and an OECD Working ~~Parting~~ PARTY stated the accuracy of the method to be ±10% at levels >100 $\mu g/m^3$. However, as summarized in Table 1-1, numerous sources of errors have been encountered in the practical application of the method in collecting data for the British National Survey over the past 15-20 years.

Certain of the sources of error listed in Table 1-1, it can be seen, resulted in relatively small errors, whereas others produced errors ranging up to 50-100% in magnitude. Also, some errors appear to have been restricted to affecting data from only limited locations (usually unspecified as to specific names of localities) or during only limited time periods. Many of these types of errors appear to have been detected fairly quickly and steps taken to successfully correct or minimize them. Still other sources of errors exist (e.g., those from reagent evaporation), which have likely affected essentially all British National Survey SO_2 data. Some of these appear to remain uncorrected to this date, in some cases more than 10 or 15 years after they were first detected and brought to the attention of Warren Spring Laboratory officials responsible for overseeing quality control for the entire National Air Pollution Survey. See Chapter 3 for a more detailed discussion of each type of error.

Taking the above information into account for present purposes, it would be extremely difficult to determine precisely which errors affected particular

National Survey data sets employed in British epidemiology and other studies discussed later in this document. That would likely require a thorough examination, on a time- and site-specific basis, of records detailing information on how each pertinent data set was collected and WSL quality control assessment reports for the data sets. Alternatively, in later evaluations of British epidemiology studies one could accept the following overall evaluation and set of conclusions by the WSL (1975) regarding British National Survey air pollution data (emphases added):

> The actual degree of accuracy attained in the Survey is not known. Input data are scrutinized by WSL staff, and subjected to computer checks, and any reflectances, titres, or air flows which are abnormally high or low or show unusually abrupt changes from one day to the next are queried and data known to be invalid are excluded from the annual summary tables. Such checks can however eliminate only some of the gross errors. More information will become available on accuracy when current (1974) plans to institute additional quality control, e.g., on reagent solutions, are put into operation. <u>However, although the accuracy of the Survey data cannot at present be quantified, many of the errors discussed in the previous paragraphs will cancel out when data are averaged over periods of a few months or a year, or for groups of sites. The remainder tend to show up as anomalies when data are compared with past or subsequent data at the same site or with data from other sites</u>; anomalies of this kind have been commented upon throughout the Reports. Members of Warren Spring Laboratory staff have devoted a large effort over the years to site visiting and checking on procedures. It is their experience that the vast majority of the instruments are maintained and operated with reasonable care and accuracy. <u>The Laboratory is therefore confident that the accuracy is sufficient for the type of data analyses carried out in the present series of reports</u>.

Presumably, it is the opinion of the WSL and British epidemiologists that the accuracy of the survey data is also sufficient to meet the original objectives of the Survey, ie. to assess the benefits accruing from the Clean Air Act of 1956, which requires use of the survey air quality data along with community health endpoint evaluations in order to define quanititative air pollution/health

effects relationships. That this presumption is likely correct is further attested to by the long history of reliance on these data by British epidemiologists, such as in the making of statements regarding such quantitative relationships in innumerable journal articles and reviews appearing during the past twenty years, up to and including the very recent review by Holland et al. (1979).

Daily Smoke Measurements of the United Kingdom National Survey--The general technique for the British Smoke shade (BS) measurement is described in detail in Chapter 2, and a detailed critical assessment of the measurement procedure is provided in Chapter 3 to allow for evaluation of the precision, accuracy, and reliability of the measurements. Also, details of the BS measurements are provided by an Instruction Manual (IM) issued by Warren Spring Laboratory in 1966. At the start of the National Survey in 1961 (WSL, 1961) it was recognized: "The daily instrument, while comparatively simple in design and operation gives reliable results in good hands* and seemed the best choice for the National Survey." WSL circulated the specifications of the apparatus and methods to all the cooperating organizations as careful, uniform work was essential if the results from the different sites throughout the country were to be comparable. However, WSL found that detailed instructions were necessary as most of the Local Authority staff making the measurements had no training in analytical techniques. These methods were reviewed by an O.E.C.D. Working Party and a report "Methods of Measuring Air Pollution" (OECD, 1964) was prepared, which was accepted into the British Standards Specification 1747, Parts 2 and 3. The Manual of Instruction (WSL, 1966) incorporated the improvements in techniques, "but apparatus and procedures are

specified in much greater detail to assist operation by observers with no technical knowledge."*

Partly due to the lack of analytical training of survey monitoring site operators, and other factors as well, various errors were encountered in carrying out BS measurements for the National Survey.

Table 1-3 summarizes information discussed in Chapter 3 on the sources, magnitudes and directional biases of errors associated with British smoke measurements during the past 30 to 40 years. For example, prior to 1961, the use of weights for sealing purposes led to highly variable errors in BS measurements due to leakage at filter clamps, and steps were taken to require screw-down clamps as standard procedure as part of the later British National Survey work implemented after 1961. It is not clear to what extent any specific British BS data sets from the 1950s may have been affected by the clamp leakage problem, but one must assume that such errors could not have often been very large or serious and that the WSL took appropriate steps to eliminate or invalidate any data in gross error as they were detected via their quality control efforts in the late 1950s. Analogously, there is evidence that WSL did take steps to inform users of pre-1961 BS data of errors arising from (1) comparing reflectance on filters to photographs of painted stains and (2) use of reflectance readings below 25 percent, where the stain was too dark to use the Clark-Owens DSIR curve. However, it also appears that only a few investigators (e.g., Commins and Waller, 1970) took steps to go back and correct published reports based on the affected pre-1961 data and to publish revised analyses taking into account corrections for the pre-1961 data errors.

*Underline added for present emphasis.

TABLE 1-3. SUMMARY OF EVALUATION OF SOURCES, MAGNITUDES, AND DIRECTIONAL BIASES OF ERRORS ASSOCIATED WITH AMERICAN SO_2 MEASUREMENTS

Time period	Measurement method	Reported source of error	Direction and magnitude of reported error	Likely general impact on American SO_2 data
1944-1968	Lead dioxide.	Humidity (RH).	Reaction rate increases with RH.	Variable positive bias, especially in summer.
		Temperature (T).	Reaction rate increases 2% per 5° rise.	Variable positive bias, especially in summer.
		Windspeed (WS).	Reaction rate increases with WS.	Variable positive bias, especially in summer.
		Saturation of Reagent (sulfation plate mainly).	Variable underestimation beyond pt. where 15% of PbO_3 on plate reacted.	Possible large negative bias, especially for 30-day samples for summer monthly readings.
		(Overall Errors).		Generally wide ± error band associated with data. Possible negative bias up to ≥100%, mainly in summer, with 30-day reading.
1969-1975 (EPA CHESS PROGRAM)	West-Gaeke Pararosanaline.	Spillage of reagent during shipment.	18% of total volume 50% of time; occasional total loss	Half of SO_2 data likely negatively biased by mean of 17%; some up to 100%.[a]
		Time delay for reagent-SO_2 complex.	SO_2 losses of 1.0, 5, 25, and 75% at 20, 30, 40, and 50°C, respectively.	Usually small (<5%) negative bias, but consistent negative summer bias up to 25% at 40°C temp, extreme.[b]
		Concentration dependence of sampling method.	Underestimation of unspecified magnitude at daily SO_2 >200 $\mu g/m^3$.	Probable general negative bias in daily, monthly, and yearly SO_2 data.[b]
		Low flow correction.	±10% to 50% variable error.	Usually error of < ±10%; occasionally up to ± 50% in daily, but dampened statistically in annual mean.[b]
		Bubbler train leakage.	Small underestimation error of unspecified magnitude.	Slight negative bias suspected.[b]
		(Overall errors).		From Nov., 1970, to Dec., 1971, data biased low by 50-100%. From Nov., 1971, to conclusion of CHESS Program in 1975, fall-winter data appear valid but summer data biased low by maximum of 60-80%. From 1972 to 1975 annual average data approximately 15-20% low. Daily data highly random, not useful.[c]

[a]November, 1970, to April, 1973, CHESS Program data impacted before error corrected.
[b]Applies to CHESS Program SO_2 data from all years 1970-1975.
[c]As summarized by Congressional Investigative Report (IR, 1976).

Probably of much greater concern than the pre-1961 BS measurement errors are those encountered after the establishment and initial implementation of the British National Survey in 1961. These include certain errors, e.g., the "computer error of 1961-1964," (see Figure 1-1), which were eventually detected by WSL and resulted in steps being taken to correct affected BS data in National Survey data banks. It is clear, however, that whereas users of the affected data may have been informed of such errors by WSL, virtually none of them have taken steps to (1) alert recipients of publications containing analyses based on the affected data of the likely inaccuracies or ranges of error involved; (2) to reanalyze the study results based on the affected data sets; or (3) to reissue or publish anew any revised analyses. In fact, even some Warren Spring Laboratory quality control literature prepared and published during the 1960s or 1970s and still in use may contain incorrect information and recommended standard procedures for BS measurements based on analyses "contaminated" by computer errors or other problems summarized in Table 1-2 and discussed in more detail in Chapter 3. One such example of this relates to ambiguities in the use of certain correction factors in calculating BS values, which have been questioned by Ellison (1968).

In regard to determining which British BS data sets and related epidemiology studies are affected by different post-1961 National Survey errors, it is again presently very difficult, as was the case with British SO_2 measurements, to specify with any confidence the nature and magnitude of specific errors impacting particular studies. This would probably require thorough examination of records and WSL quality control reports concerning each of the pertinent data sets. On the other hand one can project that certain data sets and British epidemiology studies were almost certainly affected by some subset of BS measurement errors and these are taken into account in evaluating

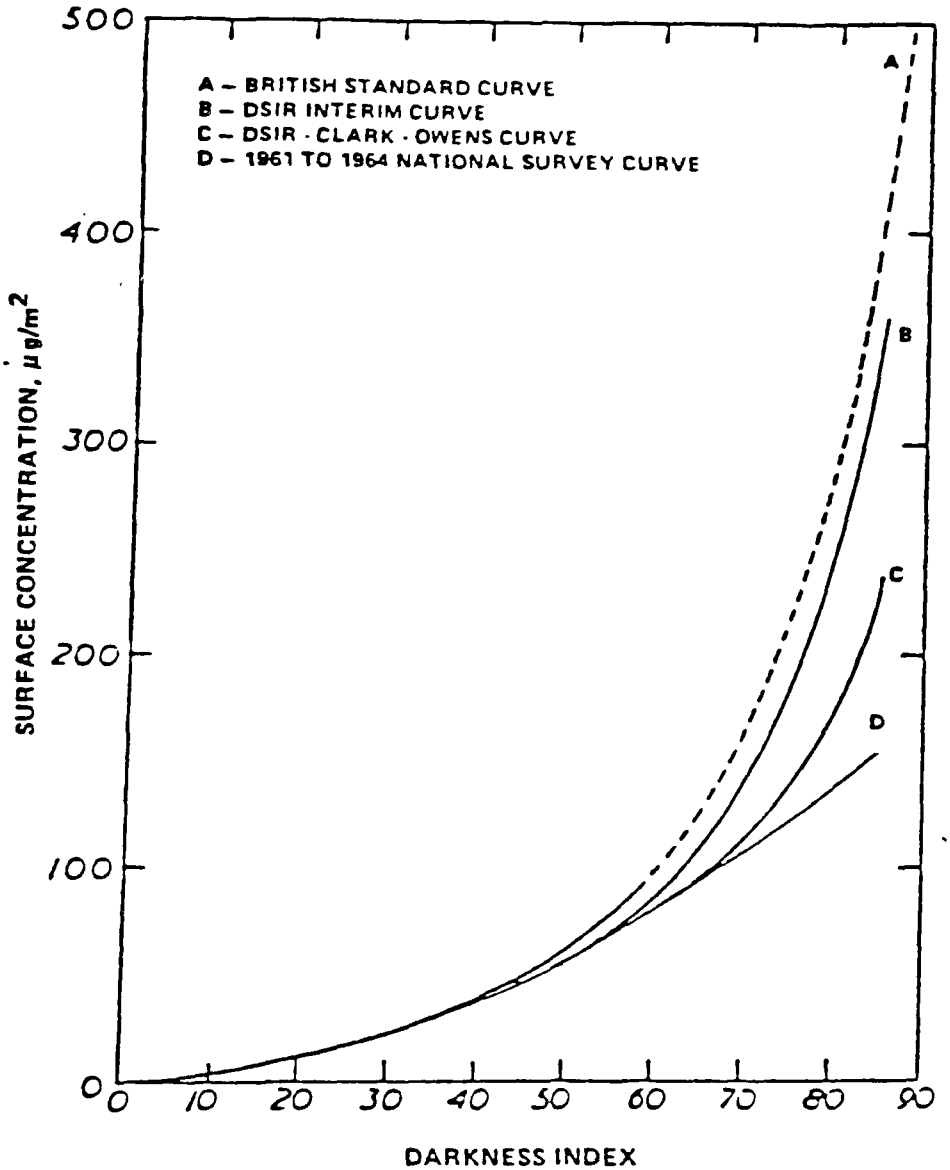

Figure 1-1. Comparison of smoke calibration curves for Eel reflectometer, Whatman No. 1 paper and a 1-in diameter filter. From WSL (1967) The computer followed curve D during 1961-64 instead of the correct curve(s) B and C.

such studies later this Chapter. For example, published reports of the "Ministry of Pensions" (1965) and Douglas and Waller (1966) studies contain specific reference to usage of National Survey data from the 1961-64 period and, therefore, the results of those studies should be reevaluated in light of measurement errors reported by the WSL for that period.

1.3.2.2 American Approaches

American SO_2 Measurements--Turning to American measurement approaches, different types of measurement methods for a given pollutant were adopted by various local, state, and federal agencies in establishing or expanding air quality monitoring systems that proliferated across the United States during the 1950s and 1960s. Rather than discuss methods used for SO_2 measurements by all of the different American air monitoring systems, main emphasis is placed here on the discussion of only certain key American applications of measurement methods for SO_x that are of crucial importance for later discussions of quantitative relationships between health effects and atmospheric levels of sulfur dioxide. These include mainly applications of SO_2 measurement methods as employed in the EPA "CHESS Program" as the single largest attempt to define quantitative relationships between air pollution and health effects.

In regard to sulfur oxides measurement approaches used in the United States, lead dioxide or other "sulfation rate" measurement methods were, as in Britain, widely employed prior to the early 1960s for assessing SO_2 air levels. However, probably to a somewhat greater extent than in Britain, sulfation rate measurement techniques continued to be used later into the mid or late 1960s by some monitoring programs in the United States or in connection with certain community health epidemiology studies, as discussed later in this

chapter. As shortcomings of the "sulfation" methods became more widely recognized, however, their use was generally abandoned and more specific methods for the measurement of SO_2 or other sulfur oxide compounds were adopted, as was done in Britain. The hydrogen peroxide acidimetric method (see OECD, 1965) selected for use in the British National Air Pollution Survey, however, was not very widely adopted in the United States for SO_2 measurements. Rather, versions of West-Gaeke (1956) colorimetric procedures were much more widely used in the USA. Conductivity measurements for SO_2 (Adams et al., 1971), based on an acidimetric method adaptation often used in automatic instruments and most suitable for measuring periods of around 24 hours, later began to be applied in the operation of some American air monitoring networks in the 1970s.

The West-Gaeke method was the method mainly employed in the EPA "CHESS Program" for determining SO_2 air levels for inclusion in analyses of community health end point data in "CHESS" epidemiology studies. The application of that method in the CHESS Program was accordingly most thoroughly discussed in Chapter 3. The types of errors in measurement associated with CHESS SO_2 data are summarized in Table 1-2, along with notation of some factors affecting earlier sulfation methods. Much of the information on the former subject is derived from a 1976 Congressional Investigative Report (IR) which contained a thorough evaluation of EPA CHESS Program air quality measurements and other aspects of the Program.

Looking at the types of errors associated with earlier American use of sulfation rate lead dioxide methods, similar effects of temperature, humidity, etc., as affected analogous British SO_2 methods are seen to apply here to American data as well.

TABLE 1-3. SUMMARY OF EVALUATION OF SOURCES, MAGNITUDES, AND DIRECTIONAL BIASES OF ERRORS ASSOCIATED WITH BRITISH SMOKE (PARTICULATE) MEASUREMENTS

Time period	Measurement method	Reported source of error	Direction and magnitude of reported error	Likely general impact on published BS data
1944-1950s	Smoke filter	Leakage at clamp. Weights used to make the seal.	Highly variable underestimation of BS levels.	Probable widespread highly variable negative bias.
Pre-1961		Comparing reflectance to photographs of painted standard stains.	Depending upon observer and value of R.	Probable widespread relatively small negative bias.
		Reflectance (R) below 25%, stain too dark with use of Clark-Owens DSIR curve.	50-100% underestimation.	Occasional 50-100% negative bias in some data sets.
1961-1964		Computer not following proper calibration curve.	≤80% underestimation at low R if not corrected by WSL (See Moulds,1961) and discussion of clamp size correction factor.	Negligible for BS <~100 $\mu g/m^3$. Increasing negative bias up to 80% as BS values increase over 100 $\mu g/m^3$.
1964-1980		Clamp correction factor for other than 1-inch clamp.	Uncertain; derivation cannot be verified. Possible ±20%.	Possible underestimate for 2-inch and 4-inch clamps. Possible overestimate for 1/2-inch and 10 cm clamps.
		Flow rate - normal 1 day.	±3% variation.	Presumed ± 3% precision level.
		Flow by 8-port with 1 reading per week.	-10% underestimation. +10% overestimation.	10% negative bias on high BS days. 10% positive bias on low BS days.
		Variability of reading reflectance.	±2 units of R	Error increases with BS level from ±10% at 50 $\mu g/m^3$ up to ±20% at 400 $\mu g/m^3$.
		Averaging reflectance instead of averaging mass/cm^2.	Highly variable underestimation due to nonlinearity of R.	Probable small negative bias at low BS levels, could be large at high BS.
		Use of coarse side of filter facing upstream.	6-15% underestimation.	Occasional negative bias of 6-15%.

TABLE 1-3. (continued)

Time period	Measurement method	Reported source of error	Direction and magnitude of reported error	Likely general impact on published BS data
		Reading of wrong side of stained filter.	50-75% underestimation.	Occasional negative bias of 50-75%.
		Leakage at filter clamp		
		a. Normal, with good care	1-2% underestimation.	General 1-2% negative bias.
		b. With inadequate care.	2-8% underestimation.	Occasional 2-8% negative bias.
		c. Careless loading where uneven stains are produced.	10-20% underestimation.	Occasional 10-20% negative bias.
		Use of wrong clamp size		
		a. Stain too light R>90%.	Highly variable overestimation.	Data usage not recommended.
		b. Stain too dark R<25%.	Highly variable underestimation.	Data usage not recommended.

Turning to American applications of SO_2 measurements since the widespread abandonment of sulfur dioxide sulfation rate methods in the mid to late 1960s, several different types of errors were identified as being associated with EPA CHESS Program SO_2 measurements via a thorough evaluation of the CHESS Program, as reported in the IR (1976). As can be seen, the magnitudes of some errors in CHESS SO_2 measurements spanned about the same range as those seen for British National Survey SO_2 measurements and, at times, derived from analogous sources of error, e.g., evaporation or other loss of reagents. In the case of the American CHESS Program data, however, the specific overall impact of the various detected errors on particular CHESS data sets appears to have been more definitively defined by the work of the IR (1976); more specifically, it appears that the CHESS data generally tended to be somewhat negatively biased in comparison to other local or state SO_2 data from monitoring sites proximal to the CHESS sites, with the local and state data judged by the IR (1976) to be reasonably accurate and reliable. The specific magnitude of the negative bias for particular years of CHESS data is summarized in Table 14-3, and appears to have been around 30-40% in some circumstances and up to around 100% in other cases.

American High-Volume TSP Sampling Measurements--As discussed earlier, the hi-volume TSP sampler, since its development in the early 1950s, has been the instrument most commonly used in United States for measurement of atmospheric particulate matter; and high-volume TSP readings have most typically been used in American epidemiology study evaluations of associated air pollution-health effects relationships. In contrast, other particulate matter measurement approaches (e.g., the coefficient of haze method) saw only relatively limited application during the 1950s and early 1960s in certain American locations

and were infrequently used in estimating quantitative relationships between airborne particulate matter and health or welfare effects. Accordingly, major emphasis is placed below on the critical appraisal of certain key applications of hi-volume TSP measurements in the United States. As before, in discussing American applications for measurement of oxides of sulfur, the present summarization focuses most heavily on evaluation of applications of TSP measurement methods employed as part of the EPA "CHESS Program," as the single most extensive and comprehensive use of such methods as part of American community health epidemiology studies. Much of the information is derived from the 1976 Congressional Investigative Report (IR), which included a thorough analysis of EPA CHESS Program TSP measurements and comments regarding certain local or state TSP measurements.

The main sources, directions and magnitudes of errors identified as possibly affecting American TSP measurements are summarized in Table 1-4. In addition to various sources of minor errors inherent to the basic TSP sampling method, certain other nuances of procedures included in the Federal Reference Method (40 CFR 50, Appendix B) may have resulted in the introduction of an additional slight negative bias in TSP data obtained by American researchers. This, more specifically, pertains to the manner in which flow rate calculations are made upon which final TSP concentration determinations are based.

The Federal Reference procedure calls for the averaging of the initial and final recorded airflow rates. However, as described in Appendix 3-A of Chapter 3, the uncontrolled flow rate drops more rapidly at the start of the run than at the end of the run. Therefore, a linear approximation leads to an overestimate of the flow rate, which will reduce the measured value. Consequently,

TABLE 1-4. SUMMARY OF EVALUATION OF SOURCES, MAGNITUDES, AND DIRECTIONAL BIASES OF ERRORS ASSOCIATED WITH AMERICAN TOTAL SUSPENDED PARTICULATE (TSP) MEASUREMENTS

Time period	Measurement method	Reported source of error	Direction and magnitude of reported error	Likely general impact on published BS data
1954-1980	High-Volume TSP	Time Off (Due to power failure).	Variable underestimation.	Negligible impact, rare negative bias.
		Weighing error.	±2% random variation.	Negligible impact.
		Flow measurement (with control).	±2% random variation.	Negligible impact.
		Flow measurement (without control)		
		a. Constant TSP--Average of flows.		
		1. Low TSP level.	2% underestimation.	Negligible impact.
		2. High TSP level.	5-10% underestimation.	Possible 510% negative bias.
		b. Rising TSP-Average of flows.	10-20% underestimation	Possible 10-20% negative bias.
		c. Falling TSP-Average of flows.	10-20% overestimation.	Possible 10-20% possible bias.
		Aerosol evaporation on standing.	1-2% underestimation.	Probable negligible impact.
		Condensation of water vapor.	5% overestimation.	Possible 5% positive bias.
		Foreign bodies on filter (Insects).	Generally small overestimation.	Possible 5% positive bias.
		Windblown dust into filter during off-mode.	Generally small overestimation.	Occasional (rare) positive bias.
		Wind speed effect on penetration of dust into the Hi-Vol shelter.	Less penetration at high windspeed.	Occasional (rare) negative bias.
		Wind direction effect due to Hi-Vol Asymmetry	Higher penetration when normal to sides.	Probable increase in random (±) error.
		Artifact formation, NO_3^- $SO_4^{=}$.	5-10 $\mu g/m^3$ overestimation.	Occasional positive bias.

TABLE 1-4.(continued).

Time period	Measurement method	Reported source of error	Direction and magnitude of reported error	Likely general impact on published BS data
1969-1975 (EPA CHESS Program).	Fed. Reference Method Standard Hi-Vol Sampler	Loss of sampling material in field.	No specific estimate of magnitude of error; but would be underestimation.	Probable slight negative bias in Utah winter data. No known impact on other CHESS TSP data.
		Loss of sampling material in mailing.	Reported 4-25% apparent loss; max. likely due to crustal (sand, etc.) fall-off from selected Utah sampling sites.	Probable general small <10% negative bias; occasional 25% negative bias.
		Evaporation of organic substances.	No specific estimate of error magnitude, but not likely to exceed 5% underestimation.	Probable slight negative bias of <5% for TSP data from urban/industrial areas.
		Windflow velocity and asymmetry.	No specific estimate of error magnitude; but most likely to increase random variation of small underestimation.	Negligible impact or slight negative bias.
		(Overall errors).		Generally <10% negative bias; occasional 10 to 30% negative bias.

all TSP data computed in this manner have a slight negative bias which is likely usually of the order of 5 percent; on occassion, however, under circumstances where the flow rate may have fallen below 40 ft^3/min, larger errors (up to approximately 15 percent) may have been introduced. Assuming that monitoring site operators in the United States adhere to the recommended Federal Reference Method procedures, then this type of bias is likely inherent in essentially all American TSP data collected without flow rate control or recording. Despite such problems, it can be seen that the maximum range of uncertainty derived from the various errors associated with American TSP measurements is generally less than 20 percent in either a positive or negative direction on a random (±) basis.

Errors in addition to general TSP measurement errors reported by the 1976 Congressional Committee Investigative Report (IR, 1976) to affect CHESS Program TSP measurements during 1969-1975 are broken out and listed seperately in Table 1-4. Some of those errors (e.g., loss of sample materials in filter removal from the field monitoring apparatus) were reported by the IR (1976) as likely affecting only very restricted CHESS data sets. Others, e.g., errors due to loss of sample in mailing, appear to have been more widespread and presumably impacted on many CHESS data sets. It is interesting to note, however, that the IR (1976) concluded that the net effect of all of the errors was to introduce, in general, a slight negative bias of 10 to 30 percent into CHESS TSP data, which is not much beyond the range of different types of errors (e.g., linear flow corrections) more generally associated with American applications of TSP measurements. Section IV C 3 of the IR (1976) further concluded that:

> "...the TSP data were by far the best quality data taken in the CHESS monitoring program. Differences measured between High and Low sites are probably reasonable estimates of the differences of TSP exposures as received by populations in these areas."

It appears reasonable to concur with the IR (1976) and, accordingly, to accept CHESS TSP measurements as reasonable estimates of TSP exposures of CHESS Program community health study populations, taking into account that such data may be biased low by no more than 10 ~~or, at most~~ to, 30 percent.

1.3.2.3 BS-TSP Comparison Studies--In Chapter 3, information is reviewed on BS-TSP comparisons derived from a number of studies published over the past twenty years and reporting results obtained from the sampling of air in many disparate geographic areas (Britain, other European countries, and the United States) and varying time periods (from the early 1950s to the mid 1970s). It was earlier hinted at and is now clear from a present perspective, with the advantage of viewing the various BS-TSP comparison data sets together in relation to each other, that a nonlinear relationship exists between BS and TSP measurements obtained with colocated samplers. That is, regardless of where or when such readings were obtained, TSP values were usually found to be two or more times higher than corresponding BS readings up to BS levels of about 100 $\mu g/m^3$. At higher levels, however, the TSP and BS readings tend to converge toward each other, such that TSP/BS approaches unity at BS levels of 500 $\mu g/m^3$ or more. Thus, above 500 $\mu g/m^3$ or so, BS and TSP readings from colocated samplers are essentially identical.

