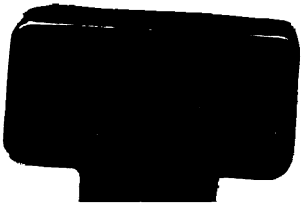
OZONE AND OTHER PHOTOCHEMICAL OXIDANTS



Medical and Biologic Effects of Environmental Pollutants

OZONE AND OTHER PHOTOCHEMICAL OXIDANTS

Committee on
Medical and Biologic Effects of
Environmental Pollutants

DIVISION OF MEDICAL SCIENCES
ASSEMBLY OF LIFE SCIENCES
NATIONAL RESEARCH COUNCIL

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This report has been reviewed by a group other than the authors according to procedures approved by a Report Review Committee consisting of members of the National Academy of Sciences, the National Academy of Engineering, and the Institute of Medicine.

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This document was written by the Subcommittee on Ozone and Other Photochemical Oxidants under the chairmanship of Dr. Sheldon K. Friedlander. The members of the subcommittee and its consultants were chosen for their competence to prepare sections of the report. The entire document was critically reviewed by the subcommittee, and it represents the combined effort and cooperation of all its members and consultants.

The authors of the individual sections were as follows: Dr. Kyle D. Bayes, chemical origin; Dr. Daniel Grosjean, aerosols; Dr. Alan Q. Eschenroeder, atmospheric concentrations of photochemical oxidants and models for predicting air quality; Dr. Peter K. Mueller, measurement and methods; Drs. Karl A. Bell and Bernard Altshuler, respiratory transport and absorption; Dr. Sheldon D. Murphy in collaboration with Drs. Bernard D. Goldstein and Margaret Hitchcock, toxicology; Dr. Jack D. Hackney, controlled studies on humans; Dr. James R. McCarroll, epidemiologic studies; Dr. Walter W. Heck in collaboration with Drs. John B. Mudd and Paul R. Miller, plants and microorganisms; Dr. Miller in collaboration with Dr. Marshall White, ecosystems; and Dr. James R. McNesby, effects of photochemical oxidants on materials. Dr. Friedlander prepared the executive summary.

The document was reviewed by the Report Review Committee of the National Academy of Sciences; by the parent Committee on Medical and

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Contents

OZONE AND OTHER PHOTOCHEMICAL OXIDANTS

1

Executive Summary

In the early 1950's, it was reported by Haagen-Smit[1] that many of the characteristics of photochemical smog could be explained by the presence of ozone and other photochemical oxidants. These substances, he believed, were formed in the atmosphere as a result of chemical reactions involving nitrogen oxides and hydrocarbons present in automobile exhaust. Significant quantities of nitrogen oxides were also emitted by power plants.

Considerable time elapsed before there was general acceptance of Haagen-Smit's important discovery, in part because of its subtle nature. For the first time, a major air pollution problem was demonstrated to be caused by a pollutant generated in the atmosphere. Its effect often did not become apparent until many miles downwind from the source. (The same suspicion has been attached to sulfate-containing aerosols for many years, but the proof that the sulfate is damaging is not as well established.) In addition, a new pollution source, automobile exhaust, had been shown to be of prime importance.

After the pioneering studies of Haagen-Smit, an extensive scientific literature developed on the properties, measurement, and effects of photochemical smog. The attempt to control engine emission has had a profound effect on the automobile and petroleum industries. Estimated costs and associated benefits of automobile emission control each run into the billions per year.[2]

By the Clean Air Act Amendments of 1970, Congress set automobile

emission standards and instructed the Environmental Protection Agency (EPA) to set ambient air quality standards. Included in the Act was a requirement to review the standards periodically. It is hoped that this report will be of value to both Congress and the EPA in discharging their responsibilities for the review of these standards.

However, the Subcommittee on Ozone and Other Photochemical Oxidants did not discuss—and does not necessarily endorse—the adoption of fixed federal standards as the prime approach to pollution control. The subcommittee also did not attempt to determine the concentration at which the standard should be set, except to recognize the difficulty of arriving at such a number. There was, however, general skepticism concerning the applicability of the concept of threshold concentration (the concentration below which there are no biologic effects).

This report deals primarily with the origins and effects of ozone and other photochemical oxidants. It is limited, more or less, to the problem of urban pollution and to such closely related topics as natural background in the earth's boundary layer. No consideration is given to the stratospheric ozone layer and the effects produced by supersonic transport (SST) emission or halocarbons.

The reference method recommended by the federal government for the determination of oxidant measures ozone, which serves as an *indicator* of photochemical smog. Other agents formed in the photochemical system include a variety of free radicals in the gas phase and sulfates, nitrates, and oxygenated organic compounds in the particulate phase. A measurement of ozone alone provides only limited information on the concentrations of the other agents because of the complex chemical and mixing processes involved. How these other agents form and what their effects are remain poorly understood. Recommendations on studies involving such agents are found throughout this report.

We still lack an adequate dose-response relationship for humans exposed to ozone, particularly at concentrations less than about 0.2 ppm. The data base for the development of such a relationship for both short- and long-term exposures is inadequate. Although some data from controlled studies are available for concentrations above 0.3 ppm, methods for extrapolating to lower concentrations are needed. Moreover, it is not clear how to weight the results of pulmonary function tests on humans, animal studies, and epidemiologic studies in a general dose-response relationship.

Despite uncertainties concerning the causative agents and their effects, we must proceed with the regulation of emissions that lead to the formation of photochemical smog. At the same time, research should continue on identifying the individual harmful agents in photochemical smog and

determining their effects. Otherwise, there is danger of focusing on an indicator (ozone) while the formation and behavior of associated pollutants, which create a major part of the problem, are not adequately understood.

Approximately the first third of this report is concerned with the origins and measurement of ozone and other photochemical oxidants and the relationship of atmospheric concentrations to emissions. The middle third deals with toxicologic studies and effects on humans, and the last with effects on plants, ecosystems, and materials.

Each chapter is accompanied by a summary and/or a set of recommendations. Some of the most important points are identified in this Executive Summary, particularly those which cut across several fields. In some cases, the recommendations have been abstracted directly from the other chapters. For detailed recommendations, however, the reader is referred to the chapters themselves.

ORIGINS AND MEASUREMENT

The extensive scientific literature covering the chemistry of smog reactions is reviewed in Chapter 2. Even in the case of a single hydrocarbon with typical concentrations of the oxides of nitrogen, carbon monoxide, water vapor, and other trace components, several hundred chemical reactions take place. The urban atmosphere contains not just one but hundreds of different hydrocarbons, each with its own reactivity and oxidation products. Only a few of the reaction mechanisms and rate constants needed to construct realistic models of polluted atmospheres have been determined in laboratory studies under carefully controlled conditions. *Serious gaps remain in the present models, and further fundamental research on kinetics and mechanisms is necessary.* For example, rate constants are needed for almost all the reactions of hydroperoxy and alkylperoxy radicals. The homogeneous and heterogeneous reactions of the oxides of nitrogen with water also need study.

Smog-chamber studies are needed for validating both detailed chemical models and lumped models. Measurements of more products and the reactive intermediates, including such free radicals as hydroxyl and hydroperoxy, will provide more stringent tests for models. *There are useful interactions among modeling studies, smog-chamber experiments, atmospheric measurements, and fundamental chemical kinetics; it is not possible to ignore one without hindering progress in the others.*

The possibility that free radicals, particularly hydroperoxy, have significant effects on biologic surfaces should be investigated.

The available information on aerosol formation in photochemical smog

is reviewed in Chapter 3. The story told there is still not complete, but there is evidence that reactions involving ozone contribute significantly to the formation of both the organic and sulfate-containing components of the aerosol. Laboratory studies show that both cyclic olefins and C_{6+} diolefins are efficient aerosol precursors that lead to the formation of difunctional oxygenated organic compounds (such as dicarboxylic acids) of low vapor pressure. These compounds have also been found in the smog aerosol. Cyclic olefins have been identified in both gasoline and auto exhaust and might be an important source of secondary aerosol organics; sources of diolefins are unknown. The role of aromatics as aerosol precursors is not understood.

Aerosol organics in the atmosphere could be reduced by control of emission of nitrogen oxides and *total* hydrocarbons. However, the identification and control of a few *specific* aerosol precursors in gasoline and other sources might prove a more efficient approach.

Our knowledge of the chemical and physical processes that govern aerosol formation in the atmosphere is limited, and further research in the field is badly needed. Attention should be focused on laboratory studies of aerosol formation from aromatic hydrocarbons. The concentrations of aerosol precursors in the atmosphere should be determined; more data on organic compounds in ambient aerosols are needed to estimate the relative importance of olefinic and aromatic hydrocarbons as aerosol precursors.

The health effects of difunctional oxygenated organic compounds should be investigated in both animal and human studies.

A critical question concerning atmospheric concentrations of ozone and other photochemical oxidants is: "What fraction of the observed values in each locale can be controlled by reduction of emissions?" Some contend that natural background concentrations exceed the federal ambient air quality standard (0.08 ppm). Another point of view is that background ozone concentrations rarely exceed about 0.05–0.06 ppm at the surface and that higher concentrations are caused by man-made sources.

The data reviewed in Chapter 4 support the second point of view. Measurements in remote areas of the Northern Hemisphere, when compared with those in the lower 48 states of the United States, support the contention that man-made sources are involved in cases where the standard is exceeded. Further measurements are needed to establish this contention with more certainty. Some of the difficulties involved in such studies become apparent when it is noted that the effect of pollution—particularly nitric oxide emission—is to *reduce* ozone concentrations locally.

Theoretical interpretation of the experimental observations will help in determining the relative roles played by stratospheric injection, plant emission, background methane, and transport to surfaces in the natural portion of the tropospheric ozone cycle.

The most complete data on ozone and other oxidant concentrations have been obtained for the Los Angeles air basin, because of the severity of the problem there. Further measurements are needed in the central and eastern areas of the United States, to broaden the foundations of a national control strategy. Such studies should be designed with specific goals in mind, and not carried out as routine monitoring exercises.

Rational air pollution control strategies require the establishment of reliable relationships between air quality and emission (Chapter 5). Diffusion models for inert (nonreacting) agents have long been used in air pollution control and in the study of air pollution effects. Major advances have been made in incorporating the complex chemical reaction schemes of photochemical smog in diffusion models for air basins. In addition to these deterministic models, statistical relationships that are based on aerometric data and that relate oxidant concentrations to emission measurements have been determined.

Improvements in deterministic (photochemical/diffusion) methods are based largely on accounting for more physicochemical effects in the structure of the model. *Specific research subjects for improved models include photochemical aerosol formation and the effects of turbulence on chemical reaction rates.* The challenge to the researcher is to incorporate the study of these subjects without needlessly complicating already complex models. How accurate a mathematical simulation is required? What, roughly, will be the effect of omitting some particular chemical or physical component? What is the sensitivity of model outputs to inaccuracies in the inputs?

One of the most important contributions of research in this field will be the development of criteria to define the limits of applicability of existing models, rather than creating a single supermodel that will incorporate all effects.

Specific goals are essential in model development and in data collection for model-testing. Examples of goals are determining oxidant isopleths and relating visibility degradation to emission sources. *Monitoring programs should be designed with specific goals of data analysis or modeling. It should not be expected that, from the data alone, useful information will emerge directly, or that someone else will spontaneously dig out the important results.* Two important steps that can be undertaken by those who produce models to encourage application and aid the user are the compilation of a catalog of air quality models that describes their

capabilities in terms of a common set of performance standards and the improvement of model output to permit easy access by the user.

Methods of measuring the components of photochemical smog are reviewed in Chapter 6. There have been significant advances in the calibration of instruments for monitoring ozone in ambient air. A method based on the absorption of ultraviolet radiation at 254 nm has been adopted by California for the calibration of air monitoring instruments. The method is based on the use of a commercially available instrument that measures ultraviolet absorption as a *transfer standard* in the calibration process.

It is important to separate (conceptually and in practice) the calibration process from the monitoring process. Photochemical oxidants consisting primarily of ozone were continuously monitored first in southern California by measuring the color change of potassium iodide solutions brought into contact with ambient air. This measurement continues to yield valid photochemical oxidant data in California. However, it has yielded questionable data at ambient air monitoring sites elsewhere in the United States. For this reason, at the end of 1971, the EPA adopted a continuous monitoring process that involves the measurement of the chemiluminescence produced when ozone in air is brought into contact with ethylene. When it is *calibrated* with the ultraviolet-absorption method, this reference procedure for monitoring ozone in ambient air is widely accepted. *The evaluation of nationally applicable primary calibration procedures for ozone measurement should continue.*

Instruments based on differential ultraviolet absorption still need to be evaluated, and possibly modified, before their acceptance for *monitoring* ozone in polluted atmospheres on a nationwide scale. The California Air Resources Board and other air pollution control agencies are evaluating ultraviolet absorption with both chemiluminescence and potassium iodide instruments.

There is no commercially available instrument for the continuous monitoring of any of the chemical species present in the particulate component of photochemical smog. Methods should be developed for the direct and continuous measurement of such species. Species of interest include sulfates, nitrates, some oxygenated organic compounds, and lead.

HEALTH EFFECTS

A great deal is known about the deposition of aerosol particles in the lung and their later clearance. Less is known about the uptake of gases such as ozone and other oxidants that can react with biopolymers in the

mucous and tissue layers. Such information is important in understanding the site and mechanism of pollutant gas action in humans and the effects of copollutants like nitrogen dioxide and ozone, and in the extrapolation of dose-response data from animals to humans. What has been done in this field is reviewed in Chapter 7, which also discusses the information necessary for improved understanding of the transport process.

The solubility of the gas is important. For example, experimental data from studies carried out with dogs show that nearly 100% of highly soluble sulfur dioxide inhaled through the nose is removed before reaching the first bifurcation in the lung, whereas 27-70% (depending on initial concentration) of ozone, which is less soluble in water, is removed in the same region. In addition to solubility, chemical reactions in the surface layers are of great importance.

A concerted effort is needed to increase our understanding of the transfer and uptake of reactive gases in the lung. A program in this field should involve *in vitro* model studies, animal experiments, and clinical studies. More information is required on the chemical, physical, and morphologic properties of the mucous layer and the kinetics of the reactions of ozone in the mucous and tissue layers. Experimental data on uptake and dosage for ozone and other oxidants are difficult to obtain for the tracheobronchial and pulmonary regions. Such data for animals and humans will be needed to test the present simple transport models, before further refinements are made.

Toxicologic research (Chapter 8) on the effects of ozone in laboratory animals has demonstrated that exposure to airborne ozone at less than 1 ppm for a few hours produces numerous changes in cell and organ structure and function. The lowest concentrations that produce these changes differ somewhat among different species of laboratory animals and with the effect under observation. However, several functional and morphologic indexes of response to ozone are altered with exposures to concentrations of about 0.2-0.5 ppm over periods ranging from a few minutes to several weeks.

Recent studies involving repeated or prolonged exposures of laboratory animals to ozone have suggested that changes indicative of chronic lung disease (such as decreased elasticity of the lungs) also require concentrations of 0.2-0.5 ppm.

Exposures to ozone for a few hours result in a marked increase in the susceptibility of animals to controlled doses of infectious organisms introduced into the lung. This is the most sensitive test of any yet reported; significantly increased susceptibility of mice to one microorganism occurred after exposure to ozone at a concentration as low as 0.08 ppm.

Other reports with different microorganisms or different species have suggested that somewhat higher concentrations are required. *These findings suggest the need for carefully planned epidemiologic studies on the incidence of lung infection in human populations exposed to oxidant air pollution.* How do such studies relate to reported cases of human adaptation to long-term oxidant exposure?

Extrapulmonary effects have also been observed in laboratory animals at concentrations of about 0.2 ppm. These include reduced voluntary activity, chromosomal aberrations in circulating lymphocytes of hamsters, increased neonatal mortality, and greater incidence of jaw abnormalities in offspring of mice exposed to ozone. The mechanisms of these effects are largely unknown. *Reports of chromosomal aberrations in hamsters and of mutagenic activity of ozone in microorganisms and tissue cultures raise the question of a possible genetic or carcinogenic hazard. This should be tested experimentally and epidemiologically.*

There is evidence that nutrition affects animal response to ozone. Increased susceptibility has been reported in animals deficient in vitamin E—or the converse (protection conferred by administration of vitamin E).

Convincing new information on the health effects of oxidant exposure has emerged from controlled studies on humans, from which tentative dose-response curves have been constructed. These data are reviewed in Chapter 9, with the types of experimental facilities now available for such measurements. *The new data show reduced pulmonary function in healthy smokers and nonsmokers after exposure to ozone at 0.37 ppm and higher for 2 h. (The federal standard is 0.08 ppm for a 1-h exposure.)* Other gases and aerosols found in an urban atmosphere were not present in these experiments.

With various tests of ventilatory function, it has been shown that healthy male college students experienced no effect of sulfur dioxide at 0.37 ppm, a 10% decline in function with ozone at 0.37 ppm, and a 20-40% decline in function with a combination of sulfur dioxide at 0.37 ppm and ozone at 0.37 ppm. Other experiments have suggested an adaptation of southern Californians to chronic exposure to ambient ozone.

Further studies are needed to give better dose-response information and to provide a frequency distribution of the population response to oxidants alone and in combination with other pollutants at various concentrations. Such studies should include the effects of mixed pollutants over ranges corresponding to the ambient atmosphere. The mixtures should be carefully characterized to be sure of the effects of trace pollutants on sulfate aerosol formation. The design of such studies should

permit extrapolation from animals to humans and from small groups of humans to populations. Further research on the possibility of human adaptation to chronic exposure to oxidants is desirable.

Safety, ethical, and legal considerations require that the utmost care be exercised in human experimentation. The risk inherent in this work can be minimized by the proper design of facilities for human exposure to reactive gases, such as ozone and sulfur dioxide, and reactive gas mixtures. *Standards for the exposure of humans to such controlled atmospheres should be discussed by national groups and agencies, such as the American Medical Association and the National Institutes of Health.*

Studies of the reactions of population groups to photochemical smog are reviewed in Chapter 10. Such studies played a major role in the establishment of the current federal standards. Included were eye irritation studies, effects on asthmatics, and the responses of groups of high-school athletes. Uncertainties in the design of these experiments and interpretation of the data make further epidemiologic studies essential.

Two major studies are being conducted by the EPA in Los Angeles on the effects of photochemical oxidants on health. The first is a survey of schoolchildren in seven communities representing different degrees of oxidant exposure. Rather detailed environmental monitoring data are being taken, and specific health characteristics are being followed, including chronic respiratory disease in adults, lower respiratory disease in children, acute respiratory disease in both children and adults, pulmonary function in children, aggravation of asthma, irritation of mucous membranes, and tissue residues of trace metals. Complete data from this study will not be available until about 1980.

The second study is only beginning and will attempt to correlate the effects of photochemical oxidants and cigarette-smoking in promoting chronic respiratory signs and symptoms in cohorts of adolescents and their families. Pulmonary function tests will be included.

These studies are being carried out by EPA as part of the CHESS (Community Health Effects Surveillance Studies) program. *The results of these studies should be released as soon as possible for evaluation by the general scientific community.* This will permit the design and initiation of additional studies with modifications where necessary to supplement what has been done. *The continuation of epidemiologic studies, including those of the CHESS program, is vital to our understanding of the effects of air pollution on health.*

Other epidemiologic studies should be designed to seek analogues in human populations of effects observed in toxicologic and clinical studies,

including the results of pulmonary function tests, evidence of chronic lung disease in animals, and evidence of increased susceptibility to microbial infection.

EFFECTS ON PLANTS, ECOSYSTEMS, AND MATERIALS

The major phytotoxic components of the photochemical oxidant system, discussed in Chapter 11, are ozone and peroxyacetylnitrate (PAN), but there is indirect evidence that other phytotoxicants are present. Considerable effort has gone into controlled exposures to ozone and into field studies. Leaf stomata are the principal sites for ozone and PAN entry into plant tissue. Closed stomata will protect plants from these oxidants. Both ozone and PAN may interfere with various oxidative reactions within plant cells. Membrane sulfhydryl groups and unsaturated lipid components may be primary targets of oxidants. Young leaf tissue is more sensitive to PAN; newly expanding and maturing tissue is most sensitive to ozone. Light is required before plant tissue will respond to PAN; that is not the case with ozone.

Oxidants reduce yields of many plants, especially sensitive cultivars. Chronic exposures to concentrations between 0.05 and 0.15 ppm will reduce soybean, corn, and radish yields. The threshold appears to be between 0.05 and 0.1 ppm for some sensitive cultivars—well within values monitored in the eastern United States. Growth or flowering effects on carnation, geranium, radish, and pinto bean have been found at chronic exposures to ozone at 0.05–0.15 ppm. Estimated costs to consumers of agricultural losses from oxidant damage are several hundred million dollars a year.

There have been three main approaches to protecting plants from air pollution. Several researchers are including pollutant stress in standard breeding programs and thus are breeding for tolerance. Interim measures involve the use of chemical sprays. Such sprays are not now economically feasible; but they are being tested, and some are protective. Cultural and land-use practices may also be used to control pollution effects, especially on a short-term basis.

A few definitive experiments are needed to complete our knowledge of acute dose-response relationships for ozone. Research is necessary in the case of PAN and other oxidants. More important is the need for studies of crop and native species over growing seasons with chronic oxidant exposures. At the same time, additional work with field chambers, filtered or nonfiltered, is needed.

There is a critical need to understand the interaction of multiple pollutants on the plant systems. These are believed to be important, but little is known about these interactions with respect to most plants.

Although the plant membrane is considered the primary site of action for the oxidants, there is no definitive work on this. The mechanism of response and the biochemical systems affected are not understood. An understanding of these responses would be supportive of breeding and spray protective programs.

Effects on ecosystems are considered in Chapter 12. The permanent vegetation constituting natural ecosystems receives much greater chronic exposure than the short-lived vegetation that makes up the agroecosystem subject to intermittent short-term fumigations. Each situation has measurable economic and aesthetic effects, but on different time scales. The single agricultural ecologic system (the agroecosystem) has little resilience to pollutant stress; losses are sometimes immediate and occasionally catastrophic. The natural ecosystem is initially more resistant to pollutant stress because of species diversity, but the longer chronic exposures disrupt the system. Simulation models of ecosystem components are under development. The study of such models and their interaction offers the possibility of determining the long-term effects of pollutants on natural ecosystems and agroecosystems.

There is convincing evidence of large-scale damage to natural ecosystems in regions downwind from Los Angeles. The injury to the mixed-conifer stands of the San Bernardino National Forest began in the early 1940's and is well advanced. A similar problem is developing in the forests of the southern Sierra Nevada. Both direct and indirect effects have been observed on most components of the forest ecosystem, including producers, consumers, and decomposers. These effects are the results of reactions in the Los Angeles urban plume, which generate oxidants as the pollutants are transported downwind. *Other cities of the western United States—namely, Salt Lake City and Denver, where basin-mountain terrain is contiguous—may show similar injury to forest ecosystems as their oxidant air pollution problems grow.*

The socioeconomic consequences of the continued degradation of natural ecosystems and agroecosystems should be investigated in more detail. *The indirect effects on man's health and the direct effects on his welfare resulting from ecosystem deterioration by oxidant injury should be taken into account in developing air pollution control strategies.* Land-use planning and air-shed classification schemes may be useful in averting further deterioration.

Dose-response relations in key primary-producer species in service-important ecosystems should be examined under field conditions.

Material damage by oxidants is reviewed in Chapter 13. In test chambers with external "ozone" generators that operate at or near atmospheric pressure, ozone is the only likely oxidizing species. In ambient air, however, the presence of other oxidants and sunlight may contribute to material damage. *Laboratory studies of the mechanisms and effects of oxidants other than ozone—including PAN, atomic oxygen, and some free radicals, such as hydroxyl and hydroperoxy—on specific materials are needed. The concentration of these agents in the atmosphere should be determined.*

The most economically important materials with respect to ozone damage are paint, elastomers (rubbers), and textile fiber-dye systems. Damage to polyethylene by ozone is considered to be negligible. The 1970 ozone damage to materials has been estimated as follows: paint, $540 million; elastomers, $569 million; and textile fibers and dyes, $84 million—for a total of over $1 billion. Thus, the total combined material and crop damage falls between $1.5 and $2 billion per year. Estimates of damage to natural ecosystems are not available.

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2

Chemical Origin

The photochemistry of the polluted atmosphere is exceedingly complex. Even if one considers only a single hydrocarbon pollutant, with typical concentrations of nitrogen oxides, carbon monoxide, water vapor, and other trace components of air, several hundred chemical reactions are involved in a realistic assessment of the chemical evolution of such a system. The actual urban atmosphere contains not just one but hundreds of different hydrocarbons, each with its own reactivity and oxidation products.

The atmosphere is complicated in other ways. The emission of primary pollutants occurs throughout the day and night (varying with time and location), adding to some of the previous day's well-aged pollutants. As the sun rises, the light intensity increases in a nonlinear fashion. The movement of air is important—vertical mixing and lateral transport from one community to another. With today's computers, it is now practical to construct a model that includes the detailed chemistry and all these variables.

Several approaches have been used to reduce the problem to manageable proportions. The chemistry of photochemical-oxidant formation can best be understood by considering laboratory experiments with one hydrocarbon (two at most) and typical amounts of the nitrogen oxides, carbon monoxide, and water vapor. A model is developed on the basis of all the chemical reactions that are thought to be relevant, with their measured

or estimated rate constants. Calculations and observations are then compared to assess the accuracy and completeness of the chemical model. Alternatively, one can reduce the chemistry to just a few generalized or lumped reactions, and then include the temporal and spatial changes that occur as a polluted air mass moves through an urban environment. Both approaches provide valuable insights.

This chapter offers a brief introduction to present understanding of the chemistry of photochemical-oxidant formation (thorough reviews are available elsewhere[1,2,13,35,36,49]), some comments on the state of knowledge of the chemistry of the polluted atmosphere, and a review of some recent developments in instrumentation that promise to increase understanding of atmospheric chemistry.

BASIC CHEMISTRY OF OXIDANT FORMATION

Brief Review

This section covers some of the more important chemical reactions that occur in the polluted atmosphere and attempts to show how these reactions result in photochemical-oxidant formation. For a more thorough understanding of the chemistry involved, the reader should consult recent reviews[1,2,35,36,49] and computer modeling studies by Demerjian, Kerr, and Calvert[13] and by Calvert and McQuigg.[9] Unless otherwise noted, the mechanisms and rate constants of these modeling studies are used in this discussion.

Three properties of photochemical smog were evident first in Los Angeles: eye irritation; haze (aerosol) formation; and the degradation of rubber products. All three are associated with oxidants, although aerosols can also be formed by other pollutants, particularly sulfur dioxide.

The photochemical oxidants that are observed in the atmosphere are ozone, O_3; nitrogen dioxide, NO_2; and peroxyacetylnitrate (PAN). Several other substances, such as hydrogen peroxide, H_2O_2, may be classified as photochemical oxidants, but their common presence in smog is not well established. The oxidants are secondary pollutants; i.e., they are formed as a result of chemical reactions in the atmosphere. Primary pollutants are those emitted directly by pollution sources.

The classes of major primary pollutants that are important in urban areas are listed in Table 2-1. The pollutants most responsible for oxidant formation in the air are the nitrogen oxides, hydrocarbons, aldehydes, and carbon monoxide. The internal-combustion engine is a major source of emission of these primary pollutants, although many stationary sources,

TABLE 2-1 Classes of Major Primary Pollutants in Urban Areas

HYDROCARBONS
Alkanes: *n*-butene, isopentane, isooctane
Cycloalkanes: cyclohexane, methylcyclopentane
Olefins (sometimes called "alkenes"): ethylene, propylene, butene
Cycloolefins: cyclohexene
Alkynes: acetylene
Aromatics: toluene, xylene
CHLORINATED HYDROCARBONS
ALDEHYDES, RCHO:[a] formaldehyde, acetaldehyde
KETONES, RCOR:[a] acetone, methylethylketone
NITRIC OXIDE, NO[b]
CARBON MONOXIDE, CO
SULFUR DIOXIDE, SO_2

[a] R = a hydrocarbon group such as methyl, CH_3, or benzyl, C_6H_5.
[b] "NO_x" is often used to indicate "oxides of nitrogen." In practice, this usually means the sum, NO + NO_2, although it should include such other forms as NO_3, N_2O_5, and HNO_3. Nitrous oxide, N_2O, is relatively inert in the lower atmosphere and is not included in NO_x.

such as electric power generating plants, also contribute heavily to emission of nitrogen oxides.[60-63]

The amount of oxidant formed in the atmosphere has a complex dependence on time of day, meteorologic conditions, and amounts of the various primary pollutants. A typical time dependence for a smoggy day in Los Angeles, California, is shown in Figure 2-1. Early in the morning the concentration of ozone is very low. As the day progresses, the ozone increases. The complementary behavior of nitric oxide and nitrogen dioxide is of major importance here. The rapid increase in nitric oxide is the result of the morning rush-hour traffic. The nitric oxide concentration then falls, even though automobile emission is still strong, and nitrogen dioxide begins to increase. As the nitrogen dioxide concentration increases, so does that of ozone.

It should be noted that the concentrations shown in Figure 2-1 represent averaged hourly values. Recent continuous monitoring of nitrogen dioxide at one site shows a more complex time dependence, seen in Figure 2-2. These rapid fluctuations in concentration within a few minutes are probably the result of the movement of air masses and varying emission from local sources.[64] Figure 2-2 underlines the importance of air transport and variable emission, as well as of chemical changes, in the modeling of concentration at a single monitoring station.

The time dependence of the oxidant concentrations shown in Figure 2-1 can be mimicked in laboratory studies. The results of a typical smog-chamber experiment are shown in Figure 2-3. A sample of air initially

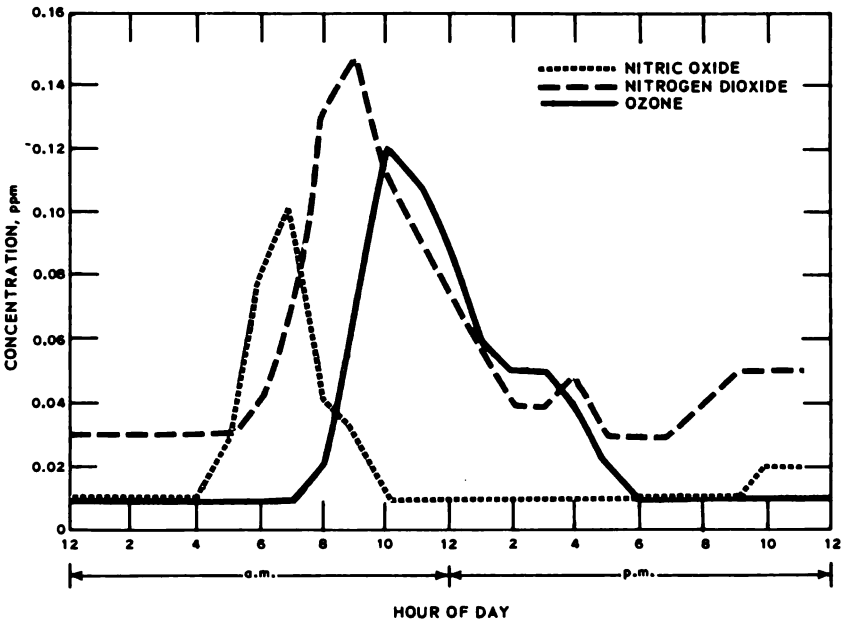


FIGURE 2-1 Diurnal variation of nitric oxide, nitrogen dioxide, and ozone concentrations in Los Angeles, July 19, 1965. Reprinted from *Air Quality Criteria for Photochemical Oxidants.* [66]

containing propylene at 2.2 ppm and nitric oxide at 1.0 ppm is irradiated starting at time zero. Although the hydrocarbon begins to disappear almost immediately, ozone does not develop until almost all the nitric oxide has been converted to nitrogen dioxide. This relationship between nitric oxide, nitrogen dioxide, and ozone is of central importance in attempts to understand the chemistry of these systems. Note also in Figure 2-3 the development of PAN, $CH_3CO_3NO_2$.

Laboratory experiments of this type have the great advantage that the initial conditions can be well defined (although often they are not[13]), in contrast with the average sample of urban air, which is a mixture of new and old pollutants. Also, in laboratory experiments, the same sample of air is observed over a long period, which is not possible with most air pollution monitoring networks. For these reasons, most attempts to understand the chemistry of oxidant formation have concentrated on smog-chamber experiments, rather than the real atmosphere.

The major oxidant in smog is ozone, and early research efforts concentrated on the mechanism of its formation. Attention was focused on the nitrogen oxides, specifically nitrogen dioxide, because it was known

that the nitrogen dioxide molecule could absorb blue and near-ultraviolet sunlight and break apart (undergo photolysis):

$$NO_2 + \text{light}(\lambda < 430 \text{ nm}) \rightarrow O + NO. \quad (2\text{-}1)$$

In the lower atmosphere, the oxygen atoms react quickly with molecular oxygen to form ozone:

$$O + O_2 + M \rightarrow O_3 + M. \quad (2\text{-}2)$$

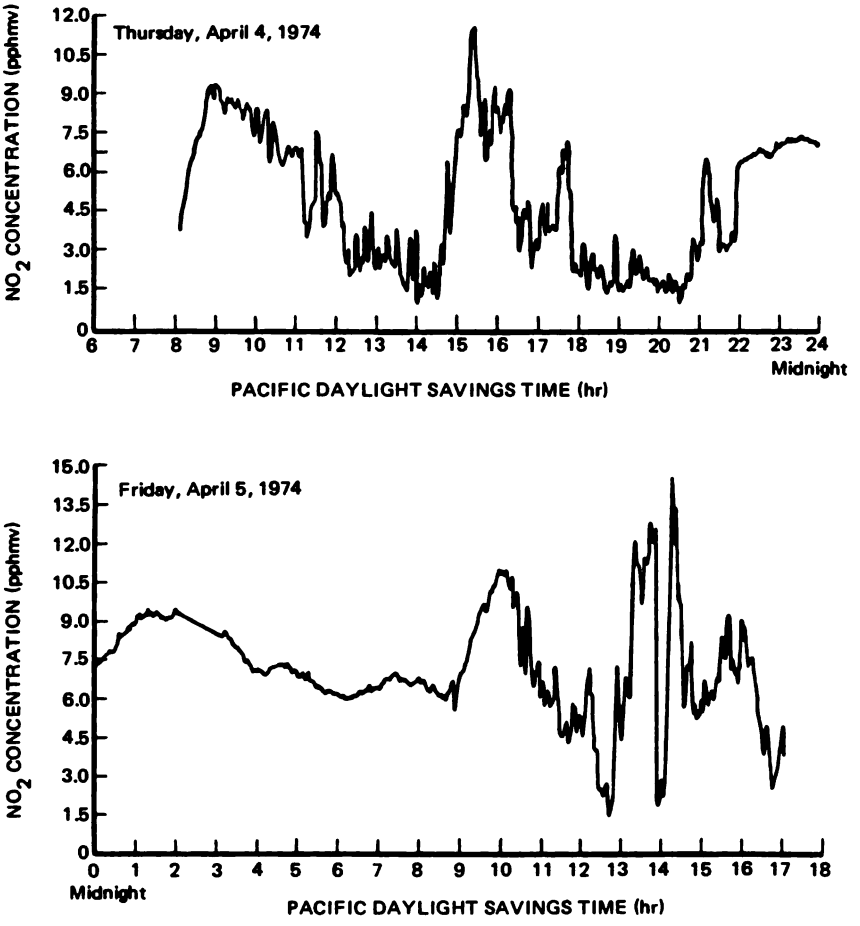


FIGURE 2-2 Atmospheric nitrogen dioxide concentrations, El Segundo, California, April 4–5, 1974. Reprinted with permission from Tucker *et al.*[64]

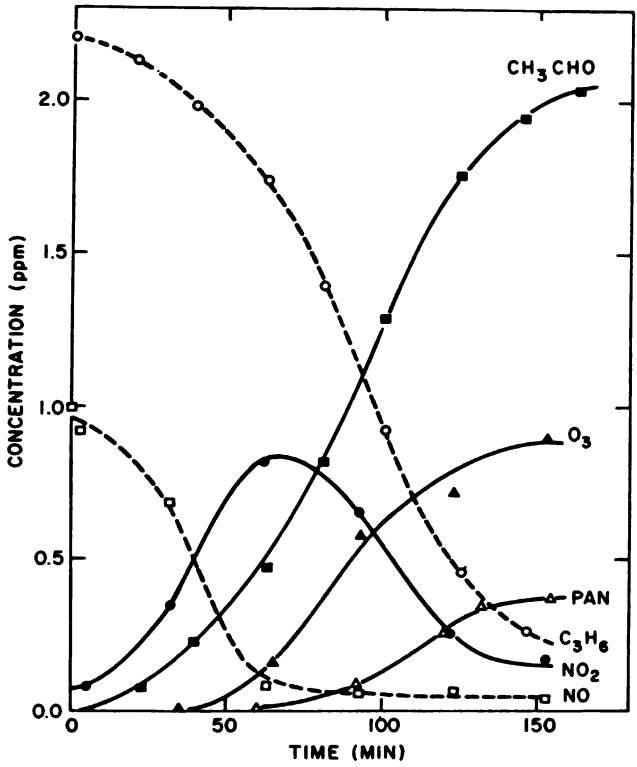


FIGURE 2-3 Typical concentration-time profiles for irradiation of a propylene-NO_x mixture in a smog chamber. Reprinted with permission from Niki *et al.* [44]

Reaction 2-2 is actually a three-body process, in that another molecule (M), usually nitrogen or oxygen, is necessary to carry off the energy released in the newly formed bond. These two reactions then form a mechanism for ozone formation in the atmosphere. They would not be complete without the additional reaction,

$$NO + O_3 \rightarrow NO_2 + O_2, \qquad (2\text{-}3)$$

which is known to be fast. Because Reaction 2-3 consumes the molecule of ozone that was formed in Reaction 2-2 and regenerates the molecule of nitrogen dioxide that was photolyzed in Reaction 2-1, the three reactions form a cycle, which is shown schematically in Figure 2-4. The net result of this cycle is that the absorbed sunlight is degraded into thermal energy. Several other minor reactions occur in this system, but they are not discussed here.[13(p. 17)]

Reactions 2-1 through 2-3 show the most common chemical reactions that occur in the polluted atmosphere. The reason is that nitrogen dioxide is the strongest absorber of sunlight. At a latitude of 40°, the typical turnover lifetime for nitrogen dioxide is about 1.4 min. This means that, every 1.4 min on the average, half the nitrogen dioxide molecules are photodissociated (Reaction 2-1) and reformed (Reaction 2-3). No other molecule in smog is so active.

An important consequence of this rapid turnover is the establishment of a steady-state concentration of ozone. One can express this dynamic equilibrium as follows:

$$[O_3] = \frac{k_1\,[NO_2]}{k_3\,[NO]}, \tag{2-4}$$

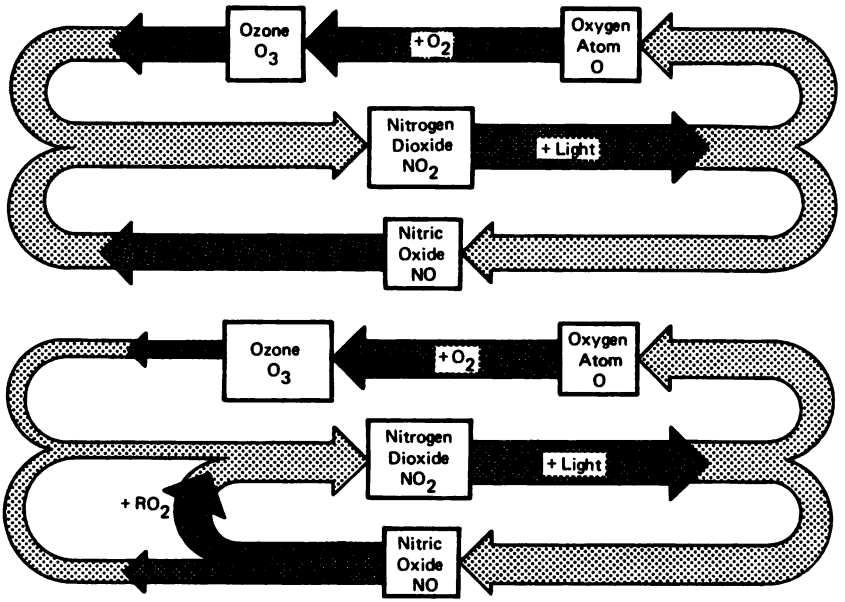


FIGURE 2-4 The NO-NO_2-O_3 cycle in air contaminated with NO_x only (above) and with NO_x and hydrocarbons (below). Above, the dissociation of nitrogen dioxide by sunlight forms equal numbers of nitric oxide molecules and oxygen atoms. The latter are rapidly converted to ozone molecules. The ozone then reacts with the nitric oxide, again on a 1:1 basis, to reform nitrogen dioxide. Only a small steady-state concentration of ozone results from this cycle. Below, when hydrocarbons, aldehydes, or other reactive contaminants are present, they can form peroxy radicals that oxidize the nitric oxide, pumping it directly to nitrogen dioxide. This leaves very little of the nitric oxide to react with the ozone, so the ozone builds up to large concentrations.

where k_1 is the rate at which sunlight dissociates nitrogen dioxide, k_3 is the rate constant for Reaction 2-3, and the brackets indicate concentrations. With a typical value for k_1 (8×10^{-3}/s) and the known rate constant for Reaction 2-3, Equation 2-4 becomes:

$$[O_3] = (0.021 \text{ ppm}) \frac{[NO_2]}{[NO]}. \qquad (2\text{-}5)$$

Modeling studies show that Equation 2-4 should be obeyed quite closely.[13] Tests of this equation on atmospheric data show good agreement, at least for ozone concentrations of 0.1 ppm or less.[14,57] At higher ozone concentrations, deviations have been observed, although it was suggested that the method of averaging was responsible, rather than a real failure of Equation 2-4.[14]

Equation 2-4 does appear to explain, at least qualitatively, the time dependence of the ozone concentration. For example, as long as the ratio $[NO_2]$:[NO] is less than 1:1 (Figure 2-3), the ozone concentration is very low. However, when most of the nitric oxide has been converted to nitrogen dioxide, the ozone concentration increases rapidly. Similar behavior is observed in the Los Angeles atmosphere (Figure 2-1).

Although of central importance in smog chemistry, Reactions 2-1 through 2-3 cannot by themselves explain the buildup of ozone. If only these three reactions were important, the photodissociation of nitrogen dioxide would rapidly establish a small ozone concentration within a few minutes, after which no further changes would occur. During this ozone buildup, the nitrogen dioxide concentration would only *decrease*. Contrast this behavior with the observations in Figures 2-1 and 2-3. The actual ozone buildup occurs over a period of hours and is accompanied by an *increase* in the nitrogen dioxide concentration. Thus, the photolysis of nitrogen dioxide alone cannot explain the ozone buildup, even though it is the *mechanism* of ozone formation. The dominant factor in these systems is the ratio of nitrogen dioxide to nitric oxide. The challenge, then, is to explain the conversion of nitric oxide to nitrogen dioxide. Once that is done, the ozone concentration will follow the $[NO_2]$:[NO] ratio.

Another difficulty was apparent in the early chemical studies on polluted air.[64] It was known from laboratory studies that both ozone and the ground-state oxygen atoms that are formed in Reaction 2-1 would attack reactive hydrocarbons. However, the experimentally observed rate of loss of the hydrocarbons was often greater than could be explained by the attack of ozone and oxygen atoms. Figure 2-5 shows this effect for the case of propylene. Note that the discrepancy is especially large in the earlier parts of the reaction.

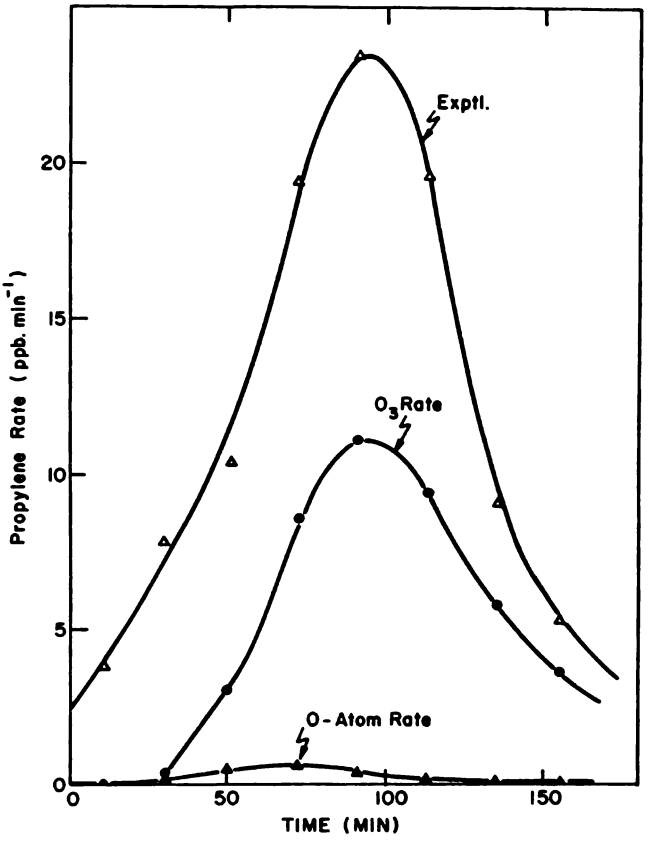


FIGURE 2-5 Comparison of the experimentally observed rate of propylene loss with that calculated for its reactions with ozone and oxygen atoms. Reprinted with permission from Niki *et al.* [44]

Some new mechanism was required to explain the rapid oxidation of nitric oxide to nitrogen dioxide; and one or more reactive intermediates, in addition to ozone and oxygen atoms, were needed to explain the observed hydrocarbon loss rates. Several people suspected that these two problems were connected to some free-radical chain mechanism. Many reactive intermediates were suggested, including the hydroxyl radical, OH; the hydroperoxy radical, HO_2; the methoxy radical, CH_3O; nitrogen trioxide, NO_3; and "singlet oxygen" (meaning O_2 in one of its low-lying metastable states, $a^1\Delta$ or $b^1\Sigma$).

About 1970, two research groups suggested that hydroxyl radicals

(OH) were the solution to the above problems.[24,58] They suggested the following reaction cycle:

$$OH + CO \rightarrow H + CO_2, \quad (2\text{-}6)$$

$$H + O_2 + M \rightarrow HO_2 + M, \quad (2\text{-}7)$$

$$HO_2 + NO \rightarrow OH + NO_2. \quad (2\text{-}8)$$

Reaction 2-6 is sufficiently fast to be important in the atmosphere. For a carbon monoxide concentration of 5 ppm, the average lifetime of a hydroxyl radical is about 0.01 s (see Reaction 2-6; other reactions may decrease the lifetime even further). Reaction 2-7 is a three-body recombination and is known to be fast at atmospheric pressures. The rate constant for Reaction 2-8 is not well established, although several experimental studies support its occurrence.[11,19,48,54] On the basis of the most recently reported value for the rate constant of Reaction 2-8, which is an indirect determination,[20] the average lifetime of a hydroperoxy radical is about 2 s for a nitric oxide concentration of 0.05 ppm. Reaction 2-8 is the pivotal reaction for this cycle, and it deserves more direct experimental study.

Reactions 2-6 through 2-8 form a catalytic cycle, in that the hydroxyl radical that is used in Reaction 2-6 is regenerated in Reaction 2-8. The net results of this cycle are the oxidations of nitric oxide to nitrogen dioxide and carbon monoxide to carbon dioxide by the oxygen present in the air.

Other oxidation chains can be constructed. For example, when methyl radicals are generated by other reactions, as in the ozonolysis of olefins, the following reactions can occur:

$$CH_3 + O_2 + M \rightarrow CH_3O_2 + M, \quad (2\text{-}9)$$

$$CH_3O_2 + NO \rightarrow CH_3O + NO_2, \quad (2\text{-}10)$$

$$CH_3O + O_2 \rightarrow CH_2O + HO_2. \quad (2\text{-}11)$$

The methyl radical rapidly (in 10^{-7} s) combines with oxygen to form the methylperoxy radical, CH_3O_2. A recent study has confirmed that nitric oxide is oxidized by methylperoxy, although the rate constant is still unknown.[46] The methoxy radical, CH_3O, should then react predominantly with oxygen to form formaldehyde, CH_2O, and hydroperoxy radical. The net result of this sequence is the oxidation of one molecule of nitric oxide to nitrogen dioxide and the conversion of an alkyl radical into a hydro-

peroxy radical, which can then react as in Reaction 2-8. Similar sequences can be written for larger alkyl radicals.

There were two important innovations in the development of these oxidative cycles: the use of carbon monoxide, which had previously been considered a relatively inert molecule in the atmosphere, to regenerate the hydroperoxy radical via Reactions 2-6 and 2-7; and the use of peroxy radicals, HO_2 and RO_2, to oxidize nitric oxide to nitrogen dioxide.

These oxidative cycles have a drastic effect on the concentration of ozone, as is summarized in Figure 2-4. The peroxy radicals oxidize the nitric oxide back to nitrogen dioxide, increasing the ratio $[NO_2]$:$[NO]$ and, as a result of Equation 2-5, the concentration of ozone. Expressed in words, the photolysis of nitrogen dioxide continues to generate ozone, but the balancing reaction of ozone with nitric oxide becomes less probable, because most of the nitric oxide is reacting with peroxy radicals. As a result, more ozone is being formed than is being destroyed, and so its concentration increases. It is this interaction of the NO-NO_2-O_3 cycle with the free radicals generated from hydrocarbons and other reactive pollutants that is the basis of photochemical oxidant formation.

The participation of hydroxyl and hydroperoxy radicals in the oxidation of nitric oxide raises the possibility that these radicals might also attack hydrocarbons. In the case of hydroxyl, these reactions are known to be fairly rapid. On the basis of the rate constants that have been measured and estimates of those which have not, the rates of attack of hydroxyl and hydroperoxy radicals appear to be large enough to explain the excess consumption of propylene shown in Figure 2-5.

A detailed chemical model has been constructed with these free-radical chain reactions and all the other reactions that are thought to be important in the atmosphere. It is possible to evaluate quantitatively the various reactions that destroy the olefin and to determine which intermediates are most important. Figure 2-6 shows the results of such a summation for a mixture of *trans*-2-butene, NO_x, and aldehydes.[9] The vertical distances between lines on Figure 2-6 are proportional to the various rates of attack. The graph shows that destruction of olefin by hydroxyl radicals is a major process, although attack by ozone, hydroperoxy radicals, and oxygen atoms is also significant. Olefin attack by other intermediates, such as nitrogen trioxide and methoxy radicals, is less significant, but not insignificant.

Taking propylene as a typical example, the reactions with hydroxyl radical would be:

$$\cdot OH + C_3H_6 \rightarrow \cdot CH_2CHCH_2 + H_2O, \qquad (2\text{-}12)$$

$$\cdot OH + C_3H_6 \rightarrow CH_3\dot{C}HCH_2OH, \qquad (2\text{-}13)$$

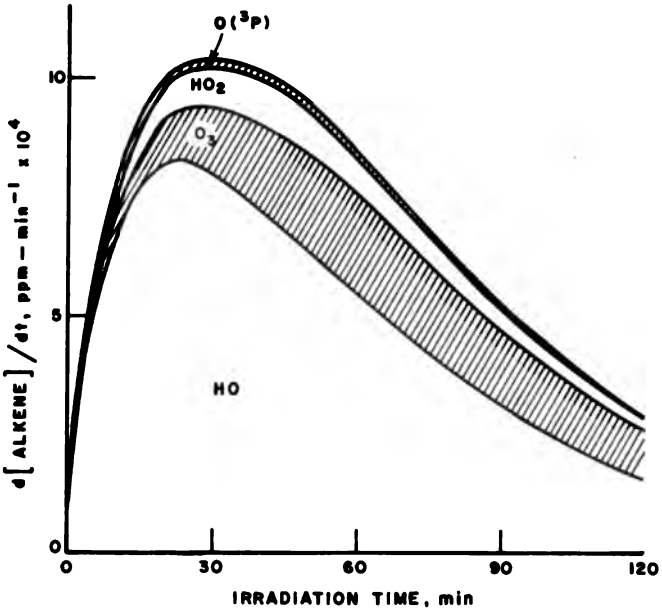


FIGURE 2-6 Calculated rates of reaction of various species with *trans*-2-butene as a function of irradiation time. The initial conditions are as specified in Table 2-2, except that the aldehydes are not initially present. Reprinted with permission from Calvert and McQuigg.[9]

where the dots on the free radicals indicate the dominant positions of the unpaired electrons. Evidence of both abstraction reactions, such as Reaction 2-12, and addition reactions, such as Reaction 2-13, has been obtained recently.[55] The relative importance of addition and abstraction will depend on the structure of the olefin; these numbers are not well established.

Each of the radicals formed in Reactions 2-12 and 2-13 will react with molecular oxygen to form a peroxy radical,

$$R\cdot + O_2 \rightarrow RO_2\cdot, \tag{2-14}$$

which can then oxidize nitric oxide:

$$RO_2\cdot + NO \rightarrow RO\cdot + NO_2. \tag{2-15}$$

The RO· formed in Reaction 2-15 is still a free radical, so it will react further.

Most free radicals contain odd numbers of electrons, and most stable molecules contain even numbers of electrons (nitric oxide and nitrogen dioxide are two important exceptions, being stable molecules with odd numbers of electrons). Therefore, in the reaction, free radical + stable molecule ⟶, another free radical is usually generated. This free-radical chain process is stopped only when one of the following types of processes occurs:

Radical-radical reactions, e.g.,

$$H\dot{O}_2 + H\dot{O}_2 \rightarrow H_2O_2 + O_2, \tag{2-16}$$

$$CH_3\dot{O}_2 + H\dot{O}_2 \rightarrow CH_3O_2H + O_2. \tag{2-17}$$

Radical-NO_x reactions, e.g.,

$$H\dot{O}_2 + NO_2 \rightarrow HONO + O_2 \text{ (or } HOONO_2?), \tag{2-18}$$

$$CH_3COO_2 + NO_2 \rightarrow CH_3COO_2NO_2 \text{ (PAN)} \tag{2-19}$$

Radical-surface reactions, e.g.,

$$H\dot{O}_2 + \text{Surface} \rightarrow \text{Absorbed radical.} \tag{2-20}$$

The rate constants for these chain-terminating steps are not well established. However, present estimates are probably not greatly in error, because radical-radical reactions tend to be fast.

What is the initial source of the free radicals that are so important for oxidant development? Calvert and McQuigg[9] attempted to answer this question by evaluating the many proposed reactions with their detailed chemical model. Although the actual importance of any particular source will depend on the concentration of pollutants assumed and the time of irradiation, they found for a typical mixture (nitric oxide, nitrogen dioxide, *trans*-2-butene, formaldehyde, acetaldehyde, carbon monoxide, water, and methane) that the following reactions were the most important radical sources:

$$HONO + \text{Sunlight} \rightarrow OH + NO, \tag{2-21}$$

$$CH_2O + \text{Sunlight} \rightarrow CHO + H, \tag{2-22}$$

$$CH_3CHO + \text{Sunlight} \rightarrow CH_3 + CHO, \tag{2-23}$$

$$O_3 + C_4H_8 \rightarrow \text{Various radicals.} \tag{2-24}$$

Of special interest here is the radical generation during the early part of the irradiation, before the oxidant concentration has developed; photodissociation of nitrous acid, HONO, and aldehydes is very important. The concentration of nitrous acid in the atmosphere due to the reaction

$$NO + NO_2 + H_2O \rightarrow 2HONO \qquad (2\text{-}25)$$

is controversial, [13 (p. 166)] but the presence of aldehydes in the urban atmosphere is not. It is established that aldehydes will absorb sunlight and dissociate into free radicals. The mechanism of the reaction of ozone with olefins in the gas phase appears to be very complicated, but recent experimental evidence shows that some free radicals are formed.[4]

Although the above reactions generate a few free radicals, most of the oxidation of nitric oxide to nitrogen dioxide is carried out by the alkylperoxy, RO_2, and hydroperoxy radicals that are formed in later reactions involving reactive hydrocarbons, aldehydes, or even carbon monoxide. One such example is shown in Figure 2-7. There is still considerable uncertainty as to the mechanism of these secondary reactions. The modeling studies should be consulted for details.[9,13]

The importance of the secondary reactions can be expressed as a radical chain length, which is the total rate of all reactions involving a particular radical divided by the primary rate of formation of that radical. For a particular set of conditions chosen in the modeling studies, chain lengths of about 4 for the hydroperoxy radical and about 8 for the alkylperoxy and hydroxyl radicals were calculated early in the irradiation period.[9] After further irradiation, the chains became shorter.

The oxidizable pollutants—such as hydrocarbons, aldehydes, and carbon monoxide—serve the function of regenerating free radicals that will react with the oxygen in the air to form alkylperoxy and hydroperoxy. Thus, these oxidizable pollutants can be thought of as pumping the nitric oxide to nitrogen dioxide. In the process, they become degraded to other compounds, some of which are still reactive (e.g., formaldehyde, CH_2O, in Figure 2-7). The amount of pumping that can be done, and thus the amount of photochemical oxidant formed, depends in a nonlinear way on both the reactivity of the oxidizable pollutant and its concentration. As the oxidant concentration builds up, the probability of ozone's reacting with hydrocarbons and various free radicals increases, and the rate of ozone accumulation decreases.

Early attention focused on the most reactive of the hydrocarbons, the olefins, because it was expected and was observed by atmospheric sampling that they were preferentially consumed during smog formation.[59] Laboratory studies confirm that olefin-NO_x mixtures are very prolific sources of ozone. However, these olefins are not essential to oxidant formation.

Both the modeling studies and smog-chamber simulations show significant oxidant formation with NO_x + aldehydes, NO_x + alkanes (except methane), or even NO_x + carbon monoxide in moist air. The development of significant oxidant from NO_x + aldehydes is particularly ominous, because aldehyde emission is not now controlled. As the modelers state:[9 (p. 139)]

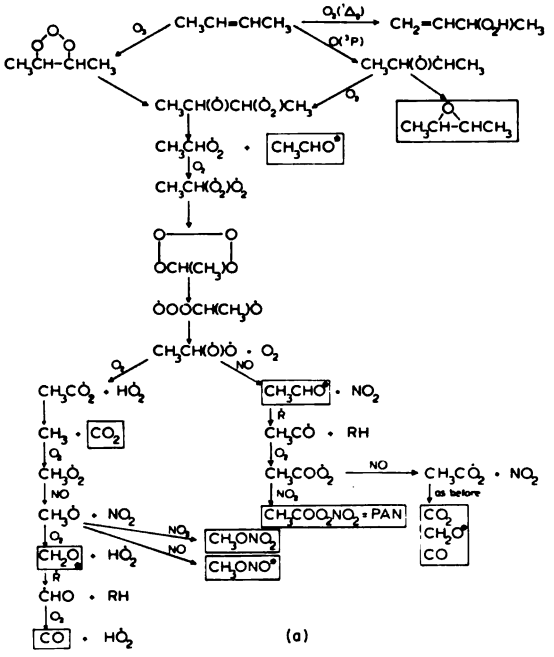
> It appears from these data that the O_3 standard [i.e., 0.08 ppm for 1 h] could not be met if the aldehydes remained high, $[CH_2O] \leq 0.10$, $[CH_3CHO] \leq 0.06$ ppm, even if nearly all of the olefinic hydrocarbon were removed. ... We should learn from these data that the "true" relationship between nonmethane hydrocarbons and maximum 1-h oxidant at low hydrocarbon levels could be a critical function of a variable which is not routinely measured now, namely the concentration of the impurity aldehydes.

A similar statement could probably be made concerning ketones. These compounds are commonly used as solvents, and they are known to form free radicals when photolyzed. Many chlorinated hydrocarbons, which are also widely used as solvents, can be attacked by hydroxyl radicals and thus contribute to peroxy radical formation.

No one pollutant can be blamed as the major cause of ozone formation. Replacing the more reactive hydrocarbons with less reactive ones would delay the formation of ozone, but would not prevent it. Reducing the NO_x concentration seems to reduce the maximal oxidant concentrations observed, but the effect is nonlinear. Heavy injections of nitric oxide into the air can temporarily reduce the local ozone concentration, as often happens in urban centers, but additional oxidant formation can be expected later downwind. Although these effects can be understood qualitatively, it is not yet possible to make accurate predictions of oxidant formation, even in laboratory experiments.

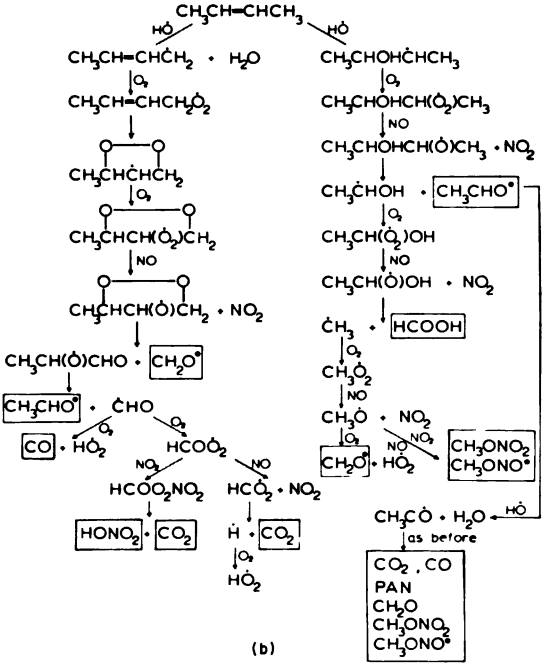
In summary: the concentration of ozone in the polluted atmosphere is controlled by the intensity of sunlight and the ratio of nitrogen dioxide to nitric oxide. Hydrocarbons and other pollutants—such as aldehydes, ketones, chlorinated hydrocarbons, and carbon monoxide—react to form peroxy radicals. These, in turn, react with nitric oxide, causing the ratio $[NO_2]$:[NO] to increase. As a consequence of Equation 2-5, the ozone concentration also increases.

This brief description of oxidant formation in polluted air is based on our current understanding of the chemistry involved. It is evident from an examination of the detailed mechanism that many of the important reactions have not been well studied. For example, the sequences of degradation reactions for the hydrocarbons are only poorly understood. As a result of these uncertainties, it is not possible to make accurate predictions of photochemical oxidant concentrations. However, with another 5 yr of progress similar to the last 5, it should be possible to construct chemical models that will permit ozone predictions accurate to within



(a)

• These products undergo significant photodecomposition in sunlight



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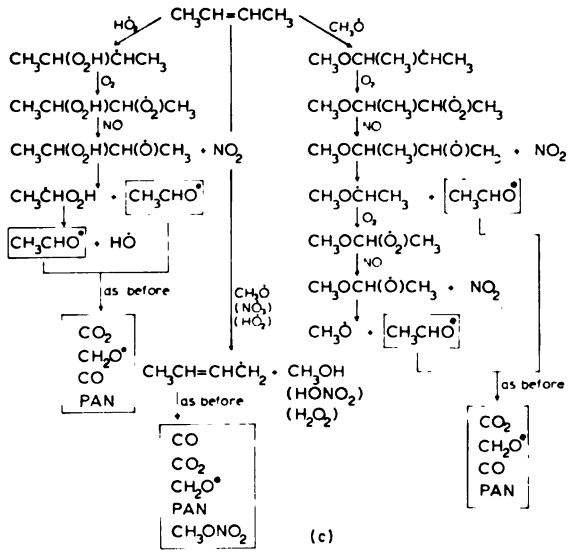

FIGURE 2-7 Major reaction paths for the degradation of *trans*-2-butene in an irradiated NO_x-polluted atmosphere. Reprinted with permission from Demerjian *et al.*[13]

30% for laboratory studies. Although many of the chemical details in the current models are certain to be altered as more experimental data become available, it seems probable that at least the backbone of our present understanding is correct.

Recent Chemical Modeling Studies—Results and Uncertainties

The state of our current understanding of the chemistry of oxidant formation can be judged by examining recent modeling studies, in which all the reactions that are considered important are combined and the resulting differential equations are integrated numerically. Experimental rate constants from the literature are accepted (unless they are judged unreasonable), and others are estimated. In the system NO_x-propylene-air, 242 different reactions are included; another 100 reactions are neglected as probably unimportant. Roughly half the rate constants used in the model are not firm experimental values.

The success of these computer simulations must be rated as quite good. Figure 2-8 compares concentration-time measurements from a smog-chamber study of NO_x-propylene-air with computer-calculated results based on the same initial conditions. The time dependences and absolute concentrations agree fairly well, but not perfectly. Note that the

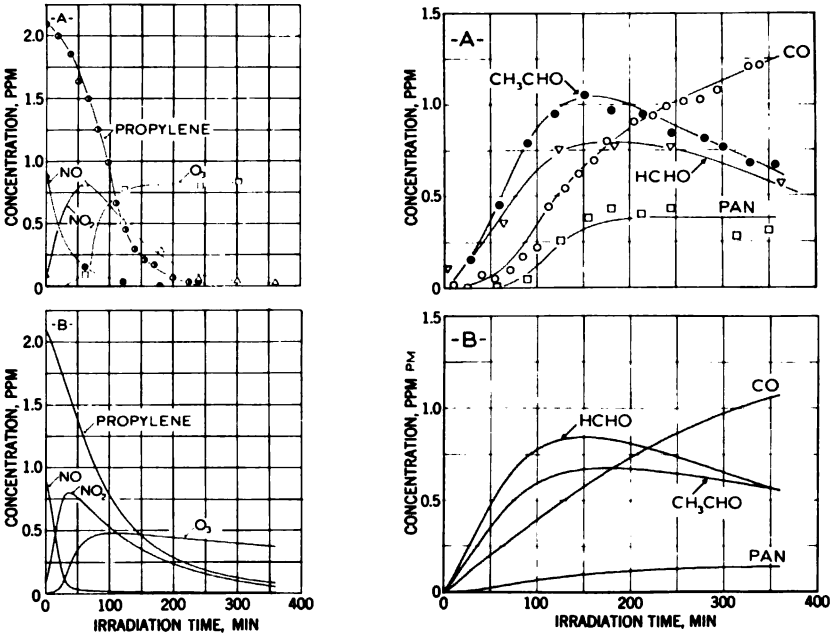

FIGURE 2-8 Photooxidation of propylene in irradiated C_3H_6-NO-NO_2 mixtures in moist air. *A*, experimental rate data from smog-chamber experiment of Altshuller *et al.*[3] Initial concentrations: C_3H_6, 2.09 ppm; NO, 0.90 ppm; NO_2, 0.09 ppm. Relative humidity at 31.5°C, 50%. *B*, computer simulation of product concentration-time curves for same initial conditions. Reprinted with permission from Demerjian *et al.* [13]

calculated maximal concentrations of ozone and PAN are less than the measured values by about a factor of 2. The rapid conversion of nitric oxide to nitrogen dioxide and the time dependence of ozone and PAN are reproduced quite well. These results are impressive, considering that the mechanism and rate constants in the model were not considered variables to be adjusted until a best fit is obtained. Once the reactions and rate constants were selected, they were used for a variety of simulations, with fair to excellent agreement.[13]

However, the number of concentrations being fitted is fairly small, compared with the number of parameters going into the model. The computer calculations can be used to predict the concentrations of other trace compounds that should be present in smog chambers. If these compounds are later found at approximately the predicted concentrations, the model will be strengthened. If not, changes will have to be made in the model. A more stringent test of the model will occur when it becomes

possible to measure the actual concentrations of the free radicals in the atmosphere.

Many of the values of the rate constants that go into the model are not critical, but some are. As the modelers state:[9 (pp. 114-115)]

> Even these systems are very complex and difficult to treat quantitatively since many reactions which appear to be important in theory have not been studied in detail, and theoretical estimates of rate constants must be made in desperation....

Some of the most serious current uncertainties are as follows:

- Direct determinations of rate constants are needed for almost all the reactions of hydroperoxy radical and RO_2.
- "The rate constants for both homogeneous and heterogeneous reactions related to the oxides of nitrogen and water vapor should be characterized carefully....Furthermore the actual levels of nitrous acid, nitric acid, H_2O, NO, and NO_2 should be determined simultaneously in real auto-exhaust-polluted atmospheres...."[13 (p. 140)]
- "The primary quantum yield of radical formation in nitrous acid, CH_2O, and CH_3CHO photolyses should be better established as a function of the appropriate wavelength range of sunlight."[13 (p. 140)]
- "The chemical details of the reaction sequence following HO radical addition and abstraction from olefins should be explored fully...."[13 (p. 141)]
- The chemical details of the reactions of representative alkyl radicals, alkoxy radicals, and biradicals with oxygen should be established. Both the rate constants and the immediate products are needed to construct realistic mechanisms for the model.
- The gas-phase stability of a variety of possible products—such as the olefin ozonide, peroxyformyl nitrate, and peroxynitric acid—is not known.

These assignments represent formidable tasks for the experimentalists, but recent developments in instrumentation and techniques suggest that substantial progress should be made soon.

The present smog models are probably most vulnerable to the possibility that some important mechanism or intermediate has been omitted altogether. The modelers themselves recognize the uncertainties involved:[9 (pp. 149-151)]

> One must not take too seriously the results from complex simulations at this stage of our knowledge. In particular the development of sound reaction schemes and realistic smog models require [sic] much more quantitative kinetic information as to the detailed reaction paths which appear to be important in photochemical smog....There is no question that as such information becomes available, present models will require substantial changes.

A realistic and detailed chemical model has great value. The stepwise addition of various primary pollutants can be made to evaluate the importance of each. The effects of various emission control strategies on the chemistry of oxidant formation can be studied easily and quickly. It is possible to calculate the importance and concentration of various reactive intermediates. One can estimate the concentrations of various compounds that have not yet been observed in smog. And it is possible to pinpoint some of the important gaps, in order to stimulate future experimental studies.

The detailed chemical model has been used to make the following observations and predictions; some of these statements are common to other models and are in accord with experimental observations:

- Complete elimination of olefinic hydrocarbons without controlling aldehyde emission will not ensure low oxidant readings in the atmosphere.[6]
- If no hydrocarbons or aldehydes were present in the atmosphere, but carbon monoxide and NO_x were present, significant ozone concentrations would develop. With reactive hydrocarbons present, the addition of carbon monoxide does not have a strong effect on oxidant concentration, unless it is added in very large amounts (2,000 ppm).[16]
- Reactions of the hydroxyl radical dominate the removal of hydrocarbons. However, several other reactants make significant contributions, including hydroperoxy radical, ozone, and oxygen atoms. (This conclusion depends on the hydrocarbon being considered: it is claimed that some terpenes in air are attacked mainly by ozone.[18])
- The hydroperoxy radical has the highest concentration of all the free radicals in smog. The concentrations of both hydroperoxy and hydroxyl radicals are rather insensitive to primary pollutant concentration.
- The concentration of ozone generated photochemically goes through a maximum as the NO_x concentration is increased.[65]
- The total oxidant dosage varies in a nonlinear manner with dilution of the primary pollutant.[17,51]
- The participation of singlet oxygen in the development of photochemical oxidant is minor, but its human health consequences should be considered.[49 (p. 8)]

A good understanding of the detailed chemistry of oxidant formation makes it possible to construct more compact chemical models. These generalized or lumped mechanism models reduce the number of individual chemical reactions by combining similar or sequential reactions and ig-

noring minor ones.[15,22,23] Averaged reactivities and averaged product yields may be used. The simplified mechanisms can also reproduce smog-chamber data very well, although the construction of the simplified model is semiempirical, so good agreement is not unexpected. These generalized mechanisms are very valuable for modeling the real atmosphere, because the reduction in chemical complexity allows the inclusion of other important variables, such as transport and pollutant inputs. (The use of these models is covered in Chapter 5.)

Which Reactive Intermediates Are Important?

The concentrations of the various reactive intermediates can be calculated with a detailed chemical model. This information is not available elsewhere, because it is not yet possible to measure these reactive intermediates in the atmosphere. Table 2-2 shows the calculated concentrations of several intermediates and ozone for a simulated polluted atmosphere. Table 2-3 gives the calculated rates of attack of the same intermediates on the olefin *trans*-2-butene for the same irradiation times. By comparing these two tables, one can judge the importance of various intermediates. For example, the concentration of singlet oxygen is high, but its rate of attack on the olefin is insignificant, compared with that of hydroxyl radical. The steady-state concentration of hydroxyl radical is very low, owing mainly to its high reactivity. Early in the irradiation, hydroxyl radicals account for 90% of the *trans*-2-butene removal and even after

TABLE 2-2 Calculated Concentrations of Reactive Species in a Simulated Smog Mixture at Several Irradiation Times[a]

	Concentration, ppb		
Species	2 min	30 min	60 min
Ozone, O_3	8.5	84	139
Hydroperoxy radical, HO_2	0.21	0.32	0.37
Singlet oxygen, O_2 ($a^1\Delta$)	3.9×10^{-3}	5.1×10^{-3}	5.2×10^{-3}
Nitrogen trioxide, NO_3	0.03×10^{-3}	2.2×10^{-3}	7.4×10^{-3}
Hydroxyl radical, OH	1.7×10^{-4}	0.88×10^{-4}	0.72×10^{-4}
Oxygen atoms, O	3.8×10^{-6}	8.9×10^{-6}	9.5×10^{-6}

[a]Data from Demerjian *et al.*[13] Initial conditions: [NO], 0.075 ppm; [NO_2], 0.025 ppm; [*trans*-2-butene], 0.10 ppm; [CO], 10 ppm; [CH_2O], 0.10 ppm; [CH_3CHO], 0.06 ppm; [CH_4], 1.5 ppm; relative humidity, 50%.

TABLE 2-3 Calculated Rates of Attack on *trans*-2-Butene by Various Reactive Species in Simulated Smog Mixture at Several Irradiation Times[a]

Species	Attack Rate, ppb/min		
	2 min	30 min	60 min
Ozone	0.026	0.16	0.16
Hydroperoxy radical	0.16	0.15	0.09
Singlet oxygen	2.9×10^{-6}	2.1×10^{-6}	1.3×10^{-6}
Nitrogen trioxide	0.05×10^{-4}	2.2×10^{-4}	2.6×10^{-4}
Hydroxyl radical	1.72	0.55	0.27
Oxygen atoms	0.013	0.018	0.011

[a]Data from Demerjian *et al.*[13] Conditions same as in Table 2-2.

60 min, when the ozone concentration has developed, they still account for 50% of the removal. The hydroperoxy radicals show both a relatively high concentration—by far the highest of the free radicals—and a significant rate of attack on the olefin.

Average Lifetimes of Reactive Intermediates

It is well established that both ozone and PAN can cause damage to biologic systems (see Chapters 8 and 11). The possibility that the reactive intermediates in smog could directly cause biologic damage has been suggested,[35] but experiments are seldom designed to test this possibility.

TABLE 2-4 Calculated Average Lifetimes of Several Reactive Intermediates in a Simulated Smog Mixture[a]

Species	Lifetimes, s
Ozone	250
Hydroperoxy radical	7
Singlet Oxygen	0.05
Nitrogen trioxide	0.1
Hydroxyl radical	0.0036
Oxygen atoms	10^{-5}

[a]Data from Pitts and Finlayson.[49 (p. 8)] Conditions same as in Table 2-2. Irradiation time, 60 min.

The lifetimes of the various reactive intermediates shown in Table 2-4 were calculated on the assumption of a sudden termination of sunlight, as would occur when air is inhaled. The long lifetime of the hydroperoxy radical means that almost all of it will survive long enough to be transported into the lungs. In contrast, the hydroxyl radicals will decay within a few milliseconds. As a computer simulation has shown, even though the half-life of singlet oxygen appears short, compared with the breathing cycle of several seconds, a significant fraction of it should survive into the lungs.[8]

Whether such species as hydroperoxy radicals and singlet oxygen will survive collisions with the surfaces of the upper breathing tract and reach the lower lungs, as ozone does, must await further experiments. Clearly, if all the ozone molecules and all the hydroperoxy radicals are eventually absorbed in the lungs, the ozone damage will dominate, unless hydroperoxy radicals are several hundred times more damaging, which seems unlikely.

Free radicals could have a significant effect on biologic surfaces, such as leaves and human skin, that are in direct contact with irradiated smog. The effective dose delivered to a surface depends on the bulk concentration of the species, the sticking coefficient (the probability that a molecule that hits a surface will be absorbed), and the damage once it is absorbed. Very little is known about sticking coefficients, especially of free radicals. It is known that singlet oxygen will survive approximately 10,000 collisions with aqueous phosphoric acid[71] and that ozone is not immediately absorbed in the upper bronchial tubes. There is an indication that hydroperoxy radicals might be absorbed readily on surfaces.[37] If the sticking coefficient of hydroperoxy radicals were unity for biologic surfaces (which might be reasonable, inasmuch as it has the ability to make hydrogen bonds) and the sticking coefficient for ozone were 10^{-4}, the actual influxes of hydroperoxy radicals and ozone to a surface might be comparable, even though the ozone concentration is approximately 300 times greater. This calculation is pure speculation, but it does show that the influx of free radicals to a sunlit surface should not be neglected.

It is known that free radicals are formed when ozone reacts with carbon-carbon double bonds. Recently, it has been suggested that PAN probably forms free radicals when it reacts with aldehydes.[72] Because hydroperoxy radicals are free radicals, they may have biologic effects similar to those of ozone and PAN. Certainly, for experiments in which the observed biologic damage cannot be attributed to the measured concentrations of ozone and PAN, free radicals or unstable compounds should be considered.

NEW EXPERIMENTAL METHODS OF STUDYING AIR CHEMISTRY

Lasers

The development of reliable lasers with a variety of wavelengths, both fixed and variable, has generated many ideas of applications to air pollution monitoring and to chemical kinetics. Most of these techniques are still in the developmental stage. Whether they will have the reliability and low cost needed for widespread use is not known. Proposed methods include the following:

- *Long-path infrared absorption*, using a tunable diode laser, which is claimed to have a sensitivity of 5 ppb for carbon monoxide over a 610-m path length.[34]
- *Differential laser absorption*, with measurement of two or more wavelengths simultaneously and claimed sensitivity in the parts-per-billion range.[41,45,47]
- *Laser backscattering*, either Raman or fluorescence, which does not require a remote detector, can thus be used for detecting atmospheric pollutants at a distance, and has sensitivity less than that with direct absorption techniques.
- *Laser-induced electronic fluorescence.* Two devices reported recently look very promising for continuous atmospheric monitoring. Sensitivities of 0.6 ppb for nitrogen dioxide and 50 ppb for formaldehyde are claimed.[5,64] Careful attention to possible interference from other species is necessary. Detection of the hydroxyl radical in air ($\sim 10^8$ molecules/cm^3) has been claimed for this technique,[67] but it has been pointed out that this concentration seems much too high,[9] especially because the air had been removed from the sunlight 6 s before analysis; spurious effects, such as photolysis of the ozone in the air by the laser beam and two-photon absorption by water vapor, might have been responsible for the hydroxyl radical that was observed.
- *Photoacoustic or optoacoustic spectroscopy*, which detects the absorption of a pulsed laser in a cell by the pressure pulses generated when the light energy is degraded to heat,[33] which is claimed to have sensitivities of 0.4 ppb for nitric oxide and 5 ppb for ethylene, and which can measure the absorption spectra of solids and dusts.[38]
- *Laser magnetic resonance*, which has already been used to detect the free hydroxyl, methynyl (CH), hydroperoxy, formyl (HCO), and amino radicals in low-pressure gases[12] and could be used to determine rate constants for the reactions of the smaller free radicals.

Recent reviews on lasers and laser spectroscopy should be consulted for details of these promising new techniques.[10,26]

Photoionization Mass Spectrometry

The use of high-intensity resonance lamps in the vacuum ultraviolet as photoionization sources for mass spectrometers allows many free radicals to be observed directly in reacting gases.[27] With the proper choice of lamp, photoionization causes no fragmentation of other molecules to interfere with the free-radical peaks—a major problem in conventional electron bombardment sources. Steady-state concentrations of free radicals have been observed when oxygen atoms react with hydrocarbons and in ozone–olefin reactions.[4,29,56,68,70] With this technique, it was possible to resolve an argument of long standing concerning the immediate products formed by the attack of oxygen atoms on ethylene (mainly methyl and formyl radicals, with about 5% formaldehyde and hydrogen).[29,31,32,50]

The photoionization mass spectrometer can detect singlet oxygen in a large excess of ground-state oxygen and nitric oxide in an excess of nitrogen dioxide.[28,30] Attempts to detect hydroperoxy radicals have not been successful, probably because of a low photoionization cross section, but the methylperoxy radical has been observed with this technique.[69]

Future research using the photoionization mass spectrometer should result in significant progress in resolving some of the uncertainties in current chemical smog models, namely, detection of the immediate products formed when hydroxyl radical reacts with olefins, determination of rate constants and mechanisms for oxygen reacting with free radicals, identification of the immediate products formed in ozone–olefin reactions, and determination of the rate constants and products of the reactions of alkylperoxy radicals with olefins and other hydrocarbons.

Computer-Controlled High-Resolution Mass Spectrometry

The powerful technique of coupling a computer to a high-resolution mass spectrometer has been used to analyze air pollutants.[52] Both particulate matter and gases can be scanned for up to 300 pollutants. Only stable compounds will be detected by this method, because the samples are concentrated before analysis.

The advantage of this technique is the rapidity of monitoring for many compounds simultaneously, including some of the liquid and solid inorganic materials—such as sulfuric acid, ammonium sulfate, and ammonium nitrate—that may be the final products of the primary pollutants nitric oxide and sulfur dioxide. Also, monitoring the many par-

tially oxidized hydrocarbons, such as aldehydes and acids, will give useful insight into the reaction mechanisms involved in the atmosphere. One disadvantage of this system is that only compounds that the computer is programmed for will be reported; unexpected compounds may be overlooked. A careful study of sampling efficiency will be needed before quantitative concentrations in the atmosphere can be reported.

PRODUCTS OF PHOTOCHEMICAL SMOG—OBSERVATIONS AND SPECULATION

A large number of compounds can be formed in the polluted atmosphere. As a result of the small concentrations involved and the great variety of possible products, very few compounds have actually been observed. The gaseous compounds for which quantitative measurements have been reported are listed in Table 2-5 with typical concentrations. Compounds observed in particulate matter are discussed in Chapter 3.

Which of the compounds in Table 2-5 are considered oxidants depends on the reactant being considered. Certainly, ozone, PAN, and hydrogen peroxide are strong oxidants when biologic materials are considered. This list is certain to grow as more sensitive analytic techniques are used and as modeling studies suggest other important species that should be present.

Table 2-6 is a list of some compounds that may be present in photochemical smog, but have not yet been reported. The presence of some of these compounds (such as PBzN and ketene) seems very probable, in that they have been observed in smog-chamber studies, whereas others are

TABLE 2-5 Compounds Observed in Photochemical Smog

Compound	Typical (or Maximal) Concentration Reported, ppm	Reference
Ozone, O_3	0.1 (0.7)	Chapter 4
PAN, $CH_3COO_2NO_2$	0.004 (0.01)	Chapter 4
Hydrogen peroxide, H_2O_2	(0.18)	7
Formaldehyde, CH_2O	0.04	Chapter 4
Higher aldehydes, RCHO	0.04	Chapter 4
Acrolein, CH_2CHCHO	0.007	Chapter 4
Formic acid, HCOOH	(0.05)	21

TABLE 2-6 Compounds That May Be Formed in Photochemical Smog

Compound	Possible Reaction of Formation	Reference
Peroxybenzoylnitrate, $C_6H_5COO_2NO_2$ (PB_zN)		25
Nitric acid, $HONO_2$		21,42
Organic hydroperoxides, ROOH	$RO_2 + HO_2$	13 (p. 75)
Organic peracids, $RCOO_2H$	$RCOO_2 + HO_2$	13 (p. 75)
Organic peroxynitrates, RO_2NO_2	$RO_2 + NO_2 + M$	13 (p. 77)
Ozonides, O_3–olefin	O_3 + olefin + M	4
Ketene, CH_2CO	O_3 + olefin	40
Nitrous acid, HONO	$NO_2 + HO_2$	39
Pernitric acid, HO_2NO_2	$NO_2 + HO_2 + M$	
Pernitrous acid, HO_2NO	$NO + HO_2 + M$	11
Sulfoxyperoxynitrate, $HOSO_2O_2NO_2$	$HOSO_2O_2 + NO_2 + M$	9

very speculative. For example, organic peroxy radicals, RO_2, are almost certainly important intermediates in the conversion of nitric oxide to nitrogen dioxide. When these radicals undergo a chain termination reaction with hydroperoxy radical, the corresponding organic hydroperoxide, ROOH, or peracid, $RCOO_2H$ (if an acylperoxy radical is involved), will be formed. Although the rate of this hydroperoxide formation is estimated[9] to be less than that of the similar formation of hydrogen peroxide by approximately a factor of 100, the potential concentration must be combined with toxicity and other information before a substance can be dismissed as unimportant.

Similarly, chain termination by $RO_2 + NO_2$ could form organic peroxynitrates. These would probably be less toxic than PAN, but they could be present in comparable amounts. The formation of organic peroxynitrites and peroxynitrates has been considered.[13 (p. 77)] but they have not been observed in smog.

If ozone–olefin adducts are stable in the gas phase, as a recent study hinted,[4] then they are almost certainly present in the urban atmosphere. Their concentrations will depend on their stability in the sunlit atmosphere. If present, they are expected to be very reactive.

Pernitric (or peroxynitric) acid is an example of a compound that could be present in significant quantities. This proposal is very speculative, because there is no evidence of this compound in the gas phase, although there is evidence of some such species in solutions.[43] The reaction of hydroperoxy radical with nitrogen dioxide is usually written

$$HO_2 + NO_2 \rightarrow HONO + O_2, \qquad (2\text{-}18)$$

which is certainly reasonable. However, at a pressure of 1 atm, the three-body recombination step,

$$H\dot{O}_2 + NO_2 + M \rightarrow HOONO_2 + M, \qquad (2\text{-}26)$$

also seems probable. The laboratory experiments that support Reaction 2-18 cannot rule out the formation of some pernitric acid. The chemical modeling studies show that, if Reaction 2-26 is assumed to be half as probable as Reaction 2-18, the formation of pernitric acid would be comparable with that of PAN.[9]

Similarly, recent experiments[11] have been interpreted to mean that about 10% of the reaction of hydroperoxy radical with nitric oxide gives pernitrous acid, HOONO, instead of nitrogen dioxide and hydroxyl radical. Because this reaction is of major importance, even 10% of a second channel would be important, although it has been argued that such compounds would not be sufficiently stable to accumulate in the atmosphere.[13 (p. 77)] Whether such peroxynitrogen compounds are stable in the gas phase and whether they can be found in the atmosphere must await further experiments.

The last entry in Table 2-6, sulfoxyperoxynitrate, is an inorganic analogue of PAN. It was suggested in a computer study that investigated the addition of sulfur dioxide to the NO_x-hydrocarbon system.[9] Although this is only speculative, such unpleasant compounds may become more important if the sulfur content of fuels is allowed to increase in areas where an oxidizing photochemical smog is common. It is known that sulfur dioxide, when present in photochemical smog, is rapidly oxidized to sulfuric acid, and a recent mass-spectrometric study also indicated the presence of organic sulfur dioxide compounds.[53]

Table 2-6 is only a sampling of the compounds that might be found in photochemical smog in the future. The possible combinations among the many free radicals and the oxides of sulfur and nitrogen are almost limitless. Many undiscovered exotic compounds *are* present in photochemical smog, but their concentration and importance remain to be established.

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3

Aerosols

Organic compounds were recognized long ago as key ingredients of the polluted atmosphere and constitute a significant fraction of the urban aerosol associated with photochemical smog.[128] Although there is no air quality standard for organic aerosols, ambient concentrations of particulate organic substances are related to the concentrations of total suspended particles, for which there are federal and state standards,* and to prevailing visibility. Primary organic compounds are emitted directly into the atmosphere, and their concentrations can be reduced through emission control. [217] Secondary organic compounds result from gas-phase photochemical reactions involving hydrocarbons, nitrogen oxides, and ozone and thus the same types of control strategies are implied as for ozone and photochemical oxidants[150] (see Chapter 5). However, control of secondary organic compounds would require a more elaborate approach, with the identification of specific hydrocarbon precursors as a necessary step (Figure 3-1). Because of their accumulation in the submicrometer range, secondary organic aerosols may be responsible for adverse health effects and contribute significantly to visibility degradation. Despite its

*The national ambient air quality standards for particulate matter are:[223] 24-h averages, not to be exceeded more than once a year: primary, 260 $\mu g/m^3$; secondary, 150 $\mu g/m^3$. Annual geometric means: primary, 75 $\mu g/m^3$; secondary, 60 $\mu g/m^3$.

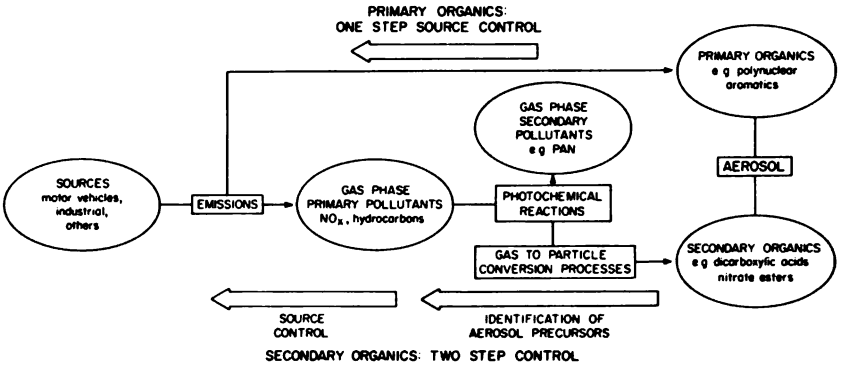


FIGURE 3-1 Control strategies for primary and secondary organic aerosols.

importance, the gas-to-particle conversion of organic pollutants has received much less attention than other aspects of air pollution. This chapter deals with the identification of secondary organic aerosols in the atmosphere and the physical and chemical aspects of their formation. The relative importance of their gas-phase hydrocarbon precursors, including naturally emitted terpenes, is discussed, and the contribution of hydrocarbon photochemical reactions to the formation of inorganic sulfate and nitrate aerosols is briefly reviewed.

ATMOSPHERIC OBSERVATIONS

Analytic Techniques

Identifying the chemical composition of urban aerosols, in which hundreds of components may be present, is a formidable task for the analyst. Although rather simple techniques can be used to quantify the aerosol composition by elemental analysis (e.g., organic carbon) and groups of compounds (e.g., nitrates, sulfates, and organics or aliphatics, aromatics, and oxygenates in the organic fraction), only more sophisticated techniques can provide individual identification of compounds present at very low concentrations. The necessary selectivity and sensitivity are then gained at the expense of a general picture of the aerosol composition.

Aerosol samples can be analyzed without further preparation for organic carbon[78] and for carbon, hydrogen, oxygen, and nitrogen (CHON)[160] by infrared spectroscopy,[39,212] by photoelectron spectroscopy,[155] and by mass spectrometry.[195] However, organic analysis is

generally conducted after solvent extraction of samples collected on glass-fiber filters.[118] The organic extract can be further fractionated by thin-layer, paper, liquid, or column chromatography, by ion-exchange chromatography,[111] or by separation in various solvents.[215] The extracts or fractions are then analyzed for organic carbon[78] or for CHON[33,38,143,178,188] by infrared,[33,38,55,134,143,178] ultraviolet fluorescence,[50,71,101,166,187,189,200,205] gas chromatography, high-pressure liquid chromatography,[141] or mass spectrometry.[19,135,165,184,201] For example, more than 70 polynuclear aromatics have been identified by mass spectrometry.[127] Some of these techniques have been used in conjuction with size distribution measurements.[33,39,143,155,166] Vacuum sublimation[6] and, more recently, ultrasonic extraction[68] have been proposed as alternative techniques for the time-consuming process that uses the Soxhlet extractor. However, most of the later analyses depend on the extraction efficiency of the organic solvent. Although nonpolar solvents were most widely used in the past (cyclohexane, but benzene for the National Air Surveillance Networks[221]), it has been found recently[78] that solvent extraction efficiencies depend on solvent polarity characteristics (Figure 3-2) and

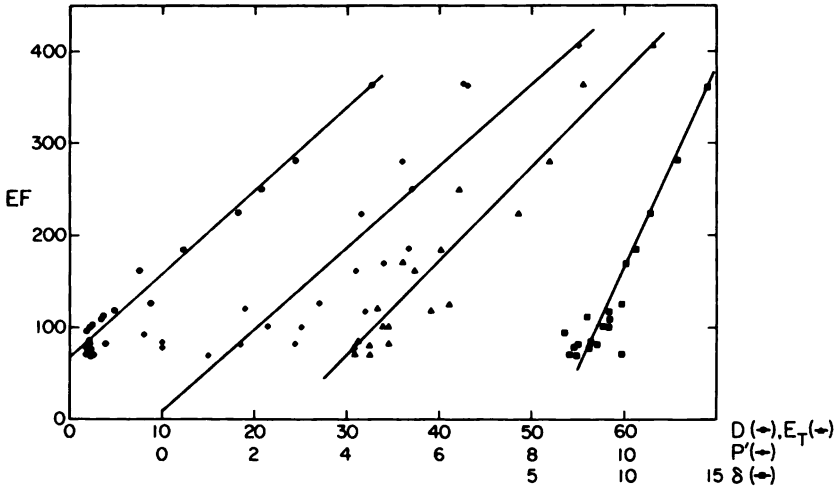


FIGURE 3-2 Solvent extraction efficiencies (EF) as functions of dielectric constants (D), solubility parameters (δ), and polarity parameters (P′ and E_T). Solvents studied: silicon tetrachloride, carbon disulfide, *n*-pentane, Freon 113, cyclopentane, *n*-hexane, carbon tetrachloride, diethylether, cyclohexane, isooctane, benzene (reference, EF = 100), toluene, trichloroethylene, diethylamine, chloroform, triethylamine, methylene, chloride, tetrahydrofuran, 1,4-dioxane, pyridine, 2-propanol, acetone, ethanol, methanol, dimethyl sulfoxide, and water. Reprinted with permission from Grosjean.[78]

that binary mixtures of a polar solvent and a nonpolar one can extract up to 48% more organics than benzene alone from samples collected in areas of heavy photochemical pollution (Table 3-1).

Primary Organic Aerosols

Primary organics are emitted to the atmosphere by industrial sources (oil refineries, chemical plants, producers and users of solvents and plasticizers), vehicles (as a result of incomplete fuel combustion, oxygenated degradation products of lubricating oil, polymers from tires), and agricultural activities (use of pesticides). An exhaustive literature survey is beyond the scope of this section, but can be found in *Air Quality Criteria for Particulate Matter;*[219] many useful references are also available.

Among the identified primary organics are linear and branched alkanes and alkenes, substituted benzenes and styrenes, quinones, acridines, quinolines, phenols, cresols, phthalates, fatty acids, carbonyl compounds, and some pesticide compounds. Diurnal concentration profiles of primary pollutants emitted in auto exhaust parallel vehicle activity and show two characteristic peaks associated with morning and evening traffic periods.[122] Because of their potential carcinogenic activity, polynuclear aromatic hydrocarbons have been extensively studied (see Melton *et al.*[141]). Recent studies have reported their identification in automobile exhaust,[19] their ambient distribution with respect to particle size,[166] the close relation between their ambient concentrations and automobile traffic in the Los Angeles area,[69,71] and their possible loss during sampling as a function of their volatility.[171] The recent observation of chemiluminescence associated with particles in auto exhaust[207] is of interest, in view of the fact that the carcinogenic action of electronically excited molecules is much greater than that of the corresponding ground state.[186]

Secondary Organic Aerosols

Organic aerosols formed by gas-phase photochemical reactions of hydrocarbons, ozone, and nitrogen oxides have been identified recently in both urban and rural atmospheres. Aliphatic organic nitrates,[79,143,156] such dicarboxylic acids as adipic and glutaric acids,[156] carboxylic acids derived from aromatic hydrocarbons (benzoic and phenylacetic acids) and from terpenes emitted by vegetation, such as pinonic acid from α-pinene,[240] have been identified. The most important contribution in this field has been that of Schuetzle *et al.*, who used computer-controlled

TABLE 3-1 Extraction Efficiencies (EF) and Organic Carbon Extraction Efficiencies (OCEF) of Various Solvents and Binary Mixtures for Atmospheric Aerosol Samples[a]

Solvent	EF	OCEF	Binary Mixture[b,c]		EF	OCEF
Silicon tetrachloride	69	—	Freon 113	2-Propanol	157	119
Carbon disulfide	70	69		Acetone	191	131
n-Pentane	71	—		Ethanol	198	134
Freon 113	76	76		Methanol	220	147
Cyclopentane	77	—	Cyclohexane	2-Propanol	210	125
n-Hexane	78	78		Acetone	230	136
Carbon tetrachloride	81	80		Ethanol	242	130
Diethylether	82	80		Methanol	252	144
Cyclohexane	84	84	Isooctane	2-Propanol	220	131
Isooctane	95	94		Acetone	241	132
BENZENE (reference)	100	100		Ethanol	255	130
Toluene	102	—		Methanol	281	144
Trichloroethylene	109	108	Benzene	2-Propanol	225	135
Diethylamine	112	—		Acetone	240	140
Chloroform	118	114		Ethanol	268	135
Triethylamine	120	116		Methanol	301	145
Methylene chloride	126	120	Chloroform	2-Propanol	218	131
Tetrahydrofuran	100-260	54-115		Acetone	248	137
1,4-Dioxane	112-262	47-110		Ethanol	260	139
Pyridine	115-295	52-116		Methanol	312	149
2-Propanol	70-327	61-167	Methylene chloride	2-Propanol	219	140
Acetone	71-388	60-170		Acetone	249	144
Ethanol	95-421	64-190		Ethanol	260	142
Methanol	85-560	69-200		Methanol	320	151
Dimethyl sulfoxide	90-520	—				
Water	183-594	74-230				

[a] Data from Grosjean.[78]
[b] 50:50% by volume.
[c] Polar-solvent EF and OCEF measured for the same sampling period:
2-propanol: 230 and 107 acetone: 260 and 120
ethanol: 291 and 122 methanol: 374 and 144

$$EF_i = 100 \times \frac{\text{Total extracted, solvent } i}{\text{Total extracted, benzene, same sample}}$$

$$OCEF_i = 100 \times \frac{\text{Extracted organic carbon, solvent } i}{\text{Extracted organic carbon, benzene, same sample}}$$

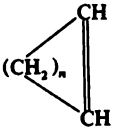
high-resolution mass spectrometry and thermal analysis,[194-196] the only available technique that combines the resolving power necessary to identify individual pollutants at very low concentrations with the ability to detect the wide range of compounds (metals, inorganics, organics) present in polluted atmospheres. Organic aerosols of secondary origin identified by the University of Washington group—Schuetzle *et al.*[194-196] and Knights *et al.*[122]—are listed in Tables 3-2 and 3-3. It can be seen that most of them are difunctional compounds, most probably formed in the atmosphere by photochemical oxidation of cyclic alkenes and alkadienes. The sensitivity of the technique permits obtaining diurnal variations of primary and secondary organics from 2-h size-resolved samples. Diurnal concentration profiles of secondary organic aerosols follow ozone variations closely. Such typical profiles are illustrated for some selected compounds in Figure 3-3.

Relative Abundance of Primary and Secondary Organic Aerosols

If primary organic aerosols are preponderant in urban areas where photochemical reactions are not significant, secondary organic aerosols are predominant in photochemically polluted areas, such as the south California basin. As part of the recently completed California Air Characterization Study (ACHEX),[94-98,100] a close relation between visibility reduction and photochemical smog was observed at eight sampling locations where the extinction coefficient, b_{scat}, due to light scattering from airborne particles was measured when ozone concentration was at its maximum (Figure 3-4). Good correlations were obtained between the organic aerosol fractions,[5,79] their carbonyl infrared absorption band intensities,[79] and ozone concentrations.

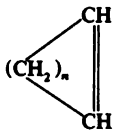
With the concept of chemical-element balance developed by Friedlander,[57] secondary organic aerosols have been estimated to account for 82% and 76% of the aerosol carbon balance in 24-h and 2-h samples (Pasadena, California, September 29, 1973[61]). During a severe photochemical episode, secondary organics reached up to 95% of the total organics (Pasadena, July 25, 1973; maximal ozone concentration, 0.67 ppm[79]). In such extreme situations, all secondary material resulting from gas-to-particle conversion (secondary organics plus nitrates plus sulfates) accounts for up to 95.5% of the total aerosol mass (Figure 3-5). Size distribution measurements on short-period samples, first developed by Lundgren,[133] show that difunctional compounds listed in Tables 3-2 and 3-3 accumulate in the submicrometer range and that a significant fraction of the organic carbon is found below 0.5 μm.[94-98] These results clearly illustrate the potential health hazards associated with human

TABLE 3-2 Secondary Organic Aerosols[a]

Compounds Identified	Possible Gas-Phase Hydrocarbon Precursors
Aliphatic Multifunctional Compounds	
1. X—$(CH_2)_n$—Y (n = 3,4,5):	1. Cyclic olefins
X / Y	
COOH / CH_2OH	
COOH / COH	
COOH / COOH	
COOH / CH_2ONO	
or[b] COH / CH_2ONO_2	
COH / CH_2OH	
COH / COH	and/or diolefins
COOH / COONO	
or[b] COH / $COONO_2$	$>C{=}CH{-}(CH_2)_n{-}CH{=}C<$
COH / COONO	
COOH / $COONO_2$	
COOH / CH_2ONO_2	
2. Others:	2. Not known; possibly from aromatic ring cleavage
$CH_2OH{-}CH{=}C(COOH){-}CHO$	
$CH_2OH{-}CH_2{-}CH{=}C(COOH){-}CHO$	
$CHO{-}CH{=}CH{-}CH(CH_3)CHO$	
$CH_2OH{-}CH{=}CH{-}CH{=}C(CH_3)CHO$	
$C_5H_8O_3$ isomers[b]	
Nitrocresols	
$C_6H_6O_2$ isomers[b]	
Aromatic Monofunctional Compounds	
3. $C_6H_5{-}(CH_2)_n{-}COOH$ (n = 0,1,2,3)	3. Alkenylbenzenes $C_6H_5{-}(CH_2)_n{-}CH{=}CHR$; also toluene for C_6H_5COOH
4. $C_6H_5{-}CH_2OH$	4. Toluene, styrene, other monoalkylbenzenes?
C_6H_5CHO	
Hydroxynitrobenzyl alcohol	
Terpene-Derived Oxygenates	
5. Pinonic acid	5. α-Pinene
Pinic acid	
Norpinonic acid	
6. Isomers of pinonic acid:[b]	6. Other terpenes?
$C_9H_{14}O_2$ isomers	
$C_{10}H_{14}O_3$ isomers	
$C_{10}H_{16}O_2$ isomers	

[a] Data from Knights *et al.*;[122] compounds identified at West Covina, California, July 24, 1974.
[b] Isomers not resolved by mass spectrometry.

TABLE 3-3 Relative Importance of Aliphatic and Aromatic Precursors[a]

Gas-Phase Hydrocarbon Precursors	Secondary Organic Aerosols				
	$X—(CH_2)_n—Y$		Concentration,[b] $\mu g/m^3$		
	X	Y	n = 3	n = 4	n = 5
	COOH	CH_2OH	2.18	3.40	0.65
	COOH	COH	1.39	2.59	0.82
	COOH	COOH	1.35	0.78	0.15
	or { COOH	CH_2ONO	1.01	0.40	0.27
	or { COH	CH_2ONO_2			
	COH	CH_2OH	0.31	0.40	0.13
	COH	COH	0.30	0.24	—
	or { COOH	COONO	0.14	0.24	—
	or { COH	$COONO_2$			
	COH	COONO	1.01	0.14	—
or/and	COOH	$COONO_2$	—	—	—
	COOH	CH_2ONO_2	0.12	0.15	—
>C=CH—$(CH_2)_n$—CH=C<	*Total*:		7.81	8.34	2.02
	COOH—CH_2—COOH		0.15		
	COOH—$(CH_2)_2$—COOH		0.57		
	Total difunctional compounds: 18.89				
C_6H_5—CH=CHR	C_6H_5—$(CH_2)_n$—COOH		n = 0: 0.38		
			n = 1: 0.41		
			n = 2: 0.52		
			n = 3: 0.03		
	Total from aromatics:		1.34		

[a] Of aerosols in Pasadena, California, September 22, 1972; sampling period, 7:30 a.m. to 12:35 p.m. Adapted from Schuetzle *et al.*[196]

[b] The same response factor (that of adipic acid) was used for all difunctional compounds.

exposure to high concentrations (≥ 300 $\mu g/m^3$) of respirable secondary particles during acute photochemical episodes.

Gas-Particle Distribution Factors

The extent of gas-to-aerosol conversion of secondary pollutants can be estimated by measuring gas-particle distribution factors for carbon, nitrogen, and sulfur species.[79] For example, $f_C = P/(P + G)$, where P = particulate organic carbon ($\mu g/m^3$ as carbon) and G = gas-phase

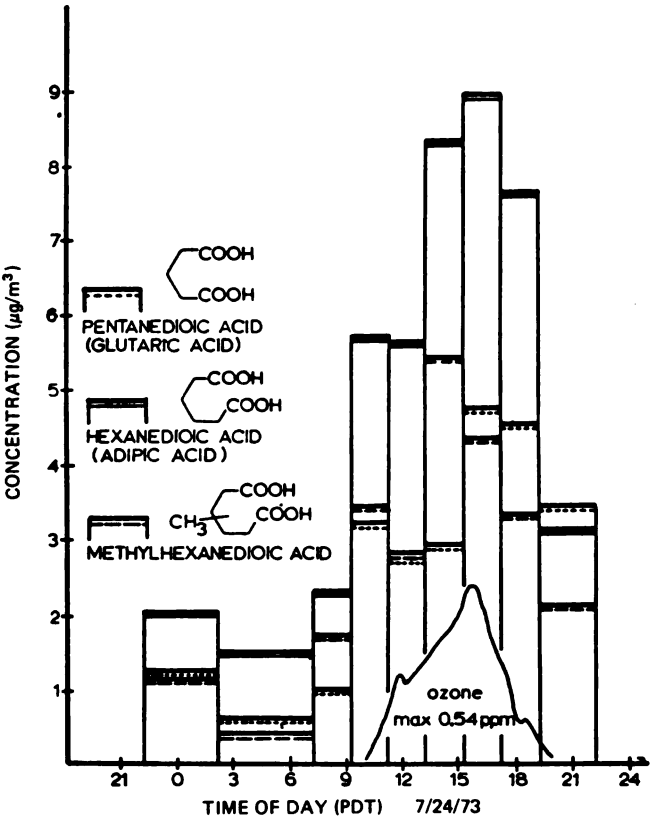


FIGURE 3-3 Diurnal profiles of dicarboxylic acids in particles less than 1 μm in diameter. West Covina, California (40 km east of Los Angeles). Reprinted with permission from Knights *et al.*[122]

reactive hydrocarbons (total hydrocarbons − [methane + acetylene]) converted from ppm as methane to $\mu g/m^3$ as carbon. It can be seen from measured values of f_N, f_S, and f_C (Table 3-4) that the extent of conversion of organic gases is much lower than that of sulfur and nitrogen oxides. Similar f_N, f_S, and f_C values were calculated from ACHEX data.[94-98,100] The maximal f_C, measured over a 1-h period when the highest ozone concentration in several years was recorded in Pasadena, was only about 6%, and average values are in the range of 1-2%. This, combined with estimates of air-mass travel times based on air trajectory analysis,[233] provides an upper limit of about 2%/h for the ambient gas-to-aerosol conversion rate of organic gases in photochemically polluted atmospheres.[79] Although organics always account for an important fraction of

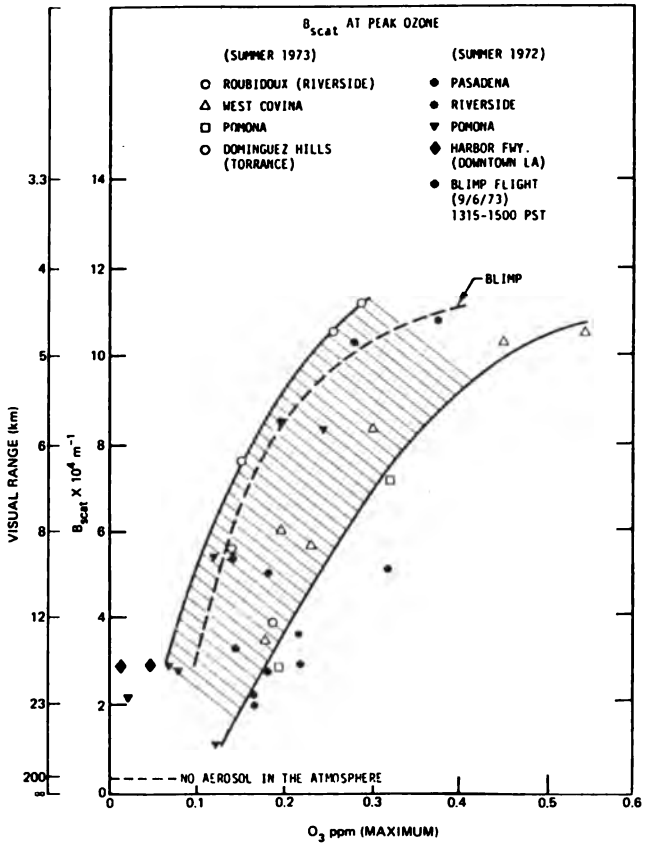


FIGURE 3-4 Correlation between B_{scat} and maximal ozone concentration. Based on 2-h averaged data taken in the Los Angeles, California, area. Reprinted with permission from Hidy.[94]

urban aerosols, only a small fraction of the organic gas is converted to particulate material. This, and the fact that most of the secondary organics identified in ambient aerosols are difunctional compounds that can be formed from a few possible precursors (such as cyclic olefins and diolefins), leads us to review next data obtained in smog-chamber studies of organic aerosol formation from olefinic and other hydrocarbons.