In carrying out analyses of BS-TSP comparison data sets, various investigators in the past generally employed linear regression analyses in an effort to define straight lines that best fit data points obtained by them over a limited range of BS-TSP values (see Figure 1-2). They also often extrapolated the thusly defined straight lines to BS-TSP values beyond the range of their emperical observations and found inconsistencies between the BS-TSP relationships implied by their line(s) and those defined by linear analyses of different

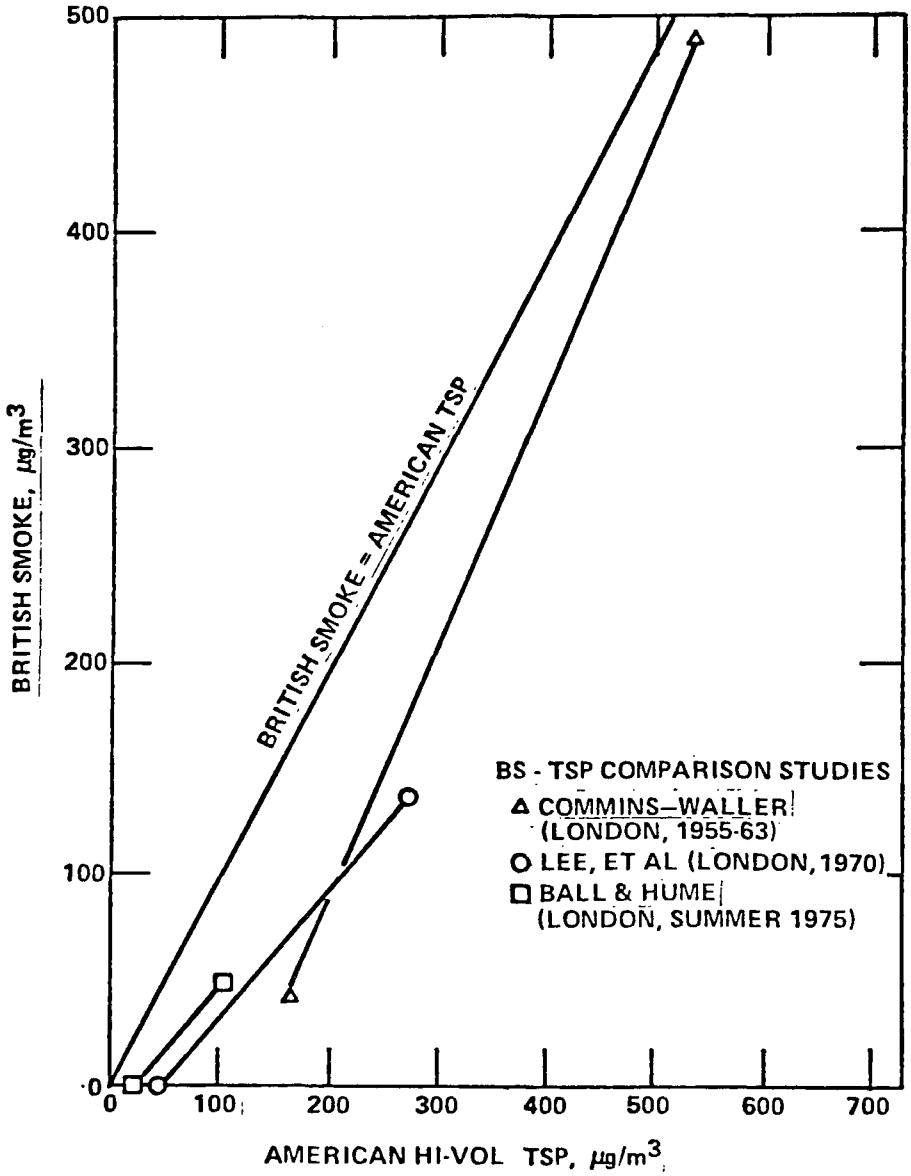

Figure 1-2. Representative examples of BS/TSP relationships defined by linear regression analyses employed to fit BS/TSP comparison data points as described in published reports.

BS-TSP comparison data sets obtained at other times or locations. Such apparent inconsistencies between BS-TSP relationships, arising from linear regression analyses of various data sets obtained over limited and often different ranges of BS-TSP values, have contributed to and reinforced the view that no consistent relationships exist between BS and TSP measurements obtained at different locations or even at different times at the same location. Paradoxically, this view has gained widespread credence despite the concurrent realization that corresponding BS-TSP readings are nonlinearly related.

Recognition of the acknowledged nonlinearity of BS-TSP relationships and the necessity to meet certain boundary conditions defined by emperical observations and certain theoretical considerations as sine-qua-non starting points led to the formation of a "bounded nonlinear model" (BNLM) proposed in Chapter 3 as a unifying concept or means by which to interconvert monthly or annual average BS and TSP values obtained under vastly different circumstances. The BNLM (see Figure 1-3) essentially employs a power function defining a nonlinear relationship that meets the boundary conditions of (1) BS $\rightarrow$ 0, where TSP $\rightarrow$ 0 and (2) BS $\rightarrow$ TSP, as BS $\rightarrow \infty$; or, in other words, when there is no particulate matter in the air, both TSP and BS readings must be zero or tend toward zero and, also, they must tend to converge toward each other as BS values become very large as observed in empirical BS-TSP comparison studies. In addition, the particular BNLM model chosen defines a curve which fits other empirically-derived observations to the effect that TSP/BS = 2 at 100 $\mu g/m^3$ BS and TSP/BS = 4/3 at 250 $\mu g/m^3$ BS. Plotting of corresponding BS-TSP values from numerous published BS-TSP comparison studies reveals that the BNLM model fits well virtually all presently available BS-TSP comparison data, in the mean.

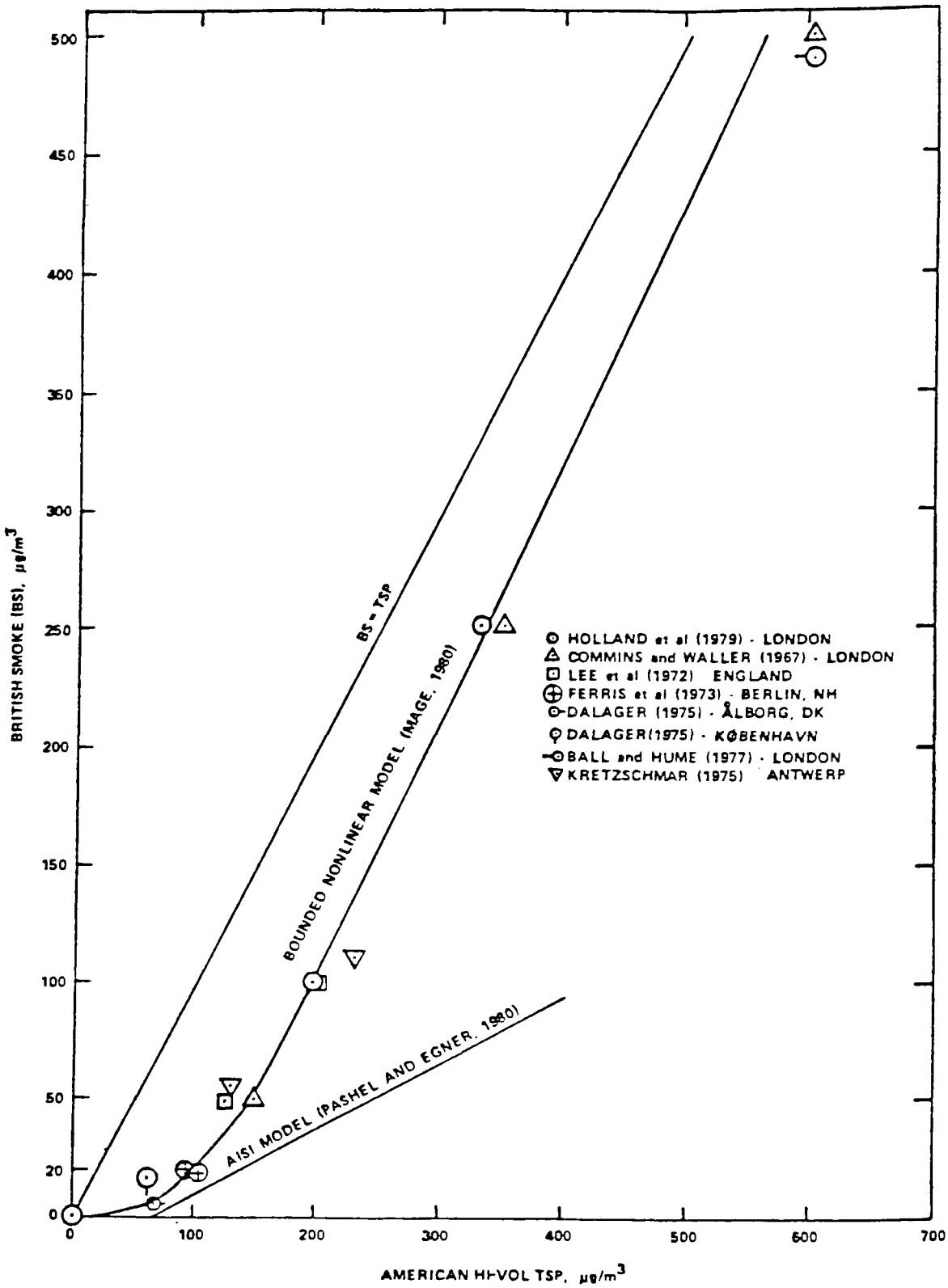

Figure 1-3. Measurements of British Smoke vs Hi-vol TSP, showing a consistent relation between these measures over the entire range of reported observations. Most points shown are annual mean values; see text for discussion.

Only the reported results from one recently obtained data set comparing BS and TSP values from colocated samplers at 16 locations in the United States appear, at first sight, to be greatly inconsistent with the BNLM model (see AISI line in Figure 1-3). Closer inspection of the study, however, reveals that numerous methodological errors were made in conducting the study, including the failure to follow published standard procedures employed in the collection of BS data earlier used in British epidemiology studies on the health effects of particulate matter and, also, in most other BS-TSP comparison studies. Nevertheless, when the particular methodological errors and other deficiencies are evaluated, it becomes apparent that some of the basic observations reported may not be as inconsistent with other published results and the BNLM model as initially seems to be the case. See Chapter 3 for a more detailed discussion of this matter.

1.3.3 Sources and Emissions

Sulfur oxides and particulate matter are emitted into the atmosphere from a variety of sources, both natural and man-made. Natural particulate matter emissions include terrestrial dust (windblown soil and rock particles), radioactive particles, sea spray, volcanoes, biosphere emanations (products of biological processes), and biomass burning. Most natural particle mass is greater than 1 μm in diameter. On a global scale, natural emissions of sulfur oxides and particulate matter into the atmosphere greatly exceed man-made emissions. The latter, however, tend to be much more concentrated on a local or regional scale.

In the United States, anthropogenic air pollutants, that is, those pollutants associated with human activities, are primarily caused by the combustion of fossil fuels and the smelting of metals. The marked increase in energy consumption since the middle of the twentieth century has led to a generally upward trend in emissions of particulate matter and sulfur oxides.

Projections indicate that secondary PM will account for an increasingly greater proportion of the total emissions over the next decade. (Secondary particles are formed by atmospheric conversion of gases.) Most of this increase will occur in the fine fraction (less that 2-3 μm), and its impact will be felt over increasingly greater geographic areas. For example, the use of higher smoke stacks will result in a longer range transport of SO_2. This long-range transport creates more secondary fine particulate matter and greater geographical distribution of particulate pollutants.

Man-made emissions of sulfur oxides from stationary sources in the United States are currently estimated at 29 million metric tons per year. Transportation sources contribute less than 1 million metric tons of sulfur oxides, or not quite 3 percent of the total emissions. Of all stationary sources, utility plants burning coal and oil are the primary contributor (about 62 percent), while other industrial processes account for about 32 percent, and residential and commercial use of coal and oil accounts for less than 3 percent. Emissions vary considerably from one region to another; over 75 percent of the total national emissions come from the eastern half of the United States. Annual emissions of sulfur oxides have increased from about 23 million metric tons in 1940 to as high as 35 million metric tons in 1973. Although the past few years have seen a slight decline in emissions, an increase to nearly 40 million metric tons is projected by 1990 unless reduced by implementatio of air regulations.

Over 90 percent of the national sulfur oxide emissions occur in the form of sulfur dioxide; the balance consists of sulfates in various forms. The quantity and composition of these emissions vary from source to source and depend on factors such as fuel characteristics, operating conditions, and emissions-control equipment.

The characteristics of particulate matter, like those of sulfur oxides, vary according to source, emissions-control equipment, and other factors. Man-made emissions of particulate matter amounted to about 12.5 million metric tons in 1975 in the United States. The distribution of sources varies geographically: the eastern half of the United States accounts for 66 percent of the national total. Forty percent of the emissions come from stationary fuel combustion sources, principally in electric generation and industry and 25 percent from mineral processing. Additional emissions come from primary metal production, land vehicles, food and agricultural processes, solid waste disposal, and other sources. In addition, there are considerable emissions of fugitive particulate matter from both industrial (3.3 million metric tons/yr) and non-industrial (4.9 million tons/yr) sources.

1.3.4 Environmental Concentrations and Exposure

To protect the public health from air pollution, it is important to know how many people are likely at risk from pollutant concentrations capable of inducing adverse effects. For financial and technical reasons it is impractical to obtain direct measurements of pollutant doses incurred by individuals. It is necessary, therefore, to rely on fixed-point air monitoring to estimate pollutant exposures in representative environments. Such monitoring is conducted predominantly outdoors--in the ambient air, within the first 100 feet above the surface. A meaningful evaluation of population exposure to sulfur oxides and particulate matter must include measurements of specific local and area-wide concentrations, data on particle size and composition, and knowledge of the number of people in specific areas and their activity patterns.

Fixed-point ambient air monitoring conducted at several thousand sites in the United States over the last three decades shows a general trend of declining pollutant concentrations. For example, in the late 1950s, monitors in five

heavily industrialized cities were recording annual arithmetic mean concentrations of total suspended particles in the range of 130 to 195 $\mu g/m^3$. By the early 1970s, those sites were recording annual means in the range of 70 to 115 $\mu g/m^3$. By 1978, only 17 percent of reporting stations in the nation were exceeding annual average particulate levels of 75 $\mu g/m^3$ and six percent were exceeding the levels of 260 $\mu g/m^3$.

Studies of urban aerosols indicate that while the mass balance of total TSP is similar from city to city, its chemical composition varies. Many of these differences arise from the type of fossil fuel used and the type of industries dominant. For example, soot produced from the combustion of fuel oil can vary from 1 to 13 percent of the TSP mass in a particular area. Coal soot, on the other hand, may account for up to 30 percent of the TSP mass in some urban areas.

Sulfur dioxide concentrations have also decreased. In 1964, a representative group of 32 SO_2 monitors across the nation averaged about 50 $\mu g/m^3$, with the peak station reporting an annual average of some 200 $\mu g/m^3$. By 1971, their composite average had decreased to about 25 $\mu g/m^3$, and the peak station reported less than 50 $\mu g/m^3$. In 1978, only one percent of reporting stations were exceeding the primary annual standard for SO_2 (80 $\mu g/m^2$); and two percent were exceeding the primary 24-hour standard (365 $\mu g/m^3$). Sulfur dioxide levels in the ambient air generally rise and fall according to the amount of coal or other fossil fuels burned other than natural gas.

Analyses of some specific constituents of air pollution, notably sulfates, nitrates, and fine particles (<2.5 $\mu g/m$ MMD) indicate that these constituents are not declining at the same rate as the gross particulate concentrations.

Sulfate, ammonium, and nitrate ions dominate the fine particle fractions in urban and rural areas. Generally, toxic elements such as arsenic and lead

are associated with the fine fractions, while less toxic elements such as calcium and iron occur more often in the coarse fraction.

At least 50 percent of atmospheric particulate sulfur occurs in the fine fraction as sulfates and often accounts for 40 percent or more of the fine fraction mass. Most of the sulfate aerosols occur as ammonium salts rather than as sulfuric acid. High sulfate levels occur more often in summer than in winter.

There are regional differences in trace metal concentrations. Vanadium and nickel, for example, are correlated with the use of fuel oil. Their concentrations are, therefore, highest during the winter months. But in areas requiring low sulfur fuels, vanadium and nickel levels are quite low, since these elements are removed during fuel desulfurization processes. Where coal is used, titanium tends to exist at somewhat higher concentrations.

1.3.5 Transmission Through the Atmosphere

Pollutants emitted into the atmosphere are transported vertically and horizontally, transformed physically and chemically, and deposited by dry and wet removal mechanisms. Since each of these processes is a function of numerous physicochemical and meteorological variables, source-receptor relationships are necessarily complex. Despite the difficulty in analyzing atmospheric transmission, certain findings have been substantiated.

Atmospheric particulate mass is distributed bimodally in relation to particle size: fine particles are smaller than 2.5 micrometers (μm) and coarse particles are larger than 2.5 μm. Comparable masses of fine and coarse particles have been measured within urban areas. Outside of urban areas, the fine particulate mass tends to exceed the coarse particulate mass, especially in the Eastern United States.

Many fine particles are formed in the atmosphere from precursor gases. Most coarse particles are emitted directly from combustion or industrial processes, or are of natural origin, and become suspended in the atmosphere through human activity.

A portion of the fine particulate mass is the result of the atmospheric conversion of sulfur dioxide, nitrogen oxides, and higher molecular weight hydrocarbons (C_6+) to particulate sulfate, nitrate, and organic aerosols, respectively. The remainder of the fine particulate mass consists of combustion-derived sulfates and carbonaceous particles, lead, small amounts of other metal-containing particles from combustion and industrial processes, and small amounts of finely divided dusts. The coarse particulate mass consists of substances emitted directly from industrial sources and from combustion processes, along with substantial amounts of suspended or resuspended dusts of various types.

Because their deposition occurs over several days, fine particles can be transported for distances up to 1000 kilometers or more from their origins or the origins of their precursor gases. Finely divided particles formed continuously by secondary atmospheric reactions are especially likely to undergo long-range transport and to exist at appreciable concentration levels. Since urban-area sources and large point sources contribute precursors of secondary particles, concentrations of such particles may become superimposed upon those of fine particles during transport.

Gas-to-particle conversion processes depend on a variety of environmental parameters, such as solar radiation, concentrations of oxidizing radicals, and humidity. Therefore, precursor gas emission rates alone are of very limited utility in estimating the mass of secondarily formed fine particles.

Although it has been known that many factors contribute to sulfate levels in the atmosphere, past results suggest that heterogeneous reactions may play an important role in determining those levels. More detailed studies are necessary to assess the role of these reactions. Past work has shown the importance of temperature, oxygen, water, and other parameters in removal rates.

1.4 WELFARE EFFECTS ASPECTS

The third volume of this document discusses welfare effects of sulfur oxides and particulate matter. This includes information summarized below on: effects on vegetation (Section 1.4.1); acidic precipitation formation and effects (1.4.2); effects on visibility and climate (1.4.3); and materials damage effects (1.4.4). Major emphasis is placed in the discussion of lengthy materials contained in Chapters 7 and 8 on vegetation effects and acidic precipitation.

1.4.1 Effects on Vegetation

Terrestrial vegetation is normally exposed to a variety of substances from the atmosphere. Among these substances are sulfur oxides, particulate matter, and other phytotoxic pollutants. More is known concerning the effects of sulfur oxides, mainly sulfur dioxide, than about the effects of particulate matter.

The sensitivity to sulfur dioxide of plant species and varieties differs because of the genetic composition of the plants and the influence of environmental conditions on their response. Temperature, light, humidity, other air pollutants, soil conditions, and the stage of plant growth all interact in affecting the sensitivity of plants to injury from sulfur dioxide and particulate matter. Since the ambient air contains many substances other than sulfur dioxide and particulate matter, interaction with these other substances must be considered when analyzing the effects of sulfur oxides or particulate

matter on vegetation. Mixtures of the substances in the ambient air, some of which may be nitrogen oxides or ozone, may have adverse growth or foliar effects of a greater magnitude than the effects from sulfur dioxide alone.

The response of a given variety or species of plants to a specific air pollutant cannot be predetermined on the basis of the known response of related plants to the same pollutant. Neither can the response be predetermined by a given response of a plant to similar doses of different pollutants. The interplay of genetic sensitivity and environmental influences must be considered for each plant and pollutant.

In general, sulfur dioxide must enter a plant to induce toxic effects, although some toxic actions on plant surface coatings can be induced by surface contact alone. It is generally accepted that entry of sulfur dioxide into a plant is through leaf openings termed stomata. Environmental conditions (e.g., light, temperature, and humidity) that favor open stomata at the time of exposure permit the assimilation of SO_2. In the plant, SO_2 is converted to sulfite or bisulfite and eventually to sulfate. Sulfite and bisulfite are approximately 30 times more toxic than sulfate.

Air pollutants, as do all plant stress-inducing agents, initiate changes within plant metabolic systems. Changes in metabolic pathways may lead to extensive physiological dysfunctions. If physiological dysfunctions are severe, visible symptoms may be manifested. Two basic forms of injury occur: (1) injury not visibly expressed that may or may not result in reduction in growth or loss in yield; and (2) injury visibly expressed as clinical symptoms that may be observed, recorded, and evaluated.

As long as the rate of absorption in plants does not exceed the rate of conversion of SO_2 to sulfate, the only effects of exposure to SO_2 may be changes in stomatal opening or closing, or biochemical or physiological changes. If SO_2 exposure concentrations are reduced, abatement of effects may occur.

Reduced growth and yield and/or predisposition to other biotic or abiotic stress-inducing agents may occur if alterations in plant metabolism or physiology persist for a period of time. Significant reductions in growth and yield of major forest tree species and agronomic crops have been reported without the presence of visible symptoms.

Visible symptoms result from both acute and chronic injury. Acute injury symptoms include necrosis or death of cells, tissues, organs, or the entire plant. Chronic injury symptoms include plant responses that usually involve chlorophyll disruption, followed by induction of chlorosis or yellowing of tissues. Pigmentation changes resulting in stippling or general discoloration characterize this type of injury. Chronic injury results from either high-dose or low-dose exposure; high-dose exposure may lead rapidly to acute injury. These terms refer to plant response rather than to the exposure conditions and the dose received. Dose-response relationships relate variations in the length of exposure and pollutant concentration to variations in plant responses as mediated by the plant response system and the environment. Dose is defined as the concentration of pollutant multiplied by the length of the period of exposure.

Variations in exposure regimens and response measurements frequently make it difficult to compare the results of different studies. The conclusions listed below are based on a synthesis of the data discussed in Chapter 7 and summarized in Tables 1-5, 1-6, 1-7. These conclusions were formed by summarizing the dose-response data without considering the confounding environmental variables: (1) Significant suppression in yield of economically important agricultural species by SO_2 concentrations in the range from 0.05 to 0.06 ppm can occur if the period of exposure is at least two weeks in length. (2) Both

TABLE 1-5. DOSE-RESPONSE INFORMATION SUMMARIZED FROM LITERATURE PERTAINING TO CULTIVATED AGRONOMIC CROPS AS RELATED TO FOLIAR, YIELD, AND SPECIFIC EFFECTS INDUCED BY INCREASING SO_2 DOSE

Conc, ppm	Exposure time	Exposure condition[b]	Plant[c]	Effects on Foliar	Effects on Yield	Species Effect[c]	Reference
0.01	10 min	EC	Corn			Stomata open wider	448
0.01	Growing season		Oats	X		Light leaf injury	159
0.015	Growing season		Wheat		X	15% decrease in grain weight yield	159
0.02	10 min	EC	Bean			Stomata open wider	448
0.035	3 hr for 8 exp, growing season	F/CC	Wheat			No effect on apparent photosynthesis, no effect on the average head length or number of grains per head	394
0.02 0.05 0.10	Growing season (2 year)	F-ZAP	Winter wheat Prairie June grass			S content increased with increase in SO_2 concentration; digestibility of dry matter was reduced by 2 years of treatment; crude protein content in winter wheat decreased significantly	473
0.03-0.10	72 hr/wk for growing season	F/CC	Barley Durham wheat Spring wheat		X	No effect on yield	473
0.3-0.06 0.10 0.15 0.20	24 hr/day for 30 days	F/CC	Winter wheat ev. Yamhill ev. Ilyslop		X X	0.03 ppm increased yield 27% 0.06-0.15 ppm, no effect 0.06 ppm decreased yield 22%) 0.20 ppm decreased yield 70%	473
0.035 1.75	8 hr	EC/SD	Broadbean			Depressed photosynthesis	40
0.05	10 min	EC	Broadbean			Stomata open wider; threshold 0.02 ppm for 10 min	37

TABLE 1-5. (continued)

Conc, ppm	Exposure time	Exposure condition[b]	Plant[c]	Effects on Foliar	Effects on Yield	Species Effect[c]	Reference
0.05	5 hr/day; 5 day/wk for 4 wk	EC/SD	Alfalfa		X	26% decrease in foliage dry weight	430
					X	49% decrease in root dry weight	
			Tobacco		X	22% decrease in leaf dry weight	
0.05 0.10 0.25	4 hr	EC/SD	Oats Radish Soybean Tobacco	X		No foliar injury	431
0.05 0.20	8 hr/day 5 days/wk for 18 days	EC/SD	Soybean	X		No effect on top fresh or dry weight; root fresh or dry weight; plant height; shoot/root fresh or dry weight ratio	
0.06	68 days	EC/SD	Alfalfa		X	28% decrease in foliage stubble, 45% decrease root dry weight 21% decrease in total protein content, amino acid content, total nonstructured carbohydrates, symbiotically fixed nitrogen	327
0.065 0.13 0.26	1-55 days	GC	Cabbage Chinese cabbage Cucumber Eggplant Lettuce Spinach	X X X X X X		Foliage injury threshold 0.13x27 days Foliage injury threshold 0.26x55 days Foliage injury threshold 0.13x26 days Foliage injury threshold 0.13x11 days Foliage injury threshold 0.13x15 days Foliage injury threshold 0.13x1 day Other parameters measured such as plant height, number of leaves, top fresh weight, number of flowers, fresh weight vs. dry weight of roots were not found to be significantly different from controls	140
0.10	20 min	EC	Bean Corn			Stomata open wider, effect also shown to occur in dark	448
0.10	1 hr	EC	Bean			Stomata open wider, water-stressed plant had wider opening of stomata compared with controls	448
0.10	8 hr	GH	Tobacco	X		Foliar injury threshold for development of fleck-like lesions	283

Conc, ppm	Exposure time	Exposure condition[b]	Plant[c]	Effects on Foliar	Effects on Yield	Species Effect[c]	Reference
0.10	6 hr/day for 133 days	F/CC	Soybean		X	No significant effect on growth or yield; 92nd-day defoliation was 12% greater; 135th-day seed weight was 1% reduced from control	169
0.10	18 days	GC	Pea		X X	3% decrease in fresh weight of shoot 5% decrease in dry weight of shoot 4% decrease in total nitrogen 30% decrease in H^+ (buffer capacity) 10% increase glutamate dehydrogenase activity 110% increase in inorganic sulfur content	204
0.10	6 hr/day 43 days 92 days 133 days	F/OT	Soybean			No significant effect on foliar injury, defoliation fresh weights, seeds/plant, or weight of seeds/plant	171
0.125-1.0	1-3 hr	EC/SD	Oats Radish Sweet pea Swiss chard	X		No foliar injury	32
0.15	18 days	GC	Pea		X X	3% decrease in fresh weight of shoot 8% decrease in dry weight of shoot 2% decrease in total nitrogen 35% decrease in H^+ (buffer capacity) 32% increase in glutamate dehydrogenase activity 140% increase in inorganic sulfur content	204
0.15	72 hr/wk for growing season	F/CC	Barley Durham wheat Spring wheat	X X	X	42% decreased yield in Durham wheat; 44% decreased yield in barley; no effect on spring wheat	473
0.25	18 days	GC	Pea		X X	32% decrease in fresh weight of shoot 26% decrease in dry weight of shoot 24% decrease in total nitrogen 42% decrease in H^+ (buffer capacity) 80% increase in glutamate dehydrogenase activity 150% increase in inorganic sulfur content	204
0.11	103.5 hr/wk for 20 wk	GC	Cocksfoot Meadowgrass Italian ryegrass Timothy		X X X X	40% decrease in total dry weight 28% decrease in total dry weight Nonsignificant 51% decrease in total dry weight (Yield reductions were related to decrease in leaf areas)	13,14