SMOG-CHAMBER STUDIES

Since the pioneering work of Haagen-Smit and co-workers more than two decades ago,[82-84] the chemistry of polluted air has been simulated in

laboratory studies and a good understanding of the reactions occurring in the complex atmospheric mix has been gained by investigating the behavior of simple pollutant mixtures under controlled conditions. However, aerosol formation in smog-chamber experiments has not been studied as much as gas-phase photochemistry. In most studies, aerosol formation was observed only by monitoring the light-scattering increase during the experiment, and aerosol chemical composition was not sought. Moreover, a quantitative comparison of data obtained by all investigators is practically impossible, in view of the differences in smog-chamber design and material, mode of irradiation, initial reactant concentrations, available instrumentation, etc. Systems investigated included ozone-hydrocarbon reactions in the dark, in irradiated mixtures of hydrocarbon with nitric oxide or nitrogen dioxide or both, in "clean" air, in ambient air, and with or without addition of sulfur dioxide, water, and ammonia. More than 80 compounds that have been investigated are listed in Table 3-5.

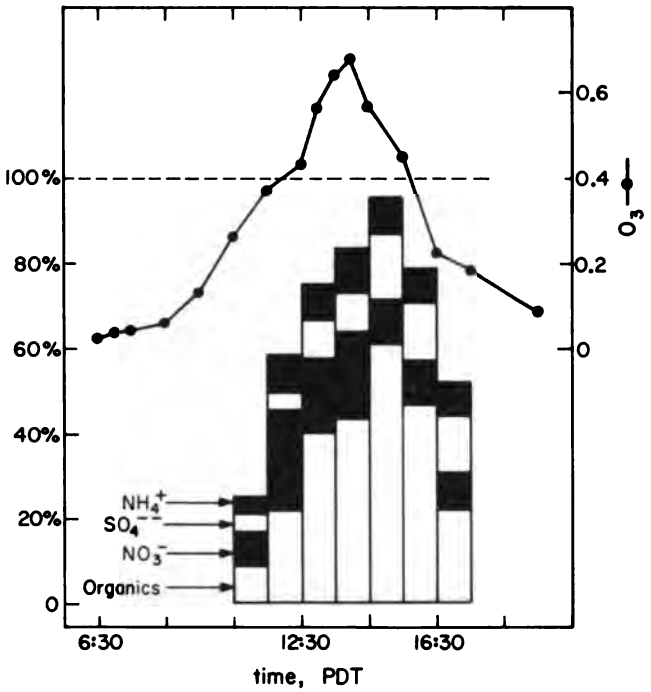


FIGURE 3-5 Hourly variations of secondary aerosol: organics, nitrates, sulfates, and ammonium as percent of total aerosol. Pasadena, California, July 25, 1973. Reprinted with permission from Grosjean and Friedlander.[79]

TABLE 3-4 Gas-Particle Distribution Factors for Organics (f_C), Nitrates (f_N), and Sulfates (f_S)[a]

Sampling Date	Concentration, $\mu g/m^3$			Gas-Particle Distribution Factor		
	Organics	NO_3^-	$SO_4^=$	f_C	f_N[b]	f_S[c]
7/12/73	22.7	3.9	49.8	0.014	0.013	—
	37.2	5.3	31.8	0.0235	0.018	0.324
	26.3	4.5	44.5	0.0175	0.017	0.333
	45.5	4.0	24.4	0.0300	0.018	0.230
	28.4	2.5	—	0.0120	0.012	—
	11.6	2.9	—	0.0090	0.015	—
7/25/73	50.5	7	—	0.020	0.019	—
	79.1	8.5	—	0.0365	0.023	—
	54.0	12	8.6	0.029	0.041	0.16
	20.0	19.7	10.7	0.009	0.074	0.13
	47.0	50	9.2	0.021	0.165	0.18
	118.5	53	24.4	0.0385	0.152	0.36
	132.5	64	27.7	0.0515	0.168	0.37
	141.0	25.4	34.1	0.0615	0.077	0.39
	85.6	19.5	24.4	0.0495	0.061	0.25
	28.7	11.2	18.5	0.020	0.037	0.26
	37.3	5.0	—	0.0235	0.019	—
10/17/73	16.6	6.3	10.3	0.0042	0.012	0.393
	15.1	3.8	9.0	0.0079	0.011	0.171
	43	14.3	12.2	0.0205	0.032	0.466
	15.1	1.8	5.4	0.0088	0.007	0.297
	8.1	4.0	—	0.0066	0.017	—

[a] Data from Grosjean and Friedlander.[79] Sampling in Pasadena, California. Sampling time: 7/12, 8:00-12:00, 12:00-14:00, 14:00-16:00, 16:00-18:00, 18:00-20:00, and 20:00-24:00; 7/25, 1 h 7:30-17:30, last sample 17:30-19:30; 10/17, 8:00-10:00, 10:00-12:00, 12:00-13:00, 13:00-15:00, and 15:00-17:00.

[b] $f_N = [NO_3^-]_P/[NO_3^-]_P + [NO_x]_G$; units, $\mu g/m^3$ as NO_2.

[c] $f_S = [SO_4^=]_P/[SO_4^=]_P + [SO_2]_G$; units, $\mu g/m^3$ as SO_2. $[X]_P$ = particulate-phase concentration: $[X]_G$ = gas-phase concentration.

Aerosol Formation from Different Types of Hydrocarbons

Extensive discussion of data represented in Table 3-5 is beyond the scope of this review, and only the most important aspects of aerosol formation are reported here and in the next few sections. Many of the conclusions presented thereafter were reached in the early studies reported by the Stanford Research Institute, Air Pollution Foundation, and Franklin Institute groups.[24,47,49,51,169,177,191-193] Results obtained in the

TABLE 3-5 Compounds Investigated in Smog-Chamber Studies, with References

Compound	References
I. OLEFINS	
Alkenes:	
Ethylene	3, 51, 76, 88, 139, 142, 169, 177, 191, 238
Propylene	23, 35, 51, 76, 88, 123, 139, 204, 229
1-Butene	3, 23, 130, 142, 169, 197, 229, 236–238
cis- and *trans*-2-Butene	34, 164, 169, 177, 191, 214, 229
Isobutene	51, 169, 177, 191, 229
2-Methyl-1-butene	229
2-Methyl-2-butene	35, 142, 177, 191, 192, 229, 238
3-Methyl-1-butene	169, 229
1-Pentene	12, 142, 169, 177, 191, 229, 238
cis- and *trans*-2-Pentene	34, 35, 169, 229
1-Hexene	12, 23, 24, 34, 130, 131, 169, 180, 214, 224, 229, 237
cis- and *trans*-3-Heptene	169, 180, 229
5-Methyl-1-hexene	34
4-Methyl-1-pentene	35, 229
cis-3-Methyl-2-pentene	169
2,3-Dimethyl-2-butene	51, 177, 191, 229
1-Heptene	76, 142, 157, 214, 236, 238, 240
cis- and *trans*-3-Heptene	76, 142, 169, 214, 238
5-Methyl-1-hexene	157
1-Octene	23, 66, 67, 76, 157
trans-4-Octene	157
2,4,4-Trimethyl-1-pentene (isooctene)	76, 169
1-Decene	23
1-Dodecene	23, 131
Cyclic Olefins:	
Cyclopentene	76, 169
Cyclohexene	23, 24, 66, 76, 131, 142, 157, 169, 177, 180, 191, 198, 214, 238, 240
Diolefins:	
1,3-Butadiene	3, 76, 169, 177, 191
Isoprene	76
1,3-Hexadiene	131
1,5-Hexadiene	131, 157, 169, 180
1,6-Heptadiene	157
2-Methyl-1,5-hexadiene	157
1,7-Octadiene	157
2,6-Octadiene	157
Dicyclopentadiene	169

TABLE 3-5 (Cont.)

Compound	References
Other Olefins:	
Indene	157
Turpentine	66
Styrene	76
α-Pinene	76, 131, 157, 180, 181, 198, 240
II. PARAFFINS	
Methane	76, 177, 191
Cyclopropane	169
n-Butane	142, 169, 238
n-Pentane	177, 191
2,2-Dimethylpropane	177, 191
Cyclopentane	177, 191
2-Methylbutane	142, 177, 191, 238
2-Methylpentane	169
3-Methylpentane	3
n-Hexane	3
Cyclohexane	3, 142, 169, 180, 238
2,4,4-Trimethylpentane (isooctane)	3, 76, 142, 236, 238
2,6-Dimethylheptane	157
III. ACETYLENIC	
1-Butyne	177, 191
IV. AROMATICS	
Benzene	32, 66, 76, 124, 142, 177, 191, 238
Toluene	3, 32, 66, 124, 131, 142, 157, 177, 180, 191, 198, 235–238, 240
o-, *m*-, and *p*-Xylene	3, 32, 66, 124, 131, 142, 157, 177, 191, 238
Ethylbenzene	76, 124, 157, 177, 191
1,3,5-Trimethylbenzene (mesitylene)	3, 76, 124, 142, 157, 214, 237, 238
Isopropylbenzene	66, 124
1,2,4-Trimethylbenzene	124
1,2,3-Dimethylbenzene	124
3-Ethyltoluene	124
1,2-Diethylbenzene	142, 238
tert-Butylbenzene	142, 238
n-Butylbenzene	142, 238
1,4-Diethylbenzene	124
1,2,3,5-Tetramethylbenzene	124

TABLE 3-5 (Cont.)

Compound	References
V. ALDEHYDES	
Formaldehyde	3
Propionaldehyde	3
Hexanal	142, 238
Heptanal	157
Benzaldehyde	66
Glutaraldehyde	157
VI. KETONES	
Mesityl oxide	235
Isophorone	235
Methylisobutylketone	235
Cyclohexanone	180

presence of sulfur dioxide are discussed toward the end of this chapter.

In the absence of sulfur dioxide, aerosol formation depends strongly on the type of hydrocarbon precursor studied. The following qualitative trends are observed:

- Most paraffins do not generate aerosol, even when irradiated at high concentrations. However, some aerosol can be formed from the more "reactive" branched paraffins having more than six carbon atoms (such as isooctane) after long irradiation periods.
- Acetylenics do not form aerosol.
- All unsaturated compounds can form organic aerosol when reacting with ozone at high concentrations, as observed by Wei and Cvetanović.[229] However, studies conducted at much lower alkene concentration (1-10 ppm) show a marked effect of alkene chain length on aerosol formation. Alkenes with fewer than six carbon atoms do not form aerosol; those with six or more carbon atoms form aerosol when they yield (after rupture of the double bond) a fragment with at least five carbon atoms. For example, 1-heptene forms much more aerosol than 3-heptene, and 2,4,4-trimethyl-1-pentene (isooctene) forms more aerosol than its isomer *trans*-4-octene. Amounts of aerosol formed from 1-alkenes increase regularly with the number of carbon atoms (Figure 3-6).

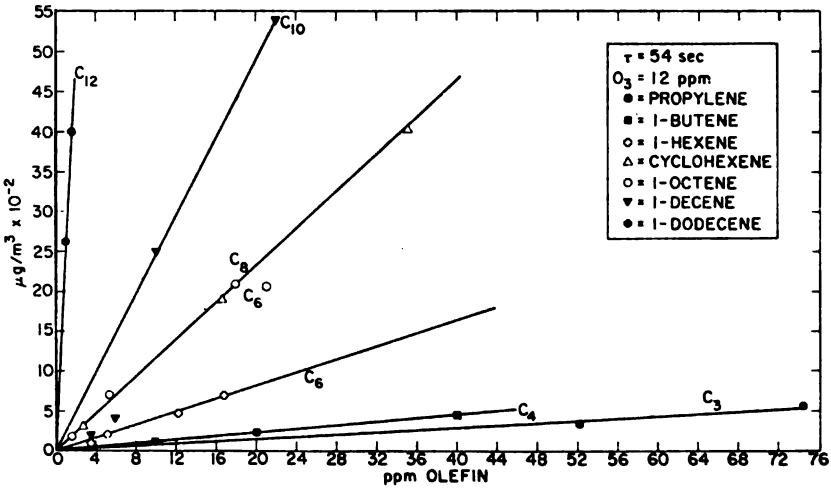


FIGURE 3-6 Mass concentration of aerosol formation from olefin-ozone reaction as a function of olefin concentration. τ, residence time in the flow reactor. Reprinted with permission from Burton *et al.*[23]

- Cyclic olefins and diolefins form much more aerosol than 1-alkenes that have the same number of carbon atoms (for example, cyclohexene $\gg$ 1-hexene, and 1,7-octadiene $\gg$ 1-octene). The same effect of chain length and double-bond position is observed for diolefins (1,7-octadiene > 1,6-heptadiene > 1,5-hexadiene, and 1,7-octadiene $\gg$ 2,6-octadiene). Heavier unsaturated cyclic compounds, such as indene and terpenes, form even more aerosol.
- Conflicting results have been reported for aromatic compounds. Aerosol formation has been reported from benzene, toluene, and other alkylbenzenes by several investigators,[32,198,238] whereas no aerosol formation was observed in other studies.[157,177] This merits further investigation, in view of the large fraction of aromatic hydrocarbons present in polluted atmospheres.
- Carbonyl compounds (ketones, C_{1-7} aldehydes, dialdehydes) do not generate aerosol.
- Data on aerosol formation from irradiated automobile exhaust[47,49,85,193,238,239] confirm the marked sensitivity to hydrocarbon type observed in individual hydrocarbon studies. Aerosol formation increases with the olefinic and aromatic[239] fuel content. However, changes in mode of engine operation (acceleration, idle) and inorganic variables (sulfur dioxide, relative humidity) have a more pronounced effect on aerosol formation than change in fuel composition.[238]

Photochemical Reactivity and Aerosol Formation Ability

We have listed in Table 3-6 the relative rates of conversion of nitric oxide to nitrogen dioxide measured by Glasson and Tuesday[64] and the amounts of aerosol formed, relative to cyclohexene, from data of Renzetti and Doyle,[177] Prager *et al.*,[169] Groblicki and Nebel,[76] Wilson *et al.*,[142,238] O'Brien *et al.*,[157] and Grosjean (unpublished data) for different types of hydrocarbon precursors. Table 3-7 summarizes the experimental conditions for data presented in Table 3-6. As mentioned before, data reported by these investigators are in good qualitative agreement for all precursor classes except aromatics. It is clear from the data in Table 3-6 that there is no straightforward relation between the amount of aerosol formed and the gas-phase photochemical reactivity of the various precursors. The overall hydrocarbon photochemical reactivity is a complex function of the rate constants of reaction with various species (ozone, oxygen atom, hydroxyl and hydroperoxy radicals, other free radicals) during the photooxidation process. Therefore, relative rates of hydrocarbon decay, of ozone (or oxidant) formation, or of conversion of nitric oxide to nitrogen dioxide can be used as empirical photochemical reactivity (PR) indexes. As we will see in a more quantitative manner later, the ability of a given hydrocarbon to form aerosol depends on the *nature* of the products formed (chemical dependence) and on their *volatility* (physical dependence), and an aerosol formation ability (AFA) index can be derived from physical and chemical aerosol data. Therefore, the amount of aerosol formed appears to be the product of two terms:

[Aerosol] = (Photochemical reactivity) × (Aerosol formation ability).

If we remember that product volatility decreases regularly when the size of the hydrocarbon precursor increases, this relation provides a rational approach for a qualitative estimate of aerosol formed from any type of precursor. Aerosol formation can be "PR-controlled" (paraffins, acetylenics; PR = 0) or "AFA-controlled" (low-molecular-weight alkenes, carbonyl compounds; high PR, but AFA = 0). In the same way, aerosol formation from 1-alkenes having similar PR is controlled by their AFA factor, which increases with chain length; whereas relative amounts of aerosol formed from hydrocarbons leading to products of similar volatility (for example, *n*-heptane, toluene, and 1-heptene, with similar AFA factors) are controlled by their relative gas-phase reactivities. Therefore, hydrocarbons providing the bulk of secondary organic aerosols in the atmosphere are not necessarily those prevailing in oxidant formation. An important consequence is that control of atmospheric organic aerosol concentrations can be achieved by two types of control strategies: *specific* control of precursors with high AFA may prove to be as efficient as *total* hydrocarbon and nitrogen oxides emission control.

TABLE 3-6 Aerosol Formation and Gas-Phase Reactivity

Precursor	Aerosol Formation Ability[a]											Gas-Phase[a,b] Reactivity
	Without Sulfur Dioxide						With Sulfur Dioxide					
	[b]	[c]	[d]	[e]	[f]	[g]	[b]	[c]	[d]	[e]	[g]	
1. OLEFINS												
Alkenes:												
Ethylene	0	2.8	—	—	—	—	12.6	63	44	1	—	48.5
Propylene	—	—	12.4	—	—	—	—	—	69	—	—	100
1-Butene	—	1.4	—	—	—	—	—	81	—	1	—	83
cis-2-Butene	—	1.4	—	—	—	—	—	86				202
trans-2-Butene	0						144		—	—	—	320
Isobutene	0	0	—	—	—	—	40	—	—	—	—	100
2-Methyl-2-butene	3.6	—	—	—	—	—	96	—	—	2.6	—	543
3-Methyl-1-butene	—	2.8	—	—	—	—	—	—	—	—	—	77
1-Pentene	0.9	2.8	—	—	—	—	96	87	—	2.7	—	60
cis- and *trans*-2-Pentene	—	0	—	—	—	—	—	86	—	—	—	187
1-Hexene	—	1.4	—	—	—	—	—	96	—	—	—	48.5
cis- and *trans*-2-Hexene	—	7	—	—	—	—	—	86	—	—	—	171 (*trans*)
cis-3-Methyl-2-pentene	—	5.6	—	—	—	—	—	—	—	—	—	—
2,3-Dimethyl-2-butene	2.7	—	—	—	—	0	100	—	—	—	—	10^3
1-Heptene	—	—	10.5	≤ 0	1	—	—	—	—	6.5	—	43
cis- and *trans*-3-Heptene	—	12.6	0	—	—	—	—	96	—	11.7	—	134 (*trans*)

5-Methyl-1-hexene	—	—	—	—	1	—	—	—	—	—	—	—
1-Octene	—	—	—	—	1	—	—	—	88	—	—	42.8
trans-4-Octene	—	—	—	—	1	—	—	—	—	—	—	94.5
Isooctene	—	94	28	—	—	—	—	94	81	—	—	—
Cyclic Olefins:												
Cyclopentene	—	75	58	—	—	—	—	—	—	—	—	657
Cyclohexene	100	100	100	—	100	100	107	100	91	100	—	100
α-Pinene	—	—	140	—	200	—	—	—	137	—	—	—
Diolefins:												
1,3-Butadiene	3.6	33	—	—	—	—	150	91	85	—	—	123
Isoprene	—	—	—	—	—	—	—	—	75	—	—	106
1,5-Hexadiene	—	104	—	—	45	—	—	111	—	—	—	—
1,6-Heptadiene	—	—	—	—	178	—	—	—	—	—	—	—
2-Methyl-1,5-hexadiene	—	—	—	—	122	—	—	—	—	—	—	—
1,7-Octadiene	—	—	—	—	200	—	—	—	—	—	—	—
2,6-Octadiene	—	—	—	—	1	—	—	—	—	—	—	—
Dicyclopentadiene	—	124	—	—	—	—	—	111	—	—	—	—
II. PARAFFINS												
Methane	0	—	—	—	—	—	0	—	25	—	—	2.86
Cyclopropane	—	4.3	—	—	—	—	—	—	—	—	—	—
n-Butane	—	—	—	—	—	—	—	2.8	—	.55	—	21.2
n-Pentane	0	—	—	—	—	—	0	—	—	—	—	26.5
2,2-Dimethylpropane	0	—	—	—	—	—	0	—	—	—	—	10.0
Cyclopentane	0	—	—	—	—	—	0	—	—	—	—	26.2
2-Methylbutane	0	—	—	—	—	—	0	—	—	0.65	—	28.0
2-Methylpentane	—	—	—	—	—	—	—	0	—	—	—	26.8
Cyclohexane	—	0	—	—	—	—	—	—	—	1.4	—	27.6
Isooctane	—	—	6.2	—	—	—	—	—	12.4	0.25	—	21.1
2,6-Dimethylheptane	—	—	—	—	1	—	—	—	—	—	—	—

TABLE 3-6 (Cont.)

Precursor	Aerosol Formation Ability[a]											Gas-Phase[a,h] Reactivity
	Without Sulfur Dioxide						With Sulfur Dioxide					
	[b]	[c]	[d]	[e]	[f]	[g]	[b]	[c]	[d]	[e]	[g]	
III. AROMATICS												
Benzene	0	—	10.5	—	—	—	4.5	—	—	10.8	—	9.4
Toluene	0	—	—	8.5	—	0	9	—	—	8.6	—	37.2
o-Xylene	—	—	—	—	9	—	—	—	—	—	—	74.5
m-Xylene	—	—	—	—	—	—	—	—	—	29.4	—	106
p-Xylene	0	—	—	—	—	—	0	—	—	—	—	60
Ethylbenzene	0	—	—	—	1	—	12.6	—	69	—	—	34.3
Mesitylene	—	—	—	9	1	—	—	—	50	9.0	—	146
1,2-Diethylbenzene	—	—	—	—	—	—	—	—	—	23.4	—	48.5
tert-Butylbenzene	—	—	—	—	—	—	—	—	—	24.4	—	16.8
n-Butylbenzene	—	—	—	—	—	—	—	—	—	22.2	—	—
IV. ALDEHYDES												
Hexanal	—	—	—	—	—	—	—	—	—	0	—	—
Heptanal	—	—	—	—	0	—	—	—	—	—	—	—
Glutaraldehyde	—	—	—	—	0	—	—	—	—	—	—	—
V. ACETYLENIC												
1-Butyne	0	—	—	—	—	—	0.9	—	—	—	—	—

[a] Relative to cyclohexene = 100. [b] Data from Renzetti and Doyle.[177] [c] Data from Prager *et al.*[169] [d] Data from Groblicki and Nebel.[76] [e] Data from Wilson *et al.*[142,238] [f] Data from O'Brien *et al.*[157] [g] Data from Grosjean (unpublished data). [h] Data from Glasson and Tuesday;[64] nitric oxide photooxidation rates.

TABLE 3-7 Experimental Conditions in Smog-Chamber Studies

Smog Chamber	Renzetti and Doyle[177]	Prager *et al.*[169]	Groblicki and Nebel[76]	Wilson *et al.*[142,238]	O'Brien *et al.*[157]	Grosjean (unpublished data)
Material	Pyrex	Glass	Stainless steel	Glass	Glass	Teflon
Volume	50 liters	640 liters	300 ft^3	200 liters	1,100 ft^3	80 m^3
Irradiation	Mercury lamps	Mercury lamps	Fluorescent tubes	Black fluorescent lamps	Black fluorescent lamps	Solar
Diluent gas	Clean air	Air	Clean air + CO at 50 ppm	Clean air	Clean air	Ambient air
Hydrocarbon impurities	?	?	< 0.6 ppm as hexane	Produce ozone at ⩽ 3 ppb	Nonmethane hydrocarbon at 1 ppb	Ambient (high)
Reactor type	Stirred flow	Stirred flow and batch	Batch	Flow	Batch	Batch
Stirring	Yes	Yes	Yes	No	No	No
Temperature	Ambient	Ambient	95° F	82° F	22° C	Ambient
Initial Concentrations						
Hydrocarbon, ppm	3	10	4	3	2	1
Nitric oxide, ppm	1	0	2	0.37	1	0.33
Nitrogen dioxide, ppm	0	5	0	0.37	0	0.16
Sulfur dioxide (when added), ppm	0.1-0.6	2	1	0.1	Not studied	0.04
Water	50% r.h.	100 ppm	3 torr	50% r.h.	70% r.h.	Ambient, 35% r.h.
Aerosol Measurement	[a]	[b]	[b]	[b]	[c]	[c]

[a] 90° light scattering relative to air.
[b] Sinclair Phoenix forward-scattering smoke photometer (logarithmic; 1 unit reading increase = mass × 10 increase).
[c] b_{scat}, integrating nephelometer (linear, reading proportional to mass).

Application of Smog-Chamber Data to the Atmosphere

Many difficulties arise in the application of laboratory data to the more complex atmospheric processes. This is illustrated by the following examples:

- Depending on meteorologic conditions, aerosol formation in the atmosphere is better approximated, but never fully simulated, by smog-chamber studies under either static (batch-reactor) or dynamic (flow-reactor) conditions.
- Small differences in light sources have definite effects on photochemical processes. Some irradiation systems match the solar spectrum poorly, thus affecting the relative importance of the various photodissociation processes occurring in the atmosphere.[107] Ambient variations in the nitrogen dioxide photolysis pseudo-first-order rate constant, K_d, are difficult to reproduce with artificial irradiation systems. "Transparent" smog-chamber materials, such as fluoroolefinic polymers, fail to transmit the entire solar radiation: Mylar absorbs strongly in the <3300 Å region,[1] and Teflon shows a pronounced decrease in transmission, owing to aging.[54] Different smog profiles were obtained when identical experiments were conducted on aluminum, stainless-steel, Pyrex, and Teflon smog chambers.[107] Furthermore, a so-called inert material, such as Teflon, reacts with ozone.[42,124]
- Mechanical stirring dramatically inhibits aerosol formation, and data obtained in smog chambers equipped with stirring accessories are questionable.
- Significant aerosol losses on the walls are observed in smog chambers with high surface:volume ($S:V$) ratios. Typical smog chambers have $S:V$ ratios of about 3:1 to 5:1 m^{-1}. Although wall losses are minimized in bigger smog chambers, with $S:V$ ratios of 0.1:1 to 1:1 m^{-1}, this is still much higher than typical ambient $S:V$ values. For example, a typical ambient concentration of 150 $\mu g/m^3$ corresponds to an $S:V$ ratio of about 9×10^{-4}:1 m^{-1} (assuming a density of 1 and a diameter of 1 μm). Formation of water layers on the smog-chamber walls would promote heterogeneous reactions and thus affect the chemical composition of the aerosol formed. Nitric oxide and, to a lesser extent, nitrogen dioxide are poorly soluble in water, whereas sulfur dioxide and nitrogen pentoxide are readily soluble and their liquid-phase oxidation to form sulfate and nitric acid, respectively, is quite different.
- Errors may be due to the instrumentation itself. Improper calibration of condensation nuclei counters may lead to poor nuclei concen-

tration data.[132] In many experiments, the aerosol mass concentration was deduced from the light-scattering measurements by using Charlson's relation,[31] which was established from data on ambient, well-aged aerosols. Use of this relation is questionable for automobile exhaust data,[141,239] and it does *not* apply to fresh aerosols generated in smog chambers.[157] Interference due to ozone and to nitrogen-containing compounds has been reported when nitrogen oxide concentrations have been measured by the colorimetric[13] and chemiluminescence[241] techniques. Significant differences have been observed in the measurement of ozone by different techniques, and a standard procedure has been recently recommended.[26]

- Use of clean air, although important for obtaining quantitative *gas-phase* data, would favor homogeneous nucleation to the detriment of the heterogeneous process, which is preponderant in the polluted atmosphere, thus providing aerosol kinetic and growth data of questionable significance. Moreover, the background composition of individual hydrocarbons in clean air is generally not specified, and only an upper limit of "total hydrocarbon" concentration is provided. Therefore, a significant fraction of the nuclei formed may well originate from a few C_{6+} alkenes or cyclic olefins present as traces in the hydrocarbon background.
- Because of the abundance of smog-chamber data on homogeneous gas-phase photochemical reactions, the possible importance of heterogeneous reactions in the formation of organic aerosols has been somewhat overlooked. However, reactions in the liquid phase and reactions catalyzed by airborne particles containing metals, oxides, and salts—similar to the reactions occurring in the well-known heterogeneous oxidation of sulfur dioxide—may be of some importance. As demonstrated by Judeikis and Siegel,[117] heterogeneous reactions can compete with and, under favorable conditions, outweigh the homogeneous gas-phase reactions that are usually considered to be important. Free radicals (hydroxyl, hydroperoxy, and so on) may be efficiently trapped by aerosol particles and react further in the particle-water layer[73] or at the particle surface.[149] These reactions have been incorporated in a recent mathematical simulation study.[73]

We have seen that aerosol data obtained in environmental chambers may be seriously affected by many empirical characteristics, such as smog-chamber design and materials, stirring, mode of irradiation, and wall losses. Moreover, only homogeneous systems have been studied, and the importance of heterogeneous organic reactions is not assessed. Therefore, *caution must be exercised when extrapolating smog-chamber data to atmospheric processes.*

CHEMICAL COMPOSITION OF MODEL AEROSOLS

As mentioned earlier, there is little information on the chemical composition of "model" organic aerosols generated from a single hydrocarbon in a smog chamber. Prager *et al.*[169] and Endow *et al.*[51] reported the presence of absorption bands due to carbonyl, carboxylic acid, and nitrate ester groups in the infrared spectra of aerosols produced from various olefins. In their study of the gas-phase olefin–ozone reaction at high concentrations, Vrbaski and Cvetanović[226] reported the formation of aerosols that initially had a peroxidic character and whose later decomposition produced carbonyl compounds identified by gas chromatography. Kopczynski[124] reported absorption bands due to carbonyl, hydroxyl, nitrate ester, and nitro groups in the infrared spectra of aerosol generated from mesitylene at 25 ppm. Barlage and Alley[12] studied, by mass spectrometry, aerosols formed from 1-pentene and 1-hexene at 10 ppm. They found peaks corresponding to masses in the range 120-160 AMU, indicating the presence of polymeric material. Groblicki and Nebel[76] and Ripperton *et al.*[180] reported similar infrared spectra for α-pinene aerosol generated by either dark reaction with ozone or irradiation with nitrogen oxides. Infrared spectra of α-pinene, 1-hexene, and dodecene aerosols were recently reported by Lipeles.[131] Chu and Orr[32] studied aerosols formed from benzene, toluene, and *o*-xylene and found similarities between *o*-xylene aerosol and diethylphthalate (infrared spectra) and between benzene aerosol and such aromatic ketones as fluorenone (mass spectra). O'Brien *et al.*[157] reported carbonyl, hydroxyl, carboxyl, and nitrate ester absorption bands in the infrared spectra of 1-octene and 1,7-octadiene aerosols and found that infrared spectra of indene aerosol resembles that of homophthalic acid, an expected product of indene photooxidation. Paper chromatography indicated the presence of several carboxylic acids in the 1-octene aerosol and the presence of acidic difunctional compounds (α,ω diacids, α,ω acid nitrate, and other ω-substituted acids) in the 1,7-octadiene aerosol. Some ammonium nitrate was found, indicating that nitric acid was formed photochemically. The molecular weight of 1,7-octadiene aerosol was determined, and its value (256) indicates that the difunctional compounds are present as dimers, or possibly higher polymers, in the aerosol.

This survey that shows highly oxygenated compounds (carbonyls, acids, nitrate esters) constitute the bulk of model organic aerosols and that smog-chamber data on chemical composition are in qualitative agreement with atmospheric observations. However, positive identification of individual organic compounds present in model aerosols cannot be achieved from infrared, carbon-hydrogen-nitrogen, and paper-chroma-

tography analysis or from limited mass-spectrometry data. Only since 1973 have firm product identifications been reported. Lipeles *et al.*[130] and Schulten and Schurath[197] studied the composition of aerosol from the reaction of ozone with 1-butene. Aerosol composition from irradiation of nitrogen oxides with cyclohexene, toluene, and α-pinene was reported by Schwartz,[198] and with cyclopentene, cyclohexene, and 1,7-octadiene, by Grosjean (unpublished data). Mass spectrometry was used in these four studies.

Ozone-1-Butene Aerosol

The Rockwell study [130] was conducted in a flow reactor with a dry nitrogen stream and typical residence times of 20–60 s. Freshly collected aerosol showed a marked peroxidic character, but would no longer oxidize iodide after several days. Polarographic analysis confirmed the presence of peroxides. Aerosol composition was obtained by a combination of gas chromatography and electron-impact mass spectrometry. Schulten and Schurath[197] also used a flow-reactor system coupled with a high-resolution-field desorption mass spectrometer. Hydrogen peroxide was positively identified. Other peroxides may have been present, but would decompose at the low ($\leq 10^{-5}$ torr) pressure in the ion source of the mass spectrometer. The complete data are listed in Table 3-8. Aldehydes, ketenes, and C_{1-4} monocarboxylic acids were identified. Schulten and Schurath observed many unidentified complex ions at the higher-mass region and postulated the presence of aggregates of aldehydes and acids held together by strong hydrogen bonds. This assumption is consistent with the release of carbonyl compounds observed when aerosols generated from alkenes decompose during gas-chromatography analysis.[226]

NO_x–Toluene Aerosol

This system was studied by Schwartz.[198] Toluene at 10 ppm, nitric oxide at 1 ppm, and nitrogen dioxide at 1.2 ppm were irradiated with ultraviolet lamps in a 17-m^3 batch reactor for 270 min. Collected aerosols were successively extracted with methylene chloride and then methanol. The methylene chloride extract was fractionated into water-soluble and water-insoluble material, and the latter fraction was further divided into acidic, neutral, and basic fractions. The acidic and neutral fractions were analyzed by gas chromatography and chemical-ionization mass spectrometry; the compounds identified are shown in Figure 3-7. The two analyzed fractions represented only about 5.5% of the total aerosol mass. It is noteworthy that "classical" nitration of an aromatic ring appears to

TABLE 3-8 Composition of Aerosol from the Reaction of Ozone and 1-Butene

Identified by Lipeles *et al.*[130]	Identified by Schulten and Schurath [197]
Carbon dioxide	Formaldehyde
Ethane	Hydrogen peroxide
Water	Ketene
Formaldehyde	Acetaldehyde
Methanol	Formic acid
Acetaldehyde	Methylketene
Formic acid	Propionaldehyde
Propionaldehyde	Acetic acid or methylformate
Acetic acid	Ethylketene
Propionic acid	Alcohol (tentative)
Other unidentified trace compounds	Butyraldehyde or ether, C_4H_8O
	Propionic acid
	Formic acid-formaldehyde cluster, $C_2H_4O_3$
	Butanoic acid
	Butanoic acid-water
	$C_2H_4O_4$: dimer of formic acid or dimer zwitterion
	$C_4H_8O_3$: ozonide
	+ Unidentified peaks from m/e = 105 to m/e = 199 ($C_xH_yO_z$ with x = 3-8, y = 5-13, z = 2-6), presumably from condensates or aggregates of aldehydes and acids held together by strong hydrogen bonds.

be an important process during aerosol formation and that both the alkyl group and the aromatic ring undergo attack by oxidizing species, thus leading to polyfunctional compounds of very low volatility.

Aerosols from Cyclic Olefins and Diolefins

Data on cyclohexene and α-pinene aerosols were reported by Schwartz[198] after a preliminary report from the Battelle Institute group.[240] The experimental conditions and analytic techniques were identical with those just described for the toluene aerosol study. Here again, only the methylene chloride-soluble, water-insoluble fractions were studied. They accounted for about 7% and 65% of the total aerosol mass generated from cyclohexene and α-pinene, respectively. Grosjean (unpublished data) has investigated the chemical composition of cyclopentene, cyclohexene, and 1,7-octadiene aerosols. Experiments were conducted in an 80-m^3 Teflon smog chamber filled with ambient air, with irradiation by

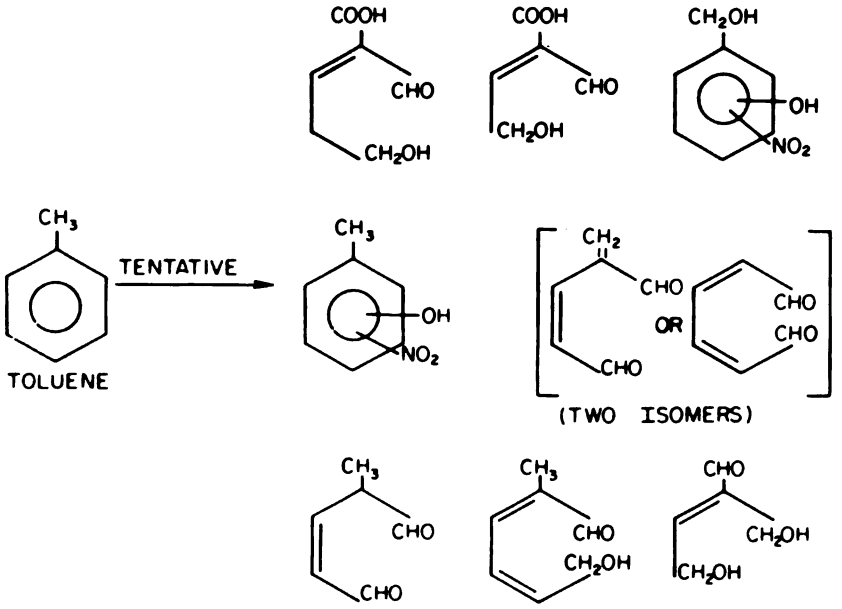


FIGURE 3-7 Composition of aerosol from the reaction of NO_x and toluene. Reprinted with permission from Schwartz.[198]

sunlight. Typical initial concentrations were 1 ppm for hydrocarbon, 0.33 ppm for nitric oxide, and 0.16 ppm for nitrogen dioxide. Aerosols were extracted after collection and analyzed without further fractionation by combined gas chromatography and electron-impact mass spectrometry and by combined gas chromatography and chemical-ionization mass spectrometry. Data obtained by Schwartz and Grosjean are listed in Figure 3-8 and Table 3-9. Most of the products are difunctional compounds bearing in many cases a carboxylic acid group. The ethylenic bond is retained in some compounds, indicating free-radical attack on the aliphatic chain. Good agreement is observed in the case of cyclohexene (the only compound common to the two studies), except for adipic acid, which was not reported by Schwartz, but was the major aerosol compound in Grosjean's study. However, adipic acid would not be expected to be present in the methylene chloride-soluble, water-insoluble fraction analyzed in the former study. Data for the 1,7-octadiene aerosol are also in good agreement with those obtained by O'Brien *et al.*[157] by infrared and paper chromatography.

It is very significant that most of these polyfunctional compounds have also been identified in ambient aerosols (Tables 3-2 and 3-3) and

α-Pinene aerosol.

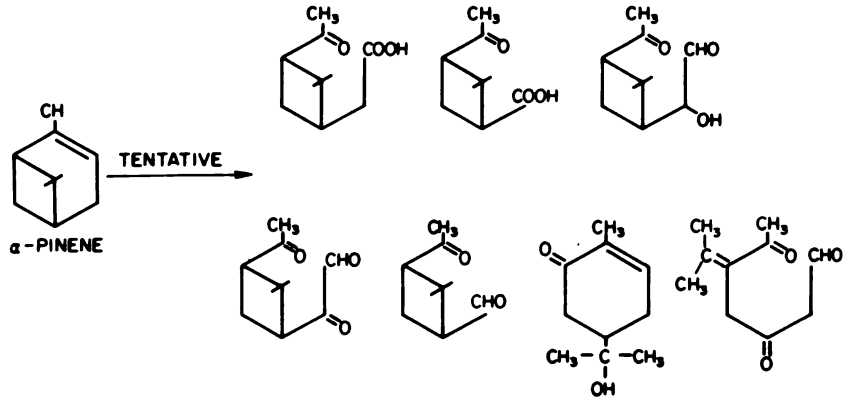


FIGURE 3-8 Composition of aerosol from reaction of NO_x and α-pinene. Reprinted with permission from Schwartz.[198]

that the gas-to-particle distribution factor, f_C, measured in smog-chamber studies for aerosol precursors, such as cyclic olefins and diolefins, exceeds by one order of magnitude those measured in ambient atmospheres.[79]

CHEMICAL MECHANISMS OF ORGANIC AEROSOL FORMATION

The Ozone–Olefin Reaction

Much evidence has been accumulated that the ozone-olefin reaction has a predominant role in aerosol formation from alkenes, cyclic olefins, diolefins, and other unsaturated compounds. Free radicals are formed in the reaction and can react further, along with nitric oxide and nitrogen dioxide, either with the various intermediates or with the olefin itself (see the recent review by Pitts and Finlayson[168]).

A mechanism has been proposed recently by O'Neal and Blumstein[158] for the gas-phase ozone-olefin reaction. This mechanism postulates that molozonide-biradical equilibrium is reached fast and postulates a competition between α-, β-, and γ-hydrogen abstraction reactions and the classical mechanism proposed by Criegee[36,37] for the liquid-phase reaction. The main features of the Criegee mechanism (Figure 3-9) are the formation, from the initial molozonide, of the major carbonyl products and a second biradical intermediate, the "zwitterion." The decomposition pathways of the zwitterion comprise unimolecular re-

TABLE 3-9 Composition of Aerosol from the Reaction of NO_x and Various Olefins

Identified by Schwartz[198]	Identified by Grosjean
From Cyclohexene Aerosol	
$COOH-(CH_2)_4-CHO$	$COOH-(CH_2)_4-COOH$ major
$COOH-(CH_2)_3-CHO$	$COOH-(CH_2)_4-CH_2ONO_2$
$COOH-(CH_2)_3-CH_2ONO_2$	$COOH-(CH_2)_4-CHO$
$COOH-(CH_2)_3-CH_2OH$	$COOH-(CH_2)_4-CH_2OH$
$CH_3-CH=CH-\underset{\underset{O}{\|}}{C}-\underset{\underset{O}{\|}}{C}-CH$	$COOH-(CH_2)_3-COOH$
	$COOH-(CH_2)_3-CH_2ONO_2$
$CH_2=CH-CH=CH-CH_2-CH_2OH$	$COOH-(CH_2)_3-CHO$
cyclopentene-2-aldehyde or	$COOH-(CH_2)_3-CH_2OH$
$CHO-CH=CH-CH_2-CH=CH_2$	$CHO-(CH_2)_3-CHO$
From Cyclopentene Aerosol (Identified by Grosjean)	
$CHO-(CH_2)_3-CHO$	$COOH-(CH_2)_2-CH_2OH$
$CHO-(CH_2)_3-COOH$	$COOH-(CH_2)_2-CHO$*
$COOH-(CH_2)_3-COOH$	$CHO-(CH_2)_2-CHO$*
$COOH-(CH_2)_3-CH_2ONO_2$	$CHO-(CH_2)_2-CH_2ONO_2$*
From 1,7-Octadiene Aerosol (Identified by Grosjean)	
$COOH-(CH_2)_4-COOH$	$CH_2=CH-(CH_2)_4-CH_2ONO_2$*
$COOH-(CH_2)_4-CH_2ONO_2$	$CH_2=CH-(CH_2)_4-CHO$
$COOH-(CH_2)_4-CHO$	$CH_2=CH-(CH_2)_4-COOH$*

* Tentative.

arrangements and bimolecular reactions, the latter including ozonide formation from zwitterion-aldehyde reaction. Other aspects of the Criegee mechanism have been discussed and reviewed elsewhere.[11, 53, 63, 72, 86, 103]

The Criegee mechanism, widely accepted for the liquid-phase reaction, does not adequately explain the available gas-phase data. O'Neal and Blumstein[158] suggested a biradical structure for the first gas-phase intermediate and proposed three types of unimolecular hydrogen abstraction reactions (Figure 3-10).

The α-hydrogen abstraction leads to an α-ketoperoxide, which has been tentatively identified by Pitts in the case of *cis*-2-butene.[7, 52] Further reactions of the ketoperoxide include the formation of the "normal products," i.e., the carbonyl products that can also be explained by the Criegee mechanism. The β-hydrogen abstraction accounts for the ob-

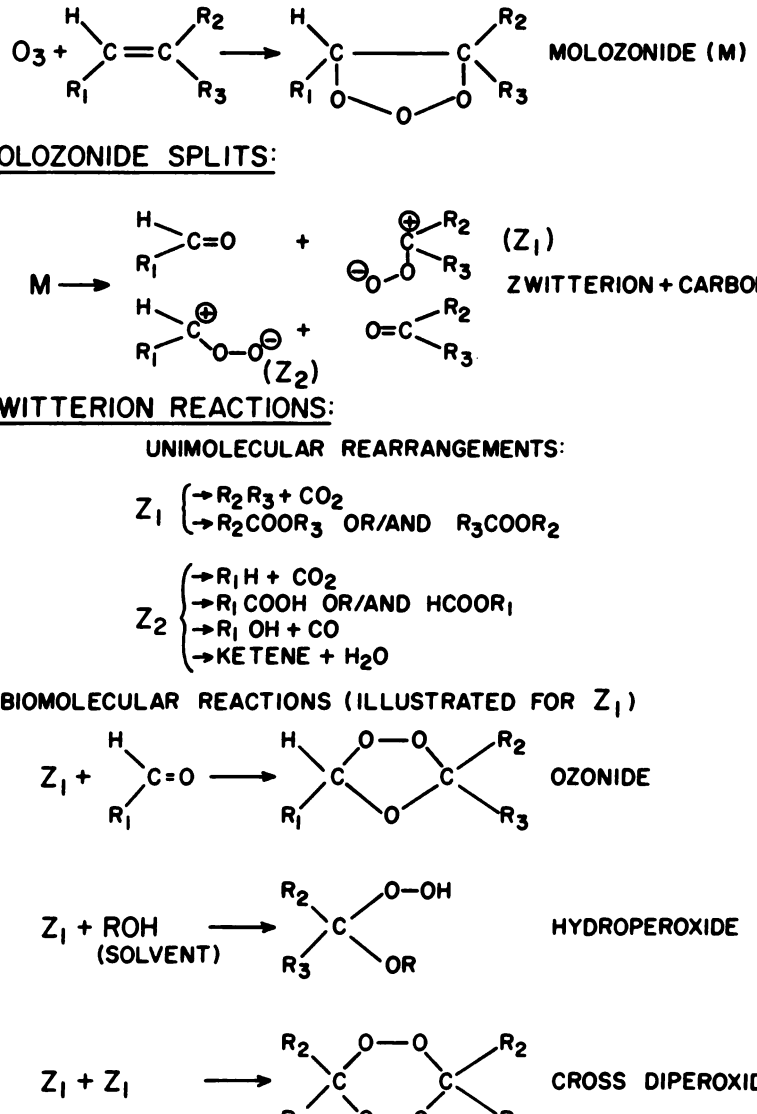
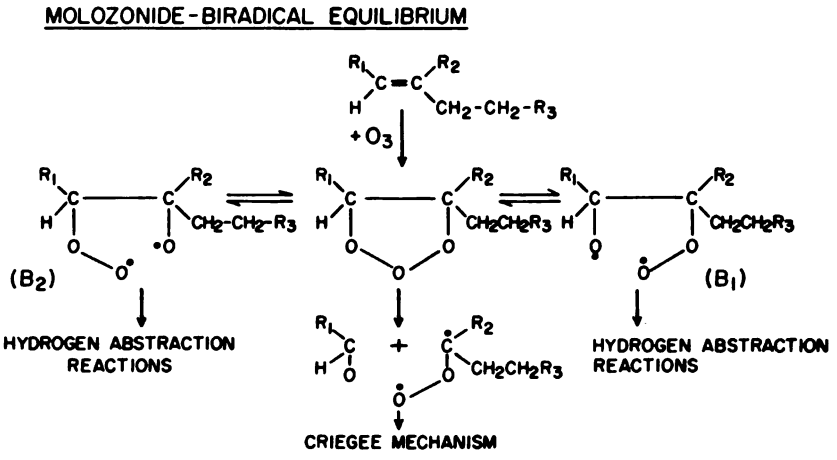


FIGURE 3-9 Liquid-phase ozone-olefin reaction: the Criegee mechanism.[36,37]



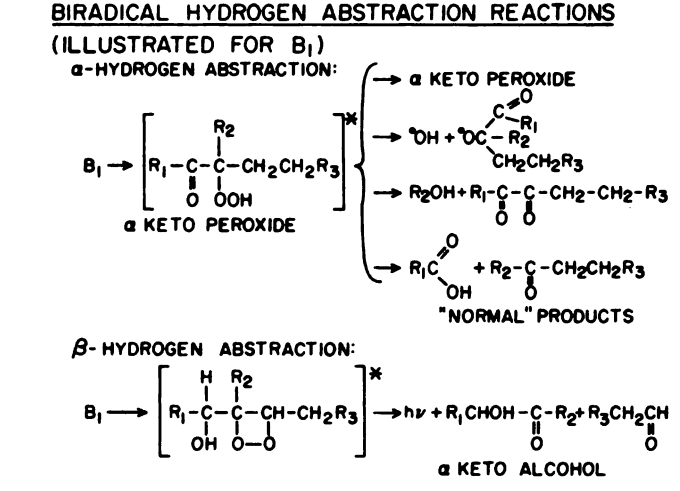


FIGURE 3-10 Gas-phase ozone-olefin reaction: the O'Neal and Blumstein mechanism.[158]

served α-cleavage products. The γ-hydrogen abstraction (possible for C_{4+} alkenes) should lead to a rather stable five-membered ring, dioxetane, whose existence has so far not been demonstrated experimentally. On the basis of calculations of the relative importance of the Criegee and abstraction pathways for both spontaneous and thermal reactions of the intermediate species, O'Neal and Blumstein concluded that the Criegee and abstraction mechanisms are of equal importance for butenes, that the former is predominant for ethylene and propylene, and that the latter is predominant for C_{5+} alkenes.

All aerosol products identified in the smog chamber can be reasonably explained in terms of the O'Neal and Blumstein and Criegee mechanisms, as is illustrated in Figure 3-11 for cyclohexene. The major difference between alkenes and cyclic olefins lies in the fact that, after opening of the cyclic olefin double bond, the original number of carbon atoms is conserved and the chain carries both the carbonyl group and the biradical intermediate, whose further reactions lead to the observed difunctional compounds.

The Hydroxyl Radical–Aromatic Hydrocarbon Reaction

Very little is known about the mechanisms governing ambient reactions of aromatic hydrocarbons. The Battelle study[198] demonstrated that aromatic-ring opening, aromatic-ring nitration, and alkyl-group oxidation may occur at atmospheric concentrations. The former possibility was supported by Altshuller *et al.*,[2] who observed a decrease of infrared absorption bands due to the aromatic ring during the photooxidation of various aromatics at 5 ppm with nitric oxide at 3 ppm. Reactions of aromatic hydrocarbons with ozone, atomic oxygen, and hydroxyl radical might account for the observed polyfunctional aerosol products. The reaction of ozone with aromatics has been studied in the liquid phase by Wibaut and co-workers (cited in Bailey[11]). They reported relative rate constants of 1.9, 10, 40, 250, and 15,000 for benzene, toluene, xylenes, mesitylene, and hexamethylbenzene, respectively. Glyoxal, methylglyoxal, and biacetyl were the major products of the ozone-*o*-xylene reactions, and their formation was interpreted in terms of the Criegee mechanism. The gas-phase reaction has received little attention, and the rate constants of only three aromatics have been measured. It appears that the reaction is very slow, compared with that of olefinic compounds. Bufalini and Altshuller[21] estimated the rate constant k_{O_3} for mesitylene, one of the most reactive aromatics, to be only about 0.37 the rate constant for ozone reactions with the least reactive olefin, ethylene. Recent data from Stedman and Niki[208] show that toluene and xylenes react even more slowly with ozone than acetylene does.

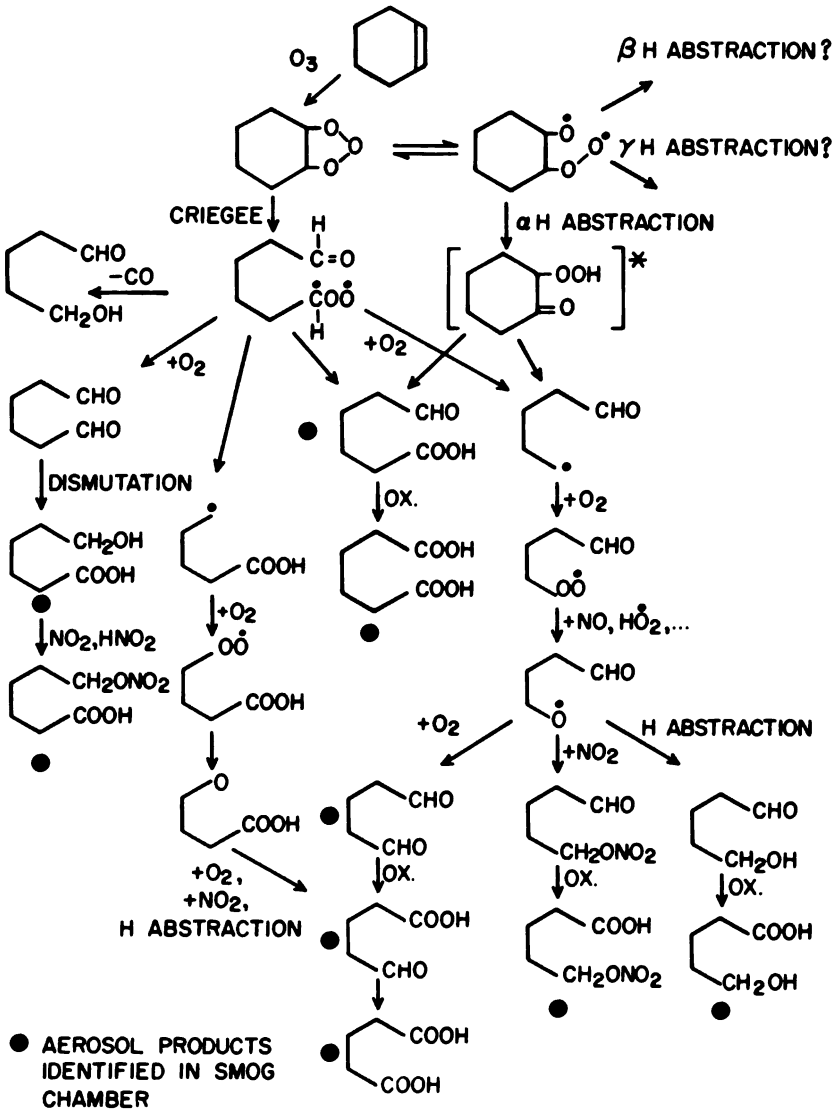


FIGURE 3-11 Possible formation pathways for cyclohexene aerosol products. After Grosjean and Friedlander.[80]

The reaction of atomic oxygen, $O(^3P)$, has been investigated in greater detail.[32,168] Cvetanović and co-workers[17,114] reported formation of nonvolatile polymeric material from benzene and toluene. Formation of the observed linear products can be explained by cleavage of the biradical initially formed by addition of atomic oxygen on the aromatic ring. Absolute rate constants for atomic oxygen addition to a series of aromatics were recently measured in the temperature range 299–392 K.[8]

The aromatic-hydroxyl radical reaction has been studied by Davis *et al.*[43] They reported rate constants for benzene and toluene and concluded that hydroxyl additions to the aromatic ring compete favorably with the abstraction of hydrogen atom from the alkyl substituent. Doyle *et al.*[48] recently published hydroxyl reaction rate constants for a series of alkylbenzenes.

Possible ozone, atomic oxygen, and hydroxyl radical reaction mechanisms, leading to linear polyfunctional products from aromatic hydrocarbons, are shown in Figure 3-12. In an attempt to assess the relative importance of the various oxidizing species in the photooxidation of olefinic and aromatic hydrocarbons, we have compared the rate constants for hydroxyl radical, atomic oxygen, and ozone reactions with the rate constants for the conversion of nitric oxide to nitrogen dioxide (Figure 3-13). Good relations are obtained between k_{O_3} and $k_{NO \to NO_2}$ for alkenes and between k_{OH} and $k_{NO \to NO_2}$ for aromatics, indicating that the structural effects prevailing in the overall conversion of nitric oxide to nitrogen dioxide are those controlling the *ozone-olefin* and *hydroxyl radical-aromatic* reactions. This is further substantiated by comparing the absolute reactivities of olefins and aromatics. The most reactive aromatics, *m*-xylene and mesitylene, react faster than ethylene and as fast as propylene and isobutene with the hydroxyl radical. The same reactivity sequence is observed for the rates of conversion of nitric oxide to nitrogen dioxide, whereas ozone-aromatic reaction rate constants are too low to account for their overall photochemical reactivity. Relevant to the postulated importance of the hydroxyl radical-aromatic reaction is the observation made by Miller *et al.*:[142] an aerosol increase from 1-heptene and an aerosol decrease from toluene when ozone is added to irradiated NO_x-hydrocarbon mixtures. In the latter case, the observed inhibiting effect results from the reaction

$$O_3 + \dot{O}H \rightarrow H\dot{O}_2 + O_2, \qquad (3\text{-}1)$$

which competes with the hydroxyl radical-aromatic reaction. Many investigators also reported that aerosol formation from olefins coincides with ozone appearance, whereas aerosol buildup from aromatics starts

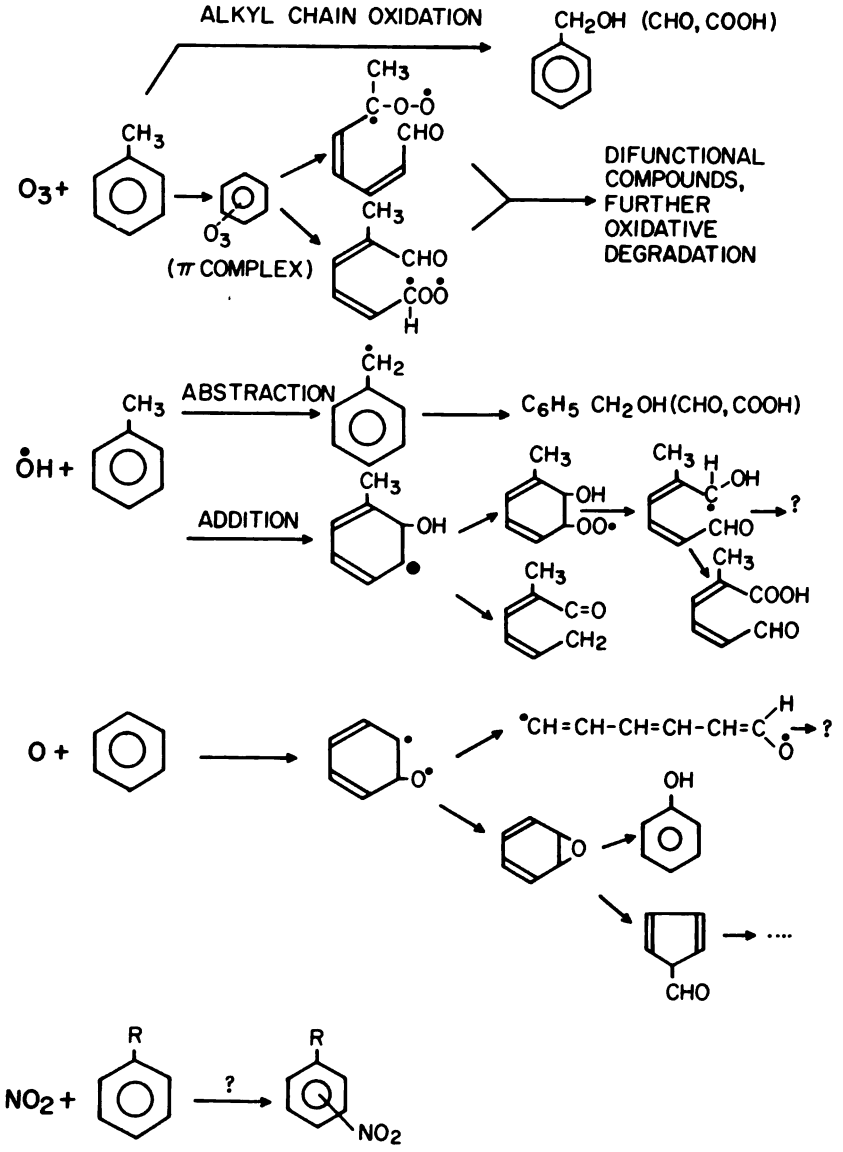
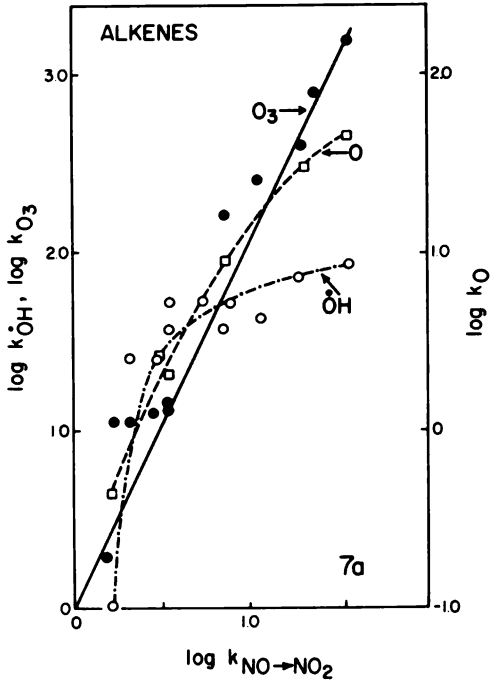
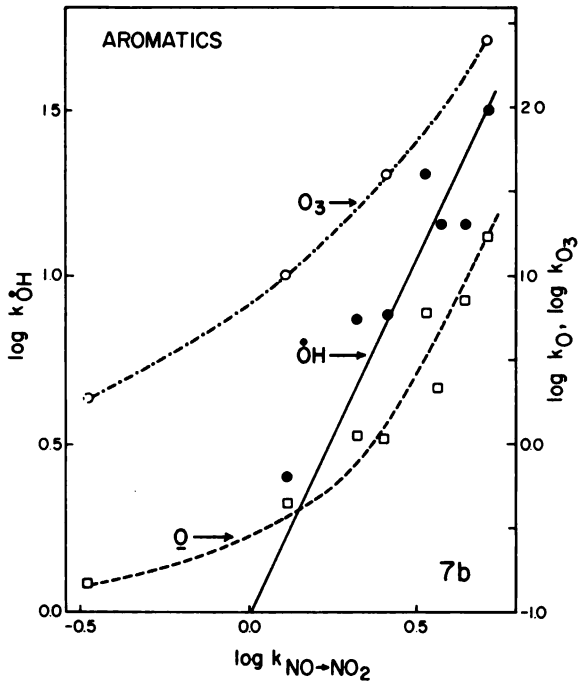


FIGURE 3-12 Possible initial steps for ozone, atomic oxygen, nitrogen dioxide, and hydroxyl-radical reaction with aromatic hydrocarbons.



A: Alkenes, $k_{NO \rightarrow NO_2}$ from Glasson and Tuesday;[64] k_O, from Japar *et al.*; [110] $k_{\dot{O}H}$ from Morris and Niki;[148] k_O from Furayama *et al.*[60]



B: Aromatics, k_O from Atkinson and Pitts;[8] $k_{\dot{O}H}$ from Doyle *et al.*;[48] k_O, liquid phase relative to toluene from Bailey[11] and Gould.[72] Gas-phase ozone rate constants have not been measured.

FIGURE 3-13 Relations between conversion of nitric oxide to nitrogen dioxide and ozone, atomic oxygen, and hydroxyl-radical reaction rate constants. Reprinted with permission from Grosjean.[77]

before ozone formation and is presumably associated with the appreciable hydroxyl radical concentrations in the early stages of the photochemical process.

From all available evidence, the hydroxyl radical plays a major role in the photooxidation and aerosol formation processes for aromatic hydrocarbons. However, much research remains to be done to improve our knowledge in this field.

PHYSICAL MECHANISMS OF ORGANIC AEROSOL FORMATION

Dynamics of Gas-to-Particle Conversion

The physical processes involved in the formation of atmospheric aerosol have been thoroughly investigated in the last few years[25,58,92,94–100,104,119,121,146,234] and are briefly summarized here, with some emphasis on recent data obtained in smog-chamber studies.

Regardless of the chemistry, there are some physical constraints on aerosol-gas interactions. Particles must be close to or at equilibrium with respect to the surrounding vapor to exist in air for any substantial period. Thus, the partial pressure of condensed species on particles must be less than or equal to the saturation vapor pressure at atmospheric temperature for stability. As shown later in this chapter, the requirement of low vapor pressure is particularly important to the stability of organic aerosols.

Accumulation of condensed material as aerosols in the atmosphere may take place by two basic processes: by condensation of supersaturated vapor or chemical reaction that leads to spontaneous formation of new particles, and by condensation, absorption, or reaction on existing particles. In the latter case, the chemical reactions may actually take place on the surface of or within existing particles.

For condensable precursors, particle formation may occur by homogeneous or heterogeneous nucleation. It is generally accepted that heterogeneous processes are most likely in the atmosphere, because of the large number of nuclei present.

Growth of particles by accumulation on existing particles can be classed as two broad processes. If the precursor is supersaturated, growth will occur at a rate limited by vapor diffusion, which depends on the supersaturation, the temperature, the particle size, and the accommodation coefficient at the surface. The proportionality of particle size changes with the ratio of particle diameter to mean free path of the suspending

gas. At one extreme, the growth depends on volume to the 2/3 power; at the other, growth is proportional to volume to the 1/3 power. When the precursor is unsaturated, growth still may take place by irreversible absorption or by chemical reactions in the particle. In this case, the rate law should be proportional to the particle volume, if the reaction is uniform throughout the particle. If the formation of material is limited by reactions in the particle, the conversion ratio should not depend on the concentration of the gaseous precursor.

Sufficient information is not available to determine the rate law or physical mechanism most likely to predominate in atmospheric aerosol growth. However, there are clues to differences in the processes from the Los Angeles data. The shape of the particle volume number distribution of tropospheric aerosol is such that the 1/3 (diameter) and 2/3 (surface) moments are concentrated in the submicrometer fraction, whereas the first moment (volume) is weighted toward larger particles. Thus, the observed accumulation of organic carbon on the small particles in smog (Figure 3-14) suggests a process controlled by surface or vapor diffusion.

It is of interest that the influence of thermodynamic equilibrium must enter the growth process of particles. If the radius of the particles is too

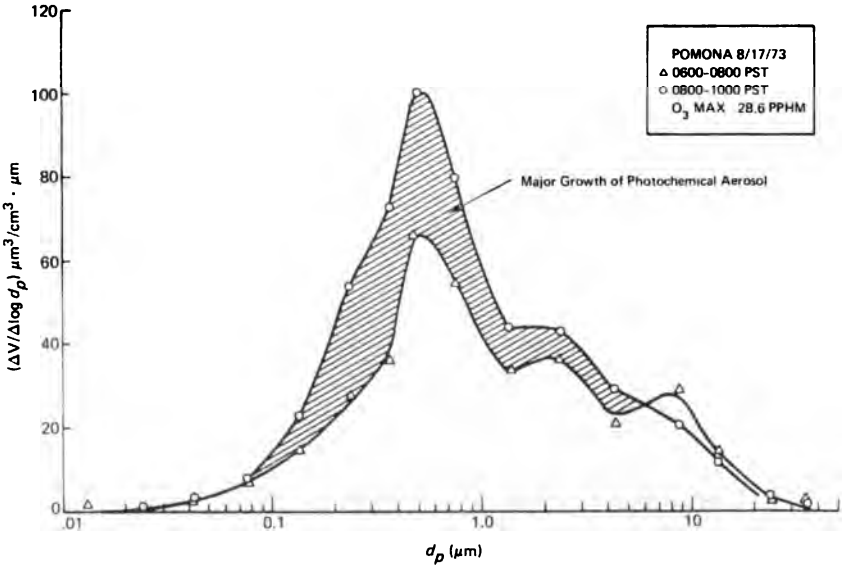


FIGURE 3-14 Evolution of the volume distribution of ambient smog aerosol. Compare with Figures 3-24 and 3-26. Reprinted with permission from Hidy.[94]

small, the partial pressure of the condensable species can increase significantly by the influence of radius of curvature (Kelvin effect). Examination of values of surface tension for a range of materials suggests that the Kelvin effect will constrain growth to particles greater than about 0.05–0.1 μm in diameter. This appears to be consistent with *available* observations of atmospheric growth and the distribution of secondary chemical components.

Growth of Secondary Organic Particles

The physical mechanisms governing the formation of organic aerosols in smog-chamber experiments have recently been studied in some detail.[90,91,157] Two types of profiles, presented in Figure 3-15, are generally observed. Aromatic hydrocarbons and alkenes with fewer than seven carbon atoms, when present at initial concentrations of about 1 ppm, produce copious quantities of nuclei that are not able to grow in the light-scattering range. On the contrary, aerosols formed from C_{7+} alkenes, cyclic olefins, diolefins, and terpenes always grow in the light-scattering range and produce appreciable visibility reduction (Figure 3-16; compare

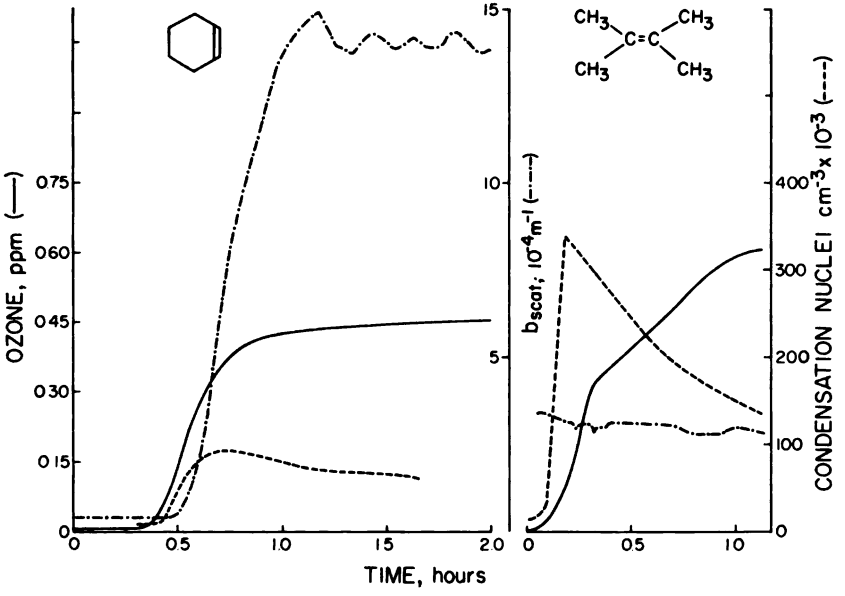


FIGURE 3-15 Smog-chamber profiles. *Left*, cyclohexene. *Right*, tetramethylethylene. Initial concentrations: hydrocarbon, 1 ppm; NO, 0.33 ppm; NO_2, 0.16 ppm.

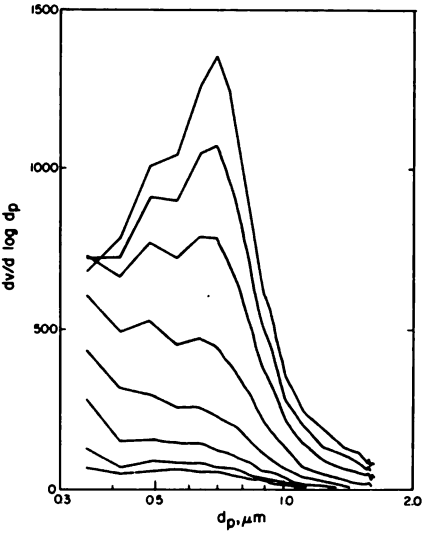


FIGURE 3-16 Evolution of the volume distribution of secondary organic aerosol generated in smog chamber with 1-ppm cyclohexene, 0.33-ppm NO, and 0.17-ppm NO_2. Time from bottom to top: 0, 203, 412, 631, 863, 1,109, 1,364, and 1,626 s. Compare with Figures 3-22 and 3-26. Reprinted with permission from Heisler.[90]

with ambient data presented in Figure 3-14). As a result of aerosol growth, the light-scattering efficiency per aerosol mass unit increases rapidly in the early stages of the experiment (Figure 3-17; compare with the light-scattering efficiency as a function of particle size for monodisperse aerosol presented in Figure 3-18).

In the case of organic constituent formation, growth of particles is governed by physical laws of condensation, provided that the precursors are formed in the gas phase. For a diffusion-limited condensation process, the rate of volume change in particles is

$$\frac{dv}{dt} = A(d_p - d_p^*)\left(1 + l\frac{2\lambda}{d_p}\right)^{-1}, \qquad (3\text{-}2)$$

where $A = \sum_i A_i$, in which $A_i = \lambda_i x_i S_i \ln S_i$; for the ith condensable species, λ_i is the particulate mole fraction, x_i is the activity coefficient, and S_i is the supersaturation ratio. In Equation 3-2, d_p is the particle diameter, d_p^* is the initial particle size below which condensation cannot take place because of the curvature effect on equilibrium vapor pressure (Kelvin effect), λ is the mean free path in air, and l is a parameter proportional to the ratio d_p/λ. This linear relation has been verified experimentally in the case of cyclic olefin and diolefin aerosols (Figure 3-19). Critical sizes of 0.13–0.24 μm and 0.26–0.28 μm have been measured for cyclohexene and 1,7-octadiene, respectively.[90,91] For such aerosol precursors, the gas-to-particle conversion process consists of the

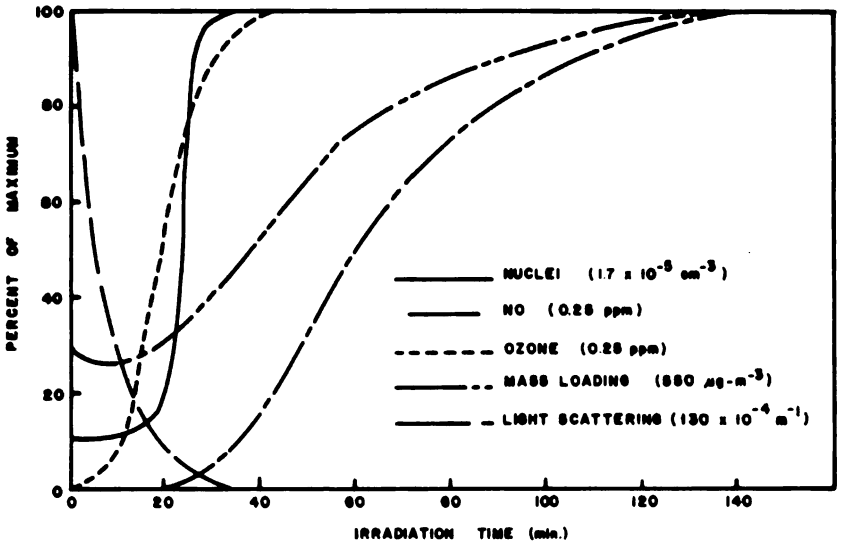


FIGURE 3-17 Increase in aerosol light-scattering efficiency as a result of aerosol growth. Compare with Figures 3-24 and 3-26. Reprinted with permission from O'Brien *et al.*[157]

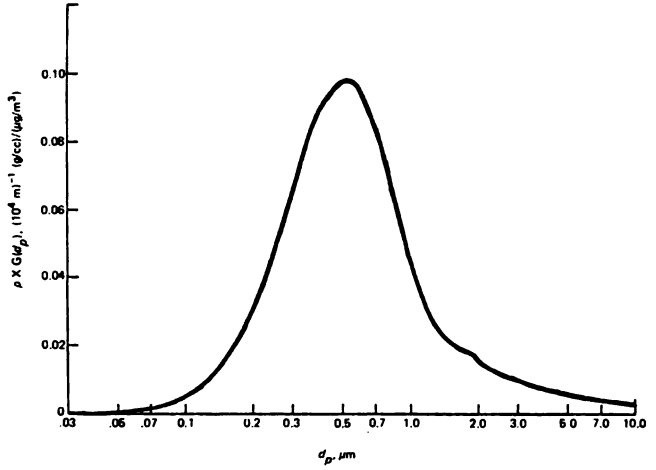


FIGURE 3-18 Ratio of light-scattering coefficient to mass concentration for uniform spherical particles of unit density. Refractive index, 1.5; diameter, d_p. Reprinted with permission from Hidy.[97]

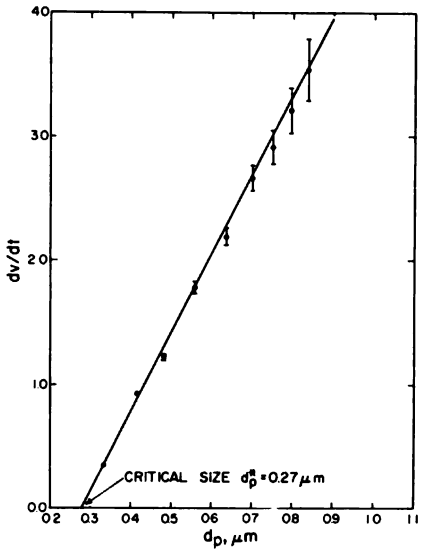


FIGURE 3-19 Growth of organic aerosol generated in smog chamber; same experiment as in Figure 3-24. Reprinted with permission from Heisler.[90]

formation of supersaturated compounds in the gas phase followed by condensation on preexisting particles.

Condensable-Species Vapor Pressure and Aerosol Formation

As discussed earlier, the equilibrium constraint of low vapor pressure is particularly important for stability of the organic aerosols. Organic aerosol formation requires accumulation of condensable species in excess to their gas-phase saturation concentrations. In turn, examination of vapor-pressure data[9,45,102,116,170,183,228] for various oxygenated compounds permits estimating the minimal hydrocarbon precursor concentration required to achieve aerosol formation in ambient atmosphere. Table 3-10 gives examples of vapor pressures of various oxygenated compounds formed in hydrocarbon photooxidation reactions. It can be seen from the table and from boiling-point data that volatility decreases regularly when the number of carbon atoms increases and that, for a given chain length, carboxylic acids have the lowest vapor pressure. Moreover, the volatility of difunctional oxygenates is several orders of magnitude lower than that of the corresponding monofunctional compound. Grosjean and Friedlander[80] estimated the minimal ambient alkene concentration required to form the corresponding carboxylic acid in excess to its saturation concentration (Table 3-11). Because a 100% gas-to-aerosol conversion was assumed and because carboxylic acids

TABLE 3-10 Vapor Pressures of Oxygenated Compounds[a]

	Vapor Pressure, mm Hg at 25° C[b]				
Carbon Number	Alkanes[c]	Aldehydes	Alcohols[c]	Carboxylic Acids[c]	Dicarboxylic Acids
1	—	—	100 (21.2° C)	40 (24° C)	—
2	—	795	31.5	16	2.2×10^{-4}
3	—	200	16	4	—
4	—	—	5	1	2.97×10^{-7}
5	—	—	1.8	0.25	—
6	—	—	1 (24.4° C)	0.02	6.14×10^{-8}
7	—	6	0.26	—	—
8	15.1 (27° C)	—	0.11	4.4×10^{-3}	—
9	—	—	7.9×10^{-2}	—	—
10	2.28 (33.6° C)	1.4×10^{-2}	4.3×10^{-2}	10^{-3} (33.6° C)	5.49×10^{-10}
11	0.48	7.2×10^{-2}	3.8×10^{-2}	—	—
12	0.117	—	10^{-2}	10^{-5} (25.3° C)	7.28×10^{-10}
13	—	—	—	8.0×10^{-5}	—
14	—	—	—	$\leqq 10^{-6}$	—
15	$\leqq 2 \times 10^{-3}$	—	—	—	—
16	10^{-3} (21.8° C)	—	—	$\leqq 10^{-8}$	—
17	5×10^{-4} (25.7° C)	—	—	—	—
18	10^{-4} (24.8° C)	—	1.45×10^{-4}	$\leqq 10^{-9}$	—

Volatility sequence for C_n *compounds* (from boiling and melting points); alkane > aldehyde > alcohol $\leqq$ nitrate ester > carboxylic acid $\leqq$ dialdehyde > diol $\leqq$ dinitrate $\leqq$ alcohol $C_{n+6} \leqq$ acid aldehyde >> dicarboxylic acid.

[a] Data from Grosjean and Friedlander.[80]

[b] Unless otherwise indicated in parentheses.

[c] Log *Vp* varies linearly with the number of carbon atoms.

actually represent only a fraction of the products, the precursor concentration data in Table 3-11 are, in fact, lowest estimates. Vapor-pressure considerations suggest, again, cyclic olefins and diolefins as the most efficient aerosol precursors. For example, cyclohexene at only about 0.1 ppb is required to form adipic acid ($Vp \leqq 6 \times 10^{-8}$ mm Hg) in excess to its saturation concentration, whereas 1-heptene at about 50 ppm (5×10^5 more than cyclohexene) would be necessary to form hexanoic acid ($Vp \leqq 2 \times 10^{-2}$ mm Hg) aerosol. Obviously, aerosol formation from C_{2-5} alkenes would require unrealistic ambient precursor concentrations, even if polymerization of the condensable species lowers the required precursor concentration by several orders of magnitude. Aggregation or polymerization (at least dimerization) of the condensable species formed from C_{6+} alkenes must take place to achieve aerosol

TABLE 3-11 Hydrocarbon Threshold Concentration[a]

Olefinic Precursor	Condensable Species	Condensable Species Vapor Pressure, mm Hg	Minimal Precursor Concentration[b]
Ethylene	Formic acid	40	52,400 ppm
Propylene	Acetic acid	16	20,960 ppm
1-Butene	Propionic acid	4	5,240 ppm
1-Pentene	Butanoic acid	1	1,310 ppm
1-Hexene	Pentanoic acid	0.25	327 ppm
1-Heptene	Hexanoic acid	0.02	26.2 ppm
1-Octene	Heptanoic acid	$\leqq 9 \times 10^{-3}$	11.8 ppm
1-Nonene	Octanoic acid	$\leqq 4 \times 10^{-3}$	5.2 ppm
1-Decene	Nonanoic acid	$\leqq 6 \times 10^{-4}$	0.78 ppm
1-Tridecene	Dodecanoic acid	10^{-5}	13 ppb
Cyclopentene	Glutaric acid	2×10^{-7}	$\leqq 0.26$ ppb
Cyclohexene	Adipic acid	6×10^{-8}	0.08 ppb
Methylcyclohexene	Methyladipic acid	$\leqq 2 \times 10^{-8}$	$\leqq 0.03$ ppb

[a] Estimated lowest ambient olefin concentration required to form the corresponding condensable species in excess to its saturation concentration. Data from Grosjean and Friedlander.[80]
[b] Assuming that there is 100% gas-to-particle conversion, that the formed condensable species is the one with the lowest vapor pressure (carboxylic acid), and that there is no vapor-pressure lowering by condensable species polymerization or other effect.

formation. On the contrary, no polymerization (not even dimerization, although it may occur) is necessary to form aerosols from cyclic olefins, even when present as traces (1 ppb or below) in ambient air. *Therefore, there is a threshold concentration, below which no organic aerosol is formed for each hydrocarbon precursor.* The concept of precursor threshold concentration is supported by qualitative experimental observations on nuclei formation[131] (Figure 3-20) and occurrence of light scattering[157] (Figure 3-21) as a function of the precursor concentration. The low-volatility constraint for organic condensable species has major consequences for the ability of the various precursor classes to form organic aerosol in the atmosphere:

- Cyclic olefins and diolefins form aerosol even when present at very low concentrations, as confirmed by smog-chamber studies for cyclohexene[180] and 1,6-heptadiene.[157]
- Alkenes of C_{2-6} do not form organic aerosol in parts-per-million concentrations. Aerosol data obtained at higher concentrations, although useful for mechanistic studies, *cannot be extrapolated to atmospheric concentrations* for these low-molecular-weight alkenes.

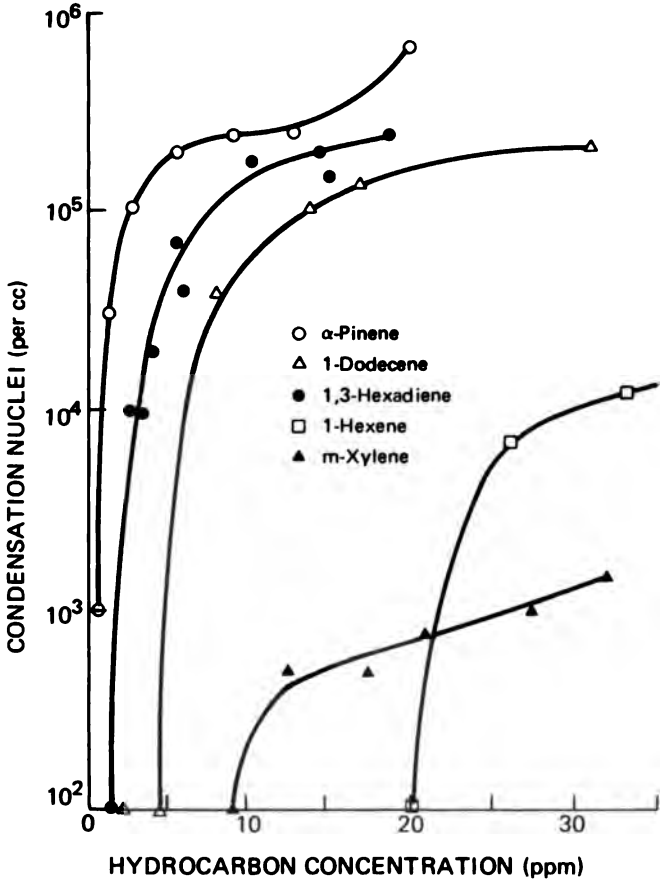


FIGURE 3-20 Threshold concentration for condensation nuclei formation from various hydrocarbons. Reprinted with permission from Lipeles *et al.*[131]

- Threshold concentrations for C_{7+} alkenes are in the parts-per-hundred-million to parts-per-million range. Determination of their *gas-phase concentration* is required for assessing their contribution to atmospheric organic aerosol.
- Nothing is known about the threshold concentration for aromatic hydrocarbons. No vapor-pressure data on the polyfunctional products are available. However, a *possible* threshold concentration of several parts per million seems indicated by data of Kopczynski (mesitylene aerosol at 25 ppm, no aerosol at lower concentration),[124] O'Brien *et al.* (mesitylene aerosol at 10 ppm, no aerosol at 2 ppm),[157] Schwartz

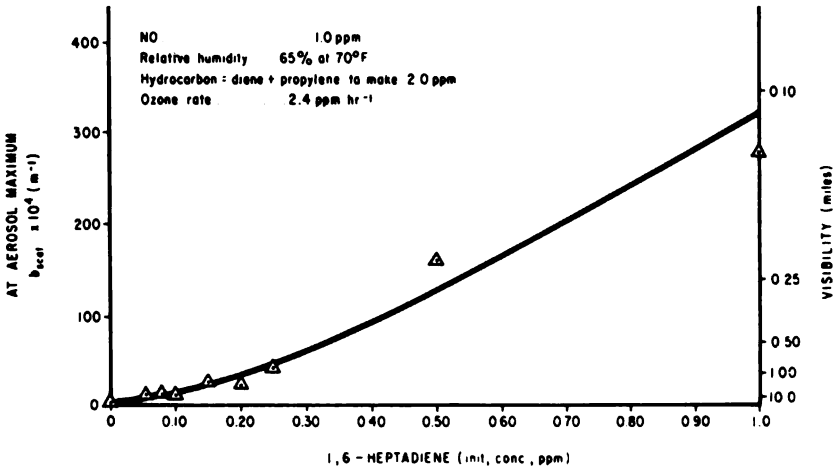


FIGURE 3-21 Threshold concentration for aerosol formation with 1,6-heptadiene. Reprinted with permission from O'Brien *et al.*[157]

(toluene aerosol at 10 ppm),[198] and Grosjean (no toluene aerosol at 1 ppm; unpublished data). The situation is further complicated by the low reactivity of aromatic compounds. Monofunctional compounds formed early would stay in the gas phase, whereas polyfunctional compounds formed later by further oxidation would accumulate in the aerosol phase. Therefore, aerosol formation from aromatics would be favored by meteorologic conditions that allowed long irradiation periods. Further research is necessary for assessing in a more quantitative manner the contribution of aromatic hydrocarbons to atmospheric aerosol.

KINETICS OF ORGANIC AEROSOL FORMATION

Assuming that some of the physical and chemical mechanisms just reviewed are predominant in the formation of organic aerosol, various schemes can be derived that permit a more quantitative description of the time evolution of atmospheric organic aerosol. For example, a kinetic scheme has been proposed recently (Grosjean and Friedlander, unpublished data) for aerosol formation from olefinic precursors that may be applied in principle to other hydrocarbon classes. Starting with this system,

$$\begin{matrix} \diagdown & & \diagup \\ & C{=}C & \\ \diagup & & \diagdown \end{matrix} + \begin{cases} X_1 \xrightarrow{k_1} \\ \vdots \\ X_n \xrightarrow{k_n} \end{cases}, \qquad (3\text{-}3)$$

the olefin concentration is given by:

$$-\frac{d[\text{ol}]}{dt} = \sum_i k_i[X_i][\text{ol}], \tag{3-4}$$

where k_i is the rate constant of olefin reaction with the ith species X_i (O_3, O, $\dot{O}H$, $H\dot{O}_2$, NO_3, ...). However, not all the rate constants k_i have been measured, and the olefin consumption can be estimated from empirical data, such as the nitric oxide to nitrogen dioxide conversion rate constants measured by Glasson and Tuesday.[64] Note also that Equation 3-4 can be written

$$-\frac{d[\text{ol}]}{dt} = \sum_i a_i(t) b_i k_{O_3}[O_3][\text{ol}], \tag{3-5}$$

where $k_i = b_i k_{O_3}$, and that $[X_i] = a_i[O_3]$, where a_i is only a function of time and can be calculated from computer simulation data.

The Ozone–Olefin System

With Equation 3-5, the general problem can be reduced to the ozone-olefin system:

$$\text{C=C} + O_3 \xrightarrow[\text{slow}]{k} \text{I} \begin{cases} \xrightarrow{k_{1i}} \sum P_{1i} & i = 1, p \\ + R_j & \\ \xrightarrow{k_{2j}} \sum P_{2j} & j = 1, q \end{cases} \tag{3-6}$$

where $\sum P_{1i}$ *and* $\sum P_{2j}$ are the products (including other intermediates) that result from unimolecular and bimolecular reactions of the intermediate I, *whose formation is the rate-determining step.*

Assuming a steady-state concentration for I,

$$I = k[O_3][\text{olefin}] \Big/ \left(\sum_i k_{1i} + \sum_j k_{2j}[R_j] \right), \tag{3-7}$$

where

$$R_j = O, O_3, O_2, \dot{O}H, H\dot{O}_2, NO_2, SO_2, \text{ etc.}$$

The condensable species are generally only a fraction of the products:

$$\sum CS_{1i} = \alpha_1 \sum P_{1i} \quad \text{and} \quad \sum CS_{2j} = \alpha_2 \sum P_{2j}.$$

Assuming that the rate of gas-to-particle conversion of *any* condensable species is greater than its rate of formation in the gas phase (which is the case for heterogeneous nucleation predominant in the atmosphere, but may not be valid for homogeneous nucleation in "clean-air" smog-chamber studies):

$$\frac{d[\text{aerosol}]}{dt} = \frac{\sum d\,\text{CS}_{1i}}{dt} + \frac{\sum d\,\text{CS}_{2i}}{dt}$$

$$= [\text{I}](\alpha_1 \sum k_{1i} + \alpha_2 \sum k_{2j}[\text{R}_j]). \qquad (3\text{-}8)$$

From Equation 3-7:

$$\frac{d[\text{aerosol}]}{dt} = k[\text{O}_3][\text{olefin}]\frac{\alpha_1 \sum k_{1i} + \alpha_2 \sum k_{2j}[\text{R}_j]}{\sum k_{1i} + \sum k_{2j}[\text{R}_j]}. \qquad (3\text{-}9)$$

The term

$$\alpha = \frac{d[\text{aerosol}]}{dt}\frac{1}{k[\text{O}_3][\text{olefin}]}$$

is not constant, but is a function of $[\text{R}_j]$. However, α can be constant in the following cases:

- $\alpha \equiv \alpha_1$: if $k_{2j}[\text{R}_j] \ll k_{1i}$ (fast unimolecular decomposition) or if $\alpha_2 = 0$ (no condensable species are formed in the bimolecular pathways)
- $\alpha \equiv \alpha_2$: if $k_{2j}[\text{R}_j] \gg k_{1i}$ or if $\alpha_1 = 0$
- $[\text{R}_j] \doteq$ constant, if the species R_j leading to condensable species by reaction with the intermediate are either in great excess (pseudo-first-order) or at steady-state concentrations (free radicals).

Note that the proposed scheme is general and that Equation 3-9 can be applied to any simpler system. For example, using sulfur dioxide $\equiv \text{R}_1$, $\alpha_1 = 0$, $\alpha_{2j} = 0$ for $j \neq 1$, and $\alpha_{21} = 1$ (all sulfur trioxide formed leads to sulfuric acid aerosol), we find the Cox and Penkett relation:[34]

$$\frac{d[\text{aerosol}]}{dt} = \frac{d[H_2SO_4]}{dt} = k[\text{olefin}][O_3] - \frac{k_{2i}[SO_2]}{\sum k_{2j} + k_{21}[SO_2]}, \quad (3\text{-}10)$$

which has been experimentally verified for the system

$$\text{olefin} + O_3 \xrightarrow{k} I \begin{cases} \xrightarrow{k_{1i}} \sum P_{1i}, \text{ no } CS_{1i} \\ \xrightarrow[+R_j]{k_{2j}} \sum P_{2j}, \text{ no } CS_{2j} \\ \xrightarrow[+SO_2]{k_{2i}} SO_3 \rightarrow H_2SO_4(\text{aerosol}) \end{cases} \quad (3\text{-}11)$$

A relation similar to Equation 3-9 has been found to account satisfactorily for organic aerosols formed from cyclopentene, cyclohexene, and 1,7-octadiene in smog chambers. The aerosol organic carbon concentration as a function of time was measured (Grosjean and Friedlander, unpublished data) with an organic-carbon analyzer (Figure 3-22). The relation obtained was:

$$\frac{d[\text{organic aerosol carbon}]}{dt} = \alpha_C \alpha k [O_3][\text{olefin}], \quad (3\text{-}12)$$

where α_C is the percentage of organic carbon averaged over all condensable species.

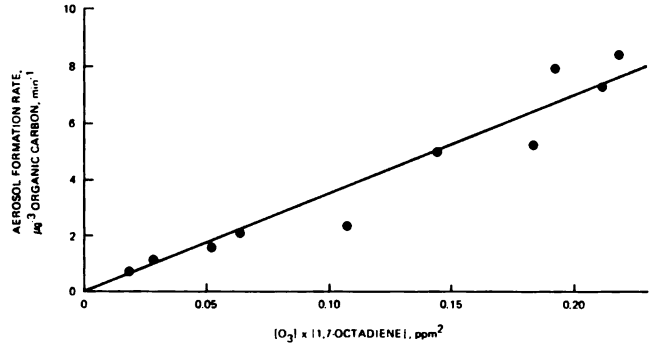


FIGURE 3-22. Aerosol formation rate as a function of reactant concentrations. Initial concentrations: NO, 0.17 ppm; NO_2, 0.33 ppm; 1,7-octadiene, 1.18 ppm.

The Ozone–Multihydrocarbon System

The same kinetic scheme can be used for the most general situation involving multicompetitive olefin–ozone reactions:

$$
O_3 + \begin{cases}
ol_1 \xrightarrow{k_1} I_1 \begin{cases} \xrightarrow{k_{1i}^1} \sum P_{1i}^1 & i = 1, p \\ \xrightarrow[+R_j]{k_{2j}^1} \sum P_{2j}^1 & j = 1, q \end{cases} \\
ol_2 \xrightarrow{k_2} I_2 \begin{cases} \xrightarrow{k_{1i}^2} \sum P_{1i}^2 \\ \xrightarrow[+R_j]{k_{2j}^2} \sum P_{2j}^2 \end{cases} \\
ol_n \xrightarrow{k_n} I_n \begin{cases} \xrightarrow{k_{1i}^n} \sum P_{1i}^n \\ \xrightarrow[+R_j]{k_{2j}^n} \sum P_{2j}^n \end{cases}
\end{cases} \tag{3-13}
$$

In this case, the ozone concentration is such that it satisfies simultaneously the m relations:

$$[I]_m = k_m[ol_m][O_3]/\left(\sum k_{1i}^m + \sum k_{2j}^m[R_j]\right) \qquad m = 1, n \tag{3-14}$$

With the only assumption being, as before, that the intermediate formation step is slower than any further reactions, including transfer of condensable species into the aerosol phase, we obtain the relation

$$\frac{d[\text{aerosol}]}{dt} + \sum_{m=1,n} \frac{d[\text{aerosol}]}{dt} m = [O_3] \sum_{m=1,n} \alpha_m k_m [ol]_m, \tag{3-15}$$

where, as before:

$$\alpha_n = \alpha_1^n \sum k_{1i}^n + \alpha_2^n \sum k_{2j}^n[R_j]/\left(\sum k_{1i}^n + \sum k_{2j}^n[R_j]\right).$$

Note that Equation 3-15 expresses in a more quantitative manner the previously discussed dependence of aerosol formation on the product of a gas-phase reactivity term, k_m, and an aerosol formation ability term, α_m.

Applications to the Atmosphere

The general relation of Equation 3-15 can be used for estimating amounts of organic aerosol formed from a multihydrocarbon mixture, provided that four parameters—$[O_3]$, α_m, k_m, and $[ol]_m$—are known. Ozone concentration can be readily measured. Aerosol formation ability factors, α_m, can be measured in smog-chamber experiments or estimated (α_m = 0 for paraffins, acetylenics, and C_{1-5} alkenes). Note also that the measured ambient gas-to-particle distribution factor f_C[79] is an upper limit for $\sum \alpha_m$. Rate constants have been measured for some olefins.[14,21,34,86,93,110,227,229] Unknown rate constants for the ozone–olefin reaction can be estimated from published linear relations between k_{O_3} and alkene ionization potentials and between k_{O_3} and the rate constants for other electrophilic additions. The last parameter, olefin concentration, can be measured or estimated from data on the composition of gasolines and automobile exhaust (see the following section). When combined with an estimation of air-mass trajectories and residence time derived from meteorologic data, use of this type of kinetic relation would permit predicting the amount of secondary organic aerosol present at a given location of an urban basin by measuring the local ozone concentration. However, it is not assumed so far that such a relation as Equation 3-15 is valid for all types of unsaturated and aromatic precursors, and its application to the atmosphere is limited by the scarcity of data on ambient precursor concentrations.

GAS-PHASE HYDROCARBON PRECURSORS

More than 100 compounds are released in the atmosphere of urban areas by automobiles, and there is a close relation between the atmospheric hydrocarbon composition and the composition of gasolines and automobile exhausts. The full range of compositions of gasolines has been reported by Sanders and Maynard.[137,185] They identified 180 of the 240 compounds separated by capillary-column gas chromatography. Detailed fuel compositions were reported by other investigators,[65,141,152] and exhaust hydrocarbon compositions were reported by Neligan *et al.*,[151,152] McEwen,[138] and, more recently, Papa *et al.*,[159] Jacobs,[105] and Dishart.[46] Exhaust compositions were found to be very sensitive to vehicle regime[138] and fuel composition. Methane, ethane, ethylene, and acetylene due to fuel cracking always represent at least 30% of the exhaust. Ambient hydrocarbons with no more than six carbon atoms are now measured on a routine basis,[4,125,126,151,202,210,211,218] but data on C_{7+} hydrocarbons have been limited to the paraffins and aromatics,[4,125,126,202]

Figure 3-23 illustrates the wide variety of hydrocarbons found in gasoline, auto exhaust, and urban air.

Alkenes

Surprisingly, in view of the wealth of publications dealing with the hydrocarbon composition of polluted atmospheres, there is virtually no information on the existence and ambient concentrations of C_{7+} alkenes. Because of their reactivity and the stringent restriction on the olefinic content in gasolines sold in California, C_{7+} alkenes have not been found in California ambient air. Only a few of them have been identified elsewhere.[16,75,115,176] On the basis of the difference between nonmethane hydrocarbon concentrations and the sum of all $C_{<6}$ hydrocarbons measured individually, the C_{7+} fraction (paraffins + olefins + aromatics) accounts for about half the nonmethane hydrocarbon concentration. In the absence of any data for C_{7+} alkenes, their *possible* concentrations were estimated by scaling up gasoline and auto-exhaust data to match the morning ambient concentration of 1-hexene. This crude estimate indicates that C_{7+} alkenes may be present at about 100 ppb in the atmosphere during the morning traffic period (Grosjean and Friedlander, unpublished data). These alkenes are listed in Table 3-12.

Cyclic Olefins

Five cyclic olefins have been identified in gasolines and auto exhaust, cyclopentene being the only one detected in the atmosphere.[151,210,211] The five cyclic olefins account for only about 0.5% by weight of gasolines and 0.6% by volume of exhausts. Their ambient concentrations were estimated by scaling up gasoline and auto-exhaust data to match the measured ambient morning cyclopentene concentration and from the measured ambient concentrations of C_{5-7} aerosol difunctional compounds. The two estimates agree and indicate that cyclic olefins may be present in the morning atmosphere at 10–50 ppb. On the contrary, C_{6+} diolefins are not present in gasolines and exhaust gases and have not been found in the atmosphere (Table 3-13).

Terpenes

The possibility of widespread haze formation by sunlight irradiation of terpenoid compounds from vegetation was first suggested by Went.[230] Went,[231] Rasmussen and Went,[175] and Rasmussen[173] estimated the annual worldwide contribution of forest hydrocarbon emission and con-

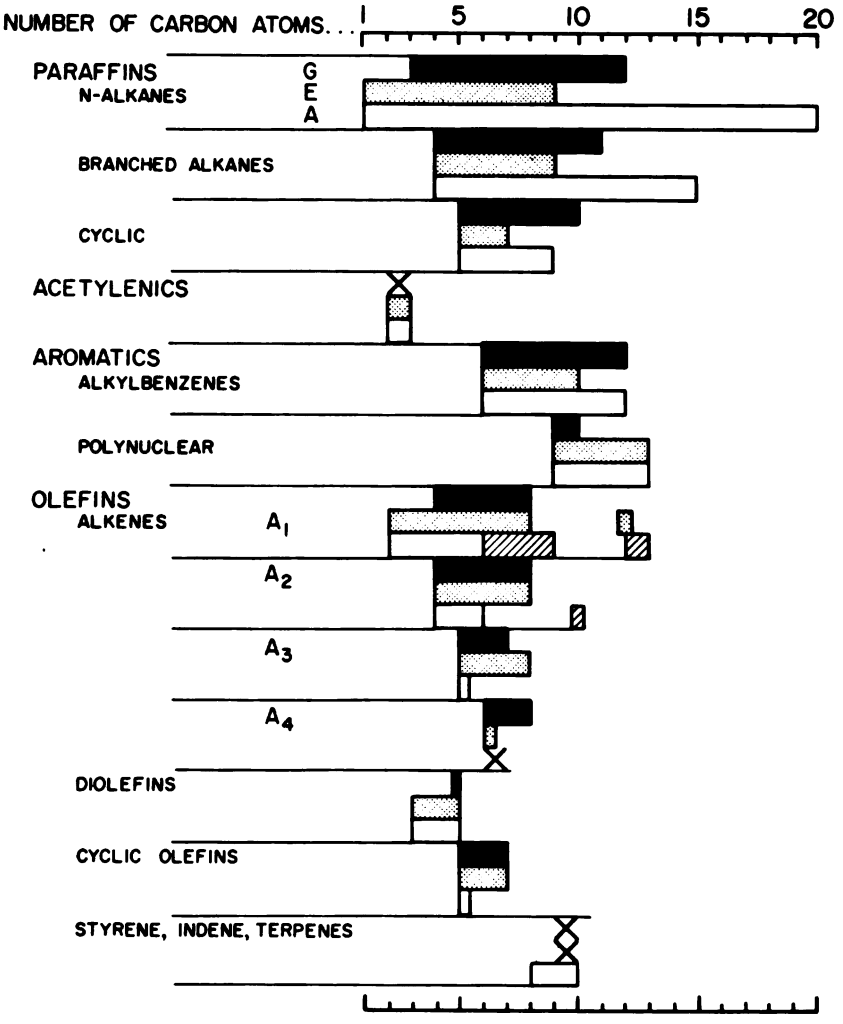
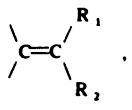


FIGURE 3-23 Hydrocarbons in gasolines (G), automobile exhaust (E), and ambient urban air (A). A_1, A_2, A_3, A_4 = mono-, di-, tri-, and tetra-substituted alkenes. X = not found. Hatching = not found in California.

TABLE 3-12 Potential Aerosol Organic Precursors Identified in Gasolines and Automobile Exhaust

Alkenes[a]	Gasolines	Auto Exhaust	Ambient Air	Expected Photochemical Products
2,3,3-Trimethyl-1-butene	+	+	—	
3-Methyl-2-ethyl-1-butene	+	—	—	
2,3-Dimethyl-1-pentene	+	—	—	
2,4-Dimethyl-1-pentene	+	—	—	
3,3-Dimethyl-1-pentene	+	—	—	Monofunctional compounds:
3,4-Dimethyl-1-pentene	—	+	—	
4,4-Dimethyl-1-pentene	+	—	—	aldehydes
2-Ethyl-1-pentene	+	—	—	
3-Ethyl-1-pentene	+	—	—	ketones
2-Methyl-1-hexene	+	—	—	
3-Methyl-1-hexene	+	—	—	nitrates
4-Methyl-1-hexene	+	—	—	
5-Methyl-1-hexene	+	+	—	carboxylic acids
1-Heptene	+	+	+[b]	
2,4,4-Trimethyl-1-pentene	—	+	—	
2-Ethyl-1-hexene	+	—	—	(also unsaturated oxygenates
cis-2-Octene	—	+	—	if allylic oxidation occurs)
trans-2-Octene	+	+	—	
1-Octene	+	+	+[b]	
Isodecene	—	—	+[b]	
1-Dodecene	—	+	+[b]	
Diolefins				
Propadiene	—	+	+	
1,3-Butadiene	—	+	+	
2-Methyl-1,3-butadiene	+	+	+	Dicarboxylic acids and other
1,3-Pentadiene	—	+	—	difunctional compounds
α,ω-C_5 + diolefins	—	—	—	
Cyclic olefins				
Cyclopentene	+	+	+	
1-Methylcyclopentene	+	—	—	
3-Methylcyclopentene	+	—	—	Dicarboxylic acids and other
Cyclohexene	+	+	—	difunctional compounds
3-Methylcyclohexene	—	+	—	
4-Methylcyclohexene	—	+	—	

[a] Alkenes leading to a C_6+ fragment:



with $R_1 + R_2 \geq 5$ carbon atoms.
[b] Not identified in California.

TABLE 3-13 Estimated Cyclic Olefin Contribution to Ambient Organic Aerosol[a]

Precursor	Gasoline, wt %[b]	Exhaust, ppb in 2-ppm NMHC[b]	Ambient Air, ppb	
			Measured[b]	Estimated (morning Los Angeles)[c]
	0.13–0.18	1.0	2–9 Los Angeles 4.4 Riverside	5
	0.04–0.08	+	—	2
	0.14–0.18	+	—	5
	0.14–0.55	5.0	—	12
	+		—	6
		6.0		
	+		—	6
Total	0.45–1.0	12		36

[a] Data from Grosjean and Friedlander (unpublished).
[b] +: identified, but concentration not measured; —: not found.
[c] From gasoline and automobile exhaust data; assuming an average molecular weight of 82 (that of cyclohexene), 100% gas-phase reaction, 30% gas-to-aerosol conversion, 36-ppb cyclic olefins would form aerosol difunctional compounds at about 36 $\mu g/m^3$.

cluded that more reactive hydrocarbons are released by tree foliage than by man's activities. The major compounds emitted are monoterpenes (C_{10})—like α-pinene, β-pinene, limonene, and myrcene[62,173]—and the hemiterpene (C_5) isoprene.[172] The fate of these gaseous olefins in the atmosphere is still undetermined. Went[232] noticed that irradiated mixtures of nitrogen dioxide and α-pinene produced ozone and fine particles and postulated that terpene–nitrogen oxide reactions, similar to the olefin–NO_x reaction of polluted urban areas, may take place in forested rural areas. Rasmussen and Holdren[174] indicated that, on an individual basis, monoterpene hydrocarbon concentrations are in the low parts-per-billion range in rural air.

The gas-phase reactivity of various terpenes has been measured. Stephens and Scott[213] were the first to include two terpenes (pinene and α-phellandrene) with their study of the relative reactivity of various hydrocarbons. Both monoterpenes showed the high reactivity predicted by their olefinic structure. Conversion of nitric oxide to nitrogen dioxide in the presence of isoprene is at a rate intermediate between those for ethylene and *trans*-2-butene,[64] and Japar *et al.* reported rate constants for the α-pinene and terpinolene–ozone reactions.[109] Grimsrud *et al.*[74] measured the rate con-

stants for the reaction of various terpenes with ozone and with nitric oxide, the latter at the low concentrations (10-ppb hydrocarbon and 7-ppb nitric oxide) observed in rural areas. Their data are listed in Table 3-14. Structural effects have a major influence on terpene reactivity, and olefinic terpenes react with ozone at rates comparable with those of the most reactive alkenes, such as tetramethylethylene. Grimsrud *et al.* also established that the atmospheric reactivity of very reactive terpenes is due to their reaction with ozone, whereas atmospheric reactivity of less reactive terpenes is controlled by other mechanisms that involve free radicals. Therefore, photochemical reactions of terpenes and nitric oxide may contribute, in part, to the high ozone concentrations ($\geq$20 ppb, and sometimes exceeding 80 ppb) observed in rural daytime air.[179,206]

Aerosol formation from terpenes has not been extensively investigated. Only one olefinic terpene, α-pinene, has been studied.[76,131,180,181,198] Ripperton and Lillian[181] reported condensation nuclei formation from irradiated mixtures of 0.1-ppm nitrogen dioxide and 0.5-ppm α-pinene. Fewer condensation nuclei were obtained when increasing the water vapor concentration. Infrared spectra of α-pinene aerosol[76,131,180] indicate the presence of organic compounds, such as carbonyls, carboxylic acid, and nitrate ester. As mentioned earlier, the composition of α-pinene aerosol was studied in great detail by Schwartz (Figure 3-8). Similar polyfunctional compounds of low volatility are expected to be formed from other olefinic terpenes (β-pinene, limonene) and probably constitute a major fraction of the natural blue haze aerosols formed over forested areas. It is noteworthy that α-pinene and its aerosol products are also present at significant concentrations in urban air.[122] Diurnal profiles of the products are indicative of their secondary origin (Figure 3-24). It has not been determined whether tree foliage, solvent use (turpentine?), or transport from nearby rural regions is responsible for the presence of terpenes in urban areas.

Relative Importance of Various Hydrocarbon Classes in the Formation of Secondary Organic Aerosols

The contribution of the various classes of hydrocarbons to the formation of particulate organic compounds is a complex function of their relative ambient concentrations, gas-phase reactivity, and ability to form products whose physical properties, especially vapor pressures, are of prime importance in the physical mechanisms controlling the gas-to-aerosol conversion process. In view of the results discussed previously, cyclic olefins appear to be the most important class of organic aerosol precursors. This is due to their high gas-phase reactivity and their ability

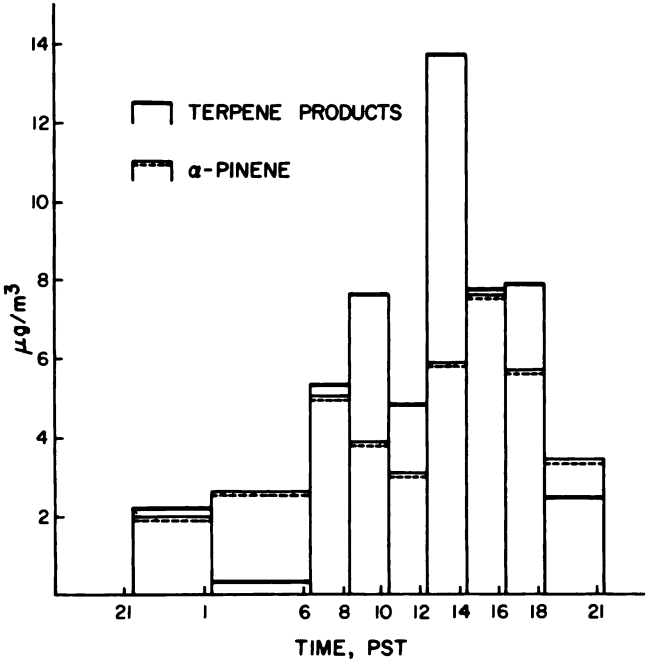


FIGURE 3-24 Diurnal profiles of α-pinene and total terpene products. West Covina, California, July 24, 1973. See ozone profile, Figure 3-11. Adapted from Knights *et al.*[122]

to form difunctional compounds of very low volatility. Such difunctional compounds constitute the major fraction of ambient secondary organic aerosols, and most of them have been identified in smog-chamber experiments.

The observed ambient organic aerosol formation rates are also consistent with those estimated by extrapolation of smog-chamber kinetic data. Other heavy unsaturates, such as styrene and indene, are present in the atmosphere and may contribute, in part, to the formation of benzoic acid and homophthalic acid, respectively. Diesel exhaust and industrial emission are possible sources of such heavy unsaturates. Diolefins of C_{6+} are not present in gasolines and exhaust gases and have not been found in the atmosphere, and their possible role as precursors of the C_{5-7} difunctional acidic compounds is seriously challenged. Lower diolefins are emitted in automobile exhaust. Examination of vapor-pressure data indicates that the bulk of their expected photooxidation products remains in the gas phase, including most of the less volatile C_{3-4} dicarboxylic acids.

TABLE 3-14 Photoxidation and Ozonolysis Rates of Monoterpene Hydrocarbons[a]

Hydrocarbon	Photooxidation Rate (k),[b] $s^{-1} \times 10^4$	Ozonolysis Rate		
		k,[c] $ppm^{-1}\ s^{-1}$	$k \times 30$ ppb, $s^{-1} \times 10^4$	Ratio,[d] $R_{O_3}:R_{NO_x}$
p-Menthane	0.11	—	—	—
p-Cymene	0.25	—	—	—
Isobutene	0.84	3.6×10^{-4}	0.11	0.13
β-Pinene	1.1	9×10^{-4}	0.27	0.25
Isoprene	1.3	—	—	—
α-Pinene	1.3	3.6×10^{-3}	1.1	0.85
3-Carene	1.4	3×10^{-3}	0.9	0.64
β-Phellandrene	1.9	4.4×10^{-3}	1.3	0.69
γ-Terpinene	—	7×10^{-3}	2.1	—

Carvomenthene		2.1	1.3 × 10⁻²	3.9	2.0
Limonene		2.4	1.6 × 10⁻²	4.8	2.0
Dihydromyrcene		3.0	1.7 × 10⁻²	5.1	1.7
Myrcene		3.8	3.1 × 10⁻²	9.3	2.4
Ocimene		5.3	5 × 10⁻²	15	2.8
Terpinolene		11	2.5 × 10⁻¹	75	6.8
α-Phellandrene		12	2.9 × 10⁻¹	87	7.2
α-Terpinene		55–110	2.2	630	6.3–12.0

[a] Data from Grimsrud *et al.*[74]
[b] Irradiation vessels contained 7-ppb NO_x and 10-ppb hydrocarbon.
[c] Ozone and hydrocarbon initial concentrations, 0.1–5 ppm.
[d] For a typical rural ozone concentration of 30 ppb.

TABLE 3-14 Photoxidation and Ozonolysis Rates of Monoterpene Hydrocarbons[a]

Hydrocarbon	Photooxidation Rate (k),[b] $s^{-1} \times 10^4$	Ozonolysis Rate		
		k,[c] $ppm^{-1}\ s^{-1}$	$k \times 30$ ppb, $s^{-1} \times 10^4$	Ratio,[d] $R_{O_3}:R_{NO_x}$
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[a] Data from Grimsrud *et al.*[74]
[b] Irradiation vessels contained 7-ppb NO_x and 10-ppb hydrocarbon.
[c] Ozone and hydrocarbon initial concentrations, 0.1–5 ppm.
[d] For a typical rural ozone concentration of 30 ppb.

Although the ambient concentrations of alkenes are about 10 times higher than those of cyclic olefins, their contribution to the formation of organic aerosols is much lower in both smog-chamber experiments and the ambient atmosphere. Here again, examination of vapor-pressure data reveals that vapor pressures of monofunctional oxygenates expected from alkenes exceed by several orders of magnitude those of the difunctional compounds that have the same numbers of carbon atoms. Thus, the saturation concentration for monofunctional compounds and their later gas-to-aerosol conversion can be reached only under "favorable" conditions, i.e., at high precursor (alkene) concentrations. However, ambient concentrations of C_{6+} alkenes have not been measured, and their role as aerosol precursors cannot be ruled out. The importance of aromatic hydrocarbons remains to be determined. A "compensation effect" is expected between their low reactivity and the possible aromatic ring cleavage that leads to multifunctional compounds of low volatility. On the basis of all available ambient-air and laboratory data, cyclic olefins can be reasonably postulated as the most important class of secondary organic aerosol precursors. However, aromatics and, to a lesser extent, alkenes may be important after a sufficiently long period, when the more efficient aerosol-forming precursors have disappeared. Unfortunately, there are no laboratory data based on periods long enough to simulate the slow transport of air masses ($\geqslant$12 h) encountered in some urban areas (such as the eastern part of the Los Angeles basin) where aromatics may outweigh cyclic olefins as major organic aerosol precursors.

FORMATION OF SECONDARY INORGANIC AEROSOLS IN PHOTOCHEMICAL SYSTEMS

Recent studies suggest that potentially harmful health effects may be associated with moderately high concentrations of sulfates[222] and nitrates[56] and that both sulfate and nitrate aerosols contribute more than organic particles to visibility degradation in urban areas.* [79,94-98] Nitrates and sulfates in atmospheric aerosols can be formed by a wide variety of homogeneous and heterogeneous reactions. Heterogeneous reactions have been thoroughly studied and were considered, until recently, as the major pathways for sulfur dioxide and NO_x removal and particle formation. However, studies of various homogeneous reactions initiated in the last few years suggest that some of these reactions can compete effectively with heterogeneous processes. The purpose of this section is

*J. Barone, personal communication.

to examine the possible relation between ozone and the two major secondary inorganic aerosol species, sulfates and nitrates. Accordingly, only gas-phase photochemical reactions relevant to sulfate and nitrate formation are reviewed, and their relative importance in the overall aerosol formation process is assessed where possible. Information on the corresponding heterogeneous reactions can be found in several reviews.[22,29,30,94-98,100,147]

Nitrate Aerosols

The two important aerosol nitrate precursors are nitrous acid and nitric acid, formed mainly in the reactions

$$NO + NO_2 + H_2O \rightarrow 2HONO, \quad (3\text{-}16)$$

$$N_2O_5 + H_2O \rightarrow 2HONO_2, \quad (3\text{-}17)$$

$$NO_2 + OH + M \rightarrow HONO_2 + M. \quad (3\text{-}18)$$

With currently accepted rate constants for these reactions, Calvert[30] estimated that Reaction 3-18 is of principal importance for nitric acid formation in smog. Nitric acid has recently been identified in both smog chambers[29] and Los Angeles air.[144] However, both nitrous acid and nitric acid have high vapor pressures,[120] and it does not appear possible that particulate nitrogen species will exist in the atmosphere in pure form as acids. Infrared, x-ray, and chemical analyses indicate that ammonium nitrate is the major constituent of the ambient particulate nitrate fraction.[70,94-98,133,209] Ammonium nitrate is also found in aerosol formed from NO_x-hydrocarbon mixtures irradiated in the presence of ammonia in smog chambers.[157] Thus, the production of aerosol nitrate requires neutralization by ambient ammonia. It is not certain whether the reaction takes place in the gas phase or in the liquid phase after fast nitric acid diffusion into the aerosol droplets. Urban ammonia concentrations have been estimated at up to 0.2 ppm (140 $\mu g/m^3$), with averages of one-tenth that value.[106] High aerosol nitrate concentrations are observed in the eastern part of the Los Angeles basin,[94-98,209] where ammonia emission from feed lots is thought to be important. Also, diurnal aerosol nitrate profiles do not correlate with nitrogen oxide concentrations. Therefore, aerosol nitrate formation may be limited by ambient ammonia, rather than by any of the nitrogen oxide species.

The relative importance of the heterogeneous and photochemical nitrate formation pathways can be assessed from recently measured aerosol nitrate profiles.[79,94-98] On humid days, a midmorning nitrate

peak is usually observed, which tracks rather poorly the morning rush hour NO_x peak and indicates that heterogeneous processes are predominant. On oxidant episode days, however, particulate nitrate concentrations correlate with ozone concentrations. Diurnal profiles of several nitrogen species are presented in Figure 3-25.[79,87] In this case, photochemical production of aerosol nitrate at a rate of about 10 $\mu g/m^3$ per hour was observed.

Sulfate Aerosols

As mentioned before, heterogeneous sulfur dioxide oxidation reactions

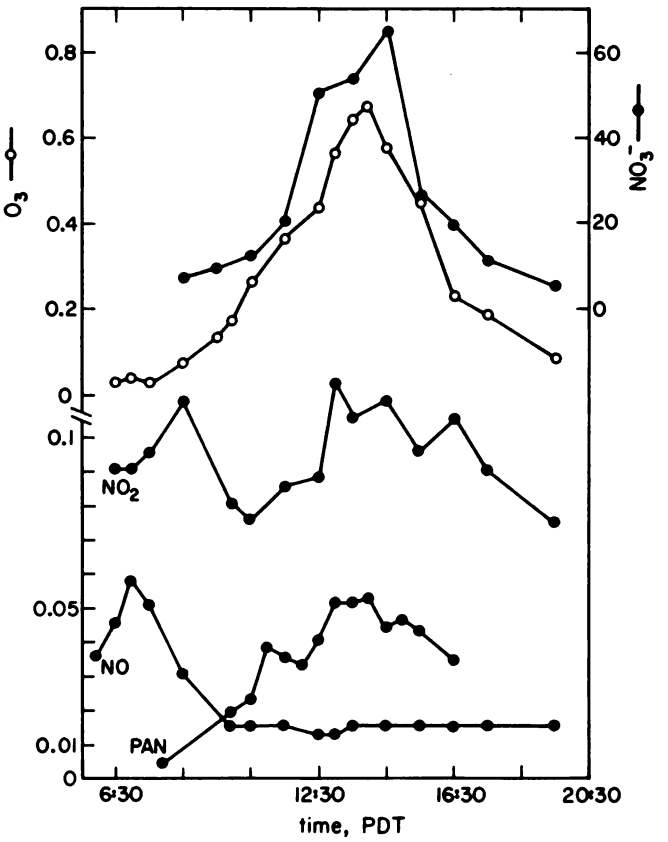


FIGURE 3-25 Diurnal profiles of nitrogen compounds, Pasadena, California, July 25, 1974. NO, NO_2, O_3, and PAN in ppm; inorganic aerosol nitrates in $\mu g/m^3$. Data from Grosjean and Friedlander[79] and Hanst *et al.*[87]

have been extensively studied.[22,29,30,100,147] Two reactions of this type are thought to be particularly important: the aqueous oxidation of sulfur dioxide in water droplets and the catalytic oxidation of sulfur dioxide adsorbed on carbon particles. Because ozone is involved in the former reaction and carbon particles are generated by incomplete combustion of hydrocarbons, these two reactions will be examined hereafter. Novakov *et al.*[153,154] have observed that significant amounts of sulfate ion can be found on carbon particles generated by combustion of hydrocarbons in air enriched with sulfur dioxide in the parts-per-million range. It is difficult to assess the significance of carbon or organic particles for the sulfur dioxide oxidation in the atmosphere. There is little doubt that adsorption of sulfur dioxide on carbon particles freshly generated by combustion can provide a surface-catalyzed oxidation medium. Indeed, such experiments as those of Yamamoto *et al.*[243] have shown that sulfur dioxide oxidation can be as high as 30%/h on activated-charcoal particles less than 5 mm in diameter. Their work also indicates that this rate is strongly reduced by sulfuric acid collection in the micropores of the charcoal. The work of Yamamoto *et al.* further emphasizes that such a heterogeneous oxidation mechanism depends on a variety of factors, including grain size of the carbon, temperature, and concentrations of sulfur dioxide, water vapor, and oxygen, as well as the micropore structure of the particle surface. It would seem that oily, gummy, wet particles collected from the atmosphere would be poorly suited for nonaqueous reactions to form sulfate, in that their micropore structure would be minimal. Yet such a mechanism cannot be ruled out.

The class of reactions that have been used most often to explain high sulfur dioxide rates in the presence of aerosols that contain water is the system involving sulfur dioxide absorption in water followed by oxidation by dissolved oxygen and/or ozone to form sulfate. Catalysis of the oxidation by heavy metal salts, such as Mn^{++} ion, can realize rates of oxidation in excess of 1%/h in clean water solutions (e.g., Johnstone and Coughanowr[113]). The absorption of sulfur dioxide can be promoted by the buffering effect of simultaneous absorption of ammonia. Scott and Hobbs[199] have shown that the aqueous sulfur dioxide oxidation process can be enhanced significantly by ammonium ion. Indeed, the estimates and experiments of Miller and de Pena[145] suggest that rates of sulfur dioxide oxidation can be achieved in fog approaching 10%/h.

It is well known that ozone is quite soluble in water. Therefore, one expects that ozone absorption with sulfur dioxide would contribute to significant oxidation of sulfur dioxide. Experiments of Penkett[161] have shown that oxidation of sulfur dioxide in air at 7 ppb when absorbed in water droplets with ozone, which is present in surrounding air at 0.05 ppm, can be as large as 13%/h. Thus, foggy or cloudy air mixed with

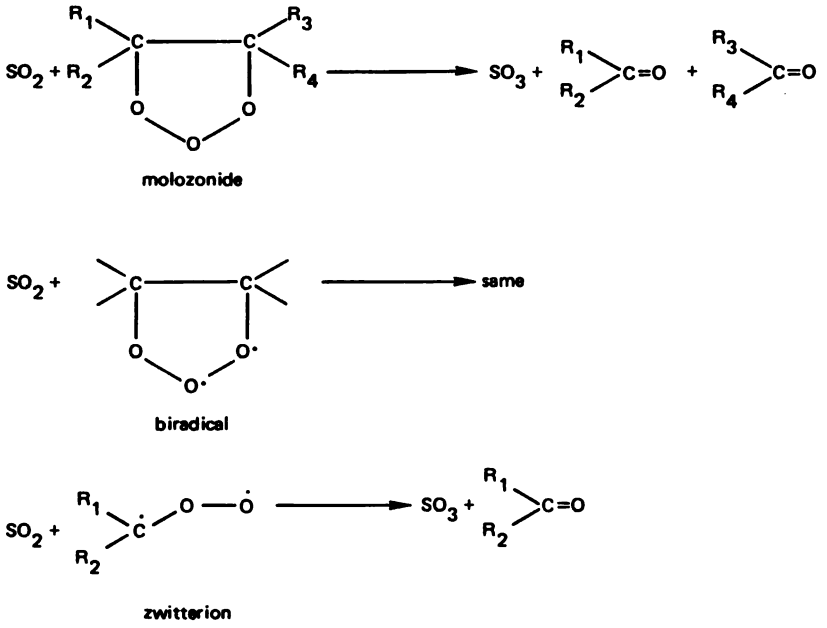
photochemical smog, such as occurs sometimes along the Pacific Coast, could well be an important medium for sulfate ion formation. Furthermore, such an aqueous mechanism could be significant at middle altitudes over continents even at background ozone concentrations.

The reported rates of sulfur dioxide oxidation in clean water droplets must be considered maximal. It is questionable whether they can ever be achieved in the atmosphere, inasmuch as such aqueous reactions have been shown to be suppressed significantly by organic contaminants. The work of Fuller and Crist[59] and later of Schroeter[190] has indicated that the aqueous absorption of sulfur dioxide and its later oxidation are reduced by as much as an order of magnitude by dissolved organic acids or alcohols that are known to be present in atmospheric aerosol. Much experimental evidence has been accumulated on homogeneous sulfur dioxide oxidation reactions. All investigators report aerosol sulfate formation when sulfur dioxide is added to mixtures of NO_x and hydrocarbons in smog-chamber experiments (see, for example, Table 3-5). The observed enhancement of sulfur dioxide oxidation depends strongly on the hydrocarbon structure (Table 3-6). Alkenes with fewer than seven carbon atoms, which constitute the bulk of ambient unsaturates and do *not* form organic aerosol, always produce sulfate aerosol when sulfur dioxide is added. On the contrary, there is no significant sulfate formation and neither the nature nor the yields of organic aerosol formed by cyclic olefins are affected by adding sulfur dioxide. An "intermediate" class is represented by C_{7+} alkenes (for example, 1-heptene[236]), which produce some organic aerosol in the absence of sulfur dioxide and both organic and sulfate aerosol when sulfur dioxide is present. Addition of sulfur dioxide does not seem to affect aerosol formation from aromatic hydrocarbons significantly.[142,239] Such a striking hydrocarbon effect is not well understood and may reflect the differences in the nature or the stability of the intermediates involved for each hydrocarbon class.

Various homogeneous reactions have been postulated to account for sulfur dioxide oxidation in irradiated NO_x-hydrocarbon-sulfur dioxide sytems. Gas-phase inorganic reactions of sulfur dioxide with oxygen plus light, ozone, oxygen atoms, nitrogen dioxide, nitrogen trioxide, and nitrogen pentoxide have been considered severely rate-limited on the basis of available rate data (e.g., Calvert[29,30]). Listed in Figure 3-26 are other reactions that appear to be important in the atmosphere. These reactions are divided into two subcategories whose end products are sulfur trioxide and organic sulfur species. The first three reactions in Figure 3-26 correspond to the interpretation of Cox and Penkett's[34] observations that sulfur dioxide is oxidized at appreciable rates in the dark in ozone-olefin-air mixtures. The higher rate of 3%/h was found for *cis*-2-pentene,

I. PRECURSOR = SULFUR TRIOXIDE

1. Reactions with intermediates of the ozone–olefin reaction:



2. Reactions with organic free radicals:

$$SO_2 + HO_2 \rightarrow OH + SO_3$$
$$SO_2 + RO_2 \rightarrow OR + SO_3$$

II. PRECURSOR = ORGANOSULFUR SPECIES

$$SO_2 + OH \rightarrow HOSO_2$$
$$SO_2 + OR \rightarrow ROSO_2$$
$$SO_2 + HO_2 \rightarrow HO_2SO_2$$
$$SO_2 + RO_2 \rightarrow RO_2SO_2$$

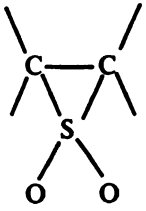
FIGURE 3-26 Atmospheric SO_2 oxidation to aerosol sulfate homogeneous gas-phase organic reactions.

whereas the lower rate of 0.4%/h was found for propylene. Cox and Penkett suggested that either the ozonide or the zwitterion intermediates were involved in sulfur dioxide oxidation. However, Calvert's estimated lifetimes of these olefin-ozone intermediates do not favor their importance as oxidizing agents. Other radical species—such as hydroxyl, alkoxy, hydroperoxy, and alkyperoxy—may well account for sulfur dioxide oxidation.

The importance of the hydroxyl-sulfur dioxide reaction was postulated by Wood *et al.*[242] With measured rate constants for hydroperoxy-sulfur dioxide (Davis *et al.* [44]) and other reactions (James *et al.*;[108] S. Gordon and E. Hamilton, personal communication), Calvert estimated sulfur dioxide oxidation rates by hydroperoxy, methylperoxy, hydroxyl, and methoxy radicals to be 0.85, 0.16, 0.23-1.4, and 0.48%/h, respectively.

It is generally accepted that aerosol sulfate formation requires formation of sulfur trioxide and later fast sulfur trioxide reaction with water. However, consideration should be given to other possible sulfur dioxide and sulfur trioxide reactions. For example, Urone and Schroeder[225] and Bourbigot *et al.*[18] reported nitrosylbisulfate, $NOHSO_4$, formation during photolysis of sulfur dioxide-NO_x mixtures, whereas Daubendiek and Calvert[41] reported the formation of an unidentified white solid in the sulfur trioxide-nitrogen dioxide reaction. The significance of this compound in urban aerosol formation should be evaluated. Moreover, it appears from reactions listed in Figure 3-26 that *various organic sulfur species must also be considered as potential sulfate aerosol precursors.* The radical addition products, such as $OHSO_2$, should react rapidly with other species to form sulfuric acid, peroxysulfuric acid, alkylsulfates, and mixed anhydrides, such as $HOSO_2ONO_2$. Any of these ultimately should lead to sulfate in the presence of water.

Although no conclusive evidence has been reported so far, the possible importance of organic sulfur species as sulfate aerosol precursors is supported by several observations. Sulfuric acids, sulfonic acids, and other organic sulfur compounds are formed in sulfur dioxide-hydrocarbon reactions at high concentrations.[40,112,216] Organosulfur radical species, such as RSO_2 and RO_2SO_2 have been postulated as intermediates for these reactions.[10,162,203] Suzuki (see Penzhorn *et al.*[163]) observed polymer formation from



units in the sulfur dioxide–*cis*-butene reaction. More recently, Schulten and Schurath[197] reported several organosulfur compounds in aerosol formed from the sulfur dioxide-ozone-1-butene system. They tentatively identified these compounds as zwitterion-sulfur dioxide addition products and/or mixed anhydrides of sulfuric acid and sulfurous acid with several organic acids. These results are most interesting, inasmuch as the system was studied at concentrations approaching those of interest in polluted atmosphere.

Summing all the known homogeneous reactions for sulfur dioxide oxidation, it is possible to rationalize a theoretical rate of sulfate formation in the range 1.7-5.5%/h for moderate photochemical smog conditions. Sulfur dioxide oxidation rates in nonphotochemically polluted areas, such as central Europe[163] and St. Louis,[100] are in the range of 0.5-1.0%/h. Much higher sulfur dioxide oxidation rates, up to 13%/h, have been measured in the Los Angeles basin for days of low humidity and high oxidant[182] (see also gas-particle distribution factor f_S in Table 3-4). A strong influence of photochemistry is demonstrated for submicrometer sulfate aerosol by the systematic increase in $f_S < 0.5\,\mu$m with ozone concentration at various locations in the southern California air basin (Figure 3-27). It appears, therefore, that conversion of sulfur dioxide to sulfate is significantly enhanced by homogeneous reactions in photo-

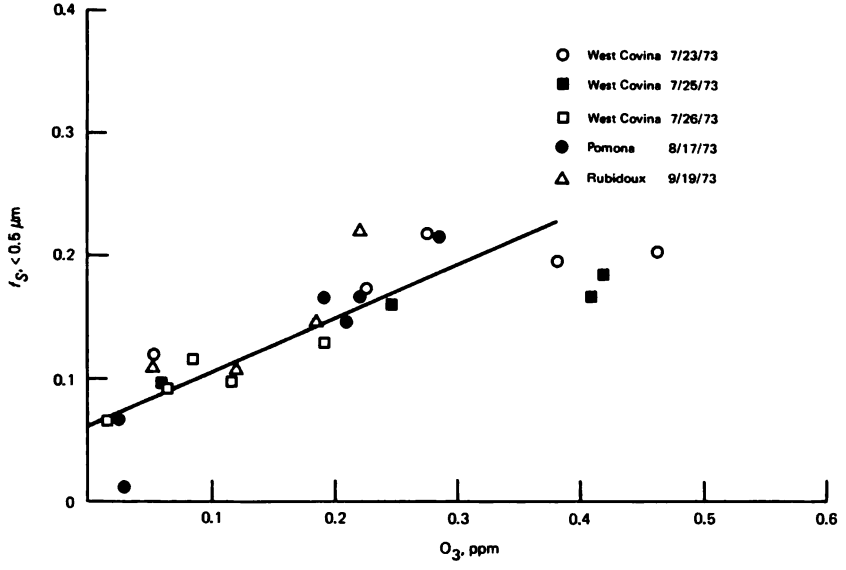


FIGURE 3-27. Scatter diagram of the conversion ratio f_S based on particles smaller than 0.5 μm vs. 2-h averaged ozone concentration. Data from Hidy.[97]

chemically polluted atmosphere. It is possible, however, that the sulfate problem in the next few years will be dominated by other factors, such as the use of high-sulfur fuels and the use of oxidizing catalysts that cause conversion of fuel-bound sulfur to sulfuric acid aerosol.[15,27,28,167,220] More quantitative data on aerosol formation in sulfur dioxide-NO_x-hydrocarbon systems at atmospheric concentrations are necessary to estimate whether significant reduction in sulfate concentrations can be achieved through the control of oxidants.

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4

Atmospheric Concentrations of Photochemical Oxidants

The main purpose of this chapter is to survey atmospheric concentrations of photochemical oxidants, with emphasis on surface concentrations and the distribution patterns associated with them. The reason for that emphasis is that the photochemical oxidants that affect public health and welfare are largely concentrated in this region. The whole subject of stratospheric ozone (and its filtering of ultraviolet light and interactions with supersonic-transport exhaust products), nuclear weapon reaction products, and halogenated hydrocarbon decomposition products is not treated here.

As in *Air Quality Criteria for Photochemical Oxidants*,[59] our concern will be with the broad subject of oxidant pollution in the atmosphere and the net oxidizing ability of the contaminants in an air sample. The standard corrections or adjustments to remove interferences from the data have already been implied in the primary references in most cases. Therefore, we need not refer to the early distinction between "oxidant" or "total oxidants" and "corrected or adjusted oxidant."

It should be noted that there are still unresolved discrepancies in oxidant data owing to differences in primary standards. Iodometric calibration techniques for ozone monitors were compared by an *ad hoc* committee appointed by the California Air Resources Board (CARB).[14] The committee set out to find an accurate method for measuring ozone, to relate the recommended method to earlier data, and to recommend

procedures for calibrating ozone monitors in the field. It examined calibration techniques used by the U.S. Environmental Protection Agency (EPA), the CARB, and the Los Angeles County Air Pollution Control District and concluded that the Los Angeles County method reads low by about 4% and that the CARB and EPA methods read high by about 25–30%. An ultraviolet procedure was used as an absolute standard, and a commercially available ultraviolet ozone monitor was calibrated to serve as a secondary standard. The committee stated that multiplication of previous CARB ozone measurements by 0.78 and of previous Los Angeles County Air Pollution Control District ozone measurements by 1.04 would yield results accurate within 10%. (A seemingly disproportionate amount of attention must still be given to measurements available from California, because that data base continues to be the largest available.)

Ozone measurement as an indicator of the concentration of oxidants in general is widely accepted. This is not to presuppose that ozone is the cause of all oxidant-related health effects or damage, but that it serves as a surrogate compound for a complex mixture of substances that are characterized by their oxidizing ability and by the effects attributable to this property. Consequently, oxidized organic compounds like aldehydes and organic nitrates are also in the category of our subject matter. Implicit in the belief that ozone is an indicator compound is the assumption that the concentrations, or at least the adverse effects of concentrations, of nonozone oxidants bear some relationship to the ozone concentration. This assumption is a measure of the inability to analyze for the other compounds and of the sparse medical knowledge of their effects on human beings. The use of ozone as a surrogate should be carefully reevaluated.

Measurements of natural-background photochemical oxidants are extremely difficult to find in the literature. Measurements that once were thought to represent the truly rural background are now suspected of having been contaminated by pollutants of man-made origin. This constitutes a critical question in the interpretation of atmospheric concentration, because some of the ozone that is present is controllable and some is not. Some of the literature cited in this chapter contains assertions that it will be impossible in some urban areas to achieve the national ambient air quality standard by local-source reductions, because "natural-background" concentrations already exceed the standard. If these are not in fact natural-background concentrations, they may be reducible by limiting emission of chiefly man-made precursor pollutants in upwind areas. Because the measurements are incomplete, no specific section in this chapter is devoted to natural-background concentrations of ozone; however, measurements that are available are cited and interpreted.

It is currently advocated that atmospheric concentrations of air pol-

lutants be expressed in micrograms per cubic meter. For ozone, the factor for converting from parts per million to micrograms per cubic meter is 1,962 for 25° C and 760 mm Hg; that is, 1 ppm = 1,962 $\mu g/m^3$. In this chapter, conversions are not always made, because of uncertainties as to the temperature and pressure at which readings were taken. These uncertainties become especially severe under conditions of high altitude. In this context, parts per million is on a molar basis.

This review begins with a summary of the sources of monitoring data operated primarily by public agencies. The spatial and temporal patterns of oxidant concentrations are then discussed—urban versus rural and indoor versus outdoor relationships, diurnal and seasonal patterns, and long-term trends. The chapter includes brief discussions of photochemical oxidants other than ozone and of data quality and concludes with a set of recommendations for guidelines in future monitoring of atmospheric concentrations of ozone and other photochemical oxidants.

SOURCES OF MONITORING DATA

One of the earliest organized efforts to acquire data on photochemical oxidants was that of the Los Angeles County Air Pollution Control District, which began in the middle 1950's and has produced the largest data base now available for these studies. In 1961, the California Department of Public Health set up a 16-station Statewide Cooperative Air Monitoring Network (SCAN).

The national network of air pollution measurements is keyed to the 247 air quality control regions (AQCR's), which were classified according to the relative severity of their pollution problems.[62] The classification is a ranking of measured ambient air concentrations or the estimated air quality in the area of maximal severity. The priorities for air quality problem severity are as follows:

Priority I: Ambient concentrations significantly above primary standards.

Priority I*a*: Ambient concentrations significantly above primary standards and due to emission from point sources.

Priority II: Ambient concentrations significantly above secondary standards.

Priority III: Ambient concentrations below secondary standards.

For photochemical oxidants, there were 55 AQCR's in the Priority I

classification and 192 AQCR's in the Priority III classification in October 1973.[60] This indicates how many stations showed a severe oxidant air pollution problem.

A 1973 report issued by the EPA summarized the history of the federal air quality program by presenting a comprehensive analysis and interpretation of data collected from federal, state, and local air quality surveillance activities.[61] Because of data-reduction lags, the report carried information up to 1971, when 183 continuous oxidant monitoring stations were in operation. It proposed 458 monitoring stations for 1974, compared with the 208 legally required. Statistics presented indicate that 9 AQCR's were exceeding the primary oxidant standard in 1969; the number grew to 12 in 1970 and to 15 in 1971. Under the federal regulations, the states submit to the EPA on a quarterly basis all the air quality data obtained from their monitoring systems. The regional EPA offices edit the data for inconsistencies and errors and then forward them for inclusion in the National Aerometric Data Bank (NADB).

Of the six federal monitoring programs in operation when the report was written, two (the National Air Surveillance Networks, NASN, and the Continuous Air Monitoring Program, CAMP) were analyzed for trends. The NASN monitors total suspended particulate matter and sulfur dioxide at over 200 stations. Nitrogen dioxide is monitored at some sites. Oxidant data obtained on a national basis are available from the CAMP, which has been operating in six major urban areas for more than 10 years. Data from the EPA's air monitoring networks described above and from networks operated by state and local governments were submitted for storage in EPA's Storage and Retrieval of Aerometric Data (SAROAD) format. Significant quantities of the information from the states were not expected to be transmitted until the summer of 1973, so the report had only limited information in this category. Extensive tabulations of air quality data and of the monitoring installations are provided.

Other data in this chapter are from special monitoring programs and from scattered reports from networks operated by public agencies. The special programs generally are experimental and are designed to elucidate specific features of the oxidant problem. The scattered data from public monitoring networks have been obtained from a variety of international sources.

A DAMAGE INDEX FOR OXIDANT IMPACT

The health effects of photochemical oxidant pollution should be measured by some kind of aggregate index that involves a weighting of pol-

lutant concentrations according to spatial and temporal distribution. The following sections therefore examine typical values of oxidant concentration as they depend on location and time. The national ambient air quality standard concerns itself simply with the worst place at the worst time. However, in the future, damage associated with each specific effect should be incorporated in the form of a summation or in an integral, such as

$$D_{ij} = \int\int \rho_j(x, t)\Phi_{ij}(c_i)\, d\sigma dt, \tag{4-1}$$

where D_{ij} = index of damage due to the action of the ith species on the jth type of receptor,
ρ_j = population density of jth receptor,
Φ_{ij} = impact function for ith species on jth receptor,
c_i = concentration of ith species,
x = location,
t = time, and
σ = area.

This objective index goes beyond the simpler requirements now imposed by ambient air standards; however, it can be related to ambient air standards by normalizing the impact function. For example, suppose that the impact of pollutant i on receptor j increases as the nth power of c_i, so that the expression

$$\Phi_{ij} = (c_i/c_{is})^n \tag{4-2}$$

permits a comparative evaluation of all species if c_{is} is the ambient air standard. Contributions can be aggregated to state the total damage on receptor j by summing all the D_{ij} over all i. These interactions of time and space patterns with long-term health effects are discussed further in Chapter 10. The application of this index[20] to the analysis of entire systems requires the spatial and temporal distribution of pollutants through the c_i, which implicitly depends on these variables. The sections that follow consider some of the distributions that might enter this type of damage formula.

SPATIAL PATTERNS OF OXIDANT CONCENTRATION

The early siting of monitoring stations in central business districts overlooked the fact that nitric oxide emitted by primary polluters reacts very rapidly with ozone to cause localized decreases in ozone concentration.

Consequently, the ozone concentration is often higher in suburban and even rural areas than in urban areas. Before we examine detailed patterns within a given urban area, it is important to look at the degrees of pollution typically found in cities throughout the United States and elsewhere.

The original criteria document for photochemical oxidants[1] included tabulations of maximal oxidant concentrations and cumulative frequency distributions of hourly average oxidant concentrations. Tables 4-1 and 4-2 show the results. The ranking of stations by yearly average concentration differs from the ranking that might come about from using peak concentrations only. This is borne out by the cumulative frequency distribution: at the high-frequency end, there is very little differentiation among the cities in Table 4-2; but for the rare event, the cities are widely separated in concentration values. This must reflect the differences in mechanisms for forming the ozone that is largely responsible for the oxidant readings. This distinction may be drawn between the nonurban ozone formation processes leading to low concentrations and the urban photooxidation processes leading to the high concentrations of low frequency.

An international expert panel[40] has issued an air quality criteria document for photochemical oxidants and related hydrocarbons that builds on the U.S. Department of Health, Education, and Welfare (DHEW) air quality criteria document for photochemical oxidants. It discusses oxidant concentration patterns in the context of the same tabular material presented earlier. New information is added for the city of Delft:* the monthly means of daily maximums of hourly ozone concentrations are shown in Table 4-3, and the monthly average ozone concentrations are shown in Table 4-4. As in other cities, the worst month seems to be August, with a mean daily maximum (of hourly concentrations) of 0.071 ppm (140 $\mu g/m^3$). Table 4-5 compares the number of days in May through July 1971 when the ozone concentration at one or more sites reached or exceeded the hourly average of 200 $\mu g/m^3$ or of 100 $\mu g/m^3$ (from NATO data[40]). A comparison is made between Delft and five other monitoring sites in the Netherlands. Amsterdam had a peak value of 0.18 ppm in March 1971.

Measurements from a downtown site in Frankfurt-am-Main and another on a nearby 800-m mountain, Kleiner Feldberg, both in the Federal Republic of Germany, are plotted in Figure 4-1. As opposed to the pattern in New York State,[11,56] the ozone concentration at the urban

*The Delft data were provided to the NATO/CCMS Panel on Air Quality Criteria by L. J. Brasser of the Research Institute for Public Health Engineering, Delft, The Netherlands, in April 1973.

TABLE 4-1 Summary of Maximal Oxidant Concentrations in Selected Cities, 1964-1967[a]

Station	Total Days with Available Data	Total Days with Maximal Hourly Average ≥ Concentration Specified						Maximal Hourly Average, ppm	Peak Concentration, ppm
		0.15 ppm		0.10 ppm		0.05 ppm			
		No. Days	% of Days	No. Days	% of Days	No. Days	% of Days		
Pasadena, California	728	299	41.1	401	55.1	546	75.0	0.46	0.67
Los Angeles, California	730	220	30.1	354	48.5	540	74.0	0.58	0.65
San Diego, California	623	35	5.6	130	20.9	440	70.6	0.38	0.46
Denver, Colorado[b]	285	14	4.9	51	17.9	226	79.3	0.25	0.31
St. Louis, Missouri	582	14	2.4	59	10.1	362	62.2	0.35	0.85
Philadelphia, Pennsylvania	556	13	2.3	60	10.9	233	41.9	0.21	0.25
Sacramento, California	711	16	2.3	104	14.6	443	62.3	0.26	0.45
Cincinnati, Ohio	613	10	1.6	55	9.0	319	52.0	0.26	0.32
Santa Barbara, California	723	11	1.5	76	10.5	510	70.5	0.25	0.28
Washington, D.C.	577	7	1.2	65	11.3	313	54.2	0.21	0.24
San Francisco, California	647	6	0.9	29	4.5	185	28.6	0.18	0.22
Chicago, Illinois	530	0	0	24	4.5	269	50.8	0.13	0.19

[a] Derived from U.S. DHEW.[59]

[b] Eleven months of data beginning February 1965.

TABLE 4-2 Cumulative Frequency Distribution of Hourly Average Oxidant Concentrations in Selected Cities, 1964–1965[a]

City	Percent of Hours with Concentrations ≥ Stated Concentrations, ppm								1964–1965 Yearly Average, ppm
	90	70	50	30	10	5	2	1	
Pasadena, California	0.01	0.01	0.02	0.04	0.12	0.18	0.23	0.26	0.042
Los Angeles, California	0.01	0.01	0.02	0.04	0.10	0.14	0.18	0.22	0.036
San Diego, California	0.01	0.02	0.03	0.04	0.08	0.10	0.12	0.14	0.036
Denver, Colorado[b]	0.01	0.02	0.03	0.04	0.06	0.08	0.10	0.12	0.036
St. Louis, Missouri	0.01	0.02	0.03	0.04	0.06	0.07	0.09	0.11	0.031
Philadelphia, Pennsylvania	0.01	0.02	0.02	0.03	0.06	0.08	0.11	0.14	0.026
Sacramento, California	0.01	0.01	0.02	0.04	0.06	0.08	0.10	0.12	0.030
Cincinnati, Ohio	0.01	0.02	0.02	0.04	0.06	0.07	0.08	0.10	0.030
Santa Barbara, California	0.02	0.02	0.03	0.04	0.06	0.08	0.09	0.10	0.036
Washington, D.C.	0.01	0.01	0.02	0.03	0.06	0.07	0.09	0.10	0.029
San Francisco, California	0.01	0.01	0.02	0.03	0.04	0.05	0.06	0.07	0.019
Chicago, Illinois	0.01	0.01	0.02	0.03	0.05	0.06	0.08	0.08	0.028

[a] Derived from U.S. DHEW.[59]
[b] Eleven months of data beginning February 1965.

TABLE 4-3 Mean Daily Maximum of Hourly Ozone Concentrations in Delft, 1970[a]

Month	Ozone Concentration	
	$\mu g/m^3$	ppm
January	4	0.002
February	23	0.012
March	90	0.046
April	104	0.053
May	116	0.059
June	121	0.062
July	88	0.045
August	140	0.071
September	113	0.058
October	58	0.030
November	46	0.023
December	29	0.015

[a] Reprinted with permission of NATO.[40]

TABLE 4-4 Monthly Averages of Ozone Concentrations in Delft, 1970[a]

Month	Ozone Concentration	
	$\mu g/m^3$	ppm
January	2	0.001
February	13	0.007
March	50	0.026
April	58	0.030
May	48	0.024
June	45	0.023
July	46	0.023
August	61	0.031
September	50	0.026
October	32	0.016
November	25	0.013
December	10	0.005

[a] Reprinted with permission of NATO.[40]

TABLE 4-5 Days When Ozone Concentration in Delft Reached or Exceeded 200 $\mu g/m^3$, 1969–1971[a]

1969, day-mo	Maximal Hourly Average Ozone Concentration, $\mu g/m^3$	1970, day-mo	Maximal Hourly Average Ozone Concentration, $\mu g/m^3$	1971, day-mo	Maximal Hourly Average Ozone Concentration, $\mu g/m^3$
9-4	310	10-3	190	15-4	248
5-5	395 (440)[b]	2-5	190	4-5	200
15-6	306	14-5	287	6-5	191
16-6	285	20-6	218	11-5	214
1-7	205	21-6	313	31-5	275
16-7	370	30-7	198	1-7	259
23-7	196	1-8	190	5-7	192
4-8	191	13-8	230	8-7	208
9-9	192	29-8	217	9-7	226
13-9	204	30-8	227	11-7	239
		31-8	190	23-7	193
		18-9	237	30-7	249
		19-9	247	19-8	249
		20-9	237	21-8	245
		29-9	211	22-8	244
				29-9	186
				2-10	228
				3-10	364 (>410)[b]

[a] Derived from NATO data.[40]
[b] Maximal concentration measured.
In 1969, 340 hourly averages were >100 $\mu g/m^3$ and 22 hourly averages were >200 $\mu g/m^3$.
In 1970, 550 hourly averages were >100 $\mu g/m^3$ and 30 hourly averages were >200 $\mu g/m^3$.
In 1971, 845 hourly averages were >100 $\mu g/m^3$ and 48 hourly averages were >200 $\mu g/m^3$.
In 1972, 236 hourly averages were >100 $\mu g/m^3$ and 9 hourly averages were >200 $\mu g/m^3$.

sampling site was far greater than that observed at the mountain site. The peak in Frankfurt lasts from 10 a.m. to 6 p.m. Readings from two stations in Berlin are compared in Figure 4-2. One station, Steglitz, is in an area with high traffic; the other, Dahlem, is in the suburbs. It was concluded that the lower oxidant concentrations at the urban station were due to the high concentrations of hydrocarbon there. In reality, they were probably caused by the high concentration of nitric oxide that reacted with the ozone. For both cases, the concentrations of ozone were extremely low, with a maximum of 0.0132 ppm at Dahlem.

The NATO report also supplied ozone information for Amsterdam, University City in Rome, and Ankara, Turkey. These data are shown in Figures 4-3, 4-4, and 4-5.

An air pollution episode in Windsor, Ontario, was recorded on August

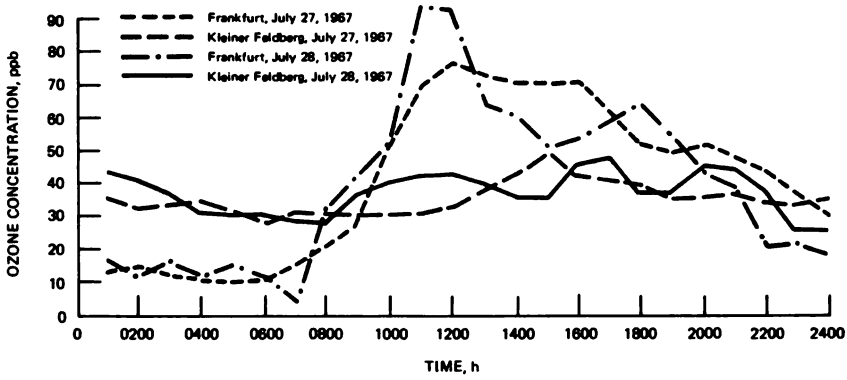


FIGURE 4-1 Diurnal variation in ozone concentration in Frankfurt-am-Main and at Kleiner Feldberg mountain station on July 27 and 28, 1967. Reprinted with permission from NATO.[40]

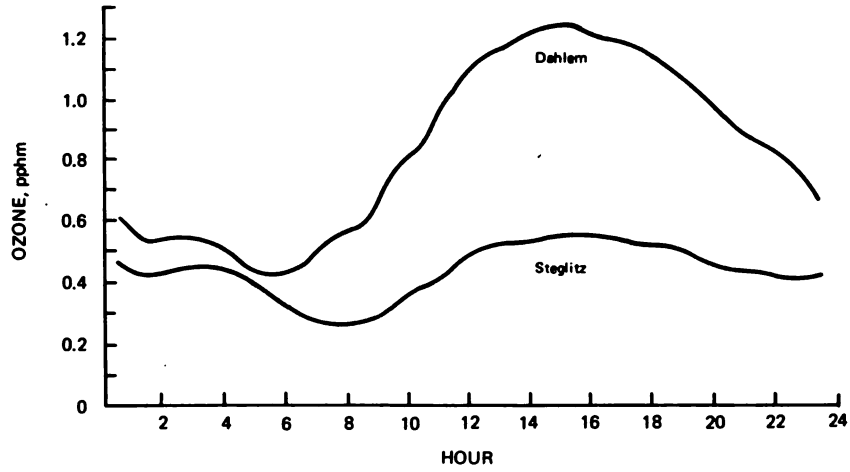


FIGURE 4-2 Diurnal variation in ozone concentration at two sites in Berlin, 1966–1967. Reprinted with permission from NATO.[40]

17–19, 1971.[17] Figure 4-6 shows total-oxidant peaks ranging from 0.12 to 0.20 ppm and comparable peaks for the oxides of nitrogen. An interesting feature of these measurements is the relationship between oxidants and hydrocarbon. This interpretation must not be taken too seriously, because of the large quantity of relatively unreactive material in the total-hydrocarbon reading. Figure 4-7 refers to a pollution episode at

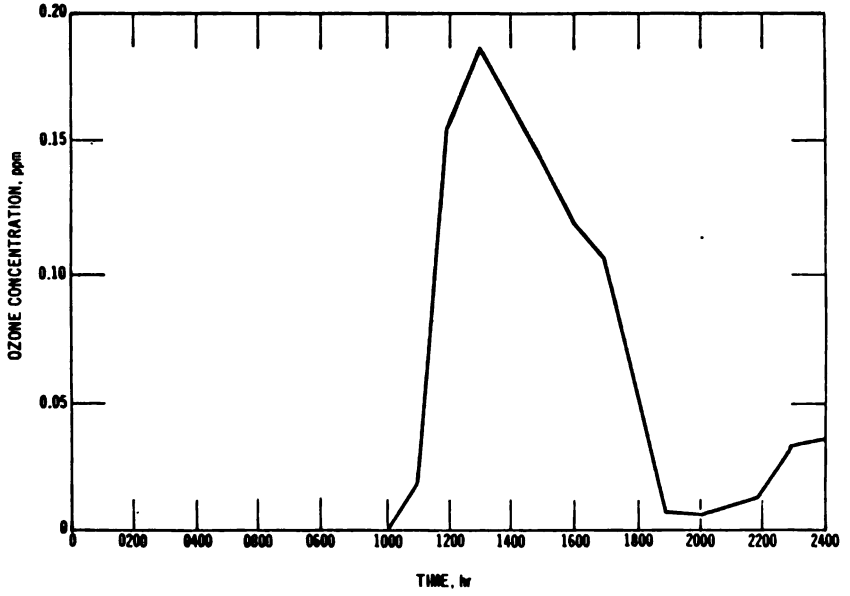


FIGURE 4-3 Ozone concentrations in Amsterdam, March 10, 1971. Reprinted with permission from NATO.[40]

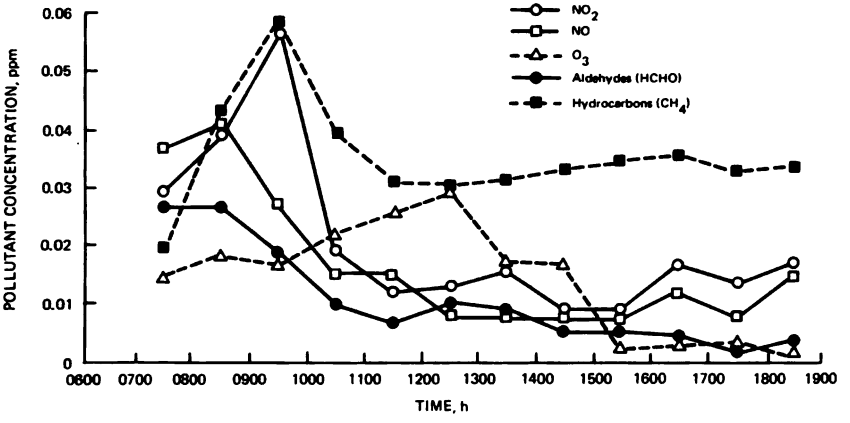


FIGURE 4-4 Nitrogen dioxide, nitric oxide, ozone, aldehyde, and hydrocarbon concentrations in University City, Rome, Italy. Reprinted with permission from NATO.[40]

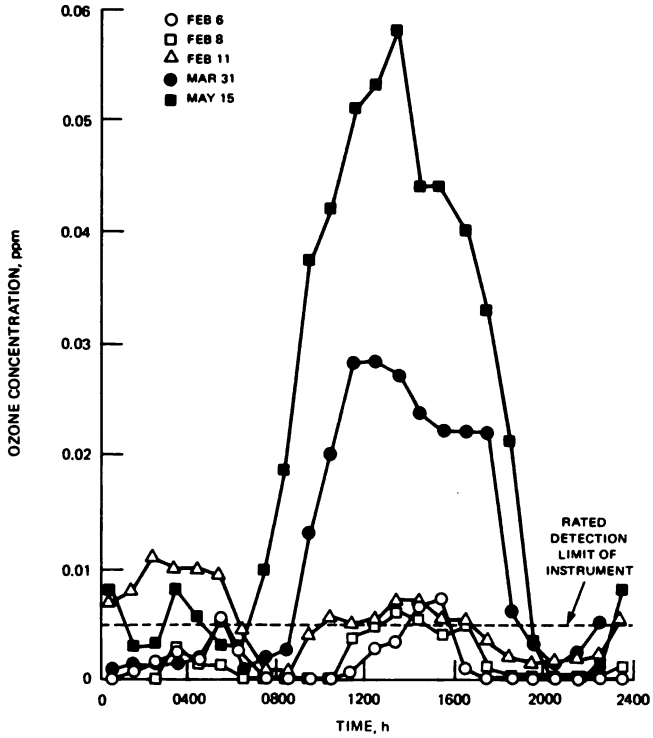


FIGURE 4-5 Diurnal variation in ozone concentration in Ankara, Turkey, on selected days of 1972. Reprinted with permission from NATO.[40]

Tunney's Pasture in Ottawa in 1973, in which oxidant concentrations approached 0.14 ppm. This episode took place on a July weekend, and the nitrogen dioxide and NO_x concentrations appear to be very small. Nonmethane hydrocarbons, however, were relatively high, about 0.6 ppm.

Bilger has documented ozone and other oxidant measurements in Australia[4] and compared them with those in other cities. Table 4-6 shows the portion of hours during which threshold concentrations were exceeded in Sydney in 1971, 1972, and 1973. Low concentrations were recorded before 1970, probably because of the proximity to nitric oxide emission sources in the central portion of the city. It is also noteworthy that, despite the calm conditions and strong inversion in the winter months (in the Southern Hemisphere), high-oxidant days were relatively infrequent. Figure 4-8 outlines this seasonal variation in oxidant concentration. In Melbourne, Australia, however, a high-ozone episode was observed during

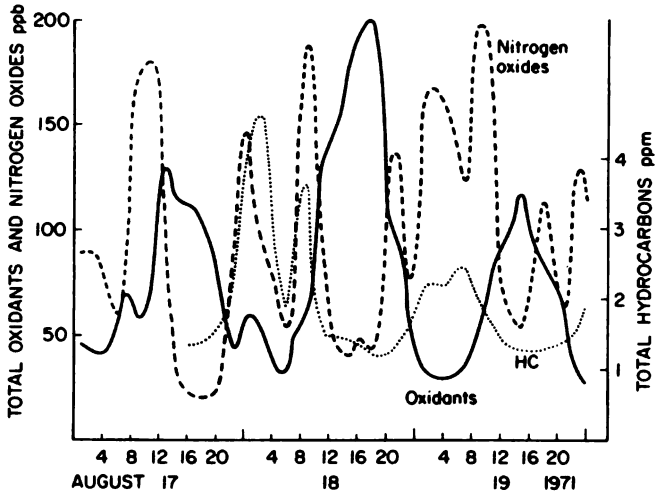


FIGURE 4-6 Oxidant, nitrogen oxide, and hydrocarbon concentrations in pollution episode in Windsor, Ontario. Redrawn from Dickson and Quickert.[17]

December 1973. The diurnal concentration variations shown in Figure 4-9 illustrate the episode between December 10 and December 13, with a peak of 0.28 ppm on December 11, the second day of the episode. Relatively low concentrations were observed in other cities.

In addition to the analysis of oxidant problems in the United States and Australia, the Organization for Economic Cooperation and Development's Air Management Sector Group[41] has compiled oxidant concentrations measured in Japan during 1970–1972. Table 4-7 summarizes maximal hourly oxidant concentrations and the frequencies of concentrations of 0.15 ppm or more. The figures show that, despite the high latitude, rather large maximal hourly concentrations are observed in several of the cities, with the highest being 0.38 ppm in Tokyo in 1972, during which year the oxidant concentration reached or exceeded 0.15 ppm on 25 days.

Another high-latitude area where substantial ozone concentrations have been observed is London. Derwent and Steward[15] reported ozone concentrations in excess of 0.1 ppm. These were recorded at Harwell, Brookshire, on July 3 and July 7, 1971, by Atkins *et al.*[3] The main subject of Derwent and Steward's paper, however, deals with measurements in central London. Figure 4-10 (from Derwent and Steward[15]) shows the time variation of photochemically active pollutants for 3 days in central

London. On the second day, the late afternoon peak exceeded 0.1 ppm. It is apparent that some conversion of nitric oxide to nitrogen dioxide was responsible for the ozone buildups, but it is not as clear a chemical pattern for London as it is for cities in the western United States. The paper did not mention the method of ozone measurement; however, qualitative descriptions of the weather patterns suggest that the days of high ozone were characterized by light winds and considerable sunlight.

Coupled closely with the effect causing horizontal distributions are the vertical distributions of ozone concentrations. These distributions have an intimate influence on the urban-rural interchange of ozone. Miller and Ahrens[35] presented detailed vertical time and space cross sections of ozone concentrations at altitudes up to 2,500 m. A low-altitude temperature inversion may actually lead to lower concentrations of oxidant, because the destruction rate can be increased by the injection of nitric

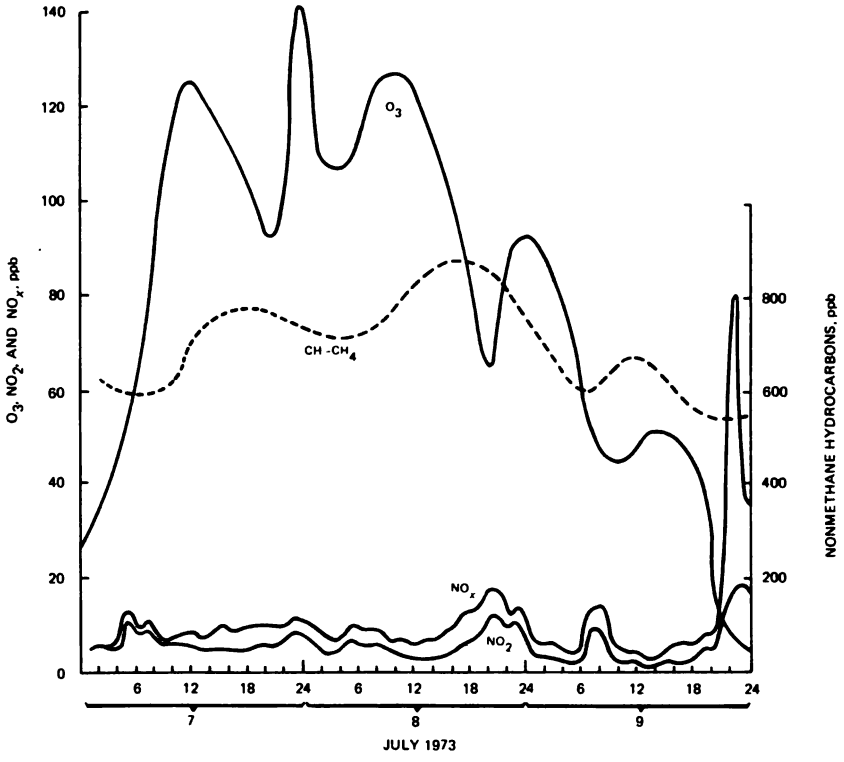


FIGURE 4-7 Ozone, nitrogen dioxide, NO_x, and nonmethane hydrocarbon concentrations in pollution episode at Tunney's Pasture, Ontario. Redrawn from Quickert *et al.*[44]

TABLE 4-6 Oxidant Concentrations in Sydney[a]

Year	Portion of Hours with Concentration, %			Annual Maximal 1-h Average, ppm
	>0.05 ppm	>0.10 ppm	>0.15 ppm	
1971	4.1	0.2	0.01	0.17
1972	5.6	0.9	0.2	0.24
1973	6.1	0.9	0.3	0.28

[a] Derived from Bilger.[4]

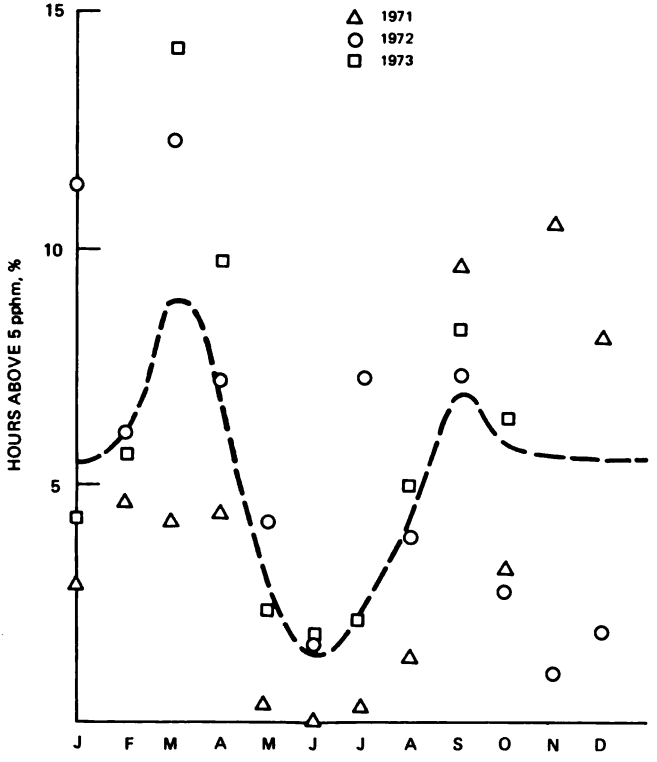


FIGURE 4-8 Seasonal variations in oxidant concentration in Sydney, Australia. Redrawn from Bilger.[4]

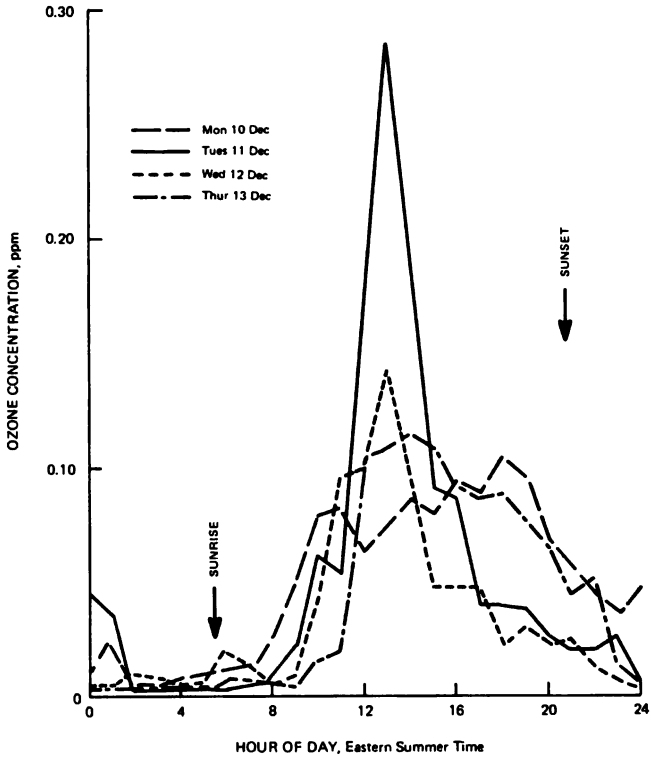


FIGURE 4-9 Diurnal variation in ozone concentration during December 1973 at Parliament Place, Melbourne, Australia—a 3-min average on the hour. Redrawn from Bilger.[4]

oxide into a shallow mixing layer. Very high oxidant concentrations were observed at the edge of the marine inversion, where the mixing layer is rather deep. Possible high-altitude transport of ozone within the inversion layer and absence of destruction mechanisms could lead to a buildup aloft. Later fumigation caused by surface heating could cause increased ozone concentrations at downwind locations. Horizontal spatial patterns were mapped out with aircraft measurements to indicate the connection between transport from areas where precursor emission occurs and the downwind buildup of ozone after the photochemical reaction.

The compensating factors leading to increased daily maximal oxidant concentration over the years include both decreased destruction rates and increased precursor concentration. The decreased destruction rates

TABLE 4-7 Oxidant Concentrations in Japan, 1969-1972[a]

City	Maximal Hourly Oxidant Concentration, ppm			
	1969	1970	1971	1972
Tokyo	0.27	0.34	0.29	0.38
Osaka	0.26	0.24	0.24	0.29
Kanagawa	—	0.21	0.36	0.33
Chiba	—	—	0.25	0.32
Saitama	—	—	0.32	0.28
Aichi	—	—	0.165	0.20
Hyogo	—	—	0.21	0.24

No. Days in 1972 with Oxidant Concentration ≥0.15 ppm	
Tokyo district:	av. 25
Osaka district:	av. 11
Ise district:	av. 5
Ibaragi:	16
Okayama:	3
Ehime:	3

[a]Derived from OECD.[41]

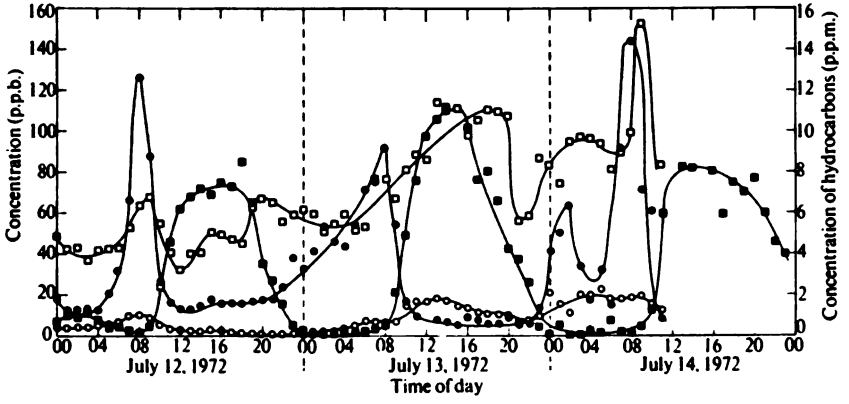


FIGURE 4-10 Diurnal variations of air pollutants measured in London from July 12 to July 14, 1972. ■, Ozone ppb; ●, nitric oxide, ppb; □, nitrogen dioxide, ppb; ○, hydrocarbons, ppm. Reprinted with permission from Derwent and Steward.[15]

theoretically result from urbanization, which tends to destabilize the air and increase vertical mixing. Thus, the very mechanism that leads to greater dispersion also leads to higher ozone concentration. The ozone aloft that comes from polluted air that is transported upward into the inversion has a relatively long lifetime, compared with that at low altitudes.

To determine the relationship of ozone concentrations in the Sierra Nevada mountains with those in the central valley of California, airborne and ground-station measurements were taken of total oxidant, temperatures, and wind from August 17 to August 27, 1970. Miller *et al.*[36] reported the results in a paper that coordinated the measurements on the basis of ozone transport. Figure 4-11 shows concentration equaling or approaching the national ambient air quality standard for oxidants at stations at 351 and 2,287 m. Comparison with concentrations in an urban center in the central valley, with an altitude of 99 m, shows that the peak concentrations at these very different elevations roughly approximate one another during the afternoon and early evening. At the urban location, however, nighttime values decrease dramatically, probably because of reaction with nitric oxide emitted locally. These results suggest that the ozone-laden layer moves along the terrain, rather than touching the surface at some altitude near the mixing depth. A highly plausible transport mechanism is found in the afternoon upslope flows induced by surface heating. Aircraft profile measurements at Mineral King (2,287 m) indicated that both temperature and oxidant increase slightly near the surface. The evidence suggests that photochemical oxidant is transported from population centers in the central valley into the higher-altitude

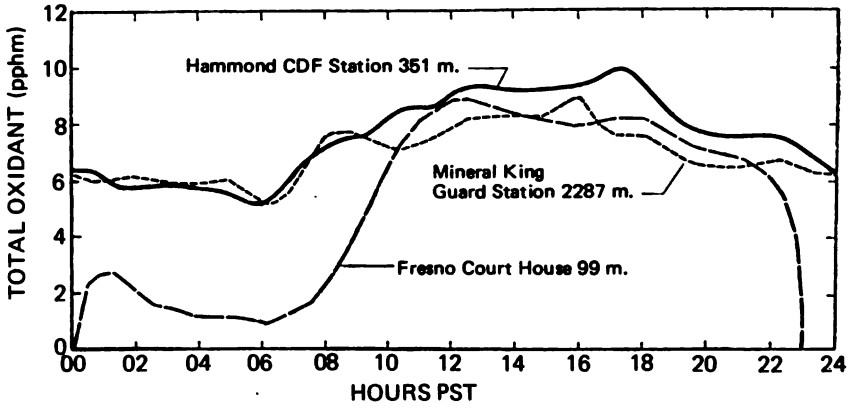


FIGURE 4-11 Comparison of 10-day means of hourly total oxidant concentrations at Fresno, Hammond, and Mineral King, California. Reprinted with permission from Miller *et al.*[36]

mountain valleys. The primary pollutants (oxides of nitrogen and hydrocarbons) may have been the specific pollutants advected (i.e., moved horizontally), and the abundant light in the ultraviolet range at the higher altitudes could have been significant in increasing ozone production during the early morning.

Blumenthal *et al.*[6] obtained aircraft data by taking vertical soundings over various airports in the Los Angeles basin area. Figure 4-12 shows the results for a vertical sounding over Hawthorne Airport at 9:15 a.m. on September 20, 1972. The vertical distributions of condensation nuclei and temperature suggest a mixing layer approximately 800 ft (244 m) deep. At the same altitude, there is a decided increase in ozone concentration that persists up to a few thousand feet in altitude. This morning profile of ozone up into the inversion layer corroborates other findings of high ozone concentrations in regions that are not well mixed with nitric oxide from the ground. The profiles of carbon monoxide concentration and b_{scat} (a measurement of light scattering in air) also indicate the location of the mixing layer.

Blumenthal and co-workers[8] used the earlier data and a case study of Denver, Colorado, to develop arguments as to the source of high concentrations of ozone or ozone precursors that are found in some nonurban areas. They believe that downwind areas as long as 260 km can exceed the standard because of precursor emission from an urban source. Although they do not present any ozone production estimates related to photochemistry, their data analyses confirm hypotheses of transport from urban to nonurban areas. They point out that rural areas have had concentrations as high as 0.3 ppm with no local source of reactant.

The Los Angeles Reactive Pollutant Program (LARPP) obtained three-dimensional distributions of ozone by using helicopters and ground-based instruments. The instrument platforms were directed to follow an air parcel through the Los Angeles basin by means of radio commands based on the radar tracking of tetroon arrays that were released below the inversion base and allowed to float along with the air. (A tetroon is a neutral-buoyancy balloon that follows winds along constant density contours.) Preliminary plots of these data[18] show that each vertical distribution of ozone is very nonuniform during the morning, but becomes very uniform toward midday as the values increase. This behavior is shown in Figure 4-13. Vertical profiles of temperature, dew point, and ozone, shown in Figure 4-14, illustrate a case in which the ozone concentration in the inversion layer 1,000–2,000 ft (305–610 m) above mean sea level was approximately three times the concentration near the ground. This finding lends further support to the hypotheses advanced by others regarding the horizontal transport of ozone at high altitudes.

In rural areas of western Maryland and West Virginia, hourly ozone concentrations as high as 0.12 ppm were recorded[46] in August and September 1972. The concentration exceeded the national ambient air quality standard of 0.08 ppm 11% of the time. During one episode, this standard was exceeded for 26 consecutive hours. Precursors were always near background concentrations, and the horizontal spread of ozone was extensive. After mentioning stratospheric sources and ground sinks, the report hypothesizes that clouds of precursors emitted from distant urban areas undergo transformation to ozone during their long journey. Mechanical turbulence is suggested as a transport mechanism that brings the resulting ozone to the surface.

Airborne measurements made for transects across the Los Angeles basin by Edinger[19] confirm the earlier hypotheses that ozone concentrations go through a spatial maximum above the base of the inversion in many situations. Figures 4-15 and 4-16 show space sections of the potential temperature and the oxidant concentrations in a vertical cross section running approximately west to east from the Pacific Coast to Rialto, California. These results are for June 20, 1970, at 4:30 p.m. The temperature field exhibits a distinct inversion base that roughly follows the terrain, but increases slightly moving inland—which is typical of the heating effect that occurs during onshore flows. The oxidant field corresponding to it shows peaks in excess of 0.35 ppm at altitudes approaching 1,000 m. At the same time, ground concentrations gradually increase to a maximum

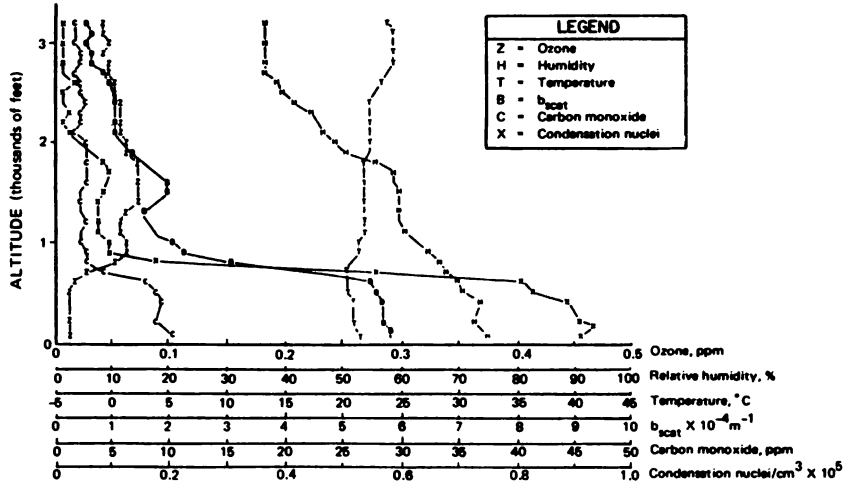


FIGURE 4-12 Data from vertical sounding over Hawthorne Airport at 9:15 a.m., September 20, 1972. Reprinted with permission from Blumenthal *et al.*[6]

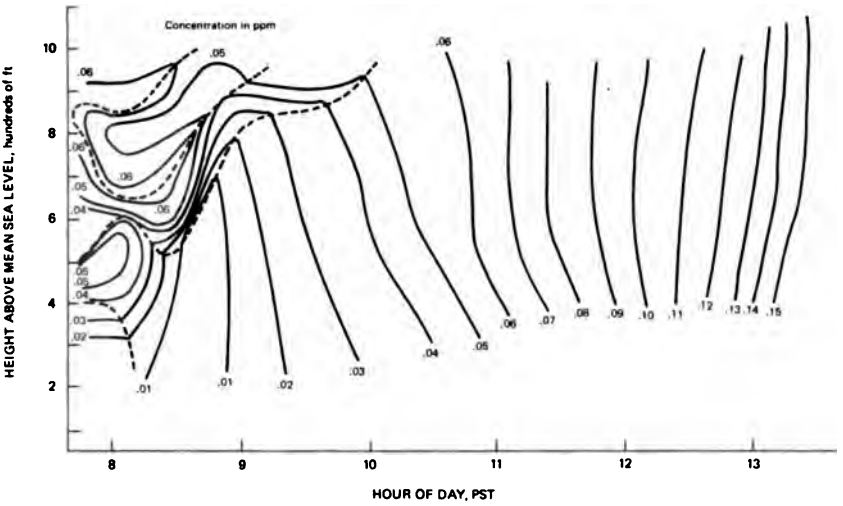


FIGURE 4-13 Vertical time section for ozone, operation 33, November 5, 1973. Redrawn from Edinger.[18]

of 0.25 ppm 60 miles (97 km) onshore. These data suggest that the oxidant within the inversion layer has been advected into that layer from horizontal injections in the vicinity of heated mountain slopes. This explanation is preferred to vertical mixing, because of the extreme stability within the inversion layer. In fact, that stability insulates the high-ozone air parcel from nitric oxide, which could react rapidly with the ozone and thereby decrease it. Entrapment in the inversion layer also suggests an explanation for long-range horizontal transport that can ultimately lead to the large nonurban ozone concentrations that have been reported.

Ozone and ozone precursor concentrations at nonurban locations in the eastern United States were studied extensively.[47] The three parts of the study were field measurements, a quality assurance program, and an airborne monitoring program. The main objective of the study was to establish a data base for nonurban ozone and precursor concentrations. Simultaneous statistical summaries of the concentrations of nitrogen dioxide and nonmethane hydrocarbons were also provided. Another objective was to search for relationships between ozone concentrations and nitrogen dioxide and nonmethane hydrocarbon concentrations.

Monitoring sites were selected in northwestern Pennsylvania, central Ohio, western Maryland, and southern West Virginia. The field program was conducted between June and September 1973. Ozone was measured at each of the four sites, and precursor concentrations were measured at

three of them. The diurnal patterns of ozone concentration were not similar to the rural site measurements reported for New York State.[11,56] Instead of a gentle ozone peak just after midnight, there was a late afternoon peak that was several times the minimal concentration of ozone, which generally occurred early in the morning. On selected dates, however, nighttime ozone concentration maximums were observed at the Maryland and Ohio sites. It was noted that the National Ambient Air Quality Standard for photochemical oxidants was exceeded during 37, 30, 20, and 15% of the hours at the sites in Maryland, Pennsylvania, Ohio, and West Virginia, respectively, during the sampling interval. Correlation coefficients among hourly ozone concentrations measured at the four stations at the same time were in the range of 0.468–0.678. Evidently, high ozone con-

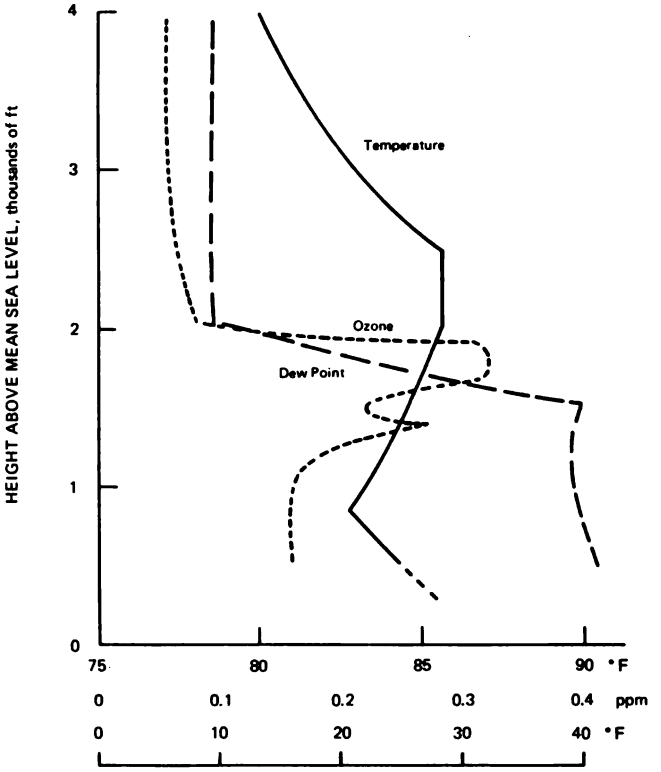


FIGURE 4-14 Vertical temperature, dew point, and ozone sounding for operation 14, Glendale, California, October 4, 1973, 1:01–1:25 p.m. Redrawn from Edinger.[18]

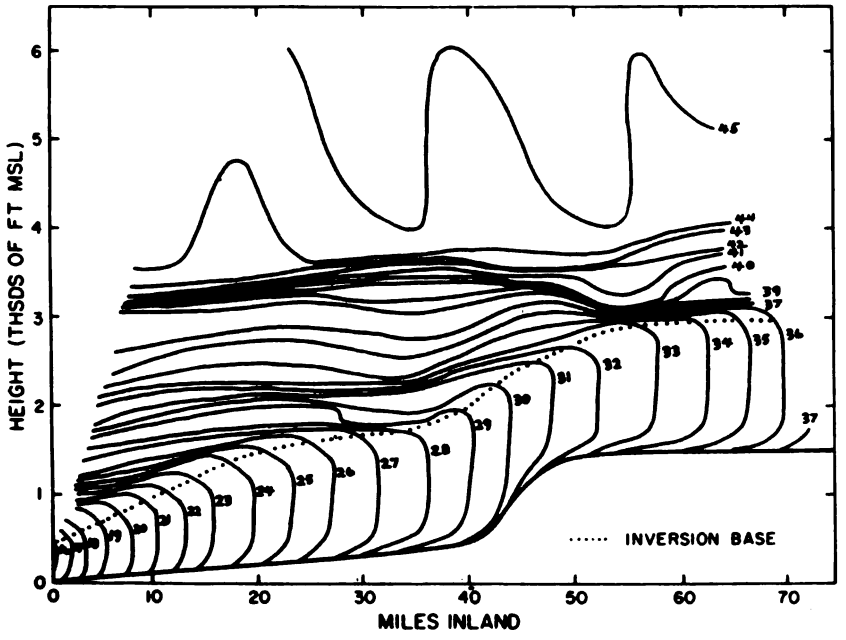


FIGURE 4-15 Field of potential temperature (°C) in the vertical cross section from Santa Monica to Rialto-Miro, California, 4:30 p.m., June 20, 1970. Reprinted with permission from Edinger.[19]

centrations at the nonurban locations were spread over extensive areas of the four-state region.

The quality assurance phase of the program was conducted to maximize the comparability of the data generated at the four monitoring sites, as well as data from the mobile laboratory. Real-time data processing permitted quick analysis of the performance of the analyzers. Because the nitrogen dioxide values were very low and the hydrocarbon data from the fixed sites were declared invalid, only ozone was subjected to statistical analysis. The bias of measurements between fixed- and mobile-site analyzers was estimated. The greatest bias observed during the program, 23%, was attributed to span drift of one of the analyzers.

The airborne monitoring program concentrated on the measurement of ozone to provide supplementary air quality data for various altitudes over the fixed sites. The airborne measurements were conducted in a C-45 aircraft that carried a solid-face chemiluminescent ozone monitor. The ozone meter was cycled every 2 min to provide calibration, purge, measurement, and purge at equal intervals. The sparseness of the airborne data precluded

detailed analysis, but the comparisons between ozone aloft and ozone at the ground stations showed a high degree of regularity. The airborne values were comparable with those read on the ground 100–600 ft (30–183 m) below. A general conclusion on the vertical data is that the ozone generated from ground-source precursors is predominant over ozone that might be transported from the stratosphere.

Gloria *et al.*[25] studied photochemical air pollution with an instrumented aircraft in various air bases in California. They estimated background ozone concentrations for the southern coast air basin to be about 0.03 ppm. Ozone was measured by ultraviolet absorption with a Dasibi ozone photometer. Total oxidant, carbon monoxide, oxides of nitrogen, hydrocarbons, dew point, and temperature were measured simultaneously. East-west ozone sections were made from series of flights following survey patterns mapped on Figure 4-17. Figure 4-18 shows the vertical cross section of ozone isopleths for August 11, 1971, indicating higher

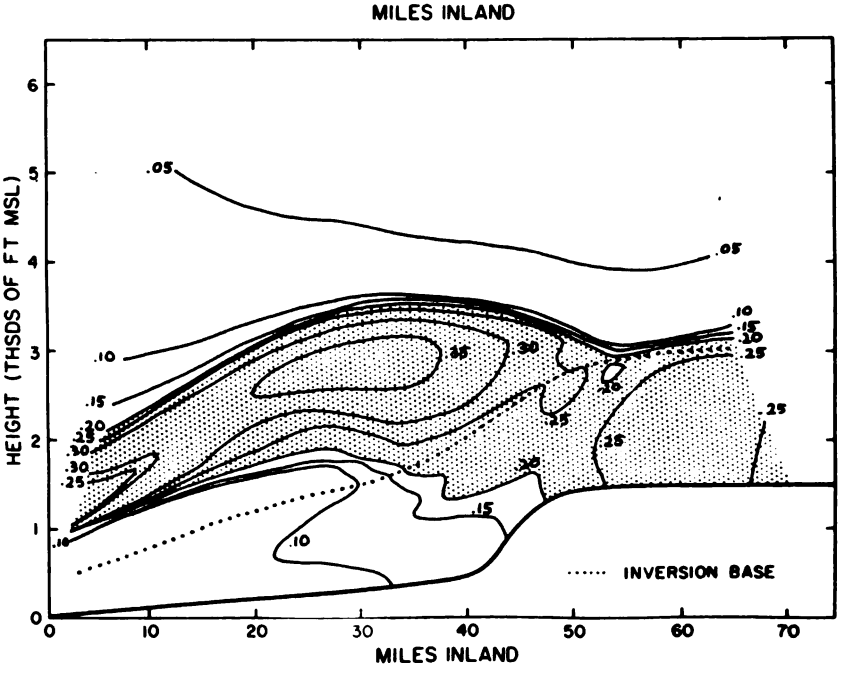


FIGURE 4-16 Field of oxidant concentrations (ppm) in the vertical cross section from Santa Monica to Rialto-Miro, California, 4:30 p.m., June 20, 1970. Reprinted with permission from Edinger.[19]

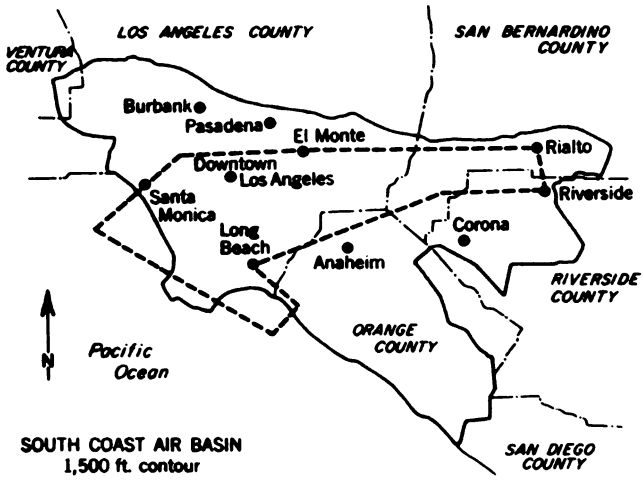


FIGURE 4-17 Outline map of the Los Angeles basin area traversed during survey flights, showing general flight paths. Reprinted with permission from Gloria *et al.*[25]

ozone concentrations next to the surface than up near the inversion base. Layering of ozone at higher altitudes is shown in the profiles measured at Riverside, California, on August 10–12, 1971. These profiles, illustrated in Figure 4-19, show the entrapment of high ozone concentrations far above the surface in strata. Similar ozone layering was observed in the San Francisco Bay area during the same study.

Atmospheric ozone concentration was measured simultaneously at two rural and four urban sites in New York State.[56] It was noted that during the period August 1–17, 1973, the average hourly ozone concentrations at the rural sites stayed well above the urban concentrations throughout each day and that the urban values peaked at approximately the rural values in the early afternoon (see Figure 4-20). The average rural ozone concentration for the period of observations was around 0.05–0.07 ppm. The authors concluded that violations of the present ambient air quality standard for photochemical oxidants may not be prevented completely by reducing anthropogenic hydrocarbon emission in the state. They compared the weight equivalent of 0.24-ppm hydrocarbon and 0.08-ppm ozone. These numbers were derived from the air quality standards and suggested that one weight of nonmethane hydrocarbon is capable of generating an equivalent weight of ozone photochemically. The questionable nature of these assumptions is brought out by a comparison of the ozone potential for all anthropogenic emission of hydrocarbon in the nation

with the influx of ozone by advection into New York State. The two ozone values are approximately equivalent. Clearly, the photochemistry of nitrogen oxides must be included in such estimates. Explanations of the relatively high rural ozone concentrations center on speculation based on the subsidence of stratospheric ozone or on ozone generation by the photooxidation of natural precursors. It is argued that transport of ozone from other urban areas and later reactions with further precursors do not appear to constitute the dominant mechanism. These arguments apparently were based on the assumed weight equivalence between hydrocarbon precursors and ozone in the atmosphere.

The authors later continued the discussion with the same New York State data.[11] There were close correlations between rural ozone concentrations and some of the urban peak values for the August 1-17, 1973, period, as shown in Figure 4-21. Time correspondences between maximal wind and maximal ozone concentration were cited as evidence to support the hypothesis that high urban ozone concentrations in New York State result from the transport of background ozone into these areas. Correlation coefficients of 0.83 for wind and temperature, 0.81 for wind and ozone, and 0.87 for ozone and temperature were obtained for readings in Kingston, New York. The correlations for the Welfare Island monitoring site in New York City were not nearly as clear as the ones for Kingston. It was concluded that the correlation of photochemical ozone in one area with that in another does not necessarily indicate transport. An alternative

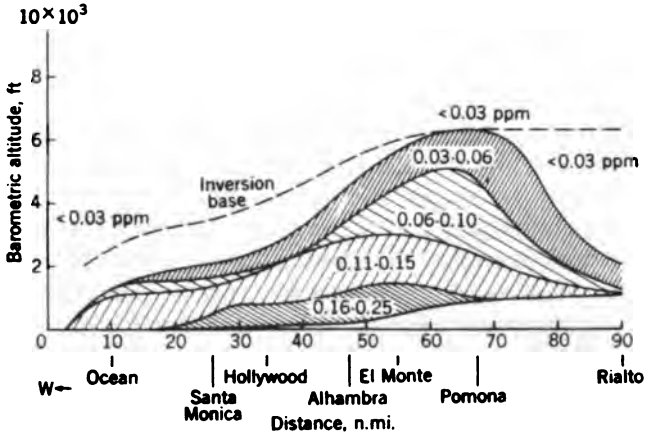


FIGURE 4-18 Ozone isopleths (vertical cross section) from southern California coast air basin survey, August 11, 1971. Reprinted with permission from Gloria *et al.*[25]

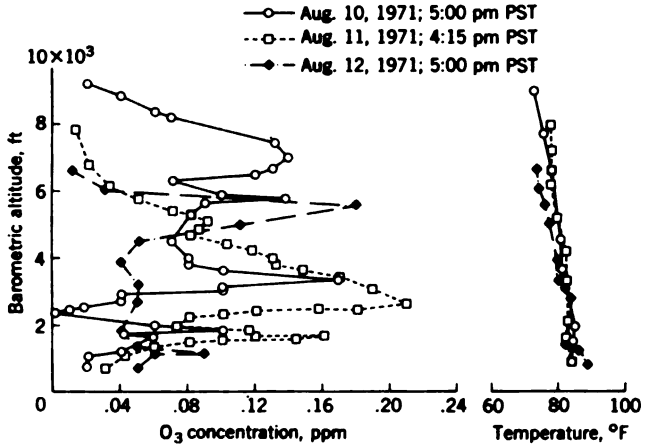


FIGURE 4-19 Ozone profiles, Riverside, California, August 10–12, 1971. Reprinted with permission from Gloria *et al.*[25]

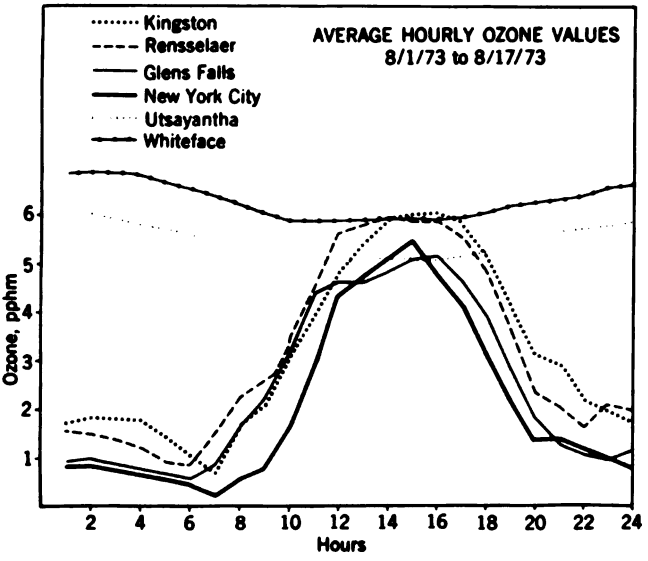


FIGURE 4-20 Average hourly ozone concentrations during August 1–17, 1973, at selected sites in New York State. Reprinted with permission from Stasiuk and Coffey.[56]

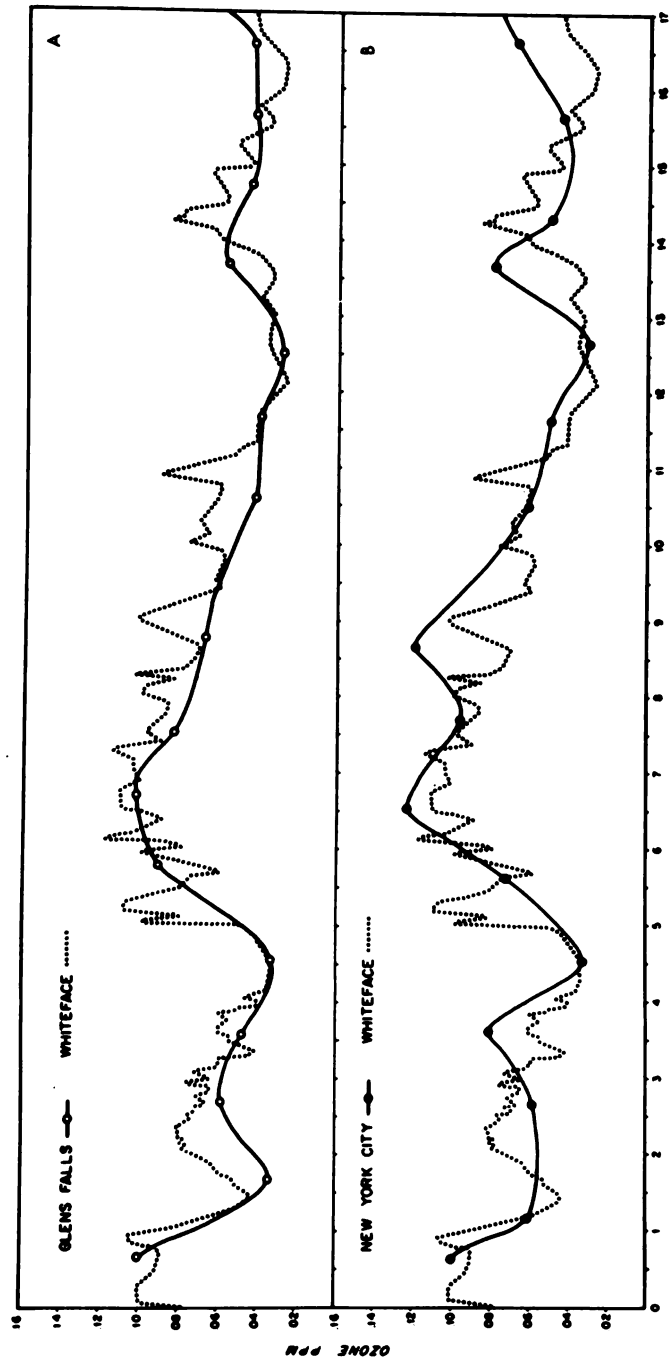


FIGURE 4-21 Close correlations between the Whiteface, New York, ozone concentrations, August 1-17, 1973, and the smooth curve drawn between the Glens Falls and New York City daily ozone maximums. Reprinted with permission from Coffey and Stasiuk.[11]

mechanism was based on a belief that the observed correlations imply a common ozone source. It was suggested that ozone concentrations exceeding the federal ambient air quality standard exist in rural areas and are transported into urban areas, but no direct evidence was offered beyond the concentration graphs.

Air breakdown due to coronal discharges around high-voltage transmission lines has been considered as a possible rural source of ozone. Several investigations[22,23,47,49,55] suggested that this cannot be a significant source.

In a paper presented at the EPA scientific seminar on automotive pollutants,[42] Pitts pointed out that, until the controversy on calibration methods was resolved, most people believed that the urban plume moving eastward from Los Angeles was accompanied by increasing ozone concentrations. This is illustrated in Figure 4-22 for downtown Los Angeles, Pasadena, Pomona, Azusa, Riverside, and San Bernardino. Figures 4-22 through 4-24 all show the numbers of days on which the maximal hourly average oxidant concentration equaled or exceeded 0.20 ppm in 1973. Pitts assumed that all data were adjusted to the Los Angeles Air Pollution Control District (APCD) calibration method by multiplying the non-Los Angeles (i.e., Riverside and San Bernardino) data by 5/7. Figure 4-23 shows that this procedure lowers the numbers for the two easternmost stations relative to those for the eastern portion of Los Angeles County (stations 2, 3, and 4—Pasadena, Pomona, and Azusa). Proceeding to another alternative, Pitts multiplied the numbers for stations 1–4 by 7/5 as a way of converting Los Angeles APCD data to the ARB calibration method. The results, illustrated in Figure 4-24, show the numbers for the two easternmost stations to be lower than those for the stations in Los Angeles County. Without saying which agency is correct in the controversy, Pitts pointed out that a consistent approach with either method produces a conclusion different from the previous one. Previously, it was believed that the eastern counties experienced higher ozone concentrations. Now it appears that the ozone concentration goes through a peak in the eastern portions of Los Angeles County and decreases as the air parcel moves into the counties east of Los Angeles. These results show that extreme care must be taken in comparing ozone or oxidant measurements taken at different places with instruments that are calibrated by different methods.

The production of ozone in power-plant plumes has been suggested to explain ozone spatial distributions in nonurban areas.[12,13] Comparison of oxidation mechanisms competing for sulfur dioxide suggests that three reactions—

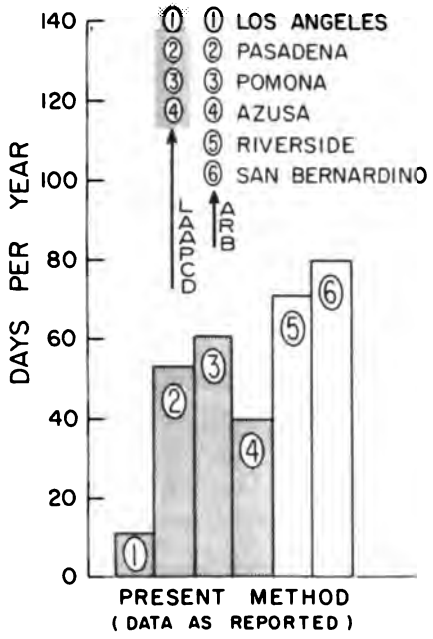


FIGURE 4-22 Number of days in 1973 on which the maximal hourly average oxidant concentration equaled or exceeded 0.20 ppm at six air monitoring stations in the southern California coast air basin. Data as reported: Los Angeles, Pasadena, Pomona, and Azusa reported by Los Angeles County APCD; Riverside reported by Riverside County APCD; and San Bernardino reported by San Bernardino County APCD. Reprinted with permission from Pitts.[42]

$$SO_2 + HO_2 \rightarrow SO_3 + OH \qquad (4\text{-}3)$$

$$SO_2 + OH + M \rightarrow HSO_3 + M \qquad (4\text{-}4)$$

$$SO_2 + CH_3O \rightarrow SO_3 + CH_3O \qquad (4\text{-}5)$$

—are principally responsible for sulfur dioxide removal. Reaction 4-4 is thought to be the first step in a chain that converts nitric oxide to nitrogen dioxide, causing an ozone buildup via the photostationary-state mechanism involving the nitrogen dioxide-nitric oxide-ozone cycle. Oxidized species up to HSO_6 are postulated as being responsible for the conversion.

Descriptions of field studies of power-plant stack plumes were given by Davis *et al.*[13] The ozone concentration appears to be lower in regions of high sulfur dioxide content. At 32 km downwind from the stacks, it was claimed that the ozone concentration in the plume (now 11 km wide) is higher than the ambient concentration ($\sim$0.08 ppm) by approximately 0.02 ppm. Simultaneous measurements of nitric oxide and nitrogen dioxide were integrated across the plume. Values of the ratio of nitric

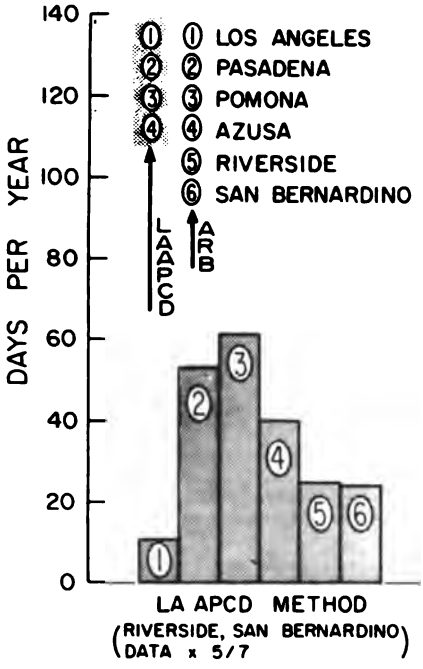


FIGURE 4-23 Number of days in 1973 on which the maximal hourly average oxidant concentration equaled or exceeded 0.20 ppm at six air monitoring stations in the southern California coast air basin. Non-Los Angeles APCD data multiplied by 5/7 to convert to Los Angeles APCD scale. Reprinted with permission from Pitts.[42]

oxide to NO_x concentration decreased monotonically from 0.9 at 2 km to 0.2 to 15 km and beyond. Other, similar observations were cited.

It has been suggested by the analysis of organic components in rainwater that naturally emitted terpene compounds may be the source of some photochemical oxidant.[52] Gas chromatography was used to separate individual components of rainwater that had been purged by helium gas as a carrier. The individual components thus separated were identified by their mass spectra. Rainwater samples were taken at the end of a mid-August 1973 smog episode in the Washington, D.C., area. The episode was characterized by stagnant weather over a large area east of the Appalachian Mountains between New Jersey and North Carolina. It ended on August 13, when a gentle rain occurred and samples were taken. The dominant compound in the rainwater samples was 3-methylfuran. Plausibility arguments based on chemical reaction mechanisms were advanced for the existence of 3-methylfuran as a product of the photooxidation of isoprenoid compounds. Some aromatics in the sample were attributed to vehicular hydrocarbon emission, but the relative concentrations suggested that terpenes could have been the most important compound in the smog. No definite conclusions were drawn, but it

was inferred that naturally occurring hydrocarbons could cause summer photooxidant smog. This is highly unlikely, in view of measurements made on rain that originated from an air mass different from that in which the smog episode occurred.

In addition to observations in Los Angeles, Blumenthal and White[7] have reported measurements of a power-plant plume and an urban plume 35 and 46 km downwind from St. Louis, Missouri. Figure 4-25 shows the evidence of extensive ozone buildup in the urban plume. Simultaneous measurements of scattering coefficient, b_{scat}, trace the spread and dilution of suspended particulate material. It is interesting that in the urban plume, which spreads to 20 km in width, the ozone increases while the particulate matter decreases; this suggests considerable photochemical production at an altitude of 750 m. Contrary to the statements of Davis and co-workers reported above,[12,13] the power-plant plume causes a decrease, rather than an increase, in ozone. Nitric oxide in the plume reacts with the ozone as it mixes. This is clearly indicated by the distribution of particulate matter, which acts as a tracer.

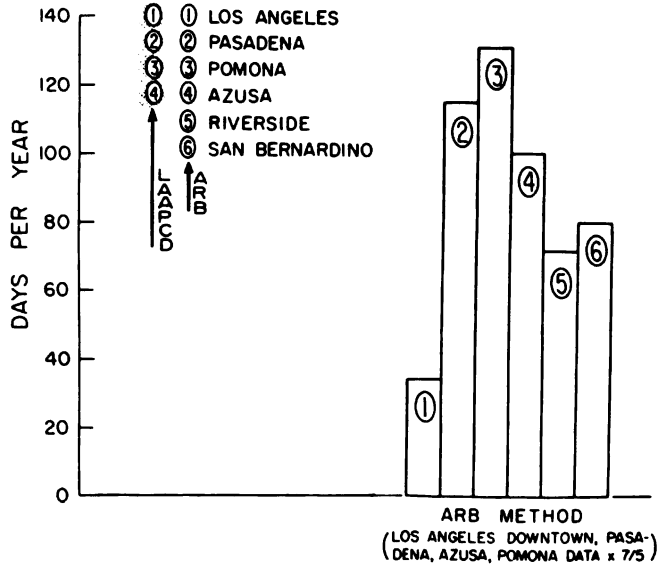


FIGURE 4-24 Number of days in 1973 on which the maximal hourly average oxidant concentration equaled or exceeded 0.20 ppm at six air monitoring stations in the southern California coast air basin. Los Angeles APCD data multiplied by 7/5 to convert to the ARB-EPA scale. Reprinted with permission from Pitts.[42]

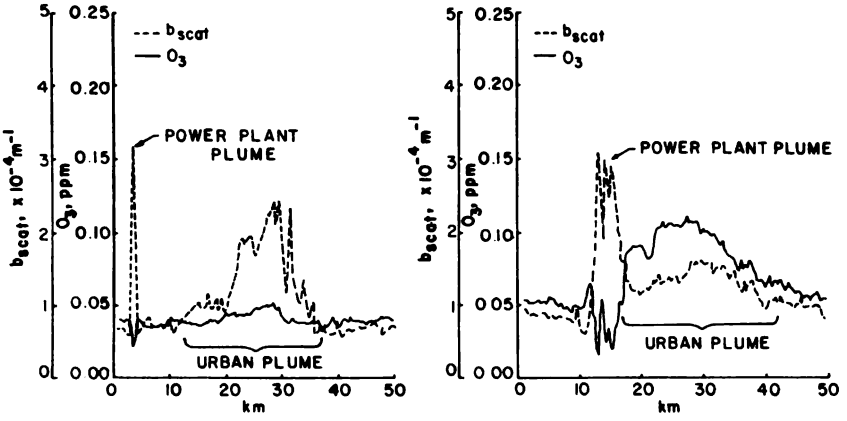


FIGURE 4-25 Plume measurements on September 6, 1973. Left, traverse at 750 m above mean sea level (msl) 35 km downwind (SW) of Arch, St. Louis, Missouri, 11:17-11:37 a.m. Right, traverse at 750 m above msl 46 km downwind (SW) of Arch, St. Louis, Missouri, 3:30-3:49 p.m. Reprinted with permission from Blumenthal and White.[7]

Rough plots of ozone isopleths from measurements over rural areas of Connecticut have been constructed by Rubino *et al.*[50] Increased concentrations (~0.31 ppm) are traced across the state during the June 10, 1974, episode. The concentrations that built up throughout the day are associated with the trajectory of an air mass that was over the metropolitan New York area during morning peak traffic. The conclusion was that the city is an origin of precursors that cause high ozone concentrations downwind. This is an interesting contrast with (but perhaps not a contradiction of) the suggestions of Coffey and Stasiuk,[11] who believed that ozone of stratospheric origin is transported into the metropolitan New York area. One difference between the two studies is that the Connecticut work traced geographic movement of increased ozone, whereas the New York paper showed temporal variations in urban and rural areas.

Sticksel[57] discussed vertical profile measurements of ozone in the stratosphere and the troposphere over the last several years. Transient ozone maximums in the troposphere are illustrated and explained by three possible mechanisms: a channel-like region conducted ozone from the stratosphere into the troposphere; ozone-laden air descended from the stratosphere and was compressed as it subsided; and ozone-rich layers leaked through the break between the polar and middle tropopauses by differential advection. Surface variations of ozone soundings were mostly attributed to anthropogenic pollution; however, relatively thick high-

altitude layers were held out as a potential natural source of high surface ozone concentrations. Sticksel concluded that further investigation was needed to ascertain whether the stratosphere can play a significant role in raising nonurban ozone concentrations.

Johnston[38 (pp. 76-82)] has pointed out possibilities of significant transport and transformation of oxidant precursors from the Los Angeles or San Francisco metropolitan air quality control regions to the central valley of California. Emission rates per unit area from sources in Los Angeles County and the Los Angeles metropolitan AQCR are approximately 10-20 times the national average for photochemically active pollutants. The emission rates per unit area in the California central valley are about the national average values for hydrocarbon and carbon monoxide, but only half the national average for NO_x. Despite the much lower emission rates in the central valley, compared with Los Angeles, the valley's ambient concentrations of carbon monoxide, NO_x, and oxidant are half those in Los Angeles. Johnston suggested that a combination of urban-rural transport and confinement without substantial ventilation is responsible for the ambient pollutant concentrations, which are relatively high, compared with emission rates per unit area.

In considering the more general case of ozone in nonurban locations, Johnston considered the problem of downward transport of ozone from the stratosphere, which contains ozone at a peak value of about 10 ppm. He cited Fabian and Pruchniewicz's[21] measurements of ozone at 19 nonurban sites in Europe and Africa—from Tromso, Norway, to Hermanus, South Africa—in 1970-1972. The 19-station network in Europe and Africa averaged approximately 0.022 ppm in the summer. However, some of the central European areas occasionally had higher values.[21] In another survey, a 12-site network was operated from 1963 to 1964 between Thule, Greenland, and Balboa, Canal Zone.[28-30] Profiles were measured at intervals from ground level to about 30 km. Table 4-8 shows the number of ozone profiles that were made at 0-1 km and the number of ground-level ozone readings that equaled or exceeded 0.08 ppm. Note that the average of the 835 values corresponds closely to the summer values reported from the European-African network. Table 4-9 details the seven readings that equaled or exceeded 0.08 ppm. Five of them occurred at the Bedford, Massachusetts, station.

Johnston argued that, if ozone were formed in the stratosphere and downward transport occurred, there would be a positive concentration gradient extending from 0.5 km to 5 km in altitude. Most of the cases in Table 4-9 obey this situation; however, for the seven high-ozone cases in Table 4-9, the reverse is true. This seems to indicate that the occur-

TABLE 4-8 Ozone Concentrations at Ground Level, 12 Widely Spaced Stations in the Northern Hemisphere, 1963-1964[a]

Station Location	Latitude, deg W	No. cases	Ozone Mole Fraction, ppm			No. Cases at or above 0.080 ppm
			Min.	Max.	Av.	
Thule, Greenland	76.5	50	0.010	0.050	0.022	0
Fairbanks, Alaska	64.8	58	0.010	0.050	0.024	0
Churchill, Canada	58.8	67	0.010	0.060	0.026	0
Goose Bay, Labrador	53.3	71	0.010	0.060	0.021	0
Seattle, Washington	47.4	83	0.010	0.100	0.029	1
Madison, Wisconsin	43.1	29	0.010	0.060	0.031	0
Bedford, Massachusetts	42.5	124	0.010	0.110	0.031	5
Fort Collins, Colorado	40.6	93	0.010	0.050	0.023	0
Albuquerque, New Mexico	35.0	102	0.005	0.060	0.026	0
Tallahassee, Florida	30.4	73	0.005	0.080	0.035	1
Grand Turk, West Indies	21.5	31	0.010	0.040	0.019	0
Panama, Canal Zone	9.0	54	0.010	0.050	0.017	0
Total		835	5	110	26	7

[a] Derived from Hering and Borden.[28-30]

TABLE 4-9 Ozone Concentrations (1963-1964) Equal to or Greater than 0.080 ppm at Ground Level (0-1 km)*

Station Location	Date	Ozone Mole Fraction, ppm	
		0 km	5 km
Bedford, Massachusetts	June 26, 1963	0.100	0.075
	July 3, 1963	0.080	0.075
	January 20, 1964	0.100	0.065
	July 15, 1964	0.090	0.060
	August 26, 1964	0.110	0.040
Tallahassee, Florida	September 11, 1963	0.080	0.045
Seattle, Washington	April 17, 1964	0.100	0.090

*Derived from Hering and Borden.[28-30]

rence of ground ozone concentration exceeding 0.08 ppm is characterized by formation reactions near the ground, which suggests that stratospheric sources are not responsible for these cases.