TABLE 1-5. (continued)

Conc, ppm	Exposure time	Exposure condition[b]	Plant[c]	Effects on Foliar	Effects on Yield	Species Effect[c]	Reference
0.15	24 hr	EC/SD	Corn Rice			Absorbed SO_2 remained in water-soluble form and very difficult to assimilate to protein	490
0.15- 0.30	7 days	EC/SD	Barley	X		Severe foliar injury	288
			Bean	X		No injury	
			Corn	X		Severe foliar injury	
0.15	14 days	GH	Celery	X		Increased peroxidase activity, caused chlorosis of leaves	356
			Big plantain	X		Increased peroxidase activity, decreased buffering capacity of cells, caused necrotic leaf injury	
			Bean	X		Caused necrotic leaf injury	
			Big mallow	X		Caused necrotic leaf injury	
0.17	2 hr	EC/SD	Broadbean			Decreased photosynthetic rate, decreased stomatal resistance if RH > 40%, increased stomatal resistance if RH < 40%	39
0.20 0.25	1 hr	EC/SD	Alfalfa			No effect on photosynthesis	471
0.25	2 hr	GC	Alfalfa Barley Oats			Threshold dose for inhibition of photosynthesis	30
0.20 0.30 0.40 0.50 0.60 0.70	2 hr		Alfalfa Barley			No effects at these doses	30
0.20	15 days	GC	Tomato	X		Threshold dose for initial symptom of tissue death, decrease or change in vitamin B_1, B_6, and nicotinic acid content	449
0.20	To maturation	EC/SD	Kidney bean		X	15% decrease in total yield	33
0.218	4.5 hr/day for 4 days	F-ZAP	Soybean	X		No visible damage	201
					X	20% decrease in yield	

TABLE 1-5. (continued)

Conc, ppm	Exposure time	Exposure condition[b]	Plant[c]	Effects on Foliar	Effects on Yield	Species Effect[c]	Reference
0.23	14 days	GH	Buckwheat	X		Caused necrotic leaf injury	356
			Lucerne	X		Caused necrotic leaf injury	
			Red clover	X		Caused necrotic leaf injury	
			Little stinging nettle			Increased peroxidase activity	
			Ryegrass			Increased S content in leaves	
0.25	1 hr		Broadbean			Slight swelling of stroma thylakoids of chloroplast, effect reversible	464
0.25	2 hr		Broadbean			Stroma thylakoids spread to granum thylakoids, effect reversible	
0.25 0.40 0.80 1.20	Once every wk (3 hr) to once in 5 wk (3 hr)	F/CC	Alfalfa Barley Durham wheat Spring wheat		X	No effect on yield	473
0.25	4 hr	EC/SD	Broccoli	X		1% leaf injury	432
			Tobacco, Bel B,	X		6% leaf injury	
0.25	8 hr	EC/SD	Tobacco			Chlorophyll a decreased more sharply than chlorophyll b	40
0.25	24 hr	GC	Broadbean			Stomata opened faster and wider in light condition; stomata opened longer in darkness	286
0.25	1 hr	EC	Begonia	X		Foliar injury	431
0.25 0.40 0.80 1.20	3 hr every 2 wk for growing season	F/CC	Alfalfa Barley Durham wheat Spring wheat		X	No effect on yield	473
0.25	Unknown	EC	Pea			50% decrease in net photosynthesis	62
0.25	Unknown	EC	Sunflower			10% decrease in photosynthetic rate	450
			Morning glory			20-30% decrease in photosynthetic rate	
			Corn			No effect	
			Sorghum			No effect	
0.29	15 days	GC	Lettuce	X		Foliar injury, 30% decrease in thiamine content	449

TABLE 1-5. (continued)

Conc, ppm	Exposure time	Exposure condition[b]	Plant[c]	Effects on Foliar	Effects on Yield	Species Effect[c]	Reference
0.30	5 hr/day	EC/SD	Barley			11% foliar injury; 38% decrease in dry weight shoot	292
	6 day/wk		Bean			1% foliar injury; 38% decrease in dry weight shoot	
	12 days		Sunflower			5% foliar injury; 41% decrease in dry weight shoot	
	26 days		Barley			21% foliar injury; 26% decrease in dry weight shoot 2% foliar injury; 15% decrease in dry weight shoot 16% foliar injury; 29% decrease in dry weight shoot	
0.35	1 hr	EC/SD	Alfalfa			80% decrease in apparent photosynthesis	472
0.35	21 days	EC/SD	Pea			Increase in glutamine content, decrease in glutamic acid and protein content inorganic S accumulated	205
				X	X	Necrosis and growth inhibition at 0.35 x 14 days	
0.38 1.15 1/90	14 days	0	Radish	X		Decrease injury at 0.38 and above, inhibited seed germination, formation of green leaflets of sprouts, and root growth	111
0.40	3 hr		Oats	X		Threshold for leaf injury	174
0.40 0.50 0.60	4 hr	EC/SD	Tomato			Increase accumulation total and soluble S content	29
0.40	6 hr	EC/SD	Apples			No effect	227
0.40	6 hr/2 wk (1-2 exposures)	EC/SD	Alfalfa Cotton Pecan Pepper			No difference found in total N, protein/total N ration, chlorophyll, all plants, all treatments	424
0.45	3 hr for 7 exposures for growing season	F/CC	Wheat		X	No accumulative effect on yield, no effect on average head length or number of grains/head	394
0.46	7 hr	EC	Buckwheat	X		Injury threshold	498
0.50-6.00	30 min	F/CC	Soybean	X	X	Very significant negative linear relationship between percent leaf area destroyed and percent crop loss; 0.66% yield loss for every 1% increase in foliar injury; asymtomatic plots increased yield by 6.02% over controls	102

TABLE 1-5. (continued)

Conc, ppm	Exposure time	Exposure condition[b]	Plant[c]	Effects on Foliar	Effects on Yield	Species Effect[c]	Reference
0.50	1.5 hr	EC/SD	Soybean	X	X	7% decrease in short fresh weight; trace foliar injury	172
0.50	1.5 hr		Oats		X	Inition of leaf injury	174
0.50	2 hr	EC	Begonia			No effect	2
			Petunia		X	30% decrease in flower number; 15% decrease in shoot weight	
			Coleus			No effect; 12% decrease in shoot weight	
			Snapdragon		X	11% decrease in number of flowers; no effect	
0.50	2 hr	EC	Grape			190% increase in stomatal resistance	371
0.50	4 hr	EC/SD	Alfalfa	X		19% leaf injury	431
			Broccoli	X		4% leaf injury	
			Radish	X		1% leaf injury	
			Tobacco	X		1% leaf injury	
			Tomato	X		1% leaf injury	
0.50	4 hr	EC/SD	Oats	X		Foliar injury occurred to all crops	441
			Radish				
			Soybean				
			Tobacco				
0.50	4 hr/day for 14 days	EC/SD	Oats		X	32% decrease top dry weight; 13% decrease in root dry weight; number of heads unchanged	175
0.50	5 hr/day		Barley			24% foliar injury; 42% decrease in dry weight shoot	292
	6 days/wk		Bean			7% foliar injury; 31% decrease in dry weight shoot	
	for 12 days		Sunflower			18% foliar injury; 44% decrease in dry weight shoot	
	26 days		Barley			36% foliar injury; 45% decrease in dry weight shoot	
			Bean			12% foliar injury; 34% decrease in dry weight shoot	
			Sunflower			26% foliar injury; 35% decrease in dry weights shoot	

TABLE 1-5. (continued)

Conc, ppm	Exposure time	Exposure condition[b]	Plant[c]	Effects on Foliar	Effects on Yield	Species Effect[c]	Reference
0.50 1.00 1.50	4 hr	GC/G	Red Clover			Increase in vitamin A, fat, protein content Significant change in plant nutritional components	196
0.50	5 hr/day 6 days/ week for 14 days	EC	Broadbean Sunflower Tobacco			Under drought conditions exposure caused wider opening of stomata, no effect on diffusive resistance	379
0.50	6 days	0	Pea			Increased glutamate dehydrogenase; increased peroxidase activity	465
0.56 0.77 0.92	4 hr	EC/SD	Cucumber			Accumulation of significant total and soluble sulfur	29
0.60	6 hr	EC/SD	Apples	X		7.3% increase in foliage injury; 5% increase leaf abscission	227
0.60	6 hr/day 5 days/week for 14 days	EC/SD	Soybean White bean	 X		No effects Bifacial necrotic lesion on mature leaves	183
0.70	8 hr/day for 3 days	EC/SD	Broadbean			20% decrease in photosynthesis after 2 hr fumigation; after 3-day fumigation, 1 hr to full recovery in light condition; no foliage injury	24
0.70	6/12, 24 hr/day 1-7 days	EC	Bean	X		Increase in total amino acids and ammonium, decrease in aspartic acid glutamic acid and protein synthesis, all before visible injury present	
0.75	3 hr	EC/SD	Alfalfa	X		No injury developed	192
0.80	2 hr		Alfalfa	X		Threshold dose for foliar necrosis; 25-50% decrease in net photosynthesis	30
0.80-2.00	4 hr, 20 min	F/ZAP	Soybean		X X X	4.5% decrease in yield at 1.4 ppm 11% decrease in yield at 1.7 ppm 15% decrease in yield at 2.0 ppm	310
0.80-2.00	4 hr, 20 min	F/ZAP	Soybean	X	 X	Epidermal and mesophyll cell death, the number of dead mesophyll cells highly correlated with increase in SO_2 Highest SO_2 concentration, significant decrease in seed yield	

TABLE 1-5. (continued)

Conc, ppm	Exposure time	Exposure condition[b]	Plant[c]	Effects on Foliar	Effects on Yield	Species Effect[c]	Reference
0.90	2 hr	EC/SD	Broadbean			26% decrease in net photosynthesis under saturated light conditions; 52% decrease in net photosynthesis under nonsaturated light conditions	39
1.00	2 hr		Barley	X		Threshold dose for foliar necrosis; 30-60 decrease in net photosynthesis	30
1.00	3 hr	GC	Poinsettia eight cultivars			No effect	177
1.00	3 hr	EC/SD	Alfalfa	X		Leaf necrosis at 315 ppm CO_2 was 2.8x that induced under 645 ppm CO_2	192
1.00	2 hr	EC	Begonia			No effect	
			Petunia		X	30% decrease in flower number; 19% decrease in shoot weight	
			Coleus		X	27% decrease in flower number; 19% decrease in shoot weight	
			Snapdragon		X	14% decrease in flower number; 16% decrease in shoot weight	
1.00	4 hr	EC/SD	Broccoli	X		38% leaf injury	
			Bromegrass	X		65% leaf injury	431
			Cabbage	X		70% leaf injury	
			Lima bean	X		25% leaf injury	
			Radish	X		46% leaf injury	
			Spinach	X		49% leaf injury	
			Tomato	X		33% leaf injury	
1.0	1 hr/2 days for 4 days	EC/SD	Geranium			Rapid closing of stomata in low-RH air after exposure; slow closing in high-RH conditions, stomata remained open	47
1.0	6 hr/day for 3 days	EC/SD	Strawberry			No effect on growth and development	
				X		Necrotic lesions, lower leaf surface	358
1.00	1.5 hr	EC/SD	Soybean	X	X	9% decrease in shoot fresh weights, 4% leaf	
	3 hr				X	injury 21%-29% decrease in shoot fresh weight	172
1.50	0.75-3 hr	EC/SD	Soybean	X	X	24-94% decrease in shoot fresh weight; 63-93% foliar injury	172
1.50	3 hr	EC/SD	Alfalfa	X		Leaf necrosis at 315 ppm CO_2 was 2.5x that induced under 645 ppm CO_2	192

TABLE 1-5. (continued)

Conc, ppm	Exposure time	Exposure condition[b]	Plant[c]	Effects on Foliar	Effects on Yield	Species Effect[c]	Reference
1.50	To maturation	EC/SD	Kidney bean		X X	20% decrease in root dry weight; 14% decrease in legume dry weight; 17% decrease in seed dry weight; 10-30% decrease in apparent photosynthesis. Increase in chlorophyll a and b content	33
2.00	2 hr	EC	Begonia		X	14% decrease flower number; 22% decrease in shoot weight	2
			Petunia		X	32% decrease in flower number; 24% decrease in shoot weight	
			Coleus		X	30% decrease in flower number; 20% decrease in shoot weight	
			Snapdragon		X	15% decrease in flower number; 15% decrease in shoot weight	
2.0	3 hr	GC	Poinsettia eight cultivars	X		0-18.3% foliar injury	177
2.5	6 hr	EC/SD	Apples	X	X	17% increase in foliar injury; 62% increase in lead abscission; 19% decrease in shoot growth	227
3.0	1 hr	GC	Poinsettia eight cultivars	X		0.13.8% foliar injury	177
	2 hr	GC		X		1.8-26.8% foliar injury	
	3 hr	GC		X		18.8-96.5% foliar injury	
4.00	2 hr	EC	Begonia		X	27% decrease in flower number; 33% decrease in shoot weight	2
			Petunia		X	42% decrease in flower number; 32% decrease in shoot weight	
			Coleus		X	30% decrease in flower number, 21% decrease in shoot weight	
			Snapdragon		X	20% decrease in flower number; 19% decrease in shoot weight	

[a]Table arranged by increasing SO_2 concentration as first-order and exposure time as second-order divisions. Doses within a single study that did not induce specifically different effects are listed along with the lowest SO_2 concentration that induced said effect.

[b]F = field or forest surveys
F/CC = Field, closed chambers
D/OT = Field, open-top chambers
F/ZAP = Field, zonal air pollution system
G = Greenhouse
GC = Growth chambers
EC = Exposure chambers
EC/SD = Exposure chamber, special design
O = Other

[c]See Appendix A for most scientific latin binomials of plants.

[d]X indicates study examinated foliar and/or yield effects. The X does not necessarily imply that an effect was found.

[e]Most prominent or significant effect reported.

the quality and quantity of the crop can be negatively affected by the concentration of pollutant multiplied by the length of the exposure period. Unfortunate variations in exposure regimens and response measurements make it difficult to compare the results of different studies.

The concept of dose-response can be demonstrated by a synthesis of the data presented in Tables 1-5, 1-6, 1-7. The following conclusions were formed by summarizing the dose-response data without designating specific associated exposure conditions:

- Yield of economically important agricultural spe cies can be significantly suppressed by SO_2 concentrations in the range of 0.05 to 0.06 ppm if the exposure period is sufficiently long (2 weeks). Both crop quality and quantity can be negatively affected.
- Fluctuating, long-term (seasonal, annual) SO_2 exposures averaging 0.05 ppm or less can cause economically and ecologically undesirable effects on productivity and stability of range and forest ecosystems.
- As SO_2 concentrations increase to 0.25 ppm, a variety of agricultural crops (such as alfalfa, timothy, range grasses, soybean, barley, wheat, cabbage, lettuce, spinach, tobacco, cucumber, eggplant, pea, and kidney bean) respond with necrotic foliar injury or suppressed yield. Approximately 70 percent of the cultivated agronomic crop species exposed to 0.25 ppm or less respond to the SO_2 exposure of 1 hour or longer with changes in stomatal aperture (leaf openings), foliar injury or yield effects. Foliar injury on vegetables and suppression of yield are directly related to economic values.
- Forest trees species (such as pine, spruce, fir, beech, alder and poplar, representing coniferous and deciduous forest ecosystems) respond to 0.25 ppm or less of SO_2. Approximately 90 percent of the

TABLE 1-6. DOSE-RESPONSE INFORMATION SUMMARIZED FROM LITERATURE PERTAINING TO FOREST TREE SPECIES AS RELATED TO FOLIAR, YIELD, AND SPECIFIC EFFECTS INDUCED BY INCREASING SO_2 DOSE

Conc, ppm	Exposure time	Exposure condition[b]	Plant[c]	Effects on Foliar	Effects on Yield	Species Effect[c]	Reference
0.001	10 yr avg.	F	Forest trees	X	X	No injury	~~118~~ 272
0.003-0.09	Annual avg	F	Scotch pine	X	X	Decreased photosynthesis leading to the death of tree	355
0.09-0.12	Annual avg						
0.004 0.35	Annual avg Annual avg (exposed 5 mo)	F	Eastern white pine			No significant difference in S content of foliage	369
0.006	Growing season	F	White birch			No effect on foliar S content	273
0.007-0.01	Annual avg	F	Fir forests		X	20 ± 5% growth increase	297
0.007-0.01	Annual avg	F	Fir forests	X	X	20 ± 5% growth decrease Premature defoliation	296
0.008	10 yr avg	F	Forest trees	X	X	Very little chronic foliar injury	~~118~~ 272
0.011	Growing season	F	White birch	X		Increased foliar S content; trace to light foliar injury	273
0.017	10 yr avg	F	Forest trees	X	X	Mostly chronic foliar injury; some acute injury	~~118~~ 272
0.015	Annual avg	F	Conifers		X	30% decrease in growth	454
0.019	Annual avg	F	Conifers		X	52% decrease in growth	454
0.023	Annual avg	F	Conifers		X	54% decrease in growth	454
0.025	6 hr	EC/SD	Eastern white pine	X		Threshold dose for needle damage; most sensitive clones only	193

TABLE 1-6. (continued)

Conc, ppm	Exposure time	Exposure condition[b]	Plant[c]	Effects on Foliar	Effects on Yield	Species Effect[c]	Reference
0.025-0.037	Annual avg	F	Fir	X	X	Death of groups of trees	297
0.026	Growing season	F	White birch	X		Moderate to severe foliar injury	273
0.026-0.037	Annual avg	F	Fir	X	X	Rapid death of groups of trees	296
0.035	5 mo	F	Eastern white pine	X		Foliar injury	369
0.038-0.057	Annual avg	F	Scotch pine		X	Species occurrence negatively correlated with SO_2 ambient conc.; foliar S content positively correlated with SO_2 ambient conc.; foliar S content positively correlated with SO_2 ambient conc.; by distance from source	133
0.045	Growing season	F	Jack pine	X		Reduced chlorophyll content, tissue death	273
0.045	10 yr avg	F	Forest trees	X	X	Acute and chronic foliar injury	~~118~~ 272
0.048	Growing season	F	White birch	X		Severe foliar injury; foliar S concentration 3x normal	273
0.05 0.15	6 hr	EC/SD	Eastern white pine	X		60% of tolerant clones foliar injury developed	193
0.05	49 days		Norway spruce	X		Foliar injury	491
0.05	10 wk	F/CC	Spruce		X	No effects	224
0.05 0.10 0.20	5 mo	F/CC	Beech	X		Increase in S concentration proportional to increase in SO_2 exposure conc.; terminal bud death	223
0.05	9 mo	F/CC	Spruce			Decreased foliar buffering capacity; in- peroxidase activity	222
0.05-0.10	9 mo	F/CC	Scotch pine			Decreased photosynthesis	222
0.05 0.10 0.20	9 mo	F/CC	Fir			Decreased pollen viability	222

TABLE 1-6. (continued)

Conc, ppm	Exposure time	Exposure condition[b]	Plant[c]	Effects on Foliar	Effects on Yield	Species Effect[c]	Reference
0.069	Annual avg	F	Conifer		X	70% decrease in growth	454
0.07	3 days	EC	Eastern white pine	X		Chlorotic spotting and death of needle tips	19
0.10 0.20	10 wk	F/CC	Spruce		X	Decreased CO_2 uptake; positive correlation between CO_2 uptake and cambium growth; increase in conc. induced annual ring width	223
0.10	76 days		Black alder			Increase phenoloxidase activity	491
0.15 0.30	9 wk		Poplar		X	Decreased leaf area index and foliar growth	209
0.18- 0.20	24 hr		Jack pine			Inhibited foliar lipid synthesis, inhibition reversible; increase in dose = increase in recovery time	286
0.20- 1.00	1 hr		Azalea Firethorn White ash White birch			No appreciable effect on foliar sorption of SO_2	367
0.20	12 hr/day for 7 wk	EC/SD	Hybrid poplar		X	Slightly decreased height; decreased relative growth rate, relative leaf growth rate, and relative leaf area expansion rate	208
0.20	110 days		English birch			No effect on phenyloxidase activity	491
0.025	2 hr	EC/SD	Eastern white pine Jack pine Red pine	X X X		6.5% foliar injury 4.5% foliar injury 0.5% foliar injury	34
0.25	2 hr	EC/SD	Loblolly pine Shortleaf pine Slash pine Virginia pine	X X X X		All equally sensitive; most sensitive period 8-10 wk of age or older	35
0.27	3 mo	EC	Pin oak White birch		X X	45% decrease in height growth 107% increase in height growth	368
0.35	3 hr	EC/SD	Trembling aspen	X		2% foliar injury	217
0.40 0.50	74 hr	EC	Yellow pine Eastern white pine			Chlorophyll content varied inversely with concentration	100

TABLE 1-6. (continued)

Conc, ppm	Exposure time	Exposure condition[b]	Plant[c]	Effects on Foliar	Effects on Yield	Species Effect[c]	Reference
0.45	6 hr	EC/SD	Eastern white pine	X		All tolerant clones developed foliar injury	193
0.45	9 hr/day for 8 wk	EC	Ponderosa pine	X		Severe needle tip chlorosis and necrosis	128
0.5	15 min		Red pine			Decreased primary needle chlorophyll content.	78
	30 min					Decrease dry weight of primary needles and cotyledons	
	60 min					Further increase of all of above effects	
	120 min						
0.50	2 hr	EC/SD	Eastern white pine	X		12% foliar injury	34
			Jack pine	X		11% foliar injury	
			Red pine	X		2% foliar injury	
0.50	3 hr	EC/SD	Trembling aspen	X		11% foliar injury	217
0.50	5 hr	GC	Austrian, Ponderosa Scotch pine, Balsam, Fraser, White fir Blue, White spruce Douglas fir	X		No injury	399
0.50	30 day	GC	Chinese elm	X		Severe chlorosis and necrosis	416
			Gingko	X		Moderate marginal chlorosis	
			Norway maple	X		Moderate marginal chlorosis	
			Pin oak	X		Slight overall chlorosis	
0.65	3 hr	EC/SD	Trembling aspen	X		23% foliar injury	217
1.00	4 hr	GC	Austrian, Ponderosa, Scotch pine, Balsam, Fraser, White fir, White spruce, Douglas fir	X		Less than 4% foliar injury all species	399
1.00	8 hr	EC	American elm			Inhibition of stomatal closing	335
1.07-6.41	30 min to 6 hr	O	Scotch pine *P. pinea* *P. nigra*	X		Visible injury was proportional to foliage S content	65
1.83	50 min	O	Pine Spruce	X		SO_2 absorbed by exposed foliage in winter-time; S stored in new shoots	298

TABLE 1-6. (continued)

Conc, ppm	Exposure time	Exposure condition[b]	Plant[c]	Effects on Foliar	Effects on Yield	Species Effect[c]	Reference
2.00	2 hr	CG	Austrian, Ponderosa Scotch pine, Balsam, Fraser, White fir, Blue, white spruce, Douglas fir	X		No foliar injury on Douglas fir, firs, spruce Pine foliar injury threshold, necrotic tips	399
2.00	6 hr	GC	American elm	X	X	Induce severe foliar injury; defoliation in older leaves; significant reduced expansion of new leaves; number of emerging leaves and root dry weight reduced	76
2.00	6 hr	GC	American elm			No significant reduction in lipid content; significant decrease in new leaf protein content; significant decrease in leaf, stem, root carbohydrate content	79
2.00	6 hr	GC	Chinese elm	X		100% leaf necrosis	416
2.00	6.5 hr	O	Ginkgo			Water-stressed plant increased uptake of SO_2	335
2.00	12 hr	O	American elm			Induced stomatal closing; S content increased in plants fumigated in light	416
3.00	6 hr	GC	Ginkgo Norway maple	X		50% leaf necrosis	416

[a] Table arranged by increasing SO_2 concentration as first-order and exposure time as second-order divisions. Doses within a single study that did not induce specifically different effects are listed along with the lowest SO_2 concentration that induced said effect.

[b] F = field or forest surveys
F/CC = field, closed chambers
D/OT = field, open-top chambers
G/ZAP = field, zonal air pollution system
G = greenhouse
GC = growth chambers
EC = exposure chambers
EC/SD = exposure chamber, special design
O = other

[c] See Appendix A for most scientific latin binomials of plants

species tested in this range of SO_2 concentrations (with 2 hour exposures) responded with physiological modifications, suppressed photosynthesis, foliar injury, death of buds, or suppressed foliar or woody growth.

- Non-woody components of native ecosystems such as lichens and grasses also respond to SO_2 concentrations below 0.025 ppm. Responses include suppressed growth, death, and reduced diversity in lichen populations and suppressed photosynthesis and growth of leaves, tillers, and stubble of grasses.
- At SO_2 concentrations between 0.25 and 0.50 ppm (1-8 hours exposure), less than 50 percent of the agronomic species tested responded negatively to the sulfur dioxide treatments. At SO_2 concentrations less than 0.25 ppm but for multiple days, 70 to 90 percent of the species responded negatively.

A comparison of the species response to the latter treatments at the concentrations ranging between 0.25 to 0.50 ppm with the response to concentrations below 0.25 ppm might be interpreted as suggesting that plant response is not positively correlated with dose. This is not the case. Exposure durations used in the studies at the higher SO_2 concentrations generally ranged from 1-8 hours while multiple-day exposures were frequently used at the lower concentrations of SO_2. All agronomic species responded to a concentration of 0.50 ppm at exposure durations ranging from 1.5 to 5 hours. A variety of responses occurred, including physiological modifications, foliar injury, and suppressed growth. These trends suggest that as the SO_2 concentration is increased (a) a shorter exposure duration is sufficient to elicit a plant response equal to or greater than that which occurred at a lower concentration; (b) responses become more severe; (c) plants tolerant at lower concentrations become sensitive.