Evidently, the high nonurban ozone concentrations observed in 1971-1973 in the continental United States were not of natural origin. This conclusion is supported by comparisons of the data from the 1963-1964 North American study and those from the 1970-1972 European-African study. Because increased concentrations were not traceable to natural processes, anthropogenic sources may be necessary to explain the concentrations of ozone currently observed in the United States. Inhibition of the photochemical smog reaction in the atmosphere by increased NO_x emission could delay the oxidant formation until air parcels move out of urban regions. (The early portion of the vehicle emission control plan had increased NO_x emission, but decreased hydrocarbon and carbon monoxide emission.) Similarly, hydrocarbon substances in the lower paraffin series that are relatively abundant may be reactive over long periods and behave synergistically with other hydrocarbons. The high ozone values in nonurban locations may well result from these processes.

Nonurban oxidant measurements in Ohio were reported by Neligan and Angus.[39] Concentrations of 0.18 and 0.12 ppm were reported for rural sites in Wilmington and McConnelsville, respectively. At the same time, urban sites had similar concentrations. However, the nonurban sites violated the ambient air quality standard more frequently than the urban sites. Trajectory analysis showed that ozone concentrations of 0.04-0.06 ppm were found in air masses that had not passed over anthropogenic hydrocarbon sources. These may have been examples of naturally occurring oxidant. Airborne hydrocarbon bag samples were obtained over 6-min

periods, and ozone was also measured. At 4,000 ft (1,220 m) above msl, ozone concentrations exceeding 0.20 ppm were observed over northern Ohio and Pennsylvania. Wind was predominantly out of the Southeast. Forty-eight-hour reverse trajectories (apparently based on surface winds) showed that a developing high-pressure system carried air from central New York State and seaboard metropolitan areas into the test region. The correspondence of locations with high ozone concentration and locations with high acetylene, carbon monoxide, and chlorofluoromethanes ("Freons") concentrations suggests anthropogenic emission that leads to increased ozone concentrations. Early in the morning, the rural ozone concentration is at a maximum. Profiles at 7:00–9:00 a.m. show increased ozone in nonurban areas at altitudes of several thousand feet and relatively low concentrations at the surface. Daytime profiles show increases in ozone at the surface, with the late afternoon concentrations exceeding the midday concentrations.

Deep mixing of stratospheric ozone can occur during episodes of frontal passage and jet stream interaction. The mechanism for this transfer is the downward advection of air parcels rich in ozone and unaffected by removal processes until they reach the ground. The meteorologic conditions during these events are totally different from those attending high-oxidant episodes over extensive nonurban areas. Therefore, the invocation of stratospheric transport for high nonurban ozone concentrations cannot explain the long-term increases in ozone observed in the studies described above.

INDOOR VERSUS OUTDOOR OXIDANT CONCENTRATIONS

An important aspect of the relationship of indoor to outdoor oxidant concentration is the scavenging of the oxidant by surfaces, e.g., in ventilating systems and in indoor areas.

Ozone decay was measured in an office, a home, and several metal test facilities.[37] Measurements were carried out with a Mast ozone meter and an MEC chemiluminescence ozone detector. The latter was calibrated with a stable ozone source and the EPA neutral buffered potassium iodide procedure. (It was noted over a wide range of concentrations that the MEC meter measurements were consistently higher than those of the Mast meter by a factor of 1.3. That this is essentially identical with the findings of the DeMore committee[14] is interesting.) Ozone generated by a positive corona ionizer was introduced into the test facilities. Ozone decay in a metal-walled room was found to be first-order, with the rate constant

highly dependent on the preconditioning of the metal walls with respect to ozone exposure. An activated-carbon filter in the air cleaner for the room markedly increased the rate of decay of ozone. Increases in ambient humidity or temperature also markedly increased the rate of ozone decay. Decay rates in the bedroom and office environment were much greater than those in rooms with aluminum or stainless-steel walls.

During a series of smoggy days in Los Angeles, indoor concentrations of ozone were measured to study the phase relationship of indoor to outdoor ozone buildup.[51] The general mechanism of ozone decay within buildings was investigated with a Dasibi ozone instrument. Various air filters were evaluated with respect to capacity for removing ozone. A computational model was suggested for correlating indoor and outdoor concentrations of ozone as a function of time. Buildups of ozone within buildings lagged behind ambient outdoor buildups by 3 or 4 h, and the maximums indoors were not as high as those outdoors. For example, a small number of measurements suggest that concentrations in private residences reach only about 70% of the peaks attained outside. Sabersky *et al.*[51] constructed a table of ozone decomposition rates on several common materials. Activated-charcoal and Purafill filters performed better in their tests than fiberglass filters. Approximately 95% of the ozone was removed by the activated charcoal under a wide range of flow conditions. A differential equation model was proposed to calculate concentration-time relationships for indoor ozone concentration. First-order reactions with empirical constants were assumed, and the predicted ozone concentration patterns were similar to those based on measurement.

A ventilation model for relating indoor to outdoor pollutant concentration was proposed on the basis of an extension of earlier work by these authors.[54] When the outdoor pollutant concentrations change slowly, compared with the indoor changes, a rather simple equation can be used to relate indoor to outdoor concentrations. Further findings of the model study suggest that reductions in indoor concentrations down to 20% of the outdoor concentrations are feasible. Such a program would limit the number of days on which a threshold value of 0.1 ppm is exceeded to 4 or 5 per year, instead of the typical 200 or more days per year for outdoor concentrations. The model was tested with the assumption that the air flow in a building can be approximated by a well-stirred chemical reactor, which is influenced by constant sources and first-order reactions.[26] Monitoring measurements were taken for a laboratory building at the California Institute of Technology to test the model. For a series of locations within the building, the model predicted a good upper-bound curve for the observed indoor pollutant concentrations. It appeared that ozone concentrations were lower in rooms with excess furniture, books, and

papers than in rooms that had concrete floors and contained few books or papers.

The use of ozonizers for deodorizing indoor air has been discussed and evaluated with respect to potential health hazards.[31] In a normal 40-m^3 room, an ozone concentration of 0.1 ppm is established after 3.5 h of operation of one of these devices. Evidence on health effects was cited to support the conclusion that inhalation of the quantities of ozone produced by these air conditioners should be avoided and that certainly no beneficial effects should be attributed to ozone inhalation.

PERIODIC TEMPORAL PATTERNS OF OXIDANT CONCENTRATION

Although the previous discussion on spatial differences in ozone contains some references to time dependence, this discussion explores periodic time dependence further. Specifically, diurnal and seasonal variations are explored here, with data on various cities in the United States.

The diurnal variations in mean hourly average oxidant concentration are illustrated in Figures 4-26 and 4-27. Several factors influence the shapes of these curves. The primary influence is that of sunlight intensity, inasmuch as photons in the ultraviolet are responsible for the primary photochemical process that leads to ozone formation. Note that the St.

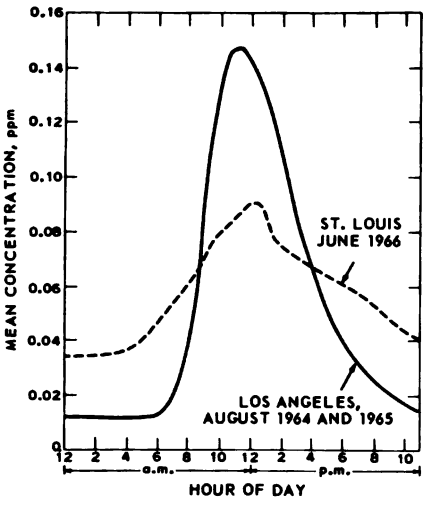


FIGURE 4-26 Diurnal variation in mean hourly average oxidant concentration in Los Angeles, California, and St. Louis, Missouri. Reprinted from U.S. DHEW.[59]

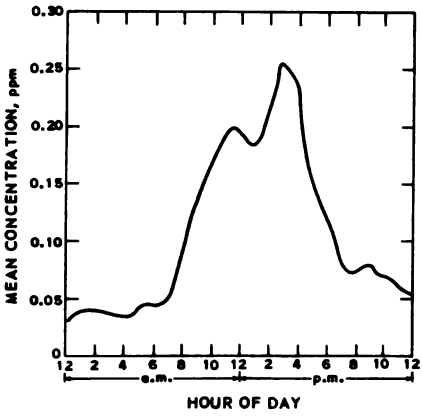


FIGURE 4-27 Diurnal variation in mean hourly average oxidant concentration in Philadelphia, Pennsylvania, August 6–8, 1966. Reprinted from U.S. DHEW.[59]

Louis curve for June 1966 is broader than that for Los Angeles in August. This is explained by differences in sunlight intensity distribution throughout the day.

Another factor in the curve shapes is the relative proximity of sources of nitric oxide, which reacts with ozone locally and suppresses it. Figure 4-20 shows how the diurnal variations are larger in a city where nitric oxide emission dominates than in a rural area, where the ozone is relatively unaffected. This brings us to a third factor influencing the shape of these curves: advective transport. If movement of ozone from another area is dominant in the local photochemical oxidant concentration, then the wind direction and speed have a great influence on the curve shape. Figure 4-28 shows that, in a central urban area like downtown Los Angeles, the ozone pulse shape is relatively symmetric about the middle of the day, because of the dominance of local production. In outlying areas, however, such as Azusa and Riverside, the pulse is skewed to the later hours of the afternoon, because of the greater role of transport to the area. Riverside shows this most decidedly, with its own locally produced ozone rising about 8 a.m. and a large secondary peak at 4 p.m., presumably the result of advection from areas to the west. Palm Springs peaks after 8 p.m.

Monthly patterns that show the seasonal variation in oxidant concentration for three cities are presented in Figures 4-29 and 4-30. Again, the combination of availability of sunlight and degree of ventilation governs the shape of these curves. Los Angeles is subject to high late-season oxidant production, because the late spring and early summer months are heavily affected by stratus, which obscures the sunlight. Denver, Colorado, however, has a more symmetric seasonal distribution that follows the sunlight pattern. Phoenix, Arizona, has relatively

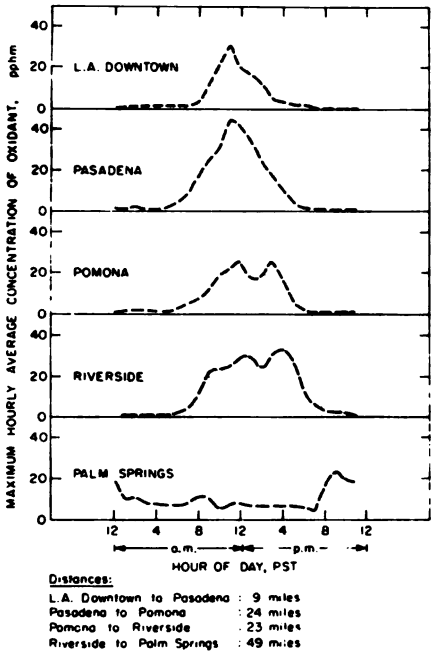


FIGURE 4-28 Diurnal variation in mean 1-h average oxidant concentration at selected California sites, July 25, 1973. Reprinted with permission from Pitts *et al*.[43]

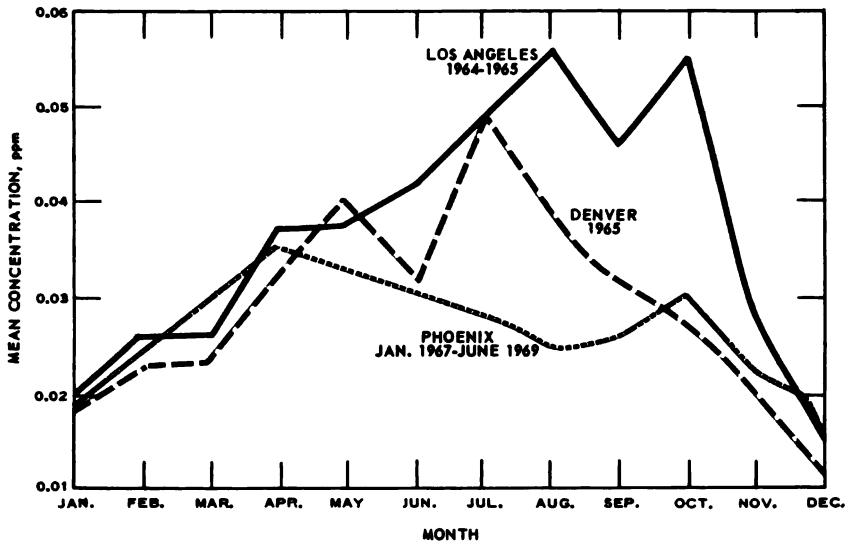


FIGURE 4-29 Monthly variation in mean hourly oxidant concentration in three selected cities. Reprinted from U.S. DHEW.[59]

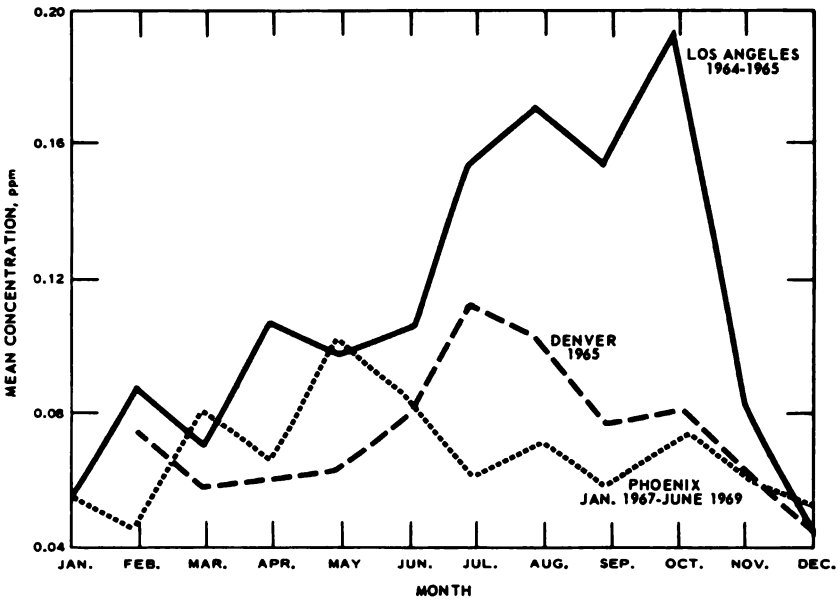


FIGURE 4-30 Monthly variation in mean daily maximal 1-h average oxidant concentration in three selected cities. Reprinted from U.S. DHEW.[59]

lower oxidant peaks in July, August, and September, because of the dominance of convectively driven mixing, which counteracts the effect of the higher solar intensity.

LONG-TERM TRENDS OF AMBIENT CONCENTRATIONS

The limited sample of photochemical oxidant and ozone data precludes extensive trend analysis for all but a few regions in the United States. Some such analysis has been done by the California Air Resources Board.[32] Its analysis can be applied on a temporal basis to any given location; however, spatial intercomparisons are subject to the problems pointed out by Pitts.[42]

Both annual average and 3-yr moving average values of oxidant concentration were plotted in a CARB report by Kinosian and Duckworth[32] for several stations in the southern California coastal air basin during 1963-1972. These are shown in Figures 4-31, 4-32, and 4-33. The 3-yr moving average is used to smooth the data. Comparing the three cities—Los Angeles, Azusa, and Riverside—they found a distinct downtrend in

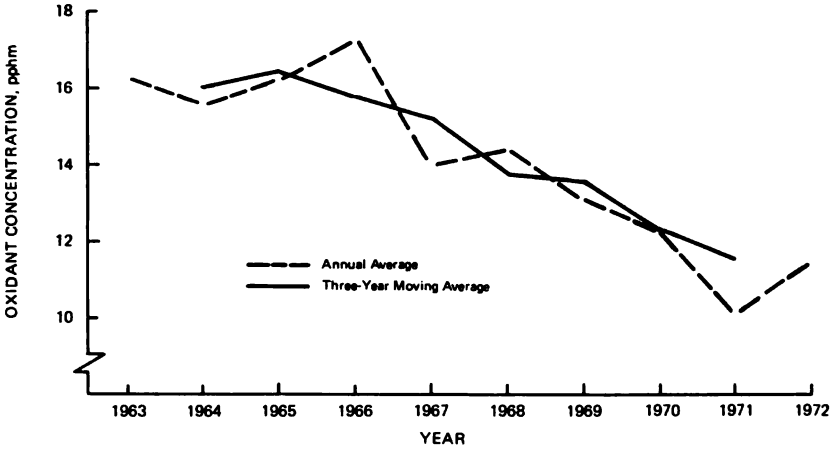


FIGURE 4-31 Oxidant trends in Los Angeles, California, 1963-1972. Annual and 3-yr moving averages of daily maximal 1-h concentrations for July, August, and September. Reprinted with permission from Kinosian and Duckworth.[32]

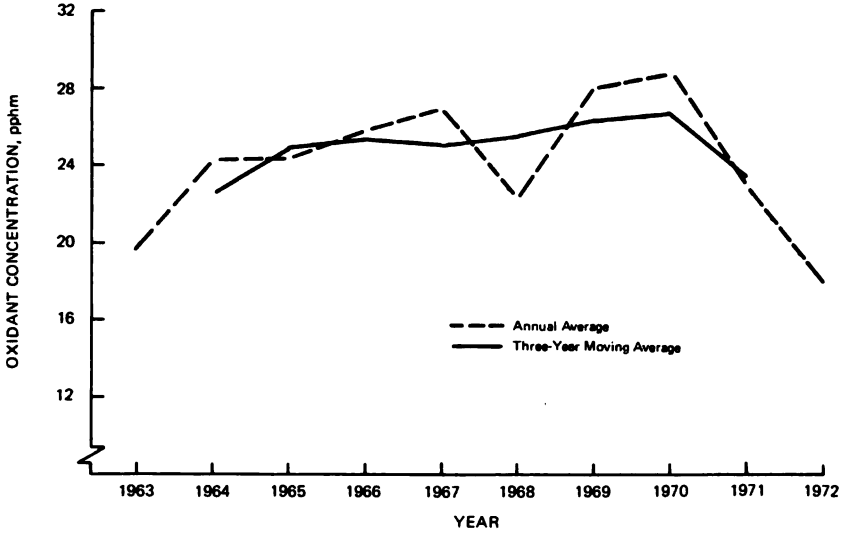


FIGURE 4-32 Oxidant trends in Azusa, California, 1963-1972. Annual and 3-yr moving averages of daily maximal 1-h concentrations for July, August, and September. Reprinted with permission from Kinosian and Duckworth.[32]

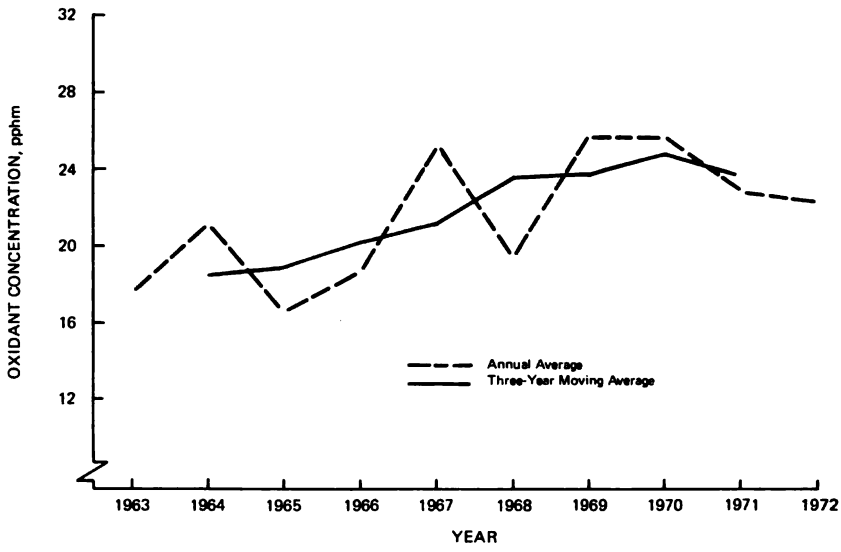


FIGURE 4-33 Oxidant trends in Riverside, California, 1963-1972. Annual and 3-yr moving averages or daily maximal 1-h concentrations for July, August, and September. Reprinted with permission from Kinosian and Duckworth.[32]

the 3-yr moving average for Los Angeles, a nearly level trend for Azusa, and a slight uptrend for Riverside. It should be pointed out that the variability due to weather is not completely removed by the 3-yr averaging process. Thus, the report applied an adjustment factor for temperature aloft and concluded that low oxidant concentrations in 1968 were due to the weather, whereas the lower ones observed in 1970 and 1971 were not.

A report by the National Academy of Sciences Coordinating Committee on Air Quality Studies to the U.S. Senate[38] concluded that "available air monitoring data do not allow conclusions to be drawn about photochemical oxidant trends on a nation-wide basis." This report relied heavily on California data to illustrate trends, because so much information was available for that region. Maximal 1-h concentrations in the New Jersey cities of Bayonne and Newark were compared for 4-yr periods between 1966 and 1973. These two cities exhibited 24% and 46% decreases, respectively. Emphasis was placed on differences in calibration procedure in various jurisdictions of air pollution control agencies. It is indicated that trend analysis for each station is still valid, despite the differences in calibration procedure. But values from different places must be compared cautiously.

CAMP data presented in Table 4-10 show a generally decreasing trend in total oxidant concentration as measured by neutral buffered potassium iodide. The southern coastal air basin in California is also known as the metropolitan Los Angeles AQCR. The Figure 4-34 map shows the location of air pollution monitoring stations. However, the boundaries shown in that figure include more than the AQCR referred to. Of Santa Barbara County, only the southern strip bounded on the north by the coastal mountain range is included in that basin; and of San Bernardino and Riverside Counties, only the partially urbanized areas in the western portions are included. Figure 4-35 shows the oxidant trends in the southern coastal air basin. Two selection techniques were used for stratifying the air monitoring data.[27] One is based on the "rule 57" day, which is defined as a day having an inversion base at 4:00 a.m. lower than 1,500 ft (457 m), a maximal mixing height below 3,500 ft (1,067 m), and an average wind speed between 6:00 a.m. and noon below 5 mph. Another selection technique stratifies the data for an inversion base less than 3,500 ft. The number of days in each year that exceed an oxidant concentration of 0.25 ppm as defined by these two systems shows a distinct downward trend in the graph.

As a result of recent control activities, the downtown Los Angeles station has experienced successively lower oxidant concentrations,[32,58] as shown in Figure 4-31. It should be noted that Riverside and San Bernardino Counties show great fluctuations in annual average oxidant concentrations, with Riverside actually having an increase. Again, it should be emphasized that differences in calibration methods from place to place will not affect the temporal trend analysis presented here.

It has been argued that the smog reaction has been inhibited by the higher NO_x:hydrocarbon ratios brought about by early emission control systems, thereby increasing concentrations at downwind locations. It has also been argued that increased NO_x concentrations could contribute to the high nonurban values by interacting over long periods with natural methane or terpene to produce ozone.[10,48] It is probable that nitric oxide emission reacts locally to decrease preexisting atmospheric ozone; however, there is little doubt that increased NO_x over a long-time (long-range) trajectory will ultimately make more oxidant, if more hydrocarbon is introduced. In summary, added nitric oxide inhibits ozone locally, but can enhance it regionally.

Altshuller[1] evaluated oxidant results from throughout the United States. CAMP concentration data were analyzed for 8 or more years of measurements available for the period 1964–1973. Tables 4-11 and 4-12 summarize the measurements. Data from 2 yr were rejected, because the method of eliminating sulfur dioxide interference was not in operation. Inter-

TABLE 4-10 Total Oxidant Concentrations Measured at CAMP Stations by Neutral Buffered Postassium Iodide Method[a]

Station Location	Average of 99*th* Percentile, $\mu g/m^3$			Average of Annual 2*nd* Highest Value, $\mu g/m^3$		
	1962–1966	1967–1971	Change, %	1962–1966	1967–1971	Change, %
Chicago, Illinois	128.1	166	+30	263	299	+14
Cincinnati, Ohio	192	177	−8	333	287	−14
Denver, Colorado	—	—	—	—	—	—
Philadelphia, Pennsylvania	212	270	−20	459	299	−35
St. Louis, Missouri	—	—	—	—	—	—
CAMP Averages	177	171	−3	352	295	−16

[a] Derived from National Academy of Sciences Study.[38]

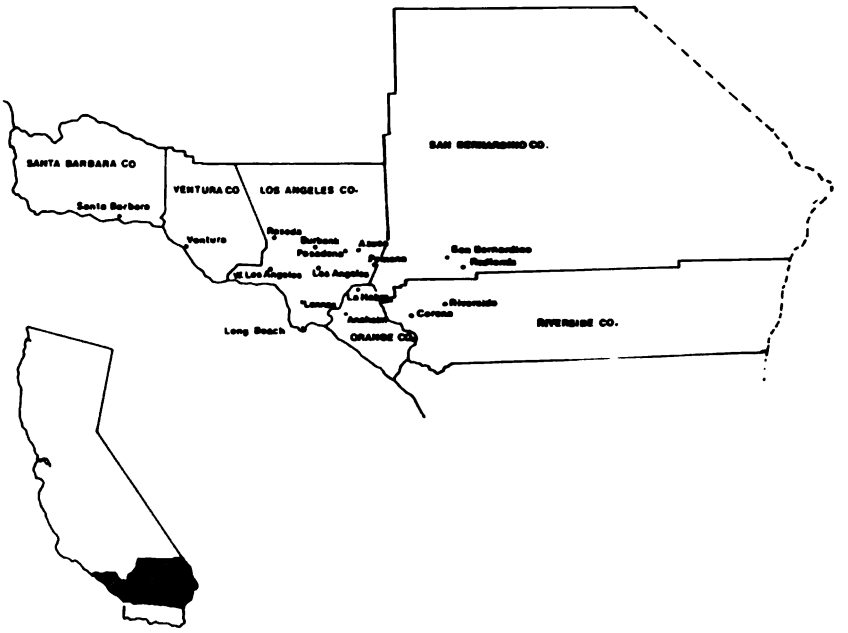
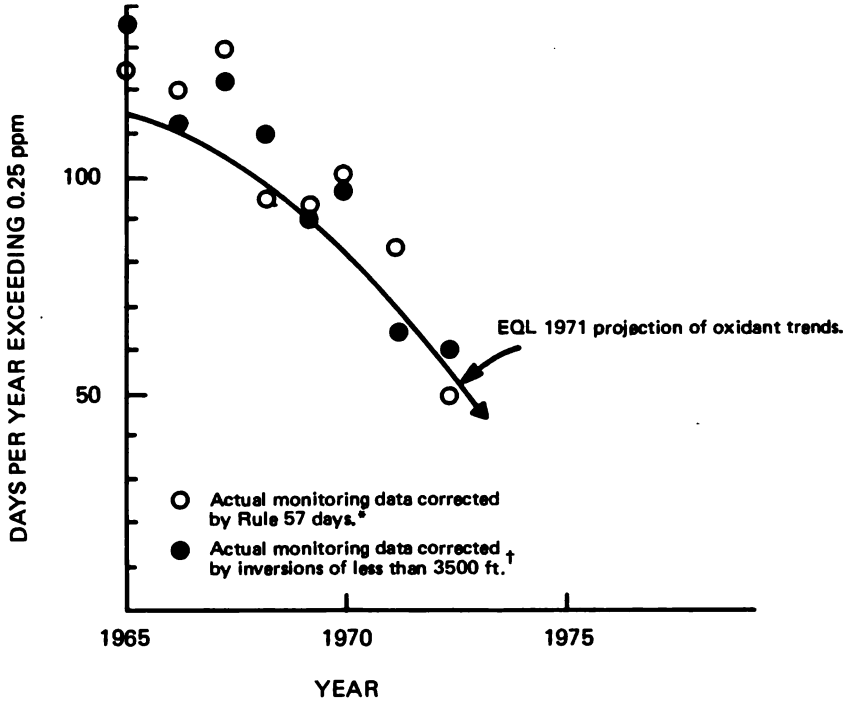


FIGURE 4-34 Air pollution monitoring network for southern coastal air basin, California. Reprinted with permission from a National Academy of Sciences study.[38]

ference from nitrogen oxides was removed from the data by subtracting 20% of the combined nitric oxide and nitrogen dioxide concentrations from the total oxidant reading, because all the data used were from the colorimetric potassium iodide method of measurement. Where comparisons were available, colorimetry oxidant data averaged 0.015 ppm higher than the chemiluminescence measurements. It was observed that this difference should be expected, because of possible additional interferences from organic peroxides or peroxyacylnitrates.

The fractions of days with valid oxidant data for May–October and for June–August were tabulated for the CAMP sites for 1964–1973. The average over all years and all sites was 60%. Seasonal effects were shown by distributing according to month the days with oxidant concentrations exceeding 0.08 ppm. Sunday oxidant concentrations and comparisons of results by location were also discussed. Annual trends in oxidant concentration for the various sites were represented by plotting the number of days with oxidant concentrations over 0.08 ppm (or the California state standard of 0.10 ppm) for each year at each site (see Figure 4-36 and Table 4-13). It was suggested that the relatively infrequent high

oxidant concentrations observed in Chicago were due to a site effect involving the lake breeze ventilation and the high concentration of automobile-generated nitric oxide. The higher frequency of high ozone concentrations in Denver was attributed to Denver's high altitude and attendant increases in ultraviolet intensity. No distinct Sunday oxidant effect was noted with respect to exceeding the 0.08-ppm concentration. Neither precursor concentrations nor favorable reactant ratios should lead one to expect more favorable conditions for oxidant formation on Sundays, according to the paper. However, it was noted that in the Los Angeles basin alert concentrations (0.6 ppm) or near-alert concentrations (0.4–0.6 ppm) are never observed on Sundays. Extensive discussion was de-



* A Rule 57 day is one on which the inversion base at 4 AM (PST) is lower than 1500 feet, the maximum mixing height is not above 3500 feet and the average surface wind speed between 600 AM and 12 noon (PST) does not exceed 5 MPH.

† This correction has been adopted by the California Air Resources Board in an attempt to remove this meteorological variability from the analyzing oxidant trends.

FIGURE 4-35 Southern California coastal air basin oxidant trends. Reprinted with permission from Heitner and Krier.[27]

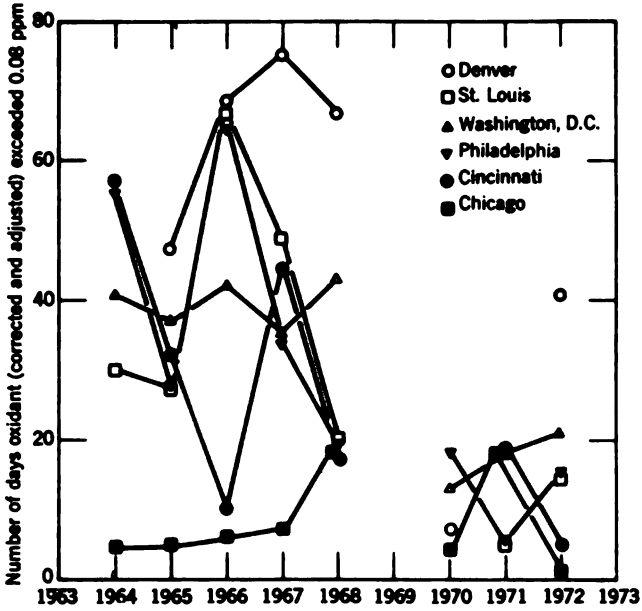


FIGURE 4-36 Trend in oxidant concentration by year at CAMP sites. Reprinted with permission from Altshuller.[1]

voted to the apparent downward shifts in oxidant measurements for central urban areas, in contrast with the nearly constant or rising oxidant concentrations in suburban and rural areas.

The latest Los Angeles County APCD profile gave further insights into the measurement of trends and their interpretation. It used California's ambient air quality standard of 0.10-ppm oxidant (hourly), instead of the federal standard of 0.08 ppm. Table 4-13 shows the numbers of days on which the state standard for total oxidant (by the potassium iodide method) was equaled or exceeded in the Los Angeles basin (confined here to the County of Los Angeles). From 1957 to 1973, a distinct downtrend in the number of days was observed. On an average over this 17-yr period, there were 258 days of violation per year. During the last 3 yr tabulated—1971, 1972, and 1973—the numbers were 218, 211, and 185, respectively. This trend is qualified by the observation that climatologic conditions during 1973 were favorable for smog formation, with the lower frequency of inversion and weak winds. It is perhaps more realistic to consider the final 3 yr as a group, rather than using 1973 to interpret the trends. Another index that the Los Angeles County APCD uses is the total number of days on which eye irritation was recorded in

TABLE 4-11 Summary of Total Oxidant Concentrations Recorded at CAMP Sites, 1964–1972[a]

City	Year	No. Days of Valid Data	No. Days with at Least One Hourly Average ≥ 0.05 ppm	0.10 ppm	0.15 ppm	Maximal Hourly Average Oxidant Concentration, ppm
Chicago, Illinois	1964	254	149	15	0	0.13
	1965	275	120	9	0	0.13
	1966	235	52	6	3	0.19
	1967	255	113	16	1	0.16
	1968	211	113	17	5	0.18
	1969	24	15	0	0	0.07
	1970	200	97	4	3	0.20
	1971	276	90	14	2	1.17
	1972	312	79	4	0	0.14
Cincinnati, Ohio	1964	303	137	36	5	0.26
	1965	310	182	19	5	0.17
	1966	208	54	1	0	0.10
	1967	228	122	24	1	0.20
	1968	86	65	7	0	0.14
	1969	48	23	5	1	0.16
	1970	7	1	0	0	0.08
	1971	221	96	10	1	0.16
	1972	257	69	3	1	0.15
Denver, Colorado	1965	285	226	51	14	0.25
	1966	298	187	46	9	0.19
	1967	166	76	12	4	0.21
	1968	151	149	28	5	0.26
	1969	108	70	2	0	0.13
	1970	141	69	9	1	0.18
	1971	184	63	5	1	0.20
	1972	209	94	26	1	0.18

Philadelphia, Pennsylvania	1964	269	124	37	9	0.20
	1965	266	109	23	4	0.33
	1966	315	145	52	19	0.52
	1967	282	124	28	3	0.17
	1968	140	88	18	3	0.21
	1969	92	37	3	0	0.11
	1970	112	29	10	0	0.13
	1971	260	47	5	0	0.14
	1972	47	20	10	1	0.15
St. Louis, Missouri	1964	253	156	26	6	0.26
	1965	329	206	33	8	0.35
	1966	292	174	33	5	0.22
	1967	289	185	38	4	0.20
	1968	163	100	7	2	0.23
	1969	95	47	5	0	0.12
	1970	96	20	0	0	0.08
	1971	294	77	1	0	0.13
	1972	203	77	9	2	0.16
Washington, D.C.[b]	1964	293	163	40	4	0.20
	1965	284	150	25	3	0.21
	1966	325	134	27	2	0.16
	1967	322	137	27	5	0.26
	1968	217	134	40	9	0.25
	1969	71	30	1	0	0.10
	1970	167	95	10	2	0.16
	1971	299	144	17	0	0.13
	1972	179	45	6	0	0.13

[a] Derived from NATO data.[40]
[b] Site moved to another section of the city at end of 1968.

TABLE 4-12 Number of Days and Percentage of Days with Corrected Oxidant Concentration over 0.08 ppm at CAMP Sites, 1964–1973[a]

Corrected Oxidant Concentration, ppm	Days[b] Chicago	Cincinnati	Denver	Philadelphia	St. Louis	Washington	Total
	No.	No.	No.	No.	No.	No.	No.
0.085–0.095	15(2)	75(8)	64(16)	53(14)	92(18)	87(14)	386(72)
0.10–0.12	22(5)	52(5)	70(10)	77(14)	56(9)	95(12)	372(55)
0.125–0.145	7(3)	32(2)	31(1)	26(2)	19(8)	19(4)	134(20)
0.15–0.195	6(1)	7(2)	20(1)	17(3)	6(1)	11(2)	67(10)
0.20–0.245	0	0	6(0)	3(2)	3(0)	2(0)	14(2)
0.25–0.295	0	1(0)	0	1(0)	0	0	2(0)
0.30–0.345	0	0	0	1(0)	0	0	1(0)
Total	50(11)	167(17)	191(28)	178(35)	176(36)	214(32)	976(159)
	%	%	%	%	%	%	%
At or above 0.10 ppm	70(82)	55(53)	67(44)	70(60)	48(50)	59(56)	60(55)
At or above 0.125 ppm	26(36)	24(24)	30(7)	27(20)	16(25)	15(18)	22(20)
At or above 0.15 ppm	12(10)	5(12)	14(4)	12(14)	5(3)	6(6)	9(8)
At or above 0.20 ppm	0(0)	1(0)	3(0)	3(0)	2(0)	1(0)	2(0)

[a] Derived from Altshuller.[1]
[b] Sundays in parentheses.

TABLE 4-13 Number of Days on Which State Standard 1-Hour Average of 0.10 ppm for Total Oxidant[a] was Equaled or Exceeded in Los Angeles Basin[b]

	Year																
Month	1957	1958	1959	1960	1961	1962	1963	1964	1965	1966	1967	1968	1969	1970	1971	1972	1973
January	15	26	27	13	23	17	9	9	9	11	10	8	8	5	10	2	0
February	21	21	14	15	22	9	22	16	17	13	22	13	7	11	12	17	1
March	26	15	28	25	18	12	18	12	10	23	18	18	17	19	17	26	3
April	24	25	26	23	22	23	16	15	17	26	14	24	22	19	17	22	12
May	20	30	29	25	24	20	23	22	21	26	22	26	26	26	17	22	26
June	28	30	30	30	29	26	21	27	25	29	24	26	22	27	26	26	25
July	31	31	31	31	31	31	29	31	31	31	31	31	30	31	31	30	31
August	31	31	30	30	30	31	31	30	31	31	31	29	31	31	31	29	29
September	30	29	28	30	25	29	26	30	24	28	28	25	30	29	26	24	27
October	25	29	28	26	22	26	27	27	28	30	31	26	27	24	18	8	24
November	26	27	27	19	22	24	16	10	15	17	20	20	14	17	13	4	7
December	28	28	18	19	15	19	20	3	8	6	8	6	12	2	0	1	0
Total	305	322	316	286	283	267	258	232	236	271	259	252	246	241	218	211	185

[a] By potassium iodide method.
[b] Derived from Birakos.[5]

the Los Angeles basin. This is summarized in Table 4-14. For the same 17-yr span, the average number of days characterized by eye irritation is 163. However, during the final 3 yr, the numbers were 125, 125, and 124, which seem to correspond to the ozone measurement trend. It is of interest to note the similar statistics (Table 4-15) for violation of the state visibility standard—namely, for one observation, visibility should not be less than 10 miles (16 km) when the relative humidity is less than 70%. The data in Table 4-15 are reported on the basis of the 1970 state standard through May 1972 and later on the basis of the June 1972 state standard. The average number of days on which the standard was violated over the 12-yr period is 328. For the last 3 yr of the reporting period, the numbers were 333, 311, and 298. This confirms the improvement trend exhibited in the other two tables.

It is also of interest in interpreting trends to look at the rare event and at the frequency distribution, as well as average values. This can be done by examining the number of ozone alerts called for the series of years under analysis. For a first alert for ozone, the Los Angeles County APCD has set the value at 0.50 ppm. Ninety alerts were declared from the inception of the alert program in 1955 through December 1973. The 1955–1972 average is five alerts per year. However, in 1972 and 1973, only one alert was declared each year. The highest number of alerts posted during a single year was 15, in 1955. The highest frequency of alerts occurs during September. January and February have never experienced ozone alerts. Alerts are most frequent on Friday and have never occurred on Sunday.

Frequency distributions of ozone concentrations for Azusa and Goleta, California, are shown in Figures 4-37 and 4-38, respectively. (These curves were prepared by J. R. Martinez of Environmental Research and Technology, Inc.) The Azusa distribution consists of points for 8 yr (each taken as an individual sample). The Goleta data are for 1 yr only. A year-to-year scatter of approximately ±15% about a mean curve is observed for Azusa; both curvature and slope difference distinguish it from the Goleta distribution. The primary reference standard for the latter followed the CARB procedure; that for Azusa followed the Los Angeles APCD procedure. Although the DeMore *et al.* report[14] stated that zero offsets are suppressed and that direct multiplicative corrections should be applied, one must wonder whether nonlinearities may still emerge from the calibration procedure, in view of the differences in curvature. Another explanation may be that, because of the higher concentrations in Azusa, actual atmospheric nonlinearities occur. But this will not account for the large slope discrepancy at low concentrations. It should be noted that all Los Angeles APCD distributions qualitatively resemble Azusa's and that other CARB distributions are like Goleta's.

TABLE 4-14 Number of Days on Which Eye Irritation Was Recorded in Los Angeles Basin[a]

Month	Year																	
	1956	1957	1958	1959	1960	1961	1962	1963	1964	1965	1966	1967	1968	1969	1970	1971	1972	1973
January	12	3	6	11	3	12	10	4	3	2	2	6	2	3	1	8	0	0
February	11	12	6	3	7	12	7	11	7	11	3	6	7	1	4	4	6	0
March	14	11	2	14	19	9	5	8	4	4	18	10	5	9	7	6	15	0
April	8	13	8	18	17	16	19	8	6	15	18	1	7	5	6	7	11	9
May	12	8	20	7	15	8	16	11	13	12	17	11	13	16	12	8	17	18
June	23	17	17	23	24	21	21	13	13	9	22	15	17	12	17	16	17	16
July	23	27	21	28	26	22	28	23	28	24	27	23	27	21	26	23	18	21
August	23	27	31	23	26	28	29	22	22	29	23	26	19	31	26	23	20	23
September	28	25	26	12	21	15	25	19	23	20	20	15	16	24	20	17	15	17
October	17	11	21	22	18	19	23	22	21	22	19	23	13	10	11	7	2	17
November	12	10	16	18	14	13	18	7	6	12	6	13	8	4	4	6	4	3
December	16	6	20	9	8	11	13	13	1	4	1	0	0	1	0	0	0	0
Total	199	170	194	188	198	186	214	161	147	164	176	149	134	137	134	125	125	124

[a] Derived from Birakos.[5]

TABLE 4-15 Number of Days on Which State Standard for Visibility[a] Was Equaled or Exceeded[b]

Month	Year											
	1962	1963	1964	1965	1966	1967	1968	1969	1970	1971	1972	1973
January	25	29	27	26	26	28	28	24	21	28	26	22
February	18	22	24	24	26	28	26	22	23	24	28	15
March	26	26	24	28	26	29	28	24	27	28	29	16
April	29	21	24	22	30	24	29	28	27	27	27	20
May	25	28	26	31	28	30	30	31	30	28	28	29
June	29	28	29	29	29	28	30	29	30	30	28	28
July	31	31	31	31	31	31	31	31	31	31	28	31
August	31	31	31	31	31	31	30	31	31	31	31	30
September	30	29	30	29	29	30	30	30	29	29	26	30
October	30	30	30	28	31	31	30	28	30	29	24	27
November	29	25	20	19	29	27	27	22	27	29	21	24
December	31	29	21	26	25	26	26	26	26	19	15	26
Total	334	329	317	324	341	343	345	326	332	333	311	298

[a] Visibility < 10 miles (16 km) when relative humidity is < 70%.
[b] Derived from Birakos.[5]

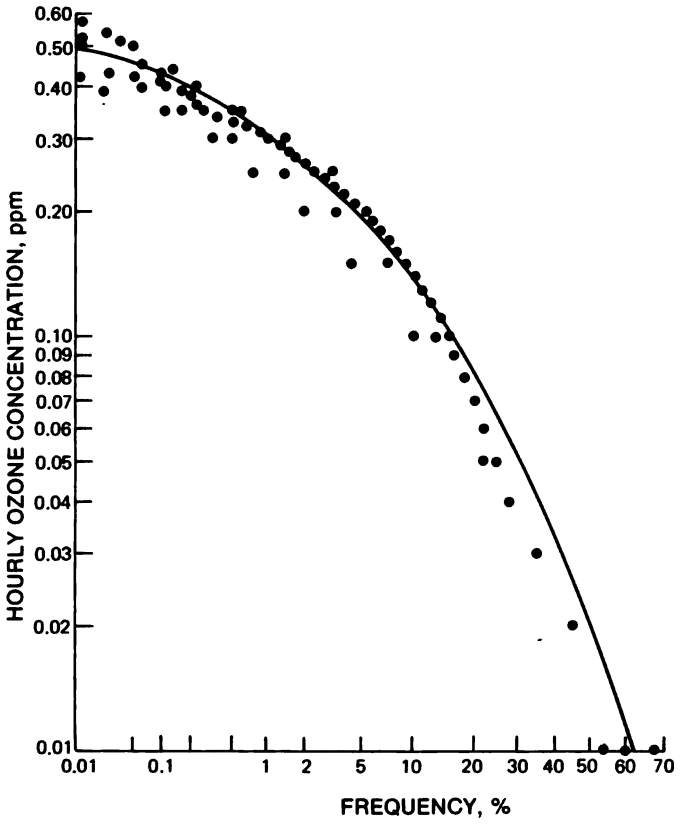


FIGURE 4-37 Hourly ozone frequency distribution, Azusa, California, 1965-1972.

PHOTOCHEMICAL OXIDANTS OTHER THAN OZONE

As mentioned previously, the scope of photochemical oxidants extends to organic nitrates and other carbonyl compounds. Among the organic nitrates, the one most often cited is peroxyacetylnitrate (PAN). Electron-capture detector techniques applied to the gas chromatograph were used to measure PAN concentrations in Los Angeles late in 1965.[34]

On each of 16 weekdays in September and 19 in October, seven measurements were made. The daily means are plotted in Figure 4-39. Note that the PAN concentrations are considerably below the total oxidant concentrations.

At the University of California at Riverside, the same technique was

used to measure PAN, beginning in June 1966. Hourly samples were collected between 7:00 a.m. and 4:00 p.m. Figure 4-40 shows the PAN distribution to exhibit double peaks, which sometimes occur with oxidant concentrations.[59] Note that the relationship between the PAN curve and the oxidant curve in Figure 4-40 is very similar to that in Figure 4-39. Figure 4-41 shows the seasonal variations in PAN concentration and oxidant concentration for portions of 1966 and 1967 at Riverside.

Aldehydes may also be thought of as photochemical oxidants. The definition here becomes a bit hazy, because aldehydes in themselves are photooxidative reactants, as well as secondary pollutants that have adverse health effects. Referring to Figure 4-4, we note that aldehyde concentration throughout the day in Rome, Italy, seems to decay at roughly the same rate as the nitric oxide concentration. It would be expected to track the reactive fraction of the hydrocarbons, and this is also borne out approximately by the Rome data. A maximal formaldehyde concen-

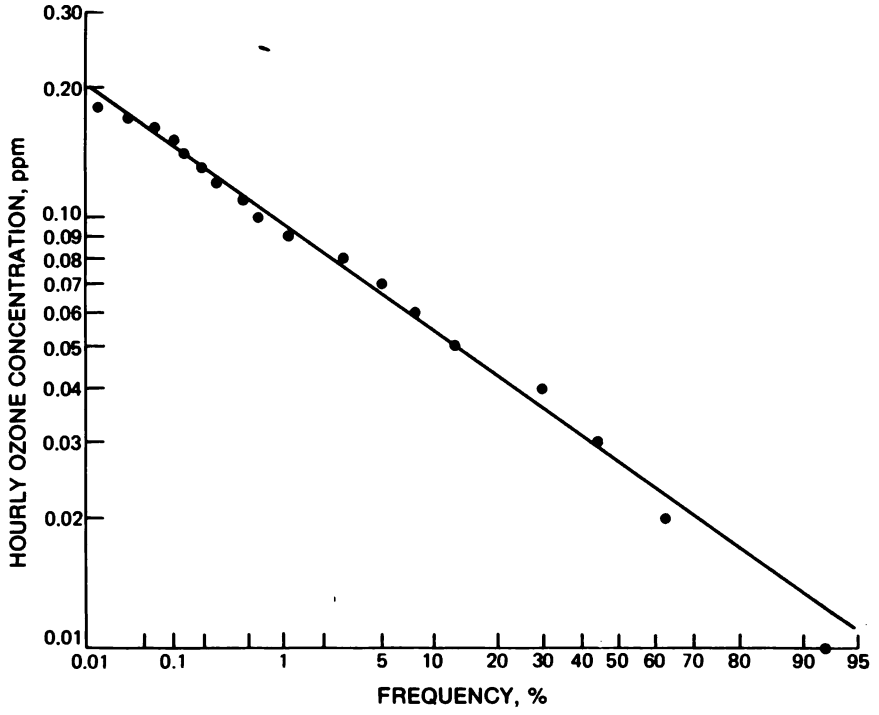


FIGURE 4-38 Hourly ozone frequency distribution, Goleta, California, 1973.

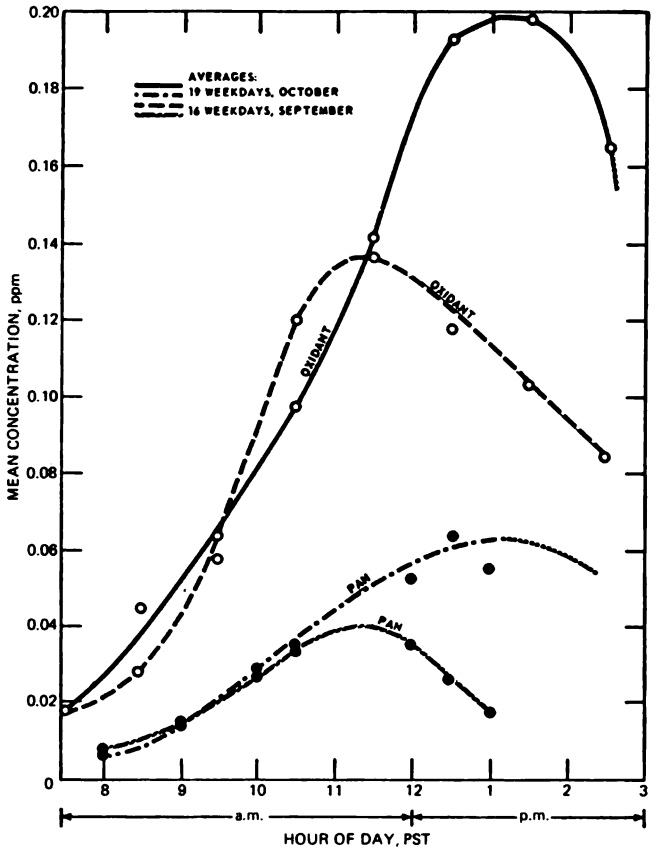


FIGURE 4-39 Variation in mean 1-h average oxidant and PAN concentrations, by hour of day, downtown Los Angeles, California, 1965. Reprinted from U.S. DHEW.[59]

tration of 39 $\mu g/m^3$ (0.032 ppm) was measured in Berlin, Germany, in 1967 on a street with high traffic density.[33]

Measurements were conducted in Rotterdam during the period January-March 1973, to determine the ambient concentrations of aliphatic aldehydes. The 24-h average concentrations were around 5 $\mu g/m^3$, and the 8:00 a.m.-4:00 p.m. average concentration was 8.4 $\mu g/m^3$ (data from L. J. Brasser).

Dickinson[16] measured total aldehydes in Los Angeles by the bisulfite procedure and formaldehyde by the chromotrophic acid procedure.

Renzetti and Bryan[45] measured total aldehydes, formaldehyde, and

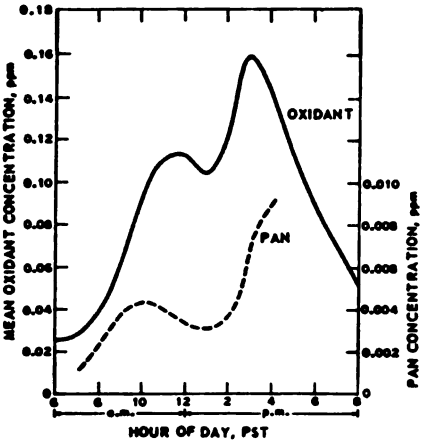


FIGURE 4-40 Variation in mean 1-h average oxidant and PAN concentrations, by hour of day, at the Air Pollution Research Center, Riverside, California, September 1966. Reprinted from U.S. DHEW.[59]

acrolein in Los Angeles. The maximal acrolein concentration observed was 25.2 $\mu g/m^3$ (0.011 ppm), and the maximal total aldehyde concentration was 0.36 ppm for a 10-min sample. The formaldehyde concentration never exceeded 130 $\mu g/m^3$ (0.10 ppm).

Altshuller and McPherson[2] used spectrophotometry to analyze aldehydes in the Los Angeles atmosphere in the fall of 1961. Table 4-16 shows the diurnal variation in both formaldehyde and acrolein concentrations. Both rise early, remain relatively constant throughout the day, and decrease

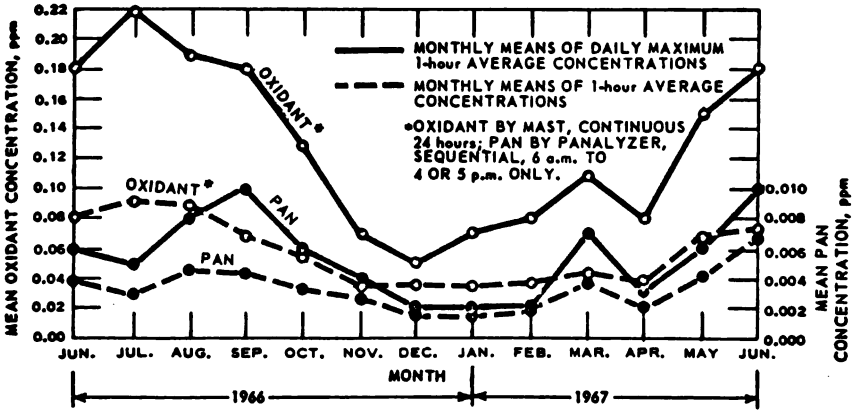


FIGURE 4-41 Monthly variation in oxidant and PAN concentrations at the Air Pollution Research Center, Riverside, California, June 1966–June 1967. Reprinted from U.S. DHEW.[59]

TABLE 4-16 Average Aldehyde Concentrations by Hour in Los Angeles, September 25 through November 15, 1961[a]

Sampling Time	Formaldehyde		Acrolein	
	No. Days	Average Concentration, ppm	No. Days	Average Concentration, ppm
7 a.m.	7	0.041	2	0.007
8 a.m.	18	0.043	3	0.009
9 a.m.	21	0.045	3	0.009
10 a.m.	28	0.044	5	0.008
11 a.m.	27	0.051	5	0.008
12 noon	23	0.044	3	0.005
1 p.m.	25	0.041	7	0.008
2 p.m.	27	0.034	5	0.007
3 p.m.	25	0.026	4	0.004
4 p.m.	15	0.019	5	0.004

[a] Derived from Altshuller and McPherson.[2]

in the later part of the day. Acrolein apparently accounts for only about 10% of the total olefinic aldehyde in the atmosphere, with most of the latter concentration being accounted for by formaldehyde.

Another nonozone photochemical oxidant observed in urban atmospheres is hydrogen peroxide. Bufalini *et al.*[9,24] found this compound to be present at concentrations up to 0.04 ppm in the air in Hoboken, New Jersey, and up to 0.18 ppm on a smoggy day in Riverside, California. Figure 4-42 shows that the diurnal hydrogen peroxide variation in Riverside on August 6, 1970, nearly paralleled that of total oxidant. Figure 4-43 indicates, however, that on at least one occasion (August 11, 1970) it peaked as early as 10:30 a.m.

A dramatic departure of ozone measurements from total oxidant measurements has been reported[53] for the Houston, Texas, area. Side-by-side measurements suggested that either method was a poor predictor of the other. Consideration was given to known interferences due to oxides of nitrogen, sulfur dioxide, or hydrogen sulfide, and the deviations still could not be accounted for. In the worst case, the ozone measurements exceeded the national ambient air quality standard for 3 h, and the potassium iodide instrument read less than 15 ppb for the 24-h period. Sulfur dioxide was measured at 0.01–0.04 ppm throughout the day. Even for a 1:1 molar influence of sulfur dioxide, this could not explain the low oxidant values. Regression analysis was carried out to support the conclusion that the ozone concentration is often much higher than the nonozone oxidant concentration.

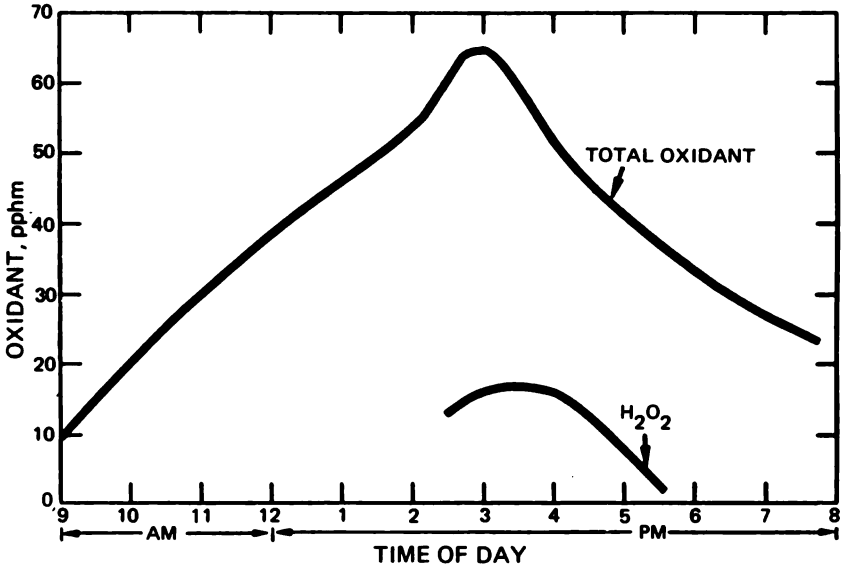


FIGURE 4-42 Measured oxidant at Riverside, California, August 6, 1970. Reprinted with permission from Bufalini *et al.*[9]

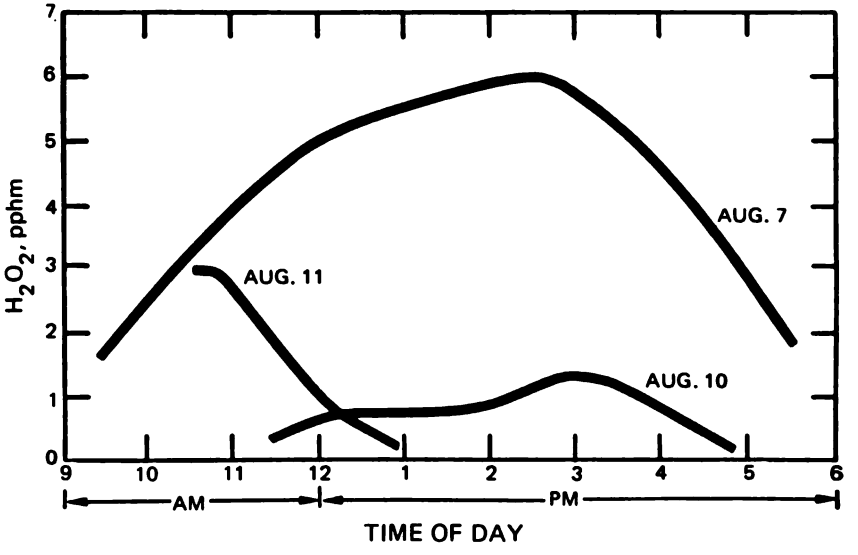


FIGURE 4-43 Hydrogen peroxide concentrations at Riverside, California, in August 1970. Reprinted with permission from Bufalini *et al.*[9]

TABLE 4-17 Percentage of Days for Which Oxidant Data Are Available from CAMP Sites, May-October and June-August[a]

Site	Percentage of Days[b]										
	1964	1965	1966	1967	1968	1969	1970	1971	1972	1973	Average
Chicago, Illinois	60(75)	85(85)	70(80)	75(75)	75(75)	15(30)	90(95)	90(85)	85(85)	65(85)	70(75)
Cincinnati, Ohio	80(80)	90(80)	50(45)	70(55)	35(35)	15(0)	5(0)	85(85)	70(65)	80(80)	55(55)
Denver, Colorado	—	90(90)	75(65)	30(15)	50(30)	15(20)	60(55)	25(1)	55(55)	65(70)	50(40)
Philadelphia, Pennsylvania	85(85)	55(45)	80(80)	70(70)	45(55)	25(20)	25(45)	75(75)	15(15)	25(50)	55(50)
St. Louis, Missouri	90(80)	90(95)	75(60)	80(80)	60(50)	30(30)	30(15)	75(85)	40(60)	85(95)	65(65)
Washington, D.C.	85(80)	80(85)	90(90)	95(100)	75(100)	15(15)	80(90)	95(100)	20(30)	85(80)	70(75)
Average	80(80)	80(80)	75(70)	70(65)	55(55)	20(20)	50(50)	55(70)	50(50)	70(75)	60(60)

[a] Derived from Altshuller.[1]
[b] Rounded to nearest 5%. June-August values in parentheses.

DATA QUALITY

Chapter 6 covers most of the questions influencing data quality. At least three factors enter into the selection of data: the instrumental technique used for measurement, the exposure of the station and the location of the sampling inlet, and the choice of standard calibration method.

Because of interaction with nitric oxide, local decreases in ozone concentrations can occur near large sources, such as power plants or freeways. Consequently, ozone monitoring should not be undertaken near any of these sources if a representative regional ambient concentration is desired. The early portions of the CAMP (Continuous Air Monitoring Program) were evidently focused on the measurement of community exposure to pollutants, inasmuch as monitoring sites were set up in the centers of urban areas. Because of the source interaction referred to above, this often resulted in an underestimate of the ozone problem. It is now known that stations around the perimeter of a central business district or in suburban areas give higher ozone readings because of the absence of local sources and because of the time required for photochemical reactions to result in ozone buildups in the air. Dark reactions in the inlet manifolding of the sampling train can also distort an instrument's ozone measurements. Large-diameter high-velocity flow systems made of low-reactivity materials should be used to avoid this source of data distortion.

The problems with various primary calibration standards are still being resolved. The earlier discussion of DeMore *et al.*[14] indicates the work undertaken by the CARB in comparing oxidant calibration procedures. It was recommended that all oxidant analyzers in the California network be calibrated by a secondary standard consisting of an ultraviolet ozone analyzer. The primary standard recommended is ultraviolet photometry. These recommendations have been adopted by the CARB. Potassium iodide, indicated as a second choice, requires the application of a correction factor of 0.78.

The fraction of valid data days has been summarized by Altshuller[1] for the six CAMP sites. He noted that, in Cincinnati in the summers of 1969 and 1970 and in Denver in 1971, no results were reported for oxidant. At best, for Washington, D.C., and Chicago, 70–75% valid data were obtained; at worst, 40–50% valid data were obtained for summer months in Denver and Cincinnati. The findings are summarized in Table 4-17. Altshuller called attention to the large corrections that must be applied to colorimetric oxidant analyzers for peaks that occur at night late in the fall and in winter. He pointed out that the NO_x response correction becomes a major part of the reading, because the instruments are func-

tioning mostly as analyzers of NO_x. What might be considered a valid data base extends much further back in time for the Los Angeles basin. A high fraction of the data for nearly 20 years is available for that area. Before a final judgment on the validity of the data, however, one must carefully assess the problems of calibration consistency and monitoring station siting. It will be found in most local monitoring systems outside California that the bulk of the valid oxidant data has been obtained since 1970. Thus, in 1975, valid data are available for only a few years, because of lags in data processing and editing.

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5

Models for Predicting Air Quality

Trends in air pollutant concentrations can be predicted with simple empirical models based on atmospheric and laboratory data. Concentrations of nonreactive pollutants from point sources can be predicted *with accuracy well within a factor of 2*; predictions are more likely to be *too high* than *too low*, especially predictions of concentration peaks. Concentrations of reactive pollutants, such as ozone and other photochemical oxidants, can be predicted reasonably well with photochemical-diffusion models when detailed emission, air quality, and meteorologic measurements are available; most such predictions of air pollution in Los Angeles, California, have been accurate to within approximately 50% for ozone. Detailed performance analyses are found elsewhere in this chapter.

Statistical models based on data correlations and on Markov chains are being actively developed and their fidelity evaluated by several research groups. Photochemical-diffusion models based on deterministic equations are also being developed, but because of their complexity will probably be used only as research tools for some time.

As expressed in Equation 4-1,[117] the spatial and temporal distributions of pollutants must be known to assess their damage to receptors. Traditionally, monitoring station measurements are used to estimate the concentrations for entire regions. Statutory mandates, however, require

the prediction of future concentrations under differing emission conditions. More and more refined predictions will be required as the standards become more specific.

An air quality model is a method of relating air quality to emission under specific environmental conditions. There are many types of air quality models, and the purposes of this chapter are to describe models that are available for the analysis of photochemical oxidants and to give some information on how well the models perform.

We must first gain a perspective on present modeling capabilities. The photochemical-diffusion computer programs are aimed at prediction of ozone and nitrogen dioxide concentrations. It must be understood that ozone is used as an indicator or as a surrogate for other oxygenated organic compounds or radicals that may actively cause adverse effects. Most models do not treat aldehydes and oxygenated organic aerosols specifically, although they do address the problems of peroxy-radical and hydrogen peroxide concentrations in some cases.

Because of the dominance of distributed sources over local single sources in the production of photochemical oxidants, point-source models are not discussed here. Related research regarding the measurement of diffusion or the development of atmospheric chemical submodels are not emphasized. Chapter 2 is devoted to the chemical processes that govern atmospheric transformation and removal, and this aspect of the models is not repeated here.

Numerous reviews of air quality models have appeared in the literature in the last several years. Wanta[73] considered the meteorologic role in air pollution, including the effects of vertical temperature structure, topography, windfields, and dispersion mechanisms. He tabulated in detail numerous characteristics of the mathematical air pollution models that existed in 1967. The relationship of meteorology to air pollution was also reviewed in papers by Pack[50] and Neiburger.[45] Theories of diffusion in the lower atmosphere were outlined by Gifford,[18] who stressed the empirical aspects of both differential equation diffusion theory and Gaussian diffusion formulas, traced the historical connections between the two, clarified their relationship to atmospheric turbulence, and reviewed cases of plume formulas to cover special phenomena, such as plume fluctuation, looping, coning, fumigation, and lofting.

Various meetings on urban diffusion models have been held in recent years. One was a symposium[70] sponsored by the Environmental Protection Agency. Its *Proceedings* included studies of Gaussian plume and puff modeling techniques available in 1969. Each paper on a specific model gave some detail of the mathematical assumptions and the types of measurements that were used to test it. Several participants noted a

need to develop finite-difference numerical techniques to handle the nonlinearities of atmospheric reactions. Another review resulted from a series of meetings and working groups related to Project Clean Air, which was carried out by the University of California in 1969 and 1970. One of the task force reports[46] is especially helpful in its review of mathematical simulation modeling up to 1970. The stated purpose of the review was to identify future research needs for California's Project Clean Air. A tabulation summarizes 15 simulation schemes by commenting on each of 9 points of information, including statements of working equations and quantitative aspects of cell size, time resolution, and verification tests. It is gratifying to note that substantial progress has been made on each of the 10 recommendations given for future research.

Another useful collection of papers is the *Proceedings of the Second Meeting of the Expert Panel on Air Pollution Modeling*, sponsored by NATO.[47] The volume contains 15 papers from both the first and second meetings of the panel. Three of the main topics are: air quality modeling projects that were going on in Ankara, Turkey, and Frankfurt, West Germany; applicability of physical models that use hydraulic or aerodynamic replications of flow fields; and problems in atmospheric chemistry of pollutants, with particular emphasis on photochemical transformation. In his review of atmospheric transport processes, Reiter[54] stressed the behavior of the diffusion equations under the combined effects of frictional, buoyant, and Coriolis forces. The distortion of the velocity profile as it approaches the geostrophic wind was discussed. The main approach was theoretical and was based on K theory, but results of Gaussian plume research were also cited. The fluid dynamic limitations of the Gaussian plume formulas were reviewed critically, and extensive references for various special aspects of the formulas were given.

In another review, Hoffert[29] discussed the social motivations for modeling air quality for predictive purposes and elucidated the components of a model. Meteorologic factors were summarized in terms of windfields and atmospheric stability as they are traditionally represented mathematically. The species-balance equation was discussed, and several solutions of the equation for constant-diffusion coefficient and concentrated sources were suggested. Gaussian plume and puff results were related to the problems of developing multiple-source urban-dispersion models. Numerical solutions and box models were then considered. The review concluded with a brief outline of the atmospheric chemical effects that influence the concentration of pollutants by transformation.

Motivated by statutory mandates for environmental evaluation of transportation systems, Darling[8] solicited information from each originator of an air pollution model by a questionnaire advertised in the *Commerce*

Business Daily, a widely circulated publication in the United States. Of the 78 questionnaires sent out, 44 were completed and returned. The questionnaire dealt in some detail with computer programs involved with each model, in addition to the analytic foundations of each approach. Principles, implementation, applications, and validation were discussed in the report. Whenever information was available, there was comparative analysis of the models. An important conclusion of the work was that in 1972 there had been very little performance evaluation of models related to transportation-generated air pollution.

Johnson[30] reviewed EPA programs in air quality simulation modeling in 1972, covering the various policy questions that models can help to answer, summarizing modeling approaches, and outlining advantages and disadvantages of the various techniques. The mission of UNAMAP (*U*ser *N*etwork for *A*pplied *M*odeling of *A*ir *P*ollution) was described as a system that would provide easy user access to the models for practical applications.

The third meeting of NATO's Expert Panel on Air Pollution Modeling[48] updated reports of modeling efforts for Cologne, St. Louis, Milan, Ankara, Frankfurt, Stockholm, Oslo, and Manchester. It also included research papers on model-related meteorologic topics and reviews of various national programs and facilities represented by the participants. The fourth meeting of the panel[49] departed from the format of the others, in that it consisted of five workshop sessions, which covered applications of modeling and users' needs, validations of air quality simulation models, regional air pollution studies, empirical-statistical modeling of air quality, and the question of simplicity versus sophistication in air quality modeling. The *Proceedings* summarized each workshop discussion in narrative form, and it included several prepared papers.

Lamb and co-workers[35] reviewed techniques of diffusion modeling for air quality with relation to transportation-generated pollution. They discussed the theory and structure of models, presented a series of tabulated comparisons, analyzed the function and design of each model, and offered simple diagrams to illustrate the functions and problems of the various techniques. The report was intended to survey a great deal of unpublished material and therefore was important in bringing the earlier surveys up to date (to about mid-1973).

Air quality simulation models for photochemical pollutants were reviewed by Johnson *et al.*[31] for a new edition of *Air Pollution*. Some of the models developed for simulating photochemical smog were reviewed from the viewpoints of module logic and evaluation. The Los Angeles-based developments were outlined, including the format and preprocessing of emission inventory data and meteorologic data. Lumped-param-

eter chemical approaches were described, and smog-chamber kinetics validations were outlined.

One of the purposes of this chapter is to add recent material to that collected in the reviews just described. In contrast with previous reviews, however, this chapter emphasizes the critical evaluation of performance. The sections that follow deal with objectives of models (from research to applied control systems), the elements of schemes for predicting air quality, specific methods of modeling, and the evaluation of prediction techniques.

OBJECTIVES OF MODELS

At the heart of the problem of relating improvements in air quality to reductions in pollutant emission is a reliable method of prediction. Only with such a method can there be rational planning for air pollution control through regulation of transportation, indirect sources, and stationary sources. Decision-makers need it as a tool and must specify it in their regulations. Otherwise, their administration of an air quality plan would be based on sheer guesswork tempered by political negotiation.

Pollutant concentrations are related to sources under specified meteorologic conditions by using what is called an air quality model. Models vary from simple arithmetic exercises to complex computer simulations. There are many paths to the needed answers. Which of the available methods is appropriate depends on the specific problem. Most agencies charged with enforcement of air quality rules have used only the most rudimentary models. In some cases, short deadlines have forced the situation. But the recognition of the social costs of air pollution control has led to deeper interest in scrutinizing the results of air quality models, and it is not likely that casual calculation will be accepted by regulatory agencies—especially if the results (in the form of abatement strategies) involve severe socioeconomic dislocation and large financial outlays.

In examining how well a model meets its objectives, one must ask several questions:

- Who will use the model?
- What questions must the model be able to answer?
- How well is the model adapted to the intended application?
- Has the model been thoroughly tested against an adequate data base for validations?
- How readily accessible is the model with respect to practical calculations and computer implementation?

This section examines various purposes of developing and using air quality models.

Scientific Purposes

In any environmental management plan, it is essential to understand the pertinent quality criteria and their relationship to the variables that can be manipulated directly. The construction of mathematical models imposes the requirement of a logical framework that connects causes and effects, identifies all pertinent variables, and defines their interrelationships. Even if a particular technique never becomes practical, the discipline imposed by the logical structure is valuable in highlighting important relationships.

Methods for predicting air quality were first applied in conjunction with field measurement programs and routine monitoring programs. Such applications have emphasized both the strengths and the weaknesses of various modeling schemes and elucidated the main points of technical understanding that must underlie the establishment of control systems. Considering the responses of models to various inputs, "parametric sensitivity evaluations" carried out in parallel with supporting field programs will continue to be helpful in the development of the understanding on which pollution abatement plans can be based.

Experience has followed an iterative pattern in playing the model exercises against field measurements. Usually, the first indication of the relative importance of variables is seen in bodies of observational data. The next step is to build a model on the basis of either intuition or a deterministic physical equation that reflects the trends seen in the data. The model is then used for the range of conditions in the data base, and uncertainties as to the correctness or completeness of the model become evident. The questions that arise can usually be answered only through further field experimentation. Thus, the models themselves are used in the design of both laboratory and field experiments that will ultimately provide a basis for the improvement of the modeling art.

Regulatory Purposes

Long-term air quality forecasts are implicit in any scheme that is designed to improve the atmospheric environment through specific sanctions on primary pollutant emission. The writing of legislation and regulations is an obvious application of air quality modeling. For example, vehicular emission controls are specified in the U.S. Clean Air Act Amendments of 1970. Originally, a modeling scheme was used to

specify the control that would be required to achieve improvements in air quality. One of the difficulties with such legislation is that the ambient air quality standards are set by an administrative process, whereas the vehicular emission standards are set by the original law. In this case, the extent of control was specified before the establishment of the target reduction of pollution for the future.

A broader regulatory application of air quality modeling is the examination of regional plans for abatement of air pollution. Ideally, each plan should be tested by a reliable model that will tell what the pollutant concentrations will be in the future if emission is reduced as specified by the plan. Therefore, alternative approaches must be evaluated both with respect to their effectiveness in cleaning the air and with respect to their social costs, and cost models and possibly even damage models must be used concurrently with the air quality model. Only in this way can a least-cost abatement strategy be implemented.

The successful application of quantitative predictions to the design of strategies requires full cooperation of the scientific community with the decision-makers. Regulatory and legislative bodies need the most reliable tools available to assess the impact of their decisions. The assessment of environmental impact is an integral part of the process of engineering design, whether the object of the process is a steam generator, a highway, or an airport. As a result of the new requirements, many branches of government—not only the EPA—must be able to predict air quality.

Urban Planning

Human land-use patterns and meteorologic conditions both directly determine the degree of air pollution. Land-use planning includes the design of transportation systems. Air quality models must be able to predict the pollution that will be caused by various patterns of land use. In this application, the predictive scheme is used inversely; i.e., it has to answer the question: Given air quality standards, what patterns of land use are acceptable? Experience has shown that conventional wisdom can be dangerously misleading in regard to the impact of various land-use and transportation plans on air pollution. In the models to evaluate proposed changes, one gains an understanding of the sensitivity of environmental variables to different types of facilities and to where they are placed.

Emerging from analyses like these is a new concept of population holding capacity. Earlier notions of maximal population density were based on availability of space for transportation systems, subsoil conditions related to the support of buildings, availability of off-street park-

ing space, and many other considerations. With the new set of requirements imposed by environmental quality standards, the earlier criteria will be superseded, in effect, by the output of a model that specifies how great a population can occupy a given region, and where and when. In many urban areas, air quality may already be a controlling factor in the determination of this holding capacity. Hence, accurate forecasts are important.

Episode Control

Systems are now being devised that require a real-time system that uses model logic to control emission sources to maintain acceptable air quality for all meteorologic conditions. In the United States, such control systems may be needed for large combustion sources of air pollution. Such systems use meteorologic and power-demand data to produce load schedules by fuel type. Indeed, the concept of computer-aided controls could be used for the control of sources throughout an urban area, as has been proposed in some prefectures in Japan. For real-time warning systems or control systems, the air quality model provides the logic in the feedback loop that links air quality improvement to emission reduction under specific predicted meteorologic conditions. With the coming need for flexibility in fuel logistics, these systems may become more and more prevalent.

The operational logic for an episode control system must have much finer resolution and higher reliability than that for long-term applications. For forecasts covering a decade or two, progress in a cleanup campaign can be closely monitored, and emission controls can be continually adjusted. Episode management, however, is far more difficult. On the one hand, if too many errors are made by the system, unduly high public exposure to harmful pollutants will result. On the other hand, if the episode control system generates too many false alarms, the social costs in industrial operational modification will become so onerous that public support for the system may fade.

Implicit in the success of any episode control system is the ability to predict weather accurately. Conventional applications of air quality models that use a wide variety of meteorologic information require that such details as wind speed and direction and mixing depth be reliably predicted. Some of the statistical-empirical models that are discussed below obviate such a large mass of prediction by selecting a few sensitive measurements and bypassing the detail of deterministic logic. The episode-control application of models clearly illustrates the need to develop a multiplicity of air quality models. For example, a model that finds a natural application in the regulatory or planning process may be very poorly suited to the control of discrete episodes.

ELEMENTS OF AIR QUALITY PREDICTION SCHEMES

Structure of Deterministic Models

All deterministic schemes have some elements in common. The completeness or detail of any of these elements varies greatly from model to model, but a diagrammatic representation of the basic structure will clarify the relationships among the various techniques to be presented. Figure 5-1 shows that three streams of input information enter the preprocessing module, and the background and initial pollutant concentrations may become unimportant if the simulation is run for a long enough period and covers a large enough area. The preprocessing module renders this information useful for the main computational part of the air quality model, transmitting to the main program the various kinds of data shown in Figure 5-1. Depending on the complexity of the logic, one or more of these kinds of data may be unnecessary. Pollutant concentrations constitute the output of any of the models. The form of this output varies in detail, ranging from a single concentration averaged over a long period for an entire region to hourly maps of concentration in three dimensions over the region.

Classification of Types

There are many ways of categorizing air quality models. One differentiation is between statistical and deterministic models. The structure of statistical models is based on the patterns that appear in the extensive measured data. The structure of deterministic models is based on mechanistic principles wherever possible. Most deterministic models contain some degree of empiricism. For example, few models, if any, use turbulent-diffusion formulations that are based on first principles, but rather use measured values of dispersion. The same is true in regard

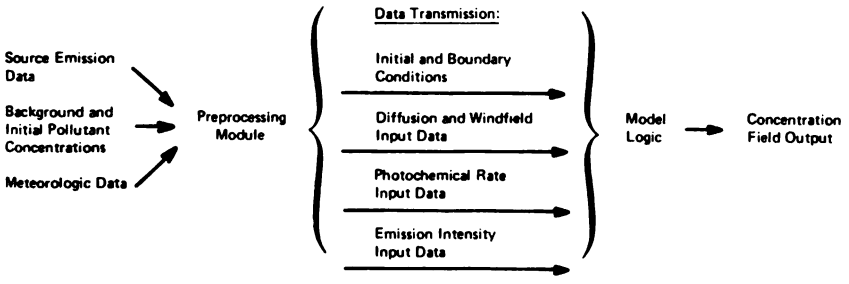

FIGURE 5-1 Diagram of elements of deterministic model.

to the atmospheric chemistry of photochemical formulations. A common caution is that, if too many disposable quantities exist in a deterministic model, it becomes a statistical model in disguise, because all it accomplishes is a version of curve-fitting.

Another classification of model is related to the time and space scales of interest. Ambient air quality standards are stated for measurement averaging periods varying from an hour to a year. However, for computational purposes, it is often necessary to use periods of less than an hour for a typical resolution-cell size in a model. Spatial scales of interest vary from a few tenths of a meter (e.g., for the area immediately adjacent to a roadway) up to hundreds of kilometers (e.g., in simulations that will elucidate urban-rural interactions). Large spatial scales are also warranted when multiday simulations are necessary for even a moderate-sized urban area. Under some climatologic conditions, recirculations can cause interaction of today's pollution with tomorrow's. Typical resolution specifications couple spatial scales with temporal scales. Therefore, the full matrix of time scales and space scales is not needed, because of the dependence of time scales on space scales. Some typical categories by scale are as follows:

- A roadway impact model in the microscale (~0.1 km; ~10 min to 1 h).
- Large-point-source or indirect-source model (~10 km; ~1 h).
- Urban regional scale model (~50 km; ~1 h).
- Urban-rural regional scale model (~300 km; 1 h to 1 week).

The reviews by Johnson[30] and by Seinfeld[65] give helpful guidelines in the classification of models by space and time scale.

Perhaps the most fundamental way to classify models is by methodology. Examples of specific methods are discussed later, but a brief summary is appropriate here. Great emphasis on historical data as embodied in empirical formulas is found in the methodology of statistical and rollback models.[2,4-6,20,42,43,53,61,62,71] Rollback models embody the principle that reductions in emission are reflected by improvements in air quality, as may be shown by a straight line, a curved line, or a complex surface that expresses some proportionality relationship. These models work best if the geographic and temporal distribution of the emitters is not changed. The straight-line versions can apply only to pollutants that do not undergo chemical transformations in the atmosphere. An example of a proportional linear rollback is a rapidly instituted retrofit control of carbon monoxide emission imposed on an entire vehicle population. Corrections for irregularities in distribution caused by nonuniform growth,

however, have been suggested.[5,6] The great advantages of the linear rollback model are its use of aggregated emission statistics and its mathematical simplicity. Suggested improvements of the rollback to account for chemical change have been based on the use of air quality data or smog-chamber data.

Dispersion models may take the form of a simple box model[19,21-23,37] or a Gaussian formula. Dispersion models that use superposition of Gaussian plumes improve the rollback approach for nonreacting species by accounting for geographic distribution of emission sources. Emission generally consists of an array of area elements or effective point sources, each characterized by an output intensity. The plumes from the several sources contribute in an additive manner to the pollution at any downwind field point. Summation of the contributions over all sources constitutes the superposition aspect of the approach. Computationally, this is convenient, because it allows sequential consideration of each source element, but the same feature renders the method inapplicable to multireacting systems undergoing chemical transformations. The Gaussian puff approach removes the limitation of steady-state assumptions from the calculation and treats discrete emissions as puffs that spread according to the Gaussian law.

Special balance-equation models combine the effects of diffusion, advection, and some chemistry and normally may use finite-difference techniques for the solution.[10,11,13-15,17,32,36,39,41,72,74] Currently, these are the most elaborate simulations applied to air quality analysis. Both time and space are subdivided into cells in these models, so that the cumulative effects of emission, transport, and (when chemistry is included) reactions are simultaneously accounted for. The potential danger in using this kind of model is that the available data base will be outstripped by mathematical detail. When that happens, the large volumes of data output can delude the user into a high degree of confidence when, in reality, only very sparse data bases are available for verifying the model. The advantage of finite-difference models is the potentially greater fidelity where greater detail exists in the input data base and in the validation testing.

Emission Description

A quantitative expression for the introduction of primary pollutants into the atmosphere is basic to any air quality model. Emission is most generally described as a geographic, temporal, and chemical distribution that requires a rather massive array of numbers. Some simple models need only the aggregated numbers found in ordinary tabulations

of emission inventory (e.g., kilograms of carbon monoxide, NO_x, etc., per day in a large air quality control region). A geographic breakdown of emission is needed, however, if we are to determine key "hot spots" of high pollution to which ambient air quality standards are directed. This spatial distribution of emission is often given on a grid or on a traffic-flow network system. Similarly, the hourly variation in emission must be known if we are to predict the peak hourly averages that are often mentioned in the standards.

For purposes of characterization, emission sources are generally divided broadly into stationary and mobile or transportation sources. Stationary sources are further divided into point and area emitters. Typical point sources must include petroleum refineries and electric power plants. Commercial solvent emission and gasoline marketing emission may generally be represented as area sources. A third category has been defined recently—indirect sources—that takes into account hybrid sources like sports arenas and shopping centers. These have fixed locations, but the traffic that is generated by or attracted to such a facility constitutes the source of emission that is combined with the emission of the facility itself.

Gathering emission data and putting them in condition for use in air quality models are often among the most tedious and time-consuming parts of their handling. For this reason, the preprocessing module is identified as a separate automatic operation in procedures outlined in Figure 5-1.

Transport Formulation

The movement of pollution from one place to another and its dilution by atmospheric mixing are both based on the meteorologic conditions of the airshed in question. Air flow patterns are in turn based on the interaction of the large-scale flow with the topographic details of the region, with regard to altitude variation, roughness of surface, and heating characteristics. The part of the flow-surface interaction that influences the degree of pollution must be taken into account in a model. Rather than computing the local weather as part of the prediction, most models use meteorologic measurements to construct atmospheric flow fields that represent, on a local scale, the driving factor of the transport mechanisms.

With the velocity field and the atmospheric dispersion mechanisms given, the basic equation is that for mass balance for an individual species, which can be expressed in the following form:

$$\frac{\partial c_j}{\partial t} + \sum_{l=1}^{3} \frac{\partial (c_j v_{jl})}{\partial x_l} = w_j + S_j \qquad (j = 1, 2, 3, \ldots, s), \tag{5-1}$$

where

c_j = mass concentration of jth species,
t = time,
l = an index referring to each coordinate direction,
v_{jl} = velocity of jth species in lth direction,
x_l = distance in lth direction,
w_j = net molar production rate of jth species per unit volume by chemical reaction,
S_j = source strength for emitters of jth species at some location above the ground, and
s = number of species.

The derivation of the mixture-balance laws has been given by Chapman and Cowling[7] for a binary mixture. Its generalization to multicomponent mixtures, as in Equation 5-1, uses a determination of the invariance of the Boltzmann equation. This development has been detailed by Hirschfelder *et al.*[25-28] These derivations were summarized in the notes of Theodore von Kármán's Sorbonne lectures given in 1951–1952, and the results of his summaries were stated in Penner's monograph.[52] For turbulent flow, the species-balance equation can be represented in the Boussinesq approximation as:

$$\frac{\partial \tilde{c}_j}{\partial t} + \sum_{l=1}^{3} \tilde{v}_l \frac{\partial \tilde{c}_j}{\partial x_l} = \sum_{l=1}^{3} \frac{\partial}{\partial x_l} \left(K_{ll} \frac{\partial \tilde{c}_j}{\partial x_l} \right) + \bar{w}_j + S_j \quad (j = 1, 2, 3, \ldots, s), \tag{5-2}$$

where overbars denote time-averaged values and K_{ll} is the eddy diffusivity that relates the flux of a diffusing species to the species concentration gradient, both acting in the same direction. The Boussinesq approximation is best applied to large-scale turbulent flow or motions typifying the problems in urban and regional areas.

The development of this form of the equation was given by Bird *et al.*[3] The species-mass-conservation models use numerical integrations of various forms of these equations.

The Gaussian plume formulations, however, use closed-form solutions of the turbulent version of Equation 5-1 subject to simplifying assumptions. Although these are not treated further here, their description is included for comparative purposes. The assumptions are: reflection of species off the ground (that is, zero flux at the ground), constant value of vertical diffusion coefficient, and large distance from the source compared with lateral dimensions. This Gaussian solution to Equation 5-1 is obtained under the assumption that chemical transformation source and sink terms are all zero. In some cases, an exponential decay factor is applied for reactions that obey first-order kinetics. A typical solution (with the time-decay factor) is:

$$\tilde{c}_j(x_1, x_2, x_3) = \frac{Q_j/\rho}{2\pi\sigma_2\sigma_3\bar{v}_1}\left\{\exp\left[-\frac{x_1}{v_1\tau} - \frac{1}{2}\left(\frac{x_2}{\sigma_2}\right)^2 - \frac{1}{2}\left(\frac{x_3 - h}{\sigma_3}\right)^2\right] + \exp\left[-\frac{1}{2}\left(\frac{x_3 + h}{\sigma_3}\right)^2\right]\right\}, \quad (5\text{-}3)$$

where

Q_j = emission rate of jth species,
ρ = air density,
h = source height,
τ = chemical decay time for first-order reaction, and
$\sigma_i = (2K_{ii}x_1/\bar{v}_1)^{1/2}$.

The three coordinate directions—1, 2, and 3—are taken to be downwind, crosswind, and vertical. The origin is fixed to the ground at the location of the source.

Physiochemical Transformation Simulation

The atmospheric chemical processes undergone by most pollutants are not readily describable by first-order kinetics. Hence, the simple Gaussian plume solution in Equation 5-3 is inapplicable in most cases where physiochemical transformations significantly alter concentrations on a time scale or space scale appropriate to an urban airshed.

The general case must be solved by numerical integration with finite-difference schemes or other approaches to the solution of Equation 5-2 for the species of interest. As written, this equation requires that the partial-differential equation be solved for each species in the reactive

mixture. In reality, however, the number of partial-differential equations that must be solved can be reduced by imposing stationary-state assumptions. That is, some species are so reactive that their rate of production nearly equals their rate of depletion, and these rates may be effectively equated. This being the case, algebraic expressions are used to relate the stationary-state species concentration to all the reactive compounds and radicals that are responsible for its production and removal. For multicomponent mixtures, these are simultaneous nonlinear algebraic equations that must be solved by numerical techniques, such as Newton's method for finding roots of equations.

A generalized notation for representing chemical reactions is:

$$\sum_{j=1}^{s} \nu_{ij} A_j \xrightarrow{k_i} \sum_{j=1}^{s} \nu'_{ij} A_j \qquad (i = 1, 2, 3, \ldots, r) \qquad (5\text{-}4)$$

where, for the jth species participating in the ith reaction,

ν_{ij} = reactant stoichiometric coefficient,
ν'_{ij} = product stoichiometric coefficient,
A_j = chemical formula of jth species,
r = number of reactions, and
k_i = rate coefficient of ith reaction.

The generality of the index notation permits any specification of a chemical mechanism that the user desires. With the notation, the source term becomes:

$$w_j = (\mu_j/\mu) \sum_{i=1}^{r} \beta_{ij} k_j \prod_{\lambda=1}^{s} (\mu \bar{c}_\lambda/\mu_\lambda)^{\nu_{i\lambda}} \qquad (j = 1, 2, 3, \ldots, s) \qquad (5\text{-}5)$$

where

μ_j = molecular weight of jth species,
μ = molecular weight of mixture (air),
$\beta_{ij} = \nu'_{ij}$, and
λ = dummy species index.

Because of fluctuations in turbulent flows, Equation 5-5 is only an approximation. Research is under way to correct this deficiency. The use of Equation 5-5 instead of specific terms retains a degree of generality in the computer program that greatly simplifies alterations of the mechanism.

For stationary-state species, the partial-differential equation (5-2) is

replaced by the source term in Equation 5-5 set equal to zero. This provides the algebraic means for solving for individual species concentrations. These algebraic formulas are carried along in the calculation as constraints on the remaining partial-differential equations.

Discussions of specific chemical mechanisms are found in Chapter 2, and examples of working mechanisms that have been used in models can be found in Friedlander and Seinfeld, [17] Eschenroeder and Martinez, [13,14] Wayne *et al.*,[74] and Reynolds *et al.* [55,57]

EXAMPLES OF SPECIFIC METHODS

Rollback, Statistical, and Box Models

Rollback Both linear rollback and modified rollback models were used by Barth[2] to examine federal motor-vehicle goals for standards governing carbon monoxide, hydrocarbon, and oxides of nitrogen. The linear rollback principle was suggested and applied to these primary pollutants:

$$R = \frac{(GF)(PAQ) - (DAQ)}{(GF)(PAQ) - B}, \tag{5-6}$$

where R is the fractional reduction required, GF is the growth factor, PAQ is present air quality, DAQ is desired air quality, and B is background concentration. Linear rollback and related models have been heavily emphasized in the regulatory approaches taken to date.

Linear rollback involved direct application of Equations 5-6 to carbon monoxide, hydrocarbon, and nitrogen oxide emission. Modified rollback implies reading from a graph of peak oxidant versus hydrocarbon concentration the needed hydrocarbon reduction and then using Equation 5-6 to relate hydrocarbon emission to atmospheric concentrations. The reduction in nitric oxide emission was chosen to achieve the nitrogen dioxide ambient air quality standard, and the reduction in hydrocarbon was tailored to achieve the oxidant standard via the Schuck *et al.* diagram[61] following the modified rollback scheme. This diagram gives envelope curves of the maximal 1-h oxidant observed versus the 6:00–9:00 a.m. average hydrocarbon concentration in five cities. To avoid the inaccuracies of the linear rollback scheme as applied directly to oxidant, Schuck and Papetti[62] have refined the nonlinear or modified rollback scheme. First, they modified the nonlinear rollback method to include 1968–1971 air quality data from the Los Angeles basin. The new approach to modified rollback used an eight-station concentration aver-

age in response to the objection that the hydrocarbon concentrations that caused the oxidant buildup occur somewhere other than the oxidant station location. That is, winds will generally sweep the air with the morning hydrocarbon contamination to a location some distance away. Therefore, averaging eight stations tends to distribute this inaccuracy and, it is hoped, cancel it. The upper-limit curve is stated to be in agreement with regression analyses done at Chevron Research.[43] Because of NO_x inhibition effects, decreasing hydrocarbon emission faster than nitric oxide emission will reduce ozone even more than is predicted by the curve. Working against this bonus, however, is the effect of growth that must be used to correct the percentage hydrocarbon reductions read directly from the curve. The original rollback formula, of course, included gross growth factors. The nonlinear rollback approach has serious deficiencies and should not be used for planning purposes.

Another approach that uses a linear rollback relating primary pollutants to ambient air quality was described by Hamming *et al.*,[20] who presented a series of graphs to show photochemical smog effects in terms of primary pollutant concentrations. Contours of constant oxidant, eye irritation, peak nitrogen dioxide concentration, and time-to-nitrogen dioxide peak are plotted on planes of NO_x versus hydrocarbon. The data for the graphs were based on interpretation of the Los Angeles County Air Pollution Control District experimental-chamber measurements, as well as those of Korth *et al.*[34] and of Dimitriades.[9] It was cautioned that the chamber results cannot be transferred to the atmosphere on a one-for-one basis, because of wall effects, dilution effects, and other experimental artifacts. The 6:00–9:00 a.m. concentrations of hydrocarbon and NO_x were traced out on graphs. Projections of hydrocarbon and NO_x concentrations were made for future years, but the method of carrying out these projects was not described. Presumably, linear rollback was used to relate emission to precursor concentrations. Hence, in theory, this procedure is one step more nearly complete than the modified rollback used in the oxidant envelope curves. It reflects the influence of the hydrocarbon:nitric oxide ratio on the four photochemical smog effects contoured on the plots. This would have been a direct extension of the modified rollback method, if atmospheric data had been used: however, the authors cited atmospheric trends that tended to support the conclusions that they drew from chamber-data plots. The lack of any explicit means of considering mixture-ratio effects is a major drawback in the use of modified rollback for the present oxidant control strategies, which impose more rapid reduction in hydrocarbon than in NO_x emission. The technique of Hamming *et al.* holds out the possibility of eliminating this deficiency.

Chang and Weinstock[5] represented the general rollback formula as

integrals over time and space of a population of emitters weighted with influence functions. The influence functions (or source-receptor interaction functions) contain the elements of meteorologic and geometric effects. The analysis was restricted to carbon monoxide or any other pollutant that may be nonreactive. The role of an inhomogeneously distributed growth factor was discussed, and a method of correcting rollback for this effect with a diffusion model to obtain the source-receptor interaction functions was proposed. This followed very closely a method introduced earlier by the same authors.[6] The rollback formula was corrected for the nonuniformity of growth with the diffusion model and contrasted with the usual straight tonnage approach. The result was a lessening of the requirement for percentage reductions in emission to achieve a specific air quality goal. For cases with regularity in the forms of the source-receptor interaction function, there appears to be a good chance to make general use of this modified rollback approach.

Statistical Statistical relationships between air quality measurements and meteorologic variables are also used for calculating future air quality. It is important to note, however, that the statistical relationships are usually related to a fixed-emission-source distribution pattern or one that changes with time in some regular manner. Peterson[53] analyzed 24-h averaged sulfur dioxide concentrations at 40 sites in St. Louis for the winter of 1964–1965. Assuming some linear relationships, he isolated three basic patterns that accounted for most of the variance in the observations. The method of empirical orthogonal functions was used to get regression equations that related pollutant patterns to meteorologic variables. Because this approach performed better than a diffusion model, it appeared to be useful for forecasting, although it contains no explicit relationship to source emission. It might be possible to add an adaptive logarithm to a model like this one and use continuously updated data to reflect changing emission patterns. With meteorologic forecasts, it could then be useful for short-term pollution forecasts, but it would not be applicable to the long-range study of control strategies.

Forecasting formulas for pollutant concentrations at the downtown Los Angeles monitoring station were developed by Merz *et al.*,[43] who used a time series analysis of the monitoring data. The logarithm of oxidant was fit to the logarithms of hydrocarbon and nitric oxide concentrations up to second-order terms in the logarithm for each of the dependent variables. The formula obtained was based on 624 data points for eight 3-month periods and yielded a correlation coefficient of 0.55 with a standard error of estimate for the oxidant concentration logarithm (base 10) of 0.5. The diagram was constructed to provide a graphic

working basis for the formula derived. This model relates pollutants with one another, but does not relate emission to air quality and therefore cannot be used for planning emission control strategies.

Trijonis[71] determined statistical empirical relationships for primary and secondary pollutants. The primary-pollutant formula assumed linear rollback for each concentration along the frequency distribution. This was based on the usual supposition that the pollutant is inert and that changes in emission are proportional in space and time. The method was applied to nitrogen dioxide for central Los Angeles on the basis of NO_x emission projections. Another relationship was concerned with photochemical secondary pollutants, specifically oxidant. It followed the assumption that the probability of exceeding a prescribed concentration depends on the extent of emission of primary pollutants that react to form oxidant. The specific oxidant-precursor relationship was implicit in a series of probability curves that depended on morning NO_x concentration in central Los Angeles. Curves were developed for various constant concentrations of morning hydrocarbon (adjusted for methane). The relationships were derived from 5 years of monitoring data. This approach constitutes another method of accounting for the influence of hydrocarbon:NO_x ratios on smog predictions.

Correlations of ozone concentrations with solar radiation, wind speed, and temperature data were prepared by Bruntz *et al.*[4] for a monitoring station at Welfare Island in New York City. Insights into these correlations were gained by means of a "weathervane plot," which displays ozone versus solar radiation with circular symbols whose diameters are proportional to temperature. Lines on each circle were applied to represent wind speed and direction vectorially. An equation of ozone proportional to (solar radiation)$^{\theta_1}$ × (wind speed)$^{\theta_2}$ × (temperature)$^{\theta_3}$ is suggested by an examination of the graph. Interesting correlations emerge from this work, but no relationship to emissions is provided.

McCollister and Wilson[42] proposed two time series models based on forecasting some future event by applying a linear formula to a past event. One model uses today's maximal value of peak carbon monoxide or oxidant to predict tomorrow's peak value. The other model uses data averaged over each hour today to predict concentrations for corresponding hours tomorrow. Each model is evaluated by dividing the mean absolute error by the mean value for days in 1972 using coefficients derived from years before 1972. Evaluation results are described later. Again, nothing in this model explicitly shows how emission affects air quality.

Box The box model is one of the simplest forms of solutions of Equation

5-2 that appear in air quality simulation techniques. It assumes that the air bounded by the ground and the mixing height is uniform. It further considers that the source intensity of pollution emanating from the ground is constant and that the wind speed is constant. The consequence of these assumptions is a simple formula that states that the ambient concentration of some pollutant is directly proportional to the source emission rates and inversely proportional to the wind speed. The constant of proportionality is determined from mixing height or is derived empirically. One form of this type of model was suggested by Hanna.[22] He assumed the constant of proportionality to be taken as the width of the region divided by the average mixing depth appropriate to the scale of the region. This constant is approximately 225 for many cities. Gifford and Hanna[19] conducted tests of the simple model for particulate matter and sulfur dioxide predictions for annual or seasonal averages versus diffusion-model predictions. Hanna[21] went on to apply the simple dispersion model to the analysis of chemically reactive pollutants. This required that each reaction achieve a steady state within the space and time scale of the airshed of interest. It was concluded that chemical concentrations indicate the lack of steady state for nearly calm conditions of low mixing depth, but that the chemistry does not significantly interfere with the use of a simple model for sunny and windy conditions. For calculational purposes in the example taken for Los Angeles, the reactive hydrocarbon emission was assumed equal to the "published propylene emission." Gifford and Hanna stated that "detailed urban diffusion models developed so far have the property that they generate more pollution variability than is actually observed to occur. This seems to us to be a strong argument for the use of simpler models." "Variability" was undefined, and the degree of variability that is generated by the simpler model was not stated.

After applying the simple model to chemically reactive pollutants,[21] Lamb and Seinfeld[37] disagreed sharply with the contention that the simple dispersion model proposed by Hanna could be applied to the photochemical smog problem. They argued that the rate of change depends on more variables than concentration alone, that spatial variations cannot be neglected, that steady-state conditions are highly unlikely, that the height of the pollution layer does indeed extend to the edge of the mixing layer, and that more than local sources influence ground-level pollution concentrations.

In reply to these criticisms, Hanna[23] addressed the objections point by point. He stated that the use of regionally averaged variables is a necessary first step and has no special limitations. He asserted that the simple model formulation does not assume steady-state conditions, but

that such conditions often occur. He restated the belief that local sources are the chief influence on ground concentrations. The number of chemical parameters available with the simple model depended on the complexity of the mechanism and was therefore considered to be arbitrarily complex. It was further contended that the reason that there are few restrictive assumptions listed for the simple model is that it has few limitations. Further comparisons with more complex models were drawn on the grounds that the correlation coefficients for observed versus predicted data were comparable or better with the simple model.

On the basis of these discussions, it does not appear that the simple model is applicable to chemically reactive pollutants.

Finite-Difference Simulations

Correct modeling of variable diffusivity, time-dependent emission sources, nonlinear chemical reactions, and removal processes necessitates numerical integrations of the species-mass-balance equations. Because of limitations of dispersion data, emission data, or chemical rate data, this approach to the modeling of air pollution may not necessarily ensure higher fidelity, but it does hold out the possibility of the incorporation of more of these details as they become known.

An early analog-computer study of the solution to the species-mass-balance equations was made by Karplus *et al.*[32] This work consisted of a one-dimensional time-dependent diffusion equation with chemical source terms representing a multicomponent atmospheric kinetic system. An electronic analog computer was used, with one integrator at each node between space cells to handle the combined effects of mass transfer and chemical reaction. Results were obtained for a simple mechanism, but no tests of validity were made. Ulbrich[72] also used a series of boxes that were coupled in a model of the species-mass-balance equations. He integrated a data-management system with a model system for real-time prediction and control. A long-term cost-minimizing strategic command and control system was formulated. Adaptive features were built into this control system. The system model conceived for Los Angeles was a series of seven boxes, each covering 288 square miles (746 km^2). Each box was assumed to be well mixed and advectively coupled to its neighbors. A smog delay time of 1.5 h was set into the system. The only emission that was considered was that of nitric oxide.

Similarity solutions of the species-mass-balance equations were assumed by Friedlander and Seinfeld[17] for a simple photochemical-smog reaction scheme. (This scheme assumed a steady-state condition for ozone.) Demonstration runs were shown for parametric variations in the

system of ordinary differential equations that emerged from the partial-differential equations. Analytic solutions yielded atmospheric reaction criteria that were usable for correlating smog variables.

Pollutants emitted by various sources entered an air parcel moving with the wind in the model proposed by Eschenroeder and Martinez.[13,14] Finite-difference solutions to the species-mass-balance equations described the pollutant chemical kinetics and the upward spread through a series of vertical cells. The initial chemical mechanism consisted of 7 species participating in 13 reactions based on smog-chamber observations. Atmospheric dispersion data from the literature were introduced to provide vertical-diffusion coefficients. Initial validity tests were conducted for a static air mass over central Los Angeles on October 23, 1968, and during an episode late in 1968 while a special mobile laboratory was set up by Scott Research Laboratories.[63,64] Curves were plotted to illustrate sensitivity to rate and emission values, and the feasibility of this prediction technique was demonstrated. Some problems of the future were ultimately identified by this work,[15] and the method developed has been applied to several environmental impact studies (see, for example, Wayne *et al.*[74]).

Concern is sometimes expressed regarding the role of surface reactions on aerosol as an interference in the buildup of photochemically produced ozone. An upper-limit analysis was carried out by Eschenroeder, Martinez, and Nordsieck[15] to assess the effect of 200-$\mu g/m^3$ loading of 0.5-μm-diameter particles. Ratios of surface-reaction rates to gas-phase rates for 100% surface reaction efficiencies were computed. Early in the day, nitric oxide and nitrogen dioxide surface-reaction rates can be a few hundred times the gas-phase reaction rate, under the assumption that each molecular collision with the particle surface constitutes a successful reaction event. In the middle of the day, nitrogen dioxide and ozone surface-reaction rates in this limit are 10–50 times the corresponding gas-phase reaction rates, again assuming 100% efficiency. The efficiencies of surface reactions are more nearly in the 10^{-6}–10^{-4} region. Therefore, the high upper-limit values must be reduced considerably to reflect realistic conditions. It is therefore likely that the ozone buildup is affected little by aerosol surface reactions; however, the formation of nitrate in the aerosol, which is observed to take place in the morning, may be marginally important, compared with the gas-phase reaction.

Another Lagrangian photochemical model was developed by Wayne *et al.*[74] This photochemical model uses a moving "cell" or "air mass" that follows an air trajectory either from a specified source or to a specified receptor. The emission input from the ground-based sources is ex-

pressed as a time-dependent influx of primary pollutants. Diffusive spread need not be computed in this model, because the air in the cell is assumed to be homogeneously mixed at all times. This brings about a tremendous advantage in reduced computing time. The chemical mechanism represents reactive hydrocarbons as propylene and lumps others into a generic dummy hydrocarbon. Reactivity is handled by specifying the mix of propylene and generic hydrocarbon. Performance evaluation results were reported for September 30, 1969, on the basis of arbitrarily assigned initial values for hydrocarbon concentrations and neglecting stationary emission of NO_x.

Mahoney and Egan[11,39] developed a two-dimensional time-dependent diffusive and advective model that neglects vertical velocity. Chemical reaction is also neglected. The source term is an effective volume source in the bottom grid mesh cell of the calculation. The authors discussed the pseudodiffusion errors that arise with the large grid spacing that is appropriate to urban-scale calculations. They pointed out that this error is orders of magnitude larger than natural diffusion. A puff example from a volume source was presented in the paper. The scheme proposed avoids pseudodiffusion by using moments of the concentration distribution in the governing equations. The concentration profile was reconstructed by using the computed first and second moments. Egan and Mahoney[10] applied the model to estimate ground concentrations under different meteorologic conditions. Velocity profiles and vertical-diffusivity profiles were introduced on the basis of various stability conditions. Two-dimensional time-dependent solutions with variable advective velocity were obtained. Test cases were presented, including the effects of wind-velocity vector changes with height and the effects of this velocity field distortion on the dispersion of air pollutants. Elevated inversions and time-dependent mixing heights were also investigated. The height variation of the velocity field was shown to be important under stable conditions. Although this model does not treat air chemistry, it can resolve subgrid scale elements because of the moment method used.

A solution to the species-balance equation was generalized by Lamb and Neiburger[36] to allow for the space and time distribution of pollutant emission, diffusion coefficients, and windfield. Pollutant removal at the ground and leakage through the upper boundary were also allowed for in this model. A transformation to a Lagrangian coordinate system was made, and the model was adapted to various source emission configurations. It was tested for carbon monoxide concentrations in the Los Angeles basin for September 23, 1966. It considered vehicular sources based on freeway and surface-street traffic counts. The authors plotted

observed versus predicted concentrations for four stations and found that the morning "peak" in concentration was exaggerated by the model, but that the midday "valley" was underestimated.

Seinfeld *et al.*[66] and Reynolds *et al.*[57] discussed theoretical aspects of urban air pollution modeling in terms of the species-mass-balance equations cast into a problem requiring specification of initial and boundary values of field quantities. Restrictions of K theory in turbulence approximations were reviewed. The vertical coordinate system was mapped between the ground and the inversion base by a linear stretching transformation. The methods were detailed for interpolating discrete data on winds and vertical stability to obtain field values needed for the calculations. The kinetic mechanism that uses lumped parameters for hydrocarbon change was outlined. Eulerian difference equations were integrated numerically, and a method of fractional steps was described. Explicit differencing was used for horizontal coordinates, and implicit differencing for the vertical terms of the equations. In a later paper in the series, Roth *et al.*[60] gave a detailed description of an emission model and an inventory for the Los Angeles basin. Automotive emission was discussed with a breakdown into surface-street and freeway categories. Average-trip-speed correction factors were allowed for, and cold-start corrections for automotive emission factors were introduced. Spatial distributions of daily vehicle miles traveled were laid out on a 2-mile grid for the Los Angeles basin. An overall temporal distribution was adopted, and deviations from this at various locations were shown to be small. Ground and flight operations of aircraft were discussed, but only ground emission was included in the actual application of the model. Reactivity of hydrocarbon from aircraft was considered to be the same as that from automobiles. Stationary sources were categorized as power plants, refineries, and distributed sources. This paper serves as a prototype for future source-emission inventory processes that are intended to supply modelers with input data.