TABLE 1-7. DOSE-RESPONSE INFORMATION SUMMARIZED FROM LITERATURE PERTAINING TO NATIVE PLANTS AS RELATED TO FOLIAR, YIELD, AND SPECIFIC EFFECTS INDUCED BY INCREASING SO_2 DOSE

Conc, ppm	Exposure time	Exposure condition[b]	Plant[c]	Effects on Foliar	Effects on Yield	Species Effect[c]	Reference
0.006 0.018	6 mo	F	Lichens		X	Loss of chlorophyll, decreased growth	255
0.01- 0.02	Annual avg	F	Lichens			Elimination of many lichen species	256a
0.015	Annual avg	F	Bryophytes Lichens			Decreased lichen diversity	397
0.017	6 mo	F	Bryophytes			Elimination of species	147
0.02	29 days 22 days in 2 consecutive growing seasons	GC	Ryegrass		X	No effect on net photosynthesis, dark respiration, transpiration coefficients, number of tillers and yield	87
0.14	29 days 22 days in 2 consecutive growing seasons	GC	Ryegrass	X	X	As above effects except visible foliar injury and reduction of specific leaf area	
0.02	85 days	GC	Ryegrass			Increased organic S content	89
0.02	Growing season	GC	Ryegrass			Increased organic and inorganic S content	86
0.03 0.04 0.08 0.15	10 wk	GC	Ryegrass		X X X X	Alleviated S deficiency Alleviated S deficiency Alleviated S deficiency Reduction in yield without symptoms	277
0.03 0.04	6 mo	F	Lichens	X		Tissue death	256a
0.04 0.15	51 days	GC	Ryegrass			Decreased concentration glycine and serine; inhibited photorespiration pathway	234
0.05	Growing season	GC	Ryegrass	X		Alleviated S deficiency symptoms	86

TABLE 1-7. (continued)

Conc, ppm	Exposure time	Exposure condition[b]	Plant[c]	Effects on Foliar	Effects on Yield	Species Effect[c]	Reference
0.06	Growing season	GC	Ryegrass		X	Increase in photosynthesis, respiration and chlorophyll content; light increase in productivity	135
0.067	26 wk	EC	Ryegrass		X	Increase in dry weight of leaves, number of tillers, dry weight of stubble and leaf area; 51% decrease in yield	24
0.073	26 wk	EC	Ryegrass			50% decrease in shoot dry weight 24% decrease in chlorophyll a content; 26% decrease in chlorophyll b content	25
0.074	18 hr	O	Spiderwort			Increase in chromosome aberration rate of germinating pollen	282
0.08	13 hr/day for 28 days	EC	Foxtail grass	X		Foliar injury as caused by heavy metals was increased by SO_2 exposure	236
0.09 (peak)	115 days	F/CC	Ryegrass	X		Decrease in weight; accelerated leaf senesence	42
0.11	4 wk	O	Cocksfoot		X	30% decrease in leaf area; 45% decrease in dry weight; decrease in number tillers; decrease in number of green leaves; decrease root/shoot ratios	12
0.11	4 wk	O	Ryegrass		X	20% decrease in leaf area; 40% decrease in dry weight; decreased root/shoot ratio	14
0.11	103 5 hr/wk for 20 wk		Grass		X	Significant decrease in leaf area and all dry weight fractions; decrease in number of leaves and tillers	13
0.12	9 wk	EC	Ryegrass			Decrease in dry weight of leaves, number of tillers, dry weights of stubble and leaf area; 46% decrease in yield	24
0.13 0.25 0.50 1.00	6 wk	EC/SD	Ryegrass	X	X	Foliar necrotic lesions and decrease in net primary productivity at 0.13 ppm and above	135
0.13	6 wk	EC/SD	Ryegrass	X	X	Decreased productivity	135

TABLE 1-7. (continued)

Conc, ppm	Exposure time	Exposure condition[b]	Plant[c]	Effects on Foliar	Effects on Yield	Species Effect[c]	Reference
0.15	6 wk		Duckweed		X	Decrease in starch content and size of fronds	131
0.15	51 days		Duckweed		X	Decrease in starch content and growth; decrease in surface area dry weight	131
0.15	Growing season	GC	Ryegrass			Alleviated S deficiency symptoms; increase in S content of foliage, free amino acid content, and N/S ratio	85
0.20	2 hr	GC	Kentucky bluegrass	X		Visible foliar injury	320
0.20 (peak)	55 days	F/CC	Ryegrass	X	X	Decrease in weight; accelerated leaf sensence	42
0.25	5 wk	EC/SD	Ryegrass		X	No effect on number of tillers; 17% decrease in yield	188
0.27	14 days	EC/SD	Ryegrass			Increase in free amino acid content	11
0.27	8 wk	EC/SD	Ryegrass		X	38% decrease in green weight; 30% decrease in total dry weight; no reduction in number of tillers; 2x senesence	188
0.38 (peak)	6-43 wk	EC/SD	Ryegrass		X	36% decrease in total dry weight	93
0.50-11.00	2 hr	F/CC	87 Desert species	X		Most plants required more than 2.00 ppm SO_2 to produce foliar injury	182
0.71	1 hr 2 hr 5 hr	EC/SD	Lily			Inhibited pollen tube elongation at all exposure durations	294
2.00-0.00	8 hr	EC/SD	Diplacus meteromeles			Increase in SO_2 dose induced a progressive decrease in photosynthesis and transpiration	478
3.50	1 hr	O	Acacia	X		Foliar injury	337

TABLE 1-7. (continued)

[a] Table arranged by increasing SO_2 concentration as first-order and exposure time as second-order divisions. Doses within a single study that did not induce specifically different effects are listed along with the lowest SO_2 concentration that induced said effect.

[b] F = field or forest surveys
F/CC = field, closed chambers
D/OT = field, open-top chambers
G/ZAP = field, zonal air pollution system
G = greenhouse
GC = growth chambers
EC = exposure chambers
EC/SD = exposure chamber, special design
O = other

[c] See Appendix A for most scientific latin binomials of plants.

The lack of short- or long-term monitoring data makes it difficult to assess the results of dose-response studies in the field. When data are not available to determine whether short-term spike concentrations occurred, then only long-term averages have been used to define the dose. Obvious differences between forests in areas with high SO_2 concentrations have been observed. There is usually no exact dose information for short-term influences; therefore, in most field studies, only long-term averages are used to define the dose.

As the dose of SO_2 increases, plants develop more predictable and more obvious visible symptoms. Foliar symptoms advance from chlorosis or other types of pigmentation changes to actual necrotic areas, and the extent of necrosis increases with exposure. Studies of the effects of SO_2 on growth and yield have demonstrated a reduction in the dry weight of foliage, shoots, roots, and seeds, as well as a reduction in the number of seeds. At still higher doses, reductions in growth and yield increase. Extensive mortality has been noted in forests continuously exposed to SO_2 for many years.

The amount of sulfur accumulated from the atmosphere by leaf tissues is influenced by the amount of sulfur in soil relative to the sulfur requirement of the plant. After low-dose exposure to SO_2, plants grown in sulfur-deficient soils have exhibited increased productivity.

Sulfur dioxide and particulate sulfate are the main forms of sulfur in the atmosphere, and a plant may be exposed to these pollutants in several different ways. Dry deposition of particulate matter and wet deposition of gases and particles bring sulfur compounds into contact with plant surfaces and soil substrates. The effects of such exposures are more difficult to assess than those associated with the entry of SO_2 through plant stomata. Plant response to dynamic physical factors such as light, leaf surface moisture, relative humidity, and soil moisture may influence stomatal opening and closing,

and hence, play a major role in determining sensitivities of species and cultivars or the time of sensitivity of each on a seasonal basis. Dose-response relationships are significantly conditioned by environmental conditions before, during, and following exposure to SO_2.

Sulfur accumulation in plants has been suggested as a tool for determining the levels of sulfur in the atmosphere of a given area over time. Such data, however, cannot accurately define the dose received by a plant.

Very few studies have been conducted to determine the sensitivity of microorganisms to SO_2 or to explore the interactions of SO_2 with plant pathogens such as fungi, nematodes, or bacteria. Both inhibition and enhancement of disease processes have been reported, but more data are needed to provide reliable information on trends.

In ambient atmospheres, SO_2 and other pollutants usually exist as diverse mixtures in which a multiplicity of chemical combinations can take place. Therefore, with the possible exception of a point source in which vegetation is exposed to a high dose of SO_2, the direct and indirect influences of other air pollutants in combination with SO_2 must be considered. Major phytotoxic air pollutants that have been studied in combination with SO_2 include ozone, oxides of nitrogen, and hydrogen fluoride. The interactions of SO_2 and O_3 have been most extensively investigated because of the incursions of ozone and other oxidant precursors into many rural areas, as well as their presence in urban areas. Many studies have demonstrated more than additive effects in symptom expressions, but relatively few studies have attempted to evaluate the impacts on growth and yield. Additionally, pollutant combinations with SO_2 have caused less than additive and/or additive effects depending upon doses applied. The influence of various physical and biological factors of the environment increase in complexity as the pollutants are combined together during exposures.

A few studies have examined effects of combinations of particulate matter and SO_2 or particulate matter and other pollutants: (1) increase foliar uptake of SO_2; (2) increase foliar injury of vegetation by heavy metals; and (3) reduce growth and yield. Because of the complex nature of particulate pollutants, conventional methods for assessing pollutant injury to vegetation, such as dose-response relationships, are poorly developed. Studies have generally reported vegetation responses relative to a given source and the physical size or chemical composition of the particles. For the most part, studies have not focused on effects associated with specific ambient concentrations. Coarse particles such as dust directly deposited on the leaf surfaces result in reduced gas exchange, increased leaf surface temperature, reduced photosynthesis, chlorosis, reduced growth, and leaf necrosis. Heavy metals deposited either on leaf surfaces or in the soil (and subsequently taken up by the plant) can result in the accumulation of toxic concentrations of the metals within the tissue.

Particulate matter is heterogeneous in size and composition, ranging in mean diameter from <0.005 μm (molecular clusters) to >100 μm (visible dust particles). Particles occur in both solid and liquid phases and vary in chemical composition from a single chemical species (e.g., H_2SO_4) to complex combinations of chemical species. They are produced directly from stationary and mobile sources and are also formed secondarily in the atmosphere through chemical reactions.

While coarse particles (>2.0 μm in mean diameter) settle rapidly, fine particles (<2.0 μm in mean diameter) have prolonged atmospheric residence times and are not strongly influenced by gravity. Because of the complex nature of particulate matter, dose-response studies are very difficult to conduct and data, therefore, are not available for making generalized statements.

Vegetation within terrestrial ecosystems is sensitive to direct SO_2 toxicity, as evidenced by changes in physiology, growth, development, survival, fecundity, and community composition. Responses of individual organisms reflect both direct or indirect effects. Habitat modification by SO_2 results in indirect effects occurring. Nutrient cycling appears to be a sensitive indicator of subtle, yet important, environmental modification. The removal of certain lichen species can reduce nitrogen fixation in forest ecosystems.

At the community level, chronic exposure to SO_2 may result in a shift in the species composition due to the elimination of individuals or populations sensitive to the pollutant. The tendency for SO_2 derivatives to accumulate in the soil may have consequences for the microbiota inhabiting the upper soil horizons. The gradual accumulation of pollutant derivatives may cause a change in soil chemistry and influence nutrient cycling and ecosystem productivity.

Particulate emissions have their greatest impact on terrestrial ecosystems near large emission sources, and particulate matter in itself constitutes a problem only in those areas where deposition rates are high. Most of the material deposited by wet and dry deposition on foliar surfaces in vegetated areas is transferred to the soil. Foliage may serve as a transitional site of accumulation if previously deposited dry material becomes highly concentrated during precipitation. Ecological modifications may occur if the particles contain toxic elements, even though deposition rates are moderate. Solubility of particle constituents is critical, since water-insoluble elements are not mobile within an ecosystem. Soils are long-term sites for the retention of many constituents found in particulate matter. Accumulation in the soil-litter layer influences ecological processes such as decomposition, mineralization, nutrient cycling, and primary production.

1.4.2 Acidic Precipitation

Acidic precipitation is a major environmental concern in many regions of the United States, Canada, northern Europe, and Japan. It has caused measurable damage to aquatic ecosystems in Scandinavia, eastern Canada, and the northeastern United States. Acidic precipitation has, by acidifying lakes, induced the extinction of fish, caused the breakdown of nutritional food webs, and reduced life in lakes to a few acid-tolerant species. Acidic precipitation, in addition, has damaged national monuments and buildings made of stone. It also has the potential for impoverishing sensitive soils, degrading natural terrestrial ecosystems and for damaging forest ecosystems over the long-term (several decades).

In an atmosphere relatively free of natural or man-made emissions of sulfur and nitrogen oxides, precipitation would be expected to have a pH of 5.6 due to the presence of carbonic acid formed when atmospheric carbon dioxide dissolves in water vapor. The precipitation has become acidic chiefly due to the large amounts of sulfur and nitrogen oxides being emitted from the combustion of fossil fuels (particularly coal and oil). In addition, substances present in other gases, aerosols, and particulate matter from natural and man-made sources also contribute to the acidity of precipitation.

Precipitation acts as a scavenger, bringing to earth substances present in the atmosphere. The chemical composition of rain, therefore, depends on the substances present in the atmosphere. On a global scale natural emissions far exceed man's contributions; however, man-made emissions are localized in specific geographic areas, where they may be concentrated in the atmosphere or transported meteorologically to other areas downwind.

Sulfur and nitrogen oxides are transformed in the atmosphere to sulfates and nitrates. Sulfates and nitrates upon hydrolysis in the the atmosphere

contribute hydrogen ions (H^+). If hydrogen ions are present in significant quantities, precipitation becomes acidic. The acidity of precipitation is a reflection of the balance between the major cations and anions in precipitation, however, when determining the pH of precipitation all cations and anions should be measured. Currently the acidity of precipitation in the northeastern United States is between 4.0 and 5.0.

The ratio of sulfuric to nitric acids in precipitation varies from time to time and place to place. In much of the eastern United States, the average annual ratio of sulfuric to nitric acids is currently 2:1; however, nitric acid is apparently becoming progressively more important as a contributor of hydrogen ions. Preliminary estimates suggest that two-thirds of the sulfur emitted into the atmosphere of eastern North America is probably deposited there with the remainder leaving the atmosphere of the region and moving primarily to the east.

Tall stacks (some as high as 1200 ft.) from power plants have decreased local pollution problems but may have increased the widespread wet and dry deposition of sulfur and nitrogen oxides by permitting them to be carried long distances by air streams. Analysis of air-mass movements and chemical transformations in the atmosphere indicates that acidic precipitation in one state or region of the United States or Canada results in large part from emissions which enter the atmosphere in other states or regions, often many hundreds of miles from the original source.

Acidic precipitation is only one special feature of the general phenomenon of atmospheric deposition. In addition to precipitation (wet deposition), dry deposition also occurs. The major chemical substances that are transferred into ecosystems via acidic precipitation (wet deposition) are also transferred into ecosystems by dry deposition when it is not raining or snowing. It is,

therefore, impossible to chemically distinguish the biological effects of acidic precipitations from those of dry deposition.

The increased deposition of acidic substances into aquatic ecosystems has, as of 1979, caused hundreds of lakes in the Adirondack Mountain region of New York State, certain lakes in northern Minnesota, and many hundreds of lakes in various parts of southern Ontario and Quebec to show acid stress. Reduction or extinction of fish and other plant and animal populations has occurred. Lakes and streams in other regions of the United States and Canada are also potentially vulnerable to stress by acidic precipitation. Damage or injury to aquatic or terrestrial organisms is most likely to occur when a particularly sensitive life form or life stage (one with a narrow range of tolerance), developing in poorly buffered waters or soils, coincides in time and/or space with major episodic injections of acidic precipitation or other injurious substance.

The disappearance of fish from lakes and streams usually follows two general patterns. One pattern results when sudden short-term shifts in pH occur, the other results from the long-term decrease in the pH of the water. A major episodic injection of acids and other soluble substances occurs when these substances present in polluted snow are released in the meltwater during warm periods in winter or early spring. The release of pollutants can cause major and rapid short-term changes in acidity and chemical properties of stream and lake waters. Fish kills are a dramatic consequence of such episodic injections into aquatic ecosystems.

Equally dramatic long-term changes in aquatic ecosystems also occur from the wet and dry deposition of acidic substances because the chemical composition of precipitation and dry deposition determine in part the chemical composition of lake, stream, and ground waters. The terrestrial watershed

also plays a significant role as the chemical composition of precipitation is modified by chemical and biological weathering and exchange processes which take place as precipitation washes over vegetation, percolates through the soil and interacts with the underlying bedrock of the drainage basin in which the precipitation occurs. The situation is analogous to a gigantic, regional scale titration with the lakes and streams acting as receiving vessels for acidic additions from the atmosphere. The titration end point of each lake is predetermined by its hydrology and the capacity of the soils in the drainage basin to assimilate the incoming acid. If the soils and drainage basin can no longer assimilate the incoming acids, the lake and stream waters are changed from conditions that are favorable for fish and other aquatic organisms to conditions that inhibit reproduction and/or recruitment of populations of fish and other aquatic organisms, some of which are food for fish.

Prolonged acidity interferes with reproduction and spawning so that changes in the structure of a population occur over time. These changes include a decrease in population density and a shift in size and age of the population to one consisting primarily of larger and older fish. The process is insidious, and effects on yield are often delayed and not recognizable until the population is close to extinction. This is particularly true for late maturing species with long lives. Large increases in the mortality rate are not necessary to bring about population extinction. Even relatively small increases (5 to 50 percent) in mortality of fish eggs and fry can decrease yield and bring about population extinction. Many populations of freshwater fish become extinct at a pH below 5.0 while the reproduction of many species of aquatic organisms is inhibited at pH's between 6.0 and 5.0. Increasing acidity of freshwater habitats causes shifts in species, populations and communities of most aquatic organisms to occur. Virtually all trophic (food) levels are affected.

Interference with normal reproductive processes in fish occurs, not only because of acidity of the water, but also due to increased concentrations of certain metallic cations, notably aluminum, which become mobilized in acidified lakes and streams. The metallic cations may originate from the lake or stream watershed or be introduced in wet and dry deposition. Among the inorganic substances are elements such as manganese, zinc, copper, iron, boron, flourine, bromine, aluminum, lead, iodine, nickel, cadmium, vanadium, mercury, and arsenic. The underlined elements in the list above are essential micronutrients that are required by plants in small amounts. However, at concentrations above the amount required, these same elements, can be toxic to plants and animals. The non-esssential elements can also be toxic to plants or animals when present in large amounts or when their mobility and solubility is increased due to soil acidity. Also the deposition of these metallic substances in precipitation can affect the foliage and roots of plants and injure microorganisms or animals that may ingest the plants. In addition, these substances can harm animals (including man) that may drink water containing these elements as well as aquatic animals (especially fish) that live in the water.

An indirect effect of acidification, which is potentially of concern to human health is the possible contamination of edible fish and of human water supplies. Studies in the United States, Canada and Sweden have revealed the presence of high mercury concentrations in fish from acidified regions. Lead and copper have been found in household plumbing systems with acidified water supplies. Persons drinking the water could suffer from lead or copper poisoning.

The drainage basins, soils, and wet and dry deposition of acidic substances link aquatic and terrestrial ecosystems. Effects of acidic precipitation on soils may indirectly influence plant productivity by altering the supply and availability of soil nutrients. Increased additions of hydrogen ions may

result in a gradual acidification of the soil. Soil acidification increases leaching of plant nutrients (such as calcium, magnesium, potassium, iron, and manganese) and increases the rate of weathering of most minerals. It also makes phosphorous less available to plants. Acidification also decreases the rate of many soil microbiological processes such as nitrogen fixation and the breakdown of organic matter. Various processes important in nutrient cycling and critical in most ecosystems are known to be inhibited by increasing the soil acidity. Included among these processes are: nitrogen fixation by Rhizobium bacteria on legumes and by the free-living Azotobacter; mineralization of nitrogen from forest litter; nitrification of ammonium compounds; and overall decay rates of forest floor litter.

Acidic precipitation increases the solubility and mobility of many cations in the soil, thus increasing the concentration of trace metal cations such as aluminum, manganese, and zinc to toxic concentrations in soil solutions. Solubility and mobility of other heavy metals is also enhanced. These toxic or nutrient ions leached from the soil are transferred into surface and ground waters from which they may enter lakes or streams and drinking water. Plant nutrients leached in the same way are lost to vegetation.

Large quantities of hydrogen ions are added to soils as acidic precipitation and, also, as a result of soil amendment and fertilization practices. Acidification by these processes can be readily controlled through normal soil management practices such as liming. Large areas of the United States, however, are not cultivated and have soils that are poorly buffered. These soils are susceptible to further acidification. Many of these soils occur in forest and wilderness areas. Some of these soils could benefit from significant quantities of plant nutrients, including nitrogen and sulfur being added to soil in precipitation and by dry deposition. In some ecosystems these additions may

be important in the overall nutrient budgets; however, the additions are subject to the vagaries of wind and weather. Some substances, such as ammonium and sulfate ions, are acidifying in their effects when taken up by plants from the soil.

Various specific biological effects of simulated acidic rain have been demonstrated in controlled field and laboratory experiments. But reliable evidence of economic damage to agricultural crops, forests, and other natural vegetation and to biological processes in soil by naturally occurring acidic precipitation have been very rarely reported.

Dry deposition of toxic gases, aerosols, and particulate matter causes substantial damage to crops in certain regions of the United States. The possible effects of acid deposition must be considered together with the serious economic crop damage caused by sulfur dioxide, ozone, oxides of nitrogen, fluoride, and hydrogen chloride.

Direct and indirect injury to crops and forests has been reported based on laboratory, greenhouse, and field experiments in which simulated acidic rains were used, the following biological effects were observed:

- Formation of necrotic lesions and spots on foliage.
- Accelerated erosion of waxes on leaf surfaces.
- Loss of nutrients due to leaching from exposed plant surfaces.
- Inhibition of nodulation of legumes leading to decreased nitrogen fixation by symbiotic bacteria.
- Reduced yield of marketable crops.
- Reduced rates of leaf litter decomposition leading to decreased mineralization of organically-bound nutrients.

Soils differ by orders of magnitude in their susceptibility to acidification. Acidic additions are unlikely to damage calcareous (calcium carbonate) soils, but metal deposition may. Soils with very low cation exchange capacities are very susceptible to increased acidification. In addition, the consequences of acidic additions to soils vary greatly. Such variations depend upon the rates and recent history of the acidic additions, the character of the vegetation, natural rates of acid formation in the soil, and the physical-chemical properties of the parent material of the soil.

Acidic precipitation plays an important role in the deterioration of stone buildings, monuments, and a variety of materials. Stone has traditionally been considered one of the most durable building materials used by man. What is forgotten is that the structures built with stone which were not durable have long since disappeared.

High acidity promotes corrosion of metals because hydrogen ions act as a sink for the electrons liberated during the critical corrosion process. Acidic precipitation forms a layer of moisture on the surface of material and by adding hydrogen and sulfate ions increases corrosion. Atmospheric sulfur compounds react with the carbonates in limestone and dolomites, calcareous sandstone, and mortars to form calcium sulfate. Blistering, scaling, and loss of surface cohesion occurs, which in turn induces similar effects in neighboring materials not in themselves subject to direct attack. Acid rain may also leach ions from stonework just as acidic runoff and ground water leaches ions from soil bedrock.

1.4.3 Effects on Visibility and Climate

Pollutants released into the atmosphere alter the environment in several ways, such as by reducing visibility and affecting climate. Visibility refers to various characteristics of the optical environment such as clarity and

trueness of color, as well as the distance over which one can see distant objects. Climate is defined as the long-term manifestation of weather at a given location over a specified period, usually several decades.

Meteorologically, visibility refers to the greatest distance at which a black object can be distinguished against the horizon sky. Visibility and visual range are reduced by atmospheric particles which scatter and absorb light. Gases play a relatively minor role in visibility reduction. Data on scattering and absorption are used to determine the extinction coefficient, a measure of the visible range.

Light scattering by particles, especially fine particles, is the most important cause of lowered visibility. Particles with diameters similar to the wave-length of light (0.1 to 1.0 μm) are the most efficient light scatterers per unit mass. Sulfates generally fall within this size range. An aerosol composed of particles of 0.5 μm diameter scatters about a billion times more light than does the same mass of air.

Sulfates reduce visibility on both local and regional scales. Considerable evidence from chemical-mass balance methods indicates that sulfates, which constitute approximately 50 percent of the fine aerosol mass in the atmosphere, cause more visibility degradation than do other chemical species. Ambient sulfate concentrations and sulfur oxides emission trends from the early and middle 1960s to the early 1970s closely parallel visibility trends. A 30-year record of spatial and temporal trends of coal use suggests that increases in haziness have been associated with increases in sulfur oxides emissions since the 1950s.

Visibility or visual air quality can be measured by determining total extinction, which is the sum of light scattering, and absorption. In many cases, absorption is assumed to be small and total extinction is estimated by

light scattering. Visibility measurement approaches include: the observe method, in which a black object is viewed against the horizon; the contrast telephotometric method, in which the brightness of a black object is compared with the brightness of the horizon; and the long-path extinction method, in which the decrease in intensity of a beam of light is measured as a function of range. Indirect methods to measure visibility include scattering and absorption measurements. The nephelometric method measures the scattering component of extinction. Absorption can be measured in several ways, such as by determining the difference between extinction and scattering; however, no single method has been proven totally effective.

Pollutants released into the atmosphere may lead to slow and subtle changes in atmospheric composition and, possibly, climate. For example, a fraction of the solar radiation may be absorbed by aerosols, further reducing the amount of radiation reaching the earth's surface and, at the same time, heating the aerosol layer itself. On a hazy day, direct solar radiation is reduced to about one-half of that on a clear day. Most of this light is diffused as skylight, while there is an overall loss of up to about 10 to 20 percent of the radiation reaching the surface. Changes in certain measures of daylight in the Eastern U.S. are consistent with observed patterns of haziness and can be attributed to man-made fine particles, including sulfates and nitrates. A possible link between haziness, decreased solar radiation, and decreased surface temperature in the East Central U.S. has been pointed out recently.

Atmospheric aerosols, primarily those with strong water affinity, influence cloud formation. Essentially all water vapor condensation occurs via nucleation, i.e., by deposition on cloud condensation nuclei or ice nuclei. Both types of nuclei are aerosol particles emitted from natural or manmade sources. Their

quantity and nucleation properties have an effect on cloudiness, which in turn influences the amount of solar energy reaching the earth's surface. In urban areas, increases in cloudiness and the quantity of precipitation are well established. The incorporation of particles into cloud and fog droplets can change the quality and quantity of precipitation by altering its chemical composition. Effects on visibility and climate of the type summarized above may exert significant impacts on aesthetic and economic values.

1.4.4 Materials Damage and Soiling Effects

Atmospheric sulfur oxides and particulate matter damage materials through deterioration and soiling. Physical and economic damage functions have been developed to estimate materials damage from sulfur oxides and particulate matter. Their accuracy is hampered by problems in identifying dose-response relationships for specific damage from specific pollutants because of many variables influencing exposure in the environment. Damage functions indicate that reductions in SO_2 and particulate matter will decrease economic damage. In most cases, the cost of replacing a product that has suffered premature damage is far greater than the cost of using protective measures or alternative materials resistant to damage.

A principal deleterious effect of sulfur oxides is to accelerate the corrosion of metals to form metal sulfates. Soluble sulfates in rust can stimulate further corrosion because of their hygroscopicity and electrical conductivity, while insoluble sulfates in rust provide corrosion protective properties.

Laboratory studies show that corrosion is most severe under conditions of high SO_2 concentration and high humidity. Field studies show that corrosion rates are related to the amount of sulfur compounds on exposed surfaces of susceptible metals.

Many nonmetallic materials are also damaged by sulfur oxides and/or soiled by particles. These materials include paints and other protective coatings, fabrics, building materials, electrical components, paper, leather, plastics, and works of art.

The chemical action of SO_2 erodes paint layers; light and ozone cause degradation of the polymer. Paint films permeable to water may be penetrated by sulfur dioxide and aerosols containing sulfuric acid. SO_2 can sensitize dried paint film, permitting water to be absorbed during the weathering cycle, especially at high humidity.

Cotton, rayon, and nylon fabrics are damaged by acids derived from SO_2 ~~Polyester, acrylic, and polypropylene fibers are damaged by ammonium sulfate particles by acid hydrolysis.~~

Certain types of building stone adsorb SO_2 and undergo chemical changes that weaken the material and lead to erosion. Concrete reacts with SO_2 and suffers erosion and spalling if not protected by paint. Concrete is also subject to chemical damage by sodium sulfate. The action of SO_2 has been implicated in the deterioration of ancient buildings. Sulfate damage has been found in medieval stained glass windows, bronze sculptures, marble and stone statues, and fresco paintings on lime plaster.

Sulfur dioxide and particles have deleterious effects on electrical contacts. To reduce damage, contacts are electroplated with corrosion-resistant metals.

Sulfur dioxide is readily absorbed by paper and oxidized to sulfuric acid by metallic impurities; the paper then hydrolyzes and loses strength. Leather also has a high capacity for absorption of SO_2; the material is weakened by hydrolysis of the proteins that make up the collagens in leather. The weathering of plastics has been attributed in part to the joint action of SO_2 and light.

Airborne particles contribute to corrosion by producing acid electrolytes; by functioning as nuclei to promote the condensation of water containing SO_2 or sulfates; and by forming a solid structure to retain active pollutants such as chlorides, organic matter, and sulfates. Deposition of dust and soot on building materials reduces the esthetic appeal of structures and can also result in erosion and direct chemical attack. Fabrics soiled by airborne particles require more frequent cleaning, which leads to increased costs and reduces their life.

Exterior paints are soiled by particles of soot, tarry acids, and various other constituents such as sulfates of iron, copper, calcium, and zinc. Staining and pitting of auto finishes have been traced to iron particles from nearby industrial operations and to alkali mortar dust from buildings being demolished. It has been suggested that particles promote the chemical deterioration of paint by acting as wicks to transfer SO_2 to the underlying surface. Acid smut, emitted mainly from large industrial operations such as oil-fired boilers, has been shown to cause significant damage to auto finishes, paints, and fabrics in the area near the source. The effect is localized because the emitted particles are very large and tend to be deposited quickly. Damage may be severe because the smut, which may contain as much as 30 percent sulfuric acid, is highly corrosive.

A number of investigators have produced estimates of the economic costs of materials damage. A significant economic cost has been attributed to this damage, although the estimates produced vary greatly.

1.5 HEALTH EFFECTS ASPECTS

In the first three chapters (Chapters 11, 12, and 13) of the fourth volume, information is assessed regarding the uptake, deposition, and absorption of sulfur oxides and particulate matter and various health effects demonstrated to be associated with these pollutants by means of animal toxicology and human clinical studies. Such studies offer the advantage of being able to study biological processes specifically associated with particular pollutant exposures under highly controlled laboratory conditions.

The animal toxicology studies are particularly valuable in providing both qualitative characterization of the full ranges of health effects caused in mammalian species by SO_2 and particulate matter exposures and information on the mechanisms of action underlying such effects. However, considerable caution must be applied in extrapolating quantitative dose-effect relationships defined in animal studies to humans.

Of course, some such definition of quantitative dose-effect relationships can be more directly ascertained by means of human clinical studies. Such studies, however, are also somewhat limited, in terms of the kinds of health effects potentially characterized by them. More specifically, only the effects of short-term (a few hours) exposures or perhaps a few repeated short exposures are typically investigated in such studies. Also, the nature of the effects studied are generally limited to detection of onset of relatively transient changes in pulmonary or cardiac functions and, at times, related physiological or biochemical parameters. In addition, restrictions arising from human rights considerations often result in limitations that preclude thorough investigation of health effects experienced by the most sensitive members of the population.

Community health (epidemiology) studies offer several advantages that go beyond what can be determined by animal toxicology or human clinical studies, in that health effects of both short- and long-term pollutant exposures (including the presence of other pollutants) can be studied and sensitive members of populations at special risk for particular effects identified. In addition, epidemiology evaluations are not limited to the study of more or less transient physiological or biochemical effects but also include investigation of both acute and chronic disease effects induced by SO_x and particulate matter pollution and associated human mortality as well. Information from epidemiology studies, then, together with the results from animal and human clinical studies, help to provide more complete understanding of the health effects of environmental air pollutants such as sulfur oxides and particulate matter.

1.5.1 Respiratory Tract Deposition and Biological Fate

Airborne particles and sulfur dioxide are deposited in the various regions of human and animal respiratory tracts. Particles are deposited by gravitational settling, impaction, diffusion, interception, and electrostatic attraction. Gases are deposited due to convective and diffusional processes. Chemical and physical properties of particles and SO_2, respiratory tract anatomy, and airflow patterns during respiration also influence deposition. In addition, deposition is influenced by individual respiration and anatomical features, which can vary considerably. Nevertheless, the state of knowledge concerning the deposition of inhaled aerosols and gases is sufficient to predict regional deposition.

The respiratory tract is usually described in terms of three functional regions: (1) nasopharyngeal, (2) tracheobronchial, and (3) pulmonary. The deposition of inhaled material depends on its aerodynamic properties. Material

soluble in body fluids will readily enter the blood stream. Hygroscopic and deliquescent solid particles can grow in the humid respiratory tract, depositing in greater proportion and in larger airways than can insoluble or hydrophobic particles.

Relatively insoluble material deposits in various regions according to particle size. If it lands on ciliated epithelium, either in the nasopharyngeal or tracheobronchial airways, it will be moved by mucous flow to the throat and be swallowed. Relatively insoluble material deposited on nonciliated surfaces in the pulmonary regions may be phagocytized by alveolar macrophages, may enter the interstitium and remain in the lung for an extended period, or may be translocated via lymphatic drainage. Some material from the pulmonary region may enter the tracheobronchial region and be cleared via the mucociliary conveyor. Very insoluble particles deposited in the pulmonary region may remain there for months or years, while similar particles deposited in the tracheobronchial or nasopharyngeal regions are cleared in a few days.

Sulfur dioxide may be deposited directly in the airways or enter into a variety of gas-particle chemical and physical reactions. SO_2 may dissolve in liquid droplet aerosols or hygroscopic and deliquescent particles and thereby lead to increased deposition deep in the respiratory tract. The aerodynamic properties of particles and the route of breathing affect deposition.

A nose-breathing person taking 15 breaths per minute with a tidal volume of 1450 ml would have deposited in the deep lung: 35% of the 0.2 μm particles inhaled; 25% of the 1 μm particles inhaled; 10% of the 5 μm particles inhaled; and almost none of the 10 μm particles inhaled. During mouth breathing these deposition percentages would be expected to be about 35 percent, 30 percent,

55 percent and 10 percent, respectively. This difference shows that pulmonary deposition of large particles is greater during mouth breathing than during nose breathing. Some particles that normally are collected in the nasopharyngeal region during nose breathing may pass the glottis and be deposited in the upper part of the tracheobronchial tree during mouth breathing. One ~~study~~ investigator (Lippmann, 1977) ~~indicated~~ calculated that about ~~50~~ 10 percent of 15 μm unit density spheres may enter the tracheobronchial tree during mouth breathing, while almost none of these particles enter the tracheobronchial tree during nose breathing.

Since many biological studies of inhaled aerosols and SO_2 involve experimental animals, it is important to understand how humans and animals differ with respect to pulmonary deposition and clearance. Small rodents tend to have smaller lung deposition fractions of particles less than 3 μm D_{ar} and somewhat higher pulmonary clearance rates than humans. In dogs, however, particle deposition and clearance parallel that in humans. Mouth-breathing people bypass most of the nasal filtration of SO_2 and have much higher lung exposure than nose-breathing experimental animals. This may be a very important factor for humans under heavy work or exercise conditions. The three functional regions of the respiratory tract can each be characterized by mechanisms of deposition, clearance, and potential biological responses. The respiratory tract is itself a target of inhaled particles and gases, and it is also the portal of entry to other organs that may be affected.

1.5.2 Animal Toxicology Studies

Although major gaps exist in the animal toxicological data base on sulfur oxides (SO_x) and particulate matter (PM), such data can be useful in:

(1) delineating the full range of toxicological effects of SO_x and PM, including the effects of both very high-level exposures and prolonged low-level exposures;

(2) elucidating potential mechanisms of toxicity and defining structure-function relationships between physico-chemical properties of these agents and particular health effects;

(3) supporting the findings of human clinical and epidemiological studies investigating analogous health endpoints; and

(4) investigating of health effects (e.g., potential mutagenesis or carcinogenesis effects), which neither can be experimentally induced in human clinical studies nor easily or precisely assessed with current epidemiology methods.

The use of the toxicological data base from animal studies in order to make comparisons between man and animals involves assumptions that a chemical has ultimate mechanisms of toxicity involving chemical and biological structures which are similar in man and animals. Although the precise mechanisms underlying most effects are currently unknown, it is typically acknowledged that common biochemical events are probably involved for both man and other mammalian animal species. For example, if a pollutant is observed to cause toxicity by destroying a chemical structure essential for normal activity of a cell membrane in one mammalian species, then that cell structure is likely at risk in other mammalian species and humans. The critical issue then becomes delivery of the pollutant to that structure in different animal species and human beings. Thus, qualitative extrapolation of the type of effect from animal to man has a theoretical basis; and that basis becomes even stronger if the same effect is observed in a number of animal species, since species differences in susceptibility can exist. As will be discussed later, certain experimental evidence indicates the occurrence of similar effects in man and animals do occur in cases where the same biological endpoint has been examined.

The current animal toxicological data base on the effects of particulate matter and sulfur oxides is relatively limited. In addition, sulfuric acid studies which used whole body exposure chambers ~~are~~ may be confounded by an ammonia-induced conversion of H_2SO_4. Since such an event would not occur to an equivalent degree in human exposure chambers, direct quantitative comparison of animal and human H_2SO_4 exposure effects would not be accurate, and failure to find an effect in animal studies does not rule out its possible occurence in humans in the absence of ammonia. ~~or other agents more often found in animal living and testing conditions~~. On the other hand, many animal studies which have been conducted with SO_2, $(NH_4)_2SO_4$, and sulfuric acid used very high concentrations relative to ambient air and with exposures of short duration (less than 1 day) and the direct relevance of such exposure conditions to assessment of quantitative dose-effect/dose-response relationships for humans can be questioned. Turning to a summary of animal study results, note that certain studies have looked at the effects of relatively high levels of SO_2, particles, or aerosols on pulmonary system morphology or function in various animal species. Some animal studies have also examined the effects of such exposures on immune system functions and susceptibility to bacterial or viral infections. Certain key findings are summarized below.

1.5.2.1 <u>Effects of Acute and Chronic Exposure to Particles or SO_2</u>--The results of most of the animal studies discussed in Chapter 12 regarding the effects of acute and chronic exposure to various particles or SO_2 alone (at concentrations ≤1 mg/m³ or 5 ppm respectively) are summarized in Tables 1-8, 1-9, and 1-10. Key findings and their possible implications for human health assessment are discussed in what follows.

TABLE 1-8. SUMMARY OF EFFECTS OF ACUTE EXPOSURE TO ≤ 1 mg/m^3 PARTICLES[a] IN ANIMALS

Concentration	Duration	Species	Results	Reference
0.05 mg/m^3 $CdCl_2$	2 hr	Hamster	Decreased ciliary beat frequency in trachea.	Adalis et al.[157]
0.1 mg/m^3 $NiCl_2$	2 hr	Hamster	Decreased ciliary beating frequency in trachea.	Adalis et al.[156]
0.1 mg/m^3 $CdCl_2$ or 0.2 mg/m^3 $CdSO_4$	2 hr ($CdCl_2$) 3 hr ($CdSO_4$)	Mouse	Increased susceptibility to streptococcal lung infection.	Gardner et al.[154] Ehrlich et al.[179]
0.1 mg/m^3 H_2SO_4	1 hr	Guinea pig	41% increase in flow resistance and 27% decrease in compliance. (Higher concentrations did not cause these effects in a similar study, Reference 53).	Amdur et al.[172] Amdur[173]
0.19-1.4 mg/m^3 H_2SO_4	1 hr	Donkey	Bronchial mucociliary clearance was slowed.	Schlesinger et al.[222]
0.19 mg/m^3 $CdCl_2$	2 hr	Mouse	Decreased number of antibody-producing spleen cells.	Graham et al.[160]
0.25 mg/m^3 $NiCl_2$	2 hr	Mouse	Decreased number of antibody-producing spleen cells	Graham et al.[160]
0.25 mg/m^3 $ZnSO_4$, $(NH_4)_2$ SO_4	1 hr	Guinea pig	22% increase in flow resistance	Amdur and Corn[123] Amdur[173]
0.4-2.1 mg/m^3 $(NH_4)_2SO_4$	1 hr	Donkey	No change in pulmonary resistance or dynamic compliance	Schlesinger et al.[222]
0.43 mg/m^3 $CuSO_4$	1 hr	Guinea pig	No significant change in flow resistance. 11% increase in compliance.	Amdur et al.[130]
0.5 mg/m^3 $(NH_4)_2SO_4$	1 hr	Guinea pig	23% increase in flow resistance and 27% decrease in compliance.	Amdur et al.[130]
0.5 or 1.0 mg/m^3 H_2SO_4	1 hr	Dog	At the lower concentration, tracheal mucociliary transport was accelerated, but 1 wk later, it was depressed. At 1 mg/m^3, the rate was depressed, even at 1 wk post-exposure.	Wolff et al.[224]
0.5 mg/m^3 $NiCl_2$	2 hr	Mouse	Increased susceptibility to streptococcal lung infection.	Adkins et al.[155]

TABLE 1-8 (continued).

Concentration	Duration	Species	Results	Reference
0.6 mg/m^3 $CuSO_4$	3 hr	Mouse	Increased susceptibility to streptococcal lung infection	Ehrlich et al.[179]
0.8-1.51 mg/m^3 H_2SO_4	1 hr	Donkey	No change in pulmonary resistance or dynamic compliance.	Schlesinger et al.[222]
0.9 mg/m^3 H_2SO_4	2 hr	Hamster	Decreased tracheal ciliary beat frequency.	Grose et al.[181] Schiff et al.[182]
0.9 mg/m^3 H_2SO_4	2 hr	Mouse	No effect on susceptibility to infectious bacterial pulmonary disease.	Gardner et al.[145]
0.91 mg/m^3 $ZnSO_4$	1 hr	Guinea pig	41% increase in flow resistance.	Amdur and Corn[123] Amdur[170]
0.93 mg/m^3 NH_4HSO_4	1 hr	Guinea pig	15% increase in flow resistance and 15% decrease in compliance	Amdur et al.[130]

[a]The toxicity of particles is dependent on particle size. For simplicity, this table does not address this issue completely. Data are presented for lowest effective concentration tested. See the text and tables of Chapter 12 for details. (See Table 12-8 for more complete details on effects of particles on airway resistance in guinea pigs.)

TABLE 1-9. SUMMARY OF EFFECTS OF CHRONIC EXPOSURE TO ≤ 1 mg/m^3 PARTICLES[a] IN ANIMALS

Concentration	Duration	Species	Results	Reference
0.08, 0.1 mg/m^3 H_2SO_4	52 wk, continuous	Guinea pig	No effects on hematology, pulmonary function.	Alarie et al.[92,197]
0.38, 0.48 mg/m^3 H_2SO_4	78 wk, continuous	Monkey	No hematological changes. At 0.38 mg/m^3 there were bronchiolar epithelial hyperplasia and thickening of the respiratory bronchioles. Particle size influenced the effects. Exposure to 0.48 mg/m^3 altered distribution of ventilation early in the exposure period. Respiratory rate increased at 0.38 mg/m^3. Other functional parameters were not affected.	Alarie et al.[197]
0.89 mg/m^3 H_2SO_4	21 hr/day, 225 or 620 days	Dog	No morphological changes at 620 days. At 225 days, CO diffusing capacity was decreased. At 620 days, CO diffusing capacity was decreased and other pulmonary function measurements were affected.	Lewis et al.[89,104]
0.1 mg/m^3 H_2SO_4	1 hr/day, 5 days/wk, several mo	Donkey	After 4 wk erratic bronchial mucociliary rates were observed. During the second 3 mo of exposure, clearance was slowed in animals never pre-exposed.	Schlesinger et al.[223]
0.01, 0.15 mg/m^3 Pb_2O_3; 0.01 mg/m^3 $PbCl_2$, 0.11 mg/m^3 $NiCl_2$, 0.12 mg/m^3 NiO	3 mo continuous for Pb_2O_3; 12 hr/day, 6 days/wk, 2 mo	Rat	Decreased number of alveolar macrophages after Pb_2O_3. NiO increased the number of alveolar macrophages. The soluble metals did not change the number of alveolar macrophages.	Bingham et al.[152,153]

[a]The toxicity of particles is dependent on particle size. For simplicity, this table does not address this issue completely. Data are presented for lowest effective concentration tested. See the text and tables of Chapter 12 for details.

TABLE 1-10. SUMMARY OF EFFECTS OF EXPOSURE TO $\leq$13.1 mg/m^3 (5 ppm) SULFUR DIOXIDE IN ANIMALS

Concentration	Duration	Species	Results	Reference
0.26 or 2.62 mg/m^3 (0.1 or 1 ppm)	7 hr/day, 5 days/wk, 25 days	Rat	0.26 mg/m^3 (0.1 ppm) accelerated tracheobronchial clearance at day 10 and 23. The higher concentration accelerated clearance at day 10, but at day 25, clearance was decreased.	Ferin and Leach[110]
0.37, 1.7 or 3.35 mg/m^3 (0.14, 0.64, or 1.28 ppm)	78 wk, continuous	Monkey	No effects on pulmonary morphology or morphology.	Alarie et al.[90,91]
0.42 or 0.84 mg/m^3 (0.16 or 0.32 ppm)	1 hr	Guinea pig	Increase in flow resistance.	Amdur and Underhill[124] Amdur et al.[130]
2.62, 5.24, 13.1 mg/m^3 (1, 2, or 5 ppm)	1 hr	Dog	Increased bronchial reactivity to aerosols of a bronchoconstrictor agent (acetylcholine).	Islam et al.[102]
2.62 mg/m^3 (1 ppm)	1.5 hr/day	Dog	Decreased mucous flow.	Hirsch et al.[111]
2.62 or 13.1 mg/m^3 (1 or 5 ppm)	24 hr	Rat	Phagocytosis of alveolar macrophages increased after 3 or 4 days in culture.	Katz and Laskin[195]
9.43 mg/m^3 (3.6 ppm)	7 days, continuous	Mouse	Exposure to SO_2, whether alone or in combination with a virus, produced weight loss.	Lebowitz and Fairchild[109]
13.1 mg/m^3 (5 ppm)	6 hr/day, 20 days	Guinea pig	No change in bacterial clearance.	Rylander[107] Rylander et al.[192]
13.1 mg/m^3 (5 ppm)	3 hr/day, 1-15 days and 24 hr/day, 1-3 mo	Mouse	No change in mortality due to a laboratory-induced streptococcal infection.	Ehrlich[178]

As indicated by the results of certain studies discussed in chapter 12, ~~summarized in Tables 1-8 and 1-9,~~ virtually all particles examined to date cause health effects at relatively high exposure levels (≥ 1 mg/m³). The type and severity of the effects are chemical-specific and concentration dependent, with relatively few having been observed at concentrations < 1 mg/m³. For two classes of effects (histamine release and increased susceptibility to infectious disease), the cation of a given particle species was found to have more influence on the toxicity than the anion. For example, in regard to increasing susceptibility to infection, $CdSO_4$ was more toxic than $ZnSO_4$, while $NaSO_4$ and $(NH_4)_2SO_4$ had no significant effect even at high concentrations. Guinea pigs experienced approximately a 40% increase in airway resistance with exposure either to 0.1 mg/m^3 H_2SO_4 or 0.5 mg/m^3 $ZnSO_4(NH_4)_2SO_4$. ~~$ZnSO_4$ and $(NH_4)_2SO_4$~~. The ranking of toxicological potency varies with site of deposition or physiological process. For example, the ranking of sulfates for airway resistance does not precisely agree with that for effects on susceptibility to pulmonary infection. These findings illustrate the complexity of toxicological responses and show the need for a broad data base.

The size of the particle plays a role in the health effects observed. Unfortunately, no known experimental data compare the effects of particles which would predominately be deposited in the head vs. the respiratory tract. Studies generally have been conducted with particles that tend to enter the deep portions of the lung where gas-exchange occurs. In studies employing H_2SO_4 or $ZnSO_4$ and $(NH_4)_2SO_4$ particles of various sizes that would predominantly deposit in the gaseous exchange region, it was found that pulmonary flow resistance increased as particle size decreased.

As for particulate matter dose-effect relationships, in contrast to results discussed below for SO_2 alone, acute (1-2 hr) exposure of the several different animal species listed to much lower levels of different particulate

substances have been shown to lead to various ~~patho~~physiological changes, e.g., slowed mucociliary clearance, increased flow resistance and compliance, ~~and increased flow resistance and compliance~~, and increased susceptibility to an infectious bacterial agent. Such effects occurred with exposures to such particulate substances as H_2SO_4, $ZnSO_4$, $CuSO_4$, and Ni and $CdCl_2$ at levels as low as 100-200 $\mu g/m^3$. Also chronic exposures of various animal species to sulfate aerosols, as shown on Table 1-8, results in such effects as bronchiolar epithelial hyperplasia, slowed mucociliary clearance rates, and decreased CO diffusing capacity. Certain of these changes occur at chronic exposure levels of 100 to 500 $\mu g/m^3$ of sulfate aerosol. Other effects, e.g., altered numbers of alveolar macrophages, have also been observed with chronic exposures to lead and nickel oxide particle concentrations of around 100 $\mu g/m^3$.

Some, but not all, animal and controlled human studies have shown that acute exposure to high (>1 ppm; 2860 $\mu g/m^3$) concentrations of SO_2 increases airway resistance. At lower concentrations, only in animal studies on the guinea pig, has increased airway resistance been observed, i.e., at 420 to 840 $\mu g/m^3$. It appears, however, that the mechanism of SO_2-induced flow resistance is likely similar in man and animals, since atropine inhibits the response in both types of subjects. This suggests that the effect of SO_2 is mediated by parasympathetic motor pathways which alter airway smooth muscle tone.

In contrast to the above, human and animal studies of effects of H_2SO_4 on pulmonary function, have yielded variable results. Certain concentrations of H_2SO_4 increased resistance in some guinea pig studies, but not in human studies. In guinea pigs, but not in humans, H_2SO_4 is far more potent than SO_2 in increasing resistance. In guinea pigs, salt (NaCl) potentiates the response to SO_2. In some human studies, a similar potentiation occurred. If the hypothesis of

H_2SO_4 formation as a possible explanation were true, then H_2SO_4 can likely affect human airway resistance. However, in other human studies, SO_2 plus NaCl was not found to result in potentiation.

Some possible reasons for this difference in H_2SO_4 action between man and animals can be offered. Perhaps the mechanism for H_2SO_4 has a species specificity. In animals histamine release is one of the hypothesized mechanism. Man also has histamine stores and reacts to histamine release. Nonetheless, the detailed metabolism and action of histamine in man and animals is not fully known. Another possible difference between human and animal studies is the extent neutralization of H_2SO_4 by ammonia in exposure chambers, with much higher ammonia levels present in animal chambers from the urine of the test subjects. Both human and animal SO_2 studies, however, have shown approximately a 10 percent incidence of more responsive individuals. It could be possible that the positive animal studies had an unusually high incidence of these "sensitives" and the humans an unusually low incidence of "sensitives". Another possibility is that the negative guinea pig studies more closely reflect the human condition, and neither the resistance of man nor animals is highly affected by H_2SO_4.

Mucociliary clearance has also been investigated. In dogs, rats, and man, short-term exposure to lower concentrations of SO_2 and H_2SO_4 generally accelerated bronchial clearance of particles, while higher concentrations slowed clearance. In donkeys, single or repeated exposures to low concentrations of H_2SO_4 appeared to slow bronchial clearance. Such findings would imply that these chemicals might increase the residence times of substances in the lungs that are normally removed faster. This slowed clearance could influence susceptibility to infectious disease. Guinea pigs exposed to SO_2 experienced an increase in laboratory-induced viral pneumonia. Some animal

studies indicate that acute exposure to a variety of sulfates increases susceptibility to infectious (bacterial) pulmonary disease in mice, probably by affecting alveolar macrophages and additional host defense mechanisms. Metal sulfates caused these effects, but much higher concentrations of H_2SO_4, $(NH_4)_2SO_4$, and SO_2 did not.

1.5.2.2 Effects of Exposures to Combinations of SO_x and Particles--Some investigations with animals have focused on the effects of SO_2 in combination with particles. The results of such studies are summarized in Table 1-11. Long-term exposure of mice to carbon plus SO_2 or carbon alone caused alterations in the immune system which were more extensive than those caused by SO_2 alone. Combinations of H_2SO_4 and carbon resulted in greater morphological effects in the lungs of mice than exposure to carbon alone. Similar combination exposures caused more effects on tracheal tissue than did carbon or H_2SO_4 alone. In mice exposed acutely to O_3 and then to H_2SO_4, an additive effect was observed on susceptibility to infectious respiratory disease. However, this same regimen resulted in antagonistic effects on reduction of tracheal ciliary beating frequency. For long-term exposure, the effects of O_3 plus H_2SO_4 on lung morphology species were attributed to O_3 alone. The mechanisms for the toxicological interactions described above are not known; the effects of ammonia neutralization of H_2SO_4 during long exposure studies, however, cannot be ruled out.

A variety of other chronic studies have been conducted with single chemicals and pollutant combinations. At concentrations below 13.4 mg/m^3 (5.12 ppm) SO_2 for up to 18 months, there were no morphological or pulmonary functional changes in monkeys. A similar level of SO_2 caused increased pulmonary flow resistance and decreased lung compliance in dogs after 225 days of exposure.