Reynolds *et al.*[55] described the process of evaluation of the airshed photochemical model. Kinetic mechanisms were checked against smog-chamber tests, yielding branching factors and rate parameters for the simplified lumped-parameter scheme. A microscale model was established to correct for local effects around monitoring station sites. An evaluation procedure involved preparing data, preparing initial and boundary conditions, checking for agreement with carbon monoxide data, and testing computed versus observed values for reactive pollutants. The results agreed rather well with observations, but no statistical performance measure was used at this stage of the work.

Sklarew *et al.*[69] used a particle-in-cell K theory (PICK) approach to

calculate atmospheric diffusion and reaction. The method was applied to carbon monoxide pollution in Los Angeles and predicted daily averaged concentrations reportedly within 20% of measured averages for 12 monitoring stations. Photochemical smog simulation was demonstrated, but the results were hampered by the use of the early prototype of lumped-parameter chemical mechanisms,[14] which did not adequately represent the influence of differing hydrocarbon:NO_x ratios. Also, the carbon monoxide results required inordinately high carbon monoxide emission by the Pacific Ocean.

Knox[33] summarized recent developments at the Lawrence Livermore Laboratory. The computation using the Lagrangian large-cloud dispersion model was shown to agree moderately well with the measurement of gross beta-particle activity in the cloud of a nuclear test burst. This is a model for calculating the time-dependent airborne concentration, surface-air concentration, and dry and/or wet deposition of pollutants from large clouds. A three-dimensional atmospheric-diffusion particle-in-cell code was described in which marker particles are traced through an Eulerian grid that was distorted to fit topography. A grid followed the cloud center to minimize the number of cells and to minimize pseudo-diffusion. Mass-consistent wind-field modeling was also discussed. This form of modeling considers the confining effects of an elevated inversion and the variable topography on the surface wind measurements. A multi-box regional air pollution model was run for the San Francisco Bay area for carbon monoxide, and the calculated surface concentrations were approximately 20–50% above the observed concentrations. The frequency distribution predicted for the concentrations, however, paralleled closely the observed frequency distributions.

Gradient diffusion was assumed in the species-mass-conservation model of Shir and Shieh.[67] Integration was carried out in the space between the ground and the mixing height with zero fluxes assumed at each boundary. A first-order decay of sulfur dioxide was the only chemical reaction, and it was suggested that this reaction is important only under low wind speed. Finite-difference numerical solutions for sulfur dioxide in the St. Louis, Missouri, area were obtained with a second-order central finite-difference scheme for horizontal terms and the Crank–Nicolson technique for the vertical-diffusion terms. The three-dimensional grid had 16,800 points on a 30 × 40 × 14 mesh.

PERFORMANCE OF PREDICTION TECHNIQUES

Before we examine the performance of various models for predicting ambient air quality, it is important to review the criteria for selecting

a particular model. In this selection process, it is essential to consider the following factors:

- The precision required for forecasting air quality impact.
- The conditions of high concentrations of air pollutants in the selected region.
- The severity of vehicle-generated pollution relative to that from other sources.
- The availability of meteorologic and air quality data for the selected region.
- The availability of geographic and temporal distributions of emission sources.
- The effect of stack heights associated with large point sources on regional air quality.
- The relative influences of spatial emission distribution, time dependence, and chemical reactions on regional pollution patterns.
- The time and money available for modeling.

Many standard statistical tests are available to evaluate the performance of models against observations. It should be pointed out, however, that graphic checks on the performance of models are also necessary. Anscombe[1] has brought this out strikingly with specific numerical examples. He showed that pure regression analysis can be extremely misleading and demonstrated what can happen in purely numerical analysis of data. In his paper, four artificial data sets of x and y values were given; they are reproduced in Table 5-1. Suppose that x is an observed pollutant concentration, and y is a concentration computed from a model. If standard statistical techniques are used to analyze these four sets, it can be shown that they possess identical values for the following calculations:

- Number of observations: 11
- Mean of the x's: 9.0
- Mean of the y's: 7.5
- Equation of the regression line: $y = 3 + 0.5x$
- Sum of squares of $x - \bar{x}$: 110.0
- Estimated standard error of slope of regression line: 0.118
- Multiple correlation coefficient: 0.667

Figures 5-2 through 5-5 show the great contrasts in the actual data that are revealed by a graphic presentation, but are totally concealed in the above results of standard analytic procedures. Figure 5-2 shows a typical scatter plot, but the other three reveal regularities that may be

TABLE 5-1 Anscombe's Sample Data Sets[1]

Set 1		*Set 2*		*Set 3*		*Set 4*	
x^a	y^b	x	y	x	y	x	y
10.0	8.04	10.0	9.14	10.0	7.46	8.0	6.58
8.0	6.95	8.0	8.14	8.0	6.77	8.0	5.76
13.0	7.58	13.0	8.74	13.0	12.74	8.0	7.71
9.0	8.81	9.0	8.77	9.0	7.11	8.0	8.84
11.0	8.33	11.0	9.26	11.0	7.81	8.0	8.47
14.0	9.96	14.0	8.10	14.0	8.84	8.0	7.04
6.0	7.24	6.0	6.13	6.0	6.08	8.0	5.25
4.0	4.26	4.0	3.10	4.0	5.39	19.0	12.50
12.0	10.84	12.0	9.13	12.0	8.15	8.0	5.56
7.0	4.82	7.0	7.26	7.0	6.42	8.0	7.91
5.0	5.68	5.0	4.74	5.0	5.73	8.0	6.89

[a] x = Observed pollutant concentration.
[b] y = Model-computed concentration.

missed by linear regression analysis—a smooth curve, a different straight line with an outlier, and x values all the same except for an outlier.

The meaning of this example for model performance evaluation is significant: if measures are confined to the usual statistical tests, a great deal of information can be lost. The loss of information tends to perpetuate biases in the calculations, which might otherwise be elimi-

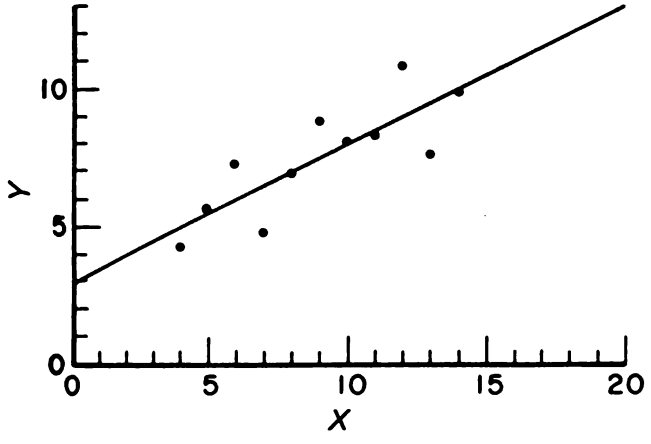

FIGURE 5-2 Graph of data in Anscombe's sample set 1[1] (see Table 5-1).

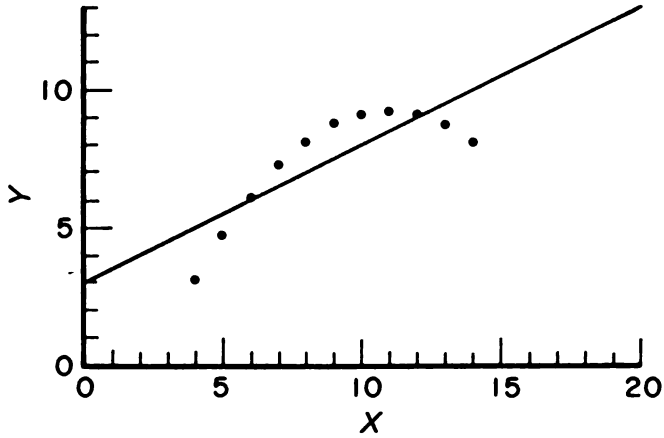

FIGURE 5-3 Graph of data in Anscombe's sample at 2^1 (see Table 5-1).

nated. The message is clear. One should beware of correlation analysis alone. Questions of data regularity that can be resolved by graphic presentations must be pursued, as well as those which pertain to regression analysis alone.

Rollback-, Statistical-, and Box-Model Performance

Eschenroeder[12] compared linear *rollback* results with photochemical-diffusion model results in assessing the effects of various stages of the

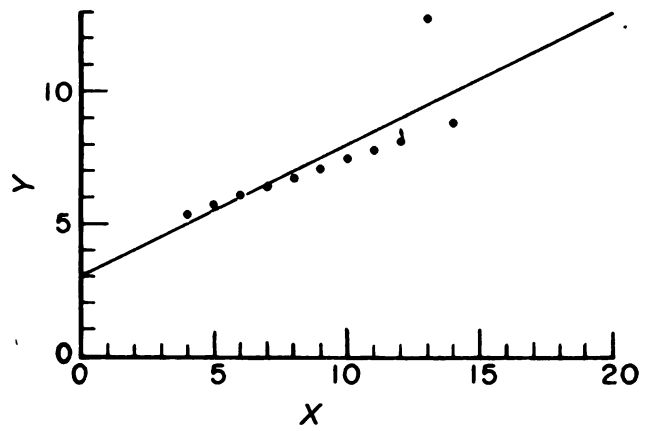

FIGURE 5-4 Graph of data in Anscombe's sample set 3^1 (see Table 5-1).

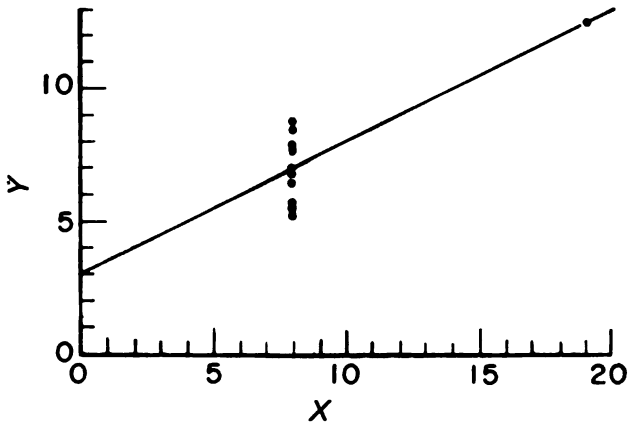

FIGURE 5-5 Graph of data in Anscombe's sample set 4[1] (see Table 5-1).

original transportation control plan formulated by a federal regulatory agency. Technologic controls without a reduction in the number of vehicle miles traveled (VMT) were considered as a first stage, and successive percentages of VMT reduction were considered as further stages. For the first stage of control, the photochemical-diffusion models suggested more rapid reduction in peak photochemical oxidant concentration than linear rollback, because the latter fails to account for nitric oxide inhibition effects. Reactive hydrocarbon (RHC) is the only precursor considered by the rollback method. In reality, the ratio of RHC to NO_x also has a decided influence on the oxidant production. Excess NO_x slows the production of oxidant. Successive stages of emission control brought about by reductions in VMT show less than proportional reductions in photochemical oxidant according to the photochemical-diffusion model. This occurs because RHC and NO_x are being reduced in the same ratio, which has a weaker influence on oxidant than in the case of more rapid reductions in RHC. This comparison illustrates the need to consider nonlinearities in the case of reactive pollutants when rollback models are used.

In another study, Martinez and Nordsieck[40] found that comparing linear rollback with a photochemical model gave very nearly the same results for peak oxidant for the year in the distant future when extensive controls are imposed. The agreement between the two methods, however, was poor in a case in which NO_x controls were frozen at 1974 values instead of being progressively applied as rapidly as RHC controls. This case is designated 1980*d* in Table 5-2, which summarizes the results for forecast ozone concentrations on the basis of both linear roll-

TABLE 5-2. Baseline Values of Maximal Hourly Average Ozone Concentration in Vicinity of Riverside, California[a]

Year	Maximal Hourly Average Ozone Concentration, ppm	
	Diffusion-Kinetics Model	Linear Rollback
1980	0.16	0.16
1990	0.15	0.13
2000	0.15	0.13
1980*d*	0.12	0.17

[a] Derived from Martinez and Nordsieck.[40]

back and the photochemical-diffusion model. The frozen-controls case (1980*d*) shows the large disparity between the methods because of the failure of rollback to consider the effects of mixture ratios of precursor pollutants on oxidant formation.

Reynolds and Seinfeld[58] compared the *statistical* model of Trijonis[71] with their dynamic model[55,57,60,66] and with linear rollback. Considering 1977 emission with and without one of the EPA abatement plans in effect, they obtained a reduction from 80 to 20 in the number of days on which the oxidant standard is exceeded and from less than 15 days to less than 10 days for exceeding the nitrogen dioxide standard. (The oxidant standard is defined on the basis of hourly average, and the nitrogen dioxide standard is defined as an annual average.) These findings, which were obtained with the Trijonis model, were limited to one monitoring station because of the method by which that model was calibrated. The dynamic model was run with two ways of specifying future initial conditions and grid boundary conditions for the calculations. It was not stated whether linear or nonlinear rollback was used. Comparisons were then made between 1969 (the reference year) and 1977 both with and without the EPA abatement plan in effect in 1977. The dynamic-model calculation showed a 76% reduction in carbon monoxide from 1969 to 1977 with the EPA abatement plan in effect and only a 65% reduction without it. Rollback agrees with the 65% reduction, because the conditions for its validity with an inert pollutant are fulfilled. Nitrogen dioxide peak concentrations were reduced by 5–8% without the plan and by 60–76% with the plan, according to the dynamic model. Rollback predicted only a 34% reduction with the plan. For ozone, the dynamic-model results suggested that the abatement plan is more effective than the rollback model would predict; that is, up to a 94% reduction in maximal ozone was predicted by the dynamic model,

and a 74% reduction was predicted by rollback. The 1969–1977 ozone reductions without the abatement plan in effect are 39% according to rollback and 65–75% according to the dynamic model. As was the case with the Hamming *et al.*[20] and Eschenroeder[12] calculations, this illustrates for ozone that it is necessary to consider both RHC and NO_x in the rollback calculation, rather than only RHC. If the RHC emission is reduced faster than the NO_x emission, the ozone concentrations at a given station may decrease in greater proportion than the RHC concentrations.

Peterson[53] used the skill score to evaluate the performance of his empirical statistical model based on orthogonal functions. The skill score equals 1.0 when all calculated and observed concentrations agree, but 0 when the number of correctly predicted results equals that expected by chance occurrences. The statistical technique had a skill score of 0.304. An 89-day, 40-station set of the data was used to check a Gaussian diffusion model, and this technique gave the diffusion model a skill score of only 0.15. (Recall that the statistical empirical model was used for 24-h averaged sulfur dioxide concentrations at 40 sites in St. Louis for the winder of 1964–1965.)

The time-series analysis results of Merz *et al.*[43] were expressed in first-order empirical formulas for the most part. Forecasting expressions were developed for total oxidant, carbon monoxide, nitric oxide, and hydrocarbon. Fitting correlation coefficients varied from 0.547 to 0.659. As might be expected, the best results were obtained for the primary pollutants carbon monoxide and nitric oxide, and the lowest correlation was for oxidant. This model relates one pollutant to another, but does not relate emission to air quality. For primary pollutants, the model expresses the concentrations as a function of time.

Bruntz *et al.*[4] applied multiple regression analysis and found that the method of least squares yielded a set of coefficients that produced a 0.84 correlation of ozone concentration with the data. Adding mixing height to the correlation yielded no statistically significant improvement in agreement with the assertions of Hanna.[21]

The first time-series model of McCollister and Wilson[42] yields results of about 0.4 for the mean absolute error divided by the mean value for the days in 1972, on the basis of parameters derived before 1972. By comparison, a persistence model yields about 0.5 for the same parameter, and the Los Angeles APCD forecasts lie between the time-series results and the persistence-model results. The hourly oxidant time-series model yields errors from 0.3 to 0.45, whereas persistence yields errors of 0.4–0.5. For carbon monoxide, the hourly-model results lie between 0.15 and 0.5—consistently below the persistence results.

Gifford and Hanna[19] tested their simple box model for particulate matter and sulfur dioxide predictions for annual or seasonal averages against diffusion-model predictions. Their conclusions are summarized in Table 5-3. The correlation coefficient of observed concentrations versus calculated concentrations is generally higher for the simple model than for the detailed model. Hanna[21] calculated reactions over a 6-h period on September 30, 1969, with his chemically reactive adaptation of the simple dispersion model. He obtained correlation coefficients of observed and calculated concentrations as follows: nitric oxide, 0.97; nitrogen dioxide, 0.05; and RHC, 0.55. He found a correlation coefficient of 0.48 of observed ozone concentration with an ozone predictor derived from a simple model, but he pointed out that the local inverse wind speed had a correlation of 0.66 with ozone concentration. He derived a "critical wind speed" formula to define a speed below which ozone prediction will be a problem with the simple model. Further performance of the simple box model compared with more detailed models is discussed later.

Species-Mass-Balance Model Performance and Comparative Evaluations

Sklarew *et al.*[69] evaluated their particle-in-cell K-theory approach for atmospheric diffusion of carbon monoxide and for photochemical smog. All-day averages of carbon monoxide concentration were predicted to be within 20% of the measured averages at 12 monitoring stations, and the correlation coefficient of measured with observed concentrations was 0.73.

TABLE 5-3. Test of Simple Model for Particulate Matter and Sulfur Dioxide Predictions[a]

City	No. Sampling Sites	Source Area Size, km^2	Correlation Coefficient, Observed vs. Calculated Concentrations	
			Simple Model	Detailed Model
Memphis, Tennessee	9	25	0.68	0.73
Nashville, Tennessee	16	25	0.91	0.80
Ankara, Turkey	10	9	0.63	0.59
Bremen, West Germany	4	16	0.65	0.05
"Test City"	8	25	0.98	0.96

[a] Derived from Gifford and Hanna.[19]

In their survey,[31] Johnson *et al.* covered evaluation studies and summarized the results of correlation coefficients and root-mean-square (rms) errors from a linear regression between observed and calculated values for three photochemical models.[16,56,75] The statistical comparisons are shown in Tables 5-4 and 5-5 for carbon monoxide and ozone, respectively. It is notable that the correlation coefficients are considerably higher than those reported for many Gaussian models. Similarly, the regression lines have slopes closer to unity than those from the Gaussian models. Extreme caution must be exercised in comparing performance measures directly, because of the intrinsic differences between trajectory and grid models and between evaluation test designs. Nappo[44] evaluated

TABLE 5-4 Statistical Comparison of Model Calculations and Observations of Carbon Monoxide Concentration[a]

Model[b]	Ref.	No. Data Points	Correlation Coefficient	RMS Error, ppm	Regression Line[c] ($y = ax + b$) a	b
SAI	56	514	0.79	3.4	1.09	0.68
PES	75	71	0.82	3.0	0.91	−0.70
GRC	16	149	0.80	3.7	1.01	−0.37

[a] Derived from Johnson *et al.*[31]
[b] SAI = Systems Applications, Inc.; PES = Pacific Environmental Services, Inc.; GRC = General Research Corporation.
[c] y = observed, x = calculated.

TABLE 5-5 Statistical Comparison of Model Calculations and Observations of Ozone Concentration[a]

Model[b]	Ref.	No. Data Points	Correlation Coefficient	RMS Error, ppm	Regression Line[c] ($y = ax + b$) a	b
SAI	56	574	0.69	0.063	0.76	3.9
PES	75	63	0.49	0.069	0.46	−7.6
GRC	16	151	0.92	0.021	0.84	2.3

[a] Derived from Johnson *et al.*[31]
[b] SAI = Systems Applications, Inc.; PES = Pacific Environmental Services, Inc.; GRC = General Research Corporation.
[c] y = observed, x = calculated.

eight mathematical models and 24-h persistence with six different measures of performance evaluation for carbon monoxide predictions. The usual measure of correlating the time-averaged concentrations was applied to the computed versus observed values. Table 5-6 summarizes these results, with computer time and computer costs estimated for an IBM 360/65 system. This is one of the few attempts to supply information that could enter a cost-effectiveness analysis. The data base used by Nappo consists exclusively of computed and observed concentrations that are reported in the literature (see Table 5-6 for references). The averaging time for each data set varied according to the time interval for which each model was run. For example, if a model was designed to predict hourly averages and was run for an 8-h interval, the averaging time for the data set was chosen to be 8 h.

In addition to the temporal correlation coefficient, the spatial correlation coefficient was calculated approximately for fixed values of time. Except for one of the mathematical models,[24] all techniques showed a better temporal correlation than spatial correlation. The two correlation coefficients are cross plotted in Figure 5-6. Nappo stressed that correlation coefficients express fidelity in predicting tends, rather than accuracy in absolute concentration predictions. Another measure is used for assessing accuracy in predicting concentrations: the ratio of predicted to observed concentration. Nappo averaged this ratio over space and over time and extracted the standard deviation of the data sample for each. The standard deviation expresses consistency of accuracy for each model. For example, a model might have a predicted:observed ratio near unity,

TABLE 5-6 Model Evaluation Based on Temporal Characteristics[a]

Model	Ref.	Average Temporal Correlation Coefficient	Computer Time for 24-h Prediction, min	Computer Cost for 24-h Prediction, $
Multibox persistence	38	0.37	106	350
		0.47	0	0
Primitive equation	59	0.52	60	200
Simple model ATDL	24	0.60	0	0
Particle-in-cell	68	0.65	49	160
Primitive equation	51	0.66	20	70
Primitive equation	56	0.73	30	100
Trajectory	16	0.73	15	50
Trajectory	36	0.90	35	115

[a] Derived from Nappo.[44]

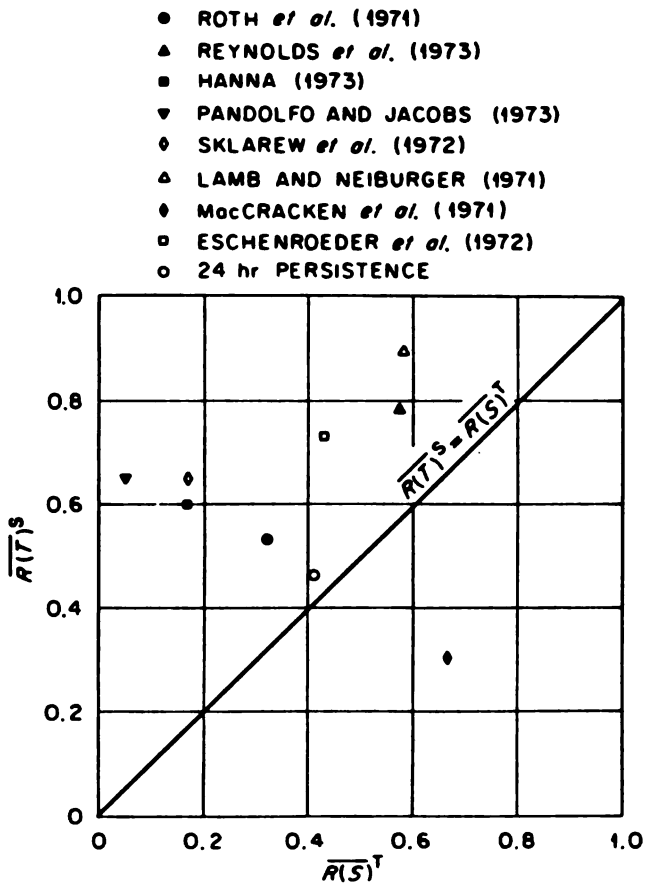

FIGURE 5-6 Space-averaged temporal correlation coefficient $\overline{R(T)}^S$ versus time-averaged spatial correlation coefficient $\overline{R(S)}^T$. Points are averages for each model tested. Reprinted with permission from Nappo;[44] reference numbers changed to conform with this volume.

but with a wide variability of the ratio about its mean value. All the models tested have ratios that average within ±40% of unity (including 24-h persistence), with two exceptions—the Hanna simple model[24] and the Lamb and Neiburger trajectory model.[36] It should be noted that this measure of model performance produced a rank ordering very different from that of the usual temporal correlation test. Figure 5-7 shows the mean ratios of predicted to observed concentration with uncertainty bars characterized by the standard deviation of the ratios about their mean. Figure 5-8 shows the space-averaged temporal standard deviation

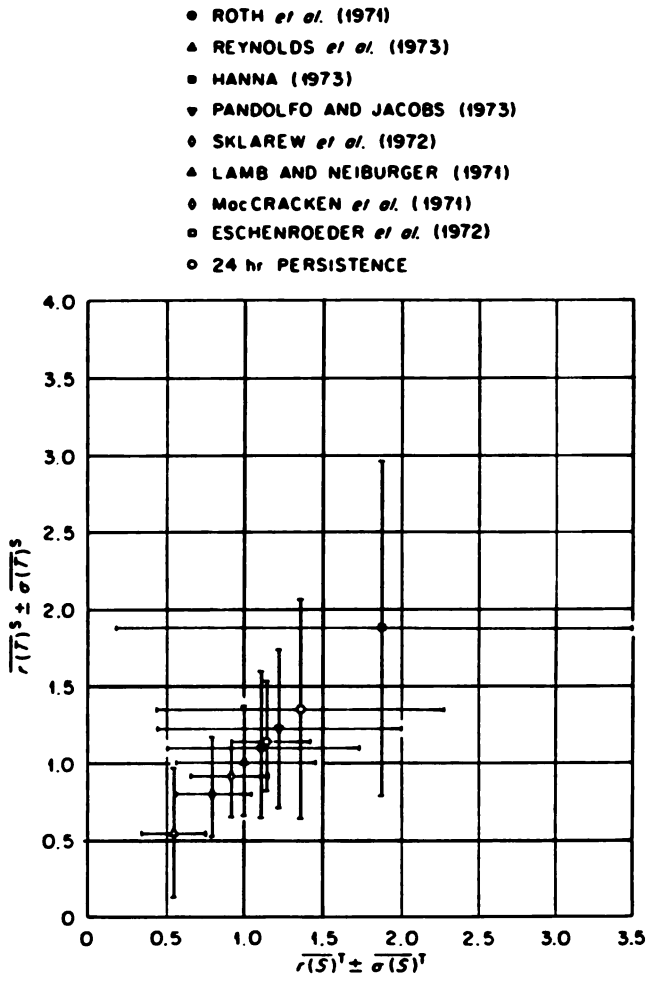

FIGURE 5-7 $\overline{r(T)}^S \pm \overline{\sigma(T)}^S$ versus $\overline{r(S)}^T \pm \overline{\sigma(S)}^T$. Points are averages for each model tested. Reprinted with permission from Nappo;[44] reference numbers changed to conform with this volume.

plotted against the time-averaged spatial standard deviation of these ratios. The models have both standard deviations less than 0.5 (except those of Roth *et al.*,[59] Pandolfo and Jacobs,[51] and Hanna[24]) and 24-h persistence.

Whitney[76] used actual measurements to evaluate studies of predicted values derived by photochemical models. To assess the validation tests sponsored by the EPA for three different approaches,[16,56,75] Whitney

proposed several statistical evaluation measures of model performance. In addition to the correlation coefficient and the standard deviation, the chi-square test is imposed. Table 5-7 shows the comparison for four pollutants modeled. The symbol SAIC denotes station values selected from the Systems Applications, Inc., validation runs[56] to be in correspondence as nearly as possible with the station values computed in the Pacific Environmental Services, Inc., validation runs (PESC).[75] This was done in an effort to compare models on a common-denominator basis. Likewise, trajectories are traced through the SAI hourly grid results, to form the SAIT data set to compare with the General Research Corpora-

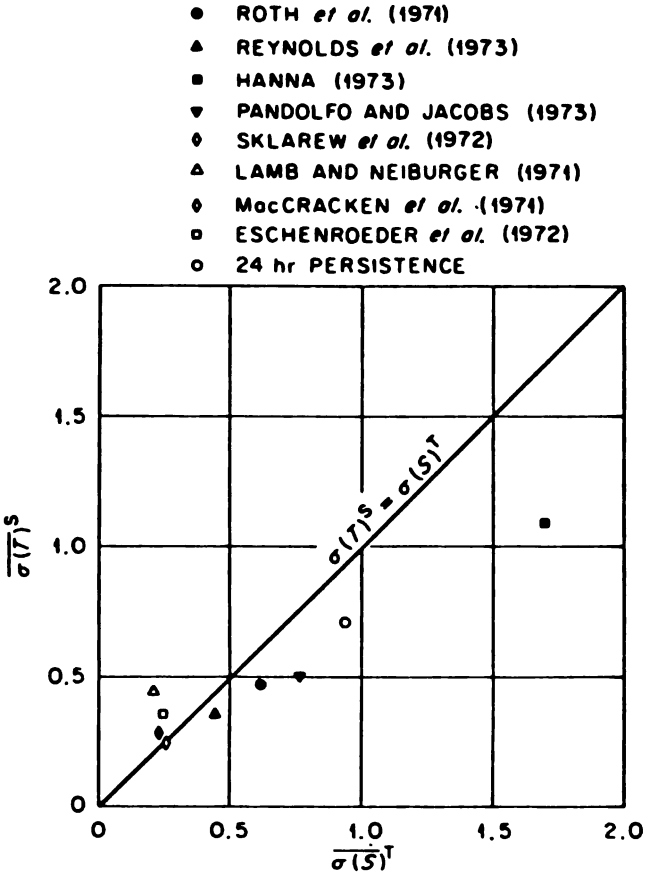

FIGURE 5-8 $\overline{\sigma(T)}^S$ versus $\overline{\sigma(S)}^T$. Points are averages for each model tested. Reprinted with permission from Nappo;[44] reference numbers changed to conform with this volume.

TABLE 5-7 Statistical Analyses of Photochemical Air Quality Model Performance for the Los Angeles Basin[a]

Model[b]	Correlation Coefficient[c]	Standard Deviation[d]	Degrees of Freedom	Measured Chi-Square[e]	Standard Chi-Square[f]
Carbon monoxide					
PESC	0.68	4.27	143	233.75	171.62
SAIC	0.84	3.52	290	287.35	330.44
GRCT	0.82	3.39	172	75.12	203.32
SAIT	0.79	3.18	175	84.42	206.58
Nitric oxide					
PESC	0.77	3.18	49	227.67	66.05
SAIC	0.87	9.13	277	1,081.01	316.54
GRCT	0.87	8.70	171	1,053.03	202.23
SAIT	0.86	8.13	173	1,042.44	204.41
Nitrogen dioxide					
PESC	0.68	8.36	49	157.15	66.05
SAIC	0.65	6.82	276	310.56	315.47
GRCT	0.43	10.05	172	316.91	203.32
SAIT	0.52	12.62	175	159.81	206.58
Ozone					
PESC	0.50	8.26	53	380.85	70.71
SAIC	0.60	8.56	255	3,480.79	292.97
GRCT	0.91	3.82	127	237.74	154.02
SAIT	0.69	8.75	131	387.30	158.43

[a] Data from Whitney.[76]
[b] Acronyms are identified in text.
[c] Correlation of predicted and actual measurements.
[d] Difference of predicted and actual measurements.
[e] Normalized by σ, where σ is assumed to be $0.7 + 0.1 \times$ (observed value).
[f] A 90% confidence level was assigned for the purposes of the chi-square test.

tion validation runs (GRCT).[16] Clear trends are difficult to identify, but, as the authors pointed out, the SAI model probably excelled in carbon monoxide prediction because it has more detail in advective diffusive coupling, and the GRC model did best with ozone prediction probably because of careful calibration. The PES model had the minimal standard deviation with nitric oxide prediction because the data sample centered on midday concentrations away from the morning traffic peak, which is difficult to predict. The chi-square test suggested that most of the time none of the models adequately represented the data on the basis of a 10% chance that the results are randomly distributed. Exceptions to this finding are noted with GRCT and SAIT, both passing the chi-square test at a 90% confidence level for carbon monoxide.

Other performance measures were carefully chosen to reflect accuracy of prediction. They consisted of scatter plots of predicted versus observed concentrations and four other types of plots involving analyses of residuals. The residual study took the (predicted vs. observed) values and displayed them in histograms in plots of time against predicted concentrations and against observed (or interpolated) concentrations. The results of the residual analysis are too extensive to review here, but the user who is interested in modeling a particular pollutant or who is considering one of the specific models tested should examine the computer printouts to answer specific performance questions. Whitney has devised a useful set of model performance measures for the task of evaluating the earlier evaluations. The results of similar tests on log statistics would have been interesting. It was not stated whether the residuals were normally distributed.

The sensitivity of diffusion-model output to variations in input has been assessed by workers at Systems Applications, Inc., and at the California Department of Transportation. In each case, reports are in preparation and are therefore not yet available. It is important to distinguish between sensitivity and model performance. True physical or chemical sensitivity that is reflected by the simulation-model equations is a valid reflection of reality. But spurious error propagation through improper numerical integration techniques may be regarded as an artificial sensitivity. Such a distinction must be drawn carefully, lest great sensitivity come to be considered synonymous with unacceptable performance.

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6

Measurement Methods

Photochemical oxidants are atmospheric pollutants produced by a series of reactions between hydrocarbons and oxides of nitrogen in the presence of sunlight. The recognized photochemical oxidants that have been measured in ambient air are ozone, the peroxyacylnitrates (mostly as PAN), and hydrogen peroxide.[24,51]

Chemical radicals—such as hydroxyl, peroxyhydroxyl, and various alkyl and aryl species—have either been observed in laboratory studies or have been postulated as photochemical reaction intermediates. Atmospheric photochemical reactions also result in the formation of finely divided suspended particles (secondary aerosols), which create atmospheric haze. Their chemical content is enriched with sulfates (from sulfur dioxide), nitrates (from nitrogen dioxide, nitric oxide, and peroxyacylnitrates), ammonium (from ammonia), chloride (from sea salt), water, and oxygenated, sulfurated, and nitrated organic compounds (from chemical combination of ozone and oxygen with hydrocarbon, sulfur oxide, and nitrogen oxide fragments).[45]

The chemistry of the photochemical reaction milieu, including the formation of secondary aerosols, is covered in Chapter 3. Table 6-1 lists the substances that have been identified and associated with the impact of photochemical oxidants on breathing, eye irritation, plant damage, and visibility reduction. It is important to recognize this chemical com-

TABLE 6-1 Substances and Monitoring Practices for Species Present in Photochemical Smog as Precursors or Products

Substance	Formula or Symbol	Monitoring Practice
Gaseous		
Ozone	O_3	Routine
Peroxyacetyl nitrate (PAN)	$CH_3\overset{O}{\overset{\|}{C}}OONO_2$	Occasional
Hydrogen peroxide	H_2O_2	Research
Nitrogen dioxide	NO_2	
Nitric oxide	NO	Routine
Hydroxyl	$HO\cdot$	Research
Peroxy hydroxyl	$HOO\cdot$	None
Alkyls	$CH_3(CH_2)_n\cdot$, $CH_3CO\cdot$, etc.	None
Aryls	$C_6H_5CH_2\cdot$, etc.	None
Aldehydes		
Formaldehyde	CH_2O	Occasional
Acetaldehyde	CH_3CHO	Occasional
Acrolein	$CH_2=CHCHO$	Occasional
Benzaldehyde	C_6H_5CHO	Occasional
Hydrocarbons		
Total	THC	Routine
Methane	CH_4	Routine
Total nonmethane	NMHC	Routine
Alkanes	C_2H_6 to C_nH_{2n+2}	Occasional and research
Olefins	C_2H_4 to C_nH_{2n+1}	Occasional and research
Diolefins	C_4H_6 to C_nH_{2n-2}	Occasional and research
Acetylene	C_2H_2	Occasional and research
Aromatics	C_6H_6 to C_nH_{2n-6}	Occasional and research
Particulate		
Sulfate	$SO_4^=$	Routine
Nitrate	NO_3^-	Routine
Ammonium	NH_4^+	Occasional
Chloride	Cl^-	Occasional
Water	H_2O	Research
Oxygenated organics	$HOOC(CH_2)_nCH_2OH$	Research
	$HOOC(CH_2)_nCOOH$	Research
	$HOOC(CH_2)_nCHO$	Research
	$HOOC(CH_2)_nCONO_2$	Research
	Many others	

plexity when formulating strategies for controlling the emission of the primary pollutants. It is also important that a review of the issues relates air pollutants to their sources. This requires the measurement not only of end products but also of their precursors. Table 6-1 shows in general terms the present state of monitoring practice for each of these substances.

The effects of photochemical smog initially observed in Los Angeles were severe haze formation, eye irritation, plant damage, and rubber-tire cracking. Haagen-Smit[7] was the first researcher to recognize that the severity of photochemical smog could be quantified by measuring oxidants. This oxidizing property of smog was later more efficiently monitored by measuring the increase in color intensity produced by the iodine released from potassium iodide solutions in contact with air.[35,47] As a consequence, photochemical oxidants have been defined by air pollution agencies as atmospheric substances that oxidize specific reagents.

The reagent most frequently used is a neutral phosphate-buffered potassium iodide solution, calibrated with a known source of ozone. This reagent, which is particularly sensitive to ozone, is also somewhat responsive to other atmospheric oxidants, such as nitrogen dioxide, the peroxyacylnitrates, and, to a lesser extent, hydrogen peroxide.[24,51] Reducing agents present in smog (e.g., sulfur dioxide, SO_2) have an effect on the reagent solution opposite to that of oxidants.

Various terms are used to describe photochemical-oxidant concentrations. Two of these, *oxidant* and *total oxidant,* are used because the measurement method (usually the potassium iodide method) cannot distinguish between oxidizing and reducing agents, which in combination produce the "net" oxidizing ability of the air. The terms *corrected oxidant* and *adjusted oxidant* are used to indicate that a measurement has been corrected for responses to particular components known to be present (usually sulfur dioxide and nitrogen dioxide), other than ozone, peroxyacetylnitrate, or hydrogen peroxide.[3] None of the terms *photochemical oxidant, total oxidant, oxidant, corrected oxidant,* and *adjusted oxidant* has an exact meaning, because the reaction measured by the potassium iodide method is produced by the presence of a number of substances, each responding differently. Ideally, each of the major oxidants (ozone, nitrogen dioxide, PAN, and hydrogen peroxide) and each of the major reductants (sulfur dioxide and hydrogen sulfide) should be measured separately.

The potassium iodide method has been used extensively in California to measure the net oxidizing properties of atmospheric pollutants without identifying the particular species of oxidizing or reducing agents present. Studies have shown, however, that in California ozone is the major oxidant.[11,24] At most sampling locations in California, the negative

interference from sulfur dioxide and the positive interferences from other oxidants have not substantially altered the perception of the photochemical smog problem.[24,51] This is both because the concentrations of sulfur dioxide are in general small, compared with the relatively high observed ozone concentrations, and because the positive interference from nitrogen dioxide tends to offset the negative interference from sulfur dioxide. In other parts of the United States where the peak ozone concentrations have generally been much lower, the potassium iodide measurement method has produced data that have been useful in establishing that an oxidant pollution problem exists. But, because it lacks sufficient specificity (T. D. Graedel, personal communication; N. Lewis, personal communication), it is unreliable for measuring the ozone occurrence in these areas. In contrast, there are specific measurement methods that provide reliable relationships between ozone concentrations and other aerometric variables.[9]

In addition to the specificity of the monitoring method, an important requirement for the measurement of atmospheric pollutants is the accuracy of the calibration technique. The calibration procedure for the measurement of oxidants or ozone utilizes a stable and reproducible sample of dilute ozone in air. The ozone concentration of this sample is established with a reference method that is not necessarily suitable for monitoring ambient air. This reference method must agree with the scientifically accurate measurement of ozone in the calibration sample.

Whether ozone or oxidant measurements can be correlated with other variables depends heavily on the choice of sampling site and the manner of sample transfer. The importance of these criteria is discussed in Chapter 5, and the desirable specifications are described in this chapter.

The emphasis here is on the various routinely used measurement processes. These create huge banks of data that become influential in deciding public policy. Measurements made only occasionally or during research (Table 6-1) are more easily scrutinized at the time of data application and therefore are not dicussed here. Specifications for most types of available instrumentation have been compiled and discussed by the Environmental Instrumentation Group, Lawrence Berkeley Laboratory.[33]

UNITS OF EXPRESSION

Ozone or oxidant concentrations are commonly reported in the following units: volumes of ozone per million volumes of air (ppm) and weight of ozone per cubic meter of air ($\mu g/m^3$). The scale for the units in volume

per volume or weight per volume may be varied to avoid small decimals or large whole numbers.

The commonly used units of expression are explained in Table 6-2. Expressing concentration data in micrograms per cubic meter facilitates relating ambient concentrations to emission. This practice is generally accepted as standard by the Environmental Protection Agency (EPA) in the United States and similar agencies in other countries.

Expressions of volume per volume units (ppm, pphm, or ppb) simplify measurements, because their value is independent of atmospheric temperature and barometric pressure. The volume units are equivalent to the ratio of the number of molecules of ozone to the number of molecules of air. This facilitates quantification of the atmospheric chemical reactions that lead to the formation of ozone. These units are also preferable when the molecular weight of a substance is uncertain, as in the reporting of total nitrogen oxides or total aldehydes.

Particulate matter cannot be expressed on a volume-to-volume basis, but it can be expressed on a mole-per-unit-volume basis, μmoles/m^3, if the molecular weight is known. This would be a convenient unit to use in investigating the relationship between particles and gaseous species.

TABLE 6-2 Units of Ozone Concentration and Their Interconversion Factors

Units	Explanation	Units
ppm	Volume of ozone per million volumes of air (or molecules of ozone per million molecules of air)	= μl O_3/l air = 100 pphm = 1,000 ppb = 1.96 mg O_3/m^3 air* = 1960 μg O_3/m^3 air*
μl/l	Microliters of ozone per liter of air	= ppm
pphm	Volume of ozone per hundred million volumes of air	= 0.01 ppm
ppb	Volume of ozone per billion volumes of air	= 0.001 ppm
mg/m^3	Milligrams of ozone per cubic meter of air at 25° C and 1 atm pressure	= 0.51 ppm
μg/m^3	Micrograms of ozone per cubic meter of air at 25° C and 1 atm pressure	= 0.00051 ppm = 0.051 pphm = 0.51 ppb
160 μg/m^3	Ozone air quality standard	= 0.08 ppm

* When the temperature is 25° C and the barometric pressure is 1 atm.
Note: Temperature and pressure do not influence the value of the volume-per-volume units.

The conversion of ppm to $\mu g/m^3$ is calculated by Equations 6-1 and 6-2:

$$\mu g/m^3 = \frac{(ppm)\ 1.219 \times 10^4 PM}{T} \quad (6\text{-}1)$$

and

$$ppm = \frac{(\mu g/m^3)T}{(1.219 \times 10^{-4})PM} \quad (6\text{-}2)$$

where M is the molecular weight of the gaseous substance being measured (for ozone, O_3, M = 48 g/mole),
P is the total gas pressure in atmospheres (atm),
T is the temperature in Kelvin (degrees Celsius + 273), and
1.219×10^4 is the reciprocal of the gas law constant, R, in the unit moles T/atm m^3, consistent with the units used in equations.

As these conversion equations show, in addition to the molecular weight of a measured gaseous pollutant, the temperature and pressure at the time of the measurement must be known. Because this information is often not given in the literature, no attempt has been made in this report to convert to a common unit.

SAMPLE-GATHERING

Several sample-gathering techniques are available for oxidant and ozone measurements. Typically, outdoor air taken with a sampling probe is ducted to a sampling manifold in a temperature-regulated space. Air is withdrawn from the manifold either continuously into automated ozone-monitoring instruments or intermittently with cumulative ozone absorbers. Because ozone reacts quickly with some substances, reliable measurement requires careful attention to the details of sampling-site selection, sampling frequency, and sampling-train materials. Ozone measurements are made indoors, outdoors near ground level, and from aircraft, for different but related purposes. Nationwide uniformity in sampling techniques is being achieved through cooperation among the various control agencies in specifying sampling conditions.

The location of the probe necessitates careful design and documentation to ensure that surrounding environmental conditions do not interfere with the subsequent interpretations of the measurement data. Examples of such minimal site descriptions are given in Table 6-3. Some of this information is now included with all the data logged into the EPA's National Aerometric Data Bank, and some was provided by the control agencies. An example of a site description in southern California based on the requirements of the California Air Resources Board is shown in

TABLE 6-3 Monitoring Site Description

Location and Agency	Site Number	Land Use and Site Description	UTM*	Elevation above SL, mi
1. New York (AQCR 043) State Dept. of Environmental Conservation, Div. of Air Resources				
Welfare Island	7093-03	Residential; fairly open area free from obstructions;	589.4 E	
(NYC East River)		northern tip of Welfare Island in East River	4513.2 N	
Merrick Ave.	2950-10	Residential, near Roosevelt Raceway; open area, no	619.3 E	46–61
Eisenhower Pk., L.I.		obstructions; level topography all around	4511.2 N	
Mammareseck	5956-01	Residential and commercial; low spot but fairly level;	603.9 E	
5th Ave. at N.Y.S.		surrounded by houses; located in small basin in	4531.8 N	
Twy. Exit 9		which the land rises 50 ft within an 800-ft radius		
2. New Jersey (AQCR 043) State Dept. of Environmental Protection, Bureau of Air Pollution Control				
Bayonne		Mostly residential between bay and open park 0.5 mi	574.4 E	46
(Hudson County Park)		long; over mile from Newark	4503.5 N	
Newark		Center city, commercial, surrounded by obstruction,	569.6 E	122
(Brandford Place)		on one corner of a parking lot 1 block square; moved mid-May 1975	4509.5 N	
Linden		Suburban, industrial adjacent to refinery	566.0 E	61
(Tremley Point Sewage Plant)			4495.4 N	
3. Houston (AQCR 216) Texas Air Control Board				
Deer Park	4515-60	Industrial, open area free from obstruction, near	285.1 E	64
(Mae St.)	(Connie 1)	Federal Rd. and IH 10, level topography	3295.4 N	
Aldine	(Connie 8)	Residential, open area free from obstruction; 1.6 km	275.4 E	579
(N. Houston)		west of Hwy. 29; 3.2 km of airport; prevailing SE wind	3310.2 N	
4. Los Angeles (AQCR 024) Los Angeles County Air Pollution Control District (see Table 6-4)				

*Universal Transverse Mercator, 1/10 km units.

Figure 6-1. In addition to the site description, there is a statement of all the conditions being monitored, the methods used, and the numerical specifications for the sampling probe, both for the sampling manifold and for the connections from the manifold to the instruments. A third page (not included here) shows a schematic drawing with the dimensions and locations of the bends in the ducting. The sampling-probe specifications currently in effect at four major air pollution control agencies are summarized in Table 6-4.

Date July 1, 1974 Prepared by WDH

Air Monitoring Station No 60 City Azusa

Street Address 803 North Loren Avenue, Azusa, California

Longitude W 117 Deg. 55 Min. 22 Sec., Latitude: N 34 Deg. 8 Min. 9 Sec.

Nearest Street Intersection Loren Avenue and Foothill Boulevard

Date Operation Initiated at this Location January 30, 1957

Name of Operating Agency Los Angeles County Air Pollution Control District

Address of Operating Agency 434 South San Pedro Street, Los Angeles, California 90013

District Los Angeles Basin South Coast

SITE DESCRIPTION

CATEGORY

☐ CENTER CITY ☒ SUBURBAN

☐ RURAL

☐ REMOTE

SUBCATEGORY (Dominating Influence)

☒ Industrial ☐ Residential ☐ Commercial ☐ Mobile

☐ Near Urban ☐ Agri ☐ Comm ☐ Ind ☐ None of these

Type of Street Traffic Arterial

(Residential, Expressway, Commercial, Downtown, Arterial)

Outstanding Landmarks and Relation to Site:

Station location at foothills of San Gabriel Mountains. Aerojet plant S.E. 2 miles; Foothill Freeway a mile south.

Surrounding terrain and community characteristics (hills, valleys, flat, bodies of water, residential, industrial, commercial, rural, open, forested, row crops, grassland, orchard, etc.).

San Gabriel Mountains four miles to the north; small manufacturing plants around station site. Some residence near site - single family next door to south, trailer park across Loren to the east. Commercial area of Azusa 2 miles to the east. Flat, gently sloping terrain.

Possible nearby sources (refineries, stacks, chimneys, gasoline stations, power plants, parking lots, traffic, agricultural operations, etc.)

Source	Direction	Distance	Pollutants
Small Iron Foundry (usual equipment)	southeast	1000 feet	Normal
Large Gravel Plant	northwest	1½ miles	Particulate
Large Gravel Plant	southwest	3 miles	Particulate
Freeway	south	1 mile	Auto Exhaust
Limited Parking	east and north	Adjacent	Auto Exhaust
Metal Melting Furnace (no permits on file so apparently a minor source)	southwest	100 feet	Normal - Operated intermittently, regular schedule

Attach 8½" X 11" portion of local street map indicating station location.

FIGURE 6-1 State Air Resources Board sampling site report.

When a monitoring site is selected, it is important to take account of environmental features. For example, ozone measured in or near automotive traffic can drop to 50% of the areawide value, owing to reaction with the nitric oxide from exhaust emission. Ozone measured 7.5 m from a large tree in green leaf can drop to 70% of the areawide value, but it may also be reduced within 1 m of shrubs and grass. Paint, asphalt, concrete, dry soil, and dead vegetation are not as reactive and so have less effect. Peak ozone values observed in sunlit windscreened,

Air Monitoring Station No. 60

Gaseous Measurements

Gases		Method	Air Flow l/min.
O_3	____	_____	_____
O_x	X	Potassium Iodide - Colorimetric	3.7
NO	X	Modified Saltzman - Colorimetric	0.225
NO_2	X	Modified Saltzman - Colorimetric	0.225
NO_x	X	Addition of NO and NO_2 from above	_____
THC	X	Flame Ionization	0.05
THC-CH_4	X	Flame Ionization	0.05
CO	X	Non-dispersive Infrared	0.5
SO_2	X	Conductivity - Hydrogen Peroxide	20 CFH
Other	____	_____	_____

Instruments located on 1st floor of 1 story building.

PROBE DESCRIPTION

PROBE

Size 1 inch I.D.

Material Pyrex pipe

Flow rate 30 l/min.

Length 23 ft.

Sample Residence Time in Probe 7.1 sec.

Number of Changes in direction 1

Number of Instruments connected 5

AIR INTAKE

Vertical _____ (up, down)

Angled 45° down (degrees, up, down); Direction _____ (N,S,E,W)

Horizontal X Direction S (N,S,E,W)

Height above: Ground level 7 ¼ ft.

sea level 607 ft.; roof - ft.

Horizontal distance to nearest street curb 43 ft.

If the probe does not extend above the roof, indicate the intake distance down from the roof 4 ft.; out from the building wall 1¼ ft.

Note: If more than one probe utilized, provide the above information for each. Attach a single line schematic diagram of probe arrangement(s) indicating inside diameters and cumulative air flows of all segments.

Three to four feet of ¼-inch Teflon tubing connects each instrument to sampling man. Are wind instruments on same mast as station probe(s) inlet(s)? XXX No. If yes how much higher? - - ft. If no, distance and direction from probe(s). 25 ft., direction W.

Height above: Ground level 24 ½ ft., roof 13 ft., sea level 624 ft.

Nearby obstruction(s) to wind flow: None

Type(s), distance(s), direction(s), size(s)

FIGURE 6-1 (Cont.)

TABLE 6-4 Sampling Probe Design

Item	Los Angeles	New York	New Jersey	Houston, Texas
Agency	Los Angeles County Air Pollution Control District (LAAPCD)	State Division of Air Resources	State Bureau of Air Resources	Texas Air Control Board
Height above ground, m	2.2-22, mostly 3-6	4-6	3-4	6
Material	Glass	Glass	Glass	Glass
Shape	Inverted U down	Inverted U down	Inverted U down	Inverted U down
Inside diameter, cm	2.5	7.6	5-7.6	5
Total length, m	7-16	7-11	7-9	13
Flow rate, liters/min	15-30	560-850	283-400	283
Sum of sampler flows, liters/min	14	5-10	<10	*ca.* 10
Water removal	See no condensation	See no condensation	See no condensation	Manifold lies in trailer light-well
Relative humidity controls	None	None	None	Heat obviates condensation
Temperature controls	±3° C	±2° C (20-22° C)	±3° C	±2° C
Inlet filters	None	Screen, no filter	No filter, no screen	None

concrete-and-asphalt courtyards tend to be higher than those recorded on an adjacent rooftop.

A preferred sampling location is open to the free movement of the ambient air, at least 3 m above grass, 8 m from shrubs, 40 m from large trees, and 120 m from heavy automotive traffic. The flat roof of a one- or two-story building is ideal.[51] A probe that projects through a wall and extends about 2 m beyond is an acceptable alternative approximating the roof.

To minimize undesirable gas-phase reactions in the probe, the gas flow must be regulated to keep the transit time in the sampling ducts as short as possible. When a probe is longer than 30 cm, passage of the ozone-air sample should take less than 20 or 30 s. Losses can also occur from reaction with the probe itself or with the accumulated dust that coats the inside.

The materials of which probes are constructed must be sufficiently inert to trace amounts of ozone to prevent adsorption, absorption, and reaction between the ozone-air sample and the sample line during the transit period. Teflon and glass are both relatively inert, but Teflon has the advantage of being unbreakable and thus more durable. Nalgon vinyl tubing is satisfactory for connectors and even for a short probe, if nothing else is available. Ozone reacts with Tygon vinyl, stainless steel, and aluminum, all of which should not be used.

To prevent the formation of reaction products from the interaction of the ozone-air sample with filters, they are intentionally not used at probe inlets (see Table 6-4). Some of the newer instruments, however, require filters at the inlet of their sampling ports to prevent the particulate matter in the ambient air from fouling reaction-chamber cells or from clogging the gas-flow controllers. When the same type of filter also precedes the calibration and zero gas sampling ports (which has not always been the practice), the problem is minimized to the extent that similar events occur during the calibration and sampling.

CALIBRATION

Most currently used oxidant and ozone monitors need to be calibrated with a predetermined concentration of ozone in air. Regardless of the principle used to measure ambient ozone or oxidant concentrations, the primary reference standard for calibrating each monitoring device or system should be identical everywhere. This requirement remains to be achieved in practice. Up to June 1975, at least seven calibration procedures were practiced in the United States. These are listed in Table 6-5

TABLE 6-5 Primary Reference Standards for Calibration of Ozone or Oxidant Analysis

Principle	Abbreviation	Agency/Application	Status	Ref.
1. Colorimetric; 1% neutral buffered potassium iodide	1% NBKI	USEPA/air monitoring	Official, EPA	53
2. Colorimetric; 2% neutral buffered potassium iodide	2% NBKI	California ARB/air monitoring	Official, California until June 1975	10
3. Titrimetric; 2% unbuffered potassium iodide	2% UBKI	Los Angeles County/air monitoring	Official	1
4. Gas-phase titration of nitric oxide with ozone	GPT	USEPA/verification	Tentative EPA method	25, 43
5. National Bureau of Standards ozone generator	SRM-O_3*	Several/with (1) in air monitoring	Developmental	27
6. Extinction coefficient of ozone in the ultraviolet range at 254 nm	UV-254	California ARB/air monitoring; research labs.	Official in California since June 1, 1975	14
7. Extinction coefficient of ozone in the infrared range at a frequency of 1053 cm^{-1} (9480 nm)	IR-1053	Research labs.	Research	21, 42

*Standard reference material.

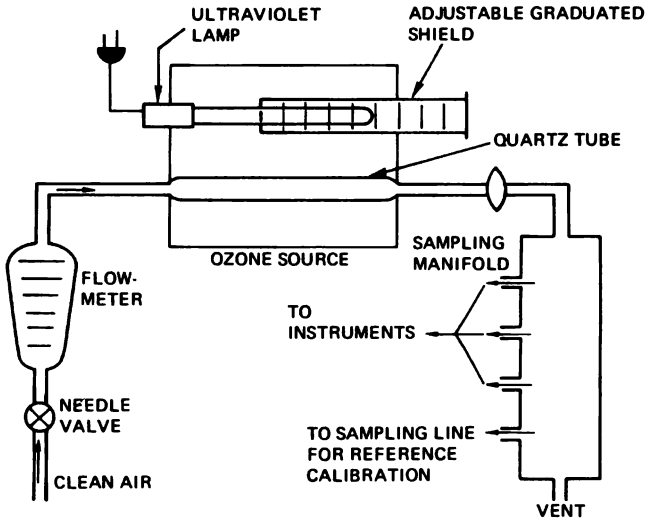

FIGURE 6-2 Ozone source and manifold system. Adapted from Hodgeson *et al.*[27]

with the agency, primary use, and current status of each method. Comparisons of these methods were recently completed.[14,16,26,41,42]

Ozone Generation

For reliable calibrations, it was necessary to develop a stable and reproducible ozone source that could produce ozone in air at concentrations smaller than parts per million. After this was accomplished,[27] several different versions were engineered; they are now available commercially from vendors of most ozone-monitoring instruments.* The factors affecting the production and survival of oxygen species other than ozone are discussed in Chapter 12. Care must be taken to prevent these species from creating interference when ozone is generated for instrument calibration.

A typical source (see Figure 6-2) consists of an ultraviolet mercury lamp that irradiates a quartz tube through which clean air flows at 5–10 liters/min. A small amount of the oxygen in air is converted to ozone by photolysis. It is important that the incoming air be free of moisture, nitrogen oxides, sulfur oxides, hydrocarbons, and particles, to

*Bendix Corp., Ronceverte, W.Va.; Dasilu Corporation, Glendale, Calif.; McMillan Electronics Corporation, Houston, Tex.; Monitor Labs., Inc., San Diego, Calif.; Ultra-Violet Products, Inc., San Gabriel, Calif.

avoid producing inadvertent interferences with subsequent sensors. Ozone concentrations from 0 to 1 ppm can be generated by varying the ultraviolet radiation intensity by means of an adjustable shield around the lamp envelope. The air-stream flow rate is controlled by a needle valve and measured by a rotameter or mass-flow meter. The ozonized air passes to a manifold from which the testing monitor draws its sample. The ozone concentration produced in this way is solely an empirical function of the settings of the lamp shield and air flow rate, the air temperature, and the humidity. Therefore, the output concentration must be measured with a scientifically acceptable reference standard.

Ozone Analysis

All the iodometric reference methods (items 1, 2, and 3, Table 6-5) are similar in principle. They assume that ozone (O_3) when in contact with iodide ions (I^-) in aqueous solution releases a stoichiometric amount of iodine (I_2), according to the following chemical equation:

$$O_3 + 2I^- + H_2O = I_2 + O_2 + 2OH^-, \qquad (6\text{-}3)$$

where O_2 stands for oxygen and OH^- for hydroxyl ions.

In California, the LAAPCD and the California Air Resources Board (CARB) both began continuous air monitoring for oxidants more than 20 years ago. Oxidant monitoring at urban sites was begun more than 5 years ago by the National Air Pollution Control Administration and continued by the EPA. All these groups initially used instruments containing the same absorbing solutions, but their iodometric calibration methods differed in detail.

California Air Resources Board (CARB) *Procedure (before June 1975)*[10] The CARB reference procedure for ozone uses a 2% neutral buffered potassium iodide reagent. Ozone is generated in air previously humidified to about 50% relative humidity. The air sample to be analyzed is drawn through the reagent in a midget impinger, and the ozone present in the sample air liberates iodine from the iodide reagent. The quantity of iodine liberated is determined with a spectrophotometer that has been calibrated with an iodine solution standardized with sodium thiosulfate solution, which, in turn, has been standardized against primary-grade potassium biiodate.

U.S. Environmental Protection Agency (EPA) *Procedure*[53] The EPA reference procedure for ozone is similar to the CARB procedure, except

that the ozone is produced from dry air, the reagent is 1% neutral buffered potassium iodide, and the iodine solution is standardized with primary-standard-grade arsenous oxide. Oxidant monitors outside California have generally been calibrated with this procedure.

Los Angeles County Air Pollution Control District (LAAPCD) *Procedure*[1] The LAAPCD reference procedure for ozone uses a 2% unbuffered potassium iodide reagent. Ozone is generated in air previously humidified to about 50%. The air sample to be analyzed is drawn through the reagent in an impinger of LAAPCD design, and the ozone present in the sample air liberates iodine from the iodide reagent. The quantity of iodine liberated is determined by titrating the reagent with sodium thiosulfate solution. The sodium thiosulfate used in the titration is standardized with potassium dichromate solution. The procedure used by the LAAPCD was modified in January 1974 to include the use of dry ice in the standardization of the dilute sodium thiosulfate, to improve the precision of the titrations. This does not affect the mean values obtained with this procedure (M. Imada, personal communication).

These iodometric calibration methods are based on the assumption that there is a stoichiometric reaction between ozone and the iodine in the various potassium iodide procedures. Three essentially independent methods have been used to test the accuracy of this assumption: measuring the absorption of ultraviolet radiation at 254 nm by ozone in air, measuring the absorption of infrared radiation at 9,480 nm by ozone in air, and determining the ozone concentration in air by titration with nitric oxide.

The first two determinations by radiation absorption require accurate measurements of the extinction coefficients of ozone (a measurement of the absorption efficiency of the incoming radiation at a maximal absorption wavelength) in the ultraviolet and the infrared. Three different principles have been used over the last 20 years to measure the extinction coefficient of ozone in the ultraviolet at 254 nm: manometric, decomposition stoichiometry, and gas-phase titration. The manometric method, which is based on pressure measurements of gaseous ozone, requires (in at least one case[29]) a substantial and somewhat uncertain correction for decomposition; and the method of decomposition stoichiometry depends on the pressure change that accompanies the decomposition of ozone to oxygen, $2O_3 \rightarrow 3O_2$. Clyne and Coxon[12] determined ozone concentrations in a flow tube by titration with nitric oxide, a method essentially equivalent to gas-phase titration. These methods with their results are summarized in Table 6-6. The best value for the ultraviolet extinction coefficient at 254 nm is considered to be 134 cm^{-1} atm^{-1} at O° C and 1 atm (STP).[20]

TABLE 6-6 Ozone Extinction-Coefficient Measurements

Authors	Extinction Coefficient, cm^{-1} atm^{-1} STP, base 10	Method[a]
Inn and Tanaka[29]	133	Manometric
Hearn[22]	134	Decomposition stoichiometry
DeMore and Raper[15]	135	Decomposition stoichiometry
Griggs[19]	132	Manometric
Clyne and Coxon[12]	136 (250 nm)	Gas-phase titration
Becker, Schurath, and Seitz[4]	135	Manometric

[a] Method used to establish the ozone concentration.

Measuring the Absorption by Ozone in Air of Ultraviolet Radiation at a Wavelength of 254 nm. With this value for the extinction coefficient, a laboratory photometer is used to measure the primary reference concentration of the ozone in the gas stream used for calibration.[14] Figure 6-3 gives a schematic for this arrangement. Highly accurate measurements are attainable with this type of photometer, because the pressure, temperature, and path length of the light beam in the cell can all be controlled and measured precisely.

The ozone concentration, in parts per million, is given by:

$$O_3\ (\text{ppm}) = \frac{10^6\ T}{273Pkl} \log \frac{I_o}{I_t}, \tag{6-4}$$

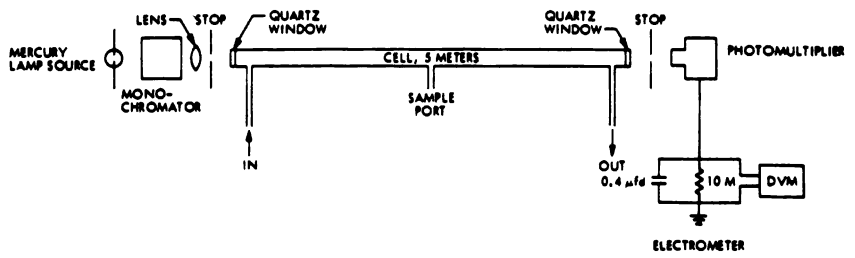

FIGURE 6-3 Absolute ultraviolet ozone photometer. Reprinted with permission from DeMore and Patapoff.[14]

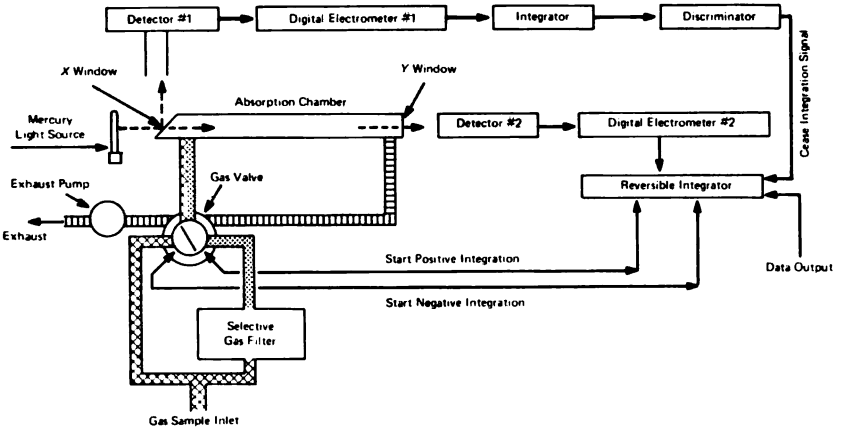

FIGURE 6-4 Dasibi ozone monitor, block diagram.[13]

where T = temperature, K,
P = total pressure, atm,
k = extinction coefficient, 134 cm^{-1} atm^{-1} at STP for ozone,
l = path length, cm,
I_o = intensity with carrier gas only, and
I_t = intensity with O_3 present.

Because, at concentrations smaller than parts per million, the radiation intensities, I_o and I_t, have to be measured with very great precision, the determination with absolute photometry requires a physical-chemistry laboratory with an experienced staff.

Calibrations at monitoring sites, therefore, require a transferable standard. The use of a commercially available photometer that displays the difference in signals between the ultraviolet light absorbed at 254 nm with and without ozone in the sample cell is one approach. It has proved to be stable in performance, even after transport between different laboratories.[16,26] A schematic diagram of the Dasibi Model 1003-AH ozone monitor is shown in Figure 6-4. The operating principle of the Dasibi instrument has been described by Behl,[5] Bowman and Horak,[6] and Hodgeson *et al.*[26] An evaluation of this instrument has been carried out by de Vera, Jeung, and Imada.[17]

As it enters the instrument, the sample is diverted by the valve into a chemical scrubber that changes any ozone present to oxygen. This

ozone-free sample then passes through an absorption chamber 71 cm long, where a detector measures the amount of ultraviolet light transmitted through it at 254 nm. The integrated intensity of the transmitted light is digitized and stored electronically as the reference signal. On completion of this measurement, the valve is opened, and the absorption chamber is flushed with the ozone-containing sample. The digital counter now records the reduction of ultraviolet light due to the ozone in the sample. This value is subtracted from the stored reference measurement, and the difference is displayed in concentration units equivalent to ozone at parts per million.

The ozone concentration as measured by the Dasibi ozone meter is in principle given by Equation 6-4, which can be expressed in terms of the instrument itself as:

$$O_3 \text{ (ppm)} = \frac{22.4T}{kP} \ln \frac{S}{S-R}, \tag{6-5}$$

where T = temperature, K,
S = span setting of instrument, $\propto I$,
R = instrument reading, $\propto (I_0 - I_i)°$,
k = extinction coefficient, 134 cm^{-1} atm^{-1} at STP for ozone, and
P = pressure, atm.

At low ozone concentrations (R small, compared with S), the above expression can be simplified as follows:

$$O_3 \text{ (ppm)} = (22.4\ T/kP)(R/S). \tag{6-6}$$

The quantity $22.4T/kP$ is the theoretical absolute span setting of the instrument. In practice, calibration against the ozone standard is required for maximal accuracy, both because the length of the light path is altered by possible multiple reflections off the inner wall and because the pressure, temperature, and absorption inside the instrument cannot be precisely determined. Hodgeson *et al.*[26] compared a Dasibi instrument spanned according to Equation 6-4 with the absolute photometer. they obtained the following relationship when repeated (61) measurements were made on different days at ozone concentrations of 0.05–2.5 ppm:

$$O_3 \text{ (phot)} = (1.05 \pm 0.01)O_3 \text{ (Dasibi)} + (0.025 \pm 0.013). \tag{6-7}$$

When the span setting of a Dasibi instrument was adjusted to agree with an absolute laboratory photometer and repeated measurements were made on different days, the following relationship with the absolute photometer was obtained:

$$O_3\,(\text{phot}) = (0.999 \pm 0.003)O_3\,(\text{Dasibi}) - (0.007 \pm 0.002). \quad (6\text{-}8)$$

The uncertainties refer to 95% confidence limits. These small error limits are indicative of the high precision of the readings obtainable from both instruments.

Measuring Absorption by Ozone in Air of Infrared Radiation at a Wavelength of 9,480 nm. This method, which is identical in principle with measurement of the absorption in the ultraviolet, requires an accurate measurement of the extinction coefficient of ozone at a wavelength of 9,480 nm. Making this measurement in the infrared energy region is more complicated, because the determination of the extinction coefficient is more strongly influenced by temperature, pressure, and instrumental characteristics, such as spectral slit width. Also, the extinction coefficient is much smaller than in the ultraviolet. This means that, for measurements of concentrations smaller than parts per million, the absorption-path length in the infrared has to be about 100 times greater than the 1-m ultraviolet cell (see Figure 6-3). All these factors create problems that detract from the practicality of this method as a primary reference. Nevertheless, at laboratories experienced in this technique, accurate measurements have recently been made that are in close agreement with the values obtained by absolute ultraviolet photometry.[42] Using the Dasibi ozone meter as a transfer standard, the following agreement has been obtained:

$$O_3\,(\text{Dasibi}) = (0.99 \pm 0.02)O_3\,(\text{IR}) + (0.016 \pm 0.011). \quad (6\text{-}9)$$

The uncertainties are within one standard deviation. Because ultraviolet photometry carried out with the Dasibi instrument is relatively simple, it may be advisable to calibrate even infrared analyzers by means of the ultraviolet transfer standard.

Determination of Ozone in Air by Titration with Nitric Oxide This calibration technique[25,43] is based on the application of the rapid gas-phase reaction between nitric oxide and ozone to produce a stoichiometric quantity of nitrogen dioxide, according to the following reaction:

$$NO + O_3 \rightarrow NO_2 + O_2. \qquad (6\text{-}10)$$

The reaction rate is extremely high, so that, even at concentrations smaller than parts per million, the reaction is virtually complete after a few seconds when there is an excess of nitric oxide present. Under these conditions, the amount of ozone added during the titration is equivalent to the amount of nitric oxide consumed and to the amount of nitrogen dioxide formed. The accuracy of this calibration method depends critically on the accurate measurements of the nitric oxide concentration and on the nitric oxide and ozone flow rates.

Nitric oxide at about 50 ppm compressed with very pure nitrogen in gas cylinders is provided for this purpose, and the true concentration is established by comparison with that of a compressed-gas tank that can be obtained from the National Bureau of Standards, as a standard reference material. The nitric oxide meter is calibrated repeatedly at several concentrations of nitric oxide, and the mass flow meters are recalibrated often with absolute bubble meters.

The titration is shown schematically in Figure 6-5. Nitric oxide, compressed with nitrogen in a gas cylinder, is then metered into the apparatus along with the clean-air stream but without the ozone. In this way, the initial nitric oxide concentration is measured with the nitric

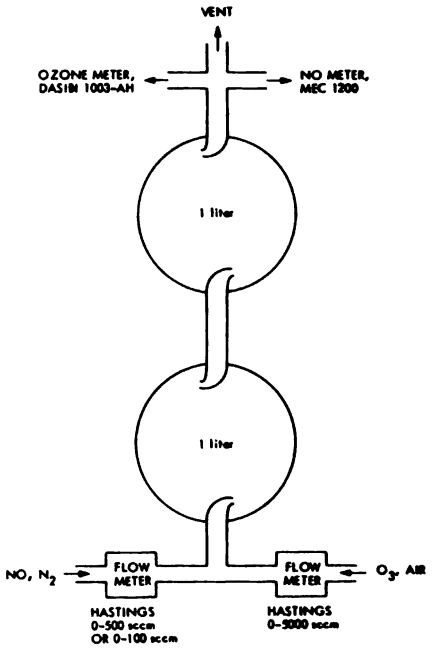

FIGURE 6-5 Gas-phase titration for determining ozone. Reprinted with permission from DeMore and Patapoff.[14]

oxide meter. When the initial nitric oxide concentration is established, the lamp shield in the ozone generator is withdrawn to various settings, and the final nitric oxide concentrations are measured. The initial nitric oxide concentration, the reaction residence times, and the maximal ozone concentration added are established such that the unreacted ozone is negligible (i.e., <1%). Under these conditions, the measured decreases in nitric oxide concentrations then determine the ozone concentrations for various generator settings and serve to calibrate the ozone source. The ozone source may then be used to calibrate ozone meters. As an option, the nitrogen dioxide concentration produced can be monitored at the same time. In fact, this is one way to calibrate nitrogen dioxide meters conveniently.

Using the Dasibi ultraviolet ozone analyzer as a secondary reference standard, DeMore and Patapoff[14] tested the prediction that the observed change in nitric oxide concentration (ΔNO) would equal the ozone concentration introduced and measured by ultraviolet absorption. They found good agreement in which

$$[O_3]_{UV} = (1.00 \pm 0.05)\,\Delta NO - (0.00 \pm 0.01). \qquad (6\text{-}11)$$

Similar conclusions were recently reported both by Hodgeson *et al.*[26] and by Paur.[41] These findings provide further validation of the ultraviolet method for calibrating air monitoring instruments and establish gas-phase titration as an alternative primary reference method.

PRINCIPLES OF MONITORING

Most of the atmospheric oxidant and ozone data—as well as the experimentally determined exposure data for vegetation, animals, and humans—have been obtained with analyzers that sample and record the ambient concentrations almost continuously during the period of observation. The response times are usually acceptable for fixed-station monitoring, because data describing hourly averages are sufficient. Faster responses are needed, however, for studying chemical reaction rates, retention on inhalation, sampling while in motion (as from aircraft), and expediting calibrations. The response times required are therefore a function of the resolution needed.

Definitions of Resolution and Response Times

Resolution: the ability to separate two closely occurring events in space or time at a signal-to-noise ratio of 2:1, expressed as percent of full scale.

Signal-to-noise ratio: the ratio of the magnitude of the response due to the pollutant concentration to the magnitude of unwanted, spontaneous, short-term responses not caused by variations in pollutant concentration.

Response times: see Figures 6-6 and 6-7.

Lag time (initial response time), t_1: the interval between the time t_o, when a step change (increase or decrease) in pollutant concentration is made, and the time t_i, when the instrument indicates a response equal to twice the noise:

$$t_1 = t_i - t_o. \tag{6-12}$$

Time to 95%, t_{95}: the interval between the time t_o, when a step *increase* in pollutant concentration is made, and the time t_{95}, when the instrument indicates a response equal to 95% of the step increase:

$$t_{95} = t_{95} - t_o. \tag{6-13}$$

Similarly, t_{90} corresponds to the time to indicate 90%, and t_{100} the time to indicate 100% of the step increase in pollutant concentration.

Time to −95%, t_{-95}: the interval between the time t_0, when a step

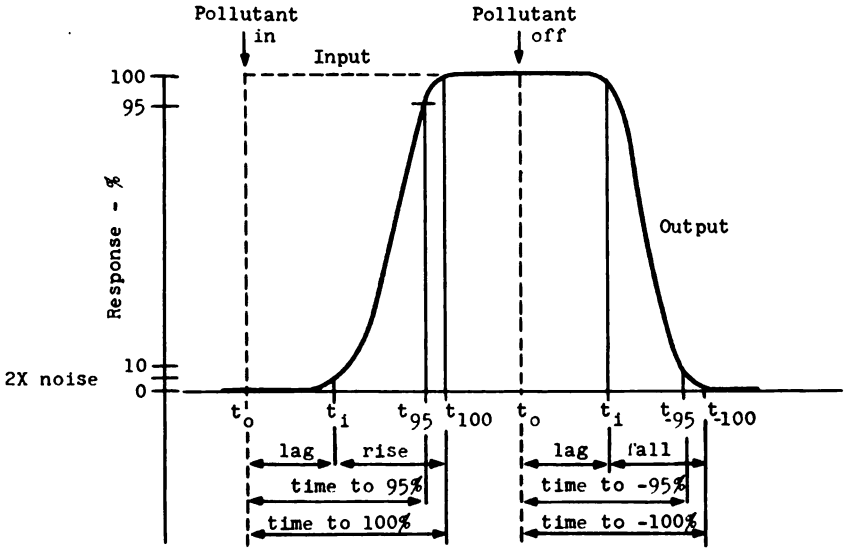

FIGURE 6-6 Visual representation and interpretation of delays in analyzer response. Reprinted with permission from Mueller *et al.*[39]

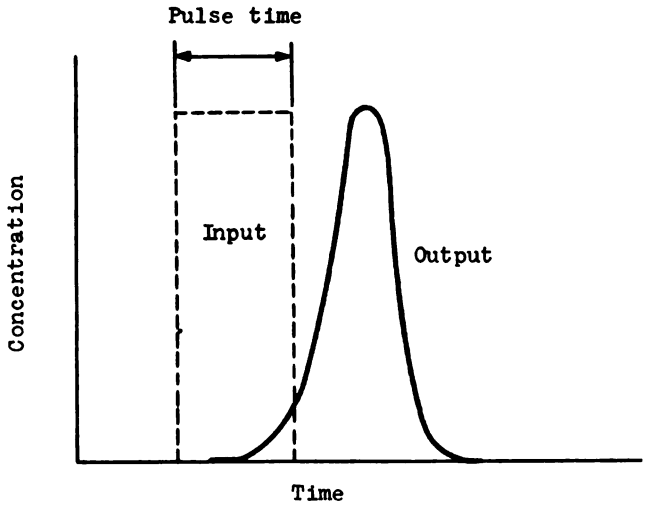

FIGURE 6-7 Diagram of pulse time. Reprinted with permission from Mueller *et al.* [39]

decrease in pollutant concentration is made, and the time t_{-95}, when the instrument indicates -95% of the step decrease:

$$t_{-95} = t_{-95\%} - t_0. \tag{6-14}$$

Similarly, t_{-90} corresponds to the time to indicate -90%, and t_{-100} to the time to indicate -100% of the step decrease in pollutant concentration.

Rise time, t_r: the interval between the time of a response equal to 100% of the step increase in pollutant concentration (t_{100}) and the lag time (t_1):

$$t_r = t_{100} - t_1. \tag{6-15}$$

Fall time, t_f: the interval between the time the instrument indicates -100% of the step decrease in pollutant concentration (t_{100}) and the lag time (t_1):

$$t_f = t_{-100} - t_1. \tag{6-16}$$

The fall time does not necessarily equal the rise time.

Pulse time: the minimal time a pollutant concentration must persist

for the analyzer to register a peak response equal to the pollutant concentration (see Figure 6-6).

For any event to be accurately recorded, it must persist for the pulse time of the instrument. This time is equal either to the rise time or to the time to 100% response, depending on the design of the instrument. For accurate data from aircraft sampling plumes, for example, it is necessary to obtain rise times of a few seconds or less. This is a very fast response for an analyzer and has only recently become possible for ozone measurements.

The analytic principles that have been applied to accumulate air quality data are colorimetry, amperometry, chemiluminescence, and ultraviolet absorption. Colorimetric and amperometric continuous analyzers that use wet chemical techniques (reagent solutions) have been in use as ambient-air monitors for many years. Chemiluminescent analyzers, which measure the amount of chemiluminescence produced when ozone reacts with a gas or solid, were developed to provide a specific and sensitive analysis for ozone and have also been field-tested. Ultraviolet-absorption analyzers are based on a physical detection principle, the absorption of ultraviolet radiation by a substance. They do not use chemical reagents, gases, or solids in their operation and have only recently been field-tested. Ultraviolet-absorption analyzers are ideal as transfer standards, but, as discussed earlier, they have limitations as air monitors, because aerosols, mercury vapor, and some hydrocarbons could interfere with the accuracy of ozone measurements made in polluted air.

Advanced electro-optical methods (e.g., laser resonance absorption) capable of measuring average concentrations over long distances still require extensive research and field testing to demonstrate their practical application to ozone monitoring. Because electro-optical methods have not yet been widely used, they are not discussed further here.

Colorimetric Analyzers

Colorimetric analyzers spectrophotometrically measure the increase in color (absorbance) of a solution resulting from contact with a measured volume of air. The absorbance is linearly proportional to the concentration of the colored species, within known limits. Continuous colorimetric analysis of total oxidants is carried out with a solution of neutral buffered potassium iodide (KI). In 1953, Littman and Benoliel[35] developed the first colorimetric oxidant recorder to come into general use. Instruments of this design, using a 20% neutral buffered potassium iodide solution, later changed to 10%, were incorporated into the Los

Angeles County Air Pollution Control District (LAAPCD) air monitoring network in the early 1950's.[51]

The continuous colorimetric instrumental method is described in detail by the Intersociety Committee[31] and the American Society for Testing and Materials,[48] and colorimetric and amperometric analyzers are discussed extensively in the papers both of Tokiwa *et al.*[50] and of Hodgeson.[24]

A typical colorimetric analyzer is illustrated schematically in Figure 6-8. Sample air is drawn at a metered rate into a contact column, where the air is scrubbed with a metered flow of potassium iodide buffered at a pH of 6.8. The reaction of oxidants with the potassium iodide solution produces the yellow triiodide ion (I_3^-). The colored solution flows to a colorimeter cell, where the absorbance of the triiodide ion is measured

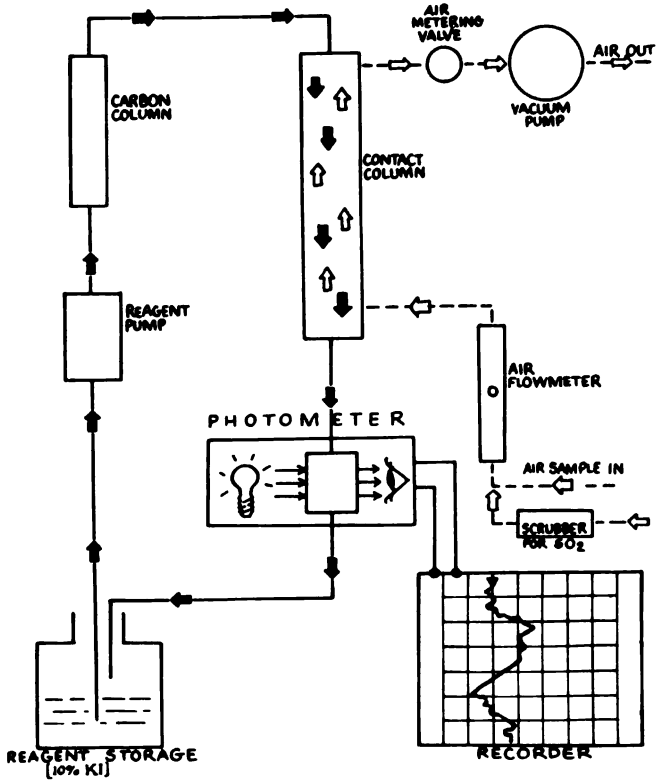

FIGURE 6-8 Colorimetric oxidant analyzer. Reprinted with permission from Tokiwa *et al.*[50]

at 354 nm. The photometer signal is then electronically recorded as oxidant at parts per million. The reaction of potassium iodide with ozone at a pH of 6.8 ±0.2 is given by Reaction 6-17:

$$O_3 + 3KI + H_2O \rightarrow K^+I_3^- + 2KOH + O_2. \qquad (6\text{-}17)$$

Colorimetric oxidant analyzers operate most reliably at atmospheric ozone or equivalent oxidant concentrations ranging from 20 to 2,000 $\mu g/m^3$ (0.01 to 1.0 ppm). A usable response can be obtained from oxidant concentrations equivalent to ozone at as much as 100 ppm, but at such high concentrations the stability and speed of response are inferior to those attainable at lower concentrations.[31]

When sulfur dioxide is present in the polluted air, it causes a negative interference equal to 100% of an equimolar concentration of oxidant. The response to the pollutant, nitrogen dioxide, varies with the reagent formulation and scrubber design. For 10% potassium iodide, nitrogen dioxide produces a positive interference of approximately 21%; for 20% potassium iodide, the interference produced is approximately 30%.[50]

Filters and scrubbers have been used for removing sulfur dioxide, but detailed information about their performance is not available. A brief review of performance characteristics of sulfur dioxide scrubbers was given by Hodgeson.[24] One device consists of glass-fiber strips impregnated with chromium trioxide and sulfuric acid.[44] An even better scrubber consists of a bed of small pellets of porous chromatography-grade firebrick or alumina impregnated with chromium trioxide.[39] These scrubbers, which convert only a portion of the ambient nitric oxide to nitrogen dioxide, require an additional correction factor. Despite these drawbacks, this is the most generally used technique for removing sulfur dioxide interference, particularly during periods of high sulfur dioxide concentration. When sulfur dioxide concentrations are less than 20% of the nitric oxide concentration, this filtering system is not recommended.[31] In this case, the correction for nitrogen dioxide interference in the oxidant reading can be determined by measuring the instrument response to a known stream of nitrogen dioxide while analyzing for nitrogen dioxide in the atmosphere; then the appropriate subtraction can be made.[48]

Amperometric Analyzers

Amperometric analyzers are often referred to as "coulombmetric" analyzers. Coulombmetry is a mode of analysis in which the quantity of electrons (charge) necessary to oxidize or reduce a desired substance

is measured. Because it is the current and not the charge that is measured by these instruments, "coulombmetric" analyzers are more properly called amperometric analyzers. This principle has been implemented for oxidant monitoring with either galvanic (Hersch) cells[23] or electrolytic (Brewer or Schulze) cells.[8,36,46] A schematic of this instrument with a Brewer cell is shown in Figure 6-9. (Instruments with Hersch or Schulze cells are not widely used.)

The operational principles of the amperometric analyzer are described

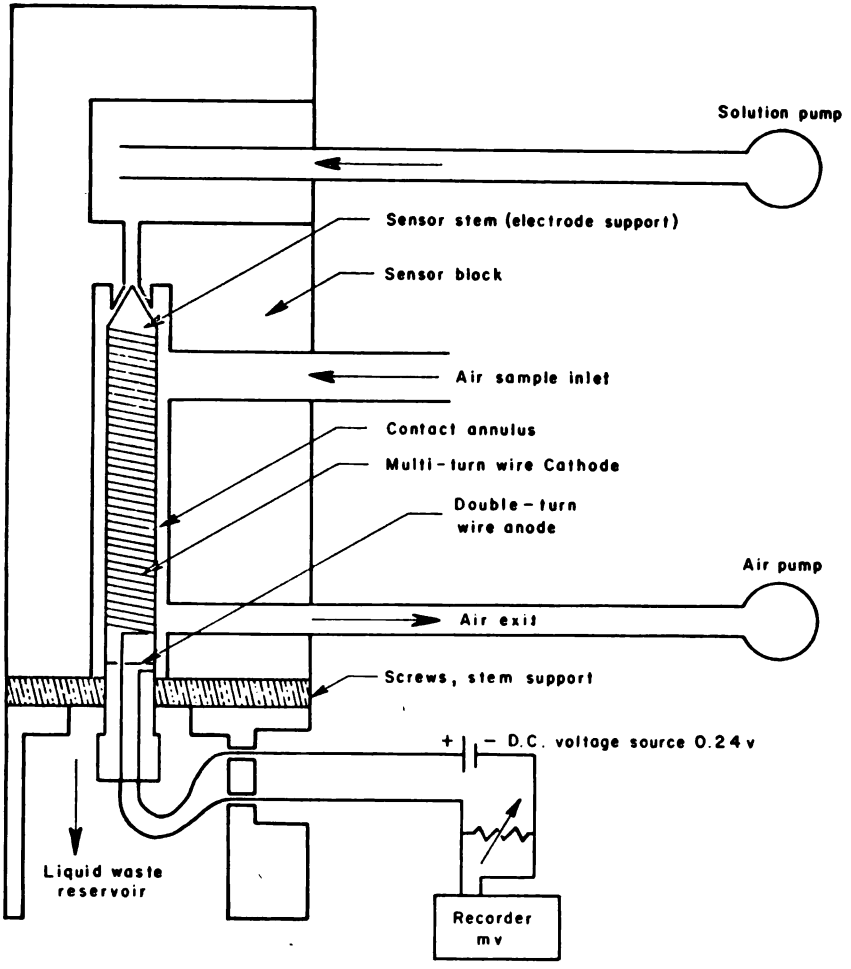

FIGURE 6-9 Amperometric analyzer with Brewer electrolytic cell. Reprinted with permission from Intersociety Committee.[30]

in the Intersociety Committee's manual of methods for ambient-air sampling and analysis.[30] The contactor-sensor consists of a rod wound with many turns of a fine platinum wire (cathode) and two turns of a heavier wire (anode) axially mounted in a plastic block with a hole about 0.6 cm in inside diameter and 5 cm long. The reagent is usually a mixture of 2% potassium iodide and 5% potassium bromide (0.01 M) buffered at a pH of 6.8 with 0.026 M disodium hydrogen phosphate and 0.018 M sodium dihydrogen phosphate.[50] Sample air and reagent pass concurrently through the annular space (about 0.2 cm) between the support rod and the cylinder wall. Ozone in the air transferred to the reagent reacts with the iodide to produce triiodide ions, which in turn react with the hydrogen, polarizing the cathode. As a result of these reactions, current flows to repolarize the cathode in proportion to the amount of hydrogen removed. This current, which is directly proportional to the ozone concentration, is recorded.[8,36]

The presence of nitrogen dioxide in the sample causes a positive interference. When the concentration of the potassium iodide reagent is 2%, then the interference is about 6% of the nitrogen dioxide concentration. This is typical for amperometric analyzers that have been marketed specifically for ozone or oxidant measurements.[50] The detector's specific response to nitrogen dioxide depends on the instrument design, the composition of the sensing solution, the operating conditions, and other unknown factors.[30] Therefore, the specific response for each detector should be determined experimentally. The oxidant measurements can be corrected for nitrogen dioxide interference by subtracting the response due to the nitrogen dioxide from the total detector response.

Sulfur dioxide in the sample causes a negative interference of approximately 1 mole of ozone per mole of sulfur dioxide, because it reduces the iodine formed by ozone back to potassium iodide. When sulfur dioxide concentrations do not exceed those of the oxidants, a method commonly used to correct for its interference is to add the amount of sulfur dioxide determined by an independent method to the total detector response. A second method is to remove the sulfur dioxide from the sample stream with solid[39,46,55] or liquid[23] chromium trioxide scrubbers. Because the data on the performance or these sulfur dioxide scrubbers are inadequate, the performance for each oxidant system must be established experimentally.

Because of the interference problems with both colorimetric and amperometric analyzers, they are being replaced by instruments based on other principles. (Colorimetric analyzers are no longer commercially available.) One such recently improved technique developed for the specific detection and measurement of ozone is the detection of the

absorption of ultraviolet radiation by the ozone molecule. Another is the measurement of the chemiluminescence produced when ozone reacts with a specific gas or solid.

Chemiluminescence

In 1965, a gas-phase chemiluminescent reaction between ozone and ethylene was reported by Nederbragt *et al.*,[40] and the sensitivity of this technique was later improved by Warren and Babcock.[54] The reaction between ozone and ethylene yields chemiluminescent emission in the 300- to 600-nm region, with maximal intensity at 435 nm.[49] The intensity of this emission is directly proportional to the ozone concentration.

A diagram of a typical gas-phase (ozone-ethylene) chemiluminescent ozone analyzer[13] is shown in Figure 6-10. The detector responds linearly to ozone concentrations between 0.003 and 30 ppm; no interferences were initially observed.[49] More recently, however, it has been established that, as the relative humidity goes from 0 to 60% and the temperature from 20° to 25° C, water vapor produces a small positive signal that results in an increase of about 8% in the ozone concentration measurement. This potential source of error can be minimized by using humidified, rather than dry, ozone in air streams when calibrating.

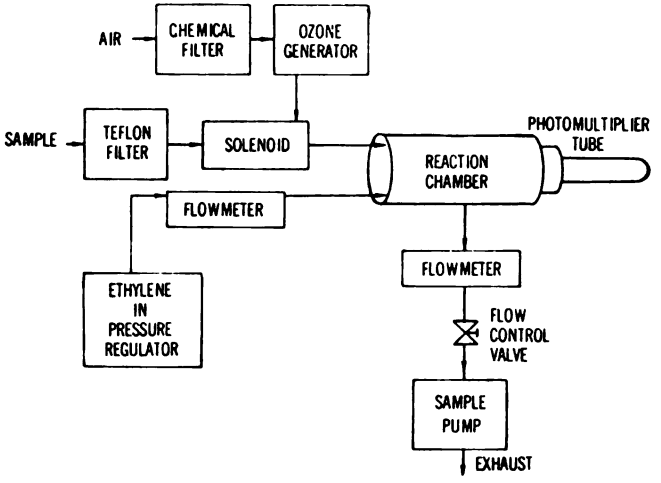

FIGURE 6-10 Gas-phase (ozone-ethylene) chemiluminescent ozone monitor, block diagram. Reprinted with permission from Coloff *et al.*[13]

Slight improvements in sensitivity can be achieved by cooling the phototubes used to detect the emitted light or by increasing the ethylene flow rate. Chemiluminescence produced by the reaction of ozone with ethylene has been designated by the EPA as the reference method for monitoring ozone.[28,53] Several different commercially produced instruments are available.

OTHER OXIDANTS AND PRECURSORS

Besides ozone, the main indicator of photochemical pollution, other important concomitant products are peroxyacetylnitrate (PAN), hydrogen peroxide, nitrogen dioxide, hydroxyl radicals and various aldehydes that are both products and primary pollutants, particles, sulfates, nitrates, ammonium, chloride, water, and various types of oxygenated organic compounds. The most important precursors of photochemical pollution are nitric oxide and hydrocarbons. The measurement procedures for the hydrocarbons are not as highly developed as those for ozone and the nitrogen oxides.

Many deleterious effects have been associated with photochemically polluted air: ozone is definitely associated with respiratory problems, plant damage, and material damage; PAN has definitely been associated with plant damage, and some other members of this class of chemical compounds have been associated with eye irritation; the hydroxyl radical is considered to be an important factor in the conversion of gas-phase intermediates to end products, such as sulfur dioxide to particulate sulfate; the particulate complex is responsible for haze formation and has also been associated with eye irritation and respiratory effects. The aldehydes have been associated with eye irritation. Ozone and PAN themselves do *not* cause eye irritation. For purposes of control, much more research is needed, in order to relate the laboratory data about the concentrations of these various materials that have significant effects to their formation in the atmosphere from emission and their atmospheric distribution. The lack of convenient measurement methods has hindered progress in gaining this understanding.

The chief precursors for both oxidant and suspended particulate matter formation in the atmosphere, which are directly emitted into the atmosphere, are nitrogen oxides, hydrocarbons and their derivatives, ammonia, and sulfur dioxide. The measurement of particulate components is discussed in Chapter 2. This section describes briefly the measurement of nitrogen oxides, hydrocarbons, free radicals, and other precursors.

Nitrogen Oxides

The technology for the routine measurement of the nitrogen oxides (nitrogen dioxide and nitric oxide) is fairly well advanced. The EPA is on the verge of officially proposing that chemiluminescence produced by the reaction of nitric oxide with ozone be the reference method for nitrogen dioxide.[28] This method is even more suitable for nitric oxide. Because no national air quality standard has been promulgated for nitric oxide, no reference method will be specified. However, its measurement in the atmosphere is crucial for establishing the relation of its emission to the formation of atmospheric ozone and other photochemical oxidants.

Before 1970, most of the data for nitrogen oxides were obtained by continuous measurements with a colorimetric analyzer that was similar in principle to the colorimetric oxidant analyzer shown in Figure 6-8. The scrubbing agent is a mixture of *N*-(1-naphthyl)ethylenediamine, sulfanilic acid, and acetic acid in aqueous solution. The color is produced when both nitrogen dioxide and nitrites react with this reagent to form an azo dye. The color is not affected by nitric oxide in the air sample.

The quantification of the nitrogen dioxide depends on the acceptance of an empirical factor that relates the response of this reagent such that 1 mole of nitrogen dioxide scrubbed from the gas phase produces the same amount of color as 0.72 mole of nitrite added in solution. With alterations in reagent composition and scrubber design, this factor may change. For most of the air monitoring activities in the United States using this reagent, this factor appears to have been verified.[32] Evaluations remain to be conducted with gas-phase titration and a known ozone source.

To determine nitric oxide by this method, the NO is oxidized to NO_2 by passing the air sample through a reaction vessel containing either potassium permanganate or chromium trioxide.[39] For an accurate measurement, however, any nitrogen dioxide originally present must first be selectively removed by passing the air sample through a triethanolamine scrubber.[34] This precaution has, unfortunately, not been the practice. Rather, the air sample containing both nitric oxide and nitrogen dioxide has been passed first through the oxidizer and then through the reagent scrubber, which gives a measurement of total nitrogen oxides (NO_x). The assumption is made that no nitrogen dioxide is lost.

In another technique, the air stream leaving the nitrogen dioxide reagent scrubber is taken to the oxidizer and then to a second reagent scrubber. This gives separate measurements for nitrogen dioxide and nitric oxide. The assumption is that all the nitrogen dioxide is retained in the first reagent scrubber and no nitric oxide is lost. Neither of the assumptions made for

these methods of measuring nitric oxide is completely correct. For this reason, there is a great deal of uncertainty about the amount of error in existing data banks. The nitrogen dioxide data, on the other hand, are believed to be reasonably accurate, particularly where there has been proper instrument calibration and maintenance.[39]

The diurnal patterns of ozone, nitric oxide, and nitrogen dioxide concentrations observed during photochemical oxidant episodes in California have been confirmed by smog-chamber studies. There may be, however, a decrease in reliability with decreasing concentration of values less than 0.1 ppm that were measured by the colorimetric method. The magnitude of these uncertainties among the various monitoring networks in the United States has still to be assessed.

In the chemiluminescent detection of nitrogen oxides, a constant source of ozone reacts with a metered air sample containing nitric oxide. Fontijn *et al.*[18] suggested that this method could also be used for ozone detection by using a constant nitric oxide source for reaction with ozone in the air sample. The ozone–nitric oxide reaction is carried out at reduced pressure, to avoid quenching the chemiluminescent reaction. Detection of the emission in the spectral region involved (600–3,000 nm) requires using a near-infrared-sensitive photomultiplier tube. The noise of such a photomultiplier tube is reduced by cooling it to about − 20° C.[2]

In the measurement of nitrogen dioxide with this technique, it is thermochemically converted to nitric oxide by reaction with molybdenum at about 200° C. The extent of possible interferences at various monitoring sites from nitrogen compounds other than ammonia, which does not interfere unless the temperature is considerably higher than 200° C, remains to be assessed. The instrumentation of this procedure is inherently more reliable than the original colorimetric analyzers. Unfortunately, the mutual equivalence in monitoring situations of data obtained by these two techniques has not yet been evaluated. This is particularly important for the data from California, where the colorimetric method has been used for more than 20 yr.

Two primary calibration standards are available for nitrogen oxides. One is a nitrogen dioxide generator in which the source is a permeation tube, certified by the National Bureau of Standards, in a temperature- and flow-controlled gas-diluter. The amount of nitrogen dioxide generated is determined by the loss in weight of the permeation tube. The other standard is a cylinder of compressed nitric oxide in oxygen-free nitrogen certified in the range of 50 ppm by the National Bureau of Standards. To obtain concentrations smaller than 1 ppm, this cylinder has to be connected to a dilution apparatus carefully regulated for flow and temperature.

The calibration of a chemiluminescent analyzer using these standards is verified when both the nitrogen dioxide and nitric oxide channels respond similarly to charges from the cylinder of diluted nitric oxide. Agreement with the response of the nitric oxide channel should also be obtained when the standard nitric oxide mixture is titrated with a previously established source of ozone. Chemiluminescent instruments have simplified monitoring of nitrogen oxides, but accurate calibration requires well-trained personnel.

Hydrocarbons

For the measurement of the hydrocarbon precursors of photochemical oxidants, the naturally occurring methane must be separated from the other so-called nonmethane hydrocarbons. Only one procedure, gas chromatography coupled with flame ionization detection, is available for this separation and measurement. Although instrumentation for routinely accomplishing this process is commercially available, its maintenance (continued operation) requires a degree of operational know-how that may be too costly for most public agencies in the United States to support. Consequently, the data currently are insufficient to relate the occurrence of photochemical oxidants and ozone accurately to some of their most important precursors, the nonmethane hydrocarbons.

In addition, it is widely recognized that, on the basis of chamber studies, some of the nonmethane hydrocarbon compounds are far more important than others in causing the formation of photochemical oxidants and aerosols. These are referred to as the reactive hydrocarbons. To date it has been possible to measure them only with a more sophisticated gas-chromatographic process than described above. Therefore, their measurement in the atmosphere has been limited to research or to short-term episodic types of studies. Although these are useful for understanding the phenomena of photochemical-pollution formation, they have been too costly for determining the effectiveness of hydrocarbon control programs with respect to the changes in the occurrence and concentration of reactive hydrocarbons in air. Consequently, there is very little information about hydrocarbon compounds in the atmosphere that may be precursors for the formation of atmospheric haze.

Particulate Sulfates and Nitrates

The sulfate and nitrate content of atmospheric particles comes primarily from the conversion of sulfur dioxide and nitrogen dioxide. Photochemically initiated atmospheric reactions and transient free radicals are

often associated with this process (Chapter 2). At over 3,500 locations in the United States, the total suspended particulate matter is regularly sampled at least once per week for a 24-h period.[28] The particles are usually collected on glass-fiber filter mats that are then sent to a central laboratory. There the sulfate and nitrate contents are extracted with hot water and determined by standardized chemical analytic procedures. Recently, a detailed review of these procedures has covered their reliability, their limitations, and the alternatives available for their improvement (emphasizing sulfates).[38]

There are several potential sources of error in these methods. The filters routinely used have a relatively high and somewhat variable sulfate content, so that, at concentrations lower than 10 $\mu g/m^3$ and sampling periods less than 24 h, the reliability of the sulfate measurement is reduced. Several different types of filtering media adsorb sulfur dioxide during the first few hours of sampling; this alters the amount of sulfate observed. This interference can become critical when sampling periods are less than 24 h and the concentration ratio of sulfur dioxide to sulfate is greater than 5:1. Interference can also be introduced by hot-water extraction when reduced sulfur compounds like sulfite are present, because they are oxidized to sulfates in this process. Another possible error source is that some of the various analytic procedures used for sulfate determination may be influenced by other substances also present in the particulate matter.

Current developments for minimizing these limitations in the analytic procedures are selecting for sampling filtering media that have low blanks and do not adsorb significant amounts of sulfur dioxide and nitrogen oxides and designing analytic methods that are specific for sulfates and nitrates in the presence of other particulate substances. In addition, more sensitive procedures for studying short-term (hourly) variations are being developed. These procedures will permit the investigation of the relation of the formation and occurrence of these particulate components to atmospheric reactions and to the acute effects when inhaled and when deposited on surfaces.

Other Substances

As shown in Table 6-1 (and reviewed in Chapter 2), other substances monitored occasionally are important in the photochemical-oxidant milieu. These are the peroxyalkylnitrates and peroxyarylnitrates and the aldehydes.

The peroxyalkylnitrates and peroxyarylnitrates have been monitored by directly injecting the air at the sampling site into a specifically designed gas chromatograph. Aldehydes (formaldehyde is the major one in

the atmosphere) are generally sampled in liquid absorbers with reagents that develop a color. These are sensed continuously with an instrument comparable in design with the colorimetric oxidant analyzer (Figure 6-8). To obtain information about specific aldehydes, the material collected in the absorber is sent to a central laboratory where individual aldehydes are analyzed by gas chromatography and mass spectroscopy.

A number of significant oxygenated organic particulate compounds and gas-phase free radicals are formed by the reactions of gas-phase hydrocarbons (see Table 6-1 and Chapter 2). The measurement methods for these substances are complicated and in the research stage. Their description is beyond the scope of this chapter. It is of major importance to develop methods for measuring hydroxyl and peroxyhydroxyl radicals, as well as the various oxygen species formed with ozone (see Chapter 12).

EVALUATION

In the final analysis, the purpose of measurement is to provide numerical values as the basis both for making policy decisions and for enforcing regulations. It is critical, therefore, to know whether measurement data are reliable. It is also essential that all the data be intercomparable. This includes data obtained from laboratory studies of chemical reactions, plant and material damage, and animal and human toxicology; from field studies of air quality and vegetation and ecosystem effects; and from population exposures. In all such studies, irrespective of the method used, the measurement of oxidants is based on a "standardized" source of ozone.

Unfortunately, and probably unavoidably, from the earliest research until the present, investigators have not used the same standardization process. Furthermore, standardization practices within different research groups have only rarely followed the meticulous series of in-house calibrations, verifications, and interlaboratory comparisons long prescribed by the community of measurement specialists. Several quantitatively crucial studies, however, have followed sound measurement procedures (see, e.g., Chapter 9).

The chief objective of this section is to provide a perspective concerning the reliability of atmospheric-oxidant data. The expected performance of atmospheric-oxidant monitors is given in Table 6-7. To judge the reliability of measurements, information about the following five factors is required:

(1) Accuracy—agreement with respect to a primary reference standard.
(2) Reproducibility—the precision with which measurements can be

TABLE 6-7 Expected Performance Specifications for Oxidant Monitors[a]

Performance Characteristic	Specification
Range	0–0.5 ppm
Noise	0.005 ppm
Lower detectable limit	0.01 ppm
Interference equivalent	
Each interferent	±0.02 ppm
Total interferent	0.06 ppm
Zero drift, 12 and 24 h	±0.02 ppm
Span drift, 24 h	±0.025 ppm
Lag time	20 min
Rise time	15 min
Fall time	15 min
Precision, standard deviation range	0.01 ppm
as % of 0.08 ppm (air quality standard)	12.5%
Accuracy with respect to primary reference standard	Not specified

[a] Data from U.S. EPA.[52]

repeated within a single measurement group and among different groups in different laboratories.

(3) Interferences—substances that exist simultaneously with ozone and alter the response of the measurement method.
(4) Comparisons among different measurement methods.
(5) Practices and maintenance of measurement operations.

The fundamental factors that govern the accuracy of primary reference standards are discussed in this chapter. Even though an improved reference standard has been advocated, most of the existing air monitoring and laboratory-exposure data have as their reference the potassium iodide procedure used by either the CARB, the LAAPCD, or the EPA. The relationship of these three variations of the potassium iodide procedure to the ultraviolet method is as follows:

$$O_{3\,(\text{CARB})} = 1.29O_{3\,(\text{UV})} - 0.005, \tag{6-18}$$

$$O_{3\,(\text{LAAPCD})} = 0.96O_{3\,(\text{UV})} - 0.032, \tag{6-19}$$

$$O_{3\,(\text{EPA})} = 1.11O_{3\,(\text{UV})} - 0.035. \tag{6-20}$$

Although the differences between these several primary reference

procedures are of some concern, the practice and performance of them since 1952 have been remarkably consistent. For instance, several studies by different investigators and laboratories have shown an average ratio of 1.37:1 between the LAAPCD and CARB methods (with extremes in this value not exceeding 5%).[16] The EPA primary reference procedure with potassium iodide has also been evaluated recently by several laboratories. When the concentration range of ozone was 0.005–0.5 ppm, the standard deviation among the laboratories during a 4-day continuous measurement of the same atmosphere was ±6–10%.

With respect to stabilized ozone generators supplied and calibrated by the National Bureau of Standards, the participating laboratories obtained values that were about 15% lower on the average, at an ozone concentration of about 0.2 ppm.[37] Therefore, although acceptable repeatability within and among laboratories can be achieved even with the potassium iodide bubbler method as a reference procedure, there are unpredictable variables inherent in this procedure that contraindicate its continued acceptance.

Differences in measurement methods include analyzer systems based both on the same and on different measurement principles. The average standard deviation in the performance of different chemiluminescent ozone instruments that are sampling the same ambient air both with and without an added ozone concentration of 0.002–0.5 ppm is 6–10%. Field studies comparing an ultraviolet monitor with several chemiluminescent monitors showed correlation coefficients for hourly averages of 0.80–0.95 between various pairs of instruments. Hourly averages for about 500 pairs of values at ambient ozone concentrations of 0.005–0.100 ppm showed deviations of 3–23% between the average values for paired instruments.

Typical colorimetric and amperometric analyzers have been compared both in the laboratory, with ozone concentrations of 0–0.6 ppm (with and without added nitrogen dioxide at a similar concentration range), and at field locations where the oxidant concentration was 0–0.2 ppm.[50] When both instruments were corrected for nitrogen dioxide interference, the field results showed highly correlated ($r = 0.96$) hourly averages. Nevertheless, the colorimetric readings were consistently 6% higher than the amperometric readings. The responses showed the following relationship:

$$O_3\,(\text{amp}) = 0.942O_3\,(\text{col}) - 0.0038\ \text{ppm}. \qquad (6\text{-}21)$$

To summarize, the results of comparing and evaluating air-oxidant analyzers indicate that, when the use of similar primary reference pro-

cedures is coupled with meticulous operational practices, agreement within about 20–30% can be expected. A monitoring network operated by a single tightly managed group, however, can achieve even better agreement.

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7

Respiratory Transport and Absorption

This chapter discusses the transport and absorption of ozone and other photochemical oxidants within the respiratory tract. It includes lung morphology and flow aspects of respiratory physiology and emphasizes methodologic approaches to modeling.

Although the theory of the uptake of inert gases is well developed, there is no adequate theory for gases like ozone, which are reactive and metabolized by body tissues and fluids. Thus, it is not possible to predict at critical airway sites local tissue dosage that can be reliably related to toxic effects. In this chapter, the background information necessary for the development of a realistic model of lung uptake of reactive gases is presented, and subjects needing further study are identified. A successful model would help in correlating observations in animal and man and estimating the effective dose at reactive sites in man. Combined with a knowledge of biologic mechanisms of toxicity, it would allow the prediction of health effects in man outside the range of conditions in which observations are made.