TABLE 1-11. SUMMARY OF EFFECTS OF COMBINATIONS OF PARTICLES[a] ($\leq$1 mg/m^3) AND SULFUR DIOXIDE ($\leq$13.1 mg/m^3, 5 ppm) IN ANIMALS

Concentration	Duration	Species	Results	Reference
0.79-0.84 mg/m^3 (0.3-0.32 ppm) SO_2 + 0.9 mg/m^3 $(NH_4)_2SO_4$, NH_4HSO_4, or Na_2SO_4	1 hr	Guinea pig	Additive effect on increased flow resistance.	Amdur et al.[130]
0.94 mg/m^3 (0.36 ppm) SO_2 + 0.4 mg/m^3 $CuSO_4$	1 hr	Guinea pig	Potentiation of increased flow resistance	Amdur et al.[130]
2.62 mg/m^3 (1 ppm) SO_2 + 1 mg/m^3 NaCl	1 hr	Guinea pig	At 80% relative humidity, NaCl potentiated the response to SO_2 flow (increased flow resistance).	McJilton et al.[141]
2.62 mg/m^3 (1 ppm) SO_2 + aerosols of metal salts	1 hr	Guinea pig	Manganous chloride, ferrous sulfate and sodium orthovanadate potentiated the response to SO_2 flow (increased flow resistance).	Amdur and Underhill[96]
2.6 mg/m^3 (1 ppm) SO_2 + 0.9 mg/m^3 H_2SO_4	18 mo, continuous	Monkey	No effects on hematology or pulmonary function. Morphological changes in bronchial mucosa. Addition of 0.41 mg/m^3 fly ash did not alter the results.	Alarie et al.[92]
0.08 mg/m^3 H_2SO_4 + 0.45 mg/m^3 fly ash	12 mo, continuous	Guinea pig	No changes on hematology, pulmonary function, or morphology.	Alarie et al.[92]
0.28, 2.62, or 13.1 mg/m^3 (0.11, 1, or 5 ppm) SO_2 + 0.56 mg/m^3 fly ash	78 wk, continuous 52 wk, continuous	Monkey Guinea pig	No effects on pulmonary function, morphology, or hematology.	Alarie et al.[199]
5.24 mg/m^3 (2 ppm) SO_2 + 0.56 mg/m^3 carbon	100 hr/wk, 192 days	Mouse	Alterations in the pulmonary and systemic humoral immune system.	Zarkower[115]

[a]The toxicity of particles is dependent on particle size which is not presented on this table. For details, see the text and tables of Chapter 12.

TABLE 1-11. continued).

Concentration	Duration	Species	Results	Reference
Various combinations, including 1.1 mg/m^3 (0.42 ppm) SO_2 + 0.09 mg/m^3 H_2SO_4	16 hr/day 68 mo	Dog	No changes in pulmonary function after 18 or 36 mo of exposure. Some alterations in pulmonary function after 61 mo. Thirty-two to 36 mo after exposure ceased, morphological effects were observed (i.e., enlarged air spaces and increased number and size of interalveolar pores, loss of cilia, nonciliated bronchiolar cell hyperplasia and loss of interalveolar septa in alveolar ducts). These changes appear similar to centrilobular emphysema.	Lewis et al.[104,105] Hyde et al.[185] Vaughan et al.[186]

[a]The toxicity of particles is dependent on particle size which is not presented on this table. For details, see the text and tables of Chapter 12.

Long-term exposure of monkeys to H_2SO_4 (as low as 0.38 mg/m^3) caused morphological and some pulmonary function changes. A higher concentration (0.89 mg/m^3) caused no structural changes in dogs, but pulmonary function decrements were observed. Guinea pigs were not affected after a one year exposure to 0.1 mg/m^3 H_2SO_4.

Effects were also noted after chronic exposure to pollutant combinations, but in most cases the contributions of the individual chemical species to the effects of the mixture are obscure. Although various mixtures of SO_2, H_2SO_4, and fly ash caused pulmonary morphologic changes, no effects on pulmonary function were found in monkeys. It appeared that fly ash did not contribute to the effects. Fly ash, when combined with SO_2 or H_2SO_4 did not significantly affect guinea pigs. When dogs received mixtures of SO_2 and H_2SO_4 for 620 days, the effects on pulmonary function were attributed to the H_2SO_4 (0.89 mg/m^3). Several morphological changes were observed in the lungs of dogs 32 to 36 months after a 68 month exposure to 1.1 mg/m^3 (0.42 ppm) SO_2 plus 0.09 mg/m^3 H_2SO_4 ceased. It was hypothesized that these changes were analagous to an incipient stage of human proximal acinar (centrilobular) emphysema. It is not known whether the disease state progressed, abated, or remained stable over this post-exposure period. A shorter exposure (approximately 18 months) to a higher concentration of H_2SO_4 (0.9 mg/m^3) plus 2.62 mg/m^3 (1 ppm) SO_2 caused less serious morphological effects in monkeys. Concentration-time relationships of effects in the above studies are not clear. But from the above-mentioned dog and monkey study, it appears that time of exposure is an important factor in the development of disease. However, concentration does play a role, as evidenced from the concentration-dependent effects after either acute or chronic exposure.

1.5.3 Studies on the Oncogenic Properties of SO_x and PM

The oncogenic potential of SO_2 has been studied, but the findings are not yet conclusive. In one series of experiments, hamsters and rats were exposed for 98 weeks or a lifetime to various regimens of SO_2 and benzo(a)pyrene, a known carcinogen. No lung tumors or other pathological effects were observed for hamsters. Rats, however, had an increased incidence of tumors after exposure to 26.2 mg/m^3 (10 ppm) for 6 hr/day and 9.17 mg/m^3 (3.5 ppm) SO_2 plus 10 mg/m^3 benzo(a)pyrene for 1 hr/day. In a later study, rats exposed for 6 hr/day to 26.2 mg/m^3 (10 ppm) SO_2 and for 1 hr/day to 10.5 mg/m^3 (4 ppm) SO_2 plus 10 mg/m^3 benzo(a)pyrene had a cancer (squamous cell carcinoma) incidence of 19.6 percent. This incidence contrasts with a zero percent incidence in the group receiving air or 26.2 mg/m^3 (10 ppm) SO_2 for 6 hr/day and a 9 percent incidence in the group exposed for 1 hr/day to 10.5 mg/m^3 (4 ppm) SO_2 plus 10 mg/m^3 benzo(a)pyrene. (See Table 12-3 in Chapter 12 for details.) The reports do not specifically state whether the 26.2 mg/m^3 (10 ppm) SO_2 was given before, during, or after the exposure to 10.5 mg/m^3 (4 ppm) SO_2 plus benzo(a)pyrene. Thus, it is difficult to speculate on potential promoter or initiator effects.

In another study, mice were exposed ~~to 1310 mg/m^3 (500 ppm) SO_2~~ in a 180 liter chamber into which 1310 mg/m^3 (500 ppm) SO_2 was injected at a rate of 20 ml/min for 5 min/day, 5 days/wk for a lifetime. This exposure increased the carcinoma incidence in females from 0 to 18 percent. No such change occurred in the males. Primary pulmonary neoplasias increased in both males and females. The investigators conclude that the increased incidence of primary lung tumors is a result of an SO_2-induced inflammatory reaction, "followed by a state of apparent tolerance, which accelerates the inherent tendency of these mice to develop lung tumor spontaneously but does not justify the classification of SO_2 as a chemical carcinogen as generally understood." From the single study

of SO_2 alone in mice and the two studies of SO_2 plus benzo(a)pyrene in rats, no definitive conclusions can be drawn about the carcinogenic or co-carcinogenic potential of SO_2. However, the data reported thus far do justify some concern, and these issues need to be more extensively addressed in further experimental research.

It is commonly believed that fundamental similarities exist between the molecular mechanisms of both mutagenesis and carcinogenesis. This assumption is based on the theory that a chemical interaction with DNA and/or other critical cellular macromolecules initiates a genetic change which may lead to carcinogenic transformation. Therefore, the demonstration of mutagenic activity for a substance is generally taken as strong presumptive evidence for the existence of carcinogenic activity. It follows that an investigation of the mutagenicity of a substance may be predictive of carcinogenic potential, and may serve as an early warning of a possible threat to human health.

1.5.4 Experimental Investigations of Human Subjects

Studies of human subjects exposed to sulfur oxides and particulate matter explore physiological and sensory responses to these pollutants under controlled conditions. A major limitation of such studies is that they do not reflect the long exposure durations which typically occur in the ambient environment. However, they may reflect short-term peak exposures, such as may occur in certain urban situations. The results of human clinical studies discussed in Chapter 13 are summarized in Tables 1-12 to 1-14 below.

The typical response to SO_2 exposures is an increase in pulmonary flow resistance. Most subjects respond to short exposures of 5 ppm (13,000 $\mu g/m^3$), while other subjects react to lower levels of SO_2. Significant decreases in nasal mucous flow rate have been demonstrated at 1 ppm; 2620 $\mu g/m^3$. Pulmonary

1-12. PULMONARY EFFECTS OF AEROSOLS

Concentration	Duration of exposure (mins)	Number of subjects	Source	Effects	Reference
SO_2 (1.6 - 5 ppm) NaCl 0.22 μm MMD	5	13	Mask	Synergistic increases in airway resistance with aerosol	Toyama, 1962
SO_2 (9-60 ppm) NaCl (CMD ≈ 0.95 μm)	5	10	Mask	Airway resistance greater after exposure to aerosol than to exposure to SO_2 alone	Nakamura, 1964
SO_2 (0.5, 1.0 and 5.0 ppm) Saline particles 7.0 μm	15	9	Oral	$MEF_{50\%}$ significantly greater decreases in aerosol (NaCl) condition	Snell and Luchsinger, 1969
Ibid	30	9 (asthmatics)	(Mask (Exercise for 10 minutes)	$\dot{V}_{max\ 50\%}$, $\dot{V}_{max\ 75\%}$, $FEV_{1.0}$ and R_T decrease significantly in aerosol condition	Koenig et al., 1979
SO_2 (1.1 - 3.6 ppm) NaCl 2.0 - 2.7 μg/m^3 MMD = 0.25 μm	30	10	Oral	No effect on pulmonary functions	Burton et al., 1969
SO_2 (1-2, 4-7, 14-17 ppm) NaCl 10-30 mg/m^3 MMD 0.15 μm	30	12	Oral	Changes in pulmonary function similar to changes due to SO_2 alone not influenced by aerosol	Frank et al., 1964
SO_2 (1 ppm) NaCl 1 mg/m^3 MMD 0.9 μ σg = 2.0 μm	60	9 (asthmatics)	Oral	Significant decreases in $\dot{V}_{max\ 50\%}$ and $\dot{V}_{max\ 75\%}$	Koenig, 1979
Ibid	60	(normals)	Mask	No pulmonary effects demonstrated	Koenig, 1979
Ammonium sulfate 100 μg/m^3	150	5 (normal) 4 (ozone sensitive) 6 (asthmatics)	Chamber (exercise)	No changes in pulmonary functions	Bell and Hackney, 1977;
Ammonium bisulfate 85 μg/m^3 aerosol size distribution 0.4 μm (MMAD)	150	16	Chamber (exercise)	No changes in pulmonary functions	Kleinman and Hackney, 1978; Avol et al., 1979
0.35 - 5.0 mg/m^3 H_2SO_4 MMD 1 μm	15	15	Mask (rest)	Respiratory rates increased, max. insp. and expiratory flow rates and tidal decreased volumes	Amdur et al., 1952
3-39 mg/m^3 H_2SO_4 MMD 1-1.5 μm	10 - 60	Variable	Mask (rest) Chamber (rest)	Longer particles due to "wet mist" resulted in increased flow resistance cough, rales bronchoconstriction	Sim and Pattle, 1957
SO_2 (1-60 ppm) plus H_2O_2 to form H_2SO_4 aerosol CMD 1.8 and 4.6 μm	Variable	24	(Rest)	Airway resistance increased especially with larger particles	Toyama and Nakamura, 1964
H_2SO_4 mist (1000 μg/m^3 MMD 0.5 μm (σg = 2.59)	120	10	Chamber (exercise)	No pulmonary function changes but increased tracheobronchial clearance	Newhouse et al., 1978
H_2SO_4 aerosol 10, 100, 1000 μg/m^3 MMD 0.1 μm	10	6 normal 6 asthmatics	Oral	No pulmonary function changes, no alterations in gas transport	Sachner et al., 1978
H_2SO_4 (75 μg/m^3) MMAD 0.48 - 0.81 μm	120	6 normal 6 asthmatics	Chamber (exercise)	No pulmonary effects in either group	Kleinman and Hackney, 1978; Avol et al., 1979
H_2SO_4 (0, 100, 300, or 1,000 μg/m^3 MMAD 0.5 μm (σg = 1.9)	60	10	Nasal	No pulmonary function effects Broncial mucociliary clearance ↑ following 100 μg/m^3 but ↓ following 1000 μg/m^3 mucociliary clearance distal to trachea	Lippmann et al., 1979

TABLE 1-13. EFFECTS OF SO_2

Pollutant Concentration (PPM)	Duration of Exposure	Effects	Reference
.37	120 minutes	No pulmonary effects	Bates and Hazucha, 1973; Hazucha and Bates, 1975
.37	120 minutes	No pulmonary effects	Bell et al., 1977
.40	120 minutes	No pulmonary effects	Bedi et al., 1979 Horvath and Folinsgee, 1977
.50	180 minutes	MMFR decreased 2.7%; delayed asthma	Jaeger, et al., 1979
.75	120 minutes	~~Significant decrease in FVC, $FEV_{1.0}$, MMFR~~ significant decrease in MEFR; FVC, $FEV_{1.0}$; MMFR also decreased	Bates and Hazucha, 1973; ~~Hazucha and Bates, 1975~~
1.0	60 minutes	No effects observed	McJilton, 1976
1.0, 5.0	120 minutes	Increase in nasal flow resistance, decrease in nasal mucus flow	Andersen, et al., 1974
1.0, 3.0, 5.0	3 minutes	Light exercise potentiates effect of SO_2; MFF 40% decreased	Kreisman, et al., 1976
1, 5, 13	10-30 minutes	No changes in pulse rate, respiratory rate. pulmonary flow resistance increased at 5 and 13 ppm but less during nasal breathing;	Frank, et al., 1962
1, 5, 15	30 minutes	Increase in R1 at SO_2 above 5 ppm	Frank et al., 1964
1.1-3.6	30 minutes	Deep breathing produced no effects	Burton et al., 1969
1.0, 5.0, 25.0	up to 6 hr/day	Significant decreases in expiratory flow and $FEV_{1.0}$, decreased mucus flow	Anderson, et al., 1974
5.0	4.5 hrs.	Number of colds similar in both groups but severity less in SO_2 exposed groups	Anderson, et al., 1977
1-23	60 minutes	Bronchoconstriction	Sim and Pattle, 1969
1.3-80	10 minutes	Bronchoconstriction	Sim and Pattle, 1957
2.5-50	10 minutes	Increased inspiratory and respiratory resistance	Abe, 1967
4-6	10 minutes	Airway conductane decreased reflex effect	Nadel, 1965
1-8	10 minutes	Pulse rate, respiratory rate increased; tidal volume decreased	Amour, et al., 1953
5.0	120 minutes	MMFR decreased 8.5%; increased tracheobronchial clearance	Newhouse, et al, 1978

at 1 ppm; one subject experienced 7% increase in flow resistance; another, a 23% decrease

1-14. PULMONARY EFFECTS OF SO_2 AND OTHER AIR POLLUTANTS

Concentration	Duration of exposure (mins)	Number of subjects	Source	Effects	Reference
SO_2 (0.37 ppm) and O_3 (0.37 ppm)	120	8	Chamber (exercise)	Decrease pulmonary functions (in synergistic effect of SO_2 on O_3) FRC, $FEV_{1.0}$, MMFR, $MEFR_{50\%}$	Hazucha and Bates, 1973, 1975
SO_2 (0.37 ppm) and O_3 (0.37 ppm)	120	4 (normal) 4 (ozone sensitive) 4 (from Bates)	Chamber (exercise)	Unable to confirm synergistic effects pulmonary decrement due to O_3 alone	Bell et al., 1977
SO_2 (0.40 ppm) and O_3 (0.40 ppm	120	9	Chamber (exercise)	Unable to confirm synergistic effects changes due to ozone alone	Horvath and Folinsbee (1977); Bedi et al. (1979)
SO_2 (5 ppm) and NO_2 (5 ppm)	120	11	Chamber (exercise)	No changes in $P_{a_{O_2}}$, $P_{a_{CO_2}}$, pHa or TGr $\cdot R_t$ increased	von Nieding et al., 1979
SO_2 (5 ppm) NO_2 (5 ppm) and O_3 (0.1 ppm)	120	11	Chamber (exercise)	No changes in $P_{a_{CO_2}}$, $P_{a_{O_2}}$, pHa or TGr $\cdot R_t$ increased	von Nieding et al., 1979
SO_2 (0.12 ppm) NO_2 (0.06 ppm) and O_3 (0.025 ppm)	120	11	Chamber (exercise)	No changes in pulmonary functions	von Nieding et al., 1979

responses to SO_2 exposure apparently persist for some 5 to 10 minutes, while the change in mucous flow rates persists for several hours following exposure. Approximately 10 to 20 percent of individuals studied under controlled conditions appear to be sensitive to exposures as low as 1 ppm SO_2. Effects on certain sensory functions, (e.g., dark adaptation, odor perception) are evident at SO_2 concentrations below 1 ppm (2600 $\mu g/m^3$).

A number of factors influence responses to SO_2. Sulfur dioxide is removed more efficiently if individuals breathe by nose than by mouth. This removal is related to the high solubility of SO_2 in water; most of the SO_2 in inhaled air is absorbed in the moist linings of the nose (and upper airways). When subjects breathe by mouth, more SO_2 reaches deeper areas of the lungs. Subjects who exercise at a level requiring mouth breathing show significant decreases in pulmonary function at an SO_2 concentration of 0.75 ppm (1,950 $\mu g/m^3$).

Asthmatics may be more sensitive to SO_2, but the data on such individuals are not definitive. Sensory awareness of the presence of SO_2 may be decreased in individuals chronically exposed to SO_2.

The presence of particles with SO_2 may influence physiologic responses. Particulate matter may function as a carrier, bringing more SO_2 into the lungs, or may induce chemical reactions to convert SO_2 into sulfates. The presence of aerosols such as NaCl does not appear to modify the pulmonary response to SO_2 in most normal subjects. However, it has been found that adolescent extrinsic asthmatic subjects exhibit pulmonary function changes in the small airways when orally breathing 1 ppm SO_2 and NaCl aerosols. One study shows that brief exercise during this exposure induces changes in both large and small airways.

Results of a chamber study indicated synergistic effects on pulmonary functions in response to a combination of ozone (0.37 ppm) and sulfur dioxide (0.37 ppm). This synergisic effect has not been demonstrated in more recent studies suggesting that the effect reported in the earlier study may have been caused by the presence of some other substances in the exposure chamber.

Few studies have examined the effects of exposure to sulfuric acid and sulfates. Sensory responses to sulfuric acid have not been clearly defined. Pulmonary functions do not appear to be influenced by exposure to H_2SO_4 at concentrations up to 1,000 $\mu g/m^3$. Mucociliary transport in the airways distal to the trachea is affected more by H_2SO_4 exposure than is transport in the trachea. This effect depends upon the concentration levels of H_2SO_4, i.e., exposure to 100 $\mu g/m^3$ increases bronchial clearance, while exposure to 1,000 $\mu g/m^3$ reduces clearance. No adverse pulmonary effects have been reported in normal or asthmatic subjects after 2.5 hour exposures to 100 $\mu g/m^3$ ammonium bisulfate or 85 $\mu g/m^3$ ammonium sulfate.

1.5.5 Community Health Observational Studies

Animal and clinical studies provide incomplete information about the health effects of sulfur oxides and particulate matter. The principal limitations of animal studies are problems with extrapolating quantitative dose effect dose-response relationships from animal to man and the difficulties of mounting studies large enough to detect small health effects, particularly small mortality effects, that may be of public health significance. Health effects of chronic exposure typically cannot be assessed in human clinical studies, and ethical constraints limit the range and quality of acute exposures that can be investigated in such studies. Both types of investigations are limited in that the mix of pollutants found in the ambient air is difficult to

characterize and duplicate in the laboratory. Thus, the evidence from observational studies plays an important and crucial role in assessing the health effects of sulfur oxides and particulate matter.

Observational or community health studies also have important limitations. Since the level of exposure is not under the control of the investigator, observational studies can demonstrate associations but not necessarily cause and effect relationships. Also, since sulfur oxides and particulate matter have common sources, they frequently covary in level in the observational setting, making it difficult to distinguish health effects of these pollutants individually. In addition, other factors such as temperature or frequency of smoking may covary with the level of sulfur oxides and particulate matter, so that effects of pollutants cannot readily be separated from effects of these other factors. A final limitation of observational studies to be mentioned here is the difficulty of relating pollution levels at one or a few community air monitoring stations to health effects seen in persons living in the vicinity of those stations. Further discussion of these and other methodological issues of importance in evaluating observational studies can be found in Chapter 14.

Despite the above limitations, an enormous number of epidemiology studies have been conducted over the past 30 years in order to better define both qualitative and quantitative relationships between various health effects and sulfur oxides and particulate matter in the ambient air; and Chapter 14 of this document provides a rather comprehensive and thorough critical assessment of such studies. The information contained in Chapter 14 is summarized below.

1.5.5.1 Overview Summary of Chapter 14 Contents--In Chapter 14, an extensive array of information is discussed concerning: (1) methodological considerations that must be taken into account in evaluating community health epidemiology studies (Section 14.1); (2) critical assessment of practical applications of air quality measurement techniques employed in the collection of sulfur oxides and particulate matter data utilized in related community health studies (Section 14.2); (3) critical review of such studies on mortality effects associated with acute and chronic exposures to sulfur oxides and particulates (Section 14.3); (4) critical review of studies of morbidity associated with acute exposures to the same pollutants (Section 14.4); and (5) critical assessment of morbidity effects associated with chronic exposures to sulfur oxides and particulate matter. In addition, in the summary and conclusions section of the chapter, there are discussed other published reviews and critical evaluations of the subject material and the interpretations derived from such reviews are compared.

Through the discussion in Section 14.1 of Chapter 14, it is seen that numerous methodological factors, including covarying or confounding variables, can potentially affect the results and interpretation of community health studies. It is also seen, through material summarized in Section 14.2 of the chapter, that a number of sources of errors have been identified as having affected sulfur oxides and particulate matter air quality measurements obtained in both the United Kingdom and the United States and used in British and American epidemiology studies which provide the bulk of the information reviewed in the chapter. It was further noted that while such errors in air measurements can at times be fairly large, they also often act to introduce both positive and negative biases into air quality data sets that tend to cancel each other

out, especially when considering data grouped or averaged over long time periods (monthly; annually) from the same sites or across several geographic areas classed as "low" or "high" pollution areas. At other times, however, it also became clear that certain measurement errors were such as to introduce either consistently negative or positive bias into particular British or American sulfur oxides or particulate matter data sets used in various community epidemiology studies providing information on quantitative air pollution/health effects relationships. It was further noted that such biases due to air quality measurement errors must be taken into account in evaluating such epidemiology studies -- not for the purpose of discrediting such studies but rather to understand better the error limits likely associated with the reported quantitative findings derived from them and to thereby allow for more accurate interpretation of overall patterns of pertinent results.

Turning to the critical assessments of pertinent community health mortality and morbidity studies contained in Sections 4.3, 4.4 and 4.5 of Chapter 14, results of many of the better known and often cited quantitative and qualitative studies discussed in the chapter are summarized in a series of tables presented below along with summary statements regarding the results of community health studies of mortality and morbidity affects associated with short- and long-term exposures to sulfur oxides and particulate matter.

Health Effects of Acute Exposure to SO_2 and Particulate Matter--Qualitative studies demonstrating increased mortality effects to be associated with sulfur oxides and particulate matter air pollution are summarized in Table 1-14. Note that these studies demonstrate, for example, associations between such pollution and significantly increased mortality from bronchitis, pneumonia and heart disease. Also note that essentially all population groups are affected,

TABLE 1-14a QUALITATIVE ASSOCIATION OF GEOGRAPHIC DIFFERENCES IN MORTALITY WITH RESIDENCE IN AREAS OF HEAVY AIR POLLUTION

Pemberton and Goldberg[223]	1950-1952 bronchitis mortality rates in men 45 years of age and older in county boroughs of England and Wales	Sulfur oxide concentrations (sulfation rates) were consistently correlated with bronchitis death rates in the 35 county boroughs analyzed
Stocks[138,164-167]	Bronchitis mortality, 1950-1953, in urban and rural areas of Britian, with adjustments for population density and social index	Significant correlation of mortality from bronchitis and pneumonia among men, and from bronchitis among females, with smoke density
Gorham[224-225]	1950-1954 deaths, 53 counties of England, Scotland, and Wales	Bronchitis mortality was strongly correlated with acidity of winter precipitation
Gore and Shaddick[8] and Hewitt[226]	Mortality in London, 1954-1958 and in 1950-1952, respectively	Duration of residence in London significantly correlated with bronchitis mortality, after adjusting for social class
Haastrom et al.[16] Zeidberg et al.[17] Sprague et al.[18]	1949-1960 deaths for each cause in Nashville, Tenn., categorized by census tract into 3 degrees of air pollution and 3 econimic classes (levels not accurately determined)	Within the middle social class, total respiratory disease mortality, but not bronchitis and emphysema mortality, were significantly assoicated with sulfation rates and social index. White infant mortality rates were significantly related to sulfation rates
Lepper et al.[227]	1964/1965 mortality rates in Chicago census tracts stratified by socioeconomic class and SO_2 concentration	Increased respiratory disease death rates in areas of intermediate and high SO_2 concentration, within a socioeconomic status, without a consistent mortality gradient between the areas of intermediate and high SO_2 concentration
Jacobs and Landoc[175]	1968/1970 mortality rates in Charleston, S.C., industrial vs. non-industrial areas	Higher total and heart disease mortality rates in industrial area

TABLE 1-14a. (continued)

Morris et al.[24]	1960-72 mortality rates compared to 1959-60 air pollution levels	Mortality higher in smokers with lower air pollution exposures
Collins et al.[287]	Death rates in children 0-14 years of age, 1958-1964, in relation to social and air pollution indices in 83 county boroughs of England and Wales	Partial correlation analysis suggested that indices of domestic and industrial pollution account for a differences in mortality from bronchopneumonia and all respiratory diseases among children 0-1 year of age
Beaker et al.[323]	Thanksgiving 1966 Fog, New York	Complaints of cough, phlegm, wheezing, breathlessness, eye irritation increased with increasing air pollution
Toyama[330]	Mortality in districts of Tokyo	Bronchitis mortatliy associated with dustfall (but not cardiovascular, pneumonia or cancer mortality)
Lindeberg[321]	Deaths in Oslo winters	Average deaths per week, 1958-65 winter, correlated with pollution

both males and females and both the young and the old; the very young (infants) and the very old, especially the infirm and those with preexisting respiratory or cardiovascular diseases, however, appear to be at most risk. Essentially the same patterns can be discerned for morbidity effects as demonstrated by qualitative studies summarized in Table 1-15.

Studies providing evidence of quantitative associations between acute health effects and air levels of sulfur oxides and particulate matter are summarized in Table 1-16. Overall, various British, Dutch, Japanese and American episodic mortality studies have yielded results that appear to suggest that mortality effects might occur at or above 300-500 $\mu g/m^3$ SO_2. The three non-episodic mortality studies listed in the table suggest that mortality effects can be seen when TSP levels reach 500 to 600 $\mu g/m^3$ and SO_2 concentrations reach 300 to 500 $\mu g/m^3$. These three studies summarize a relatively small body of data from two winters in London and five winters in New York City. The stated effect levels may be conservative (high), however, since examination of the detailed evidence from these studies presented in Section 14.3 of Chapter 14 suggests the possibility of an exposure-response relationship at lower levels of these pollutants. More complex time series studies of daily mortality have also found associations between mortality and these pollutants at lower levels. The size of the estimated effects has proved to be sensitive to model specification and choice of other adjustment variables. Although the possibility of mortality effects of TSP and SO_2 levels below those cited in Table 1-16 cannot be excluded, it is unlikely that this question can be resolved in the near future by observational studies. Thus, the minimum air levels at which acute mortality increases might be projected to be seen would be 300-500 $\mu g/m^3$ for both TSP and SO_2, based on the results summarized in Table 1-16.

TABLE 1-15. QUALITATIVE STUDIES OF AIR POLLUTION AND ACUTE RESPIRATORY DISEASE

Study	Characteristics	Findings
Angel et al.[69]	Attack rates of minor respiratory illness among 85 London workers, examined every 3 weeks, October 1962-May 1963.	Attack rates were associated with weekly average smoke and SO_2 concentrations.
Levy et al.[70]	Hospital admissions for respiratory disease in Hamilton, Ontario, correlated with sulfur oxide/particulate air pollution index.	Increased hospital admissions on heavy pollution days, except at one hospital far removed from major pollution sources.
Schoettlin and Landau[288]	137 asthmatics reporting attacks on daily occurrence of asthma, September-December, 1956, in Los Angeles Basin.	Significantly more asthma on days of heavier oxidant pollution. No adjustment was made for variations in temperature or season
Zeidberg et al.[289]	Study during 1 year of 49 adults and 34 children with asthma in Nashville, Tenn.	Doubling of asthma attack rates in persons living in more polluted neighborhoods. No adjustment for demographic or social factors.
Cowan et al.[290]	History of asthma, and skin tests of University of Minnesota students, in relation to dust from nearby grain elevator.	Significant association between grain-dust exposure and asthma attacks.
Greenberg et al.[291]	New York City hospital emergency room visits for asthma in month of September.	Emergency room visits strongly associated with onset of cold weather but not with degrees of air pollution during the one month of study.
Weill et al.[292] Carroll[293]	Retrospective study of emergency room visits to New Orleans Charity Hospital.	Periodic "epidemics" of asthma in New Orleans could not be traced to any common pollutant exposure.

TABLE 1-15 (continued)

Study	Characteristics	Findings
Phelps[294] Meyer[295]	"Tokyo-Yokohama asthma" in American servicemen stationed in Japan after World War II.	Disease primarily in smokers attributed to allergic response to atmospheric substances that could not be characterized. Patients improved after leaving the area and were immediately affected on return. Some had long-term effects afterwards.
Glasser et al.[296]	Emergency room visits in seven New York city hospitals during the November 1966 air pollution episode.	Increased emergency room visits for asthma in three of seven hospitals studied.
Chiaramonte et al.[297]	Emergency room visits at a Brooklyn hospital during a November 1966 air pollution episode.	Statistically significant increase in emergency room visits for asthma and for all respiratory diseases, continuing to 3 days after the peak air pollution concentrations.
Derrick[57]	Nighttime emergency room visits for asthma in Brisbane, Australia.	Negative correlation between asthma visits with degrees of smoke shade.
Rao[298]	Pediatric emergency room visits for asthma at Kings County Hospital, Brooklyn, October 1970-March 1971.	Negative correlation of asthma visits with degrees of smoke shade. Lack of temperature adjustments. Considerable distance of hospital district from air monitoring stations.
Goldstein and Black[58]	Emergency room visits for asthma at a hospital in Harlem and in Brooklyn, September-December 1970 and September-December 1971.	Temperature adjusted asthma rates positively correlated with SO_2 values in Brooklyn but not in Harlem. In 1971 period, 50-90% increase in asthma visits on 12 days of heaviest pollution.

TABLE 1-15 (continued)

Study	Characteristics	Findings
Finklea et al.[117]	Incidence of acute respiratory disease, determined at 2-week intervals, in parents of nursery schoolchildren residing in Chicago, December 1969-November 1970.	Acute lower respiratory illness rates were significantly lower among families living in neighborhoods where air pollution had been substantially decreased. Rates were adjusted for social class, smoking, residential mobility, and season of year. Cannot quantitate pollutant exposures.
Finklea et al.[122 123]	Daily diaries kept by 50 asthmatics in each of three New York City area communities, October 1970-May 1971.	Temperature-adjusted attack rates significantly correlated with total particulates in two of the communities. Increase in relative risk from days of light to heavy pollution was relatively small. High turnover in reporting panels.

*Reference 251

TABLE 1-16 SUMMARY TABLE - ACUTE EXPOSURE EFFECTS

Type of Study	Reference	Effects observed	24-hour average pollutant levels at which effects appear	
			TSP ($\mu g/m^3$)	SO_2 ($\mu g/m^3$)
Mortality (episodic)				
British	Table 14-1	Excess deaths	546*	994
Dutch	Table 14-2	Excess deaths	300-500	500
Japanese	Table 14-2	Excess deaths	285	1800
USA	Table 14-2	Excess deaths	570 (5 CoH)	400-532 (1 hr max: 2288)
(Non-episodic)	Martin and Bradley[11]	Increases in daily mortality	500*	300
	Martin[6]	Increases in daily mortality above the 15 moving average	500*	400
	Glasser and Greenburg[222]	Increases in daily mortality	350-450**	524
Morbidity	Martin[16]	Increases in hospital admissions for cardiac or respiratory illness	500*	400
	Lawther et al.[53]	Worsening of health status among 195 bronchitics	344* (250 BS)	300-500
	Greenberg et al.[196]	Increased cardio-respiratory ER visits	357** (260 BS)	715
	Lawther et al.[52]	Increased clinical condition in CB patients	529* (400 BS) 344* (250-350 BS)	450 300
	Stebbings and Hayes[190]	Increased symptoms in chronic bronchitis (CB) patients	200 (60 RSP) (12SS) 8 SN)	100

TABLE 1-16 (continued).

Type of Study	Reference	Effects observed	24-hour average pollutant levels at which effects appear	
			TSP ($\mu g/m^3$)	SO_2 ($\mu g/m^3$)
	Cohen et al.[55]	Increased AS attacks	150 (20SS)	200
	McCarroll et al.[163]	Increased ARI daily inc/prev	160* (1.2 COH)	372
	Cassell et al.[208 209]	Increased ARI average daily inc/prev	205* (2 COH)	452
	Stebbings and Fogleman et al.[216]	Decreased $FEV_{0.75}$ (children)	700	300

*Converted from BS (British Smoke).

Numerous studies reporting morbidity effects associated with acute exposures are also listed in Table 1-16. Worsening of symptoms in bronchitis patients and increased hospital admissions in Britain were reported to occur at TSP and SO_2 levels of 300 or 350 to 500 $\mu g/m^3$ or more. A United States study, however, found exacerbation of symptoms among bronchitics at 200 $\mu g/m^3$ TSP and 100 $\mu g/m^3$ SO_2 and asthmatics were reported to show increased attacks at 150 $\mu g/m^3$ TSP and 200 $\mu g/m^3$ SO_2. Also, spirometry tests were reported to show decreases in lung function at 700 $\mu g/m^3$ TSP and 300 $\mu g/m^3$ SO_2. However, in another study not listed, van der Lende saw improvement in lung function among adults when pollution levels were reduced from 245 $\mu g/m^3$ (TSP) and 300 $\mu g/m^3$ (SO_2). Acute upper and/or lower respiratory illness also has been reported to occur at levels as low as at 160 $\mu g/m^3$ TSP (24-hour averages). Overall, then, the summarized results suggest that (1) very severe morbidity effects, e.g., worsening of symptoms in bronchitic patients, clearly occur at TSP and SO_2 levels of approximately 300 or 350 to 500 $\mu g/m^3$, and (2) less severe but significant morbidity effects may occur with acute exposure at levels of approximately 150-300 $\mu g/m^3$. These studies do not, however, provide a basis for separately estimating the health effects of SO_2 and particulates. Since these two forms of pollution have important common sources, their levels tend to usually vary together over time.

Health Effects of Chronic Exposure to SO_2 and Particulate Matter--The results of qualitative studies on relationships between sulfur oxides and particulate matter pollution are summarized in Table 117. Many of the same types of observations as stated above for population groups at apparent special risk also apply here; that is, the elderly, the infirm, and children appear to be most severely affected health-wise by chronic exposures.

Many well-known and often-cited mortality and morbidity studies that have been reported as demonstrating quantitative associations between mortality, illnesses, or decrements in pulmonary function with chronic (monthly or annual average) levels of particulate matter of SO_2 are summarized in Table 1-18. As seen in that table, the two mortality studies suggest that mortality effects can occur at annual levels of 125 to 140 $\mu g/m^3$ or less of TSP and SO_2. In the morbidity studies, lower respiratory disease, chronic bronchitis, and reduced pulmonary function results were reported that are indicative of morbidity effects likely clearly occurring at annual average TSP or SO_2 levels of 150 to 250 $\mu g/m^3$ or more. Other study results summarized in the table suggest an association of various morbidity effects with concentrations in excess of about 70 to 80 $\mu g/m^3$ TSP and SO_2 concentration in excess of 96 to 107 $\mu g/m^3$. As with studies of acute effects, many of these studies could be further interpreted not only as demonstrating that health effects are exposure-related but also that they increase as these pollutants increase over the entire range of exposures studied and no clear-cut "no effect" level can be determined on the basis of presently available information. Also, in general, these studies cannot be used to distinguish between the effects of sulfur oxides and particulate In several studies, however, TSP effects were reported to occur in the presence of low or non-significant levels of SO_2. (Reference 188, 212, 213, 215, and 257 of Chapter 14, as shown in Table 1-18.)

Health Effects of Atmospheric Sulfates--Conversion to sulfate compounds, including sulfuric acid, has been proposed as a major pathway by which sulfur dioxide and possible other sulfur compounds may exert toxic effects. However, only a few community health studies have attempted to measure and assess health effects associated with suspended sulfates (SS). Stebbings and Hays,[190]

TABLE 1-17. QUALITATIVE STUDIES OF AIR POLLUTION AND PREVALENCE OF CHRONIC RESPIRATORY SYMPTOMS AND PULMONARY FUNCTION DECLINES

Study	Characteristics	Findings
Fairbairn and Reid[265]	Comparison of respiratory illness among British postmen living in areas of heavy and light pollution	Sick leave, premature retirement, and death due to bronchitis or pneumonia were closely related to pollution index based on visibility
Mork[266]	Questionnaire and ventilatory function tests of male transport workers 40-59 years of age in Bergen, Norway and London, England	Greater frequency of symptoms and lower average peak flow rates in London. Differences were not explained by smoking habits or socioeconomic factors
Deane et al.[267]	Questionnaire and ventilatory function survey of outdoor telephone workers 40-59 years of age on the west coast of U.S.	Increased prevalence of respiratory symptoms, adjusted for smoking and age, a larger volume of morning sputum and a lower average ventilatory function in London workers, and in the English compared with American workers. No differences in symptom prevalence between San Francisco and Los Angeles workers, although particulate concentrations were approximately twice as high in Los Angeles
Cederlöf,[39] Hrubec et al.[40]	Chronic respiratory symptom prevalence in large panels of twins in Sweden and in the U.S. Index of air pollution based on estimated residential and occupational exposures to SO_2, particulates, and CO	Increased prevalence of respiratory symptoms in twins related to smoking, alcohol consumption, socioeconomic characteristics, and urban residence, but not to indices of air pollution

TABLE 1-17. QUALITATIVE STUDIES OF AIR POLLUTION AND PREVALENCE OF CHRONIC RESPIRATORY SYMPTOMS AND PULMONARY FUNCTION DECLINES

Study	Characteristics	Findings
Bates et al.[268-270]	Comparison of symptom prevalence, work absences, and ventilatory function in Canadian veterans residing in 4 Canadian cities	Lower prevalence of symptoms and work absences and better ventilatory function in veterans living in the lest polluted city
Bates[271]	10-year follow-up study of Canadian veterans initially evaluated in 1960, and followed at yearly intervals with pulmonary function tests and clinical evaluations	Least decline in pulmonary function with age in veterans from least polluted city
Yashizo[272]	Bronchitis survey of 7 areas of Osaka, Japan, 1966, among adults 40 years of age and over	Bronchitis rates, standardized for sex, age, and smoking were greater among men and women in the more polluted areas. Bronchitis rates followed the air pollution gradient.
Winkelstein and Kantor[273]	Survey of respiratory symptoms in a random sample of white women in Buffalo, New York	In nonsmokers 45 years of age and over, and among smokers who did not change residence, respiratory symptoms were correlated with particulate concentrations obtained in the neighborhood of residence. No association of symptom prevalence with SO_2 concentrations
Ishikawa et al.[275]	Comparison of lungs obtained at autopsy from residents of St. Louis and Winnipeg	Autopsy sets, matched for age, sex and race, showed more emphysema in the more polluted city. Autopsied groups may not reflect prevalence of disease in general population
Fujita et al.[276]	Prevalence survey (Medical Research Council questionnaire) of post office employees in Tokyo and adjacent areas, 1962 and re-surveyed in 1967	Two-fold increase over time in prevalence of cough and sputum production in same persons, irrespective of smoking habits. Change was attributed to increasing degrees of air pollution

TABLE 1-17. QUALITATIVE STUDIES OF AIR POLLUTION AND PREVALENCE OF CHRONIC RESPIRATORY SYMPTOMS AND PULMONARY FUNCTION DECLINES

Study	Characteristics	Findings
Reichel,[277] Ulmer et al.[278]	Respiratory morbidity prevalence surveys of random samples of population in 3 areas of West Germany with different degrees of air pollution	No differences in respiratory morbidity, standardized for age, sex, smoking habits, and social conditions, between populations living in the different areas
Nobuhiro et al.[279]	Chronic respiratory symptom survey of high and low exposure areas of Osaka and Ako City, Japan	Higher prevalence of chronic respiratory symptoms in more polluted areas
Comstock et al.[280]	Repeat survey in 1968/1969 of east coast telephone workers and of telephone workers in Tokyo	After adjustment for age and smoking, no significant association of respiratory symptom prevalence with place of residence
Speizer and Ferris[281-282]	Comparison of respiratory symptoms and ventilatory function in central city and suburban Boston traffic pollicemen	Slight but insignificant increase in symptoms prevalence among non-smokers and smokers, but not exsmokers, from the central city group. No group differences in ventilatory function
Linn et al.[283]	Respiratory symptoms and function in office working population in Los Angeles and San Francisco, 1973	No significant difference in chronic respiratory symptom prevalence between cities; women in the more polluted community more often reported nonpersistent (<2 years) production of cough and sputum
Prindle et al.[284]	Comparison of respiratory disease and lung function in residents of Seward and New Florence, PA	Increased airway resistance in inhabitants of more polluted community. Differences in occupation, smoking, and socioeconomic level could account for these differences

TABLE 1-17. QUALITATIVE STUDIES OF AIR POLLUTION AND PREVALENCE OF CHRONIC RESPIRATORY SYMPTOMS AND PULMONARY FUNCTION DECLINES

Study	Characteristics	Findings
Watanabe[285]	Peak flow rates in Japanese school children residing in Osaka	Lower peak flow rates in children from more polluted communities. Improved peak flow rates when air pollution levels decreased
Anderson and Larsen[286]	Peak flow rates and school absence rates in children 6-7 years of age from 3 towns in British Columbia	Significant decrease in peak flow rates in 2 towns affected by Kraft pulp mill emissions. No effect on school absences. Ethnic differences were not studied
Collins et al.[287]	Death rates in children 0-14 years of age, 1958-1964, in relation to social and air pollution indices in 83 county boroughs of England and Wales	Partial correlation analysis suggested that indices of domestic and industrial pollution account for a greater part of the area differences in mortality from bronchopneumonia and all respiratory diseases among children 0-1 year of age

TABLE 1-18 SUMMARY TABLE - CHRONIC EXPOSURE EFFECTS

Type of Study	Reference	Effects observed	Annual average pollutant levels at which effect occurred	
			TSP ($\mu g/m^3$)	SO_2 ($\mu g/m^3$)
Mortality (geog.)	Winkelstein[188]	Increased mortality	125-140	not significant
	Zeidberg and colleagues[16-18]	Increased mortality	55-60	30
Morbidity				
Longitudinal and cross-sectional	Ferris et al.[41 42 46 47]	Higher rate of respiratory symptoms; and decreased lung function	180	55
Cross-sectional (2 areas)	Sawicki (1972)[31]	More chronic bronchitis, asthmatic disease in smokers; reduced FEV%	250*	125
Cross-sectional study of school-children in 4 areas	Lunn et al.[96 97]	Increased frequency of respiratory symptoms; decreased lung function in 5-year olds	260*	190
Follow-up of school-children in 4 areas	Douglas and Waller[90]	Increased lower respiratory tract infection	197* (130 BS)	130
Cross-sectional study of children in 4 areas	Hammer et al.[214]	Increased incidence of lower respiratory diseases	85-110	175-250
Cross-sectional study of high school children in 2 areas	Mostardi and colleagues[177 258]	Lower FVC, $FEV_{0.75}$ and maximal oxygen consumption	77-109	96-100
Cross-sectional (multiple areas)	Lambert and Reid[28]	Increased respiratory symptoms	160* (100 BS)	100-150
Cross-sectional (3 areas)	Goldberg et al.[109]	Increased CRD	78-82	69-160

TABLE 1-18 (continued)

Type of Study	Reference	Effects observed	Annual average pollutant levels at which effect occurred TSP (μg/m³)	SO_2 (μg/m³)
Cross-sectional (4 areas)	House et al.[108]	Increased CRD	70 (15SS)	100-150
Cross-sectional and Long (2 areas)	Sawicki and Lawrence (1977)[181]	Increased Prev CB and AS Increased persistance, Males 31-50; Increased incidence, Females, some ages	169+	114-130
Cross-sectional (3 areas)	Rudnick[182]	Increased respiratory symptoms in boys. Increased Rh in girls	221-316* (150-227 BS)	108-148
Cross-sectional and retro-long in 4 areas (children)	Nelson et al.[114]	Increased LRD	70	107
Cross-sectional 2 areas	Hammer[113 257]	Increased LRD	133 (SS=14)	<25
Cross-sectional 3 areas (children)	Shy et al.[215]	Decreased adjusted $FEV_{.75}$ in children > 8 years	78-82	69-160
Cross-sectional 2 areas (children)	Shy et al.[215] Chapman et al.[213]	Decreased adjusted $FEV_{.75}$	96-114 (45 RSP)	(= and low)

*Converted from BS (British Smoke).
**Converted from CoH.

for example, reported increased symptoms in patients with 24-hour averages of 12 μg/m^3 SS (200 TSP, 60 RSP, 8 SN, 100 SO_2). Also, Chapman et al.[212] reported increased chronic respiratory disease prevalence rates in a high pollution community with an annual average of 15 μg/m^3 SS (70 TSP and 107 SO_2). Hammer[257] further reported increased lower respiratory disease prevalence rates in a high pollution community with an annual average of 14 μg/m^3 SS (133 TSP and SO_2 >17). Thus, suspended sulfate levels of 12 μg/m^3 (daily) or more and 15 μg/m^3 (annually) might be interpreted as being important based on those results; however, certain methodological considerations discussed in Chapter 14 must be taken into account in qualifying these results.

Respirable Particulates Effects--As discussed in Chapter 11, particles below 15 μg/m^3 MMAD are important. Respirable suspended particulates (RSP) $\leq$10 μm, have been measured in only a few American epidemiology studies, e.g., those by Hammer,[113,257] Stebbings and Hayes,[190] and Shy and Chapman et al.[213,215] The later study was reported as demonstrating decreased adjusted $FEV_{.75}$ in children in an area with higher pollution with RSP of 45 μg/m^3 (96 to 114 μg/m^3 TSP and SO_2 very low). Thus RSP of 45+ μg/m^3 may be important but, again, acceptance of these results must be qualified based on certain methodological considerations discussed in Chapter 14.

1.5.5.2 Methodological Factors Impacting Interpretation of Results--If it were assumed that all of the results summarized in Tables 1-16 and 1-18 were derived from methodologically sound studies and were universally accepted as valid, then the above summary of their results could be accepted as a reasonable representation of the likely atmospheric particulate and sulfur oxides levels found to be associated with mortality and morbidity effects. However, the matter of the methodological soundness and validity of various studies has

been a matter of considerable controversy and discussion during the past decade.

Such controversy has derived, in large part, from the fact that certain additional risk factors can often be as important as the air pollution variables studied in affecting human health. For example, as was alluded to earlier and discussed more thoroughly in Chapter 14 (Sections 14.1, 14.3, 14.4), it has been strongly emphasized that smoking is one such factor, as are occupational exposures. Furthermore, age and sex co-variables can also be critical in the evaluation of health effects. Race or ethnic group characteristics likely fall into this catagory as well. In addition, numerous social variables may be highly critical in terms of their existing direct effects on human health, as well as how they may modify the health effects of environmental pollutants. Such social factors include social economic status (income, education, and occupational levels and associated social class status), migration, and household characteristics. Also, meterological variables such as sudden temperature changes or shifts in humidity levels may also be critical co-variables which, along with air pollutants, might affect health in a deleterious manner. Parental smoking and other sources of indoor pollutants may also be critical. Other less-well defined social/ environmental variables, such as a greater degree of crowding in housing conditions, too, may represent a set of "urban factors" differentially acting to affect health in comparison to "rural" conditions.

Many of the studies have attempted to control for at least some of the above and other factors. Most of the studies analyzed, however, have not controlled for all possible confounding or covarying factors, an extremely difficult task in practice. Nor have all been able to exquisitely control for

what factors were taken into account. In fact, there is not a single study that has controlled for everything that might have affected its results. Thus, the likely validity of each study has to be appraised after evaluating the importance of possible confounding variables and co-variables controlled for or not taken into account by that study. In many cases, known highly correlated variables have been controlled for or taken into account and, in other cases, comparison study groups have been chosen so as to be similar in terms of crucial characteristics, making it relatively easy to ascribe likely validity to their observed results.

In regard to evaluating other (less well-designed) studies, it should be noted that some studies exist which indicate that possible confounding variables are not always as important as they were originally thought to be. For example, follow-up studies on an adult cohort previously studied as children by one group of investigators did not confirm original social class differences between the groups to be of much significance in accounting for health findings for the groups later in life, and other studies have shown that household/familial factors are not necessarily important in all cases in accounting for observed results. Therefore, care must be taken not to over-emphasize the relative importance of potential confounding or covarying factors not ruled out as possible alternative explanations for the results of a given study. In other words, being overly critical where information is lacking to support the likelihood of a specific confounding factor or co-variable affecting the pattern of results obtained in a study at a particular time represents as much of a disservice in trying to achieve an objective, balanced appraisal of study results under discussion as would any countervailing lack of reasonable regard for the potential importance of such factors.

It must also be recognized that no single study alone, no matter how well-designed or conducted in and of itself completely establishes what comes to be accepted as a "scientific fact" defining either a relationship between two or more variables studied or a lack thereof. Rather, excellence in the design and conduction of a given study, internal consistency and biological plausibility of its results, and their consistency with other known results or information all help to heighten confidence in the likely existence of relationships indicated by that study's results. Even greater certainty is attributed to the probable existence of such relationships if further independent studies, regardless of particular individual flaws, yield results consistent with such relationships. Thus, consistency in the overall pattern of results indicative of particular relationships or the overall "weight of the evidence" from more than one study are crucial in establishing given relationships as "scientific facts" or in determining the degree of certainty ascribed to them.

1.5.5.3 Quantitative Dose-Response Relationships Defined by Community Health Studies--In order to elucidate dose-response relationships established by community health epidemiology studies of the type reviewed above, numerous attempts besides the present one have been made to examine both negative and positive information concerning such studies. This has usually been done to determine which are sufficiently sound methodologically to allow for reasonable conclusions to be drawn from them in evaluating the overall meaning of their results individually and collectively. Such attempts include critical reviews and commentaries written by Rall (1974),[245] Higgins et al. (1974),[248] Goldsmith and Friberg (1977),[247] Ferris (1978),[314a] and Waller (1978).[314b] They also include the following evaluative documents appearing in 1978: an American Thoraic Society (ATS) review of Health Effects of Air Pollution (Shy et al.,

1978);[251] a National Research Council/National Academy of Science (NRC/NAS) document on Airborne Particles by Higgins and Ferris (1978)[307] and an NRC/NAS document on Sulfur Oxides by Speizer and Ferris (1978)[308] More recent such reviews and commentary appearing in 1979 include: the 1979 World Health Organization (WHO) document, Environmental Health (8): Sulfur Oxides and Suspended Particulate Matter;[312] a report by Holland et al. (1979)[301] written for the American Iron and Steel Institute and appearing in the American Journal of Epidemiology; and a reply to that report in the same journal by Shy (1979).[313] Some of the more salient points of these reviews and commentaries are concisely highlighted below.

As will quickly become apparent through the course of the discussion below, there are certain studies that many reviews consistently rate as being methodologically sound and their results valid. Also, when those study results are viewed together, collectively, fairly consistent patterns of quantitative relationships emarge regarding exposures to sulfur oxides and particulate matter associated with the occurence of various types of health effects, including (1) mortality and morbidity effects associated with acute exposures to fairly high ranges of air concentrations of those substances and (2) morbidity effects associated with chronic exposures to lower atmospheric levels of the same agents. Given the general concensus that appears to exist regarding the validity of these studies, then, there seems to exist very good support for placing considerable confidence in the overall patterns of quantitative relationships defined by their collective evaluations.

In regard to other reasonably well-designed studies, but for which less of a concensus exists regarding their likely validity, several interesting points emerge from the subsequent discussion. First, it becomes apparent

that, beyond some small modicum of agreement among the reviews concerning problems associated with certain studies, the various reviews often differ considerably in regard to their assessments of the methodological soundness or validity of any given individual study. This derives mainly from different reviewers emphasizing or citing different possible confounding or covarying factors as potentially being important in affecting the results of a given study. However, despite whatever flaws might be evaluated by different reviewers to be associated with the particular individual studies, a surprisingly great degree of consistency exists both between most of the "flawed" study results and, also, in comparison with the findings of the other studies alluded to above as being widely recognized as being valid. In some cases, however, the results of some of the supposedly "flawed" studies point toward still lower levels of sulfur oxides and particulate matter being associated with significant mortality or morbidity effects. Thus, whereas not as much confidence can yet be placed in such findings as those from the more universally accepted studies, it is still not appropriate scientifically to completely disregard or ignore them. This is especially true in view of the fact that, all too often, relationships indicated to exist by "suggestive" evidence derived from numerous "flawed" studies are later confirmed by more carefully designed and conducted "definitive" studies.

Tables 1-19 to 1-21 summarize conclusions regarding particular study results based on critical assessments contained in Chapter 14 of this document and assessments from other reviews of observational studies of the health effects of sulfur dioxide, expressed in $\mu g/m^3$ of SO_2, and particulate matter, expressed in terms of $\mu g/m^3$ of total suspended particulates (TSP). When exposures were originally obtained in units of black smoke or coefficient of

haze, they have been converted to TSP for these tables. The conversion relationships are discussed in Chapters 3 and 14. Because information bearing on the health effects of atmospheric sulfates and the fine fraction of particulate matter is insufficient to separately assess the health effects of these fractions of atmospheric particulates, these measures of pollution level are not considered in these tables. These issues are discussed in more detail in Chapter 14.

Each row in Tables 1-19 to 1-21 corresponds to an observational study that has been cited in at least one review. These studies are grouped according to whether acute effects were studied, that is, effects associated with fluctuations in 24-hour average level, or chronic effects were studied, that is, differences in effects associated with differences in annual average level. Within each exposure category, studies showing mortality effects are listed separately from those showing morbidity effects. The geographic location(s) of the populations studied and approximate dates or time periods covered by each study are given.