The radical or reactive intermediates discussed in Chapter 2 are included in the term "photochemical oxidants" for the purpose of this chapter. Of the reactive intermediates discussed, only the hydroperoxy, HO_2, and singlet oxygen, O_2 ($a^1\Delta$), radicals have a lifetime long enough

to allow transport of significant quantities into the lungs before deactivation. Modeling of oxygenated organic aerosols is not considered in detail in this chapter. According to the discussion in Chapter 3, most of the oxygenated organic aerosols have a diameter less than 0.5 μm, the range in which diffusion is the controlling mechanism of deposition. Regional models[134,139] can account for particle deposition and clearance in the tracheobronchial tree and pulmonary regions. Work by Hounam[74] suggests that Landahl's model[92] can be used to predict the deposition of oxygenated organic aerosols that are highly diffusive, as condensation nuclei in the nasopharynx.

This chapter first reviews and discusses selected research on local dose aspects of ozone toxicity, the morphology of the respiratory tract and mucus layer, air and mucus flow, and the gas, liquid, and tissue components of mathematical models. Next, it discusses the approaches and results of the few models that exist. A similar review[65] was recently done to define an analytic framework for collating experiments on the effects of sulfur oxides on the lung. Pollutant gas concentrations are generally stated in parts per million in this chapter, because experimental uptake studies are generally quoted only to illustrate behavior predicted by theoretical models. Chapter 5 contains a detailed discussion of the conversion from one set of units to another.

EFFECTIVE TISSUE DOSE

In this section, sites of action in the respiratory tract are discussed, along with experimental studies of gas uptake in animals. Cumulative dose and dosage at critical sites of action are defined, as well as the general characteristics required for modeling the transport and absorption in the respiratory tract.

Sites of Action

The sites of action and effects of ozone and other photochemical oxidants are described in Chapters 8 and 9. Recent work with primates[42] has suggested that ozone is absorbed along the entire respiratory tract, penetrates more into the peripheral nonciliated airways, and causes more lesions in the respiratory bronchioles and alveolar ducts as the inhaled ozone concentration increases from 0.2 to 0.8 ppm. The most common and most severe tissue damage was observed in the respiratory bronchioles. The ciliated cells in the terminal bronchioles and the Type 1 cells in the epithelial layer of the proximal alveoli of rats were the

primary sites of action of ozone at 0.5 and 0.9 ppm.[125,126] Recent work on rabbits[26] and rats[120] suggests that the mucus layer in the large airways does not completely protect the underlying cells from ozone damage. Specifically, Boatman and Frank noted patches of desquamated ciliated cells along the conducting airways after acute exposures to ozone at 0.25, 0.5, and 1 ppm; these desquamated patches were most often found at bifurcations.

Table 7-1 gives an overview of various irritant and nonirritant gases commonly found in the atmosphere, their solubility in water, and their main sites of action. The Henry's law constant indicates the relative solubility in waterlike lung fluid. Although most of the information goes back to 1924, it is supported and extended by numerous studies of the effects of war gases and industrial irritants.[28,30,31,43,55,56,67,85,148]

TABLE 7-1 Physical Properties of Pollutant Gases and Their Site of Action or Absorption in the Respiratory Tract[a]

Gas	Ambient Concentration, ppm	Henry's Law Const.[b] at 37° C, 1 atm, mole fraction in air / mole fraction in water	Major Site of Action or Absorption[c]
Ammonia[d]	0.02–0.2	3.5	URT
Sulfur dioxide[d]	0.01–0.5	59.7	URT and large bronchi
Hydrogen sulfide[d]	0.03	704	URT and large bronchi
Formaldehyde[d]	0.3	791	URT
Carbon dioxide	330	2,190	PULM
Ozone	0.05–0.5	9,700	TBT and PULM
Nitrogen dioxide	0.05–0.5	Converts to nitric and nitrous acids in water	TBT and PULM
Nitric oxide	0.05–2	33,900	TBT and PULM
Oxygen	209,460	51,800	
Carbon monoxide	1–100	67,400	PULM
Nitrogen	780,840	100,700	PULM

[a] Derived from Haggard.[60]
[b] Data from *International Critical Tables*[143] and Emmert and Pigford.[46]
[c] URT = upper respiratory tract (nose, mouth, pharynx, larynx);
TBT = tracheobronchial tree (ciliated airways);
PULM = pulmonary region (alveolated airways and alveoli).
[d] For these highly water-soluble gases, Henry's law may be assumed to apply over the ambient concentration range.

Studies that have measured the uptake of pollutant and irritant gases in different regions of the respiratory tract of animals and man and in experimental airway models provide the most useful data for development and verification of gas transport models.[1,12,27,33,39,44,45,50–52,75,86,102,107,123,153–155] They generally show that the very soluble nonreactive gases—like ammonia, sulfur dioxide, and formaldehyde—are nearly totally removed by absorption in the nasal passages during normal breathing. Less water-soluble gases—like ozone and nitrogen dioxide—penetrate more peripherally into the lung and are partially exhaled. Total uptake depends on tidal volume, respiration rate, and in some cases initial inhaled concentration. For example, data from dogs[154] show that nearly 100% of sulfur dioxide inhaled through the nose is removed before reaching the first bifurcation, whereas only 27–70% of the ozone is removed in this same region.

Inhalation of gas or aerosol-gas mixtures may shift the site of action of the separate gases. Generally, the absolute quantity of irritant gases absorbed by suspended particles is negligible at ambient gas and aerosol concentrations; however, the irritation may be significantly increased by the transport of soluble gases by particles deeper into the lung. Insoluble particles, like carbon, and partially water-soluble particles may absorb ozone, sulfur dioxide, and other gases in the water phase. Likewise, partially soluble particles with catalytic properties may oxidize absorbed gases and convert them to more toxic chemical forms (e.g., sulfur dioxide oxidized to sulfur trioxide in the liquid phase of particles with the aid of metallic catalysts). Gaseous mixtures that are chemically unreactive in the environment may react rapidly with each other in the special conditions of the respiratory tract to form other toxic compounds. The synergism observed by Bates and Hazucha[20] and by Bell *et al.*[25] when human subjects were exposed to mixtures of sulfur dioxide and ozone may be caused by one of these physicochemical interactions. Another example of synergism related to physicochemical interaction of gases and particles was described by Mezentseva *et al.*,[101] who observed edema in the lungs of animals exposed to hydrochloric acid derived from the hydrolysis of titanium chloride, $TiCl_4$, but not in those exposed to similar concentrations of pure gaseous hydrogen chloride.

Indexes of Critical Dose

To model the uptake of ozone and other gases for establishing dose-response relationships at specific sites, local dose must be accurately defined.[63] In the past, this has not been done for specific sites. Fairchild

and Graham,[47] Stokinger *et al.*,[128] and Stokinger[127] have developed, for the effective dose of ozone, expressions that depend only on the inhaled concentration and exposure time. "Dose" may be the mass or moles of the toxic gas delivered to the site of interest, and the average dose may be measured in micrograms per square centimeter of surface.

For acute exposure in a specific airway, the average rate of flux to the epithelial tissue or mucus layer may be the critical quantity and is measured in micrograms per square centimeter per second (in the steady state the unit is micrograms per square centimeter per breath). Chronic effects are probably related to the time integral over the period of exposure. When sensory receptors are involved in the acute response, the local flux to the small surface areas containing the receptor sites may be crucial.

Controlling Factors in Ozone Uptake

The major respiratory factors in the control of ozone uptake are the morphology (including the mucus layer), the respiratory flow, the physical and chemical properties of mucus, and the physical and chemical properties of ozone. The next two sections discuss models of the morphology and the air and mucus flow. The physical and chemical properties of bronchial secretions have been reviewed by Barton and Lourenco[17] and Charman *et al.*[29] The relevant physical and chemical properties of ozone are its solubility and diffusivity in mucus and water and its reaction-rate constants in water, mucus, and tissue.

Solubility data for mucus are not available, but Table 7-1 indicates that the Henry's law constant for ozone in water under the conditions of the lung is 9,700. Solubility data for pure ozone and other physical properties are available from various sources.[81,137] *Air Quality Criteria for Photochemical Oxidants*[140] reports an ozone solubility of 0.494 ml/100 ml of water at 0° C for ozone at 760 mm Hg; extrapolation of data from Thorp[137] indicates 1.09 g/liter of water at 0° C and approximately 0.31 g/liter of water at 37° C for 100% ozone. The value for 37° C agrees closely with the solubility calculated from the Henry's law constant for pure ozone at 760 mm Hg.

The diffusivities of ozone in mucus, tissue, and water are unknown. As an approximation, the diffusivity of oxygen in water (2.5×10^{-5} cm^2/s) may be used for the diffusivity of ozone in water and in the mucus layer. Although reactivities of ozone in mucus and tissue are unknown, the results of Alder and Hill[5] and Hoigne and Bader,[71] which describe the decomposition of ozone in aqueous solutions, may be used as a first approximation.

MORPHOLOGY OF THE RESPIRATORY TRACT AND MUCUS LAYER

Human and Animal Airway Models and the Real Lung

The airways and the tissue lining of the human respiratory tract have a very complex structure and dynamic behavior; these vary with age, sex, and state of health.[61,66] Horsfield[72] reviewed the structure and function of the respiratory tract and described how it can be simplified into airway models for calculating gas and particle transport. Phalen[109] has summarized the limited available data concerning the similarities and differences between the airways of humans and several animal species. Hausknecht and Ziskind[65] have also reviewed airway models for gas uptake. Most mathematical models for particle deposition are based on circular cylindrical, rigid models of the conducting airways with dimensions representative of the normal adult. Diameter, length, and branching angle of the airways at the same generation of the treelike branching structures are usually assumed equal.

Although the airway model of Findeisen,[48] which was later modified by Landahl,[91] has a very unrealistic branching structure, it has been used widely for particle-deposition studies.[134] Weibel's model "A"[144] has a more realistic symmetric dichotomous branching pattern and is currently popular. His complete model has 16 generations of conducting airways in the tracheobronchial region and seven partially or completely alveolated generations in the pulmonary zone. Table 7-2 shows the numbers and diameters of the conducting airways that are representative of an adult with a lung volume of 4,800 cm^3 at 75% inflation.

Other human airway models have been developed by Davies,[37] Horsfield *et al.*,[73] Phalen *et al.*,[110] and Yeh *et al.*[152] The asymmetric model of Horsfield *et al.* is more realistic than Weibel's, but is more difficult to use for calculations. Yeh *et al.* described a Monte Carlo technique for constructing a realistic lung model from extensive morphometric data. Such a lung model should have statistical distributions of the geometric characteristics similar to those of an actual lung and will permit separate deposition calculations for each lobe of the lung. Data on the airflow distribution between lobes of the lung[6] can be used to verify the model structure. Phalen *et al.* compared the airway morphology of the human, dog, rat, and hamster. The human tracheobronchial tree was found to be more symmetric with respect to diameter ratios and branching angles than those of the other species (but closest to that of the dog). Phalen[109] found that the bronchial tree structure is variable from species to species, from lobe to lobe within a given lung, and from one depth to

TABLE 7-2 Airflow Analysis of Weibel's Model "A" for a Normal Resting Breathing Rate of 15 cycles/min and a Tidal Volume of 450 cm^3[a]

Generation (z)	No. (N_z)	Diameter (D_z), cm	Length (L_z), cm	Time-Averaged Velocity (U_z), cm/s	Reynolds Number[b] Re ($D_z \bar{U}_z/r$)	Entrances Length[c] Length of Generation 0.057 Re D_z/L_z
0[d]	1	1.8	12.0	88.6	960.7	8.21
1[e]	2	1.22	4.76	96.6	710.0	10.37
2[f]	4	0.83	1.90	105.6	528.0	13.15
3[g]	8	0.56	0.76	112.5	379.5	15.94
4	16	0.45	1.27	90.7	245.9	4.97
5	32	0.35	1.07	72.3	152.4	2.84
6	64	0.28	0.90	56.8	95.8	1.70
7	128	0.23	0.76	44.1	61.1	1.05
8	256	0.186	0.64	32.4	36.3	0.60
9	512	0.154	0.54	23.5	21.8	0.35
10	1,024	0.130	0.46	16.8	13.2	0.21
11	2,048	0.109	0.39	11.5	7.55	0.12
12	4,096	0.095	0.33	7.81	4.47	0.073
13	8,192	0.082	0.27	5.06	2.50	0.043
14	16,384	0.074	0.23	3.24	1.44	0.026
15	32,768	0.066	0.20	1.99	0.79	0.015
16	65,536	0.060	0.165	1.25	0.45	0.009

[a] Derived from Bell.[23]
[b] r = kinematic viscosity of air in lung.
[c] Equation from Langhaar.[93]
[d] Trachea.
[e] Primary bronchi.
[f] Lobar bronchi.
[g] Segmental bronchi.

another in the lung. Other animal airway models have been developed for the rat,[54,83] the guinea pig,[84] and the rabbit.[82] If bifurcations continue to be viewed as critical sites for gas and particle deposition and dose-response relationships, more refined airway models will be needed that define in more detail the structure of bifurcation regions.

Because of the complexity of the actual structures, the emphasis in modeling has been on obtaining an average representation, and the variability among individuals tends to be neglected. There are two experimental studies of variability of airway dimensions in living humans as revealed by aerosol-deposition studies. Lapp *et al.*[94] assessed the size of alveolar spaces in terms of half-life of aerosol persistence during breath-

holding and obtained a coefficient of variation of 20–25%. Palmes and Lippmann[105] report the variability of a measure reflecting the influence of anatomic factors in tracheobronchial deposition as revealed by *in vivo* retention of γ-tagged microspheres in the human thorax; they found a coefficient of 60–70%. Variability of direct anatomic measurements of numbers and sizes of airways in man was given by Matsuba and Thurlbeck,[99] Angus and Thurlbeck,[14] and Thurlbeck and Haines.[138] In addition to expressing one cause of differences in the susceptibilities of individuals, the variability in model measures indicates a bias in the results of calculations that are based only on average values of the measures. Proctor and Swift[113] described the complex anatomy of the human nose and constructed from actual casts a morphologic model, which may require simplification, of the nasal airways. Similar models need to be developed for animal upper airways.

Mucus and Alveolar Tissue Models

Because the mucus layer or the underlying cells may serve as either final accumulation sites of toxic gases or layers through which the gases diffuse en route to the blood, we need simplified models of these layers. Altshuler *et al.*[9] have developed for these layers the only available model that can be used in a comprehensive system for calculating tissue doses of inhaled irritants. It assumes that the basement membrane of the tracheobronchial region is covered with three discrete layers: an inner layer of variable thickness that contains the basal, goblet, and ciliated cells; a 7-μm middle layer composed of waterlike or serous fluid; and a 7-μm outer layer of viscous mucus. Recent work by E. S. Boatman and D. Luchtel (personal communication) in rabbits supports the concept of a continuous fluid layer; however, airways smaller than 1 mm in diameter do not show separate mucus and serous-fluid layers.

FLOW ASPECTS OF RESPIRATORY PHYSIOLOGY

Respiratory Airflow Patterns in Lung Models versus the Real Lung

FLOW ANALYSIS IN WEIBEL'S MODEL "A"

Tidal volume and respiratory frequency are used with the anatomic dimensions to model airflow patterns in the respiratory tract.

During the respiratory cycle, the volumetric flow rate of air varies from zero up to a maximum and back. Usually, the expiratory phase is longer than the inspiratory phase, and there may be intervening pauses

between the two, especially after expiration. Silverman *et al.*[122] studied the respiratory airflow patterns of healthy young men at rest and under a wide range of workloads. The maximal inspiratory flow rate increased from a mean value of 40 liters/min in sedentary subjects to 100 liters/min at an exercise workload of 622 kg/min and to 286 liters/min at 1,660 kg/min. The corresponding values for maximal expiratory flow rates were 32, 107, and 322 liters/min.

When a quiet breathing rate of 15 cycles/min and a tidal volume of 450 cm^3 are assumed in Weibel's model "A," the time-averaged velocities are as shown in Table 7-2. Reynolds number and entrance length are calculated by treating each branch as a straight smooth tube. Analysis of these data suggests that there is plug (uniform) flow in the trachea through the third generation, partially developed laminar flow in the fifth through seventh generations, and developed Poiseuille flow for an increasing fraction of each branch in the eighth through sixteenth generations. With maximal inspiratory and expiratory effort, however, velocities and Reynolds numbers (Re) may be 22–45 times larger than during quiet breathing. This suggests turbulent flow in the upper generations. Furthermore, because of variations in compliance and resistance, ventilation is not equally distributed throughout the lung; consequently, branches of the same generation may have different flow rates.

SKEWED PROFILES AND SECONDARY FLOWS

The preceding flow analysis neglects the complicated flow behavior initiated at the bifurcations and transferred to the daughter branches. Schroter and Sudlow[119] and Schreck and Mockros[118] measured the velocity profiles for steady flow in the daughter branches of airway models that were geometrically similar to Weibel's model. For Poiseuille or plug flow in the parent tube, the flow is symmetrically split by the carina, and the higher axial velocities are directed off the daughter-tube axis along the inside wall. The shear rates along the inside wall are about 4 times larger than those along the outside wall, and the peak axial velocity is twice the average bulk flow velocity. The flow is also skewed as a result of the lateral convection or secondary flows generated at the bifurcation (Figure 7-1).

Although secondary flows increase the uniformity of the flow distribution distally along the branch, this is significant only in the slower regions. The flow profiles in the daughter branches become more complicated when this asymmetric flow reaches a second bifurcation; however, they follow the same general trends.

Sudlow and Schroter observed secondary flows at all flow rates (Reynolds

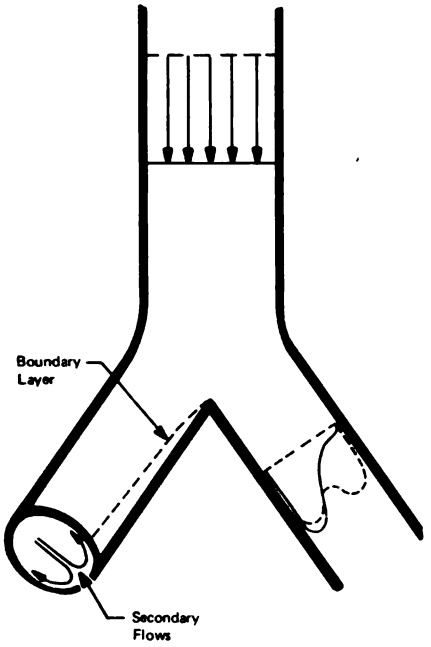

FIGURE 7-1 Schematic of flow in daughter branches of bifurcation model for steady inspiratory flow with flat profile in parent branch. Velocity profiles in plane of bifurcation (—) and in normal plane (---) are indicated in right branch. Orientation of secondary flows and position of laminar boundary layer are shown in left branch. Redrawn with permission from Bell.[23]

number, 50–4,500), regardless of the shape of the entry profile. On inspiration, a pair of symmetric vortices is formed in each daughter branch (Figure 7-1). They are strong enough to complete one helical cycle within three branch diameters. On expiration, a set of four vortices is generated in the parent tube.

These results suggest that simple parabolic flow is ensured only in the conductive airways where the Reynolds number is less than 1.0. There, the fluid inertia is negligible, and the convective fluid transport is less than the molecular transport.

TURBULENCE

The simplified flow analysis of Weibel's model "A" indicates undeveloped flow with a flat profile in the trachea for Reynolds numbers up to approximately 2,000.[117] However, this does not consider disturbances produced by the rough walls, the eccentricity of the cross section, and the larynx.

For steady inspiratory flow in hollow casts of the trachea and the first bifurcation, Dekker[40] observed that a Reynolds number of 1,800 was

required for initiation of turbulence. Similarly, for steady inspiratory flow in casts containing a larynx with the glottis in a natural open position, the required Reynolds number was 450. West[145] observed turbulence in the trachea of lung casts during exhalation at a Reynolds number of 800, although his hollow casts did not include a larynx. As the position of the vocal cords changes in the real lung,[156] the glottis of the larynx functions as a variable orifice. During inspiration, a jet of turbulent air enters the trachea and is directed against its ventral wall. Although the length of the trachea would be insufficient for the complete development of turbulent flow, the additional turbulence created by the jet and the corrugated walls may cause the turbulence to approach a fully developed state by the end of the trachea for a Reynolds number greater than 3,000.

A model developed by Owen[104] indicates that turbulence will gradually decay in any branch in which Re is less than 3,000. Assuming a peak Re of 1,865 in the trachea—corresponding to a 1.9-s, 450-cm^3 inhalation—Owen's model predicts a 10% decay in the trachea and in each of the first two generations of bronchi. According to Batchelor's theory[18] for the change in turbulent energy at regions of rapid flow contraction, decays of 15, 16, and 10% occur in the first three generations of bifurcation, respectively. Therefore, turbulence generated in the trachea at an Re of 1,865 will have approximately 50% of its initial intensity when it enters the third generation of bronchi. At inspiratory flow rates with Re greater than 3,000, decay would be even slower.

These decay calculations neglect the possible effects of the strong secondary flows generated at the bifurcation. The regions of very high and very low shear rate caused by the secondary flows could also be regions of high and low turbulence and dissipation. However, Pedley, Schroter, and Sudlow[108] argued that the boundary layer remains laminar in the daughter tube for Re less than 15,000 and had experimental evidence to verify this assumption for a parent-tube Re up to 10,000. Thus, the turbulent eddies are localized in the core, and the arguments given above are sufficient for predicting their rate of decay.

Detailed descriptions of the convective airflow patterns in cast replicas of the human respiratory tract during steady inspiration were given by Olson *et al.*[103] Their results show that the effect of the larynx is such that flow patterns typical of smooth bifurcating tubes (secondary motions and high shear rates along the inside wall) do not occur until the lobar bronchi are reached. Turbulent eddies produced by flow separation below the larynx do not decay as rapidly as predicted by theory.[104] Indeed, small eddies were observed as far down as the sublobar bronchi with 200-ml/s flows in the trachea.

WALL AND FLOW OSCILLATIONS

Another flow complication is the effect of heartbeat. West[145] measured flow oscillations in the segmental bronchi attributed to beating of the heart and found that they were detectable only during breathholding or during pauses between inspiration and expiration. The peak oscillatory flow rate observed was 0.5 liter/min, which is approximately 20% of the peak flow rate in the segmental bronchi during quiet breathing. These oscillations will improve gas mixing.

The minor variation in airway dimensions during expansion and contraction of the lungs in breathing can generate radial velocities that would be significant only in the peripheral airways with the smallest axial velocities.

Schroter and Sudlow[119] estimated that the macroscopic corrugations in the airways are below the critical protuberance height at which laminar flow can be disturbed.

QUASISTEADY FLOW

Most mathematical models for particle and pollutant transport assume steady flow conditions. However, flow actually varies approximately sinusoidally over time, and breathing frequency ranges from 8 breaths/min for sedentary conditions to 50 breaths/min during sustained work and exercise.

Quasisteady flow may be a more accurate description. This means that the pulsatile flow in the lungs can be analyzed as a continuous sequence of steady-flow profiles. However, there are different and sometimes conflicting criteria for quasisteady flow in the airways.[79,119,149]

Bell[23] suggested that the quasisteady flow is probably a valid approximation at quiet-breathing frequencies and that velocity and pressure profiles in the lung during quiet breathing can be obtained from experimental steady-flow data. The same conclusions cannot be generally applied to experimental particle deposition or gas-transfer measurements.

For regions in which the flow is not quasisteady, a transient-flow solution may be possible. For example, Lakin[89] and Lakin and Fox[90] developed a two-dimensional transient-flow solution for an idealized symmetric bifurcation during the period at the end of inspiration and before expiration. Their finding that vorticity decreases at the carina or bifurcation apex suggests that particle- and gas-deposition rates may be increased at these sites in the respiratory tract. It also suggests that reactive-gas deposition rates during normal oscillatory breathing differ

significantly from those predicted for steady flow—a view suggested by Bell[23] for particles.

CONVECTIVE AND DIFFUSIVE TRANSPORT AND MIXING

Another complication with unsteady, periodic flows is the mixing of residual air in the respiratory tract with the tidal air. The secondary flows and turbulence both increase the mixing of inhaled gases and particles with dead-space air. There have been numerous theoretical and experimental studies of convective and diffusive gas or aerosol mixing in the respiratory tract, but there is no general agreement on their relative significance in specific regions of the tract.

Work by Altshuler *et al.*[10] with 0.4-μm particles and a tidal volume of 500 ml showed that only about 11–27% of new air in each successive breath actually mixes with residual air. Theoretical particle-deposition models developed by Altshuler,[8] Beeckmans,[22] and Davies[38] have accounted for the mixing of inhaled aerosol with residual air.

Whipple, Chen, and Wang[146] showed that the distribution of an inhaled aerosol bolus depends on the orientation of the successive airway bifurcations and the volume of the bolus. On the basis of skewed velocity profiles, they made theoretical calculations of the distribution of aerosol boli in branching airways that were in fair agreement with the experimental data. Their results suggested that slow and shallow breaths should show greater differences in dispersion of irritant gases in the airways.

Baker *et al.*[16] theoretically analyzed simultaneous gas flow and diffusion in Weibel's symmetric model. They applied a time-varying flow with simultaneous longitudinal diffusion and concluded that convective mixing is much less important than mixing induced by molecular diffusion.

By analogy with heat-transfer data in curved tubes[121] and branching systems, the local transfer rates of easily absorbed gases are expected to be significantly affected by convective mixing in the large conducting airways. In the terminal bronchi and pulmonary regions, where convection is very slight, molecular diffusion is clearly dominant.

In a recent paper by Taulbee and Yu,[135] convective mixing of particles or gases in lung airways was defined in terms of an apparent diffusion coefficient. This was derived by assuming that the inhaled particles or gases follow the average air velocity in each airway and assuming that the average velocity is normally distributed among airways of the same generation. Their calculations indicated that this apparent diffusion coefficient is dominant and accounts for the pulmonary air mixing process.

As pointed out by Altshuler,[7] the flexibility and curvature of the airway walls and the gross inhomogeneities in the expansion and contraction of the lung structure cause flow separation and vortex motion that are directly related to convective mixing and flow irreversibility. To estimate the transport and uptake of inert and irritant gases within the pulmonary region better, one could use the alveolar-duct model and the calculation methods proposed by Altshuler for convective and diffusive mixing. He pointed out the significance of the fact that almost the entire wall of the alveolar ducts is open to the alveoli.

Wilson and Lin[147] described precisely the three transport mechanisms that act during the flow of a nonuniform gas in a tube: pure convection, Taylor diffusion (where radial diffusion and axial convection are coupled to produce an effective block or plug flow), and axial diffusion. They defined regions in the respiratory tract where each of these mechanisms dominates. By using Weibel's airway model [144] and these three mechanisms, they developed and analyzed a model to describe the transport of inert gases within the conducting airways. For quiet breathing, pure convection dominated the transport in the zero- through seventh-order generations, Taylor diffusion dominated in the eighth through eleventh, and axial diffusion dominated in the twelfth through the seventeenth.

Later research by La Force and Lewis[88] showed that gaseous concentration gradients are negligible during quiet breathing (contradicting the work of Cumming *et al.*[34]). Their anatomic models of the alveolated airways and their calculation methods should be compared with those used or proposed by Altshuler[7] and Taulbee and Yu[135] in the establishment of a model for pollutant-gas uptake in the alveolated airways.

AIRFLOW IN THE NOSE AND NASAL AIRWAY MODELS

The complex anatomic structure of the nose is ideal for humidification, temperature regulation, and pollutant scrubbing of inspired air. Proctor and Swift[113] studied nasal airflow by observing and measuring the flow of water through a clear plastic model of the walls of the nasal passages. They used steady flow with a Reynolds number equivalent to that for air in the human nose. For an inspiratory flow of 0.4 liter/s (quiet breathing), the linear inspiratory velocity at the nasal entrance reached at least about 4.5–5 m/s and at most 10–12 m/s. These values are significantly larger than the peak linear velocity of 2 m/s in the bronchial tree during quiet breathing.

As the cross-sectional area expands beyond the entrance, flow separation occurs and results in turbulence and eddies, which continue as the air goes through the passages around the turbinates. The linear velocity

also decreases sharply in this region, and the air stream then bends downward into the nasopharyngeal region. Because of these complex flow patterns and the large surface area of the nasal mucosa, the nose effectively scrubs particles and some gases from the inspired air.

Proctor and Swift's nasal-passage model[113] and their charts of the direction and linear velocity of airflow in the model could be used for estimating the local uptake of gaseous pollutants and the total scrubbing efficiency of the nose. The degree of swelling of the nasal mucosa significantly affects the scrubbing efficiency, so more refined airway models should simulate the morphology and flow behavior during different states of swelling.

Mucus Flow Patterns in the Respiratory Tract

The dynamic properties of the mucus fluid, serous fluid, and epithelial layers of the respiratory tract are important for the transport, absorption, and desorption of reactive gases. The cilia beat at a fairly constant frequency within the stationary serous layer and cause the outer mucus layer to move up the respiratory tract. Clearance of deposited particles and absorbed gases in the ciliated tracheobronchial tree depends partly on the movement of this mucus layer.

There have been a number of studies of the thickness and velocity of the mucus layer, with different results. Dalham[36] reported a thickness of 5 μm in the trachea of rats. Similarly, Alder *et al.*[4] reported 10 μm and less in cats, and Comroe[32] reported 10–15 μm. Velocity has been measured at 13.5 mm/min in rats,[36] 0–35 mm/min in cows and 5–14 mm/min in dogs,[69] 10.5 ± 3.7 mm/min in cats,[4] and 15 mm/min in the human trachea and 3.75 mm/min in the human main bronchus.[69]

Clearance in the upper, or ciliated, region is governed by the rate of mucus transport along the airways. These rates have been measured in the human nose[53] and in dogs, rats, and other species. Asmundsson and Kilburn,[15] Hilding,[70] and Iravani[76] established that mucociliary clearance rates increase from the distal bronchi toward the trachea. Because bronchial openings retard mucus flow, bifurcations receive an accumulation of mucus and associated particles. The rate of mucus production and mucus thickness and velocity vary from one person to another. Thickness increases and velocity decreases greatly when some toxic elements are present in the airway.[129–133]

New techniques have been developed for the direct measurement of mucociliary transport rates in the trachea. Yeates *et al.*[150] used an external gamma camera to follow a bolus of labeled microspheres deposited in the large airways by aerosol inhalation; they fitted a log-normal distri-

bution to their measurements and obtained a geometric mean of 3.6 mm/min and a coefficient of variation of 75% among 42 healthy nonsmokers. (The short-term coefficient of variation was considerably less, with a value of 25%.) Santa Cruz *et al.*[116] used a cinebronchofibroscopic method on subjects whose larger airways were anesthetized and observed the movement of small Teflon disks (0.68 mm in diameter and 0.13 mm thick) that were blown into the trachea through the fibroscope; they reported an arithmetic mean of 21.5 mm/min (standard deviation, 5.5 mm/min) in 16 normal nonsmokers and a much smaller value of 1.7 mm/min in older patients with chronic obstructive lung disease. The large discrepancy in the values reported for normal nonsmokers has several sources: different statistical distributions, different methods, and a complicated heterogeneity in local mucus velocities. It appears that the invasive aspect of the bronchofibroscopic technique caused some of the increase in the measured transport rate.

Mucus flows in the bronchial airways have not been directly measured. The measurements of particle clearance for radioactively tagged particles depend on a mixture of deposition sites and mucus flow rates. However, such measurements have shown reproducibility in the individual and a large variation among individuals.[3]

Altshuler *et al.*[9] developed a method for estimating the thickness and velocity of mucus throughout the tracheobronchial region. They matched a particle clearance time with Landahl's lung model and assumed a constant rate of mucus production per unit surface area and a uniform mucus thickness throughout the tract, except for terminal bronchioles. The calculated values are given in Table 7-3. From these values, mucus velocity in each region can be obtained by dividing the length of the region by the corresponding transit time. Jacobi,[78] Thomas,[136] and Haque and Collinson[62] also devised mucus clearance models for estimating the lung-tissue dosage of short-lived alpha emitters.

The calculated velocities are based on the assumption that mucus flows axially in each region. Actually, the bronchial openings represent obstructions to this parallel flow. Some have observed that the mucus stream has a spiral path sweeping over the carina.[64] According to Hilding,[68] mucus streams move axially and parallel in each section of airway. The streams that intersect the carina bend 90°, pass parallel to the carina, and then move upward. Others continue their flow undisturbed. Hilding stated that the reasons for this flow behavior may be the change in direction of cilia beat and the presence of small whirlpools in the middle of the margin of bronchial openings.

Some studies indicate that the mucus layer is not a continuous blanket. Direct observations of the airways of normal and bronchitic

TABLE 7-3 Anatomic Model of the Bronchial Tree and Mucus Layer[a]

Region	Diameter, cm	Length, cm	Total Circumference, cm	Total Surface Area, cm^2	Mucus Transit Time, min	Mucus Thickness, μm
Trachea	1.6	12	5.0	60	8	7
Main bronchi	1.0	6.0	6.3	38	6	7
Lobar	0.4	3.0	15	45	11	7
Segmental	0.2	1.5	63	94	37	7
Subsegmental	0.15	0.5	360	180	82	7
Terminal	0.06	0.3	11,000	3,400	1,980	4.2

[a] Derived from Altshuler *et al.*[9]

rats *in vitro* by Iravani and Van As[77] and Van As and Webster[142] failed to find a mucus blanket at any level of the tracheobronchial tree.

Further work by Van As and Webster[141] supports the discontinuity of mucus and shows that it is transported in well-defined streams in the larger airways of the rat. In addition to small regions of stagnation, local retrograde movement was observed. One must also consider the possibility that some of the serous fluid and mucus may be reabsorbed as it moves up the respiratory tract. This could influence the local tissue and mucus layer concentration of absorbed gas with time. Morphologic examination confirmed that mucus is present as flakes, droplets, and plaques. Droplets 0.5–1 μm in diameter are believed to be the primary unit of mucus, and they aggregate to form flakes, which in turn form plaques. The smaller flakes may be transported individually over individual metachronal fields of cilia, but the plaques are transported *en masse* by the combined action of numerous metachronal fields. The assumption of a continuous, stationary, and uniform layer of serous fluid also needs critical examination, especially in nonciliated areas.

Recent unpublished studies by Boatman and Luchtel (personal communication) in rabbits show that the mucus field is indeed continuous in the medium and smaller airways. Their morphologic techniques are currently being extended to the large airways. Differences in techniques may account for the discrepancy between this work and that of Van As and Webster.[141] Further studies of the structure of the mucus layer in animals are therefore needed to resolve the continuity-discontinuity question.

Mucociliary clearance from the nose or upper airways of man has been measured and described by Proctor and co-workers,[111–114] Ander-

son *et al.*,[11,13] and Quinlan *et al.*[115] Through the use of radioactive particles as tracers, mucus has been observed to move everywhere toward the nasopharynx, although indirectly. The mucociliary stream from the paranasal sinuses joins the nasal stream all along the middle meatus and above the posterior end of the middle turbinate. This flow field ensures that the region of the nose receiving the greatest deposition of toxic gases and aerosols is better protected. Mucus that reaches the nasopharynx, where the cilia disappear, is moved downward during swallowing, because the soft palate wipes the posterior nasopharyngeal wall.

Andersen *et al.*,[11] with a saccharine-particle method, found a weak positive association between tracheobronchial clearance and nasal clearance. A strong positive correlation would have indicated that information about the tracheobronchial clearance rate can be derived by studying clearance rates in the nose, which is more accessible. The saccharine method was shown to be a useful clinical tool for evaluating the status of the nasal mucociliary function in human subjects exposed to ambient pollutants or to controlled concentrations of specific pollutant gases or aerosols.

Effects of Inhaled Irritants and Airway Abnormalities on Air and Mucus Flow

A particular pulmonary irritant may alter air or mucus flow, and this in turn is one of the factors determining the local tissue dosage of the irritant. Such positive or negative feedback effects should also be incorporated into a dynamic model.

Acute exposure to irritant gases or particles present in urban air and in cigarette smoke at high concentrations can change the physical and chemical properties of mucus and cause retardation or cessation of mucociliary clearance.[12,35,36,80,124] Chronic exposure to some of the same irritants appears to cause hypertrophy of the mucus-secreting elements and glands of the upper airways, with a parallel increase in the production and secretion of airway mucus; and the bronchioles may show a marked increase in the goblet cells, resulting in excessive mucus production and airway obstruction, owing to ineffective clearance.[98]

Bronchoconstriction caused by acute exposures to ozone or sulfur dioxide may be expected to change the ventilation distribution, local aerodynamics, and tissue dosage. Edema resulting from exposures to toxic gases may alter the gas-absorptive capacity of the airways, in addition to the aerodynamics. Reaction of irritant gases with surfactant material in the alveoli may alter the absorptive capacity and physical prop-

erties of the surfactant, influence edema formation, and alter the clearance of inhaled particles.

GAS PHASE OF MODELS FOR POLLUTANT-GAS TRANSPORT IN THE RESPIRATORY TRACT

This section is concerned mainly with the approach to modeling the gas-phase behavior of single reactive gases. The basic approach can also apply to sulfur dioxide, ammonia, and other pollutant gases in which water solubility alone controls the rate of uptake. The simpler case of inert gases has been reviewed in a conference report edited by Papper and Kitz.[106]

Boundary Conditions

The simplest boundary condition between the gas and liquid phases is the assumption that the gas concentration at the surface of the mucus is zero. This same boundary condition is used for particles, so the deposition theory for highly diffusive particles may also be applicable. It may be adequate for predicting the net uptake of highly soluble gases like sulfur dioxide during single-breath inhalations at low gas-phase concentrations and could also apply to cases in which a pollutant gas undergoes rapid chemical reaction at the surface of the mucus (i.e., if the rate of chemical reaction exceeds the rate of transfer from the gas phase). Experimental data[19,154] have suggested that the rate of transfer of ozone from the air to the mucosal lining may be partially influenced by the capacity of ozone to undergo chemical transformation in the liquid phase. If this zero-concentration boundary condition is to be used, data are needed on the rate of chemical transformation of ozone in mucus.

A realistic boundary condition must account for the solubility of the gas in the mucus layer. Because ambient and most experimental concentrations of pollutant gases are very low, Henry's law ($y = Hx$) can be used to relate the gas- and liquid-phase concentrations of the pollutant gas at equilibrium. Here y is the partial pressure of the pollutant in the gas phase expressed as a mole fraction at a total pressure of 1 atm; x is the mole fraction of absorbed gas in the liquid; and H is the Henry's law constant. Gases with high solubilities have low H value. When experimental data for solubility in lung fluid are unavailable, the Henry's law constant for the gas in water at 37° C can be used (see Table 7-1). Gas-absorption experiments in airway models lined with water-saturated filter paper[75] gave results for the general sites of uptake of sulfur dioxide

and nitrogen dioxide that agree with uptake and histopathologic data on animals.

The Henry's law constant in water was used in the McJilton *et al.* uptake model[100] to determine the equilibrium concentration of ozone and sulfur dioxide at the surface of a simulated mucus film along the airways in Weibel's symmetric model.[144] It is also used to determine the concentration of absorbed gas at the surface of the mucus when the pollutant gas undergoes a homogeneous or heterogeneous chemical reaction within the mucus layer.

An additional complexity that has not been modeled is the simultaneous inhalation, absorption, and chemical reaction in the gas or liquid phase of two or more gases (e.g., sulfur dioxide and ozone). For sufficiently dilute mixtures, Henry's law can be used for each gas. If droplet aerosols and one or more reactive gases are simultaneously present, absorption with or without chemical conversion in the droplets must be considered.

Transport Equations

As noted earlier, air-velocity profiles during inhalation and exhalation are approximately uniform and partially developed or fully developed, depending on the airway generation, tidal volume, and respiration rate. Similarly, the concentration profiles of the pollutant in the airway lumen may be approximated by uniform partially developed or fully developed concentration profiles in rigid cylindrical tubes. In each airway, the simultaneous action of convection, axial diffusion, and radial diffusion determines a differential mass-balance equation. The gas-concentration profiles are obtained from this equation with appropriate boundary conditions. The flux or transfer rate of the gas to the mucus boundary and axially down the airway can be calculated from these concentration gradients. In a simpler approach, fixed velocity and concentration profiles are assumed, and separate mass balances can be written directly for convection, axial diffusion, and radial diffusion. The latter technique was applied by McJilton *et al.*[100]

To calculate more precisely the average uptake or the local variation in uptake in each airway, the local variations in velocity and concentration profiles must be taken into account. For example, thin momentum and concentration boundary layers occur at bifurcations and gradually increase in thickness with distance downstream. Bell and Friedlander[24] showed that particle and gas transfer to the airway wall is greatest where the boundary layers are thinnest, e.g., at the carina or apex of bifurcations.

Experimental data (e.g., from Yokoyama and Frank[154]) demonstrate that the concentrations of ozone and other gases reaching the trachea depend heavily on whether nose or mouth breathing is used. Detailed gas-transport equations for the nose and mouth are difficult to formulate. A simple approach used by La Belle *et al.*[87] assumed that the nose behaved like a scrubbing tower used in chemical processing. By varying the number of transfer units (defined as a portion of the dose sufficient to permit equilibration between gas and liquid phases) and the molar ratio of inhaled air to liquid, one may control the uptake of a soluble gas to match experimental data. Another simple approach was used by Aharonson *et al.*[1] A quasisteady state was considered for unidirectional flow in a one-dimensional model of the nose. A local mass balance was made for the transfer of the soluble vapor from the air to the mucous-tissue layer. The local transfer rate was strictly proportional to the local partial pressure of the vapor in the gas phase, and the average transfer rate—i.e., the overall uptake—could be determined by integrating the local transfer rates over the entire length of the nose.

Local and Average Mass-Transfer Coefficients

The rate of mass transfer of a pollutant gas from the gas phase to the wall of an airway can be written

$$dm/dt = -K_G A(C - C^*), \tag{7-1}$$

where K_G is the average mass-transfer coefficient, in centimeters per second, over the entire exposed surface area, A, of the airway; C is the average pollutant mass concentration in the gas phase in the airway; and C^* is the concentration of pollutant gas at equilibrium with the absorbed gas in the wall.[46] Local mass-transfer coefficients can be similarly defined for subsegments of an airway wall.

K_G can be defined as a gas-phase transfer coefficient, independent of the liquid layer, when the boundary concentration of the gas is fixed and independent of the average gas-phase concentration. In this case, the average and local gas-phase mass-transfer coefficients for such gases as sulfur dioxide, nitrogen dioxide, and ozone can be estimated from theoretical and experimental data for deposition of diffusion-range particles.[23] This is done by extending the theory of particle diffusion in a boundary layer to the case in which the dimensionless Schmidt number, ν/D, approaches 1 (ν is the kinematic viscosity of the gas, and D is the molecular diffusivity of the pollutant). Bell's results[23] in a tubular bifurcation model predict that the transfer coefficient depends directly on the

square root of the average airway velocity, the diffusion coefficient raised to the 2/3 power, and the bifurcation angle.

The prediction of such "hot spots" of gas transfer at bifurcations is supported by experimental data on ozone-exposed rabbits.[26] Longitudinal slices of airways from these rabbits showed at low magnification that desquamation of the ciliated epithelial cells was focal and sometimes more intense at a bifurcation.

The dependence of the local and average transfer coefficients on the square root of the average airflow rate is supported by the experimental data and analysis of Aharonson *et al.*[1] for ozone, acetone, ether, and sulfur dioxide.

Values for the average vapor-transfer coefficient from the gas phase to the airway epithelium can also be estimated from heat-transfer data in straight, curved, or bifurcating cylindrical tubes by using the analogy between heat transfer and mass transfer. Such an approach has been used by Yeh[151] to predict the diffusional deposition of small particles in the conducting airways.

LIQUID-TISSUE PHASE OF MODELS FOR POLLUTANT-GAS TRANSPORT IN THE RESPIRATORY TRACT

The important properties of the mucus and serous layers for gas-transfer models are thickness, viscosity, velocity gradients, the diffusion coefficients of pollutant gases in the mucus and serous fluid, and the chemical properties of these layers in the case of gases. Nonreactive gases like sulfur dioxide must diffuse through this liquid layer and the underlying cellular tissue layers before being absorbed by the blood. Figure 7-2 is an idealized cross-sectional model of part of a conducting airway showing separate tissue- and blood-layer components of the liquid-tissue phase. In preliminary studies designed to predict the average uptake in each generation, complexities like detailed velocity gradients in the liquid phase are unwarranted. Velocity gradients could be important in predicting local dosage to tissue; however, there are no experimental data. The mucus and serous layers may or may not be continuous and may constitute a homogeneous layer in some airways; thus, a single homogeneous liquid layer of constant thickness in each airway should be assumed until a more detailed description seems justified. Similarly, in the future it may be advantageous to subdivide the tissue layer into cell layers to reflect the pathologic evidence of ozone damage on some cell types and layers.

Aharonson *et al.*[1] combined the mucus and tissue phases in their

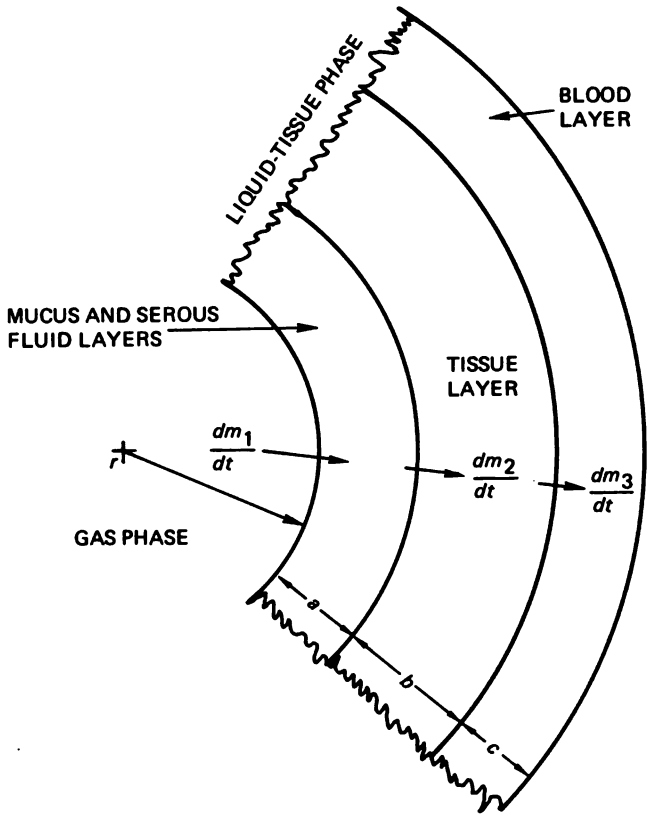

FIGURE 7-2 Cross-sectional model of part of a conducting airway in the respiratory tract, showing a gas phase and a liquid-tissue phase subdivided into mucus and serous-fluid, tissues, and blood layers. Derived in part from McJilton *et al.*[100]

conceptual model of the nose into one layer that separates the air and blood. The different resistances of each interface and layer are lumped into their local transfer coefficient.

Figure 7-2 illustrates a three-compartment structure assumed by McJilton *et al.*[100] for describing radial diffusion. It consisted of a gas phase in the lumen of the airway, a liquid layer that lined the airway, and a tissue compartment. The rate of movement of the gas into the liquid layer, dm_1/dt, is a function of the solubility of the gas in the liquid, as defined by the Henry's law constant. The rate of movement of the gas molecules across the liquid layer to the tissue compartment, dm_2/dt, is a function of the diffusion coefficient of the gas in the mucus and serous

layer. The concentration of ozone was assumed to be zero at the liquid-tissue boundary. This means that ozone is instantaneously converted by chemical reaction when it reaches the tissue layer, but undergoes no chemical reaction within the mucus layer. Such a model may be useful for ozone, if the rate of chemical reaction is very low. Ozone is known to react with hydroxyl ions at a very low rate,[5] but it probably reacts more rapidly with organic molecules in the mucus.

The mass-transfer coefficient in Equation 7-1, K_G, which averages over the interface between the gas phase and the mucous-fluid layer, is given by $K_G = 1/[(1/k_g) + (H/k_l)]$. where k_g is the gas-phase mass-transfer coefficient and k_l is the liquid-phase coefficient. This is the two-film model for interfacial mass transfer in which a gas molecule encounters resistance from both phases as it diffuses from the bulk vapor to the bulk liquid.[46,95] McJilton *et al.* assumed that $k_g \gg k_l/H$ and used empirical data to evaluate k_l for several gases. An alternative procedure is to evaluate k_l from penetration theory:[46]

$$k_l = \sqrt{4D/\pi t}, \tag{7-2}$$

where D is the diffusivity of the absorbed gas in the mucus layer and t is the gas-liquid contact time during inhalation or exhalation. Using the diffusivity of the absorbed gas in water may overestimate the actual transfer rate, because diffusivity may be much smaller in a viscous mucous fluid. Values of k_g can be determined as described in the previous section.

If the gas is converted by chemical reaction in the liquid layer, k_l is modified according to the order of the reaction and whether it is reversible or irreversible. For example, if ozone reacts rapidly and irreversibly with organic molecules in the mucus layer, k_l could be 10–100 times higher than the estimate based on penetration theory.

To model sulfur dioxide absorption by the blood through the walls of the upper airways, as demonstrated by Frank *et al.*,[52] one must include the transport rates of sulfur dioxide across a mucus-tissue interface, a tissue layer, and a tissue-blood interface (Figure 7-2). For the case of release of dissolved gas back into the exhaled air, which is depleted of gas in the lower lung, the mucus layer would still represent the greatest resistance to transfer. Consequently, the overall transfer coefficient, kg, would still be given by k_l/H.

The processes of convection, axial diffusion, radial diffusion, and chemical reaction in the liquid and tissue layers all occur simultaneously. A rigorous approach requires solution of several simultaneous differential equations. To avoid this complexity in preliminary models, the transfer

processes can be calculated in successive steps, as was done by McJilton *et al.*

The average dose rate or mass flux to tissue in each airway generation, defined as the mass transferred per unit time to the surface area of the generation, is given by dm_2/dt, the rate of the mass transfer across the liquid–tissue interface in Figure 7-2. The dosage to tissue is found by integrating the mass flux over time or a number of breaths. The local dose rate and dosage are defined in analogous ways.

DISCUSSION OF RESULTS WITH VARIOUS MODELS

Most models of gas uptake in the respiratory tract have been concerned with carbon dioxide, carbon monoxide, oxygen, and anesthetic gases like chloroform, ether, nitrous oxide, benzene, and carbon disulfide (e.g., see Lin and Cumming[96] and Papper and Kitz[106]). Unfortunately, there are only a few preliminary models of pollutant-gas transport and uptake in the respiratory tract.

Models of Nasal Uptake

La Belle *et al.*[87] modeled the absorption of various gases in the nasal passages of rats by applying principles of scrubbing-tower design. Their important characteristics were the Henry's law constant, the molar ratio of inhaled gas to absorbing liquid in the nose, and the number of transfer units. One transfer unit was defined as a portion of the nose sufficient to permit equilibration. The blood flowing through the nasal epithelial linings, rather than the moving mucus layer, was assumed to be the principal absorbing liquid, and Henry's law constants for water were used. The number of transfer units, N, was guessed to be between 1 and 10. Results of the calculations for various gases are summarized in Table 7-4. For oxygen through acrolein (relatively insoluble through moderately soluble gases), the penetration was controlled largely by the Henry's law constant. For ozone, with a Henry's law constant of 9,700 at 37° C, the La Belle *et al.* model predicts 99% penetration. For more soluble gases like sulfur dioxide and ammonia, the penetration also depends on the number of transfer units and the molar ratio of gas to blood.

This model appears inadequate, for a number of reasons. Although experimental data show that less sulfur dioxide than ozone penetrates the nasal passages in animals, as predicted by the model, much more ozone is predicted to penetrate than was demonstrated by Yokoyama and

TABLE 7-4 Calculated Penetration of Gases through the Nasal Passages in Rats[a]

Gas	Henry's Law Constant, mole fraction gas / mole fraction in solution	Fraction of Gas that Penetrates to Lung,[b] % $N = 1$	$N = 5$	$N = 10$
Oxygen	50,000	100	100	100
Nitric oxide	31,000	100	100	100
Nitrogen	10,000	99	99	99
Ozone	9,700	99[c]	99[c]	99[c]
Nitrous oxide	2,560	98	98	98
Carbon dioxide	1,900	95	95	95
Hydrogen sulfide	610	90	90	90
Chloroform	475	90	90	90
Bromine	160	80	80	80
Ethyl ether	68	70	70	70
Acetaldehyde	25	60	60	60
Acrolein	20	52	50	50
Chlorine	12	28	25	20
Sulfur dioxide	10	26	18	9
Acetic acid	7	25	7	2
Formaldehyde	3.8	10	4	0.2
Hydrogen cyanide	2.5	8	2	0.05
Ammonia	1.5	6	1.1	0.02
Phenol	0.4	5	0.8	0.01
Hydrogen bromide	0.003	5	0.7	0.01
Hydrogen chloride	0.001	5	0.6	0.01
Hydrogen iodide	0.0008	5	0.6	0.01

[a] Derived from La Belle *et al.*[87]
[b] N = number of transfer units.
[c] Extrapolated values.

Frank[154] in dogs. The most likely explanation is that the model does not account for chemical reactions of ozone in the mucus and epithelial tissue. Another problem is that the nose is believed to behave more like a scrubbing tower with fresh liquid at each level, inasmuch as the blood supply is not continuous for the entire length of the nose,[27] as assumed in the model. Neglecting the surface area, volume, flow, and thickness of the mucus layer in the nose will probably also give erroneous results for soluble gases with a small diffusion coefficient in mucus and for single-breath inhalations of a low concentration of any gas.

However, recent work by Loring and Tenny[97] partially supported the La Belle *et al.* model by suggesting that the properties of the mucus layer

in the nose may be irrelevant for modeling the absorption of relatively water-insoluble gases, such as nitrogen, oxygen, carbon dioxide, and nitrous oxide. They observed that the flux of these gases from the frontal sinuses of cats was explained best by a perfusion-limited blood-absorption mechanism.

An accurate nasal model must also account for the airflow rate and the concentration of the inspired gas. Aharonson *et al.*[1] conclusively demonstrated that the "uptake coefficient," or average mass-transfer coefficient, over the entire nose for acetone, ozone, sulfur dioxide, and ether increased with increasing airflow rate.

Yokoyama and Frank,[154] Frank *et al.*,[51] Brain,[27] and Egle[44,45] largely overlooked this flow-dependent relationship, because they did not normalize their retention data into the average-transfer-coefficient form. In fact, failure to do this led Yokoyama and Frank to the erroneous conclusion that " the uptake of O_3 was inversely rated to flow."

Aharonson *et al.*[1] discussed four explanations for the increased uptake with increased flow: misinterpretation of data owing to a dependence of the average transfer coefficient on vapor concentration, decrease in the gas-film resistance, increased perfusion of nasal tissue, and increase in the effective surface area for uptake. For gas like ozone, which is fairly insoluble but probably highly reactive in the mucus layer, the gas film or concentration boundary layer represents the major resistance to uptake. As discussed earlier, the gas-phase average or local transfer coefficient in airways of the tracheobronchial tree is predicted to depend on the square root of the average airflow rate in the airway and is independent of gas-phase concentration. Because the data analyzed by Aharonson *et al.* agree roughly with this square-root dependence, the properties of the boundary layers in the nose may be similar to those in the tracheobronchial tree. The vapor concentration enters only in calculation of the average flux to the tissue from the product of the transfer coefficient and the concentration gradient between the gas phase and the liquid-tissue phase. The overall transfer coefficient, K_G, in Equation 7-1 may be concentration-dependent, if the vapor is reacting reversibly in the liquid layer or reacting reversibly with a second dissolved vapor in an inert liquid layer.

Models of Tracheobronchial Uptake

There are no published models that adequately describe ozone uptake in the tracheobronchial tree. To present the methodology, a few published and unpublished models of the uptake of various gases are reviewed.

The McJilton *et al.*[100] model of ozone uptake has been widely cited,

although not formally published. It is described here because it was the first attempt to model the absorption of pollutant gases in each generation of the tracheobronchial tree.

They used mass-balance expressions in finite-difference form to approximate the convection and diffusion of the pollutant gas in a 25-segment airway model that started at the trachea and was a modified version of Weibel's model. A sinusoidal breathing cycle and uniform plug flow were assumed in each of the first 20 segments. Beyond the twentieth segment, where the segmental volume was greater than 5 ml, uniform convective mixing was assumed. The cylindrical airways were assumed to be lined with a stationary mucus-fluid layer and a tissue layer as shown in Figure 7-2. The mucus was assumed to have the properties of water and thickness of 10 μm in the upper generations, 3–5 μm in the alveolar ducts, and 0.3 μm in the alveoli. There were no chemical reactions in the mucus.

Finite-difference techniques were also used to calculate the rate of diffusion of the pollutant gas from the airway to the mucus layer and through the mucus layer to a perfectly absorbing sink at the mucus-tissue interface. The mass of pollutant lost from the airway or transferred across the air–mucus interface during each breathing cycle was divided by the segmental surface area to obtain the dosage, in micrograms per square centimenter per breath.

Although convection, axial diffusion, and radial diffusion actually occur simultaneously, a multistep procedure was adopted in the finite-difference calculation. For each 5-cm^3 increment in tidal volume and for each time increment Δt, the differential mass-balance equations were solved for convection, axial diffusion, and radial diffusion in that order. This method may slightly underestimate the dosage for weakly soluble gases, because the concentration gradient in the airway may be decreased.

Although the authors viewed their results with this model as only preliminary, a few of the results are presented here to contract the expected behavior of water-soluble gases (e.g., sulfur dioxide) with that of fairly insoluble gases (e.g., ozone).

Figure 7-3 shows the percentage of total gas uptake for the steady state (after five or six breaths of 500-cm^3 tidal volume with 2-s inspirations). Uptake increases from about 75% for a relatively insoluble gas with a Henry's law constant of about 10^4 to a peak of 95% for soluble gases with a Henry's law constant of 20 or less. Figure 7-4 shows why there is only a 20% variation in uptake over a wide range of the Henry's law constant (10–10^4). The model predicts that the dosage of gases of low solubility is fairly uniform throughout most of the airways until the

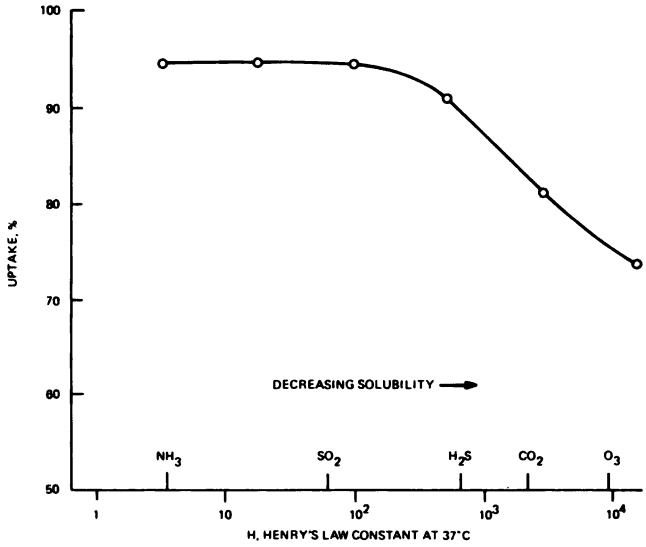

FIGURE 7-3 Uptake of pollutant gases in the entire tracheobronchial tree and pulmonary region at steady state as a function of Henry's law constant. Modified from the model results of McJilton *et al.*[100]

alveoli are reached, whereas gases of high solubility are predominantly removed in the upper airways of the tracheobronchial tree.

The model also predicts an increase in uptake as tidal volume increases over a constant breathing period. As the breathing period increases at a constant tidal volume, the uptake also increases. In the former case, increased ventilation of peripheral airways with a high surface:volume ratio increases uptake. In the latter, the period for radial diffusion is increased in every segment.

Figure 7-5 contrasts the steady-state uptake of sulfur dioxide and ozone per breath in each segment of the McJilton *et al.* model when the inhaled-gas concentration at the entrance to the trachea is 1,000 $\mu g/m^3$. The patterns for the uptake of the highly soluble sulfur dioxide and the relatively insoluble ozone are strikingly different. The segmental dosage of sulfur dioxide peaks at 1.2×10^{-3} $\mu g/cm^2$-breath in segments 11 and 12 (immediately beyond the lobar bronchi). Ozone dosage is fairly uniform around 8.5×10^{-6} $\mu g/cm^2$-breath in segments 1-18. It then dips sharply to 5×10^{-6} $\mu g/cm^2$-breath in segment 22 and peaks again at 8.3×10^{-6} $\mu g/cm^2$-breath in respiratory bronchioles. A much smaller dosage of 2×10^{-7} $\mu g/m^3$-breath is calculated in the alveoli, mainly because of the large surface area.

There are no experimental data to verify the detailed dosage distribution among airway segments; however, experimental data on mouth-breathing animals and man support the general concept of rapid absorption of sulfur dioxide and greater peripheral absorption of ozone.[27,52,153]

Recently, Corn *et al.*[33] measured an overall mass-transfer coefficient for sulfur dioxide and nitrogen dioxide in the upper airways of the tracheobronchial tree in cats during controlled respiratory cycles. Their measured transfer coefficient for sulfur dioxide was nearly 100 times larger than the average transfer coefficient predicted between the trachea and segmental bronchi from Figure 7-5. Their coefficients for sul-

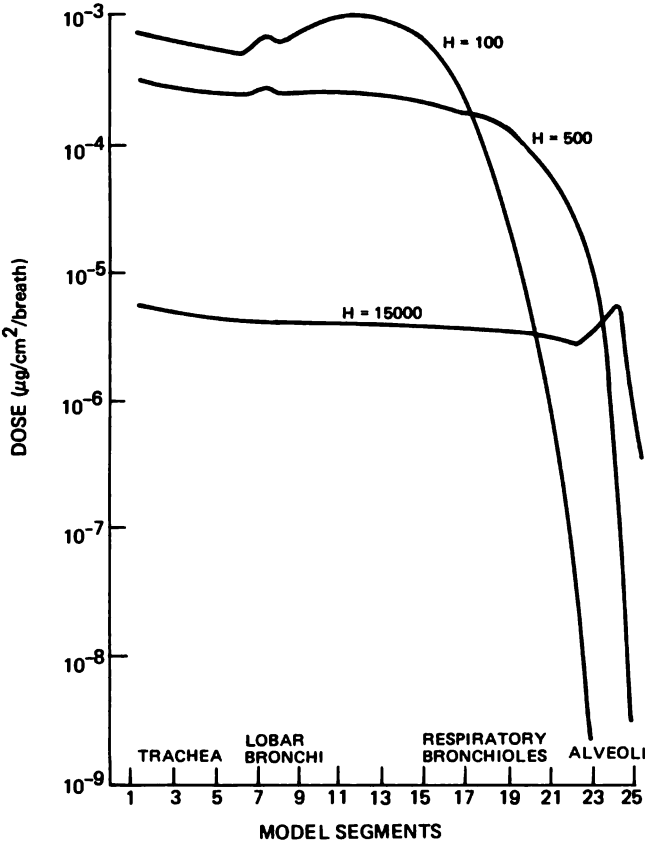

FIGURE 7-4 Uptake or dose predicted for each model segment by McJilton *et al.* for gases of different solubilities at 37° C. Tidal volume = 500 cm³. Inspiration time = 2 s. Modified from McJilton *et al.*[100]

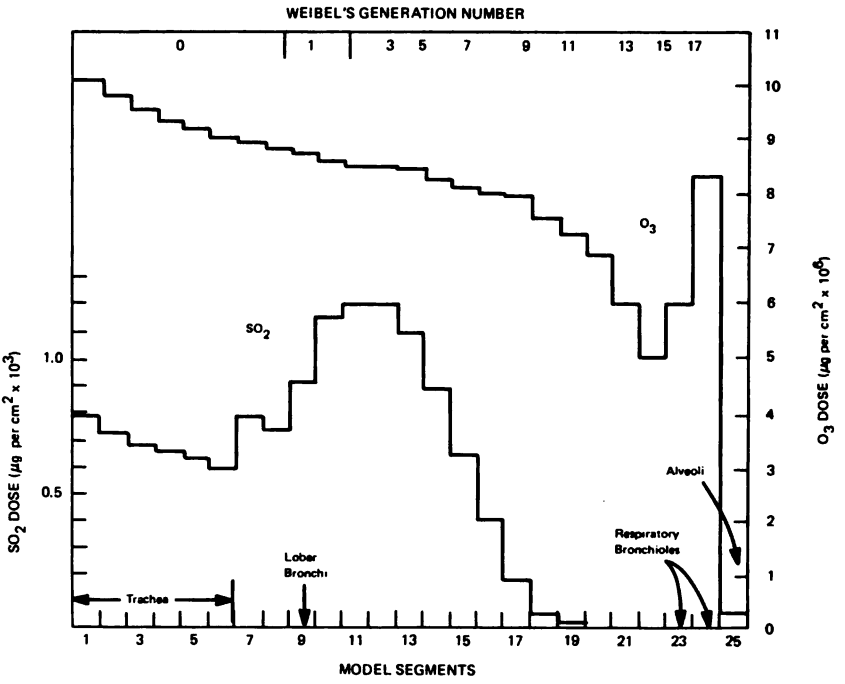

FIGURE 7-5 Comparison of dosage of sulfur dioxide and ozone predicted per breath by McJilton *et al.* for each model segment. Inhaled concentration = 1,000 $\mu g/m^3$. Tidal volume = 500 cm^3. Inspiration time = expiration time = 2 s. Reprinted with permission from McJilton *et al.* [100] Weibel's generation numbers added.

fur dioxide were also slightly dependent on concentration, but they were independent of concentration for nitrogen dioxide. At a fixed tidal volume, the measured transfer coefficients were consistent with the square root of the breathing frequency or average flow rate, as predicted by the previously discussed theory based on the boundary-layer model, and as shown by the uptake data for sulfur dioxide in the nasopharyngeal region.

In general, the McJilton *et al.* model appears to be useful for estimating the uptake in the tracheobronchial tree and pulmonary region of water-soluble and relatively water-insoluble gases that are nonreactive with the mucus layer. Nonreactive gases that are only partially soluble in tissue or blood (sulfur dioxide, oxygen, carbon dioxide, and anesthetic gases) may exert a backpressure that inhibits the gas uptake from the airways. Modifications, including the local blood flow rate and the tissue thickness (Figure 7-2), are required to handle these gases properly.

The major weakness is the requirement of nonreactivity of gases in the mucous layer. Very weakly reactive gases may be treated as nonreactive. However, the uptake of ozone, which is known to decompose in water and is expected to react rapidly with biopolymers and other organic molecules in the mucus layer, is probably underestimated in the upper airways and overestimated in the terminal airways of their model. Thus, their model represents a worst-case estimate of dosage of ozone to the terminal airways, which are unprotected by mucous. Too little is known of the chemical and physical properties of the mucus layer, and there is great uncertainty in the values of the diffusivity of ozone or other gases to be used in the liquid phase of gas uptake models.

Gases that do not react irreversibly with epithelial tissue, such as anesthetic gases, may diffuse into the bloodstream and will ultimately be eliminated from the body. A different and earlier model developed by DuBois and Rogers[41] estimates the rate of uptake of inhaled gas from the tracheobronchial tree in terms of diffusion through the epithelial tissue, rate of blood flow, and solubility of the gas in blood. The rate of uptake from the airway lumen is determined by the equation:

$$\dot{V}_c/P_0 = \alpha DA\dot{Q}/(x\dot{Q} + DA)760, \tag{7-3}$$

where $\dot{V}_c$ is the rate of uptake of gas from the lumen, P_0 is the partial pressure of the gas in the lumen, α is the solubility of the gas in blood, D is the coefficient of gas diffusion in tissue, A is the surface area of the bronchial segment, $\dot{Q}$ is the bronchial blood flow, and x is the thickness of the bronchial epithelium between lumen and blood flow. DuBois and Rogers used this equation to calculate the uptake, during acute exposures, of several gases of different solubilities from the first 16 generations of Weibel's model.

The absorption distribution between generations of Weibel's model had the same trend for acetone, nitrous oxide, and sulfur hexafluoride, which are soluble, moderately soluble, and insoluble, respectively, in blood or water. Absorption decreased by one-third from the trachea through the third generation and then increased rapidly and continuously with depth in the tracheobronchial tree; absorption in the sixteenth generation was 15–25 times that in the trachea. The relative magnitude of absorption at each generation was directly related to solubility. The distribution for nitrous oxide, whose solubility is slightly greater than that of ozone in water, differs radically beyond the third generation from the distribution predicted by McJilton *et al.* for ozone (Figure 7-5). Similarly, the uptake distribution for acetone, whose solubility is close to that of sulfur dioxide, differs significantly from the prediction for sul-

fur dioxide (Figure 7-5). This is plausible, because the tissue thickness decreases and the blood flow per generation increases with depth beyond the third generation in the DuBois and Rogers model, whereas, in the McJilton *et al.* model, blood absorption is neglected, the mucus-layer thickness decreases only slightly with depth, and the coefficient of transfer across the air-mucous interface decreases rapidly.

An improved gas-uptake model should incorporate the features of the DuBois and Rogers model and the McJilton *et al.* model. As shown in Figure 7-2, the model for gas uptake in the airways should include separate layers for mucous-serous fluid, epithelial tissue, and blood. Development of such a model awaits reliable data and methods for predicting the coefficient of diffusion of pollutant gases in tissue and information on the rates of local perfusion of blood and lymph in the bronchial epithelium. Experimental data from humans and animals[57-59,155] on the rate of sulfur dioxide absorption in blood could be used to make improved estimates of the tissue-diffusion coefficients *in vivo*.

New or improved methods are needed to measure local uptake experimentally. Such data can be used to verify the detailed dosage distribution predicted by the models. For example, the retrograde catheter and tracheal cannula system used by Corn *et al.*[33] appears promising for transfer-coefficient measurements within segments of the tracheobronchial tree. A similar method was used by Battista and Goyer[21] to measure the absorption of acetaldehyde vapor in the dog lung.

Radioactive lung-scanning techniques that use tagged irritant gases could give regional uptake data similar to scans obtained from radioactive-particle deposition and clearance studies. With methods of chemical separation and quantitation of the radioactivity in compounds isolated from the mucus layer, the reactions of ozone with biopolymers may be determined. Autoradiographic methods may also be useful for measuring the local uptake of tagged soluble gases within specific airways.

Bell[23] described how deposition of particles by convective diffusion in the respiratory tract can be used to estimate the average and local rates of gas transfer. For example, local inhomogeneities in pollutant-gas transfer can be included in uptake models. This is done by multiplying the local-transfer coefficients for 0.088-μm-diameter particles (Figure 7-6) by $(D_{gas}/D_{particle})^{2/3}$; D is the coefficient of diffusion of the gas or particle in the gas phase. When the boundary is perfectly absorbing, the local gas-phase transfer coefficients are to be multiplied by the gas concentration, the surface area between appropriate contours in Figure 7-6, and the inhalation time to determine the local gas dosage. Figure 7-7 allows a rapid determination of the area between contours. When the boundary is not perfectly absorbing, the local gas-phase transfer coeffi-

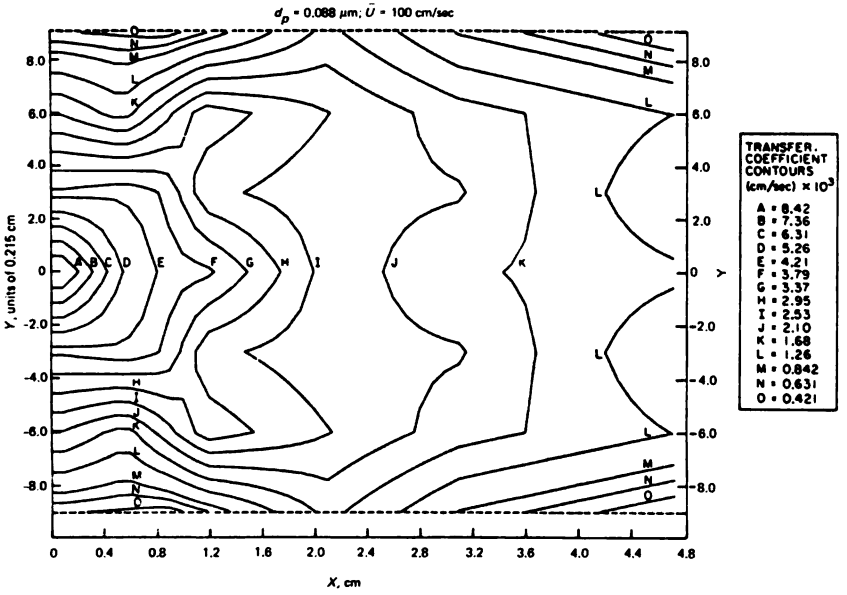

FIGURE 7-6 Transfer-coefficient contours during inhalation for 0.088-μm-diameter particle deposition in the daughter branch of the three-dimensional bifurcation model shown in Figure 7-1. The time-averaged velocity in the parent branch, $\bar{U}$, is 100 cm/sec. Total surface area = 18.6 cm². X = distance (in centimeters) downstream from the carina; Y = deposition locations around the branch circumference with Y = 0 at the carina. Each unit of the ordidnate corresponds to a distance of 0.215 cm. Adapted from Bell.[23]

cient must be substituted for k_g in the expression $1/K_G = 1/K_g + H/k_l$. Here, K_G is redefined as the coefficient of local transfer across the air-liquid interface. Local nonuniformities of gas transfer would be most prevalent in single-breath experiments, during the transient periods before equilibrium is attained, and in exposures with pollutant gases that react rapidly with the mucus layer.

Dose–Response Correlations

Modeling of gas transport is also useful for correlating dose-response data obtained under different conditions. Brain[27] suggested that the total dose of an inhaled gas is related to ventilation rate, duration of exposure, and gas concentration before inhalation. Folinsbee *et al.*[49] exposed human subjects to ozone at 0.37, 0.5, or 0.75 ppm for 2 h while they were at rest or exercising intermittently. The primary response of the subjects was an alteration in the exercise ventilatory pattern. They

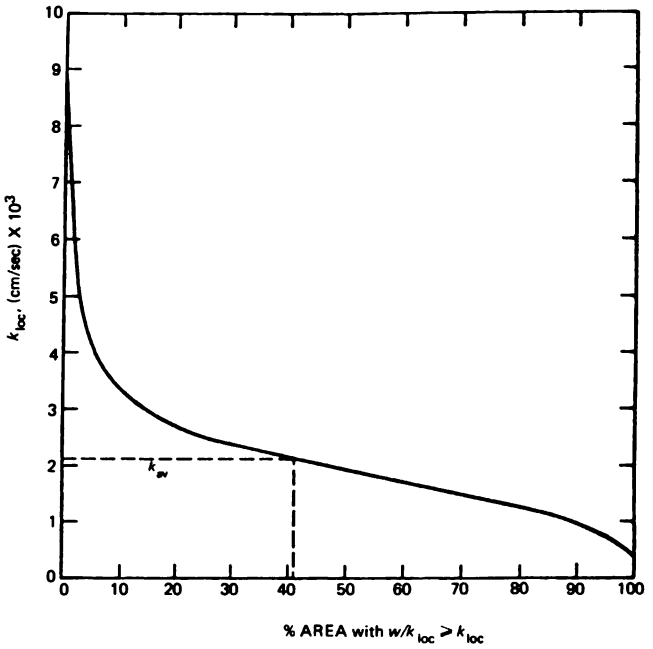

FIGURE 7-7 Cumulative surface-area distribution for Figure 7-6, showing the fraction of surface area of daughter branch that has local transfer coefficient equal to or greater than stated value d_p = 0.088 μm, $\bar{U}$ = 100 cm/sec. Reprinted with permission from Bell.[23]

showed an increase in respiratory rate and a decrease in tidal volume that were correlated with the total dose of ozone (expressed as the volume of ozone inspired during exposure). Other pulmonary-function data, like the flow at 50% of vital capacity, also appeared to be related to the volume of ozone inspired.

Fairchild and Graham,[47] Stokinger *et al.*,[128] and Stokinger[127] showed that the toxic effect of ozone in experimental animals is cumulative. They found that the effective dose depends on the product of ozone concentration and exposure duration for short-term single exposures.

When the pulmonary response is activated by irritant receptors in the nose, response for different flows and concentrations would not be expected to correlate with the volume of inspired gas, but rather with regional dosage (e.g., nasal) or the local dosage of gas to irritant receptors lining the airway.[2]

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8

Toxicology

The toxicologic aspects of oxidant air pollution have most often been examined through laboratory studies of individual constituents of the complex mixture of air contaminants that characterizes the ambient air environment. Photochemical air pollution (with automobile exhaust as the primary emission source) can be chemically characterized as oxidizing in reaction, and the chemical species that contribute to this oxidizing property are varied. Among the strongest of the photochemically formed oxidants that are present in "oxidant smog" and are stable enough to be identified and measured is ozone. Because ozone is also known to be among the most toxic of gases, it is logical that most toxicologic research related to the potential health effects of photochemical air pollution has focused on it. It has become almost a reflex to think of ozone toxicity when "oxidant toxicity" is mentioned. Nitrogen dioxide is another toxic gas present in "oxidant smog," but, by most conventional indexes, it is less toxic than ozone, especially when compared in the same proportions as their concentrations in ambient air. Indeed, ozone and nitrogen dioxide share many common toxicologic properties. Nitrogen dioxide is discussed in another monograph in this series, so its toxicology will not be reviewed here, except for specific comparisons. Other chemical species, e.g., the peroxyacylnitrates and hydroxyl free radicals, which undoubtedly contribute to the oxidizing properties of the total mixture of oxidant air pollution, may also contribute to the total biologic activity of this mixture.

However, there have been too few controlled toxicologic studies to permit an evaluation of their contribution to the toxicologic actions of photochemical oxidant mixtures. For example, the single toxicologic study on PAN that has been reported indicated that it is much less acutely toxic than ozone.[27]

The review of the experimental toxicology of photochemical oxidants in this chapter is therefore primarily a review of the toxicology of ozone. The reader should bear in mind that what is said about ozone may not apply in every case to the broader term "photochemical oxidants," but, given the current state of knowledge, ozone toxicology is the best approximation of the toxicology of the class. With this in mind, it is of value to review briefly the experimental toxicology of complex mixtures of ambient or laboratory-simulated photochemical smog.

Investigations of the potential biologic actions of a complex photochemical reaction mixture, produced by irradiating mixtures of air and auto exhaust under laboratory conditions that simulated real driving patterns and solar irradiation, were first conducted in the laboratories of the Division of Air Pollution, U.S. Department of Health, Education, and Welfare (DHEW).[102,103,106,141,144] The effects of both nonirradiated and irradiated exhaust mixtures were studied. Clearly, irradiation of the air–exhaust mixture led to the formation of photochemical reaction products that were biologically more active. The changes in chemical qualities of the mixture that accompanied the increased biologic activity were an increase in the concentration of total oxidant and an increase in the formation of irritant aldehydes. The relative proportions of the suspect biologically active chemical species varied with the total concentration of exhaust gases in the irradiated mixture and with the duration of irradiation.[141,144] Single-inhalation exposure studies lasting a few hours demonstrated in the laboratory that irradiation of exhaust mixtures led to greater effects on respiratory mechanics in guinea pigs, greater reduction in voluntary running activity of mice, and slightly greater carboxyhemoglobin formation in rats, compared with animals exposed to the same total concentration of exhaust gases that were not irradiated. The concentration of total oxidant in these experiments ranged between 0.33 and 0.80 ppm in the irradiated-exhaust mixtures; only a trace or no oxidant was detected in the unirradiated exhaust. The irritant aldehydes, formaldehyde and acrolein, were also present in higher concentrations in the irradiated atmospheres. The effects that were noted during the period of exposure (usually 4–6h) were reversible within a few hours when the animals were returned to clean air. The effects in animals exposed to the irradiated-exhaust mixture are not necessarily uniquely characteristic of ozone, but most of them could have been produced by ozone.

The nature of changes in respiratory mechanics in guinea pigs exposed to irradiated exhaust varied somewhat according to the ratio of oxidant to aldehyde concentrations (formaldehyde and acrolein were measured), and this ratio in turn varied with the duration of irradiation of the air-exhaust mixture.[141] Thus, when the oxidant:aldehyde ratio was low, the pattern of effects on guinea pig respiration resembled that produced by such upper airway irritants as formaldehyde and acrolein[6,143,145] and was characterized by increased pulmonary flow resistance, increased tidal volume, and decreased frequency of breathing. Increasing the ratio resulted in a shift in the pattern of respiration toward decreased tidal volume and increased frequency, typical of that produced by deep-lung irritants (e.g., ozone and nitrogen dioxide),[146] although the increased flow resistance typical of the aldehyde effect persisted. This interactive effect of an oxidant-aldehyde mixture could be reproduced by a simple mixture of ozone and acrolein.[141] Concentration-response relationships of these effects and other actions in laboratory animals exposed to mixed or single components of oxidant air pollution are summarized in Table 8-1.

Most experimental toxicology studies of inhaled vapors and gases report concentrations as parts per million. That convention is used in this chapter. See Chapter 6 for relations of various measurement units. From the data in Table 8-1, the conclusion could be drawn that all the functional effects observed in short-duration experiments with laboratory-produced photochemical smog mixtures could have been due to ozone alone if one considers total oxidant concentration of the mixture equivalent to ozone concentration. A possible exception is the increase in respiratory flow resistance in guinea pigs, which is more characteristic of and probably due, at least in part, to the irritant aldehydes in the exhaust mixtures. The increase in respiratory frequency in guinea pigs is most probably due to the oxidant (or ozone) content of the mixture. Comparison of the concentrations for equal effectiveness in decreasing the spontaneous running activity in mice also suggests that this action of the mixture may be largely due to ozone; and the oxidant content of irradiated auto exhaust appears to explain adequately the increase in susceptibility to respiratory infection in mice exposed to the mixture. These conclusions must, of course, be qualified by the possibility that other chemical species, such as hydroxyl free radicals, that were not measured or reported might also have produced the same effects and led to the same conclusions. Nevertheless, it is reasonable to conclude that many of the functional effects produced by short-duration exposures to complex photochemical-oxidant mixtures are due to ozone.

Hueter *et al.*[106] exposed animals to irradiated automobile exhaust (simulated photochemical smog) for periods up to 23 months. The con-

TABLE 8-1 Effects of Photochemical-Oxidant Air Pollution Mixtures (in Irradiated Auto Exhaust) and Some Individual Constituents in Single Short (6-h) Exposures*

Effect	Air Pollutant	Concentration, ppm	Reference
50% increase in respiratory flow resistance in guinea pigs	Formaldehyde	3.9	145
	Acrolein	0.6	143
	Ozone	1.1	146
	Mixture:		
	Formaldehyde	1.93	141
	Acrolein	0.09	141
	Oxidant	0.91	141
	Nitrogen dioxide	2.00	141
	Carbon dioxide	250	141
50% increase in respiratory frequency in guinea pigs	Formaldehyde	(decreased respiratory frequency)	145
	Acrolein	(decreased respiratory frequency)	143
	Nitrogen dioxide	6.5	146
	Ozone	0.68	146
	Mixture:		
	Formaldehyde	1.32	141
	Acrolein	0.06	141
	Nitrogen dioxide	0.79	141
	Oxidant	0.95	141
	Carbon monoxide	150	141

50% decrease in voluntary running activity in mice	Acrolein	0.4	141
	Nitrogen dioxide	16	146
	Ozone	0.3	141
	Carbon monoxide	250	141
	Mixture:		
	Formaldehyde	1.11–1.39	141,144
	Acrolein	0.09–0.10	141,144
	Nitrogen dioxide	1.64–2.95	141,144
	Oxidant	0.45–0.82	141,144
	Carbon monoxide	85–95	141,144
Significant increase in susceptibility to induced respiratory infection in mice	Ozone	0.08	36
	Nitrogen dioxide	3.5	54
	Mixture:		
	Oxidant	0.15	36
	Carbon monoxide	25	36

*Data shown are summarized and adapted from dose-response curves, typical concentration values, etc., given in references cited. The data should therefore be considered approximate and used only for comparison.

centrations were cycled, to simulate daily peak-and-trough pollution concentrations in urban centers. Peak daily concentrations of carbon monoxide were 20, 50, 60, and 100 ppm in four sets of exposure chambers. There was considerable loss of ozone and nitrogen dioxide on chamber walls, cages, and animal fur, so the concentrations of chemically reactive gases to which the animals were exposed probably ranged from about 0.04 to 0.2 ppm for ozone and about 0.15 to 0.5 ppm for nitrogen dioxide. Pulmonary flow resistances, tidal volumes, and respiratory frequencies of guinea pigs were measured (while the animals were breathing clean air for brief test periods) at 16-week intervals during the experiments and were not affected by the chronic exhaust exposures. Exhaust-exposed mice showed a decrease in running activity for the first few weeks of exposure, but then recovered to control activity. A decrease in mouse fertility rate and a decrease in infant survival rate occurred in the exhaust chambers, and there was an increase in the rate of spontaneous pulmonary infection in exhaust-exposed animals. There were no significant effects of exhaust exposures on mortality, histopathology, growth rate, or hematologic indexes.

Interpretation of these studies is difficult, because of surface loss of contaminants in the chambers. The decrease in mouse fertility (duplicated in a second experiment[121]), the decrease in mouse physical activity, and the increase in susceptibility to infections suggest that some functional measurements were more sensitive for detecting effects than conventional histologic, hematologic, or growth measurements. It is doubtful, however, whether any of the observed changes could be considered chronic effects, i.e., irreversible or progressive in the absence of continued exposure.

Wayne and Chambers[207] reviewed the findings of studies on experimental animals exposed throughout their lifetimes to ambient Los Angeles atmosphere. The control animals were kept in rooms that were ventilated with special filters that removed most of the ambient air pollutants. No clear evidence of chronic injury from the ambient air pollution was observed. There was some suggestion from pulmonary-function tests, electron-microscopic examinations, and pulmonary adenoma incidence that aged animals were adversely affected by ambient smog; and some reversible changes in pulmonary function of guinea pigs were noted during peak periods of air pollution. Increased 17-ketosteroid excretions suggested that breathing polluted ambient air was stressful for guinea pigs. Not only were the reported effects marginal, but some of them may have been due to variations in temperature and humidity. Although the information obtained is of little value in the quantitative assessment of the health

hazards of air pollution, some subjects for possible future consideration were revealed: the effects of air pollutants on aged animals in which physiologic repair processes are less active, and the effects of other stressful stimuli on the response to air pollutants.

Emik and Plata[55] reported on studies of the effect of breathing ambient oxidant-polluted air in Riverside, California, on spontaneous running activity of mice, as recorded over a 1-year period. The controls were mice that were exposed to highly filtered air. Monthly peak oxidant concentrations in the unfiltered ambient air were 0.05–0.24 ppm; peak nitrogen dioxide concentrations, 0.04–0.07 ppm; and peak total hydrocarbon concentrations, 2.7–4.4 ppm. The daily activity of mice breathing filtered air was consistently higher than that of those breathing ambient air, and the differences between the two groups were greatest during the months when peak oxidant concentrations were highest. Other environmental variables, especially temperature, may also have contributed to the magnitude of differences between the groups.

Nakajima *et al.*[152] studied histopathologic changes in the lungs of mice that were exposed to irradiated auto exhaust and oxidant-fortified exhaust-gas mixtures for 2–3 h/day, 5 days/week for a month. Histopathologic changes resembling tracheitis and bronchial pneumonia were observed in mice exposed to atmospheres containing oxidant at 0.1–0.5 ppm, but not in those exposed to atmospheres containing 0.1–0.15 ppm. The changes were minimal in the latter group: the main finding reported was irregular arrangement of the epithelial cells of the relatively thick bronchioles.

As shown below in greater detail, ozone alone can produce many of the effects of complex mixtures of photochemical-oxidant air pollution that have been reported from studies on experimental animals. Studies of the response of animals to these complex mixtures have the advantage that they should detect the gamut of potential toxicity of photochemical-oxidant smog. However, the study of complex mixtures has limited value for mechanism studies, the most useful type of research for laboratory animal experimentation. The mere demonstration of similarity in quality of response to complex mixtures and selected individual constituents does not ensure that the constituents studied are the only or even the primary biologically active ones. At the same time, lack of similarity, both qualitative and quantitative, between effects of a mixture and effects of individual constituents may reflect interactive effects, both synergistic and antagonistic. The prime disadvantage of attempting to study the complex mixture of photochemical smog—either ambient smog or a realistic laboratory simulation—is the difficulty of obtaining reproducible, controlled atmospheres.

EFFECTS IN THE LUNGS

Disposition and Uptake

There is little information on the exact disposition and sites of uptake of ozone. However, because it is relatively insoluble in water, it can reach the periphery of the lung and hence has the opportunity to exert damage to both the central airways and terminal lung units. Ozone is highly reactive, and, given the concentrations found in ambient air, it is highly unlikely to be exhaled as ozone under conditions of normal breathing.

Frank and Yokoyama[67,213] have attempted to define the amount and site of ozone uptake in the lung by two methods: direct measurement of the drop in concentration of the gas across the upper airway (nose and pharynx) or the remainder of the airways, and predictions based on a computer model. In their animal experiments, they used anesthetized, paralyzed, mechanically ventilated beagles. The upper airways were surgically isolated, and ozone at two concentrations (0.7–0.85 and 0.2–0.4 ppm) was administered by nose or mouth. Flow rates were either high (35–45 liters/min) or low (3.5–6.5 liters/min). Each exposure lasted 20–30 min; but, because of the slow response of the Mast ozone meter, measurements of respiratory uptake were useful only during the last 4–5 min of exposure. The following observations were made: nasal uptake exceeded oral uptake at both high and low flow rates ($p < 0.01$); uptake of ozone, both nasally and orally, was inversely related to flow rate ($p < 0.01$), except when the gas was directed through the mouth at the high flow rate; and a decrease or increase in nasal resistance induced by drugs did not significantly change the uptake. The same investigators have developed a computer model for predicting uptake of ozone, with some assumptions related to solubility coefficient, an idealized version of the morphometry of the lungs,[208] and a fixed set of ventilatory parameters. Computer analysis has shown that the dosage of ozone is greater to the peripheral airways than to the alveolar structures.[67]

Effects of Short-Term Exposure to Ozone

MORPHOLOGIC EFFECTS

Gross autopsy findings of pulmonary edema and hemorrhage after acute exposure to ozone have been known for some time. Several studies have shown that edema and an acute inflammatory response occur in several species after brief exposures to ozone. The minimal concentration of ozone

that causes the responses that have been demonstrated depends very much on the methods used for the detection of edema.

Scheel *et al.*[182] provided histopathologic evidence of injury caused by a single acute exposure to ozone at 1.0 or 3.2 ppm for 4 h in mice and by repeated intermittent exposures (8–45 ppm for 1 h) in rabbits. No gross pulmonary edema was observed in mice killed immediately after exposure to 1 ppm, but moderately engorged blood vessels and capillaries containing an excess of leukocytes were found. Mice killed 20 h after exposure showed mild edema and migration of the leukocytes into the alveolar spaces. Inhalation of 3.2 ppm produced grossly visible edema during or shortly after exposure. The perivascular lymphatic vessels were distended and filled with edematous precipitate. Hyperemia (excess blood), mobilization of leukocytes, and various degrees of extravasation of red cells accompanied the edema. Damage to the respiratory tract consisted of loss of epithelium from the bronchioles; sheets of desquamated epithelial cells were seen in the lumen. The technique for measuring the gross edema produced (wet and dry lung weights), used in earlier studies, was not sufficiently sensitive to detect a mild edematous reaction. More refined methods, based on the recovery of radiolabeled blood albumin in pulmonary lavage fluid, indicate that the ozone-exposure threshold for edema formation in rats is 0.25–0.5 ppm for 6 h.[5]

The use of sophisticated techniques of light and electron microscopy have enabled several investigators to describe in detail the nature of the inflammatory response. Boatman *et al.*[16] observed a desquamation of the ciliated epithelium throughout the airways of cats exposed to ozone at 0.25, 0.5, and 1.0 ppm for 4–6 h. The intensity of the response appeared to be dose-related. Ultrastructural changes in the airways consisted of cytoplasmic vacuolization of ciliated cells and condensed mitochondria with abnormal cristae configuration. The latter occurred most often in the medium-sized airways, 0.8–1.2 mm in diameter. Alveolar ultrastructural changes included swelling and denudation of the cytoplasm of Type I cells, swelling or breakage of capillary endothelium, and lysis of red blood cells. Type II pneumocytes appeared normal. There was a considerable variation in the degree of response, both within a single lung and from animal to animal.

Stephens *et al.*[188] have shown that, in rats, the degenerative changes in Type I alveolar cells occur after exposure to ozone at concentrations as low as 0.2 ppm for 3 h and that cells are replaced by Type II alveolar cells beginning a day after the exposure. With electron microscopy, Bils[11] noted that the swelling of the epithelial alveolar lining cells of mice occurred after exposure to ozone at 0.6–1.3 ppm for 4 h. These changes

were not accompanied by fluid accumulation in the alveolar space. There was also a focal swelling of the endothelial cells and an occasional break in the basement membrane.

Electron microscopy has also shown that, in rats exposed to ozone at 3 ppm for 4 h and in mice exposed to 4 ppm for 3 h, acute inflammatory bronchiolar lesions occur.[166,211] These concentrations were high enough to produce alveolar edema, and the observed changes were similar to those found in earlier studies that used standard histologic techniques.

EFFECTS ON PULMONARY FUNCTION

Changes in pulmonary function have been observed in a variety of species after short-term exposure to ozone, including alterations in the elastic behavior of the lungs, increased resistance to flow, and decreased carbon monoxide diffusion capacity.

Scheel *et al.*[182] exposed 75 rats to ozone at 2 ppm for 3 h and measured pulmonary function immediately after removal of the animals from the exposure chamber. Minute ventilation, tidal volume, and oxygen uptake decreased immediately after exposure and reached minimal recorded values after 8 h. At 20 h after exposure, all measurements had returned to normal. Pulmonary edema may have been responsible for the observations reported.

Murphy *et al.*[146] exposed guinea pigs to ozone at 0.34–1.35 ppm for 2 h. Respiratory rates increased and tidal volumes decreased during exposure to all concentrations. The maximal changes were significantly different ($p < 0.05$) from pre-exposure control values for every concentration used. Once a maximal response was reached, the effects tended to remain constant for the remainder of the test period. Respiratory rates and tidal volumes tended to return to pre-exposure control values when the animals were returned to clean air. Total respiratory flow resistances were not significantly altered during inhalation of ozone at 0.34 and 0.68 ppm, but increased by 47% during exposure to 1.08 ppm.

Easton and Murphy[52] observed an increase up to threefold in flow resistance within an hour in guinea pigs during exposure to ozone at 5–7 ppm. There was a 50% increase in frequency of breathing and a small increase in tidal volume. Lung compliance decreased by 50%. After exposure, all measurements returned to pre-exposure values within 3 h. These responses did not result in an increase in the work of breathing, in contrast with the response of guinea pigs to other irritants (formaldehyde and sulfur dioxide), in which the observed changes in pulmonary function resulted in an increase in the work of breathing.[7]

Recently, Watanabe *et al.*[206] observed changes in respiratory mechanics

and single-breath diffusing capacity in cats exposed to ozone at 0.26–1.0 ppm for an average of 4–6 h. The animals were anesthetized, paralyzed, and mechanically ventilated through a tracheal cannula. Increased pulmonary flow resistance was the most sensitive index of ozone exposure in this species and occurred in almost all the animals exposed to 1.0 and 0.5 ppm and in two cats exposed to 0.25 ppm. Although the magnitude of the change was dose-related, it appeared that the concentration of ozone was more critical than the duration of the exposure. The mechanism of the response to ozone at 1.0 ppm appeared to differ from that to the lower concentrations in that there was a pulsatile increase in resistance that was superimposed on a more progressive change. The underlying increase in resistance was approximately exponential with respect to time. Changes in dynamic compliance and capacity for diffusing carbon monoxide were less frequent and less marked than the change in resistance. Vital capacity did not change. The shape of the volume–pressure curve during deflation did not change, and that suggests that alveolar stability was not affected. In cats, therefore, exposure to ozone affects airway caliber at concentrations lower than those which affect transfer of carbon monoxide across the alveolar-capillary membrane or alveolar surface forces in the absence of pulmonary edema. Unlike the increase in pulmonary flow resistance that results from exposure to sulfur dioxide, the response to ozone is not mediated entirely via reflex bronchoconstriction, inasmuch as it was only partially reversed by atropine in these experiments. About half the response persisted after the administration of the drug. Thus, the small airways are implicated in the persistent increases in pulmonary flow resistance in response to ozone. This implication is further supported by the following observations: vagal innervation ends short of the peripheral airways, ozone can penetrate to the periphery of the lung, and postmortem studies show that inflammatory changes occur in the peripheral airways.

Unilateral exposure of the lung may be achieved in rabbits, because they have an intact mediastinum, which permits the unexposed lung to serve as a control for the exposed lung. The control lung may be collapsed, and ozone inhaled in a normal fashion by the noncollapsed lung; or, by selective catheterization, one lung can be made to breathe ozone through an airtight system while the other breathes ambient air. Rabbits so exposed to ozone at 12 ppm had increases in respiratory rate and minute volume and bilateral decreases in tidal volume and dynamic compliance.[4] Pulmonary-function measurements were not significantly different in the ozone-exposed lung until edema developed, as determined by an increase in the wet weight of that lung. After edema formation, tidal and minute volumes, inspiratory and expiratory flow rates, and dynamic compliance

decreased and pulmonary flow resistance increased in the lung exposed to ozone, relative to the unexposed lung.

Studies have been performed on the effects of ozone exposure on excised dog lungs.[70] This preparation offers the advantages of providing information on changes in pulmonary function in the absence of intra-alveolar edema and vagal innervation. High concentrations of ozone were used (6.8–10.3 ppm for 2.5 h and 5–10 ppm for 3 h). The changes in static-deflation volume-pressure relationships were barely detectable, whether measured in air or saline. The data suggest that exposure to ozone does not produce significant depletion or degradation of either surface-active material or tissue elastic elements in the absence of edema. Thus, it seems that the changes in elastic behavior, as determined by changes in dynamic lung compliance, are secondary to the formation of edema, unevenly distributed changes in flow resistance or compliance, and a change in tidal volume or end-expiratory lung volume.

There is evidence that ozone has a delayed effect on elastic behavior. Excised lungs of rabbits that were unilaterally exposed to ozone at 1.0 ppm for 3 h and then allowed to breathe ambient air for up to 2 weeks showed a depression in the volume-pressure curves. This change was present during most of the postexposure test period. The mechanism underlying this response was not determined.[67]

Frank *et al.*[68,69] exposed the right lungs of rabbits to ozone at 2.2–12.1 ppm for 3 h, the left lungs having been collapsed before exposure. When edema occurred in the right lungs, changes in surfactant behavior (as determined by *in vitro* measurements of the surface tension of washings from the left lungs) were observed in some animals. No such changes were observed in the absence of edema in the right lungs. These results suggest not only that ozone can induce chemical changes in exposed lungs, but that the products of such changes can cause deleterious effects in non-exposed lungs.

Effects of Prolonged Exposures to Ozone

According to Stokinger,[190] at least three effects of long-term exposure to ozone have been recognized: effects on morphology and function of the lung, lung-tumor acceleration, and aging. An additional effect, the development of tolerance after exposure to low concentrations of ozone, may also be related to chronic toxicity.

Stokinger *et al.*[195] reported that chronic bronchitis, bronchiolitis, and emphysematous and fibrotic changes in the lung tissues occur in mice, hamsters, and guinea pigs exposed daily to ozone at a concentration slightly above 1 ppm. These irreversible changes also develop in animals that have developed tolerance to acute inflammatory effects.

Freeman *et al.*[73] used tissue fixation and electron microscopy and reported on the morphologic changes that occurred in the lungs of dogs exposed to ozone at 1-3 ppm for 5-24 h/day for up to 18 months. In general, the effects reported tended to increase in severity with the concentration used, rather than with the duration of the exposure. There was no evidence of pulmonary edema. The most noticeable change was a thickening of the terminal and respiratory bronchioles. This change was barely noticeable at the lowest concentrations used, but at the highest concentrations it was accompanied by infiltration of lymphocytes, plasma cells, and fibroblasts that formed peribronchiolar collars, thus reducing the caliber of the small airways. Bronchiolar changes included an increase in the proportion of mucus-forming cells and squamous metaplasia of columnar and cuboidal cells. In addition, there was an increase in the average number of macrophages per field along the peripheral airways.

In developing rats exposed to ozone at 0.54 or 0.88 ppm for up to 3 weeks, morphologic changes were seen in the respiratory bronchioles and distal portions of the terminal bronchiolar epithelium, the entire alveolar duct, and the associated alveoli. These changes started to occur after 24 h of exposure; but, after several days of exposure, the hypertrophic bronchiolar epithelium tended to recede and was replaced by cuboidal or squamous cells. The lungs of growing rats exposed to ozone at 0.9 ppm were approximately 38% heavier than those of normal animals of the same age; within 3 weeks, 50% had died with grossly inflated lungs.[72]

Mice exposed to ozone at 2.5 ppm for 120 days showed progressive metaplasia along the tracheal bronchial tree. Return of these animals to clear air resulted in a reversal of these changes after an additional 120 days.[162]

Bartlett *et al.*[10] exposed 194 young rats (3-4 weeks old) continuously to ozone at 0.2 ppm for 28-32 days and observed that there was no effect on respiratory frequency, weight gain, tail-length increase, and external appearance in the ozone-exposed group and that, although both ozone-exposed and control groups looked healthy, 12 ozone-exposed and 11 control animals had pneumonitis at the end of the exposure period. The results with the latter animals were discarded in the later data analysis. The lung volumes of the ozone-exposed group were 16% greater than those of the control group. Air or saline static volume-pressure deflation curves at 95% or 100% of total lung capacity showed that transpulmonary pressure was significantly lower in the ozone-exposed group, thus confirming that exposed lungs were overdistended at high transpulmonary pressures. The fact that this occurred in both air- and saline-filled lungs suggested a small change in tissue elasticity, rather than abnormal surface tension. There were no apparent morphologic differences between the two groups detected by conventional staining techniques, but more

sensitive electron-microscopic techniques might have revealed some differences.

Acceleration of lung tumorigenesis (adenoma) in a strain of mice that was susceptible to lung tumors occurred after daily exposures to ozone at about 1 ppm. At 15 months, an incidence of 85% was seen in the ozone-exposed animals, compared with 38% in the controls; the average number of tumors per mouse was 1.9, compared with 1.5 in the controls.[190]

There is some suggestive evidence that exposure to ozone accelerates the aging process(es). Bjorksten[12] and Bjorksten and Andrews[13] have presented evidence that aging is due to irreversible cross-linking between macromolecules, principally proteins and nucleic acids. Aldehydes were included in the list of cross-linking agents, and these can be produced in the lung as a result of ozone exposure.[26]

Tolerance

One feature of the response to oxidants (in particular, ozone) that has stimulated considerable interest is the apparent development of tolerance to the acute effects of short-term exposure to these agents in laboratory animals. Fairchild[58] reviewed possible mechanisms of this phenomenon. Tolerance has been defined as the increased capacity of an organism that has been pre-exposed to oxidant to resist the effects of later exposures to ordinarily lethal (or otherwise injurious) doses of the same agent or of different agents (cross-tolerance) with similar toxicologic properties.

Development of tolerance to the acute toxicity of ozone in experimental animals has been demonstrated after a single, brief (1 h or less) exposure to low concentrations (0.3–3 ppm). Rats can develop a tolerance that lasts for a month or longer. In mice, a tolerance lasting up to 14 weeks has been observed. Ozone is not the only lung edematogenic agent that can produce tolerance. This phenomenon is also observed with nitrogen dioxide, phosgene, and phenylthiourea. The tolerance evoked by one agent can provide cross-protection against other irritants. For example, a single exposure of rats or mice to ozone (0.5–5 ppm for 1–5 h) will induce protection against the acute pulmonary effects of nitrogen dioxide, hydrogen peroxide, ketene, phosgene, hydrogen sulfide, and nitrosyl chloride.[58] The development of pulmonary edema is the toxic effect of all these compounds. Although pretreatment by injection of phenylthiourate will provide tolerance to inhalation of an ordinarily lethal dose of ozone, the reverse is not true; inhalation of ozone will not provide cross-tolerance to the lung edema produced by injection of thiourate.

Studies by Henschler *et al.*[101] on the development of tolerance to nitrogen dioxide in mice provided some insight into the significance of this

phenomenon. They found that mice that had been exposed only once to nitrogen dioxide were almost totally protected from effects of later exposures to high, usually fatal concentrations, whereas those which were repeatedly exposed were only partially protected. It was concluded that the first exposure protected from the effects of the following exposures, thus inhibiting increased tolerance buildup. It is significant that, in another experiment, mice tolerant to nitrogen dioxide were exposed to a concentration high enough to cause lethality, but none of the mice died with pulmonary edema. The predominant gross finding was massive hemorrhage.

In animals made tolerant to ozone and later exposed to ordinarily injurious concentrations, the usual increase in lung water and other pathologic changes associated with edema were either reduced or absent, and the usual ozone-induced changes in activity of serum alkaline phosphatase and adrenal succinic dehydrogenase were absent or slight. In addition, there was significantly less oxidation of lung-reduced glutathione in the tolerant animals.[58] It should be pointed out that there may be species differences in tolerance development. Quilligan *et al.*[174] used several different conditions of exposure and challenge and were unable to demonstrate tolerance to ozone in baby chicks; and it is not known whether humans develop tolerance to ozone.

Tolerance appears to be a local phenomenon, in that experiments with unilateral exposure showed that a lung is not protected from pulmonary edema unless it has been exposed itself. Thus, no tolerance was produced in the contralateral, unexposed lung; this suggested that there is no evidence of a circulating humoral agent that confers tolerance.[75] The same investigators also attempted to show whether induction of tolerance affected the cytotoxic effects of ozone on alveolar macrophages (discussed in the next section). They used unilateral exposure and lavage and observed no differences in the cytotoxicity of ozone between tolerant and nontolerant lungs. A twofold increase in the number of polymorphonuclear neutrophilic leukocytes occurred in the tolerant lung. Alveolar-macrophage enzyme activities were equally depressed in both tolerant and nontolerant lungs. This study suggested, therefore, that tolerance does not protect the antibacterial defense mechanisms of the lung from the effects of ozone. In addition, Murphy *et al.*[146] found that exposure to ozone, which produced tolerance in guinea pigs to the edema produced by later exposure to high concentrations, failed to produce tolerance to the effects of low concentrations of ozone on respiratory frequency and tidal volume.

It appears that the development of tolerance is a useful tool to determine the mechanism of ozone-induced pulmonary edema. However, in the light of the findings that repeated intermittent exposures of mice to nitrogen dioxide produced less protection from the effects of a lethal dose

than a single pre-exposure and the failure to produce tolerance to the effects of ozone on antibacterial defense mechanisms or on the respiratory patterns of guinea pigs, it is unlikely that tolerance development provides significant protection in human populations continuously exposed to low concentrations of oxidants.

Defense Mechanisms

It has been observed that animals challenged with aerosols of infectious organisms suffer a higher incidence of infection if they have been previously exposed to ozone, irradiated auto exhaust, or other common pollutants. The suggested explanation for this is that the various pollutants inhibit, inactivate, or otherwise impair two distinct functions: mucociliary streaming, the action of cilia in the nasal and upper respiratory passages that clears particles and thus prevents them from entering the lungs, and phagocytosis by alveolar macrophages.

Miller and Ehrlich[133] determined the effect of exposure to ozone on the susceptibility of mice and hamsters to respiratory infection caused by inhalation of *Klebsiella pneumoniae* aerosol. The ozone exposures used were 1.3–4.4 ppm for 3 h and 0.84 ppm for 4 h per day, 5 days/week for 2 weeks. The observation period for this experiment was 2 weeks. Mortality and survival time were measured. The mortality due to *K. pneumoniae* infection was significantly greater ($p < 0.05$) for every exposure regimen in which animals were exposed to ozone than for their paired controls. No deaths were caused by ozone exposure alone. Autopsy of the animals exposed to *K. pneumoniae* that died within the 14-day holding period showed the infectious organism in the lungs and heart. *K. pneumoniae* was absent in animals that survived the 14-day holding period. It was concluded that exposure to ozone significantly reduced the resistance of mice and hamsters to later respiratory infection due to *K. pneumoniae.* Statistical evaluation of the data indicated higher mortality, shorter survival time, and a lower LD_{50} (for *K. pneumoniae*) in animals exposed to ozone than in controls.

In a similar series of experiments conducted by Purvis *et al.*,[173] mice were exposed to ozone at 3.8–4.1 ppm for 3 h, 1–27 h before and 3–27 h after challenge with *K. pneumoniae* aerosol. Within 19 h after exposure to ozone, the resistance of mice to respiratory infection initiated by challenge with the aerosol was significantly reduced. The same effect was observed in infected animals exposed to ozone up to 27 h after challenge with the aerosol.

Coffin and Blommer[35] have shown that mice exhibit increased mortality

from *Streptococcus* aerosol after exposure to ozone at 0.08 ppm for 3 h. This concentration of ozone also produced a decrease in the rate of kill of bacteria deposited in the lungs and hence an increase in their later multiplication.[37] This effect was noted 4 days after exposure, but not 4 h after exposure.

Goldstein *et al.*[92] studied the effect of ozone on the *in vivo* rate of bacterial killing in the mouse lung. They used male Swiss mice infected with aerosols of *Staphylococcus aureus* labeled with phosphorus-32. The animals were sacrificed immediately or after exposure to ozone at 0.62–4.25 ppm for 4 h, and the radioisotope concentration in the lungs was determined. Increased numbers of bacteria were consistently cultured from the lungs of animals exposed to ozone, compared with controls, and the magnitude of the effect was dose-related up to 2.58 ppm. Some mice exposed to 2.2 ppm or greater yielded more staphylococci than were inhaled initially; thus, intrapulmonary bacterial multiplication had occurred. The difference between control and infected animals was significant ($p < 0.05$) at 1.10 ppm, when individual aerosol experiments were compared. When the data from all the experiments were pooled, there were significant differences in bacterial activity between control and treated mice for each ozone exposure ($p < 0.05$). Inasmuch as the ozone-exposed mice were able to remove the same number of bacteria as control mice via mucociliary streaming, the investigators concluded that the inhibition of bactericidal activity resulted from the toxic effect of ozone on the alveolar macrophages.

Alpert and Lewis[3] used unilateral exposure to study the effect of ozone on the defense mechanisms of the lungs. Rabbits were exposed unilaterally to ozone at 0.5, 0.75, and 3 ppm for 3 h. The alveolar macrophages were harvested by pulmonary lavage. Although there was a statistically significant reduction in the viability of the cells from the ozone-exposed lung, compared with the air-exposed lung, the differences were small. There was also a dose-related inhibition of the intracellular hydrolytic enzymes acid phosphatase, lysosome, and β-glucuronidase. The fraction of total cells present as polymorphonuclear neutrophilic leukocytes in the lavage fluid from the exposed lung increased in a dose-related fashion from approximately 2% at 0 ppm to approximately 28% at 3 ppm. Associated with the increase in the fraction of polymorphonuclear neutrophilic leukocytes was a reciprocal decrease in the fraction of alveolar macrophages, although the total number of macrophages per gram (dry weight) remained unchanged. The authors suggested that the depression of the intracellular hydrolytic enzymatic activity by ozone may contribute to the ozone-caused impairment of lung defense mechanisms.

Interactions with Bronchoactive Agents

The lung is relatively rich in histamine-containing mast cells, so it is not surprising that a number of investigators have assessed the role of histamine in the pulmonary toxicity observed after ozone exposure.

Among the many effects of histamine are edematogenic alterations in vascular endothelium and bronchoconstriction. The latter may be particularly pertinent, in view of its being a central manifestation of asthma attacks and of the reported association of asthma attacks with photochemical air pollution. Bronchoconstriction has also been observed in animals and man experimentally exposed to ozone or photochemical air pollution.

Dixon and Mountain[49] reported that exposure of mice to ozone at 1 ppm for 5 h resulted in a depletion of lung histamine content to as low as 75% of the control value 5 days later. They also observed that pretreatment with promethazine, an antihistaminic agent, resulted in a decrease in the amount of pulmonary edema produced by a sublethal dose of ozone. However, promethazine, in addition to being a potent antihistaminic agent, is a phenothiazine derivative and thus might act to trap free radicals or stabilize membranes.

Other investigators have been unable to demonstrate lung histamine depletion in rats exposed to ozone at 4 ppm for 4 h[41] or guinea pigs exposed to ozone at 1–5 ppm for 3 h.[52] In the latter study, it was observed that exposure to ozone at 5 ppm for 2 h followed by challenge with histamine at 0.9–1.4 mg/kg (injected 1.5–2.0 h after the end of exposure) resulted in increased mortality, compared with that in an air-exposed control group. The increased susceptibility to histamine was detectable for 120 h after the end of exposure to ozone at as low as 0.8 ppm. This concentration is about one-twentieth of that required to produce death from pulmonary edema due to ozone alone. It was found that the increased susceptibility occurred only when exposure to ozone took place before challenge with histamine. There was no increase in susceptibility in guinea pigs that received histamine before exposure.

Potentiation of the action of acetylcholine, another bronchoconstrictive agent implicated as a mediator in asthma, has also been reported in guinea pigs exposed to ozone at at least 2 ppm for 30 min before inhalation of acetylcholine;[124] the effect was observed intermittently for up to 23 h after ozone exposure.

The reported potentiation by ozone of the membrane damage produced by the indirect pathway of complement[85] might also play a role if it occurs *in vivo*, inasmuch as this complement pathway apparently mediates the allergin–reagin-induced release of histamine.[123] In view of

the fact that asthma attacks usually occur at night, whereas ozone peaks in ambient air occur late in the morning, it might be more reasonable to ascribe the epidemiologic association of photochemical oxidants with asthma (if such exists—see Chapter 10) to an ozone-induced alteration of the respiratory tract, either potentiating an otherwise subclinical effect of an allergen or increasing the reactivity of smooth muscle to bronchoactive compounds.

The vasoactive compound serotonin has also been suggested to be responsible for ozone-induced pulmonary edema by Skillen *et al.*,[187] who noted a decrease in lung serotonin after ozone exposure. Prostaglandins may also be involved in ozone toxicity, in view of a report that aspirin, a prostaglandin inhibitor, protects from ozone toxicity.[49] Prostaglandins are released from injured lungs, possibly as a result of membrane damage.[165] Recently, Tan *et al.*[197] demonstrated that injection of hydroperoxides, which results in lung damage similar to that caused by injected ozonides,[39] appeared to interfere with lung prostaglandin synthesis.

In a study of the mechanism whereby *Bordetella pertussis* vaccine increased acute ozone toxicity in rats,[59,201] Thompson[200] ascribed the effects to β-adrenergic blockade, and not to an immune-mediated response. It was further noted that both atropine and reserpine reduced mortality, which suggested that the acute lethal effects of ozone were due to shock and circulatory collapse, rather than pulmonary edema.

The release of bronchoactive compounds may result from immune reactions. A possible role for the immune system in chronic ozone toxicity was suggested by Scheel *et al.*[182] and by Stokinger and Scheel,[194] who hypothesized that ozone altered lung protein, making it immunogenic to the host. Presumably, the resulting antibody would cross-react with normal lung tissue and produce damage. Although some indirect evidence to support this hypothesis was presented, it has been questioned.[25] In addition, the suggestion that neonatal thymectomy altered ozone toxicity[95] has not been confirmed.[201]

BIOCHEMICAL ACTIONS AND MECHANISMS

Molecular Reactions

FREE-RADICAL EFFECTS OF OZONE

The idea that ozone toxicity is expressed through the formation of reactive free-radical intermediates was originally derived from studies that noted the similarity of the effects of ozone to those of radiation. Earlier studies

included the following observations: a similar and generally additive effect of ozone and X irradiation in producing chromosomal aberrations in *Vicia fava* seeds;[63] radiationlike effects on the deoxygenation of hemoglobin in the occluded digits of humans breathing ozone at 1 ppm for 10 min;[20] the protective effects of thiol radioprotective agents in ozone toxicity;[61] the ability of ozone to produce mutations and chromosomal damage; and an increase in the radiation-induced sphering of human and animal red cells *in vitro* after ozone inhalation.[21] These studies and the idea of free-radical effects of ozone have been reviewed by Stokinger,[192] Veninga,[204] Menzel,[128] Zelac *et al.*,[214] and others. As discussed in more detail later, Zelac *et al.*[214,215] have also demonstrated a nearly additive effect of ozone and X irradiation in producing lymphocytic chromosomal breaks in Chinese hamsters. Furthermore, other antioxidant and radical-trapping agents—including various quinones, ascorbic acid, and α-tocopherol—have been reported to protect against ozone toxicity.[84,86,125,129,156,176]

The exact radicals produced by ozone *in vivo* are not fully understood. Alder and Hill[1] proposed that the aqueous decomposition of ozone produced both hydrogen and hydroxyl free radicals and that the reaction was catalyzed by base. Fetner[63] suggests that hydroxyl and peroxy radicals were responsible for the observed radiomimetric effects. It should be noted that the peroxy radical dissociates to form the superoxide anion radical at physiologic pH. Recently, Mustafa *et al*[151] noted an increase in lung superoxide dismutase after ozone exposure, which indirectly suggested the presence of the superoxide anion radical. Stokinger,[192] noting the lability of sulfhydryl groups in the presence of ozone and radiation, hypothesized that free radicals might be derived from the interaction of ozone with sulfhydryls. A similar reaction pathway has been proposed by DeLucia *et al.*[45] That ozone-induced free radicals are derived from the oxidative decomposition of unsaturated fatty acids has been suggested by B. Goldstein and Balchum,[81] Menzel,[128] and Roehm *et al.*[177] B. Goldstein *et al.*[83] used electron paramagnetic resonance to demonstrate free-radical signals in ozonized linoleic acid. It is also conceivable that singlet oxygen is derived directly from ozone or via the decomposition of an ozonide. Furthermore, hydrogen peroxide, which may be formed in aqueous solution by ozone or these intermediates, has been indirectly identified in the red cells of animals that inhaled ozone.[79]

All these rapidly reacting intermediates are potentially harmful to the cell and might play a role in ozone toxicity. Furthermore, the potential for ozone-induced free-radical chain reactions exists. It appears likely that more than one radical is formed, either directly from ozone or as a result of the interaction of ozone with normal cellular constituents.

The implications of various oxidative free-radical reactions for biologic processes are being studied. Many of these reactions appear to play a role in normal cellular processes and may be of pathologic significance only if not contained by normal defense mechanisms. It is of interest that Shoaf *et al.*[185] have suggested on the basis of energetics that ozone might be derived from the superoxide anion radical. Inasmuch as superoxide is formed during some intracellular processes, ozone might be a normal transient intermediate *in vivo*. Ozonides have also been produced in the absence of ozone by the photooxidation of diazo compounds in the presence of aldehydes.[147] Further information concerning the interrelationships of ozone, free radicals, peroxides, ozonides, and singlet oxygen in biologic systems would be useful.

SULFHYDRYL COMPOUNDS AND PYRIDINE NUCLEOTIDES

In view of the oxidant nature of ozone, a number of investigators have evaluated its effects on intracellular compounds that are normally active in cellular redox reactions. Attention has focused particularly on reduced pyridine nucleotides—reduced nicotinamide adenine dinucleotide (NADH) and reduced nicotinamide adenine dinucleotide phosphate (NADPH)—and on sulfhydryl compounds, specifically reduced glutathione (GSH).

Mudd,[137] Mudd *et al.*,[139] Menzel,[127] and Nasr *et al.*[153] have reported that ozonization of aqueous solutions of NADH or NADPH results in their oxidation. However, there is a difference in their findings as to whether the resulting product is a biologically active oxidized pyridine nucleotide (NAD or NADP), as suggested by Menzel,[127] or is molecularly disrupted to the extent that it is unable to participate in enzymatic processes. Inasmuch as more drastic effects are likely to be observed *in vitro*, it is more likely that oxidation of intracellular reduced pyridine nucleotides proceeds mainly to NAD or NADP after ozone inhalation; but further resolution of this question would be of value.

Nasr *et al.*[153] failed to observe a change in the ratio of NADPH to NADP in the tracheal epithelium of rats exposed to ozone at 33 ppm for an hour. This apparently negative *in vivo* finding is not surprising, inasmuch as NADP will be rapidly reduced back to NADPH if ozone does not disrupt the structural integrity of pyridine nucleotides. In addition, *de novo* synthesis of pyridine nucleotides may also occur. The intracellular ratio of reduced to oxidized pyridine nucleotides is under fine cellular control, in that the oxidation of NADPH or NADH results in the stimulation of enzymatic activity, which restores the initial ratio. In the case of NADPH, its oxidation increases the activity of the hexose monophosphate shunt; this also occurs after the oxidation of glutathione. The rel-

evant enzymatic steps are depicted in Figure 8-1. The direct interrelationship of glutathione and NADPH occurs in the reduction of oxidized glutathione to reduced glutathione by glutathione reductase. NADPH is required in this step and is itself oxidized to NADP. Accordingly, the oxidation of either NADPH or GSH conceivably accounts for the apparent increase in hexose monophosphate shunt enzymes after repeated ozone exposure.

De Koning and Jegier[44] also measured pyridine nucleotides in a study in which exposure of *Euglena gracilis* to ozone at 0.8 ppm for an hour in the presence of light produced a 9–12% decrease in NADH formation.

$$2GSH + [O] \xrightarrow{\text{G. peroxidase}} GSSG + H_2O$$

$$GSSG + 2NADPH \xrightarrow{\text{G. reductase}} 2GSH + 2NADP$$

$$G\text{-}6\text{-}P + NADP \xrightarrow[\text{(HMP shunt)}]{\text{G-6-PD}} 6\text{-}PG + NADPH$$

GSH ≙ reduced glutathione

GSSG = oxidized glutathione

G. peroxidase = glutathione perioxidase

G. reductase = glutathione reductase

G-6-PD = glucose-6-phosphate dehydrogenase

6-PG = 6-phosphogluconate

G-6-P = glucose-6-phosphate

[O] = oxidizing moiety, e.g., hydrogen peroxide, free radical, lipid peroxide

HMP shunt = hexose monophosphate shunt

NADP = nicotinamide adenine dinucleotide phosphate

NADPH = reduced nicotinamide adenine dinucleotide phosphate

FIGURE 8-1 Some enzyme processes active in defending against oxidant stress.

Histochemical studies have demonstrated an alteration in NADPH- and NADH-diaphorase activity in the lungs of rats exposed to 0.8-ppm ozone for 7 days.[30]

A possible interaction of ozone with sulfhydryl groups *in vivo* was originally suggested by Fairchild *et al.*,[57,61] who noted that protection from lethal concentrations of ozone was conferred by the injection or inhalation of sulfhydryl or disulfide compounds. Other investigators have also noted protection from ozone toxicity by sulfhydryl compounds.[20,71,107,127] Mountain[136] observed a decrease in lung glutathione and in the activity of the sulfhydryl-containing enzyme succinic dehydrogenase after ozone exposure. The possible toxicologic implications of the reaction of ozone with sulfhydryl compounds were discussed in detail by Stokinger.[192]

Exposure of aqueous solutions of GSH to ozone results in the formation not only of GSSG, but also of higher oxidation states, including the sulfoxide.[127] This is important, in that, although the enzymes shown in Figure 8-1 are capable of recycling GSSG back to GSH, the production of the sulfoxide or the sulfone may lead to irreversible loss of glutathione. By analogy, a similar oxidation of protein sulfhydryl groups to the sulfoxide would also presumably prevent reduction by a disulfide-exchange mechanism with glutathione and therefore lead to irreversible inactivation. However, the ozone-induced production of sulfoxides or sulfones has not been reported *in vivo*.

DeLucia *et al.*[45] reported that exposure of rats to 2-ppm ozone for 4–8 h resulted in a statistically significant decrease in both protein and nonprotein lung sulfhydryl groups. But rats exposed to 0.8-ppm ozone for 10 days showed no change in lung sulfhydryl concentrations. As pointed out by the authors, this finding suggests lung adaptation and is consistent with their observation that chronic ozone exposure resulted in an increase in glucose-6-phosphate dehydrogenase (G-6-PD) concentration, despite a decrease in its activity after acute exposure. Glutathione reductase, NADH, and succinate cytochrome *c* reductase concentrations were also decreased after acute ozone exposure; this was ascribed to an effect on protein sulfhydryl groups. However, there is no firm evidence that free sulfhydryl groups are involved in the active sites of any of these enzymes in the lung. The cytochrome *c* reductases and glutathione reductases both contain flavins, which are relatively readily oxidized intermediates. Further studies on the sensitivity of flavin compounds to ozone appear warranted. In addition, there is evidence of an effect of ozone on cytochrome heme compounds, which might explain the decrease in cytochrome *c* reductase observed by DeLucia *et al.*[45]

More recently, DeLucia *et al.*[46] reported that the bulk of the glutathione oxidized in rat lung after exposure to 4-ppm ozone for 6 h was in the

form of mixed disulfides with lung protein sulfhydryl groups. No increase in the concentration of oxidized glutathione was noted. Peak oxidation of nonprotein sulfhydryl groups did not occur until about 24 h after exposure, and recovery was evident at 48 h. Effects of exposure to lower concentrations (0.8 ppm for 24 h and 1.5 ppm for 8 h) were not observed.

Further detailed studies of the effect of ozone on enzymes active in the defense against intracellular oxidation were performed by Chow and coworkers.[31-33] Chronic ozone exposure resulted in an increase in the activity of G-6-PD and 6-phosphogluconic dehydrogenase, two constituents of the hexose monophosphate shunt, and an increase in glutathione peroxidase and glutathione reductase concentrations (see Figure 8-1). Of particular note is a study in which groups of rats were exposed to ozone at 0.2, 0.5, or 0.8 ppm continuously for 8 days. The increases in G-6-PD, glutathione peroxidase, and glutathione reductase were linearly related to dose. Statistically significant differences were present after exposure to ozone at 0.2 ppm. In an additional study, exposure of rats to ozone at 0.75 ppm resulted initially in a decrease in the activity of these enzymes and then an increase, as exposure continued.[32] As discussed below, the authors related their findings to the action of glutathione peroxidase in detoxifying lipid peroxides, rather than to an ozone-induced intracellular aqueous radical or hydrogen peroxide. However, if the latter processes do occur, a similar increase in these enzymes after chronic exposure might be expected.

LIPIDS

Unsaturated fatty acids (UFA) are readily oxidized cellular macromolecules whose oxidative breakdown has been observed in a number of situations analogous to ozone toxicity, including radiation, exposure to hyperbaric oxygen,[126] and inhalation of nitrogen dioxide.[199] The classic mechanism by which free radicals and oxidative states interact with the carbon–carbon double bonds of UFA is the formation of peroxides. Their decomposition may result in further free-radical production capable of initiating peroxidation of additional UFA. The resulting breakdown of the UFA molecule results in various addition and decomposition products, including peroxides and carbonyl compounds, which may themselves be toxic to the cell. Ozonization of UFA appears in many ways similar to lipid peroxidation in the resulting products and biologic implications. However, the initial attack on the double bond proceeds by a different mechanism. This has been studied in ozonized fatty-acid methyl ester emulsions and thin films by Roehm *et al.*,[176,177] whose findings support the general mechanism originally proposed by Criegee[40] (Figure 8-2). In this scheme, ozone

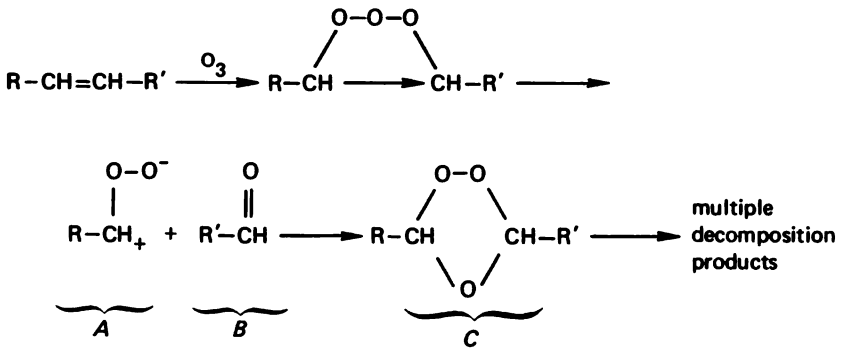

FIGURE 8-2 Mechanism of ozonolysis. *A*, Criegee zwitterion; *B*, aldehyde; *C*, ozonide.

directly attacks carbon-carbon double bonds; this results, after decomposition of the double-bond, in the formation of a zwitterion and aldehyde. These recombine to produce the ozonide. As pointed out by Roehm *et al.*,[176] in the presence of water the zwitterion may form peroxides that can then catalyze the peroxidation of additional molecules of UFA. The ozone-induced oxidative decomposition of UFA in model membranes and emulsions has also been evaluated by Menzel *et al.*[129] and Teige *et al.*,[198] and the subject has been reviewed by Menzel.[128]

It is not clear from cellular and *in vivo* experiments whether the observed ozone-induced breakdown of UFA proceeds through the direct attack of ozone on UFA, through the effects of an ozone-induced free radical that result in lipid peroxidation, or through a combination of these processes. Inasmuch as phenolic antioxidants are not as effective in protecting against the direct attack of ozone on double bonds as they are in the autocatalytic lipid peroxidation process,[177] the finding that vitamin E protects against ozone toxicity might indirectly indicate that ozone-induced lipid peroxidation does occur. However, Menzel *et al.*,[130,131] in a series of studies with preformed fatty-acid ozonides, have concluded that such compounds may be responsible for a significant proportion of ozone toxicity. Because "lipid peroxidation" is the term commonly used in the literature and the biologic implications of lipid ozonization and lipid peroxidation appear to be relatively similar, we will use the latter term in discussing the effects of ozone on UFA.

Evidence of a role of lipid peroxidation in the cellular toxicity of ozone has been obtained in *in vitro* studies in which human red cells were exposed to this oxidant gas.[9,81] The possibility that lipid peroxidation is responsible for altered permeability of bacterial cell walls after ozone exposure was proposed by Scott and Lesher[184] and has since been con-

firmed by others. Of note is the study of Teige *et al.*[198] in which red cells were lysed more readily by incubation with ozonized liposomes than they were by ozone itself. Similarly, Menzel *et al.*[130] noted that incubation of red cells in ozonized serum produced more toxicity than did direct ozonization of the red cells in the absence of serum. Menzel *et al.*[130] also demonstrated that incubation of human or mouse cells with fatty-acid ozonides produced Heinz bodies, methemoglobin formation, oxidation of thiol groups, and formation of mixed disulfides with hemoglobin. Oral administration of vitamin E protected against Heinz-body formation *in vitro*. Heinz bodies were also observed in the red cells of mice exposed to ozone at 0.85 ppm for 48 h. Lung toxicity that mimicked that observed after ozone exposure has been seen by Cortesi and Privett[39] in rats that received intravenous injections of fatty-acid hydroperoxides or ozonides.

Lung lipid peroxidation during ozone inhalation was suggested by the finding of conjugated diene bonds in an extract of the lungs of mice exposed to ozone at 0.4–0.7 ppm for 4 h.[87] The presence of thiobarbituric acid reactants, predominantly malonaldehyde (another index of lipid peroxidation), has been observed in the lungs of rats exposed to ozone at 0.7–0.8 ppm continuously for 5–7 days.[33] The malonaldehyde concentration was linearly related to the concentration of glutathione perosidase, and animals given α-tocopherol supplements did not have as great an increase in either malonaldehyde or glutathione peroxidase. The authors suggested that the increase in glutathione peroxidase concentration reflected the activity of this enzyme in detoxifying lipid peroxides, rather than a more direct ozone-induced intracellular radical or oxidizing species. This is a crucial point, particularly because in later studies there was no apparent threshold for the increase in glutathione peroxidase.[31] Lipid peroxidation is primarily a cell-membrane event, whereas GSH and its related enzymes are intracellular. Therefore, although an increase in these enzymes would presumably protect intracellular constituents against oxidation that is secondary to lipid peroxidation, direct protection of the cell membrane might not be expected. Accordingly, a lack of threshold for a lipid peroxidation-induced increase in glutathione peroxidase implies that any concentration of ozone produces lung lipid peroxidation. However, such an interpretation must be viewed with caution, for at least two reasons: the assumption of linearity in the enzyme data is open to question, particularly because the findings are not inconsistent with a curvilinear function;[31] and lung lipid peroxidation at low concentrations of ozone (e.g., 0.2 ppm) has still not been documented, and it is possible that the observed enzyme increases, rather then reflecting lipid peroxidation, are in response to other intracellular ozone effects. If the latter is true, then the increase in glutathione peroxidase could be considered

an adaptive response with no pathologic consequences to the lung, although this is open to debate. In this respect, it is of interest that Prinsloo[172] has reported an increase in lung hexose monophosphate shunt enzymes after inhalation of quartz dust.

Further indirect evidence of a role of lipid peroxidation in ozone toxicity has been obtained in studies in which animals deficient in vitamin E were found to be more susceptible to lethal concentrations of ozone and sublethal concentrations led to a more rapid utilization of this antioxidant vitamin.[84,129] Although vitamin E deficiency potentiates the effects of ozone, it is not completely clear whether supranormal concentrations of vitamin E protect against ozone toxicity. Mice given tocopherol supplements were not protected against lethal concentrations of ozone,[156] and the specific activity of lung hydrolases was found to be unrelated to dietary vitamin E concentration.[48] However, other investigators have reported that additional supplementation with vitamin E above usual dietary concentrations lessens the extent of toxicity in animals that inhale ozone.[31,33,149]

Mustafa *et al.*[150] were unable to detect lung lipid peroxides after short-term or subacute ozone exposures (0.8–2.0 ppm), but noted their presence after *in vitro* exposures of mitochondrial preparations. Dowell *et al.*[51] saw no evidence of lipid peroxidation in an alveolar macrophage preparation obtained from rabbits acutely exposed to ozone at up to 10 ppm.

The implications of an ozone-induced breakdown of UFA extend beyond the destruction of these integral components of cellular membranes. Lipid peroxides can produce hydrogen peroxide and oxidize sulfhydryl groups and other amino acid consitituents, and thus result in enzyme inactivation. In addition, lipid peroxidation has been implicated in one of the many theories that attempt to explain the aging process—that based in part on the apparent similarity of "normal" age pigments to the products of the reaction of lipid peroxides with proteins. In particular, proteins cross-linked by the dicarbonyl lipid peroxide breakdown product malonaldehyde have fluorescent spectra similar to that of lipofuchsin age pigment.[47] Bruch and Schlipköter[22] noted lipofuchsin granules in the lungs of mice exposed to ozone at 0.86 ppm for up to 10 months. Inasmuch as acceleration of aging has been reported after chronic ozone exposure,[182] it is conceivable that this is mediated through lipid peroxidation. In addition to free radicals and peroxides, the end products of the oxidative decomposition of UFA include carbonyl compounds that may also have toxic effects. Buell *et al.*[26] reported the presence of carbonyl compounds in the lungs of rabbits exposed to ozone at 1 ppm for 1 h and suggested that these compounds can cross-link collagen or elastin and so lead to altered lung function. Increased cross-linking of collagen has also been

suggested as a mechanism of the deterioration of lung function with age. Recently, Sugihara and Martin[196] reported simulation of lung aging after exposure to formaldehyde, a cross-linking agent.

PROTEINS

As described above, sulfhydryl groups are relatively susceptible to oxidation by ozone, and a decrease in lung-protein sulfhydryl groups has been reported as a consequence of ozone exposure. Free sulfhydryl groups are important constituents of many enzymes, and oxidation results in a loss in activity. In addition, cell-membrane sulfhydryl groups play an important role in the transport of water and ions, and it is conceivable that their oxidation in the lung is a factor in ozone-induced pulmonary edema.

The oxidation of cysteine, as well as other amino acids, was studied by Mudd *et al.*[138] Individual amino acids in aqueous solution were exposed to ozone; the reported order of susceptibility was cysteine, methionine, tryptophan, tyrosine, histidine, cystine, and phenylalanine. Other amino acids were not affected. This order is similar to that for the relative susceptibility of amino acids to radiation and to lipid peroxides. Evaluation of the ozonization products revealed that cysteine was converted to cysteic acid, as well as cystine; methionine to methionine sulfoxide; tryptophan to a variety of products, including kynurenine and *N*-formylkynurenine; tyrosine also to a variety of products, including dihydroxyphenylalanine; histidine to ammonia, proline, and other compounds; and cystine in part to cysteic acid. In some cases, the rate and end products depended on the pH of the solution.

Somewhat different oxidation products were observed by Previero *et al.*,[170,171] who performed the ozonization in anhydrous formic acid, rather than aqueous solution. This raises the possibility that *in vivo* effects on protein may depend on the relative hydrophobicity of the location of the susceptible amino acid, particularly in cell-membrane lipoproteins.

A number of investigators have studied the effect of ozone on the ultraviolet absorption spectra of proteins and amino acids. A decrease in the absorption of 280-nm light in a number of proteins was originally reported by Giese *et al.*[77] to be a consequence of ozone exposure; they suggested that this was due to an interaction of ozone with the ring structures of tyrosine and tryptophan. Exposure of a solution of tryptophan to ozone resulted in a decrease in 280-nm absorption, whereas the extinction coefficient of tyrosine increased. Similar results with tyrosine were reported by Scheel *et al.*,[182] who also noted alterations in the ultraviolet spectra of egg albumen, perhaps representing denaturation by ozone.

More recently, B. Goldstein and McDonagh[88] demonstrated that the native protein fluorescence (280-nm excitation, 330-nm emission) of red-cell membranes exposed *in vitro* to ozone at 1 ppm was a somewhat more sensitive indicator of ozone effect than other characteristics measured in the same system, including oxidation of cell-membrane sulfhydryl groups, loss of acetylcholinesterase activity, and formation of lipid peroxide breakdown products.

Among the specific enzymes whose activity has been reported to be decreased after *in vitro* ozone exposure are papain, glyceraldehyde-3-phosphate dehydrogenase,[127] lysozyme,[104] ribonuclease,[138] and acetylcholinesterase.[80,159] The latter enzyme appears to be particularly susceptible to free-radical and oxidative states. A loss in acetylcholinesterase activity has been reported in the red cells of humans[24] and mice[89] that inhaled ozone. However, there are only minimal amounts of this enzyme in lung tissue, and, although it has been suggested that acetylcholinesterase is important in bronchial tract ciliary activity,[26] there is no direct evidence to support this conjecture.

As described elsewhere in this chapter, alterations in the activity of a number of lung enzymes have been described after acute and chronic ozone exposure. With the possible exceptions of the sulfhydryl-containing enzyme succinic dehydrogenase and the cytochrome P-450 enzyme benzopyrene hydroxylase, it is difficult to determine whether these findings are due to a direct oxidative effect of ozone or are secondary to changes in protein synthesis, concentrations of intermediates, or destruction of cells or organelles.

NUCLEIC ACIDS

Less is known about the interaction of ozone with other macromolecules. Alterations of nucleic acids in solution have been reported by Christensen and Giese,[34] and in *Escherichia coli* by Prat *et al.*[169] in studies with relatively high concentrations of ozone. Both purines and pyrimidines are attacked by ozone. However, the detailed chemistry of the reaction, including the intermediates and end products, has not been fully elucidated. This may be important, in view of the chromosomal effects of ozone, although such effects are most likely due to the interaction of an ozone-induced intermediate with DNA, rather than to ozone itself.

Studies of DNA synthesis in lung after ozone exposure have yielded varied results. Scheel *et al.*[182] exposed rats to ozone at 9.2 ppm for 45 min and reported an initial decrease in lung DNA content, which returned to normal in 6 h. In contrast, lung RNA content was found to increase sharply (as fraction of body weight) an hour after exposure. Cronin and

Giri[41] found no change in DNA content per lung in rats exposed to ozone at 4 ppm for 4 h. Evans *et al.*[56] used a more sensitive autoradiographic procedure and reported a decrease in the fraction of mouse lung cells that synthesized DNA immediately after exposure to ozone at 0.5–3.5 ppm for 6 h. DNA synthesis had returned to normal or above normal within 72 h after exposure. More recently, Werthamer *et al.*[210] studied the uptake of tritiated thymidine, uridine, and leucine in lung homogenates obtained from mice exposed to ozone at 2.5 ppm for 2 h/day for up to 120 consecutive days. Both DNA and RNA synthesis initially declined, but protein synthesis was greatly increased. By 30 days of exposure, DNA synthesis was above normal, but RNA synthesis remained low.

CARBOHYDRATES

Even less is known about the effects of ozone on carbohydrates. Buell *et al.*[26] observed a decrease in the depolymerization of hyaluronic acid after treatment of the lungs of ozone-exposed rabbits (1 ppm for 1 h) with hyaluronidase. B. Goldstein *et al.*[85] reported a loss in membrane neuraminic acid of red cells exposed *in vitro* to high concentrations of ozone. It would be important to study the effects of ozone on respiratory tract mucus, which is rich in carbohydrates, including neuraminic acid. This could include determination of the extent to which ozone is able to penetrate mucus that is unaltered, whether the reaction of ozone with mucus results in the formation of cytotoxic intermediates, and evaluation of the interaction in mucus of ozone with other air pollutants, particularly sulfur dioxide. Of possible pertinence is a study by Falk *et al.*,[62] who observed that ozone produced a loss in the viral hemagglutinating ability of snail mucus.

Subcellular Components

Current approaches to understanding the biochemistry of organ systems depend heavily on fractionation procedures designed to separate various cellular and subcellular components. The lung is a very complex organ; it contains many cell types that presumably function in different ways and may well have different key biochemical processes. Unfortunately, the lung is difficult to homogenize and fractionate, and this, along with the multiplicity of cell types, has greatly retarded understanding of normal lung biochemistry.

Many studies have evaluated the biochemical effects of ozone and other pollutants with whole-lung homogenates. This is a somewhat unsatisfactory approach, inasmuch as a toxic effect at a specific target site may

be obscured by reciprocal adaptive changes in other pulmonary cells or organelles, as well as by an influx of leukocytes and the presence of edema. Of interest in this regard is the observation of an ozone-induced increase in the number of lung Clara cells[22,162] and a shift from Type 1 to Type 2 pneumocytes, which has been hypothesized to be related to antioxidant defense.[42] The biochemical concomitants of these morphologic alterations are unknown and are unlikely to be ascertained by using whole-lung homogenates. For a full understanding of the effects of ozone in the lung, and in particular for a determination of the lowest ozone concentrations that affect specific important indexes of toxicity, more information on normal lung biochemistry is necessary.

In general, the lung has been considered to be the major site of ozone toxicity, if for no other reason than because it is the first organ to come into contact with inhaled ozone. It is therefore of interest that, in a study by Cortesi and Privett,[39] intravenous injection of a fatty-acid ozonide in rats resulted in lung edema and hemorrhage and that on gross examination the lung was the only organ noted to be affected. Similar results were observed after injection of a fatty-acid hydroperoxide.[197] Other systemic agents that are relatively specific to the lung and whose toxicity is perhaps mediated by free-radical reactions include paraquat[114] and 3-methylindole.[28] These findings raise the possibility that there is some unknown biochemical aspect of the normal lung that, in comparison with other organs, makes it relatively susceptible to oxidative free-radical agents like ozone.

MEMBRANES

The possibility that pulmonary membranes are a primary site of ozone toxicity is suggested by a number of lines of evidence, most of which are indirect. These include observations that the membrane is the major site of ozone toxicity in plants[163] and bacteria;[184] morphologic evidence of pulmonary membrane damage after ozone exposure in a number of studies; and *in vitro* experiments with human red cells and artificial lipid membranes.

As discussed above, UFA, which are present primarily in cellular membranes, appear to be particularly susceptible to oxidative degradation by ozone. Various studies of membrane lipid peroxidation have implicated this process in damage to organelles, including mitochondria, microsomes, and lysosomes, as well as to the cell membrane itself. By analogy, it is conceivable that many of the findings in cells and subcellular components described in other sections of this chapter are secondary to ozone-induced lipid peroxidation. However, this remains conjectural.

Furthermore, the effects of ozone on the cell membrane do not appear to be limited to UFA. Protein is also affected, particularly cell-membrane sulfhydryl groups and aromatic amino acids. Whether these protein effects are secondary to cell-membrane lipid peroxidation or arise as a direct result of ozone is not clear.

No studies of the effect of ozone on a lung plasma membrane fraction have been reported. However, a few studies have evaluated the activity in whole-lung homogenates of enzymes that are believed to be associated with membranes. Scheel *et al.*[182] reported a decrease in both 5′-nucleotidase and alkaline phosphatase concentrations (expressed as amount per milligram of tissue) in rats exposed to ozone at 16.9 ppm for 1 h or 6 ppm for 4 h. Werthamer *et al.*[210] exposed mice to ozone at 2.5 ppm for 2 h/day on consecutive days. No effect on the concentration of alkaline phosphatase in lung homogenates was observed after the first day of exposure, and enzyme activity rose after 3 days of exposure. This was followed by a precipitous decrease in alkaline phosphatase activity (expressed as amount per milligram of lung protein) to about 50% of control values, which persisted for 30 consecutive days of exposure. Cronin and Giri[41] reported a decrease in lung adenosine triphosphatase (ATPase) concentration (expressed as fraction of total lung weight) in rats exposed to ozone at 4 ppm for 4 h. However, this appeared to be due to the edema, inasmuch as recalculating the data in relation to total DNA revealed no difference between control and ozone-exposed lung ATPase concentrations.

MICROSOMES

An effect of ozone on lung microsomes has been suggested by morphologic studies that indicated alterations in the endoplasmic reticulum.[11,73,105] Biochemical evidence of an effect on microsomal enzymes was originally obtained in the studies of Palmer *et al.*,[158] who demonstrated that ozone exposure (0.75–10 ppm for 3 h) resulted in a decrease in activity of Syrian hamster lung benzopyrene hydroxylase, a mixed-function oxidase that depends on cytochrome P-450. No changes in hepatic activities of this enzyme were observed, and the results were similar in animals in which high activities of benzopyrene hydroxylase had been induced. The maximal effect was not observed until a few days after the single ozone exposure. Palmer *et al.*[157] also reported a decrease in rabbit tracheobronchial mucosal benzopyrene hydroxylase activity after exposure to similar ozone concentrations.

Recently, B. Goldstein *et al.*[90] observed a decrease in microsomal cytochrome P-450 concentrations in the lungs of rabbits exposed to ozone at

1 ppm for 90 min. Again, the nadir of the cytochrome P-450 was not reached until a few days after ozone exposure. B. Goldstein and Balchum[82] had previously obtained indirect evidence that microsomal cytochrome P-450 played a role in the expression of ozone toxicity. Rats given inducing regimens of phenobarbital, which are reported to increase cytochrome P-450, were more susceptible to lethal concentrations of ozone; but injection of allylisopropylacetamide, which destroys cytochrome P-450, resulted in protection. The finding that mixed-function oxidase inhibitors protect plants against ozone injury[115] may be analogous. Other known free-radical-trapping agents shown to protect against ozone toxicity have often been given in multiple daily doses before acute ozone exposure and may also be active through alteration of microsomal intermediates. However, lung cytochrome P-450 concentrations have not been measured in conjunction with any of these agents.

Liver cytochrome P-450 has been shown to be destroyed by lipid peroxides,[181] and *in vitro* exposure of lung microsomes to ozone is accompanied by a loss in cytochrome P-450 and the formation of malonaldehyde.[90] Evidence of a possible effect of inhaled ozone on hepatic microsomes has been obtained by Gardner *et al.*,[74] who demonstrated that mice exposed to ozone at 1 ppm for 3 h on 2 or 3 successive days developed a prolongation of pentobarbital sleeping time. No effect was observed immediately after exposure on the first day, nor was there any significant difference from the control after 4–7 days of exposure. The latter finding appeared to be due to tolerance, inasmuch as increasing the dose to 5 ppm after 7 days at 1 ppm again resulted in a prolongation of pentobarbital sleeping time. The authors suggested that their findings reflect a decrease in hepatic microsomal metabolism of pentobarbital, perhaps due to an oxidizing circulating intermediate.

MITOCHONDRIA

Numerous ultrastructural studies have noted lung mitochondrial swelling and degenerative changes after ozone exposure.[11,15,16,162,166-168,183,188,189] Mitochondrial swelling occurs *in vitro* in association with lipid peroxidation of the mitochondrial membrane. However, the biochemical concomitants of ozone-induced damage to lung mitochondria have not been extensively studied.

Freebairn[71] noted a decrease in oxygen uptake of plant and bovine liver mitochondria that was reversible by glutathione and ascorbic acid. The activity of some mitochondrial enzymes, including succinic dehydrogenase and cytochrome oxidase, has been found to be susceptible to ozone.

Mustafa and colleagues have performed a series of studies on the effect of ozone on rat lung mitochondrial oxygen consumption. Acute-exposure studies (2 ppm for 8 h and 3 ppm for 4 h) revealed that an initial decrease in succinate-dependent oxygen consumption was followed by a rebound in which increase in the utilization of succinate, as well as other substrates, was noted.[148,150] This increase persisted for at least 3 weeks. No difference in the yield of mitochondria between control and exposed lungs was noted immediately after ozone exposure, but there was a definite increase in the number of mitochondria in the exposed lungs within 48 h of recovery. The authors concluded that increased lung mitochondrial oxygen consumption during recovery from ozone was due to both enzyme activation and mitochondrial proliferation, but primarily the latter. Continuous exposure to ozone at 0.8 ppm for 10 or 20 days resulted in a significant increase in lung-homogenate oxygen consumption. In a further experiment, rats were exposed continuously for 7 days to ozone at 0.2, 0.5, or 0.8 ppm.[150] The observed increase in oxygen consumption was statistically significant at all concentrations of ozone and varied directly with dose.

More recently, Mustafa[149] studied succinate-dependent mitochondrial oxygen consumption in rats fed either a diet containing vitamin E at approximately the normal American dietary intake or one with 6 times as much vitamin E. A statistically significant increase in oxygen consumption was observed in rats that received the diet lower in vitamin E when exposed to ozone at either 0.1 to 0.2 ppm for 7 days. In rats on the diet higher in vitamin E, ozone at 0.2 ppm for 7 days also produced a significant increase, but ozone at 0.1 ppm was without effect. Unfortunately, serum vitamin E concentrations were not obtained.

LYSOSOMES

The effect of ozone on lysosomal enzymes has been studied by a number of investigators. Ozone has been clearly shown to inactivate lysozyme *in vitro*, but the effects of inhaled ozone on the activity of lysozyme appear to depend on the pulmonary fraction under study. Holzman *et al.*[104] reported that exposure of rabbits or mice to ozone resulted in a decrease in the lysozyme activity of bronchopulmonary lavage samples. The effect was linearly related to product of ozone concentration and duration of exposure, although relatively high concentrations of ozone (2.0–5.5 ppm) were used in this acute-exposure experiment (1–4 h). The authors also reported that alveolar cells present in the bronchial lavage of rabbits exposed to ozone at 10 ppm for 3 h had a decrease in the rate of lysosomal

release when incubated *in vitro*. The inference that this is due to *in vivo* oxidation of intracellular lysozyme is questionable, in view of the different cell populations present in the ozone-exposed and control bronchial lavage specimens. Of interest, however, is a reported decrease in the release of lysozyme from a peritoneal polymorphonuclear leukocyte sample obtained from the ozone-exposed rabbits.

In further studies by the same group,[4,108] depression in the activity of lysosomal enzymes—acid phosphatase, β-glucuronidase, and lysozyme—was observed in alveolar macrophages obtained from rabbits exposed to ozone at as low as 0.25 ppm for 3 h. Enzyme concentrations returned toward normal 24 h after exposure. An ozone-induced redistribution of lysosomal enzyme into the cytosol was suggested by a fractionation study that demonstrated a smaller fraction of enzyme activity in the alveolar macrophage lysosomal pellet, compared with the supernatant, with increasing doses of ozone. *In vitro* studies[107] of rabbit alveolar macrophages in tissue culture revealed an ozone-induced decrease in lysosomal acid hydrolase activity, which was prevented when sulfhydryl compounds or serum were added to the tissue-culture medium. A decrease in lysozyme has also been reported in the tears of humans reacting to photochemical smog.[180]

In contradistinction to the decrease in lysosomal enzyme activity observed in alveolar macrophages and bronchial lavage fluid, Dillard *et al.*[48] reported that continuous ozone exposure (0.70–0.79 ppm for 5–7 days) resulted in an increase in the activity of some lysosomal hydrolases in rat whole-lung homogenates and lung fractions, including the soluble supernatant. The tocopherol concentrations of the diet had no effect on the findings.

Similarly, Chow *et al.*[31] observed an increase in the lysozyme activity of a soluble lung fraction and of plasma after continuous exposure of rats to ozone at 0.8 ppm for 8 days. However, no difference in lung or plasma lysozyme activity from control values was present in rats continuously exposed to 0.2 or 0.5 ppm or intermittently exposed (0.2–0.8 ppm, 8 h/day for 7 days). Histochemical evidence of an increase in lung acid phosphatase, a lysosomal enzyme, has also been reported.[29]

The apparently conflicting data on lysozyme may perhaps be explainable by considering three distinct effects of ozone: an irritative response that produces an influx into the lung of leukocytes, including the alveolar macrophage, which contain lysozyme at relatively high concentrations, thereby leading to an increase in whole-lung lysozyme concentration; a disruption of lung lysosomal membranes, resulting in the release of acid hydrolases; and oxidation of lysosomal protein, producing enzyme inacti-

vation. Evidence to support each of these processes is available, and it appears that the net observed effects will depend on the ozone dose and the lung fraction that is evaluated.

As discussed in detail by Dillard *et al.*[48] and by Mittman *et al.*,[135] the possible relationship of lysosomal proteases to chronic lung disease has been inferred from the finding of an increased incidence of emphysema in subjects deficient in serum α_1-antitrypsin factor, an α_1-globulin that can inhibit lysosomal proteases. (No effect of ozone on serum α_1-antitrypsin inhibitor was noted in rabbits chronically exposed to ozone.[160]) Thus, an ozone-induced increase in concentrations of such enzymes in the lung might produce excess proteolysis and result in eventual chronic lung disease. However, the available evidence is inadequate to support the belief that such a process occurs in humans intermittently exposed to ozone. Further studies of this potential hazard would be of value.

CONNECTIVE TISSUE

Despite physiologic evidence that chronic ozone exposure may affect lung elasticity,[10] there is very little information concerning the biochemical effects of ozone on lung collagen. Buell *et al.*[26] obtained a collagen-containing fraction from the lungs of rabbits exposed to ozone at 1 ppm for 1 h. A number of dinitrophenylhydrazine-reacting carbonyl compounds were obtained after digestion with collagenase or elastase. The authors suggested that such carbonyls might result in intramolecular or intermolecular cross-linking of collagen with an attendant decline in lung function. On the basis of present information, these carbonyl compounds are most likely derived from lipid.

In a recent abstract, Hussain *et al.*[109] presented evidence that exposure of rats to ozone at 0.8 ppm for up to a week resulted in an increased rate of collagen synthesis. Such a finding might be relevant to ozone-induced fibrosis. Obviously, further study of the biochemical effects of ozone on lung collagen and elastin are in order, particularly in conjunction with chronic-exposure experiments.

Alveolar Macrophages

Alveolar macrophages represent a major line of pulmonary defense against exogenous material, including infectious agents and particles. Although the origin of these phagocytic cells is still not completely clear, recent information suggests that they are derived from the bone marrow,[23] but are capable of further division and replenishment in the lung.[78] As discussed below, a number of investigators have reported alterations in

alveolar macrophage function and biochemistry after ozone exposure. Before a description of their observations, one potential difficulty in interpreting the effect of ozone inhalation on alveolar macrophages should be mentioned. Inhalation of ozone or other irritants results in an alteration of the number of alveolar macrophages obtained in a bronchial lavage. Presumably, this is due to a combination of cell destruction and an influx of younger alveolar macrophages in response to the irritant stress. It is well known that immature human granulocytes have functional capacities and concentrations of biochemical intermediates different from those of mature polymorphonuclear leukocytes. Unfortunately, there is no readily available morphologic indicator of alveolar macrophage maturity, as there is for granulocytes. Accordingly, observed differences between alveolar machrophages obtained after ozone exposure and those present in normal lungs may simply reflect relative immaturity secondary to pulmonary irritation, rather than a direct effect of ozone on biochemistry or function.

Interest in the effects of ozone on alveolar macrophages has been spurred by the observation that relatively low concentrations of ozone potentiate respiratory infections in animals and perhaps man. Coffin *et al.*[38] observed a decrease in the number of bacteria phagocytized by alveolar macrophages obtained from rabbits exposed to various concentrations of ozone as low as 0.3 ppm. Some suggestion of a lack of threshold is present, but it is not clear whether the difference from the controls at lower ozone concentrations is statistically significant.

These findings in rabbits are similar to those of E. Goldstein *et al.*,[91-94] who have related increased bacterial infectivity in mice to an ozone-induced impairment in the bactericidal capabilities of alveolar macrophages. They used radioactively labeled bacteria and observed a decrease in mouse lung bactericidal activity after a 4-h exposure to ozone at about 0.3–0.4 ppm. With this technique the effects of ozone and nitrogen dioxide are roughly additive,[94] and silicotic mice are no more susceptible to ozone than are control mice.[91]

Dowell *et al.*[51] reported an increase in the osmotic fragility of alveolar macrophage preparations obtained from rabbits acutely exposed to ozone at 10 ppm for 3 h or intermittently exposed to ozone at 2 ppm for 8 h/day for 7 days. Similar intermittent exposure to ozone at 0.5 ppm was without effect. A test for malonaldehyde formation was negative, but this lipid peroxide breakdown product may have been lost during the preparatory procedure.

Additional studies related to the alveolar macrophage include that of Weissbecker *et al.*,[209] who demonstrated that *in vitro* exposure to ozone at as low as 0.06 ppm resulted in alveolar macrophage cell death. How-

ever, Richmond[175] observed only a slight decrease in bacterial phagocytosis during *in vitro* incubation of alveolar macrophages with an initial ozone concentration of 0.8 ppm. Inasmuch as ozone was not administered continuously, the dose to the cells may have been minimal.

Gardner *et al.*[76] noted the ozone-induced loss of an alveolar macrophage protective component in lung lavage fluid after acute ozone exposure (10 ppm). This factor appears to stabilize alveolar macrophages in lung fluid, and its loss would result in a decrease in cell viability. As pointed out by the authors, this finding might indicate that the reported effects of ozone on alveolar macrophages are, at least in part, due indirectly to an action on an alveolar fluid component, rather than on the macrophage itself. The substantial decrease in recoverable alveolar macrophages after ozone inhalation does suggest that ozone has a direct effect on alveolar macrophages, as does the study of Huber *et al.*,[105] who noted nonspecific ultrastructural alterations in rabbit alveolar macrophages after exposure to ozone at 5 ppm for 3 h. However, other investigators have failed to observe morphologic abnormalities in alveolar macrophages after inhalation of ozone.[16,168]

Mechanisms by which ozone might interfere with bactericidal function include an alteration in cell-membrane function that produces a loss in phagocytic ability and is perhaps mediated by lipid peroxides[113] and a decrease in the ability of alveolar macrophages to kill phagocytized bacteria. Leukocytes in general appear to have a multiplicity of mechanisms for destroying ingested bacteria.

Ozone might interfere with the intracellular bactericidal capabilities of alveolar macrophages by inactivating lysosomal hydrolases, or perhaps through the destruction of heme-containing enzymes that are apparently involved in producing superoxide anion radical. Further evaluation of the process by which relatively low concentrations of ozone potentiate bacterial infection would be of value.

Extrapulmonary Effects

An appreciable body of evidence has accumulated to indicate that ozone has extrapulmonary effects. Although some of the reported effects may be secondary to the reaction of ozone with intrapulmonary neural receptors or to release of humoral substances from the lung, other findings appear to be more directly related to an oxidizing effect of ozone. The biochemical basis for the latter is unclear, particularly because the reactivity of ozone and its short-lived intermediates would make it unlikely for them to penetrate the pulmonary parenchyma. Earlier studies on the subject of extrapulmonary effects have been reviewed by Stokinger.[192]

Industrial and epidemiologic evidence associating ozone exposure with headache and drowsiness are relatively nonspecific and do not clearly indicate extrapulmonary effects. Lagerwerff[120] noted changes in a number of indexes of vision at ozone concentrations of 0.2–0.5 ppm during short-term exposures. In addition, Brinkman and Lamberts[20] reported a 50% decrease in the rate of desaturation of oxyhemoglobin in the skin capillaries of a tied-off finger during inhalation of ozone at 1 ppm (the experimental procedure was not given in detail). Neither of these studies, which have been in the literature for some time, has been replicated.

Recently, Buckley *et al.*[24] have described statistically significant changes in a number of constituents and properties of red cells and serum in human volunteers experimentally exposed to ozone at 0.5 ppm for 165 min. Their findings included an increase in red-cell osmotic fragility and a decrease in red-cell acetylcholinesterase and glutathione, all of which were consistent with previous *in vitro* or animal studies. They also observed an increase in serum thiobarbituric acid reactants consistent with lipid peroxidation and an increase in serum vitamin E that they attributed to its mobilization. The thiobarbituric acid assay is predominantly a measure of malonaldehyde. Increased serum malonaldehyde concentrations have usually been reported in man only in association with significant red-cell lipid peroxidation, such as occurs in hemolytic vitamin E deficiency, presumably because malonaldehyde is rapidly metabolized and cleared from the serum. Also consistent with acute hemolysis is the observation by Buckley *et al.* in the same subjects of increases in serum lactic dehydrogenase and in red-cell G-6-PD. In contrast with those in the lung, G-6-PD increases in the anucleate red cell are thought to represent solely a decrease in mean red-cell age consistent with reticulocytosis, which (to explain the extent of the G-6-PD increase in this study) would be consistent with a relatively significant acute hemolysis. Data necessary to interpret this possibility were not fully presented. A more recent report from this group[96] evaluating dose-response data for human 2-h exposures to ozone at 0.25, 0.37, and 0.50 ppm showed a no-effect concentration of 0.25 ppm for mean change in red-cell acetylcholinesterase activity and osmotic fragility and a linear dose-related effect on these two characteristics at the higher ozone concentrations.

A number of animal studies have revealed extrapulmonary effects. Again, there is some question as to which of these may represent an effect of ozone, or a direct ozone-induced intermediate, rather than a more indirect response to pulmonary toxicity, perhaps mediated by neurohumoral factors. Thus, for instance, the observations of altered hepatic nucleic acid concentrations,[182] shifts in the content of metals in the liver,[50] alterations in urinary pH,[99] increases in liver weight and alkaline phosphatase

activities,[142] and variations in the circulating white-cell count[17] in ozone-exposed animals conceivably represent nonspecific responses to lung injury.

Of interest is the experimental approach whereby ozone is delivered solely to one lung. The observation of pulmonary effects in the unexposed lung[68] indicates that there are extrapulmonary effects of ozone at edematogenic concentrations. However, only the exposed lung appears to develop tolerance to later ozone exposure and to exhibit impairment of bacterial defense mechanisms.[2,3,69]

Evidence of a direct oxidizing effect on the blood of mice and rats inhaling ozone was obtained in a study in which an increase in an *in vivo* red-cell catalase-reacting compound, presumably hydrogen peroxide,[79] was noted. Although this was observed only at ozone concentrations of 5 ppm or greater, the indirect assay for red-cell hydrogen peroxide used in this study is relatively insensitive and would not detect smaller degrees of ozone-induced oxidant stress. A significant decrease in the activity of red-cell acetylcholinesterase, a membrane constituent known to be sensitive to lipid peroxides, has been reported in mice exposed for 4 h to ozone at 8 ppm.[89] However, only a slight decrease in red-cell acetylcholinesterase was noted in rabbits chronically exposed to ozone at 0.4 ppm.[111] An earlier study by Brinkman *et al.*[21] noted that 30–60 min of inhalation of ozone at 0.2 ppm potentiated the sphering of animal and human red cells irradiated *in vitro*. The contention that this intriguing effect is related to an increase in aging by ozone is open to question. Ozone effects in blood also include chromosal aberrations in the circulating lymphocytes of hamsters,[214,215] an increase in plasma lysozyme,[31] higher serum concentrations of trypsin protein esterase,[161] and Heinz bodies in circulating red cells.[130,131] Earlier studies by Mittler *et al.*[134] failed to reveal any change in the hemoglobin or hematocrit of young rats chronically exposed to ozone at 2.4 ppm.

Biochemical changes in animal central nervous systems have been reported by Skillen *et al.*,[186] who noted a decrease in brain 5-hydroxytryptamine (serotonin) in rats exposed to ozone at 6 ppm for 4 h, and by Trams *et al.*,[202] who observed decreases in catecholamines and catechol-O-methyltransferase in dogs chronically exposed to ozone at 1, 2, or 3 ppm. Electroencephalographic (EEG) measurements in the same dogs were recently presented by Johnson *et al.*,[112] who noted alterations in EEG patterns at 9 months of ozone exposure, but not after 18 months of exposure. Previously, Xintaras *et al.*[212] had observed alterations in the visual evoked electric response in rats acutely exposed to 0.5–1.0 ppm. As pointed out by Johnson *et al.*,[112] it is not clear whether these findings indicate a direct neurotoxic action of ozone or are secondary to damage in other organs.

Other reported systemic effects of ozone include histologic changes in the parathyroid glands of rats exposed to ozone at 0.75 ppm for 4–8 h,[8] variations in rat thyroid function that depend on the concentration and duration of ozone exposure,[60] alterations in myocardial histology in mice exposed to 0.2 ppm 5 h/day for 3 weeks,[21] EEG changes in dogs chronically exposed to irradiated auto exhaust (containing oxidants at 0.4–0.8 ppm),[14] inhibition of gastric motility in rats exposed to ozone at 0.5 ppm for 2 h,[178] and a decrease in the release of lysozyme from peritoneal granulocytes in ozone-exposed rabbits.[104]

An interesting report by P'an and Jegier[161] noted an increase in serum trypsin protein esterase in association with pulmonary vascular lesions in rabbits chronically exposed to ozone at 0.4 ppm. This serum α_2-macroglobulin, which is synthesized in the liver, has been reported to be increased in human vascular disorders, but its physiologic significance is unknown.

In summary: the current evidence appears to support the contention that ozone or an oxidizing derivative is able to penetrate the alveolar basement membrane into the pulmonary circulation. Recent evidence, which requires replication, suggests that red-cell changes consistent with an oxidizing effect can be detected in the blood of humans exposed to ozone at ambient concentrations. There is also evidence that ozone affects other extrapulmonary organ systems, but it is not clear whether this is due to oxidizing intermediates—such as ozonides, peroxides, and the resulting carbonyl compounds carried in the circulation—or whether it is secondary to reflex arcs or to humoral processes initiated in the lung.

MUTAGENIC AND CARCINOGENIC ACTIONS

The supposition that ozone is mutagenic or carcinogenic in man is based primarily on information on the biochemical mechanism of ozone toxicity and to a lesser extent on *in vitro* and animal studies. The biochemical evidence is for the most part indirect and depends on an analogy between the free-radical nature of ozone toxicity and of radiation and other carcinogenic agents.

The production of chromosomal abnormalities by ozone was first observed in plants by Fetner,[63] who noted abnormal anaphases in the root tips of *Vicia fava* seeds exposed to ozone. Bacterial mutagenesis by ozone has been reported by a number of investigators.[43,205] Hamelin and Chung[97,98] noted an increased mutation rate in *Escherichia coli* exposed to ozone at as low as 0.05 ppm for 5 min and observed that the mutants were sensitive to ozone and X irradiation. They suggested that ozone might interact in DNA repair processes. Other studies indicating ozone-induced

chromosomal effects include the observation of mitotic inhibition in grasshopper neuroblasts[64] and abnormalities in the eggs of oysters treated with ozonized seawater.[122]

Fetner[65] has also demonstrated chromatid breaks in a human tissue-culture cell line exposed to ozone at 8 ppm for 5 min. Other tissue-culture studies include that of Sachsenmaier *et al.*,[179] who noted tetraploidy and other chromosomal abnormalities in embryonic chick fibroblasts exposed to ozone and a decrease in transplantability of mouse ascites tumor cells. In addition, Pace *et al.*[155] demonstrated an interference by ozone with mitotic activity in two tissue-culture cell lines. More recently, Booher *et al.*[19] reported that lung cells exposed in culture to ozone concentrations as low as 0.3 ppm demonstrated an inhibition in growth that was proportional to the ozone concentration.

Of particular interest are the studies of Zelac *et al.*,[214,215] in which Chinese hamsters exposed to ozone at 0.2 ppm for 5 h had an increased number of chromosomal breaks in their circulating lymphocytes. Blood samples for study were obtained immediately after exposure and 6 and 15.5 days later. The highest break frequency was observed after the longest delay. The authors compared the effects of X irradiation and ozone singly and combined, in their system. The combined effects were less than additive; this suggested some protective mechanism, perhaps analogous to that observed by Hattori *et al.*[100] When the authors extrapolated their data to acceptable industrial-hygiene exposures to ozone and radiation, ozone was found to be much more likely than X irradiation to produce chromosomal breaks in such exposures.

More recently, Merz *et al.*[132] have studied the circulating lymphocytes of humans experimentally exposed to ozone at 0.5 ppm for 6–10 h. A statistically significant increase in the number of minor chromosomal abnormalities (not breaks) was observed; it reached a peak about 2 weeks after exposure and later returned to normal. This delay in the development of chromosomal abnormalities observed after ozone exposure in both hamsters and humans differs from that observed in human radiation studies, in which aberrations tend to remain roughly constant over 3–4 weeks. This raises the possibility that the ozone-induced abnormality is related to a postreplication repair process.

Carcinogenic or mutagenic effects of ozone have also been suggested by a number of other animal studies. Stokinger[190] noted that ozone exposure resulted in an increased incidence of pulmonary adenomas in a strain of mice prone to develop these benign neoplasms. However, the experimental findings were not reported in detail. A combination of ozone and gasoline has also been shown to cause an increased incidence of lung tumor in two strains of mice,[117,118] only one of which is prone to develop pulmonary adenomas.

A mutagenic effect of ozone on germ cells was suggested by the study of Brinkman *et al.*,[21] in which female mice exposed to ozone at 0.1-0.2 ppm, 7 h/day, 5 days/week, for 3 weeks before birth, demonstrated a fourfold increase in neonatal mortality. A further report of this study noted increased blepharophimosis and jaw anomalies and a decrease in litter size.[204] An artificial oxidant smog mixture derived from irradiated auto exhaust has also been reported to cause an increase in neonatal mortality in mice.[106,121]

The toxicologic evidence indicating a possible role of ozone in human cancer may be summarized as follows:

- The biochemical mechanism of ozone toxicity appears to have many similarities with those of other agents, particularly ionizing radiation, that are known human carcinogens.
- *In vitro* studies with tissue-culture cell lines and bacteria have demonstrated ozone-induced chromosomal effects.
- Some animal studies with ozone or synthetic photochemical smog have shown chromosomal defects in circulating lymphocytes, a more rapid appearance of benign pulmonary tumors, and an increase in neonatal mortality consistent with a mutagenic effect.
- One study of humans experimentally exposed to ozone showed transient development of minor chromosomal abnormalities in circulating lymphocytes.

The toxicologic studies must be considered in the perspective of an absence of epidemiologic evidence that links photochemical air pollution with human cancer. Despite the presence of significant concentrations of ozone in southern California for three decades, no associated increase in cancer has been observed. However, the relatively recent arrival and the peripatetic nature of Los Angelenos might obscure, for the present, a carcinogenic or mutagenic effect of ozone. Furthermore, it is necessary to consider the possible toxicologic interaction between ozone and other airborne carcinogens. The reported ozone-induced decrease in lung benzopyrene hydroxylase and cytochrome P-450 concentrations could be interpreted as protecting against aromatic hydrocarbon carcinogenesis in which *in vivo* activation appears necessary. Alternatively, it could be hypothesized that ozone would directly activate aromatic hydrocarbon carcinogens (e.g., by conversion to epoxides or other free-radical intermediates) and therefore promote the presumed carcinogenic process. This speculative discussion is presented solely to illustrate the potential complexities of the problem of ozone-induced carcinogenesis. Obviously, far more information is needed on the biochemical action of ozone and the interaction of ozone with other potential carcinogens. This would

allow more confident assessment of the possible role of ozone in human carcinogenesis until definitive epidemiologic information is available.

EFFECTS ON REPRODUCTION

As noted previously, exposure of mice to simulated photochemical smog produced by irradiating diluted auto-exhaust mixtures resulted in reduced fertility and fecundity and decreased infant survival.[106] Lewis *et al.*[121] pursued this observation in greater depth and showed that the nonpregnancy rate was significantly ($p < 0.02$) greater (20 of 153) in female mice that were mated with male mice that had been exposed for 46 days to irradiated auto exhaust than in females that were mated with males that were in clean-air chambers (nine of 159). No differences in the groups with respect to the presence of copulatory plugs and absence of implantation scars in the nonpregnant females supported the authors' conclusion that the differences in pregnancy rates were due to reduced fertility. In pregnant females that had been mated with irradiated-exhaust males, there were fewer uterine implantation scars and fewer pups per litter ($p < 0.05$) than in females mated with clean-air males. In addition, offspring viability was reduced in litters that were kept in irradiated-exhaust chambers. The concentration of oxidant (ozone) varied during each day of exposure, with peaks as high as 1.0 ppm. Daily nitrogen dioxide and carbon monoxide concentration peaks were about 1.5 ppm and 100 ppm, respectively. The results of these experiments suggested a possible mutagenic effect in male germ cells resulting from exposure to the oxidant mixture.

In an earlier study of a simpler oxidant-smog mixture made by reacting ozone with gasoline vapors (oxidant concentration, 1.25 ppm in the mixture by neutral potassium iodide method), Kotin and Thomas[119] exposed male and female mice to the synthetic oxidant mixture, to Los Angeles air, and to washed air. Significant differences from controls in reproduction effects were noted only in mating pairs in the synthetic smog mixture. These consisted of reduced conception rates, litter size, and survival of newborn (that were held in the exposure chambers until weaning). These authors also alluded to possible mutagenic effects associated with organic peroxides that would be expected in the synthetic mixture; however, they felt that their data on fecundity reflected an effect primarily on the females.

Brinkman *et al.*[21] and Veninga[204] exposed mated pairs of their laboratory's inbred gray mice and C57 black mice to ozone at 0.1 or 0.2 ppm, 7 h/day, 5 days/week, for 3 weeks. There was little or no effect on litter

size, compared with air-exposed controls; however, there was a greater incidence of neonatal mortality during the first 3 weeks of life in the litters of ozone-exposed parents. Offspring (C57 blacks exposed, in addition, for 3 weeks postnatally) from the 0.2-ppm-ozone groups had increased incidence of blepharophimosis and increased incidence of jaw abnormalities. The authors concluded that this was further evidence of a similar action of ozone and ionizing radiation (which produced the same effects at a dosage of 20 R/day).

These studies, although few, suggest that exposure to photochemical oxidants can influence fertility and fecundity in animals and that the general health of newborn animals is much more likely to be impaired by exposure to oxidants than that of their parents. Whether the changes observed in reproduction variables can be related to mutagenic actions of ozone, discussed earlier, remains to be determined. In any event, it seems logical that effects of low concentrations of ozone and other photochemical oxidants on reproduction must be indirect and may be mediated by endocrine or ozone–biologic reaction products.

CENTRAL NERVOUS SYSTEM AND BEHAVIORAL EFFECTS

It is somewhat surprising that possible central nervous system (CNS) or behavioral responses to ozone exposure have not been subjected to greater investigation, in view of the reports of headache and drowsiness in humans exposed to ozone and the apparent CNS depression that occurs as an early sign of ozone interaction in animals.[191,192] Xintaras *et al.*,[212] in an application of the evoked-response technique, found a depression in the specific visual cortex and in the superior colliculus of rats exposed to ozone at 0.5–1.0 ppm for 1 h. However, Eglite[53] found no change in rat behavior or chronaxial muscle ratios after 93 days of exposure to ozone at 0.6 ppm. Lagerwerff[120] demonstrated in human volunteers that repeated exposure to ozone at concentrations of 0.02–0.05 ppm resulted in psychophysiologic or sensoriphysiologic changes, including decreased visual acuity, increased peripheral vision, altered extraocular muscle balance, lethargy, and difficulty in concentrating.

As mentioned previously, decreased voluntary running activity of mice was one of the most sensitive indexes of an effect of ambient photochemical oxidant air pollution[55] or simulated oxidant smog that was produced in the laboratory by irradiating diluted auto-exhaust gases.[106,141] Whether this effect of the mixtures was due only to ozone or to some other oxidant is not certain. It is plausible to conclude that ozone was the

effective agent, in view of the fact that Murphy *et al.*[146] showed that ozone at as low as 0.20 ppm (during a single 6-h exposure) reduced voluntary running activity of mice by 50%. The 6-h exposure used in those studies coincided with the maximal activity period of the animals (i.e., darkness, in adapted mice), so it seems that the effect of ozone overcame strong natural motivation for running activity. Furthermore, Konigsberg and Bachman[116] noted a concentration-related reduction in the gross motor activity of rats exposed to ozone at 0.1–1.0 ppm. As reported for oxidant mixtures,[106] mice exposed continuously to ozone at 0.3 ppm adapted and returned to normal activity in about a week.[146] However, the same mice were still susceptible to the activity-reducing effect of higher concentrations (0.6–0.8 ppm), and adaptation to control activity did not occur for at least 7 days of continuous exposure.[146] Fletcher and Tappel[66] reported that the voluntary running activity of rats was reduced by 84% during continuous exposure to ozone at 1 ppm for a week. Boche and Quilligan[18] also demonstrated that the oxidant mixture produced by ozonizing gasoline vapors (ozone content, 0.4 ppm or greater) caused a decrease in running activity in mice.

These studies, although demonstrating that reduction of voluntary running activity in mice is a very sensitive biologic indicator of oxidant and ozone, did not reveal the mechanism of the effect. It is possible that this effect represents only a precautionary reaction of the organism to avoid moving about when its senses detect (by odor or irritation) a strange and unpleasant stimulus. This would be consistent with the observation that mice avoided a cage ventilated with ozone at 0.6–1.1 ppm, preferring a clean-air cage.[164] Reduction of voluntary running activity might also be secondary to lung irritation or inflammation; however, the concentrations for activity reduction are below those generally associated with edema or other signs of local injury in the lung. Finally, one could evoke a systemic mechanism of action as a direct effect of circulating ozone (which seems unlikely) or of a CNS-depressant oxidant-biologic reaction product formed locally in the lung. Further studies would be required to determine whether these or other mechanisms are involved.

SUMMARY OF DOSE-RESPONSE RELATIONSHIPS

The acute lethal action of ozone is due to its capacity to produce pulmonary edema. The LC_{50} for rats and mice exposed for a single 4-h period is approximately 6 ppm, and cats, rabbits, guinea pigs, and dogs (in that order) are decreasingly susceptible to the lethal action.[190] Nu-

merous reviews have considered the toxicity of ozone at lethal and lower concentrations.[110,140,154,190-193,203]

In the foregoing discussion and in Table 8-2, attention is on studies in laboratory animals that have been exposed to ozone concentrations of about 1 ppm or less, because results of studies conducted with such concentrations are thought to be more directly relevant to ambient oxidant air pollution, which is the source of exposure for large human populations. In Table 8-2, no attempt has been made to list all studies; more comprehensive summaries can be found in some of the monographs cited. Instead, the table cites studies thought to be most useful for evaluating the health implications of exposure to low concentrations of ozone; and the concentrations listed are the lowest at which the described effects have been observed. Unfortunately, many studies have not included a sufficient range of experimental concentrations to permit construction of reliable dose-response curves or to determine what, if any, would be a "no-observed-effect" concentration for the experimental conditions used. Although it is understandable that research scientists find little stimulation in conducting exposure experiments at concentrations that fail to produce changes in the biologic system in which they are interested, the conduct (and reporting) of such experiments is extremely important for a pragmatic evaluation of the implications of positive findings for human health.

The table illustrates the wide variety of biologic effects produced in laboratory animals exposed to relatively low concentrations of ozone. Obviously, some effects have more serious health implications than others.

DISCUSSION

During the last decade, toxicologic research on the effects of ozone in laboratory animals has demonstrated that exposure for a few hours to airborne concentrations of less than 1 ppm produces numerous changes in cell and organ structure and function. The limiting concentrations required to produce these changes appear to differ somewhat among different species of laboratory animals and with the particular type of effect under investigation. However, there seems to be little doubt that several functional and morphologic indexes of response to ozone are altered with exposures to ozone concentrations of about 0.2-0.5 ppm. Many of the functional and morphologic changes produced in experimental animals exposed to ozone as a single contaminant gas have also been demonstrated to occur with exposure to complex mixtures of laboratory-produced or ambient photochemical oxidant air pollution.

TABLE 8-2 Summary of Effects of Exposure of Laboratory Animals to Ozone at Low Concentrations*

Ozone Concentration, ppm	Duration of Exposure	Observed Effects	Animal	Reference
0.08	3 h	Increased mortality from pulmonary infection with *Streptococcus*	Mouse	35
0.1	7 h/day, 5 days/week, 3 weeks	Increased incidence of neonatal mortality in litters of exposed parents (related effects and reduced fertility noted in synthetic oxidant smog)	Mouse	21, 106, 119, 121, 204
	7 days	Increased succinate-dependent lung mitochondrial oxygen consumption in rats on diet relatively low in vitamin E; no significant effect in vitamin E-replete rats at this concentration of ozone	Rat	149
0.2	3 h	Degenerative changes in Type I alveolar cells; later replaced by Type II cells	Rat	188
	Continuous for 28–32 days	16% increase in lung volumes; overdistention at high lung volumes, suggesting some change in elasticity; no change in respiratory frequency, tail length, or external appearance	Young rat	10
	6 h or continuous for 7 days	Decreased voluntary runway activity during exposure; no-effect concentration not reported; reduced gross motor activity	Mouse	146
	7 h/day, 5 days/week, 3 weeks	Increased incidence of blepharophimosis and jaw abnormalities in neonates	Mouse	204
	7 days	Increased succinate-dependent lung mitochondrial oxygen consumption	Rat	151
	Continuous for 8 days	Dose-related increased activity of lung glutathione peroxidase and glutathione reductase	Rat	31

		(glucose-6-phosphate dehydrogenase activity significantly increased at 0.5 ppm)		
	5 h	Lymphocyte chromosomal breaks	Hamster	214, 215
0.2–0.25	0.5–2 h	Increased red-cell spherocytosis after *in vitro* radiation	Mouse, rat, rabbit, man	21
0.25	4–6 h	Morphologic changes in medium-sized airways	Cat	16
0.25–0.5	6 h	Threshold for lung edema formation with ^{131}I-albumin test	Rat	5
	3 h	Decreased lysozyme, acid phosphatase, and β-glucuronidase activity in alveolar macrophages (appears to be linearly related to dose up to 1 ppm)	Rabbit	2, 108
0.26–0.5	4.6 h	Increased lung flow resistance in 2 animals at 0.26 ppm; effect in all at 0.5 ppm	Cat	206
0.34	2 h	30% increase in frequency of breathing; 20% decrease in tidal volume	Guinea pig	146
0.37–0.5	2 h	Decreased red-cell acetylcholinesterase and increased osmotic fragility (no effect at 0.25 ppm)	Man	96
0.4	6 h/day, 5 days/week, 10 months	Increased serum trypsin protein esterase	Rabbit	161
0.4–0.7	4 h	Conjugated diene bonds, suggesting lung lipid peroxidation	Mouse	87
0.5	6–10 h	Minor chromosomal abnormalities	Man	132
	3 h	Inhibition of intracellular hydrolytic enzymes of alveolar macrophages; increased fraction of polymorphonuclear leukocytes	Rabbit	3
	165 min	Alterations in blood, including red-cell membrane and enzyme changes and increased serum vitamin E and lipid peroxides	Man	24
	6 h	Decreased lung DNA synthesis	Mouse	56
0.5–1	1 h	Decreased electric response of specific areas of brain with evoked-response technique	Rat	212

TABLE 8-2 (Cont.)

Ozone Concentration, ppm	Duration of Exposure	Observed Effects	Animal	Reference
0.54–0.88	Continuous for up to 3 weeks	Morphologic changes in distal and respiratory bronchioles, alveolar ducts, and associated alveoli	Young rat	72
0.6	Not applic.	Avoidance of cage ventilated with ozone	Mouse	164
	93 days	No change in behavior or chronaxial muscle ratios	Rat	53
0.68	4 h	Decreased rate of bacterial killing in lungs *in vivo*	Mouse	92
	2h	No significant increase in respiratory flow resistance	Guinea pig	146
0.7–0.8	Continuous for 7 days	Increased acid phosphatase in specific lung areas determined histochemically	Rat	29
	Continuous for 5–7 days	Increased activity of lysosomal hydrolases in whole-lung homogenates	Rat	48
0.75	3 h	Decreased benzopyrene hydroxylase in lung and tracheobronchial mucosa	Hamster	158
	4–8 h	Histologic changes in parathyroid glands	Rat	8
0.8	Continuous for 7 days	Histochemically determined alteration in several lung enzyme activities	Rat	30
	Continuous for 8 days	Increased activity of lung and plasma lysozyme (no effect at 0.2 and 0.5 ppm)	Rat	31
	7 days	Increased activity of lung pentose shunt and glycolytic enzymes; decreased lactic dehydrogenase	Rat	32
0.84	4 h/day, 5 days/week, 2 weeks	Increased susceptibility to respiratory infection with *Klebsiella pneumoniae*	Mouse, hamster	133

0.85	4 h	Heinz bodies in circulating red cells; further exposure led to decrease in Heinz body formation	Mouse	130, 131
0.9	Continuous	Lungs 38% heavier than those of normals; 50% dead in 3 weeks	Young rat	72
1	1 h	Chemical changes in ground substance and lung protein	Rabbit	26
	4 h	Engorged blood vessels and excess leukocytes in lung capillaries	Mouse	182
	90 min	Decreased lung cytochrome P-450	Rabbit	90
	1 h	Formation of carbonyl compounds and alterations of hyaluronic acid in lung	Rabbit	26
	8–24 h/day for 18 months	Alteration in catechol-O-methyltransferase and monoamine oxidase of brain tissue	Dog	202
	3 h/day for 2–3 successive days	Prolongation of phenobarbital sleeping time (no effect after 1 or 4–7 days)	Mouse	74
	Continuous for up to 18 months	Bronchitis; bronchiolitis; emphysematous and fibrotic changes; acceleration of lung-tumor development	Mouse	190
	Continuous for 1 week	Decreased voluntary running activity	Rat	66
1–3	Continuous for 18 months	Thickening of terminal and respiratory bronchioles; barely noticeable at 1 ppm; at 3 ppm, formation of peribronchiolar collars with resulting narrowing of small airways	Dog	73
1.08	2 h	47% increase in respiratory flow resistance	Guinea pig	146
1.3	3 h	Increased susceptibility to *Klebsiella pneumoniae*	Mouse, hamster	133

*Concentrations listed are lowest for which observed effects have been reported.

Most research has focused on the effects of ozone on lung function, morphology, and biochemistry. Altered lung function, observed in studies of the mechanical behavior of lungs of several species, has been noted as the result of a few hours of exposure to ozone at 0.2–0.3 ppm. Improved techniques for detecting minimal pulmonary edema and for examining the microstructure of cells of the respiratory tract indicate that structural alterations also occur with short exposures to this range of concentrations. Furthermore, altered activities of various lung enzymes or enzyme systems, particularly those involved in defending against oxidant stress, have been noted by several investigators to occur at concentrations of 0.2 ppm. Although all these effects appear to be accentuated by higher concentrations of ozone, indicating a dose-response relationship, most investigations have not determined (or have failed to report) whether lower concentrations fail to produce such changes. In spite of the fact that biochemical, functional, and morphologic effects have all been detected at about the same low exposure concentrations, there has been little systematic investigation to determine whether these effects are causally related.

Some effects noted in these experiments might be considered adaptive, rather than injurious. For example, the increased activities of lung glutathione peroxidase and glutathione reductase that have been demonstrated after exposure to 0.2 ppm may represent an adaptation or compensation to an injurious effect, such as lipid peroxidation, which, although detected only at exposures to 0.4 ppm and above, may go undetected at lower exposure concentrations because of the insensitivity of current assay methods. In spite of these unknowns, it is noteworthy that, in the one case in which it is possible to make direct comparisons of the effects of ozone in laboratory animals and in humans in controlled experiments—i.e., lung-function studies—the minimally effective concentrations are nearly identical, around 0.3 ppm. It is tempting, therefore, to speculate that humans might also sustain the biochemical and morphologic changes observed in laboratory animals exposed to this concentration; but such a conclusion must await mechanistic information concerning the relationships between the different types of effects or independent demonstration of these effects in man.

Recent studies involving repeated or prolonged exposures of laboratory animals to ozone suggest that changes that are indicative of chronic lung diseases (such as decreased elasticity of the lungs, hypertrophy of bronchiolar epithelium, and deposition of connective tissue) also require concentrations of 0.2–0.5 ppm. At slightly higher concentrations, fibrotic changes in the lungs have been observed histologically; this is consistent with a reported increased rate of collagen synthesis in the lung. It appears,

therefore, that the changes observed with short-duration exposures, although generally reversible on cessation of exposure, are as sensitive in evaluating the injury potential of exposure to ozone as are long-term exposure studies. There have been, however, very few truly chronic exposure studies (i.e., covering a major part of the lifetime of the species) on which to base conclusions.

Another subject of active investigation concerning the effects of ozone is related to the mechanisms of defense against inhaled microorganisms. It has been shown that exposure of a few hours results in a marked increase in the susceptibility of animals to controlled doses of infectious organisms introduced into the lung. In fact, this criterion of an effect is the most sensitive of any that have been reported, with significantly increased susceptibility of mice to one microorganism occurring after exposure to an ozone concentration as low as 0.08 ppm. Other reports referring to different microorganisms or different species suggested that somewhat higher concentrations are required, but clearly this effect is noted at less than 1.0 ppm. Research in the mechanism of ozone's effectiveness in decreasing resistance to infectious agents suggests an action on the numbers and viability of lung macrophages. Aside from the obvious practical implication of these findings for carefully planned epidemiologic studies on the incidence of lung infection in human populations exposed to oxidant air pollution, they have stimulated more basic research in the physiology and biochemistry of lung macrophages that may have implications beyond the question of oxidant toxicity.

Earlier toxicity studies had suggested that the hazard from repeated ozone exposure might be reduced, inasmuch as animals became tolerant to the acute pulmonary edematogenic action of ozone. However, more recent studies, which demonstrated that tolerance to the ordinarily ozone-induced increase in susceptibility to infectious microorganisms or to the effects on respiratory mechanics does not develop, suggest that the tolerance phenomenon would have little protective value with respect to repeated exposure to ambient oxidant smog.

There are several reports of noteworthy extrapulmonary effects in laboratory animals with concentrations of about 0.2 ppm. These include reduced voluntary activity, chromosomal aberrations in circulating lymphocytes of hamsters, increased neonatal mortality, and greater incidence of jaw abnormalities in offspring of ozone-exposed mice. The mechanisms of these reported effects and whether they are due to direct actions of absorbed ozone, some secondary reaction product, or secondary responses to the stress of local actions in the lung are largely unknown. However, reported analogous effects in humans exposed to ozone, such as changes in visual acuity and headache (possibly related to the reduced activity in

animals) and minor chromosomal abnormalities in circulating lymphocytes, require that these extrapulmonary effects be considered in the evaluation of the hazard of ozone exposure. Furthermore, the chromosomal aberrations in hamsters and the reports of mutagenic activity of ozone in a variety of microorganisms, tissue cultures, plants, and insects raise the questions of the possibility of a genetic or carcinogenic hazard of this gas. Indeed, earlier studies at higher concentrations of ozone or mixtures of ozone with hydrocarbons have suggested a tumorigenic action in the lungs of mice. Although present evidence does not justify a definitive conclusion that exposure to ozone implies a mutagenic or carcinogenic hazard, prudence requires that this possibility not be dismissed until it has been definitively tested experimentally and epidemiologically.

The possibility of interactions of ozone with other environmental stresses has received relatively little recent attention. Two exceptions are noteworthy, however: reports of increased susceptibility to some of the actions of ozone in animals that are deficient in vitamin E or the converse (protection conferred by administration of vitamin E) and observations that exposure to ozone at less than 1.0 ppm reduces activity of the cytochrome P-450 mixed-function oxidase activity of lungs. Although the implications of these observations are not yet clear, they remind us that nutritional variations or exposure to foreign chemicals that are metabolized by lung mixed-function oxidases may provide bases for unanticipated qualitative or quantitative effects associated with oxidant exposures.

On the basis of the foregoing discussion, it appears that, if traditional criteria for hazard evaluation are applied to the toxicologic data on experimental animals, there is little room for complacency regarding current ambient concentrations of ozone. Functional, biochemical, and structural effects in both pulmonary and extrapulmonary systems have been reported by numerous investigators at or near concentrations that are at least occasionally achieved in some polluted urban centers. Unfortunately, there are no adequate methods for extrapolating data to obtain reliable quantitative estimates of population risk at environmental concentrations near the standard, and there is no assurance that the risk is zero.

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9

Controlled Studies on Humans

Most standards for safe concentrations of oxidants and other photochemical pollutants are based on information about their effect on human health.[67] Such information is acquired through the interplay of different fields of scientific endeavor, including epidemiologic investigation of pollutant-exposed human groups and controlled experimental study of animals or volunteer human subjects. Epidemiologic studies of humans can be used to obtain dose-response (see discussion in Chapter 7) information on polluted ambient air, but they are limited by cost factors, dose-range availability, the presence of interfering pollutant substances, and the problems caused by the presence of many uncontrolled variables. Most controlled experimental investigations have been conducted with animals. However, there is no rigorous way of extrapolating animal dose-response data to humans. There is a need, then, for comprehensive human experimental studies that are carefully controlled and documented to ensure reproducibility and to withstand challenge by proponents of other standards of safe concentrations. Such studies can establish the presence and importance of acute health responses in normal and hyperreactive people. Dose-response information, including minimal-measurable-effects concentrations, can be determined. Hypotheses can be developed and then subjected to animal or epidemiologic investigation. Typically, controlled human studies on the health effects of

specific pollutants use fixed concentrations, an absence of interfering pollutants, controlled environmental conditions, and well-characterized volunteer subjects. Risk is inherent in any research involving humans, and it is axiomatic that reasonable and proper care should be taken.[41] Prudence in the design of controlled human experiments requires that, when available, information from animal and epidemiologic work be used to delineate ways in which critical human studies are likely to be most useful.

The purposes of this chapter are to review available test methods and protocol designs for controlled human studies, to review and discuss published data, and to make recommendations for future studies.

EXPERIMENTAL METHODS

The basic design of studies on effects of pollutants should seek to maximize information relevant to public health. Tests must be reliable and sensitive, the experimental air environment must be rigorously controlled, and the manner in which subjects are exposed to this environment must simulate ambient exposure. These constraints impose complications and necessitate a focus on environmental control and monitoring, physiologic testing, and evaluation of symptoms and clinical observations.

Controlled-Environment Chambers

CONSTRUCTION AND MONITORING EQUIPMENT

A survey of the environmental control and monitoring technology used in several experimental studies[4-6,34,67] indicated significant limitations in experimental control capability. There are seven controlled-environment chambers or clean-room facilities in the United States for human exposure (community air pollution inhalation) from which studies have been reported. Another is under construction at the University of North Carolina in association with the EPA at Chapel Hill. There are three chambers in Canada of similar design.

Table 9-1 lists design features of the exposure chambers in the United States that have air-cleaning equipment. The facility at the University of Maryland Hospital, Baltimore,[42] has a chamber with activated-charcoal and high-efficiency particle filters and controlled temperature and humidity. St. Vincent's Hospital and New York University, New York City, each have a clean-room facility. The University of Pennsylvania Hospital, Philadelphia,[23] has a self-contained, reinforced-concrete

TABLE 9-1 Controlled-Environment Chambers or Clean-Room Facilities for Community Air Pollution Short- or Long-Term Human Exposure, United States[a]

Item or Condition	Facility[b]						
	H.S.C.	U. Md.	U. Pa.	R.L.A.	St. V.	St. B.[c]	N.Y.U.
Temperature control	+	+	+	+	+	+	+
Humidity control	+	+	+	+	+	+	+
Pressure control	+	−	+	−	−	−	−
Sound control	+	−	+	+	−	+	−
High-efficiency particle filter	+	+	+	+	+	+	+
Laminar flow[d]	−	+	−	[e]	−	+	−
Charcoal filter	+	+	+	+	+	+	+
Catalytic filter[f]	+	−	−	+	−	−	−
Scrubber[g]	+	−	−	−	−	−	−
Purafil filter[h]	−	−	−	+	−	−	−
Active[i]	−	+	−	+	−	+	−

[a] Based in part on information from an EPA-sponsored workshop on controlled-environment chambers, April 26-27, 1971, Research Triangle Park, N.C.
[b] H.S.C. = Hospital for Sick Children, Washington, D.C.
U. Md. = University of Maryland Hospital, Baltimore, Md.
U. Pa. = University of Pennsylvania Hospital, Philadelphia, Pa.
R.L.A. = Rancho Los Amigos Hospital, Downey, Calif.
St. V. = St. Vincent's Hospital, New York, N.Y.
St. B. = University of California, Santa Barbara, Calif.
N.Y.U. = New York University, New York, N.Y. (clean-room facility has been dismantled).
[c] More than one chamber available. This describes the plexiglass chamber used for air pollution studies.
[d] Refers to use of an intake plenum to ensure fairly uniform airflow and negligible gas concentration gradients within the region of the exposure chamber occupied by subjects.
[e] Can be operated with intake flow either through or bypassing the plenum.
[f] Heated or nonheated bed designed to oxidize carbon monoxide and light hydrocarbons (not removed by activated charcoal) to carbon dioxide and water.
[g] The liquid-acid gas scrubber is packed with polypropylene and uses sodium carbonate solution.
[h] Aluminum oxide pellets impregnated with potassium permanganate.
[i] As judged by personal communication or recent publication.

climate-controlled chamber, with pressure controls from 28 to 32 in. Hg (about 710 to 810 mm Hg, or 95 to 108 kilopascals). Rancho Los Amigos Hospital, Downey, California, has a totally enclosed steel chamber. Inlet air is heated and passed through a Hopcalite catalyst bed, filtered by high-efficiency particle filters, activated charcoal, Purafil (aluminum oxide pellets impregnated with potassium permanganate), and a Mine Safety Appliance catalytic adsorbent bed.[27] The controlled-environment facility at the Hospital for Sick Children, Washington, D.C., is a self-contained, reinforced-concrete structure that can provide clean or spe-

cifically polluted air of monitored composition along with controlled temperature, pressure, and humidity. The living area for the subjects is a two-room apartment with bath and minimal kitchen facilities (600 ft^2, or 56 m^2).[63] The University of California at Santa Barbara has two chambers for temperature- and humidity-controlled environmental studies. A third chamber is used for altitude simulation, and a fourth for air pollution studies.[19,55] The latter chamber is 6.5 ft (2 m) wide, 10 ft (3 m) long, and 19 ft (5.8 m) high and is constructed of plexiglass. A chamber complex at the University of Wisconsin in Madison is designed primarily for studies of occupational inhalation toxicology.[46,65]

A description of the environmental-control chamber for human experimental studies at McGill University, Montreal, exemplifies the other two Canadian facilities—at the Gage Research Institute, Toronto, and McMaster University, Hamilton, Ontario. It is a transparent chamber, 12 ft (3.7 m) long, 8 ft (2.4 m) wide, and 6 ft (1.8 m) high constructed of 14 6 × 4-ft (1.8 × 1.2-m) panels of plexiglass held together with angled metal. The chamber is built within an ordinary air-conditioned working laboratory, with a door in the middle of one of the 12-ft (3.7-m) sides. Near the bottom of the chamber a hole has been cut to accommodate a 9-in. (23-cm) window fan, which gives the desired incoming atmosphere. Air from the laboratory is not purified before entering the chamber. At the end of the chamber opposite the fan, two outlet pipes, 6.5 and 3.75 in. (16.5 and 9.5 cm) in diameter, run from the chamber ceiling to a fume hood. Air passes through the chamber at approximately 12,000 ft^3/h (336 m^3/h), resulting in about 20 volume changes hourly. The temperature and humidity controls are those for the building in which the chamber is housed.

For the most reliable results, chamber environment should be monitored continuously with instruments and techniques equivalent to those used in ambient-air monitoring networks (see Chapter 6). Calibration of instruments should follow recommendations by appropriate agencies and be checked by cross comparisons with those in other analytic laboratories.

Ideally, two monitoring instruments, each operating on a different principle, should be used for each gaseous pollutant under study. Ozone and nitrogen oxides can be monitored with chemiluminescence analyzers, which provide fast response and freedom from interference by other pollutants. Total oxidants can also be monitored by the neutral potassium iodide solution and Saltzman reagent methods with continuous-flow colorimetric analyzers. A nondispersive infrared analyzer and an oxidative electrochemical analyzer can be used for monitoring carbon monoxide, and light-scattering counters for monitoring particles (see Chapter 6).

ENVIRONMENTAL VARIABLES

In human experimental studies, variables to be considered and controlled include pollutant gas concentration, humidity, temperature, light intensity, noise, particles, and the presence of other gases.

The relative humidity, air temperature, noise, and artificial lighting must be controlled in environmental exposure chambers, because they can influence a subject's response directly or indirectly. At high temperature, heat stress can dominate the symptomatic and physiologic responses of human subjects. The rates of some chemical reactions of exposure gases depend heavily on temperature, so temperature should be controlled. This can be done with refrigerated cooling coils in the air-conditioning section of air supply systems, heaters, and thermocouple sensors. Extremes of relative humidity are known to cause discomfort in humans. The equilibrium size of hygroscopic aerosol particles, the degree of conversion of sulfur dioxide to sulfurous acid in sodium chloride droplets,[45] and the extent and rate of chemical reaction of mixtures of such pollutant gases as sulfur dioxide, ozone, and ammonia depend on the relative humidity of the chamber air. Cooling coils in air-conditioning units and the injection of purified steam can be used to regulate relative humidity. Artificial lighting within human-exposure chambers should be designed to ensure proper illumination, but to avoid potential experimental complications. If the intensity is too high, the air may overheat. Ultraviolet irradiation should be avoided to prevent photochemical reactions from forming uncontrolled and unknown reaction products in the chamber. If ultraviolet irradiation of gases is desired to form specific oxidant compounds for human exposures, it should be carried out in separate chambers to avoid contamination of the human-exposure chamber with intermediate products and to prevent the subjects from being directly exposed to harmful radiation. Conventional tungsten and fluorescent lighting meet the illumination requirements of human-exposure chambers.

High levels of noise known to cause discomfort to humans are unacceptable. Moderate noise also becomes undesirable in some experimental situations, if it is distracting or interferes with speech communication.

During controlled exposures of human subjects to specific compounds like ozone, the concentrations of suspended particles and trace gases must be known and minimized to ensure that health effects can be attributed solely to ozone. The air purification units for the environmental chambers are designed to remove most of the particles and pollutant gases from the ambient air. Prefilters and efficient absolute filters are used to remove 99% of the particles with diameters of 0.3 μm

or more. Catalytic beds, adsorption beds, and activated-carbon and chemical filters are used to convert carbon monoxide to carbon dioxide and to remove most of the other gaseous hydrocarbons, sulfur dioxide, nitrogen dioxide, nitric oxide, and ammonia. Condensation nuclei counters, which are commercially available, can be used to monitor the total number of particles with diameters greater than 20 Å. Light-scattering nephelometers can approximate the total number concentration of particles with diameters of 0.1–1 μm. Single-particle optical counters with multichannel analyzers can be used to monitor the number of particles in finite size intervals between 0.2 and 10 μm. Alternatively, cascade impactors and particle filters can be used to monitor the total mass concentration and the size distribution of the aerosol in the chamber. Gas monitors are commercially available for sulfur dioxide, nitrogen dioxide, nitric oxide, carbon monoxide, and ozone. Ammonia and these other gases can also be collected by bubblers and analyzed by wet chemical methods if proper precautions are taken to account for interferences. Trace hydrocarbons can be monitored by taking bag samples and analyzing them by gas chromatography, mass spectrometry, or infrared spectrophotometry.

In the design of controlled exposures to gases and particles, separately or in combination, extreme care is needed to avoid formation of interfering species. For example, in a study of the effect of a mixture of nitrogen dioxide and ferric oxide aerosol in dogs,[44] significant quantities of ammonium and nitrate ions were found in particles filtered from the air of the exposure chamber. Ammonia and trace hydrocarbon gases also emanate from sedentary and exercising humans. To prevent these contaminant gases from building up in the chamber, a continuous flow of fresh air through the chamber must be maintained.

In exposures of humans to artificially generated aerosols, where the information is to be relevant to ambient aerosols, several factors are important: the particle diameter distribution must be fairly constant and fall within size ranges typical for the given compound in the ambient air, the chemical composition of the aerosol must be stable and predictable, and the electric charge distribution of the aerosol must simulate that of normal atmospheric aerosols.

Selection and Characterization of Subjects

A minimal group size is difficult to specify. Studies on similar subjects could be expected to give the least variability in results; thus, dose–response information on relatively small groups of medically characterized subjects could provide a basis for extrapolating to essentially

similar population groups. Useful results have been obtained with as few as eight subjects.[30] Healthy young males probably should be studied initially because some variations are known to accompany aging and some problems are peculiar to females (e.g., the estrous cycle and pregnancy). Later studies of older males and females will, however, be required for complete information.

Volunteers with disease who can give informed consent and for whom some expected benefit balances the expected risk can be considered as potential subjects. If those selected for the study are smokers, this information and an estimate of the degree of smoking are needed, because systematic differences between smokers and nonsmokers have been found.[34,43]

People with known or suspected cardiopulmonary disease who are referred by a physician for diagnostic environmental stress-testing may also serve as subjects. Testing under environmental stress of persons who are suspected hyperreactors to pollutants is analogous in concept to exercise stress-testing of suspected cardiac patients. However, in contrast with the general availability of exercise stress-testing laboratories, the expense involved in an operational facility for environmental stress-testing limits its availability to research centers. Accordingly, a person who is referred for clinical testing by his physician may well become part of investigative programs and be asked to volunteer for additional procedures.

In selection of subjects, ethical considerations are dominant and limit the deliberate exposure of minors or of others who are unable legally to be volunteer subjects.

Among the criteria for selecting subjects with close similarities, socioeconomic status cannot be ignored any more than nutritional status or genetic background. Finally, place of residence and prior occupational pollution exposure may be important.

Measurement of Human Response

In addition to the visual monitoring of subjects during testing, clinical assessment can be accomplished by having the project physician interview each subject concerning symptoms, with a standard questionnaire immediately after exposure. Also, subjects can keep a record, on a standard form, of symptoms experienced during and after exposure. It is well to note here that caution should be exercised in interpreting symptoms, because these are unlikely to be "blind" studies.

Much progress has been made in recent years in the measurement and understanding of lung physiology. Usually, several methods are

available for the measurement of each characteristic of lung function of interest. Comprehensive descriptions of the various pulmonary-function tests are readily available,[8,10,18,24] so a complete review is not attempted here.

Because the respiratory tract is an initial target of any air pollutant challenge, it usually receives primary attention in tests to determine irritant effects of exposure. Other aspects of interest include hematology, blood enzyme biochemistry, eye irritation, and psychomotor performance. Constriction of the large airways, maldistribution of ventilation due to narrowing in some small airways, constriction of peripheral lung units, and mechanical or gas diffusion impairment due to edema are possible effects of insult by pollutants. A variety of pulmonary tests is required to examine the possibilities.

Volume-Time and Flow-Volume Curves[11] The characteristics measured by the maximal expiration are forced vital capacity (FVC), 1-s forced expiratory volume (FEV_1), peak expiratory flow rate ($\dot{V}_{max}$), and flow rates at 50% and 25% of the remaining FVC ($\dot{V}_{50}$, $\dot{V}_{25}$) for partial and maximal flow-volume curves. These measurements give an easily obtained, relatively reproducible evaluation of overall pulmonary mechanical performance, but provide little information on the mechanisms responsible for an observed change.

Air Resistance (R_{aw}) and Thoracic Gas Volume (TGV)[20,21] The measurement of R_{aw} is probably more sensitive than maximal flow measurements for constriction of large airways, but it is also more difficult to perform and is less stable. These problems similarly affect the measurement of TGV, which, however, may be useful for detecting gas trapped as a consequence of airway dysfunction (in combination with a gas-dilution lung-volume determination).

Total Respiratory Resistance (R_1)[22,26,72] With appropriate attention to technical problems, resistance can be measured at various pressure perturbation frequencies of 3, 6, 9, and 12 Hz. Unfortunately, this measurement is affected by changes in upper-airway configuration, which may complicate the detection of changes in pulmonary airways *per se*. As predicted by Otis,[53] the method is believed capable of detecting asynchronous mechanical behavior (unequal regional ventilatory time constants), which otherwise can be documented only by the considerably more difficult measurement of dynamic lung compliance.

Closing Volume (CV)[1,14] Buist and Ross[14] measured the lung volume at

which closure of a significant number of small airways presumably occurs, as well as estimating residual volume (RV) and total lung capacity (TLC) through the expired nitrogen concentration. In another study,[15] they presented a method to estimate the uniformity of ventilation distribution by measuring the slope of the alveolar nitrogen plateau.

Static Lung Compliance (C_s) and Dynamic Lung Compliance (C_{dyn})[47] These are measured from recordings of transpulmonary pressure and respiratory flow and volume. Dynamic compliance in the tidal range is measured in a series of breaths each at normal frequency and at other frequencies—such as 20, 40, 60, 80, and 100 breaths/min—with total volume monitored and kept constant. Static compliance is measured by interrupting airflow intermittently during an inspiration from functional residual capacity (FRC) to TLC, followed by an expiration to RV. Each determination is preceded by an inspiration to TLC to give a consistent volume history. Compliance measurements are indispensable for documentation of changes in the mechanical characteristics of the lung, particularly the development of unequal time constants. Unfortunately, the measurements are somewhat unstable and require considerable effort on the part of subjects and investigators. In some studies, these tests are performed only on a subgroup of subjects selected for motivation and performance.

Pulmonary Diffusing Capacity ($D_{L_{CO}}$) This can be determined by the single-breath carbon monoxide method according to a technique developed by Ogilvie *et al.*[52] For example, a test gas containing 0.15% carbon monoxide and 10% helium in air can be used. In calculating $D_{L_{CO}}$, correction is made for backpressure of carbon monoxide due to significant concentrations of blood carboxyhemoglobin. Helium is measured with a thermal conductivity meter, and carbon monoxide with an infrared detector or an electrochemical analyzer. Reproducibility of this test may be poor under some conditions, and it is affected by changes in ventilation-diffusing-capacity ratios, but the test offers the potential of detecting changes in the blood-air interface (such as alveolar edema) that might otherwise go undetected.

Oxygen Consumption This can be measured at rest and during exercise on a constant-load bicycle ergometer or a treadmill at a level producing a specified percentage of predicted maximal oxygen consumption.[2] Expired air is collected and measured with a spirometer to determine total expired volume, and samples are analyzed for oxygen and carbon dioxide.

Carboxyhemoglobin Concentration [HbCO] This can be estimated with the method of Jones and co-workers.[39] The subject holds a deep breath for 20 s to allow equilibration of carbon monoxide between alveolar air and blood and then expires a sample of that air into a container. The air carbon monoxide concentration may be directly related to carboxyhemoglobin concentration [HbCO]. The test can be performed before exposure in an environmental chamber to help to verify that the subject has not received inordinate ambient pollutant exposure.

Biochemical studies on human blood samples have been used by Buckley *et al.*[13] to measure response. They reported changes in red cells and serum of men after a single acute exposure to ozone at 0.50 ppm for 2.75 h. Red-cell membrane fragility and glucose-6-phosphate dehydrogenase and lactate dehydrogenase activities were increased, and red-cell acetylcholinesterase activity and reduced glutathione were decreased. Red-cell glutathione reductase (GSSRase) activity was not significantly altered; however, serum GSSRase activity was significantly decreased, and serum vitamin E content and lipid peroxidation were significantly increased.

A number of investigators[9,57,59] have argued that behavioral changes are more prevalent than frank physiologic or clinical changes as effects of air pollutants. If the ability to perform routine tasks, such as operating an automobile or a complex piece of machinery, is compromised by pollutant exposure, the setting of air pollution standards should take such effects into consideration.

In one study,[30] human subjects were tested in a controlled-environment chamber with a high (summer) temperature and with ozone, nitrogen dioxide, and carbon monoxide as pollutants. Performance on a divided-attention task given at the end of the exposure period and the subjects' heartrate variability (a potential psychophysiologic measure of attention) were evaluated. The subjects displayed a significant decrement in peripheral attention associated with increased ambient temperature. Effects attributable to pollutant gases were variable.

Subjects exposed to ambient oxidant pollution or to controlled oxidant pollution in a test chamber express their response by symptoms, as well as by physiologic changes. Because of their subjective nature, little attention has been given to symptoms in overall assessments of effects; however, with systematic collection, these experimental data lend themselves to semiquantitation.

In the study by Hackney *et al.*,[27] the investigators proposed a method for consistent gathering of symptom data by means of a checklist,

wherein symptoms are scored (according to predetermined criteria of severity) for three different periods during and after exposure. The sum of the scores for all symptoms from all periods gives a total symptom score, or discomfort index, for the 24-h period after the start of exposure. In their study, symptom scores for 42 subjects exposed to ozone at 0.25, 0.37, and 0.5 ppm revealed a dose-response relationship for higher doses that approximated the relationship demonstrated by objective pulmonary function. These results suggest that semiquantitation of symptoms is feasible in controlled pollution-exposure studies and may be a useful clinical investigative tool.

Experimental Design Considerations

Multiple stresses may be important in assessing functional and adaptive capabilities of people. An exposure protocol can be designed to simulate as closely as possible the ambient exposure of a group likely to have significant exposure, such as people working outdoors on a smoggy summer day. A 2-h exposure period is realistic, in that high ambient pollutant concentrations usually persist about that long. Other exposure periods should be chosen to simulate different pollutant episodes. Specified amounts of exercise can serve as a tool in experimental design to provide additional stress. Intermittent light exercise (sufficient to approximate a doubling of minute volume) during exposure produces a realistic degree of ventilation (to which pollutant dose is approximately proportional) during typical work. Increased temperature is another stress factor that is often present during oxidant air pollution episodes and so can be introduced into the experimental situation. The design can provide for successive days' exposures, in that deleterious effects of exposure may be cumulative. These requirements can be incorporated into a protocol in a cost-effective manner that tests several subjects on a given day. However, with a single chamber of limited size, this requires staggered exposure and testing periods, precluding "blind" studies or control measurements on the same day. Thus, sham control runs (identical protocols with exposure to purified air) precede the pollutant exposure, so that reliable baseline values of the measured characteristics can be obtained.

"Blind" studies with chemical substances that have a characteristic odor or irritant effect are difficult to manage. If, as with ozone, the odor sense tends to diminish, then "odor sham" protocols are possible. However, keeping the subjects and the investigators (other than the safety officer) truly uninformed about the nature of a particular experiment is not a trivial problem.

Cyclic variations in measurements are potentially important.[42] Adequate information is not available, for example, on normal variation over time in tests of human physiologic function. This presents a problem in designing not only short-term experiments, but especially longer-term studies. Detailed data on stability of a given measurement over periods of minutes, hours, days, weeks, months, and even years would improve rational experimental designs. Variations in measurements with changing seasons, especially summer versus winter, might be expected, because of the effect of large temperature differences on physiology and life style. Interactions may be important in design and data interpretation. The likelihood of recent prior exposure to high oxidant concentration is clearly a function of the summer season and geographic location.

There is a definite need for dose-response information on specified groups of people, if frequency distributions of responses are to be estimated. Obtaining and using such information present numerous problems, such as selection of appropriate groups and their size; the need to include persons with disease, as well as the very young and very old; and extrapolation of the data to the total population. With separate dose-response information on representative samples of major population groups, a frequency distribution of responses at each pollutant concentration might be determined by weighting the mean change for each group according to the general populations they represent. Information on frequency distribution of responses is necessary for any cost-benefit analysis that will require quantitative estimates of effects in terms of dollars.

With due regard to the factors just discussed, a test series may reasonably be designed to support or reject the hypothesis that no acute effects of realistic pollutant exposure will be detected in volunteer subjects. Results supporting the hypothesis would be useful to regulatory agencies in setting air pollution standards. If minimal effects are found in a group of "normal" subjects, an effective experimental strategy is to test a group of specified "hyperreactive" subjects, characterized by a prestudy history of cough, chest discomfort, or wheezing associated with allergy or exposure to air pollution.

Experimental data can be subjected to repeated measures like one-way analyses of variance. *Post hoc* multiple comparisons between experimental groups (e.g., the Newman-Kuels test[71]) are made when significant differences among test conditions are found. A few significant differences due to random variation may be found, because of the number of statistical comparisons usually being made; therefore, all observed, statistically significant changes must be examined critically for physiologic significance.

Ambient-Air Studies

Experiments can be conducted with well-specified ambient air, if a suitable chamber is in a highly polluted area and if ambient air can be introduced into the chamber without significant pollutant losses. Other runs with clean air and matched temperature and humidity serve as controls. Alternatively, mobile health-testing laboratories with air monitoring capabilities can go to polluted areas of interest.

Detection of short-term cardiopulmonary effects of ambient oxidant pollutant exposure is complicated by several factors. The physiologic measures of such effects exhibit significant inherent variability, which may mask the relatively small changes expected. The atmosphere contains many potentially hazardous materials, both natural and man-made, in continuously varying concentrations. Not all these substances have been identified, and many that have been identified cannot be adequately monitored. Other environmental factors, such as temperature and humidity, may influence physiologic function independently and thus complicate the detection and interpretation of pollutant effects. Variations in concentration of any given pollutant are usually closely linked to variations in concentrations of other pollutants, and this makes it difficult to assess the toxicity of a single component of the ambient mixture. These problems have led to the assertion that ambient-exposure study results cannot be interpreted reliably, owing to the incompleteness of both the atmospheric and the biologic information and the inability to control or allow for all interfering variables. This assertion is correct in principle, but it neglects the possibility of obtaining valuable partial answers to health-effects questions through ambient studies. Such improved understanding of the effects of ambient oxidants should be attainable through improved atmospheric monitoring; improved physiologic, biochemical, and clinical evaluation of exposed subjects; application of findings from concurrent controlled-exposure studies; and combination of ambient- and controlled-exposure studies.

REVIEW AND DISCUSSION OF PUBLISHED DATA

The data base on health effects of photochemical oxidants and ozone was reviewed by the Subcommittee on Ozone and Other Photochemical Oxidants in a report prepared in September 1974 for the Committee on Public Works, U.S. Senate.[48-51] The following discussion repeats some of the material in that report, to exemplify the need for further work, including controlled human studies.