Each column corresponds to one of the particular reviews referenced and discussed in Chapter 14; and the entries are the air concentrations of of SO_2 and TSP interpreted to be associated with health effects demonstrated by the different studies listed. The absence of stated quantitative values for TSP/SO_2 for a given study does not necessarily mean that it was judged to be an inadequate study by the particular reviewer(s); rather, in some cases, either no levels were clearly stated in their review or the particular study may not have been considered (some studies, for example, have appeared after the publication of several of the reviews).

TABLE 1-19. SUMMARY OF VARIOUS REVIEWERS' EVALUATIONS OF QUANTITATIVE DOSE-RESPONSE RELATIONSHIPS DERIVED FROM STUDIES OF MORTALITY EFFECTS ASSOCIATED WITH ACUTE EXPOSURES TO SO_2 AND PARTICULATE MATTER

Study	Date	Population	Rall (1974)	Higgins (1974)	Goldsmith & Friberg (1977)	Ferris (1978)	NAS SO_2 (1978)	NAS TSP (1978)	WHO (1979)	Holland et al. (1979)	Shy (1979)	Ware et al. (1980)	EPA Ch 14 (1980)
Scott/Burgess/ Gore	(1954-56)	London	2000/1144 *	2000/1000				2000/1040		1000/750			2000/1000
Lawther[13]	(1958-59)	London	750/700		750/710			750/710					
Glasser & Greenburg[222]	(1960-64)	New York						291/520	750/710			580/780	330/524
Martin & Bradley[11]	(1958-59)	London			750/710							530/300	500/300
Martin[6]	(1959-60)	London		1000/500			417BS/277		500/500	750/700		500/400	500/400
U. K. Ministry of Pensions	(1952)	U. K.							>1000/>1000				
McCarroll et al.	(1962-64)	New York	720/1500	720/1500						800/1450			570/1500
Greenburg et al.[191]	(1953-64)	New York						720/500		>570/850+			570/1000
Beuchley	(1962-66)	New York			-/500	-/300	-/300		-/500				-/500
Watanabe & Kaneko[228]	(1965-66)	Osaka											300/266
Composite Dutch	(1960s)	Rotterdam											300/500
Composite British	(1955-62)	Large U.K. cities											250/250
Composite USA	(1952-64)	Large U.S. cities											570/1000
Appling et al. & Waller	(1975)	London								546/994			500/780
Riggan et al.	(1975)	Pittsburgh											700/300

(+) Judged to be methodologically sound/valid study.
(-) Judged to be methodologically flawed/invalid study.
(?) Judged to be methodologically sound, but reservations expressed.

* Each entry represents TSP/SO_2 levels in $\mu g/m^3$ as reported by given reviewer. Exceptions: BS=British smoke shade, CoH= coefficient of haze.

TABLE 1-20. SUMMARY OF VARIOUS REVIEWERS' EVALUATIONS OF QUANTITATIVE DOSE-RESPONSE RELATIONSHIPS DERIVED FROM STUDIES OF MORBIDITY EFFECTS ASSOCIATED WITH ACUTE EXPOSURES TO SO_2 AND PARTICULATE MATTER

Study	Date	Population	Rall (1974)	Higgins (1974)	Goldsmith & Friberg (1977)	Ferris (1978)	NAS SO_x (1978)	NAS TSP (1978)	WHO (1979)	Holland et al. (1979)	Shy (1979)	Ware et al. (1980)	EPA Ch 14 (1980)
Greenberg[196]	1953	New York						3 CoH/700 *					350/715
Lawther[52]	1954	London											350-529/3-450
Lawther[53]	1954-68	London			250/250-500	350/250	350/250	500/500	250 BS/500	350/500		312/500	340/3-500
Martin[16]	1959-60	London					516/340					500/400	500/400
Waller[7]	1961-64	London		250/500						350/500			
Angel[59]/ Fletcher[274]	1962-63	London		200/250		250/250	230/250	200/400		(?)			
McCarrol[205]	1963-65	New York											160/372
Cassell[208,209]	1964-67	New York											205/452
Lawther as per Goldsmith[90]	1964-65	London			129/264					(+)			
Lawther as per Goldsmith[90]	1966-68	London			68/204					(+)			
Carnow[174]	1968	Chicago						-/700					-/700
Cohen[36]	1968-69	West Virginia				150/200	150/200		150/200	(-)			150/200
Vander Lende[74]	1969	Netherlands				230/300	245/300		140 BS/300	(?)	160 BS/-	245/300	200/300
Hammer[214] Chapman[212]	1969-71	New York						145/286		(-)			
Hammer[113,257]	1969-71	Birmingham (S.E. USA)						180/26		(-) (?)	200 BS/200		
Gervois[61]	1970s	France								(?)			700/300
Stebbings[216]	1975	Pittsburgh											

(+) Judged to be methodologically sound/valid study.
(-) Judged to be methodologically flawed/invalid study.
(?) Judged to be methodologically sound, but reservations expressed.

* Each entry represents TSP/SO_2 levels in $\mu g/m^3$ as reported by given reviewer. Exceptions: BS=British smoke shade, CoH= coefficient of haze.

TABLE 1-21. SUMMARY OF VARIOUS REVIEWERS' EVALUATIONS OF QUANTITATIVE DOSE-RESPONSE RELATIONSHIPS DERIVED FROM STUDIES OF MORBIDITY EFFECTS ASSOCIATED WITH CHRONIC EXPOSURES TO SO_2 AND PARTICULATE MATTER

Study	Date	Population	Rall (1974)	Higgins (1974)	Goldsmith & Friberg (1977)	Ferris (1978)	NAS SO_2 (1978)	NAS TSP (1978)	WHO (1979)	Holland et al. (1979)	Shy (1979)	Ware et al. (1980)	EPA Ch 14 (1980)
U.K. Ministry of Pensions		Britain		200/200*				>100/>100		(-)			
Douglas & Waller[90]	1946-65	Britain		70/90	140/130	230/120	230/120	70/90	140BS/140	(?)		230/130	
Lambert & Reid[28]	1966-68	Britain		100/100	100-200/100-200			100/100		(-)	1-200BS/-		
Lunn et al.[96,97]	1963-65	Britain				180/120	180/120	100/120	200 BS/200	330/180		260/190	
Ferris[43]	1966-67	Berlin, N.H.				180/55[a]	180/55	180/73	180/-	(?)	180/-	180/55	180/55+
Fletcher[274]	1950s-65	London			420 → 100/260	250/250	250/250			(-)			
Sawicki[181]	1966-73	Cracow				240/130	250/125		170/125	(?)	170/-	250/125	240/114
Mostardi[258]	1968	Ohio				93/98	110/110			(-)		77-109/100	
Hammer[214]	1972	New York				85-195/50-450	100/200			(-)			85-110/175-250
Shy[215]	1968	Cincinnati, OH								(-)			96-114/<20
Holland/Bennett[101-103]	1966-67	Kent, Eng.			70BS/-					(?)	<100BS/-		
Tessier[322]	1971-74	France								(?)	<100BS/-		
Colley & Reid	1966	Britain			-/100				-/150	(+)			
Suzuki & Hitosugi[312]	1970	Tokyo							1-200/60-140				
Chapman[212]	1971-72	Birmingham, AL (S.E. USA)					110/23			(-)			
Irving et al.[98]	1972-73	Britain								(?)			
Rudnick[182]	1972-74	Poland											240-340/150-180
French[306]	1969-71	Chicago								(-)			150-165/106-250
Hammer[113,257]	1969-71	Birmingham, AL (S.E. USA)								(-)			100-133/<20

(+) Judged to be methodologically sound/valid study.
(-) Judged to be methodologically flawed/invalid study.
(?) Judged to be methodologically sound, but reservations expressed.

* Each entry represents TSP/SO_2 levels in $\mu g/m^3$ as reported by given reviewer. Exceptions: BS=British smoke shade, CoH= coefficient of haze.

One very notable feature of all three tables is the variety of levels cited for the same studies by different reviewers. When one considers the continuous relationship between exposure level and response seen in many of these studies (for example, the studies by Martin discussed in detail in Chapter 14), this variation in cited levels can be attributed largely to the lack of a clear threshold level for effects being defined by these studies. The other important feature of the table is the variation among reviewers in the choice of studies considered to have demonstrated health effects. The next sections are devoted to a discussion of these differences in interpretation among reviewers and to a discussion of the studies which bear critically on differences in conclusions drawn in these various reviews.

Acute Mortality

Examination of Table 1-19 reveals that considerable agreement exists to the effect that episodic mortality has occurred definitely above levels of 750 $\mu g/m^3$ TSP and SO_2[13,247,307,312], in London. Holland et al.[301] concluded, mostly from British studies, that the critical values were 500 $\mu g/m^3$ TSP and 700 $\mu g/m^3$ SO_2. Interpretation of daily mortality by Martin et al.[6,11] indicate the effective levels in London could be as low as 500 $\mu g/m^3$ TSP and 300 to 400 $\mu g/m^3$ SO_2.[304,312] Also, new analyses contained in Chapter 14 suggest that the TSP effect levels may be as low as 200-400 $\mu g/m^3$ in the absence of significant temperature or other confounding effects. Similar studies by Glasser et al.[222] in New York City would indicate that the levels where mortality has been seen there could be 2.5 to 5 CoHs (190 to 580 $\mu g/m^3$ TSP) and 520 $\mu g/m^3$ SO_2.[307] This is not all that different from results found for London, but different reviewers may interpret these results somewhat differently. WHO concluded that levels above 500 $\mu g/m^3$ of each (SO_2 and particulate matter

expressed as BS) could definitely produce increased mortality.[312] On the other hand, some studies in the Netherlands and in Japan might indicate that such mortality increases could occur at levels of TSP around 300 μg/m^3 with SO_2 at 500 μg/m^3 or above.[100,232,302] All of these effects are tempered by the particular meteorological conditions present during the study period and whether only central pollution monitoring ~~states~~ sites or multiple numbers of geographically representative sites were used in determining the stated levels of SO_2 and particulate matter. All studies showed mortality effects to occur predominantly in the infirm, the elderly, and infants.

There is more certainty of effects occurring when either TSP or SO_2 are at or above 500 μg/m^3. Mortality effects may be less certain at levels of TSP between 300 and 500 μg/m^3 at levels of SO_2 of 500 μg/m^3.

Acute Morbidity

There is a broader range of opinion, and estimations of effect levels, associated with the studies of acute morbidity. The earlier studies by Lawther, Waller et al. of pulmonary function and of exacerbation in bronchitics show various effects when the SO_2 and BS levels were higher in London. Most reviewers agree that these effects occurred when TSP levels were 250 to 350 μg/m^3 and SO_2 levels were 250 to 500 μg/m^3. As the levels decreased, the acute effects decreased. By the winter of 1964-65, the exacerbations were slightly reduced and less consistent; daily average BS was 129 μg/m^3 (196 TSP) and daily average SO_2 was 264 μg/m^3 in that winter. Even during the winter of 1967-68, although the effects had decreased further, they were significantly correlated with pollutant concentrations; daily average BS was 68 μg/m^3 (121 TSP) and daily average SO_2 was 204 μg/m^3 during that winter. The Greenberg et al. studies in New York City in the 1960s had also shown increased morbidity during episodes

at levels above 300 μg/m^3 TSP and 700 μg/m^3 SO_2. WHO[312] concluded that daily levels above 250 μg/m^3 of SO_2 and BS (~330 TSP) would produce acute effects. However, several studies have shown effects at levels around or below those values. Studies of daily acute respiratory disease symptoms in New York City families, which included statistical analyses or controls for possible confounders, showed increased daily symptoms at levels of 160 to 205 μg/m^3 TSP and 370 to 450 μg/m^3 SO_2.[205,208,209] However, these were only average values over the winters of the study. Studies of prevalence rates of acute respiratory symptoms children in New York City, controlling for confounders indicated increased rates associated with levels of 145 μg/m^3 TSP and 286 μg/m^3 SO_2.[212,214,307] A similar study in Birmingham showed increased rates at levels of 180 to 220 μg/m^3 TSP and 26 μg/m^3 SO_2.[257,307]

Probably one of the most critical ~~study~~ studies to be considered is that of Cohen et al.[36] They studied 20 asthmatics around a coal-fired power plant. They found temperature was most important, but within temperature ranges, pollution also caused asthma attacks. The levels cited were 150 μg/m^3 TSP and 200 μg/m^3 SO_2, both daily averages. Various reviews have considered this study weak,[304,312,314b,] while others consider it a significant demonstration of competition of environmental factors.[251] After temperature and one pollutant were removed, none of the other pollutants entered into the relationship; thus, no single pollutant could be considered solely responsible for the increased attack rates.

On the basis of the above studies, one could conclude that acute morbidity could be seen at levels between 145 to 220 μg/m^3 TSP and approximately 200 to 400 μg/m^3 SO_x.

Chronic Morbidity

The studies summarized under the section of chronic morbidity include a mixture of investigations carried out in population samples composed of random samples of adults, and adults selected from working groups and populations of children selected by areas of residence. In addition, all but a few of the studies use a cross sectional method, that is, they examine the population in question at one point in time and determine the prevalence of whatever morbid condition is being assessed. Alternatively, a few of the investigations follow a group of people over a period of time. Although in these studies the opportunity to measure the onset of new morbid events exists, eventually all of the studies actually measure the prevalence of conditions at different points in time and cannot be used to determine the incidence of new conditions related to exposure to air pollutants. The morbid events in each study generally relate to the prevalence of chronic respiratory symptoms or disease states as ascertained by standardized questionnaires and/or levels or changes in levels of pulmonary function measured by generally reliable equipment.

The variation in reported levels of effective exposures from the reviews which indicate the same studies as providing reliable information probably reflect one or more of the various problems of with aerometry or health measurement errors encountered in this observational assessments (see above). The inclusion (or exclusion) of any given study in any particular review in part reflects the interpretation by the reviewers of the validity (reliability?) of the measurement of exposure or health outcomes in that particular study.

Except for the Holland report which found only one group of acceptable studies which provided information on chronic morbidity,[96,97] all of the other reviews found several studies with which to provide estimates of lowest levels

of health effects. In general, there is a range of agreement between the reviewers that health effects are measurable at levels of TSP above 180 $\mu g/m^3$ generally in conjunction with levels of SO_2 above 120 $\mu g/m^3$. There are notable exceptions in which lower levels are reported to be associated with health effects. For example, Ferris[43] reports TSP levels of 180 $\mu g/m^3$ in association with SO_2 levels of 55 $\mu g/m^3$, however, the SO_2 measures were made from 30-day sulfation rates and may not reflect considerably higher peak exposures which may have occurred.

More striking in indicating a divergence of levels is the study of Mostardi[258] and certain other studies reviewed in Chapter 14. Mostardi indicates effects at levels of both TSP and SO_2 approximately 100 $\mu g/m^3$, however, some of the reviewers rejected his findings because he did not analyze his data on pulmonary function in adolescents by race in spite of having mentioned that there were some blacks who would be expected to have lower levels of pulmonary function in the high exposure group.

The remaining studies were generally part of EPA sponsored CHESS Program investigations which have received independent peer review and have been published in the open literature over the last several years. In these studies, increases in chronic morbidity have consistently been found where levels of TSP and/or SO_2 have exceeded 100 $\mu g/m^3$. Discussions of published critiques of shortcomings of a number of these studies are included in Chapters 3 and 14 of this document and, also, in some of the other reviews listed in Tables 1-19 to 1-21. Due to considerable controversy over the results and interpretation of many of the CHESS studies, many such quantitative findings regarding air pollution/health effects relationships need to be qualified to take into account, for example, certain air quality measurement errors that tended to

bias detected effect levels in a negative direction (i.e., toward somewhat lower levels than those suggested by proximally located local air sampling monitors.) Still, at least some of the CHESS studies, e.g., that by Hammer[113,257] on children in the Southeastern United States (which was approved as a Harvard doctoral dissertation), appear to be methodologically sound, their results well-analyzed statistically and accurately interpreted, and likely valid. However, in the absence of much, if any, comment on some such studies in the published reviews listed in Tables 1-19 to 1-24, it is difficult to assign the same level of certainty to their reported effect levels as those indicated by results from others more widely critiqued to date and agreed upon as being valid. Nevertheless, it should be noted that many of the quantitative results derived from such studies, especially when known or suspected errors in aerometry are taken into account, are not badly divergent from the results obtained in some number of other studies.

The quantitative relationships defined by many of the studies listed in Tables 1-19 to 1-21 are depicted in Figure 1-4. Also provided in that figure are some indications of notable divergence of opinion between certain reviewers, especially Holland et al. (1979) versus the WHO (1979) appraisal, the present EPA (1980) evaluation and several other published appraisals. The acute and chronic exposure levels for SO_2 and particulate matter (BS translated to TSp) evaluated by the WHO (1979) to be associated with mortality and morbidity effects are indicated by dashed lines in Figure 1-4. Also, presented below are tables (Tables 1-22 to 1-24) summarizing the conclusions of the WHO (1979) regarding levels at which mortality and morbidity effects can be expected to occur and their recommended guidelines for exposure limits consistent with the protection of human health.

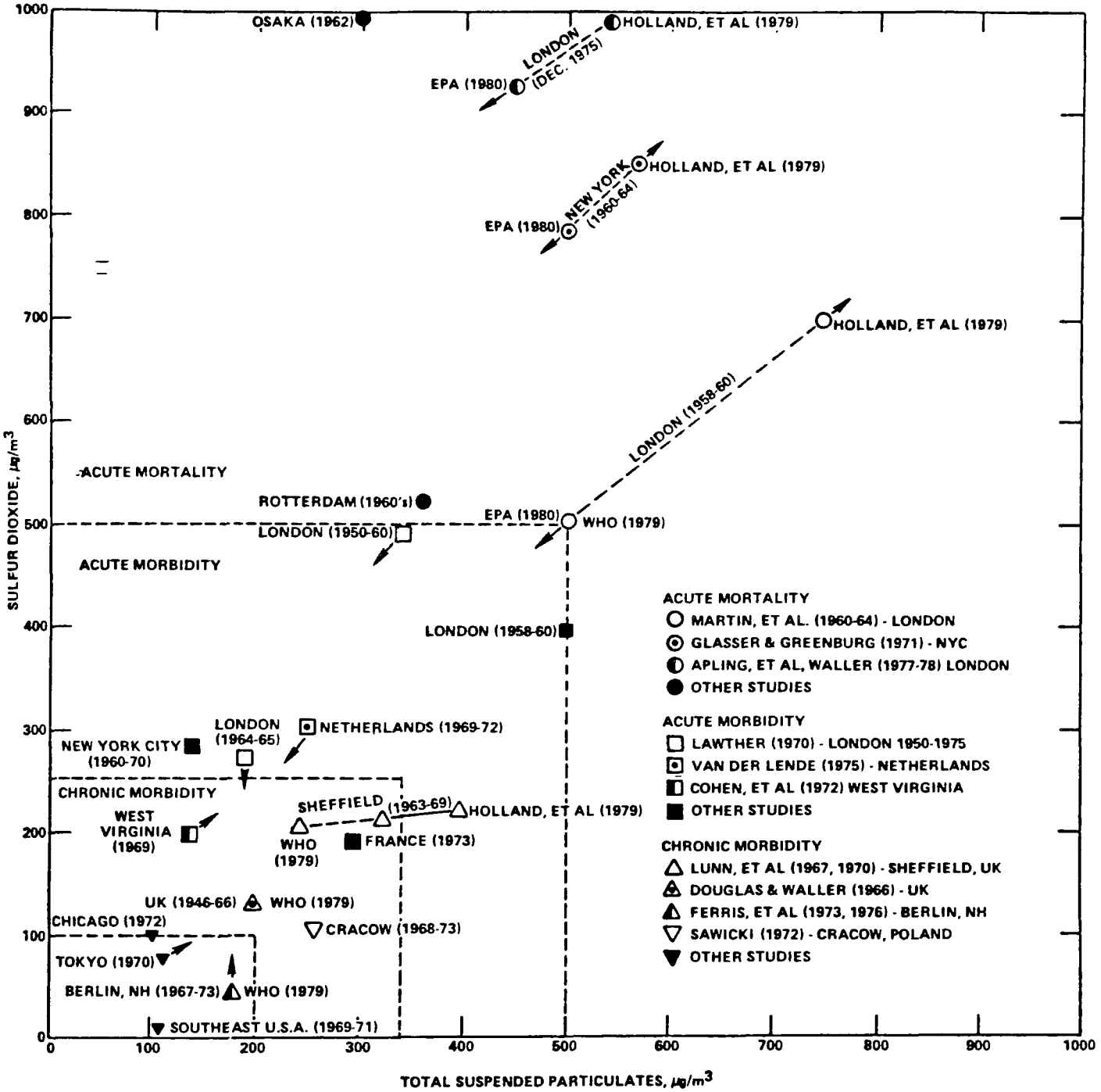

Figure 1-4. Comparison of interpretations of studies evaluated by Holland et al. (1979),[301] WHO (1979),[312] or other reviews such as those in the NRC/NAS documents[307,308] and the present chapter. Aside from the British studies noted for London and Sheffield and the 1960-64 New Youk City mortality study, Holland et al.[301] either ignored the other studies shown or evaluated them as being invalid based on methodological flaws or reinterpretation of their findings. "OTHER STUDIES" not specifically identified in the above key include those reported by: Gervois et al.[61] □ France (1973); Martin[16] □ London (1958-60); Mostardi et al.[117,258] ∇ Chicago (1972); Hammer[113,257] ∇ Southeast USA (1969-71); Suzuki and Hitosugi ∇ Tokyo (1970). The dashed lines depict WHO (1979)[312] conclusions regarding SO_2 and particulate levels associated with acute (24-hr) mortality, acute morbidity, and chronic (annual) morbidity.

TABLE 1-22. EXPECTED EFFECTS OF AIR POLLUTANTS ON HEALTH IN SELECTED SEGMENTS OF THE POPULATION: EFFECTS OF SHORT-TERM EXPOSURES[a]*

Expected effects	24-h mean concentration ($\mu g/m^3$)	
	Sulfur dioxide	Smoke
Excess mortality among the elderly or the chronically sick	500	500
Worsening of the condition of patients with existing respiratory disease	250	250

[a]Concentrations of sulfur dioxide and smoke as measured by OECD or British daily smoke/sulfur dioxide method (Ministry of Technology, UK, 1966; Organization for Economic Cooperation and Development, 1965). These values may have to be adjusted in terms of measurements made by other procedures.

*From WHO 1979 Criteria Document for Sulfur Oxides and Particulate Matter.[312]

TABLE 1-23. EXPECTED EFFECTS OF AIR POLLUTANTS ON HEALTH IN SELECTED SEGMENTS OF THE POPULATION: EFFECTS OF LONG-TERM EXPOSURES[a]*

Expected effects	Annual mean concentration ($\mu g/m^3$)	
	Sulfur dioxide	Smoke
Increased respiratory symptoms among samples of the general population (adults and children) and increased frequencies of respiratory illnesses among children	100	100

[a]Concentrations of sulfur dioxide and smoke as measured by OECD or British daily smoke/sulfur dioxide method (Ministry of Technology, UK, 1966; Organization for Economic Cooperation and Development, 1965). These values may have to be adjusted in terms of measurements made by other procedures.[312]

*From WHO 1979 Criteria Document for Sulfur Oxides and Particulate Matter.

TABLE 1-24. WORLD HEALTH ORGANIZATION GUIDELINES FOR EXPOSURE LIMITS CONSISTENT WITH THE PROTECTION OF PUBLIC HEALTH[a,*]

Expected effects	Concentration ($\mu g/m^3$)	
	Sulfur dioxide	Smoke
24-h mean	100-150	100-150
Annual arithmetic mean	40-60	40-60

[a]Values for sulfur dioxide and smoke as measured by OECD or British daily smoke/sulfur dioxide method (Ministry of Technology, UK, 1966; Organization for Economic Cooperation and Development, 1965). Adjustments may be necessary where measurements are made by other methods. ~~For example, smoke concentrations of 100-150 $\mu g/m^3$ convert to approximately 200-300 $\mu g/m^3$ TSP and smoke levels of 40-60 $\mu g/m^3$ convert to approximately 80-120 $\mu g/m^3$ TSP.~~

*From WHO[312]

Chapter 1 Introduction, Summary, and Conclusions SO_x/PM

Corrigenda

Before listing specific errata (deletions/insertions) for Chapter 1 (Volume I) of the April, 1980, External Review Draft of the EPA criteria document for sulfur oxides and particulate matter, certain general comments should be noted reguarding anticipated revisions in Chapter 1.

First, major revisions planned to be made in later current chapters (2-14) of the document, as indicated in ensuing corrigenda materials, will also be appropriately reflected in revisions to be made in Chapter 1. For example, certain major revisions in the text of Chapter 3 noted in corrigenda comments for that chapter will be appropriately reflected in revision of text in Section 1.3.2 (pg. 1-19 to 1-43). This especially includes introductory materials (4 main points) to be inserted on pg. 3-84 at the start of the discussion of comparison of particulate matter measurement techniques, as noted later in corrigenda comments for Chapter 3. Similarly, revisions noted in those corregenda comments to be made in Chapter 3 regarding the discussions of specific studies comparing COH versus TSP and BS versus TSP measurement results will be appropriately reflected in Chapter 1 revisions.

Other major revisions in Chapter 14, noted in the later corrigenda comments for that chapter, will also be reflected in revisions of Section 1.5.5 (Community Health Observation Studies) of Chapter 1. Of particular importance are major changes to be made in Tables 1-19 to 1-21 (on pg. 1-140 to 1-42) and accompanying text regarding summarization of various expert reviewers' evaluations of key quantitative community health studies. Specific changes in those tables will include the following:

(1) In Table 1-19, deletion of all entries except those for studies by Lawther[13], Glasser and Greenburg[222], Martin and Bradley[11], and Martin[6].

(2) In Table 1-20, deletion of entries for all studies except those by Greenburg[196], Lawther[52,53], Martin[16], Waller[7], and Van der Lende[74].

(3) In Table 1-21, deletion of entries for all studies except those by Douglas and Waller[90], Lambert and Reid[28], Lunn et al[96,97], Ferris[43], Sawicki[181], Mostardi,[117,258] Shy[215], and Rudnick[182].

Discussion of tables 1-19 to 1-21, in the text on pg. 1-143 to 1-152 is to be revised such that comments on quantitive air quality levels associated with observed health effects will generally be in terms of the original (COH, BS, TSP) particulate matter measurement units employed in specific studies summarized in the table, except for comments on interpretative evaluations by particular expert reviewers that involved "translation" of COH or BS units into TSP units. Further evaluative comments are to be added on whether reasonable interconversions between COH, BS, and TSP measurement units can be made and, if so, in what manner and under what circumstances. The impact of such interconversion or lack of sound bases to do so on interpretation of the epidemiology data base for SO_x/PM will then be taken into account in text revisions more specifically delineating key conclusions based on the epidemiology literature.

The implications of those conclusions, and others based on information discussed in Chapters 11, 12, and 13, for development of health criteria for sulfur oxide and particulate matter are to be delineated in an integrative health summary and conclusions chapter still in the process of being prepared for addition to the document. Relevant text summarizing the most salient features of that chapter, once completed. is to be added as the final portion of Chapter 1 (Volume 1).