The federal primary ambient air quality standard for photochemical

oxidants is 160 $\mu g/m^3$ (0.08 ppm), a maximal 1-h average concentration not to be exceeded more than once per year.[68] *Air Quality Criteria for Photochemical Oxidants*[67] catalogs and describes the overall data base for present standards. In information received by the Subcommittee (February 23-24, 1974), the following evidence was cited as the basis on which the EPA set the standards:

- When high-school cross-country runners were exposed for 1 h to photochemical oxidants at 0.03-0.3 ppm, their performance decreased with increasing concentration.[69] A statistical test for threshold values (regression using "hockey stick" functions) applied to these data gives a threshold estimate of 0.12 ppm, with a 95% confidence interval of 0.067-0.163 ppm.[3,31,54,67,69]
- At short-term maximal peak oxidant concentration of 490 $\mu g/m^3$ (0.25 ppm), subjects with asthma begin to experience significantly more attacks. These maximal daily peaks may occur with a maximal hourly average concentration as low as 300 $\mu g/m^3$ (0.15 ppm). Eight people sensitive to smog experienced increased attacks at oxidant concentrations corresponding to those at which plant damage occurs (8-h average, 200 $\mu g/m^3$, or 0.10 ppm).[60,67]
- At short-term peak oxidant concentration of 196 $\mu g/m^3$ (0.10 ppm) and above, humans begin to experience eye irritation. These peaks would be expected to occur with maximal hourly average concentrations of 50-100 $\mu g/m^3$ (0.025-0.05 ppm).[56,58]
- At short-term ozone concentration of 160 $\mu g/m^3$ (0.08 ppm) for 3 h, experimental animals (mice) exhibited increased susceptibility to laboratory-induced bacterial infections. A firm dose-response relationship was established for this effect.[16,17]
- Brinkman *et al.*[12] found that, at short-term ozone concentration of 392 $\mu g/m^3$ (0.20 ppm) for 1-2 h, experimental animals had increased sphering of red blood cells. Similarly, humans had increased sphering after 30 min of exposure to 490 $\mu g/m^3$ (0.25 ppm).
- Brinkman *et al.*[12] also found that, at long-term ozone concentration of 392 $\mu g/m^3$ (0.20 ppm) for 5 h/day for 3 weeks, structural changes in the nuclei of heart muscle fibers were produced in adult rabbits and mice. The fibers reverted to normal a month after exposure.

The EPA used the data from the first three studies listed to obtain the value of 200 $\mu g/m^3$ (0.1 ppm) for a maximal 1-h concentration—the lowest concentration at which measurable human effects are generally observed. No formal methodology was used in making this selection; it was derived as a best judgment by EPA officials.

Similarly, no published record exists to describe the procedure used

in selecting 0.08 ppm as the national primary standard for photochemical oxidants. It is the recollection of EPA officials that the standard was set midway between the highest average background concentration of ozone (0.06 ppm) and 0.1 ppm, giving a 20% "margin of safety."

There are obvious inadequacies in the established values. First, it was assumed that the oxidant standard is a surrogate standard for photochemical oxidants, with ozone as the indicator. However, it is clear that very low concentrations of specific irritants, such as peroxyacetylnitrate (PAN), are sufficient to cause eye irritation. Second, some of the statistical techniques used to determine the lowest concentration at which effects are observed were inconsistent and undocumented. A "hockey stick" function[3,54] was sometimes used to find an effect threshold.

The inadequacy of the technical data base on photochemical oxidants was realized from the start by EPA officials; Barth *et al.*,[3] describing the situation in 1971, stated that "many bits of information required to place the present National Ambient Air Quality Standards on an irrefutable and unassailable scientific basis are not yet available."

Exposure to Ozone

Studies before 1970 were reviewed in *Air Quality Criteria for Photochemical Oxidants.*[67] Details of selected controlled human studies reported later are given below.

Bates *et al.*[6] measured significant changes in lung function (decrease in maximal flow rate at 50% of the vital capacity, maximal transpulmonary pressure, and increase in total pulmonary resistance) in 10 normal male subjects aged 23-35 yr (including two smokers) exposed to pure ozone at 0.75 ppm for 2 h. Two of the three subjects who exercised intermittently at twice the resting volume showed accentuated effects. In a separate study by Hazucha *et al.*[34] on effects of short-term exposure, significant changes in lung function (decreases in forced vital capacity, forced expiratory volume at 1 s, maximal flow rate at 50% of vital capacity, and maximal midexpiratory flow rate, and increases in closing capacity and residual volume) were found in 12 normal males aged 23.6 ± 0.7 yr (including six smokers) exposed to pure ozone at 0.75 and 0.37 ppm for 2 h during alternating rest and exercise periods. The higher concentration affected smokers more than nonsmokers, whereas the lower concentration affected smokers and nonsmokers similarly. In these two studies, most subjects complained of cough, chest tightness, and substernal soreness. A few also had pharyngitis, dyspnea, and wheezing.

Hazucha[32] also measured small decreases in the same lung functions

as above in three normal male subjects of mean age 20 yr (nonsmokers) exposed to pure ozone at 0.25 and 0.56 ppm for 2 h during alternating 15-min periods of rest and exercise. Although these decreases were not statistically significant, in view of the small number of subjects involved, they were consistent and in general agreed in value with dose-response curves drawn from data at 0.75 and 0.37 ppm (Figure 9-1). The subjects

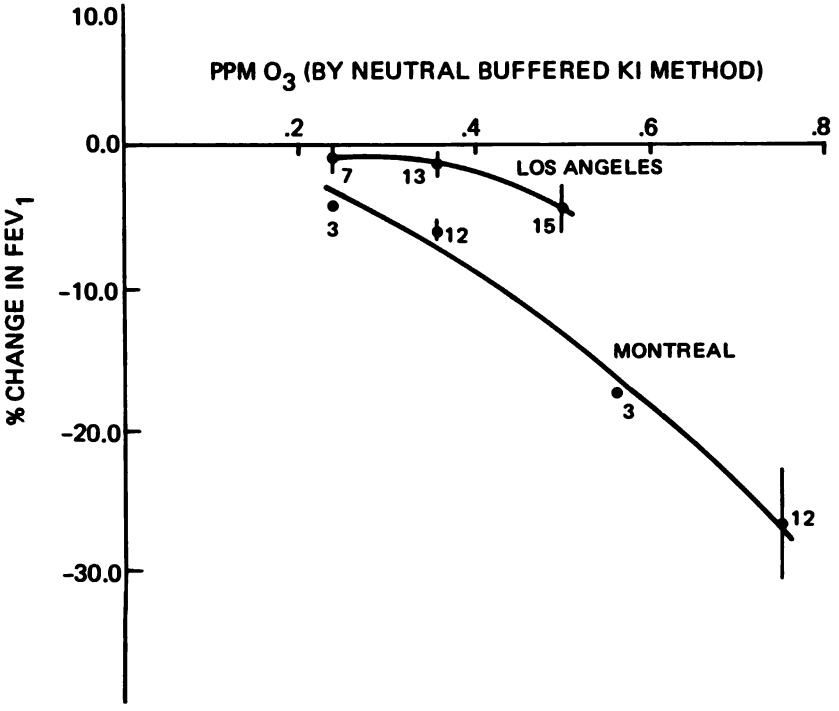

FIGURE 9-1 Behavior of FEV_1 in response to ozone expressed as percentage change from the appropriate control (sham) experiment, smokers and nonsmokers combined. Exposures 2 h, with intermittent light exercise, except some Los Angeles exposures at 0.5 ppm for 4 h with intermittent exercise. Points and error bars represent mean response (ΔFEV_1 postexposure vs. control) ± 1 standard error. Curves represent best second-order polynomial fit to mean points. Numbers adjacent to points represent sample sizes: L.A., 7 at 0.25 ppm, 13 at 0.37 ppm, 15 at 0.5 ppm; Montreal, 3 at 0.25 ppm and 0.56 ppm (error bars not given, because of small sample size), 12 at 0.37 ppm and 0.75 ppm. Mean values for L.A. and Montreal 0.37 ppm × 2-h exposures are significantly different. $t = 4.15$. $p < 0.01$. Equations for dose-response curves: L.A., second-order fit to 3 data points, $(\Delta FEV_1\%) = -7.524 + 46.546\ (\text{ppm } O_3) - 81.795\ (\text{ppm } O_3)^2$; Montreal, second-order fit to 4 data points, $(\Delta FEV_1\%) = 2.147 - 13.229\ (\text{ppm } O_3) - 35.383\ (\text{ppm } O_3)^2$. *NOTE:* These equations are only for the concentration ranges of the data. Derived from Hackney *et al.*[27,29,30] and Hazucha.[32]

coughed and complained of chest tightness, substernal soreness, increased salivation, and expectoration of mucus. The studies just described[6,32,34] were conducted in the Montreal chamber facility with general conditions of: temperature, 21–23°C; humidity, 42–49%; inside background air; and two or more Mast coulombmeters for oxidant monitoring.

Studies by Hackney *et al.*[27,29,30] used a protocol designed to simulate summer exposure in the Los Angeles southern coastal air basin and included the additional stresses of heat, intermittent exercise, and repeated exposures. Careful attention was given to environmental control, pollutant monitoring and generation, and subject selection. Four male subjects aged 36–49 yr (including one cigarette-smoker) judged by subjective criteria to have normally reactive airways completed this protocol. No obvious effects—as assessed by clinical response and measures of respiratory, cardiac, and metabolic functional change—were noted after exposure for 4–5 h to 0.5-ppm ozone, 0.5-ppm ozone and 0.3-ppm nitrogen dioxide, or 0.5-ppm ozone, 0.3-ppm nitrogen dioxide, and 30-ppm carbon monoxide.

A second group of four, aged 29–41 yr (including two cigarette-smokers), who had previously experienced clinical bronchospasm and were judged by subjective criteria to have hyperreactive airways (history of developing symptoms during light activity in smog or history of asthma) developed clinical discomfort and were unable to complete the protocol. Exposed to ozone at 0.5 ppm for 4–5 h, this group developed marked changes in pulmonary mechanics and gas distribution. Some effects were later found after exposure to ozone at 0.37 ppm, but not at 0.25 ppm.

In light of the marked clinical effects, the protocol was modified, and a third group of seven subjects, aged 22–36 yr (including two smokers), judged to have normally reactive airways were studied. This group, exposed to ozone at 0.5 ppm for 2 h, showed only minimal effects on the first day of exposure; however, five of the seven showed significant effects on a second exposure day. A fourth group of seven subjects, aged 22–41 yr (including three cigarette-smokers), exposed for 2 h to 0.25-ppm ozone, 0.25-ppm ozone and 0.3-ppm nitrogen dioxide, or 0.25-ppm ozone, 0.3-ppm nitrogen dioxide, and 30-ppm carbon monoxide showed no obvious effects. Of seven subjects, three were judged on the basis of subjective criteria to have hyperreactive airways and four were deemed to have normally reactive airways. A fifth group of five subjects, aged 27–41 yr (including one cigarette-smoker), were exposed to 0.37-ppm ozone for 2 h. Important changes with exposure were not found in most physiologic measures. However, one subject showed substantial changes

in measures of lung mechanical function, which became worse on the second day of exposure. Findings from this series of studies indicate that there is a wide range of sensitivity to photochemical pollutants and that more sensitive people develop significant symptoms, biochemical changes, and respiratory-function decrease under exposure conditions similar to those experienced during pollution episodes. The studies just described[27,29,30] were conducted in the Rancho Los Amigos Hospital chamber facility with general conditions of: temperature, 31° C; relative humidity, 35%; purified background air; and chemiluminescence and neutral potassium iodide monitoring for oxidants.

Comparison of health effects of exposure to ozone[5,29,30,32] suggests that Canadians are more reactive than southern Californians. These results are summarized in Figure 9-1. Experimental methods and subject responses were compared further in a cooperative investigation of this apparent reactivity difference. In studies conducted in California, four Canadians and four Californians were exposed to ozone at 0.37 ppm in purified air at 21° C and 50% relative humidity for 2 h with intermittent light exercise.[28] Exposures to purified air alone served as controls. Methodologic differences sufficient to explain different results of previous studies were not found. Subject responses were similar to those observed previously—Canadians on the average showed greater clinical and physiologic reactivity to exposure than Californians, who were no more than minimally reactive. Canadians also showed larger increases in red-cell fragility after exposure. These results support the existence of a real difference in reactivity between the Californians and Canadians studied to date. Although the number of subjects tested in the cooperative study is small, the good agreement between these results and previous results in the separate laboratories allows considerably increased confidence in direct comparison of the previous results from Los Angeles and Canada, providing a considerably larger data base from which to judge relative reactivity.

If the difference in response to ozone between Canadians and southern Californians studied is accepted as real, a hypothesis of adaptation in southern Californians is supported. Further support of the hypothesis requires demonstration that the subjects tested are truly representative of larger population groups residing in the same areas and that identifiable factors other than adaptation are not sufficient to explain the observations.

In studies by Kerr *et al.*,[43] 20 healthy adults—19 males and one female, aged 21-60 years (including 10 smokers and 10 nonsmokers)—were exposed to ozone at 0.5 ppm for 6 h in an environmental chamber. During this period, they engaged in two 15-min medium-exercise ses-

sions (100 W at 60 rpm) on a bicycle ergometer. Symptoms of dry cough and chest discomfort, after ozone exposure, were more commonly noted in nonsmokers. They also had a significant decrease in dynamic compliance after exposure. Subjects who experienced symptoms, in general, were the ones who developed objective evidence of decreased pulmonary function. Chest discomfort ranged from tightness on full inspiration to generalized chest pain accentuated by exercise, cough, and irritation of the nose and throat. Significant changes from control values for the group as a whole after ozone exposure were observed for several pulmonary-function tests: specific airway conductance (SG_{aw}), pulmonary resistance (R_L), FVC, and FEV_3. No significant change was observed with respect to diffusing capacity (D_{LCO}), static lung compliance (C_{st}), or the various tests derived from the nitrogen elimination rate. When the smokers were considered as a separate group, no significant decrease in pulmonary function was observed, although some individual smokers had decreases in pulmonary function. These studies[43] were conducted in the Baltimore chamber facility with general conditions of: temperature, 24°C; relative humidity, 45%; purified background air; and two Mast coulombmeters for oxidant monitoring.

Folinsbee *et al.*[25] tested the response of 28 subjects after ozone exposure to three stages of ergometer exercise with loads adjusted to 45, 60, and 75% of maximal aerobic power. The subjects were exposed to ozone at 0.37, 0.50, or 0.75 ppm for 2 h, at rest or while exercising intermittently—15 min of rest alternated with 15 min of exercise at a workload sufficient to increase ventilation by a factor of 2.5. These studies were conducted in a plexiglass chamber (Toronto). Oxidant monitoring was with a coulombmetric analyzer (Mast), which was checked periodically against neutral buffered potassium iodide. Neither submaximal exercise oxygen consumption nor minute ventilation was significantly altered after ozone exposure at any concentration. The primary response was an alteration of exercise ventilatory pattern. An increase in breathing rate ($r = 0.98$) and a decrease in tidal volume ($r = 0.91$) were correlated with the dose of ozone, calculated as the volume of ozone inspired during exposure. It was concluded that, through its irritant properties, ozone modified normal ventilatory response to exercise and that this effect was dose-dependent.

N. J. Rummo, J. H. Knelson, S. Lassiter, and J. Cam (personal communication) exposed 22 male volunteers, aged 19–27, to ozone at 0.4 ppm for up to 4 h in relatively clean ambient air or to ambient air alone. Subjects were seated during exposure, except for two 15-min exercise periods on a bicycle ergometer at 700 kg-m-min exercise that about doubled the heart rate and quadrupled the ventilation volume.

After 2 h of ozone exposure, there was a significant change ($p < 0.05$) in FVC, MMF, and airway resistance (R_{aw}). Several other measures (FEV_1, $\dot{V}_{50}$, and $\dot{V}_{25}$) were lower after 2 h of exposure, but the statistical significance was borderline. However, after 4 h of exposure, all flow measures were significantly decreased, compared with controls. After 4 h, R_{aw} increased, FVC decreased further, and FEV_1 decreased significantly. Residual volume, functional residual capacity, and total lung volume did not change as a result of the ozone exposure.

Kagawa and Toyama[40] have reported on the results of limited studies involving four normal male subjects exercising while exposed to ozone at 0.9 ppm for 5 min. A significant decrease in SG_{aw} was found during exposure and after 5 min of recovery.

All studies[6,25,27,29,30,32,34,43] reported an association between symptoms and changes in lung function. In general, people who noticed cough and substantial tightness first were the ones who developed the greatest defect in function. Ozone-induced defects in function were not usually found in the absence of definite symptoms of ozone-induced respiratory irritation.

It would be desirable at this point to discuss mechanistic interpretation of the lung-function measures—such as FEV_1 delta nitrogen, and airway resistance—that have been reported to change with ozone challenge. Unfortunately, although such measurements as FEV_1 give an easily obtained, relatively reproducible evaluation of overall lung mechanical performance, they provide little information on the mechanisms responsible for any observed change. Also, the measurement of the slope of the alveolar nitrogen plateau (delta nitrogen) cannot be interpreted beyond saying that it reflects the uniformity of ventilation distribution. Airway resistance and specific airway conductance are thought to be sensitive measures of constriction of large airways. Results of animal experiments provide additional information about mechanisms of action of oxidant pollutants on the respiratory system. These are discussed in Chapter 8.

In interpreting the results of human experimental studies with pure ozone in relation to the oxidant standard, it must be remembered that the other oxidants ordinarily present in smog were absent. Conceivably, a larger difference may be necessary between the lowest concentration of pure ozone at which an observed effect occurred and the air quality standard than between the lowest concentration of oxidant *mixtures* and the standard.

The newer experimental studies described above[7,25,27,29,30,32-34,40,43] show that significant adverse health effects occur in humans at ozone concentrations of 0.37 ppm and higher. Some limited studies[32] show

evidence of human health effects of exposure to pure ozone at concentrations as low as 0.25 ppm (see Figure 9-1). The uncertainties of extrapolating ozone effects to oxidant effects, of dose estimation, of measurement, and of subject sensitivity must be kept in mind when comparing the new data with the standard.

Exposure to Ozone and Other Pollutants

Hazucha[32] and Bates and Hazucha[7,33] reported an enhanced effect between ozone and sulfur dioxide in controlled-exposure studies of airway responses in humans. Hazucha studied subjects only in the resting state; Bates and Hazucha, during rest and exercise. With various tests of ventilatory function—including FEV_1, midmaximal flow rate, and maximal expiratory flow rate at 50% of vital capacity—the investigators showed that healthy male college students experienced no effect of sulfur dioxide at 0.37 ppm, approximately a 10% decline in function with ozone at 0.37 ppm, and a 20–40% decline in function with a combination of sulfur dioxide at 0.37 ppm and ozone at 0.37 ppm. The effect of ozone alone on human function was not clearly mainifested until subjects had been exposed for 2 h; the effect of the combination of ozone and sulfur dioxide was apparent within 0.5 h. The maximal effect was observed in the first 30 min after cessation of exposure. Care was taken to use scrubbers on the instruments that were used to measure each gas concentration. The effect of the combined gases was greater than the sum of the effects of the two gases administered separately.

Adult male volunteers were exposed to purified air,[27,29,30] to ozone alone, or to ozone in combination with nitrogen dioxide and carbon monoxide. No additional effects were detected when nitrogen dioxide at 0.3 ppm was added to ozone. The addition of carbon monoxide at 30 ppm to the ozone-nitrogen dioxide mixture produced no additional effects, other than a slight increase in blood carboxyhemoglobin content and small decreases in psychomotor performance, which were not consistent in different subject groups.

Exposure to Peroxyacetylnitrate

In 1965, Smith[62] reported increased oxygen uptake during exercise in college students exposed to PAN at 0.3 ppm. The average increase was 2.3%. More recently, Drinkwater *et al.*[19] and Raven *et al.*[55] found no significant physiologic change attributed to PAN exposure. They tested 20 young men (smokers and nonsmokers) for maximal aerobic power (treadmill walk) in a 35° C environment under four ambient air condi-

tions: filtered air, carbon monoxide at 50 ppm, PAN at 0.27 ppm, and carbon monoxide and PAN. No significant physiologic effects were noted during the PAN exposure for smokers or nonsmokers. Maximal aerobic power was not affected by any pollutant condition. Heat stress was more effective than any pollutant condition in reducing work capacity, and a 4.3% decrease in maximal aerobic power in a 35° C environment, compared with a 25°C environment, was greater than the differences in aerobic power found between filtered air and any of the pollutant conditions.

Other Experimental Studies

Holland *et al.*[38] studied 14 subjects under conditions of short-term exposures to irradiated automobile exhaust. The environmental conditions simulated the "moderate" smog episodes in the Los Angeles air basin. Oxidant concentrations were reported as 0.22-0.27 ppm (on the basis of the alkaline potassum iodide method). No significant changes attributed to exposure were found in reaction time, vital capacity, work performance, or oxygen consumption.

Henschler *et al.*[35] reported that the characteristic pungent odor of ozone was detected instantaneously at low concentrations (less than 0.02 ppm), depending on individual sensory perception acuity, and perceived at higher concentrations (0.05 ppm) for an average of 5 min. At higher concentrations, the odor was perceived as stronger and persisted for an average of 13 min.

Eye irritation has been a common complaint of people exposed to photochemical air pollution.[37] Attempts to investigate this experimentally have encountered problems, because of the subjective nature of the human response and the multiphasic photochemical reactions involved. Human studies conducted until 1970 on eye irritation were cataloged and discussed in *Air Quality Criteria for Hydrocarbons.*[66]

Several studies indicated that the major photochemical products that cause eye irritation are acrolein, PAN, and peroxybenzoylnitrate (BPzN).[36,61,64] Ozone, the principal contributor to ambient oxidant concentrations, is not an eye irritant. This is an important observation, because it might have been assumed that lacrimation would correlate with respiratory effects. In fact, however, ozone concentrations can be high enough to cause considerable respiratory effects without any irritation of the eyes having been noted. Heuss and Glasson[36] stated that the potency of PBzN as an eye irritant is 200 times that of formaldehyde. In studies by Heuss *et al.*,[37] PBzN was formed in parts-per-million amounts by irradiating automobile exhaust in laboratory apparatus. When some

aromatics were added to a low-aromatic gasoline, PBzN and greatly increased eye irritation resulted.

Wilson *et al.*[70] measured irradiated automobile exhaust and pure organic compounds and found that the addition of sulfur dioxide, although it increased aerosol formation, decreased eye irritation in many of the hydrocarbon-NO_x systems studied.

A number of components of ambient oxidant mixtures are discussed in Chapter 3. Further detailed characterization studies of ambient particulate pollution may suggest that some of these compounds are present in the organic fraction in quantities likely to be detrimental to human health. If so, further controlled health-effects studies would be indicated and might be accomplished with irradiated and diluted automobile exhaust or exposure to pure specific compounds.

Limitations of Controlled Human Studies

It is apparent that controlled human experimental studies are needed, but are cumbersome and costly. Other limitations include restrictions as to the number of measurable responses and the fact that individual pollutants are usually studied, rather than ambient mixtures. In addition, healthy subjects are usually studied, rather than sensitive population groups. The results of these studies are applicable to acute effects; but their relation, if any, to chronic effects is not known.

Ethical considerations impose design requirements that increase the cost and complexity of controlled human studies. For example, prudent operational guidelines for such studies could include:

- The investigators serving as the first subjects for each series of exposure studies.
- Pollutant exposure concentrations selected so as not to exceed documented ambient concentrations.
- The exposure environment constantly monitored by the technician who is operating the pollutant-generating equipment.
- The presence of a physician.
- Monitoring of subjects electrocardiographically from outside the chamber.
- Direct observation of subjects at all times by viewport or closed-circuit television.
- Frequent checks by the attending physician and the chamber engineer for hazardous conditions and, if they encounter any, application of corrective measures or halting of the study.
- Physician screening of prospective volunteers.

• Obtaining informed consent after the subject is given a full explanation of the experimental procedures, including known risks and discomforts; the form would describe the procedures and risks fully and specify that the subject may withdraw from the study at any time.

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10

Epidemiologic Studies

A variety of epidemiologic studies attempting to associate various characteristics of human health and functioning with daily concentrations of photochemical oxidants have been carried out during the last two decades, primarily in the Los Angeles air basin. A few of these have shown clear-cut associations involving large segments of the population. Other studies related to both the same and different health indicators have failed to demonstrate any consistent association. Failure to establish clear-cut associations may well be inherent in some aspects of the epidemiologic method. Epidemiologic studies have the advantage of being focused on the real world, using human populations in their normal setting. This permits identification of both long-term and short-term variations in health and function that can be correlated with pollutant exposures. It also permits identification of susceptible groups within the general population that may be at greater risk. Epidemiologic studies, however, have two main disadvantages: the health indexes used must inevitably be relatively crude, as opposed to controlled laboratory conditions; and it is extremely difficult in the real world to isolate the effect of one environmental factor in the presence of numerous other independent and dependent variables, which may be synergistic with or antagonistic to the environmental factor under study—e.g., other air pollutants, meteorologic factors, socioeconomic status, occupational exposures, personal

health habits, and cigarette-smoking. Within these limits, some definite conclusions can be drawn as to the association (or lack of it) of some health indicators with photochemical oxidant pollution. Because of the lack of epidemiologic information in some critical areas, use must be made of clinical and toxicologic studies on photochemical oxidants, both in setting ambient air standards and in planning future epidemiologic studies.

MORTALITY

Death is obviously the most clear-cut and significant end point for determining the effect of an environmental challenge on health. It rarely has a single, isolated cause, however, even in apparently simple events like automobile accidents, homicide, and suicide. Careful investigation will usually reveal a constellation of factors that contributed to the final event. Documented episodes of deliberate or inadvertent exposure of a sizable population to a high concentration of a known toxic substance are extremely rare, and all attempts to link death with ambient air pollution at normal concentrations must consider a multitude of factors. Furthermore, these factors often vary from person to person and from situation to situation. Among the factors that must be considered are the age and state of health of the person affected, the presence of coexisting disease states (likely for many people), the activity being pursued by the person at the time of and just before death, and individual biologic variation in response to different types of stress. Other factors that must be considered in relation to the specific challenge are the type of pollutant, the presence of other pollutants (always a factor in epidemiologic studies of populations in natural environments), the duration of exposure at various concentrations, the frequency and degree of previous exposures to similar pollutants that may initiate either tolerance or hypersensitivity to the offending agent, and the meteorologic conditions under which exposure may take place. The last factor is often shown to play an overwhelming role in determining the outcome of the challenge.

Several mortality studies on different population groups have been conducted, principally in the Los Angeles area. Studies by the California Department of Public Health (reported in 1955, 1956, and 1957)[5-7] showed a marked increase in daily mortality among Los Angeles County residents aged 65 and older during a period of high photochemical-oxidant concentrations in a 2-week period in September 1955. Temperatures during this period were examined, and it was shown that the increase in deaths occurred immediately after a marked increase in temperature; mortality returned to normal before oxidant concentrations

had returned to those prevailing before the episode. Although the photochemical-oxidant concentrations were high immediately before, during, and after the heat wave, the daily mortality decreased when temperatures dropped. During other periods when daily mortality in citizens over 65 was compared with both temperature and oxidant concentrations, no consistent pattern could be discerned, nor could a pattern be seen at other times when oxidants rose to medium and high concentrations without an accompanying increase in temperature. It must be concluded that heat, rather than photochemical-oxidant exposure, precipitated the increase in fatalities in the population studied. Nevertheless, the possibility cannot be dismissed that the concomitant high oxidant concentrations increased the death toll. Such synergistic actions between air pollutants and weather are well known.[23]

Heat has long been known to be one of the greatest stresses to which an elderly person can be subjected, and heat waves in New York, as well as Los Angeles, have been regularly shown to be followed by a tripling or quadrupling of daily deaths among persons over 65, especially before air conditioning became prevalent. Unfortunately, high temperatures are often associated with high oxidant concentrations in the Los Angeles basin. In an attempt to isolate the effects of these two environmental characteristics, Oechsli and Buechley (cited in NATO[30]) studied the mortality associated with three Los Angeles heat waves, in 1939, 1955, and 1963. It was assumed that less photochemical-oxidant pollution was present in 1939 (although no measurements were made at that time), and yet there was an equally high mortality among elderly persons in 1939 and 1955. Temperatures were similar in each of the three heat waves. A lessened mortality was observed during the 1963 heat wave, perhaps because of the increasing use of air conditioning between 1955 and 1963. The authors concluded that the high photochemical-oxidant concentrations did not augment the mortality effect of high temperatures.

Residents of nursing homes were a special group included in the California Department of Public Health study cited above.[5-7] They were considered of special interest because of the assumed presence of chronic illness in most of them and their possible greater susceptibility to photochemical-oxidant pollution. All nursing homes in Los Angeles County containing 25 or more beds were included, and daily mortality, transfer to a hospital because of worsening disease, maximal daily temperatures, and the occurrence of smog-alert days with ozone concentrations of 590 $\mu g/m^3$ (0.3 ppm) or higher were considered. Again, the striking effect on mortality during heat waves was noticed, but no correlation with smog-alert days could be demonstrated.

Massey, Landau, and Deane[22] conducted a study on two synthetic

communities within the Los Angeles air basin. These communities, with a combined population of 944,391 persons, were divided into high-pollution and low-pollution areas. The two areas had similar temperatures. The mean number of daily deaths in the low-pollution area was subtracted from the mean number in the high-pollution area, and the difference was examined by correlation and regression analyses with respect to differences in pollution. The observers were unable to detect any significant correlation between mortality and difference in oxidant, sulfur dioxide, or carbon monoxide content in the ambient air.

Studying specifically the effect of pollutant concentrations on cardiac and respiratory diseases in Los Angeles County, Hechter and Goldsmith[19] compared daily mortality from these causes with fluctuations in oxidant and carbon monoxide concentrations and temperature. The authors removed the statistical effect of season by fitting Fourier curves to the data, which were presumed to be independent of season. When residues from the fitted curves were analyzed, no significant correlations between pollutants and mortality from cardiorespiratory diseases could be found. The authors also applied lags of 1-4 days, but again were unable to demonstrate any significant correlations. It is possible that some additive effect of pollutants on mortality occurred but would not be detected by this analysis.

Another attempt to compare cardiorespiratory deaths with photochemical-oxidant pollution was carried out by Mills,[27] who compared seasonally adjusted nursing-home deaths in Los Angeles with measures of photochemical-oxidant pollution. He found a suggestive positive association between photochemical-oxidant concentration and excess deaths when pollution rose above 390 $\mu g/m^3$ (0.2 ppm). Although heat and seasonal variability were considered, the statistical analyses used make it questionable whether their effect could be suppressed.

A variety of methods have been used to assess a relationship between mortality from chronic illness and photochemical-oxidant pollution, but none has been able to demonstrate a clear-cut relationship. The possibility of a synergistic effect has not been ruled out, although the experience of areas other than the Los Angeles basin with lower concentrations of photochemical-oxidant pollution suggests that temperature is an overwhelming factor in these deaths.

Further studies on mortality in relation to photochemical-oxidant pollution are needed to delineate differences in susceptibility (especially in children and the elderly), to assess the additive effects of various weather conditions, to examine geographic variations in mortality in relation to pollution, and to determine whether excess mortality is attributable to specific diseases (e.g., emphysema), which may have been

overlooked in examinations of total mortality. Populations should also be more carefully categorized to determine whether specific subsamples are at greater risk.

HOSPITAL ADMISSIONS

Several studies have attempted to correlate increased numbers of hospital admissions with variations in photochemical-oxidant pollution. The California Department of Public Health study of excess mortality[5-7] also investigated hospital admissions as a possible health indicator of oxidant pollution. Admissions to Los Angeles County General Hospital in September through December 1954 for childhood asthma, tuberculosis, other respiratory diseases, and all other causes were examined. No significant association with oxidant concentrations was found.

Two other investigators have attempted to relate hospital admissions to increased photochemical-oxidant pollutants. Sterling *et al.*[41,42] studied admissions to Los Angeles hospitals for a 7-month period in 1961. They grouped diseases into "highly relevant," "relevant," and "irrelevant" categories. The "highly relevant" were allergic disorders, inflammatory diseases of the eye, acute upper respiratory infections, influenza, and bronchitis. "Relevant" disorders included diseases of the heart, rheumatic fever, vascular diseases, and all other respiratory diseases. All other diseases were considered "irrelevant." Because both hospital admissions and photochemical-oxidant concentrations varied by day of week, corrections for both were introduced into the analysis. Although there was a statistically significant correlation coefficient between hospital admissions for "highly relevant" and "relevant" conditions and photochemical-oxidant pollution, the differences were extremely small. Both "highly relevant" and "relevant" diseases showed some correlation with carbon monoxide and ozone, and nitrogen oxides and particles correlated with "highly relevant" diseases. Even if the relationships claimed by this study are valid, they are minimal, and confirmation by studies over a long period is needed.

Brant and Hill[2,3] also examined the number of patients who had respiratory and cardiovascular diseases and were admitted to Los Angeles County General Hospital for a 4.5-month period in 1954. These months—generally regarded as the "smog season"—included the highest photochemical-oxidant concentrations of the year. Because they accepted into their study group only persons who had resided for at least 3 yr within 13 km of downtown Los Angeles and excluded the extremely old and children under 9, the numbers they dealt with were rather small

for this brief period. Complex statistical analyses were used to compare admissions with photochemical-oxidant pollution, as measured at a monitoring station not quite 6 km from the hospital. The methods used have been questioned by many competent statisticians, and the claims of a significant correlation between periods of photochemical-oxidant pollution and hospital admissions are difficult to accept. Even more difficult to accept is the claim of a positive correlation between high concentrations of photochemical oxidants and hospital admissions 4 weeks later. No reasonable medical explanation can be given for the latter correlation.

Studies to date have demonstrated an extremely weak correlation (if any) between photochemical-oxidant pollution and hospital admissions. If such correlations do exist, observations must be carried out over considerably longer periods with adequate provisions for controlling confounding variables, such as meteorologic factors.

ACUTE RESPIRATORY INFECTION

The possibility that chronic exposure to photochemical-oxidant pollution alters host resistance and thus increases susceptibility to acute respiratory infection is a challenging one. An opportunity to examine this thesis occurred in 1968 and 1969, when an epidemic of A2/Hong Kong influenza occurred in southern California. Pearlman *et al.*[32] had the opportunity to observe five southern California communities that varied from high to low oxidant concentrations during this period. Because other pollutants, especially oxides of nitrogen and particles, have shown associations with influenza attack rates, an association with photochemical-oxidant pollution was carefully investigated. Pearlman *et al.* examined the morbidity among 3,500 elementary-school children in five communities. Although these communities showed a definite gradient in chronic oxidant exposure, there was no significant difference in oxidant concentrations immediately before or during the epidemic. Questionnaires to parents of the children between November 1968 and January 1969 asked about the presence of influenzalike illness or symptoms. Blood specimens were later obtained from the children whose parents reported upper respiratory symptoms. Titration was performed by hemagglutination inhibition and complement fixation against A2 influenza antigen. Although more children reported illness in low-pollution cities, no statistically significant difference in illness rates could be correlated with exposure to chronic oxidant air pollution. Inasmuch as acute oxidant concentrations were roughly similar in all five communities,

it may be that the children did not receive an oxidant dose sufficient to impair host defense mechanisms immediately before or during their exposure to the virus. In statistical manipulation, the investigators were able to minimize other significant covariants, but concluded that "oxidant exposure...did not alter host defense mechanisms enough to cause morbidity differences."[11]

School absenteeism is known to be due in large part to acute respiratory illness, so Wayne and Wehrle[47] followed daily absenteeism rates in schoolchildren in Los Angeles, but were unable to detect any relationship between variations in ambient photochemical-oxidant concentrations and school absenteeism.

In a long-term study, Hammer *et al.* recorded daily symptoms in healthy young student nurses for a period of nearly 3 yr (October 1961–June 1964).[16] An average of 61 student nurses in two nursing schools in Los Angeles completed a daily diary for 868 days on the incidence of cough, chest discomfort, eye discomfort, and headache—all symptoms that might be expected to accompany an acute respiratory infection. Other recorded information included incidence of gastrointestinal symptoms, physician visits, restricted activity, and menses. Daily maximal hourly concentrations of photochemical oxidants, carbon monoxide, and nitrogen dioxide and daily temperatures were also measured at monitoring stations within 2 miles (3.2 km) of the schools.

As expected, eye discomfort showed the strongest correlation with photochemical-oxidant concentrations, with one-third of the total population reporting the symptom when oxidant concentration reached 0.5 ppm. However, cough and chest discomfort also increased with maximal hourly oxidant concentration. Headache showed a positive association with photochemical-oxidant concentration, but the increase in the number reporting the symptom (3–8%) was considerably less than for other symptoms. Temperature and carbon monoxide and nitrogen dioxide concentrations could not explain the associations found. Cigarette-smoking history, allergies, and bias in reporting were suppressed as variables in the analysis. It is of particular interest that the authors were able to compute the thresholds at which the prevalence of the symptoms began to increase and found them close to the present U.S. national primary standard for photochemical oxidants, indicating little or no margin of safety. The eye irritation threshold was calculated at approximately 294 $\mu g/m^3$ (0.15 ppm). Because these were all young healthy adults, relatively free from chronic disease, the same effects in elderly persons or in those with chronic heart or lung diseases could be expected to result in greater physiologic embarrassment.

Although the observed threshold for eye discomfort is comparable

with reported results of human experimental exposure-chamber studies, it is recognized that current methods for measuring photochemical oxidants primarily measure ozone, which is not known to be an eye irritant. The authors therefore assumed that the measured concentrations of ozone reflected the concentrations of such known eye irritants as peroxyacylnitrate, peroxybenzoylnitrate, formaldehyde, and acrolein—all of which have been shown to be present in Los Angeles basin air. However, the symptoms of cough, chest discomfort, and headache have all been associated with occupational exposure to ozone, so it is possible that ozone itself is the offending agent for these symptoms. It is interesting that the observed daily maximal hourly carbon monoxide concentrations in this study were below those that have been reported to cause headache experimentally. Nevertheless, inasmuch as a significant number of student nurses did report headache associated with increased oxidant concentration, an interaction between carbon monoxide and oxidants cannot be excluded.

AGGRAVATION OF PRE-EXISTING DISEASE

The best-documented health effect of general urban air pollution is the aggravation of pre-existing disease. In the notorious Donora episode and in the many well-documented episodes of excess mortality in London's "smogs," most of those who died had had marked impairment of cardiac or pulmonary reserve. Similarly, the majority of epidemiologic studies demonstrating an association of health impairment with increased air pollution were carried out in areas where the atmosphere was characterized by a particulate-sulfate (and preponderantly chemically reducing) type of pollution. Several investigators have attempted to determine whether similar health effects are produced by exposure to atmospheres that have photochemical oxidants as the characteristic pollutants.

Studies by Motley *et al.* [28] on 66 volunteers, 46 of whom had pulmonary emphysema, were conducted in a "filtered room" from which oxidants were removed by activated-charcoal filters. Pulmonary function studies demonstrated improvement in emphysematous patients who remained in the room for 40 h or more, if they had entered it on a day when the ambient air in Los Angeles was considered "smoggy" by the investigators and the Los Angeles Air Pollution Control District. Normal subjects showed no change in pulmonary function in the filtered-air chamber, compared with measurements taken when they were breathing ambient "smoggy" air. Emphysematous patients also showed no improvement if they entered the room on a "nonsmoggy" day. The study,

although widely cited, is difficult to evaluate, because the number of patients was relatively small. In the emphysematous patients who entered on "smoggy" days, some improvement was noted in vital capacity, FEV_3, and maximal breathing capacity. The most marked change, however, was a decrease in residual lung volume.

Remmers and Balchum[33] at Los Angeles County Hospital used a specially constructed room with an air-conditioning filter system and filters that could be used at the discretion of the investigator to remove photochemical oxidants and nitrogen oxides from ambient air. Particulate matter could also be partially removed. They carried out studies during September and October 1964 and from March to November 1965. Each patient spent approximately 1 week in the room without filtration and a second week with filtration. Pulmonary-function studies were performed one or more times a day. Temperature and humidity were kept constant during both weeks. Airway resistance, diffusing capacity, other pulmonary functions, blood oxygen tension, and oxygen consumption were measured when the subjects were at rest and exercising. Examination of the data indicated that airway resistance was affected by increased oxidant concentration over a range of 100–450 $\mu g/m^3$ (0.05–0.23 ppm). Unfortunately, the data are difficult to interpret, because many of the subjects were cigarette-smokers, the subjects were exposed to ambient air containing many substances in addition to oxidant, and the observed improvement with filtered air may have been caused by the removal of other pollutants, such as aerosols, aldehydes, and particles. A more elaborate analysis of these data was carried out by Ury and Hexter.[46] It demonstrated that oxygen consumption, in most cases, increased with oxidant concentration. Unfortunately, the oxidant concentration was measured only twice a day at a station some distance from the site of the subjects' exposures. Oxidant was also measured as ozone, so the concentrations of other irritant oxidant compounds could not be determined.

A much larger group of men was followed by Schoettlin[39] in an attempt to study the long-term effects of community air pollution on persons with and without symptoms of pulmonary disease. These two groups were veterans who lived in the domiciliary unit and in the chronic-disease annex of the Los Angeles Veterans' Administration Center. A group of 528 veterans (in the domiciliary unit) who had no symptoms of respiratory disease were used as the control population. A group of 326 men were selected on the basis of having shown at least two symptoms of respiratory disease for 2 yr or longer. The symptoms included cough, sputum production, shortness of breath, wheezing, and abnormal breath sounds. The two groups were matched by age and smoking

history, and the resulting pairs were studied weekly by repeated pulmonary-function tests and response to a standardized respiratory-symptom questionnaire. An analysis of variance showed no statistically significant effects of air pollution on incidence or prevalence of respiratory symptoms or on tests of pulmonary function. Nevertheless, the maximal oxidant and oxidant precursor concentrations consistently accounted for more of the variation in frequency of symptoms and clinical signs in the group with pulmonary disease than in the control group. Up to 30% of the variation in symptoms in the group with pulmonary symptoms appeared to be correlated with the maximal oxidant-precursor concentrations, whereas no association could be demonstrated in the control group.

ASTHMA

Asthma, a disease featuring sudden and dramatic variations in respiratory symptoms and pulmonary function, is well known to be related to environmental factors in many cases. Many other factors, however, may play a significant role in the precipitation of asthma attacks, including meteorologic factors, emotional factors, infection, allergy, and physical activity. It has also been demonstrated that there is a significant ethnic variation in factors associated with acute asthma attacks.[11] Nevertheless, between 2 and 5% of the population is subject to asthma attacks, so it has been examined as an indicator of health effects of photochemical-oxidant pollution.

Schoettlin and Landau[40] carried out one of the earliest studies to determine whether aggravation of asthma could be related to increases in oxidant concentrations. They followed 137 asthmatics in Pasadena with known asthma for at least 5 yr. Over at least a 3-month period in late 1956—the usual peak oxidant period of the year—weekly reports were obtained from each patient as to the number of attacks. Of the 3,435 attacks reported by these patients, less than 5% were spontaneously associated by the patient with "smog." Although most reported attacks on days with high enough oxidant concentrations to cause eye irritation and plant damage, the majority of attacks occurred between midnight and 6:00 a.m., whereas peak oxidant concentrations occurred between 10:00 a.m. and 4:00 p.m. This lag, however, would not necessarily rule out a causative role of oxidants in asthma attacks. The conclusion of the authors was that only 8 of the 137 patients could be characterized as "smog reactors."

The oxidant concentrations later reported by Renzetti[34] were a peak of

0.25 ppm and, for the same period and same station, a maximal hourly concentration of about 0.02 ppm. It is of interest that one-third of the attacks spontaneously associated with "smog" were reported by a single patient. However, in spite of the small number of people who spontaneously associated their attacks with "smog," this study suggests that there are probably some people who do experience asthma attacks related to increased concentrations of photochemical oxidants. Further studies to confirm this, sponsored by the EPA, are under way in the Los Angeles air basin.

EFFECTS OF PHOTOCHEMICAL OXIDANTS ON HEALTHY POPULATIONS

Athletic Performance

Wayne *et al.*[48] related athletic performance of high-school cross-country runners over a 5-yr period from 1959 to 1964 to oxidant measurements for the hour of the race and 1, 2, and 3 h before the race. Temperature, relative humidity, wind velocity and direction, and oxides of nitrogen were also considered, but showed no relation to the running times. A "training effect" that might be expected to improve performance on each succeeding race was also considered. A significant relationship, however, was observed between high oxidant concentrations and the percentage of runners whose performance decreased, compared with their performances in the previous home meet. It is of interest that the deterioration in performance of these 21 runners was noted beginning at 130 $\mu g/m^3$. With increasing oxidant concentration, there was a manifest deterioration in team performance over a range of 60–590 $\mu g/m^3$. This deterioration in performance began well below the national photochemical-oxidant standard of 160 $\mu g/m^3$ (0.08 ppm). The authors readily admitted that "the observed effects may be more related to the lack of maximal effort due to the increasing discomfort than to decreased physiologic capability." Nevertheless, although they discussed the possibility that decreasing performance might be due to the detrimental effect of discomfort from eye irritation, the data provide convincing evidence that some components of the air that were measured as oxidant had an effect on team performance.

Although no threshold effect was specified by Wayne *et al.*, a later analysis by Hasselblad *et al.*[17] suggested a threshold estimate of 235 $\mu g/m^3$ (0.012 ppm). A similar study was carried out over a shorter period by Koontz[21] in Seattle, where oxidant concentrations were approx-

imately one-third those in Los Angeles. A decrease in performance of long-distance runners was noted with increasing oxidant concentrations, but these were also associated with marked temperature increases, and it was impossible to separate their relative contributions.

Automobile Accidents

Another association of oxidant concentrations with potentially serious implications is the increase in automobile accidents in Los Angeles noted by Ury.[45] He recorded accidents in each daylight hour of each weekday in the "high-smog" 3-month period between August and November for 2 yr and found a statistically significant relationship between oxidant concentrations and the number of automobile accidents. He was unable to demonstrate a similar relationship with nitrogen dioxide. Because the study compared hours with presumably similar traffic density, the likelihood that traffic density accounted for the association is probably small. The data suggest that photochemical-oxidant pollution impairs driving performance, either directly by interfering with oxygen transport or utilization or indirectly by causing eye discomfort or respiratory irritation. It is also possible that other pollutants, such as carbon monoxide emitted from the automobile tailpipe, account for some or all of the excess accidents.

Discomfort

Additional evidence of an association of discomfort or more severe health effects with increased oxidant concentrations was published by the Committee on Air Pollution of the Los Angeles County Medical Association (cited in NATO[30]). The organization circulated a questionnaire to a sample of every sixteenth physician registered to practice in the county. Although air pollution was not specifically mentioned in the questionnaire, the fact that it was sponsored by an air pollution committee probably suggested to the physicians that air polution was the principal environmental concern. The physicians were asked how many patients they had advised to move from Los Angeles County for health reasons before December 1960. By extrapolation from these questionnaires, it was estimated that more than 10,000 persons had been advised by their physicians to move from the county for health reasons and, for two-thirds of these, air pollution was given as the reason. Because this was a study of attitudes, it cannot be categorically cited as indicative of a definite health effect.

Pulmonary Function

Children have often been considered to be particularly sensitive indicators of environmental challenge and therefore to constitute a group in which adverse health effects of air pollution might be detected at lower concentrations than in adults. Two groups of elementary-school children living in the Los Angeles basin were assessed twice a week by McMillan *et al.*[26] with a Wright peak-flow meter to measure ventilatory performance. One group of 50 children lived in an area exposed to high oxidant concentrations, and the other group of 28 children lived in a less polluted area. During 11 months of the study, no correlation could be found between acute changes in oxidant concentration and ventilatory performance in either group. Unfortunately, there were significant differences between the two groups, in that the group in the more highly polluted area was ethnically mixed and had an incidence of upper respiratory tract infection three times greater than that reported in the other group. Pulmonary-function studies, as part of the Community Health Effects Surveillance Studies (CHESS) program, are under way in seven communities with graduated concentrations of photochemical oxidants in the Los Angeles basin. It is hoped that these will yield more definitive results.

A similar study on the relations between pulmonary function and exposure to photochemical-oxidant pollution was carried out by Cohen *et al.*[9] To eliminate the effects of smoking, the study was confined to Seventh Day Adventists who abstain from tobacco. They were divided into two groups: one living in the San Gabriel Valley (a high-oxidant-pollution area), the second living in San Diego, which was then considered to represent a relatively low-pollution area. Each participant was interviewed by a physician, underwent a battery of pulmonary-function tests, and completed a standardized respiratory-disease questionnaire. Daily maximal hourly average oxidant concentrations in the San Gabriel Valley were twice those in San Diego during the time of the study (San Gabriel, about 274 $\mu g/m^3$, or 0.14 ppm; and San Diego, 137 $\mu g/m^3$, or 0.07 ppm). In spite of the marked difference between the oxidant concentrations in the two areas, no significant differences could be found in pulmonary-function studies, the prevalence of chronic bronchitis, or the occurrence of respiratory-disease symptoms, as determined by the questionnaire. In this study, the differences may have been due to the absence of cigarette-smoking in both groups; some investigators have suggested that smoking is an additive factor in the effects of ambient air pollution.[14] It must also be noted that, although the peak values of oxidant pollution in the more polluted area were twice those

in the less polluted area, the mean yearly averges were essentially the same.

Kagawa and Toyama[20] in Tokyo followed 20 normal 11-yr-old schoolchildren once a week from June to December 1972 with a battery of pulmonary-function tests. Environmental factors studied included oxidant, ozone, hydrocarbon, nitric oxide, nitrogen dioxide, sulfur dioxide, particles, temperature, and relative humidity. Temperature was found to be the most important environmental factor affecting respiratory tests. The observers noted that pulmonary-function tests of the upper airway were more susceptible to increased temperature than those of the lower airway. Although the effect of temperature was the most marked, ozone concentration was significantly associated with airway resistance and specific airway conductance. Increased ozone concentrations usually occur at the same time as increased temperature, so their relative contributions could not be determined.

Eye Irritation and Lacrimation

Eye irritation and lacrimation are by far the most widespread and common symptoms clearly associated with increased photochemical-oxidant pollution. Although photochemical oxidant is customarily measured as ozone, ozone itself is not a primary eye irritant. Thus, the eye irritation associated with increased photochemical oxidants is probably due to some or many of the complex organic oxidants produced in photochemical "smog." Richardson and Middleton,[36,37] noted nearly 20 yr ago that eye irritation could be expected in a considerable portion of the population when the oxidant concentration reached 200 $\mu g/m^3$.

Renzetti and Gobran[35] carried out a controlled study on two groups of 20 female telephone-company employees working in adjacent rooms for a period of 120 days. Filters to remove eye irritants were switched periodically between the rooms, so that the two groups were alternately exposed to test and control conditions. Subjects were unaware of whether air was filtered or unfiltered at any given time, but they consistently reported eye irritation when the oxidant concentration exceeded 200 $\mu g/m^3$. Eye irritation has also been previously cited as the most commonly reported symptom in the student-nurse study of Hammer *et al.*[16]

That the problem of eye irritation in urban environments is not confined to Los Angeles is indicated by the studies of McCarroll *et al.*[24,25] and Mountain *et al.*[29] on a population in the lower east side of Manhattan. This population of approximately 2,000 persons of all ages representing the major ethnic groups of New York City was followed for a period of 3 yr with weekly interviews. Participants were queried

about the occurrence of many different diseases, disease symptoms, aggravation of pre-existing diseases, and a variety of other health factors. Frequency of reports of new eye irritation increased concomitantly with increases in oxidant concentrations in the neighborhood. Exact oxidant concentrations could not be measured in the study, because of the presence of high concentrations of interfering substances, including sulfur oxides, particles, and carbon monoxide. Substances other than oxidants might have contributed to the eye irritation reported by this population.

For the two most prevalent symptoms related to photochemical-oxidant exposure—eye irritation and lacrimation—no method of quantification has been developed. Eye irritation, although undoubtedly real, is a purely subjective response of the subject, and no measurement, other than the complaint itself, has yet been developed. Similarly, a routine objective measure of lacrimation remains to be developed. However, studies on tears have demonstrated that, when a person is experiencing eye irritation, the lysozyme content of the tears is lower than normal.[38] Measuring lysozyme content of the tears or the related pH variation appears promising, but more feasibility studies are necessary before the usefulness of the method is known.

A survey of the state of knowledge of eye irritation and lacrimation in response to photochemical-oxidant pollution was carried out by Wilson of the Copley International Corporation for the Coordinating Research Council.[49] After reviewing the studies cited above and related toxicologic work, the report concluded (p. 43):

> In many ways the understanding of eye irritation produced by photochemical smog has not kept pace with the understanding of other smog manifestations. Part of this is no doubt due to the fact that air pollution control agencies are not set up to collect data from human panels but rely exclusively on instruments. If an objective method were available for measuring eye irritation, and if this method were sensitive enough to be useful below the threshold of most humans, then a straightforward study could be done on natural smog.

Nevertheless, in spite of the many gaps in our knowledge of the components that produce eye irritation, there is remarkable uniformity in the findings of several epidemiologic studies cited, in the prevalence of the symptoms as oxidant concentrations increase, in the distress that oxidants cause the affected subjects, and in the threshold concentration at which the symptoms appear (0.15–0.2 ppm).

Photochemical-Oxidant Pollution as a Cause of Chronic Disease

Several studies attempting to relate aggravation of pre-existing chronic disease to increases in photochemical-oxidant pollution have been discussed. The possibility has also been raised that photochemical-oxi-

dant pollution plays a role in the *initiation* of some chronic diseases. In part, this possibility was suggested by laboratory studies demonstrating some radiomimetic properties of ozone. Although ozone may mimic ionizing radiation in some respects, it carries by no means all the carcinogenic implications. Nevertheless, experiments have indicated chromosomal damage to living cells by ozone[12] and to human cells *in vitro* by organic peroxides.[13] Work of Palmer and colleagues[31] has suggested that ozone is a potential cocarcinogen—a finding that carries great implications during a time of increasing rates of human lung cancer.

To assess the possible contribution of photooxidant pollution to lung-cancer mortality, Buell *et al.*[4] carried out a prospective study of lung cancer among 69,160 members of the American Legion—residents of Los Angeles County, the San Francisco Bay area, San Diego County, and all other California counties. This study was carefully adjusted for cigarette-smoking habits, occupation, and duration of residence in the same county. Death certificates for the first 5-yr period were checked for mortality from cancer of the lung and from other chronic lung conditions for a total of 336,571 man-years of observation. As would be expected, residents of the urban counties of Los Angeles, the San Francisco Bay area, and San Diego had considerably higher mortality, both from lung cancer and from other chronic pulmonary diseases. Even when adjusted for cigarette-smoking, these differences persisted, but they are compatible with other observations on the well-known "urban factor" in increased death rates from lung cancer, other cancers, and other chronic pulmonary disease. When heavy smokers (more than one pack a day) were examined, the relative risk of lung cancer was found to be greater in Los Angeles County than in other areas of California. For nonsmokers, however, the rate in the San Francisco Bay area and San Diego County was slightly higher than the rate in Los Angeles County. From these data, it was not possible to demonstrate any effect of oxidant pollution on lung-cancer mortality, if one assumes that in the years of observation (1958-1963) oxidant concentrations were higher in Los Angeles. However, because it is well known that the development of overt lung cancer is an indolent process that takes many years, the period of observation may not have been long enough to detect an environmental effect.

When the same observers examined mortality from chronic respiratory diseases other than cancer of the lung, the death rates were found to be somewhat higher in Los Angeles than in the San Francisco Bay area and San Diego County, particularly among persons who had lived for 10 or more years in the same area. It is well known, however, that socioeconomic class is an important factor in mortality from chronic respira-

tory disease,[50] and no data are available to show such differences among the groups examined in this study.

With standardized respiratory-survey techniques, Deane *et al.*[10] examined a group of West Coast outdoor telephone-company employees whose work and medical and social status were comparable with those of similar groups examined on the East Coast, in the United Kingdom, and in Japan. They noted that, in the group over 50 yr old, respiratory symptoms were more frequent in the Los Angeles and in the San Francisco populations than in the other areas studied. Twice as many telephone-company workers in Los Angeles complained of persistent cough and phlegm as in San Francisco. The groups were controlled for smoking habits. Surprisingly, in spite of the difference in reporting of symptoms, there were no important differences in the results of pulmonary-function tests between residents of the two areas in this occupational group. These findings are at variance with those of Hackney,[15] who noted seasonal differences, which were greater in San Francisco than in Los Angeles.

DISCUSSION

The EPA is conducting a major study in the Los Angeles basin on the effects of photochemical oxidants on health. It is a survey of schoolchildren in seven communities representing a gradient of oxidant exposure. In addition to comprehensive environmental monitoring data, specific health characteristics will be followed, including chronic respiratory disease in adults, lower respiratory disease in children, acute respiratory disease in both children and adults, pulmonary function in children, aggravation of asthma, irritation of mucous membranes, and tissue residues of trace metals. Complete data from this study will not be available for another 3 yr, but data from the first 2 yr may become available sooner.

A second study by the EPA will attempt to correlate the effects of photochemical oxidants and cigarette-smoking in promoting chronic respiratory signs and symptoms in cohorts of adolescents and their families. Pulmonary-function tests will be included, and this study should do much to answer the vexing questions of the relationship of chronic pulmonary disease and photochemical air pollution.

The abovementioned studies are being carried out by the EPA as part of the CHESS program. Although some of these studies have produced useful information, others are apparently not fulfilling their objectives. Specifically, these studies are designed entirely in-house by full-time

employees of the EPA at Research Triangle Park, North Carolina. The studies would have benefited by the presentation of experimental design and proposals to knowledgeable and experienced scientists outside the federal establishment for comment and criticism. Furthermore, some of the information collected in past studies and much that is now being collected is retained by the EPA to be used ultimately in standard-setting, without first being subjected to the scrutiny of the scientific community outside the federal government. To date, there has been relatively little publication in the usual scientific journals, and most data have been issued by the EPA in the form of monographs; hence, external scientific input, although sought, had little effect on the published scientific product. Because most of these studies have been related to disease or symptom prevalence, before-and-after studies of the effects of approved air quality have been impossible to carry out. This criticism applies not only to the CHESS studies, but to virtually all epidemiologic studies cited in this chapter, inasmuch as adequate environmental monitoring was rarely carried out in studies before the last decade. The epidemiologists and statisticians undertaking the CHESS studies are extremely competent and are supported by well-trained engineers, chemists, and meteorologists. Personnel turnover, however, has inevitably affected the continuity of direction of the program.

In addition to these much-needed studies, others should be designed to seek information on human populations concerning points already raised by important toxicologic and clinical studies. Although epidemiologists have been unsuccessful in relating infectious diseases to oxidant pollution, Coffin and Gardner[8] showed that ozone exposure before or after exposure to pathogenic bacteria strikingly increased mortality in mice. They found that ozone not only affected host resistance by inhibiting clearance rates, but also permitted uncontrolled growth of streptococci. This and much other toxicologic evidence that ozone interferes with host defense mechanisms in animals suggest that a similar phenomenon may go unrecognized in humans.

The evidence that ozone is a potential cocarcinogen[31] has grave implications. If the radiomimetic properties of ozone are confirmed, we may well be dealing with a nonthreshold dose-response situation in which no standard can be considered to represent a completely innocuous concentration of pollutant. The recent work of Bates and Hazucha[1,18] showing a synergistic effect of ozone and sulfur dioxide encourages the belief that pollutants cannot be considered separately when standards are promulgated and that the complex interrelationships among them must be considered. It is important to know whether other phenomena well demonstrated in animals, such as tolerance,[43] also occur in humans.

And there are suggestions that some type of cross-protection may occur, whereby exposure to ozone provides some protection against other irritant oxidants.[44] Analogues for all these phenomena should be sought in human populations, and methods should be devised for assessing their significance for human health.

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11

Plants and Microorganisms

The intent of this chapter is to give a critical review of all research related to the effects of photochemical oxidants, including ozone and the peroxyacylnitrates, on plants and microorganisms.

Injury to vegetation was one of the earliest indicators of photochemical air pollution. Injury was first observed in the Los Angeles area in 1944.[329] Since then, there has been a slow but steady increase in research efforts to understand the effects of these pollutants on vegetation. In the late 1960's and early 1970's, there has been a flood of published information.

In attempting to understand the effects of pollutant-pathogen interactions, investigators looked at the effects of oxidant pollutants on individual microorganisms and on these organisms as they infected host plants. Ozone has been tried as a fumigant to protect stored plants and plant parts against infection by parasitic and saprophytic fungi and bacteria. Research on ozone as a sterilant for water supplies and sewage systems is also related to effects on bacteria. Some studies have simply used bacteria as models for the action of ozone in biologic systems.

The chemical composition of the photochemical-oxidant complex is discussed in Chapter 2. The major phytotoxic components are ozone, nitrogen dioxide, and the peroxyacylnitrates. The latter homologous

series of compounds includes peroxyacetylnitrate (PAN), peroxypropionylnitrate (PPN), peroxybutyrylnitrate (PBN), peroxyisobutyrylnitrate (PisoBN), and peroxybenzoylnitrate (PBzN). The first four of these are known to be toxic to plants, but only PAN and PPN have been reported in sufficient quantities in ambient air to cause visible symptoms.[452] The effects of the nitrogen oxides (NO_x) are included in another NAS report.[359] Discussion of the quantitative effects of ozone and PAN will be limited to laboratory and controlled field exposures, because, under ambient conditions, the effects of these compounds are difficult to differentiate. Although ozone is, quantitatively, the primary oxidant component of photochemical air pollution, the term "oxidant" is used to include ozone and PAN in discussing biologic effects under ambient conditions. However, some studies suggest that phytotoxicants in addition to ozone and PAN are present in the photochemical complex.[182] Regardless of the number of phytotoxicants present, ozone is the most important, it has received the greatest amount of study, and its effects are better understood. The major thrust of this chapter covers research with ozone.

Before ozone[198,413] and PAN[442] were identified as specific phytotoxic components of the photochemical complex, researchers used a number of artificial chemical reaction systems to simulate the ambient photochemical-oxidant situation. These efforts involved a number of irradiated and nonirradiated reaction systems unsaturated hydrocarbon–ozone mixtures, unsaturated hydrocarbon–NO_x mixtures, and dilute auto exhaust). Most research before 1960 involved one or more of these reaction systems. This research has been well reviewed[87,88,114,180,182,184,325,328,451,459,488,505] and thus is not extensively covered here. Although the work is difficult to correlate with research on individual pollutants or combinations of pollutants, many of our present concepts were first enunciated on the basis of these photochemical-oxidant simulation systems. Any researcher concerned with the effects of oxidant pollutants on vegetation or microorganisms should be familiar with this early work.

A number of reviews of varied quality cover general or special effects of photochemical oxidants on vegetation (Table 11-1). Thomas[459] fairly comprehensively covered the available information on the effects of photochemical oxidants on plants. Middleton[325] gave the first comprehensive coverage of the phytotoxic effects of photochemical oxidants in 1961. A number of excellent reviews have appeared since 1961. Rich[406] presented an early review of ozone effects. Dugger and associates[114,120] presented the physiologic and biochemical effects of oxidants on plants. Heck[180] covered factors that influenced the expression of oxidant dam-

TABLE 11-1 Selected Review Articles—A Subject Listing

Subject	References
General	43, 86, 87, 113, 183, 195, 196, 222, 278, 312, 325-328, 348, 356, 451, 459-461, 498, 499, 504, 516, 529
Criteria documents	279, 505
Symptoms and susceptibility	184, 204, 425, 457, 459
Photochemical oxidants	87, 184
Ozone	204, 348, 406, 500
PAN	348, 360, 457
Physiology and biochemistry	113, 114, 120, 348
Effects of various factors	170, 180, 399
Plants as monitors	25, 182
Glossary of terms	154

age. Heagle[170] discussed oxidant problems with respect to interactions between air pollutants and plant parasites. Three chapters in *Recognition of Air Pollution Injury to Vegetation: A Pictorial Atlas*[184,204,457] stressed components of the photochemical-oxidant complex in terms of symptom development and relative susceptibility of plants. Reinert *et al.*[399] presented an excellent treatment of interactions between several of the photochemical-oxidant pollutants and sulfur dioxide. The American Phytopathological Society published a glossary of terms that should be useful to any research worker.[154] In addition, several recent review articles have discussed the effects of photochemical oxidants or ozone on vegetation.[183,196,451,500] Three recent books on air pollution effects on vegetation covered subjects of concern to anyone interested in the photochemical oxidants.[113,348,356] There are two criteria documents, one for the Canadian Ministry of the Environment in 1975[279] and the other for the former U.S. National Air Pollution Control Administration in 1970.[505] Both focus on the development of criteria useful for the setting of ambient air quality standards. These two reviews pertain primarily to dose-response effects and attempt to define threshold dose or specific concentration and time combinations that produce specific response measures. Neither document contains sufficient information on microorganisms to enable the development of useful dose-response information. The Canadian review[279] is generally more comprehensive than the U.S. document,[505] in that it presents a threshold dose-response curve for use with ozone and, by implication, with ambient oxidant measurements.

RESPONSES OF VASCULAR PLANTS

Injury to vegetation from photochemical oxidants was first characterized in 1944[329] as a glazing, silvering, or bronzing of the lower leaf surfaces of broadleaved plants. These symptoms were first identified in the Los Angeles area, but were soon recognized over a large segment of southern California and in the San Francisco Bay area.[327] Photochemical oxidants are known to injure plants in most, if not all, major metropolitan areas of the United States, Canada, and Mexico and probably affect vegetation in major metropolitan areas throughout the world.

Ozone is generally recognized as the most important phytotoxicant in the oxidant complex. Early studies were reported in 1864.[264] The phytotoxicity was firmly established in 1914,[251] and this was confirmed in laboratory studies in 1937.[217] It was first shown as a phytotoxic component (causing grape stipple) of the oxidant complex in 1958[413] and later as the cause of weather fleck of tobacco.[198] Ozone is now known to cause injury to a multitude of broadleaved plants and to explain several types of needle injury in both eastern and western conifer species.

The peroxyacylnitrates were first identified in 1961[442] as the primary cause of the undersurface glazing and bronzing on some broadleaved plants. PAN has since been implicated in numerous pollution episodes in southern California. Its importance in other metropolitan and rural areas in the United States and throughout the world is uncertain, although symptoms have been reported elsewhere.[385] The reason is that oxidant concentrations in other parts of the world are much lower than those reported in California, and monitoring networks have not looked for PAN or its homologues. The report by Penkett *et al.*[392] was the first to come from Europe.

Research on photochemical oxidants, including ozone, was confined primarily to California during the 1950's. By 1959, ozone was known to be an important pollutant in the eastern United States and southern Canada. It is now known as a ubiquitous pollutant and has been widely studied throughout the United States and Canada. By 1972, several other countries had recognized the potential effects of photochemical oxidants on vegetation and initiated research.

Three areas of methodology stressed here are of special concern for the study of air pollution effects on vegetation: growth of the test organisms, exposure facilities, and instrumentation. First, to determine the effects of air pollutants on many plant species, one must have a good understanding of the best cultural conditions for a given test crop; some results reflect the use of poor test specimens. Second, dynamic

airflow designs are required in chamber construction and exposure conditions must be similar to those that occur in the field. The method of preference would be a dynamic, single-pass system,[188] to avoid reactions of reactive oxidants with materials of chamber construction or emanations from the plants. Several useful chamber designs have been reported in the literature.[21,172,202,292,527] For routine experiments, temperature, light, and relative humidity should approach ambient conditions, and nutrient status should be conducive to good growth. Third, one should not assume instrument accuracy without adequate calibration, either initially or later. Most plant scientists have used the Mast oxidant instrument to measure ozone and ambient oxidants. One should be aware of the inherent variability among instruments and routinely calibrate them. Calibration of all instruments should be by the EPA standard 1% neutral potassium iodide method, and the values should be so reported. When this is not done, the actual values may be 50–100% greater than the reported values. Most papers do not state the calibration procedure, if any, and some do not mention the monitoring instrument. Investigators who use the Mast instrument over long periods without an adequate maintenance program may also report erroneous values. Calibration and instrumentation problems are reviewed in Chapter 6. For comparison, ozone at 1 ppm is equivalent to ozone at about 1,960 $\mu g/m^3$.

The foliar response of plants is discussed in terms of the visible or subtle effects on individual plants. Visible effects may be defined as identifiable, pigmented, chlorotic, or necrotic foliar patterns that result from major physiologic disturbances. Subtle effects do not produce visible injury, but may include transitory metabolic disturbances, such as changes in rates of respiration, photosynthesis, transpiration, and enzymatic processes. Subtle effects may be measurable in terms of growth or long-term biochemical changes. Both visible and subtle effects are induced by physiologic and biochemical changes in the plant. Subtle changes may also affect reproductive or genetic systems. If cumulative changes occur within individual plants as a result of visible or subtle effects, these changes may affect plant populations and communities, and this could have adverse effects on ecosystems.[168,501] Community and ecosystem responses are discussed and quantified in Chapter 12.

In general, the direct response of plants to all oxidant pollutants has been in the foliage. In several cases of long-term exposure (grapefruit after several months of exposure to ambient oxidants) or high ozone concentration over a shorter period (apple,[332] peach[436]), injury has been reported on fruit itself.

Visible Symptoms

Visible symptoms are useful in characterizing the response of vegetation to a variety of stresses, including air pollution.[499] Similar symptoms, induced by different stresses, have been well described and may confuse the diagnosis.[52,201,204] On the basis of studies to date, diagnostic techniques that use indicators other than visible injury (e.g., respiration) are less reliable than the subjective judgment of an experienced observer. Thus, it is important that researchers become familiar with injury symptoms caused by oxidants and those caused by other stresses. This section briefly describes the injury symptoms used for diagnostic purposes.

The terms "acute injury" and "chronic injury" are often confused with "acute exposure" and "chronic exposure." Their historical use and the need for brief descriptive terminology suggest that they are still terms of value. Acute injury may affect only a small part of a given leaf. It is a result of destruction of cell contents and always causes cell death. The necrotic patterns in acute injury may be characteristic of a given oxidant. These patterns at least demonstrate the presence of a chemical toxicant. Acute injury is usually associated with short exposures (hours) to specific oxidants or pollutant mixtures at concentrations that cause acute injury and usually appears within 24 h after exposure. Chronic injury, whether mild or severe, is usually associated with long-term or intermittent exposures to low concentrations of oxidants that do not produce acute injury. Normal cellular activity is disrupted, and the chlorosis or other color or pigment change that follows may eventually cause cell death. Chronic oxidant injury patterns are generally not characteristic and may be confused with symptoms caused by normal senescence, biotic diseases, insects, nutritional disorders, or other environmental stresses. These patterns may appear as early leaf senescence with or without leaf abscission. Repeated short-term exposures to ozone may cause physiologic changes that are responsible for chronic symptoms. However, such exposures may produce small but additive amounts of acute injury that are mistakenly referred to as chronic injury.

Before the identification of ozone and PAN as two major phytotoxic oxidants in photochemical air pollution, simulated "smog" (a variety of chemical mixtures) and ambient pollution were the test atmospheres used. The most complete morphologic and developmental studies were conducted during this time by Bobrov (Glater) and associates.[30-33,52,153] These were classic studies that covered a range of plant species, including table beet, annual bluegrass, oat, and tobacco. From these early studies has come our basic understanding of microscopic

changes and developmental patterns. These workers first suggested that membrane injury may have led to the initial effects on the chloroplasts. They also defined developmental patterns and showed that leaves near maximal expansion were more sensitive than young leaves (or leaf tissue) or older leaves. They suggested that maximal sensitivity was related to stomatal function, volume of intercellular spaces, and the extent of suberization of mesophyll cells. Other work has since substantiated their studies.[130,319,466,469,477]

Bystrum *et al.*[52] first described morphologic changes in leaf surface waxes of table beet exposed to photochemical oxidants; these changes were different from those associated with aphid feeding. Comparison of oxidant injury with that produced by insects has since received attention from Hibben,[201] who found that ozone injury to the leaves of four tree species produced smaller flecks, randomly spaced and darker than fleck injury along veins induced by a mesophyll-feeding leafhopper.

Although ozone and PAN are considered the two primary phytotoxic oxidants in the photochemical complex, the specific response of plants to many simulated atmospheres suggests the existence of other phytotoxic oxidants.[182] The symptoms associated with many of these reactant mixtures are closely related to those caused by ozone and PAN.[181,184] In some tests, the mixtures used would not have produced either ozone or PAN. In other cases, leaf age or the pattern of injury on sensitive test plants suggested one or more pollutants other than ozone or PAN. Field injury symptoms often resemble those reported for ozone or PAN, but the response pattern is sufficiently different that accurate diagnosis is difficult. Brennan *et al.*[42] correlated development of oxidant symptoms with aldehyde concentrations in New Jersey and suggested that aldehyde may be a major phytotoxic component of the photochemical-oxidant complex. The symptoms were probably not responses to the aldehyde, but rather to some compound or group of compounds present under the same conditions as the aldehyde.[208]

The most complete description of ozone injury symptoms is found in the *Atlas*.[204] However, several of the review articles, including the two criteria documents,[279,505] have injury descriptions. A concise description is found in Heck and Brandt.[183]

The classic ozone symptoms on angiosperms are the upper surface fleck of tobacco[198] and stipple of grape.[413] These classic symptoms were described in plants with differentiated mesophyll and are especially significant, because they were identified with palisade cells, and not with spongy cells.

The two classic symptoms are still more widely associated with the

response of dicotyledonous plants to ozone than are other symptoms. Many plants (e.g., pinto bean, cucumber, tomato, soybean, and sycamore) may have the entire upper surface covered with a bleached appearance as a result of ozone exposure, with no observable injury on the lower surface. On closer examination, the bleached area is seen to be made up of many small groups of palisade cells that are dead and contain no pigment. In other plants or under different conditions, the palisade cells may accumulate dark alkaloid pigments (stipple) coincidentally with cell death. After exposure to higher ozone concentrations or after longer periods of exposure, injury extends to the spongy cells, producing bifacial necrosis. Plants exposed to a high concentration of ozone or to a high concentration of ambient pollution during a pollution episode usually develop dark water-soaked areas in the leaf within a few hours. Leaves may show partial recovery, or these areas may form light-tan bifacial necrotic lesions within 24–48 h. Individual lesions may be small, but groups of them can extend and affect a considerable portion of the leaf.

In monocotyledonous plants (grasses and cereals) and some others, there is no division of mesophyll tissue, and injury normally appears as a bifacial fleck.[32] Some plants, after extended exposure to low concentrations of pollution (either continuously or intermittently), produce chlorotic patterns that may be distinctive of oxidant pollution or similar to symptoms of normal senescence. The early senescence seen in some plants may be a result of long-term exposure to ambient oxidants.

The foregoing discussion is not descriptive of effects noted in coniferous trees, such as pine. It is worth discussing two classic oxidant (ozone) syndromes of pine—one eastern, the other western. Ozone is the probable cause of emergence tipburn in white pine (white pine needle dieback).[24] The injury is characterized as a tip dieback of newly elongating needles and occurs throughout the range of eastern white pine. Affected trees are found at random in a stand, and symptoms develop in discrete episodes in successive years. Primary roots of affected trees often die after repeated needle injury. Costonis and Sinclair[72] reported silvery or chlorotic flecks, chlorotic mottling, and tip necrosis of needles as results of ozone exposure. Ozone may be associated with semimature-tissue needle blight (SNB),[276] but Linzon[275] found that both SNB-sensitive and SNB-tolerant white pine were first injured with ozone at about 0.6 ppm for 2 h, and the symptoms were not like SNB. Linzon[275] found that ozone injury began in young tissue with newly functional stomates, whereas in SNB[277] the necrosis begins in semimature leaf tissue and then spreads through the older needle

tissue toward the tip. Needle injury resulting in a disease called "chlorotic decline" of ponderosa pine was first noticed in 1953 and was related to oxidant air pollution by 1961.[382] The chlorotic decline was characterized by a progressive reduction in terminal and diameter growth, retention of only the current season's needles, reduction in number and size of these needles, yellow mottling of the needles, deterioration of the primary roots, and eventual death of the tree. The chlorotic decline was not associated with stresses other than ozone.[383] These symptoms were reproduced by exposing ponderosa pine to ozone at 0.5 ppm for 9 h/day for 9–18 days.[339] Similar symptoms were noted by Richards *et al.*,[414] who called the disease "ozone needle mottle of pine." Evans and Miller[131–133] and Miller and Evans[336] have conducted histologic investigations on injury development in ponderosa pine. They found that chlorotic needle mottle caused by ozone or ambient oxidants was easily distinguished from "winter fleck."[336] A comparison of needle anatomy in four pines of differing sensitivity suggested that sensitivity was related to the number of mesophyll cells per stomate.[131] They also reported an accumulation of chloroplasts and carbohydrate stain in peripheral portions of mesophyll cells of ponderosa needles before development of visible injury that was not related to stomatal presence.[133] These changes were not shown sufficiently to be specific for ozone effects.[132]

Injury from PAN was first described as smog injury by Middleton *et al.*[329] on spinach, garden beet, romaine lettuce, and chard. They reported a collapse of spongy mesophyll tissue with the development of large air pockets, especially near the stomates. The air spaces gave the leaf the appearance of a glaze and were responsible for this classic PAN symptom. Most of the descriptive work reported by Bobrov and associates[30–33,52,153] from simulated atmospheres and ambient exposures resulted from PAN injury. The classic injury pattern for PAN is glaze followed by a bronzing of the lower leaf surface. Young expanding leaves are normally more sensitive to PAN than more mature leaves. Physiologic maturity is important, as shown by the banding appearance on many susceptible leaves that do not mature uniformly. If the whole leaf has a similar physiologic age, it may be uniformly injured (e.g., pinto bean). In case of severe injury, bifacial necrosis can occur. Early senescence and leaf abscission are often associated with PAN injury. The most complete description of injury is that by Taylor and MacLean.[457] Symptoms associated with PPN and PBN are similar to those for PAN.

In several cases, symptoms have been associated with dose.[360,452,498]

At a high dose (0.5–1.0 ppm for 0.5 h), sensitive plants show complete collapse. As the dose is reduced, the plants may show necrotic banding, then bronzing and glazing with slight collapse. At 0.1 ppm, chlorosis is the major symptom. In some unpublished work,[360] an upper-surface light-yellow-to-white stippling has been described on alfalfa after exposure to PAN at 0.02–0.1 ppm for 2–6 h. The injury was intercostal on the tip of young leaves and on the base of old leaves. Drummond[112] reported a necrotic fleck and an upper-surface stipple on petunia after 1-h exposure to PAN at 0.15 ppm.

Physiologic and Biochemical Effects: Mechanism of Action

Many reports on the effects of ozone and PAN on physiologic processes (net photosynthesis, stomatal response, and water relations) and on metabolic activity (including *in vivo* and *in vitro* studies of individual enzymes, enzyme systems, metabolic pathways, metabolic pool relationships, cell organelles, and plant tissue studies) have appeared since 1964.

Stomata are the principal entry sites for ozone and PAN into plant leaves, and stomatal closure effectively protects the plant from injury.[306] Several studies suggest that oxidants may cause stomatal closure.[230,255,286,411]

Stomatal closure was associated with a genetic factor in onion wherein the stomata of sensitive plants did not close.[126] The effect of ozone and PAN on stomatal opening depends on many interacting factors; those representing water stress appear to be the most important. Dean[100] related stomatal density to the difference in sensitivity between two tobacco cultivars. Evans and Ting[135] found that maximal sensitivity of bean primary leaves was not associated with changes in stomatal number or leaf resistance. Ozone exposure caused a decrease in relative water content, but no change in resistance. Bean leaf sensitivity seemed more a function of internal activities.

Isolated enzymes and enzyme systems are affected by exposure to PAN and ozone. It is now recognized that strong oxidants interfere with various oxidative reactions within plant systems. Metabolic pools, including nitrogen and carbohydrate, appear to have some controlling influence on the response of plants to these pollutants. Sulfhydryl groups are primary targets of oxidant pollutants and may be a key to understanding the mechanism of action. Unsaturated lipid components of some membranes are sites of early action of both ozone and PAN.

OZONE

Ozone is the most nearly ubiquitous of the photochemical oxidants, at least in concentrations that cause measurable effects. It has been studied more extensively than the other oxidants.

Physiologic Effects The physiologic effects of ozone depend on its entry into the internal leaf spaces through the stomata. If the plant is resistant to ozone even when stomata remain open, mechanisms of resistance other than stomatal closure must be operative. The physiologic effects measureable with the intact tissue include effects on respiration and photosynthesis.

Todd[486] reported that the respiration of pinto bean leaves was stimulated by exposure to ozone (at 4 ppm for 40 min). The first measurements were 4 h after the ozone exposure. The respiration rate later declined to the control value. In all cases, increased respiration correlated well with visible injury. Macdowall[283] confirmed these results, but made an additional observation: during the first hour after ozone exposure (at 0.7 ppm for 1 h), and before visible symptoms appeared, respiration was inhibited. The increase in respiration took place only later, when visible symptoms appeared. Dugger and Palmer[115] reported an increase in respiration in lemon leaf tissue after 5 days of exposure to ozone at 0.15-0.25 ppm for 8 h/day. They reported no morphologic changes at that time. Anderson and Taylor[10] found that ozone induced carbon dioxide evolution in tobacco callus tissue. The threshold for evolution was about 0.1 ppm for 2 h in the sensitive Bel W_3. The ozone concentration required for maximal carbon dioxide evolution was about twice as much in the more resistant cultivar. Formation of roots decreased sensitivity.

Todd[486] and Todd and Probst[489] also measured the effects of ozone (at 4 ppm for 40 min) on photosynthesis and found that development of symptoms was associated with inhibition of carbon dioxide fixation. This effect was also confirmed by Macdowall,[283] who reported that the inhibition of photosynthesis was greater than that which could be accounted for by chlorophyll destruction. Hill and Littlefield[205] associated decreased net photosynthesis caused by ozone (at 0.06 ppm for 1 h) with both stomatal opening and rates of transpiration. These studies have generally shown that net photosynthesis can decrease without visible injury.

Pell and Brennan[387] found an initial decrease in net photosynthesis and an increase in total adenylate concentration after a 3-h exposure to an injurious concentration of ozone. Net photosynthesis returned to normal within 24 h. Respiration was usually not immediately stimu-

lated, but it was within 24 h. The authors concluded that the stimulation of respiration was a consequence of cellular injury, whereas the changes in photosynthesis and adenylate content were early events leading to the appearance of ozone injury.

The photosynthetic rate is an important indicator of vigor in ponderosa pine. A reduction in this rate may occur at the threshold dose of ozone without visible symptoms. Miller *et al.*[338] found that a daily 9-h exposure to ozone at 0.15 ppm reduced apparent photosynthetic rates by 10% after 30 days, without typical ozone symptoms. Botkin *et al.*[34] found that a threshold ozone dose for suppression of net photosynthesis in eastern white pine was a 4-h exposure to 0.50 ppm.

Ozone causes both quantitative and qualitative changes in carbon dioxide fixation patterns. Wilkinson and Barnes,[522] using carbon dioxide-^{14}C, found a reduction in radioactivity in soluble sugars and increases in free amino acids and sugar phosphates in white pine after a 10-min exposure to ozone at 0.10 ppm. Miller[333] observed a decrease in carbon dioxide-^{14}C fixation in ponderosa pines that correlated with loss of chlorophyll, after exposure to ozone at 0.30–0.35 ppm. The Hill reaction rates of chloroplasts isolated from healthy and ozone-injured ponderosa pine indicated that both light and dark reactions of the chloroplasts from ozone-injured plants were depressed. Barnes[12] found depressed photosynthesis and stimulated respiration in seedlings of four pine species of the southeastern United States after exposure to ozone at 0.15 ppm.

It is commonly observed that leaves exposed to ozone develop dark green areas—sometimes referred to as a "waterlogging" effect. If symptoms develop, they will be in these "waterlogged" areas, but symptoms are not always a consequence of the "waterlogging." In other words, there is sometimes recovery from this initial effect. The effect is due to leakage of cell contents into the intercellular spaces and, to some extent, is reversible. Many experiments with radioactive inorganic ions and organic substances have confirmed that plant cell permeability is affected by exposure to ozone.[134] Perchorowicz and Ting[393] found that, immediately after ozone exposure (at 0.4 ppm for 1 h), there was no uptake of glucose-^{14}C, but the uptake slowly increased for a period of hours. However, the metabolism of the glucose into various products (organic acids, lipids, carbon dioxide) was not changed by the ozone exposure.

Although it is widely believed that the effects of ozone on cell permeability are the basis of ozone toxicity,[472] the chemical basis of the effects on membranes is still arguable. It is still not clear whether the first effect of ozone is on the protein or lipid components of the cell.

Dugger *et al.*[116] noted that the susceptibility of pinto bean primary leaves to ozone (at 0.3–0.7 ppm for 25–90 min) is maximal at a leaf age of 10 days, when the starch content is high and that of soluble carbohydrate is low. They found that "starvation" of the leaves, by placing plants in the dark for 48–72 h, depleted the carbohydrates and conferred resistance. Susceptibility was regained by supplying sucrose through the petiole. However, susceptible leaves could be made resistant by supplying more sucrose through the petiole. They concluded that the leaf was resistant when the soluble-sugar content was either above or below the range of 1.0–4.0 mg/g of fresh weight. These results were confirmed by Lee,[268] who concluded that the protective action of high sugar content could be partly, but not completely, explained by causing closure of stomata (ozone at 0.8 ppm for 5 h). Barnes and Berry[14] reported seasonal changes in the soluble sugars of white pine that may relate low sugar contents to a prolonged sensitivity to ozone. Hanson and Stewart[163] found that several species exposed to noninjurious concentrations of ozone retained higher starch concentrations in their tissue after 12–36 h of darkness.

Macdowall[282] found that tobacco leaves were most susceptible to injury by ozone (at 0.035 ppm for 5 h) just after full leaf expansion. This point corresponded to the beginning of the decline in protein content. Lee[266] modified the nitrogen content of tobacco leaves by supplying urea and found a positive correlation of injury caused by ozone (at 1 ppm for 5 h) with nonprotein nitrogen, but not with protein. This result is in contrast with that of Ting and Mukerji,[471] who found that, in cotton leaves (in which the period of maximal susceptibility was at about 75% of full leaf expansion), the amino acid pool was low at the time of maximal susceptibility. However, ozone treatment (0.7 ppm for 1 h) increased the free amino acid pool.

It is currently impossible to give a rational explanation of the relationship between ozone injury and carbohydrate and nitrogen composition of the leaf. The compounds measured (protein and carbohydrate) may be only distantly related to metabolites that confer resistance or susceptibility.

It seems reasonable that susceptibility and resistance of different plant species and different varieties within a species should depend on concentrations of endogenous antioxidants. The results of such studies do not give a clear picture: Hanson *et al.*[165] concluded that the range of susceptibility in petunia varieties depended on the ascorbic acid concentration, but Menser[314] found that the ascorbic acid content of tobacco varieties was not related to ozone susceptibility. Ozone resistance of plants can be conferred by application of antioxidants. In the

case of white bean, ascorbic acid, cysteine, glutathione, and nickel *N*-dibutyldithiocarbamate were effective.[91,147] Field studies with tomato and tobacco showed that a number of antioxidants were effective protectors: manganous 1,2-naphthoquinone-2-oxime, cobaltous 8-quinolate, nickel *N*-dibutyldithiocarbamate, and *N*-isopropyl-*N*-phenyl-*p*-phenylenediamine.[408] These preventives do not yet present an economically feasible practice for agriculture.

Biochemical Effects It is usual to find some changes in the chemical composition of plant tissue after exposure to ozone. One cannot be certain whether the changes are associated with early reactions to ozone or are merely delayed consequences of cell injury.

A number of histologic and histochemical changes in current-year needles of ponderosa pine were detected after five to seven daily exposures to ozone at 0.45 ppm for 12 h each day.[133] Chloroplasts and carbohydrate stain accumulated in the peripheral portions of mesophyll cells; concurrently, the homogeneous distribution of proteins and nucleic acids was disrupted, and acid phosphatase activity increased. Cell wall destruction occurred in mesophyll cells after appreciable intracellular damage.

Howell[223,224] and Howell and Kremer[227] found phenol accumulation associated with the exposure of plants to ozone. Menser and Chaplin[317] also reported an increase in the phenol content of tobacco leaves injured by ambient oxidants. Hoffman[216] reported as much as an 80% increase in nicotine in leaves exposed to ozone at 0.24 ppm for 4 h. No increase was found after exposure to 0.06 or 0.12 ppm. Keen and Taylor[240] found accumulation of several isoflavonoid compounds in soybean (Harosay 63) exposed to ozone. The presence of these compounds suggests a defense mechanism against ozone that is similar to that for disease resistance.

Tomlinson and Rich[493] reported an increase in γ-aminobutyric acid in bean leaves after exposure to ozone (at 1 ppm for 30 min). In a more comprehensive study, Ting and Mukerji[471] repeated this finding in cotton leaves, but found increases in many other amino acids. The only decreases found were in phosphoserine, phosphoethanolamine, ethanolamine, phenylalanine, and alanine. There are discrepancies in the literature regarding the time after ozone treatment before detection of changes in amino acid composition. Ting and Mukerji[471] found the increase only after 24 h, whereas Tingey *et al.*[478] reported the change to be immediate. Similar differences concern soluble protein: Tingey *et al.*[478] noted a rise after 24 h, whereas Craker[78,81] found a decline in protein content. As far as specific proteins are concerned, both Tingey *et al.*[478] and Leffler and Cherry[269] found a decrease in nitrate reductase;

Yamaga *et al.*[528] reported a decrease in carbohydrase. Dass and Weaver[90] found significant increases in peroxidase and cellulase, but no change in lactate dehydrogenase; Curtis and Howell[82] reported an increase in peroxidase isoenzyme activity. Tingey has also reported an increase in the G-6-PD activity.[473] It is possible to fit the latter change into a rational scheme for the effects of ozone, but "rational" is not synonymous with "right." Chang and Heggestad[63] found that ozone at 0.35 ppm for 50–80 min impaired the activity of photosystem II in spinach and reduced the β-carotene of the chloroplasts. Coulson and Heath,[74] using isolated chloroplasts, found an inhibition of both photosystems and no penetration of the grana. They suggested that in the intact plant ozone affects only the first membrane contacted.

Chang[60–62] has made observations on the polysomes of pinto bean leaves exposed to ozone (at 0.35 ppm for 20–50 min). He found that the chloroplast polysomes were more susceptible to oxidation than was the cytoplasmic ribosomes. The sulfhydryl content of the chloroplast ribosomes was also much more susceptible to oxidation than was that of the cytoplasmic ribosomes. Finally, it was found that the effects of ozone on ribosome composition could be reproduced by *p*-mercuricbenzoate. Chang's results imply that either ozone itself or a product of ozone oxidation passes from the cytoplasmic membrane to the interior of the chloroplast before having its effect. These results connect with a number of papers on the oxidation of sulfhydryl compounds by ozone. Tomlinson and Rich[495] have reported decreases in leaf sulfhydryl groups after ozone exposure (at 1 ppm for 30–60 min). A number of investigators have found that reagents that react with sulfhydryl groups can reproduce the symptoms of ozone damage. Dass and Weaver[91] reported this effect in white bean. Tomlinson and Rich[495] reported the same effect in tobacco, and Rich and Tomlinson[410] found that conidiophores of *Alternaria solani* became more susceptible to ozone if they were pretreated with iodoacetamide. It appears that effects on sulfhydryl content may be quite early events in the toxic reactions initiated by ozone. One should remember, however, that symptoms of ozone and PAN injury are quite different, and one cannot explain the effects of both pollutants as sulfhydryl oxidizers.

Tomlinson and Rich[492,494] have also considered the reaction of ozone (at 1 ppm for 30 min) with lipid components of the leaves. They found that a change in the fatty acid composition as a result of ozone treatment was an increase in the saturated fatty acid content. It was concluded that malonaldehyde (an index of lipid peroxidation) was formed only in the later stages of ozone damage. Frederick and Heath[145] reported that the production of malonaldehyde is correlated

with loss of viability of *Chlorella sorokiana* exposed to ozone (at 1.8 ppm for 5-20 min), but with a lag after the initiation of ozone exposure. Swanson *et al.*,[445] in agreement with Tomlinson and Rich, found only small changes in the fatty acid composition of tissue exposed to ozone (at 0.3 ppm for 2 h). It is not possible to conclude from the available data that oxidation of lipid is the primary effect of ozone exposure. Tomlinson and Rich[491] have examined the effects of ozone on the complex lipids of plants. Plants were exposed to ozone at 0.25 ppm until the first symptoms were seen (2.5-3.0 h), and leaf solubles were extracted. A decrease in free sterol and increases in both sterol glucoside and acylated sterol glucoside were found.

Tingey *et al.*[478] found an increase in reducing sugars soon after the exposure of soybean plants to ozone. In experiments with ponderosa pine, Miller *et al.*[338] found that the polysaccharide content of both current and 1-yr needles, treated for 33 days with ozone at 0.30 ppm, was lowered by 40%, but there was a slight increase in soluble sugars. No relationship with absorbic acid was found. Barnes[13] reported studies with five species exposed to ozone at 0.05 ppm for 5-22 weeks. There were significant increases in total soluble carbohydrates, reducing sugar, and ascorbic acid. At 0.15 ppm, the effect on ascorbic acid was not observed. These results do not lead one to believe that ascorbic acid plays an important role in ozone resistance.

Nobel and Wang[363] reported that the permeability of the outer membranes of chloroplasts was increased by exposure to ozone (at 30 ppm for 5 min). They hypothesized that the effect was lipid oxidation. Freebairn[146] showed that ozone inhibited respiratory activities of isolated mitochondria, and Lee[267] found that the effect of ozone on oxidative phosphorylation was greater than on oxygen uptake. Mudd *et al.*[353] reported that the metabolism of UDP-galactose by isolated chloroplasts was inhibited by ozone (at 1,000 ppm for 2 min). There were differential effects on the synthesis of various galactolipids, the synthesis of monogalactosyldiglyceride being relatively resistant. This differential effect of ozone could be reproduced by chemicals that react with sulfhydryl groups.

There is some information concerning the reaction of ozone with chemicals under aqueous conditions. The information available suggests that double-bond cleavage takes place, just as it does under nonaqueous conditions, except that ozonides are not formed. Instead, the zwitterionic intermediate reacts with water, producing an aldehyde and hydrogen peroxide. In addition to double-bond cleavage, a number of other oxidations are possible. Mudd *et al.*[350] showed that the susceptibility of amino acids is in the order cysteine, tryptophan, methionine,

histidine, tyrosine, phenylalanine, and cystine (ozone at 2,000 ppm for 2–10 min). All other amino acids are resistant. Todd[485] tested the susceptibility of catalase, peroxidase, urease, and papain to ozone. Papain was the most susceptible; this result is consistent with the knowledge that a sulfhydryl group is necessary for its activity. The inactivation of avidin[350] and lysozyme[270] can be attributed to the oxidation of tryptophan residues, although, in the case of lysozyme, methionine is also partially oxidized. Enzyme inactivation caused by ozone reaction with histidine has been indicated in the case of pancreatic ribonuclease.[350] The reaction of nicotinamide derivatives with ozone has been studied by Mudd *et al.* (ozone at 0.4–800 ppm for 1–100 min).[351] The reduced forms are quite susceptible, and the oxidized forms quite resistant. The adenine moiety is quite resistant. When NADH is oxidized by ozone, the 5,6 double bond is broken, so the coenzyme is no longer biologically useful.

Studies of the reaction of ozone with simplified lipid systems have shown that malonaldehyde can be produced by direct ozonolysis.[352] The use of malonaldehyde assay as an index of lipid peroxidation is therefore invalid in ozone studies. Liposomes formed from egg lecithin and prepared in aqueous media were quite resistant to ozone, but the contribution of polyconcentric spheres to this resistance has not been fully assessed.[353] However, the bilayer configuration, with the susceptible unsaturated fatty acids shielded from ozone by the hydrophilic areas of the molecule, may be resistant. In hexane, where the fatty acid moieties are exposed, ozone reacts stoichiometrically with the double bonds. The experiments with aqueous suspensions of phosphatidylcholine gave no evidence of the formation of lipid peroxides,[458] nor did experiments with films of fatty acids exposed to ozone.[417]

Chemical Basis of Toxicity There is only one comprehensive theory for the action of ozone on biologic organisms—the theory of Chow and Tappel[64] that the initial event is the formation of lipid peroxide and that successive events are an attempt to detoxify this product. The theory was developed from experiments with animals that showed that exposure to ozone increases malonaldehyde, gluthathione peroxidase, glutathione reductase, and G-6-PD:

$$\text{lipid} \xrightarrow{O_3} \text{lipid peroxide (ROOH)},$$

$$\text{ROOH} + 2\text{GSH} \xrightarrow{\text{GSH peroxidase}} \text{ROH} + \text{GSSG},$$

$$\text{GSSG} + \text{NADPH} + \text{H}^+ \xrightarrow{\text{GSSG reductase}} 2\text{GSH} + \text{NADP}^+,$$

$$\text{NADP}^+ + \text{G-6-P} \xrightarrow{\text{G-6-P dehydrogenase}} 6\text{PG} + \text{NADPH} + \text{H}^+.$$

This theory is questionable on a number of points: there is no direct evidence of formation of lipid peroxide; GSH can be oxidized directly by ozone or used to reduce hydrogen peroxide produced from ozone reactions; and the increased activity of G-6-P dehydrogenase may be a response to the oxidation of NADPH.

PEROXYACYLNITRATES

The peroxyacylnitrates are the most phytotoxic of the known photochemical oxidants. The toxicity of the known homologues is inversely correlated with molecular weight. PAN is the best known and has been studied most, because of its usual presence in photochemical atmospheres.

Physiologic Effects Leaf age and illumination have been studied as physiologic and physical factors that affect the response of plants to PAN.[117] Leaves of most plants are most susceptible when very young. In primary leaves of pinto bean, the period of maximal susceptibility to PAN precedes that for ozone by several days (about 5–7 days from seed for PAN, as opposed to 9–13 days for ozone). The example of tomato is particularly striking, inasmuch as the terminal leaflet of the compound leaf is the oldest tissue. Thus, the youngest susceptible leaf is affected in the terminal leaflet; but, as the leaves become older, the terminal leaflet is resistant and the lateral leaflets are injured.[457]

There is no reason to believe that stomatal opening is different in leaves of different ages, so the entry of PAN into the intercellular spaces of the leaf is probably the same as the entry of ozone. One can hypothesize that the chemical composition of the leaf is such that resistance is conferred at particular physiologic states. Such a hypothesis would lead to a search for compounds that combat the oxidizing and acylating properties of PAN.

There appears to be an absolute requirement for illumination before, during, and after fumigation with PAN (1 ppm for 30 min), if symptoms are to be observed.[456] If the prefumigation light period is followed by 15 min of darkness before a 30-min exposure to PAN in the light, no symptoms are observed. Thus, at this time, a short dark period before PAN exposure prevents damage. However, the dark period after fumigation must be 1–2 h long, if damage is to be avoided. The light period after fumigation must be 3 h long, if damage is to be maximal.[452] These effects of illumination are not understood.

The action spectrum for the light requirement shows maximal quantum responsivity at 420 and 480 nm and low responsivity at wavelengths characteristic of chlorophyll (PAN at 4 ppm for 15 min).[118] The action

spectrum suggests participation of either carotenoids or flavins. It should be emphasized that the action spectrum for illumination was determined only during fumigation. It is possible that a different photoreceptor is involved in the periods before and after exposure. Photosynthesis of intact cells of *Chlamydomonas reinhardi* was inhibited by PAN (at 125 ppm for 5 min).[155] Photosynthesis recovered after the exposure, but the recovery varied with the light quality used during the exposure. Recovery was less when the irradiation was at 450 nm; this is roughly consistent with the work on the action spectrum in pinto bean leaves.[118]

Although the quantum responsivity is low at 660–700 nm, effects of irradiance at these wavelengths are noticeable. In a comparison of the effects of light at 660 and 700 nm, injury was greatest when plants were irradiated at 660 nm during exposure and lowest when they were exposed either in darkness or at 700 nm (PAN at 1 ppm for 15 min).[121] The most striking observation was that simultaneous exposure to light at 660 and 700 nm resulted in little injury. These results were interpreted as effects on photosynthetic systems I (activated at 700 nm) and II (activated at 660 nm), but effects of these wavelengths on phytochrome transformation should not be overlooked:

$$P_r \underset{700\text{ nm}}{\overset{660\text{ nm}}{\rightleftharpoons}} P_{fr},$$

where P_r is the red form of phytochrome and P_{fr} is the far-red form. It is noticeable that the action spectrum for the PAN effect is similar to that for phytochrome and for some physiologic responses mediated by phytochrome.[51,341] Interaction of PAN with the phytochrome system seems worthy of systematic study. The light effects may also be related to the sulfhydryl content of plants, inasmuch as illumination increases and exposure to PAN (at 1 ppm for 15 min) lowers the sulfhydryl content.[121]

It has been suggested that sulfhydryl compounds are susceptible to reaction with PAN (by oxidation or acetylation), and thus that conditions that increase the sulfhydryl content will increase susceptibility. This explanation for PAN injury is consistent with the finding that inhibition of photophosphorylation by *N*-ethylmaleimide, a reagent that reacts with sulfhydryl radicals, is most effective when chloroplasts are treated in the light.[310] In the Calvin and Hatch-Slack pathways of carbon dioxide fixation in photosynthesis, some enzymes are activated by light (e.g., ribulose phosphate kinase),[51] and some are inactivated by light (e.g., glucose-6-phosphate dehydrogenase, G-6-PD).[9] In both cases, the effect of light can be replicated with sulfhydryl compounds, such as

dithiothreitol. The reaction of PAN with the sulfhydryl forms of these enzymes can easily be visualized as upsetting the regulation of carbon dioxide fixation. These considerations are consistent with the effects of PAN on various aspects of photosynthesis (adenosine triphosphate, ATP, formation; nicotinamide adenine dinucleotide phosphate, NADP, reduction; and carbon dioxide fixation) and with the fact that the earliest injury found by the electron microscope was in the stroma of the chloroplasts (PAN at 1 ppm for 30 min).[467]

Biochemical Effects Several enzymes that use nicotinamide cofactors were found to be inhibited by PAN (at 125 ppm for 1 min)[345] in *in vitro* studies. These enzymes were most susceptible in the absence of substrates. In some cases, an enzyme was protected by the nicotinamide cofactor (e.g., G-6-PD plus NADP), and in other cases, by the cosubstrate (e.g., isocitrate dehydrogenase plus isocitrate). Precisely the same protection could be obtained when compounds that react with sulfhydryl compounds (e.g., *p*-mercuricbenzoate) were used instead of PAN. Thus, the evidence indicated that PAN reacted with sulfhydryl groups.

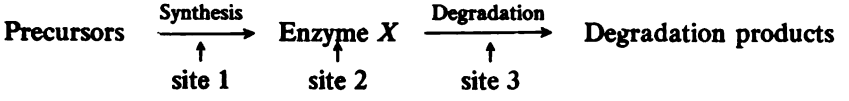
Direct evidence of the reaction of PAN with sulfhydryl compounds has since been obtained (PAN at 115 ppm for 1-10 min).[271,346,349] In the reaction with glutathione, the major products are oxidized glutathione (disulfide) and *S*-acetylglutathione. Other sulfhydryl compounds (e.g., coenzyme A, lipoic acid, and cysteine) yield only oxidation products, with no evidence of *S*-acetylation. However, acetylation reactions have been observed with alcohols[362] and amines.[515] Sulfur compounds other than thiols can undergo oxidation by PAN: methionine is converted to methionine sulfoxide, and oxidized lipoic acid (disulfide) is converted to sulfoxide.

Papain is readily inactivated by PAN (at 115 ppm for 40 min), provided that it is in the sulfhydryl form.[349] The reaction of sulfhydryl groups of hemoglobin with PAN is very similar to the reaction with *p*-mercuricbenzoate: there is more reaction at a pH of 4.5 than at a pH of 7. However, there is one striking difference between PAN and classic compounds that react with sulfhydryl groups: egg albumen is resistant to reaction with PAN.[349] Thus, enzymes that have no free sulfhydryl groups should be quite resistant to PAN. This is the case with pancreatic ribonuclease: the native enzyme was not affected by a 300-fold molar excess of PAN.

There is some difficulty in correlating results of PAN exposure *in vivo* with the above types of *in vitro* studies. For example, UDP glucose pyrophosphorylase is very susceptible to PAN *in vitro*, but *in vivo* there is a stimulation of enzymatic activity (at 112 ppm for 6 min).[161] It is also

anomalous that, whereas phosphoglucomutase of oat seedlings is sensitive to PAN both *in vivo* and *in vitro* (at 50 ppm for 4 h),[161] it is generally considered that the active site is serine, rather than cysteine. Results of enzyme analysis after *in vivo* exposure of tissues to PAN are complicated, because direct effects on the enzyme may be overshadowed by effects on protein synthesis, and these effects in turn may be directly on the protein synthesizing machinery or on the regulatory mechanisms that depress or stimulate protein synthesis.

The schematic below shows that in this dynamic system PAN could affect the synthetic process (site 1), the enzyme itself (site 2), or the degradation process (site 3). If the site of attack were site 2, the synthetic process might compensate for degradation of the enzyme by producing more. If the enzyme activity were measured as a function of time after exposure, there would be first a decrease and then recovery of activity. (Such a response has been observed for the effect of ozone on respiration.) Effects at site 3 would show first an increase in activity and then, if the system were regulated, a decline to normal, as the synthetic process slowed down. Effects at site 1 would cause a decrease in activity commensurate with the rate of enzyme degradation.



Experiments with chloroplasts showed an apparent inhibition of fatty acid synthesis by PAN (at 72 ppm for 10 min).[347] The result is difficult to interpret: the inhibition could be attributed to inactivation of one of the enzymes of the multistep system or to oxidation of the reductant (reduced NADP, or NADPH) required in the chain elongation process.

In addition to the protein and low-molecular-weight thiols that react with PAN, there are several other reactive biochemicals. Reduced nicotinamide derivatives are susceptible to oxidation by PAN (at 72 ppm for 1-5 min),[347] whereas the oxidized forms are resistant. The capability of PAN to oxidize these compounds rapidly dissipates in aqueous solution, with a half-life of 4-10 min, depending on pH. The oxidation products appear to be the biologically active forms of the nicotinamide derivatives. Purines and pyrimidines react with PAN (at 1,000 ppm for 30-120 min).[384] The order of sensitivity is thymine, guanine, uracil, cytosine, and adenine. Their reactions were studied at relatively low pH and at high PAN concentration and are probably not of biologic significance.

Various olefins and amines (undiluted) react with solutions of PAN in $CdCl_3$:[89,515]

$$PAN + R_2C{=}CR_2 \rightarrow R_2C\overset{O}{\diagup\diagdown}CR_2 + MeONO + MeNO_2;$$

$$PAN + RNH_2 \rightarrow CH_3\,CONHR + O_2 + HNO_2.$$

These oxidation and acetylation reactions have not yet been examined in a biologic context.

Chemical Basis of Toxicity The toxicity of the peroxyacylnitrates to plants can be the result of several different processes. Any chemical explanation of the toxicity will have to explain the increasing toxicity of the series PAN, PPN, PBN, and PBzN. Three hypotheses have been tested:[348] the more toxic homologues are more readily taken up by the plant; all homologues are taken up equally well, but the plant is able to reverse the actions of the lower homologues; and all homologues are taken up equally well, but the higher homologues have longer half-lives. In testing the first hypothesis, it was found that PAN and PPN were taken up equally well by plants; so that hypothesis is probably incorrect. The ability of the plant to hydrolyze thioesters of glutathione was tested; the second hypothesis predicted that the higher homologues would be hydrolyzed more slowly; but the opposite was found.[523] Thus, the second hypothesis, although not fully tested (in that the thioesters measured may not be the ones formed in the cell), does not look promising. The third hypothesis was tested by determining the half-lives of the oxidizing power of each compound in aqueous medium. PBN had the longest half-life, as predicted, but the half-lives of PAN and PPN were about the same.[348] It seems probable that half-life influences the toxicity of the peroxyacylnitrates, although definitive results have not yet been obtained.

DISCUSSION

The acute responses of plants to ozone and PAN result from disruption of normal cellular mechanisms. The initial response causes disorganization, perhaps through an attack on sulfhydryl linkages or unsaturated lipid linkages. Cellular water and salts are lost, with cell plasmolysis resulting. Cell death normally will occur; however, depending on dose and environmental conditions, membrane permeability may be restored and cell recovery take place. The extent of recovery depends on the severity of the external stresses and probably on the ability of the cells to initiate repair mechanisms. A sigmoid response has been observed in many plants exposed to ozone and probably reflects physical resistance to gas movement within the tissues. Tingey *et al.*[476] determined activity

changes in selected enzymes from soybean leaves and found that the reactions were similar to those produced by other stresses, and not to the process of senescence.

Chronic injury results primarily from secondary reactions involving membrane injury. The oxidants could cause the formation of free radicals or other, more stable oxidants (such as hydrogen peroxide), which in turn could cause secondary reactions. These secondary reactions could stimulate the production of cellular ethylene, with tissue senescence resulting.[1,77,484] These secondary reactions may predispose plants to increased injury from later acute exposures by limiting their repair capability. This predisposition concept has been noted in several reports.

Reproductive Systems and Life Cycles

Ozone and PAN are both strong oxidizing agents. Some work has suggested that ozone is a radiomimetic gas. It is known to cause chromosomal breakage in both plants and animals at high concentrations. Although the potential of ozone's mutagenic effects on vegetation at ambient concentrations is not understood, Sparrow and Schairer[437] reported that ozone appeared to be a weak mutagen in causing an increase in the pink somatic mutation rates in petals of *Tradescantia.*

Feder[137] showed an effect of ozone on tobacco pollen germination and postulated effects on reproductive capacity of tobacco. This work was not substantiated by Sinclair,[434] but the exposure techniques used were questionable. Gentile *et al.*[151] showed that germination of pollen from sensitive abcessions of *Lycopersicon pimpinellifolium* was reduced by as much as 40% with techniques described by Feder.[137] However, reciprocal cross-pollinations between ozone-exposed and ozone-unexposed populations led to normal fruit development and viable seeds. Harrison and Feder[167] found a migration of cell organelles from the outer layer of cytoplasm in pollen grain of sensitive petunia exposed to ozone. They used 0.5 ppm for 3 h and found an 80% reduction in germination. Mumford *et al.*[355] found that 0.03–0.06 ppm was the threshold for an effect on germination of corn pollen. They did extensive biochemical studies of the pollen and suggested that ozone induces autolysis of structural glycoproteins and stimulates amino acid synthesis. Cameron and associates[53,54] found major reductions in yield of some corn varieties after high-ambient-oxidant episodes occurred during tasseling. These reductions appeared to result from poor fertilization. Although preliminary, those studies point to the importance of understanding the direct effects of oxidant pollutants on reproductive processes within plant species.

A study was undertaken to determine the potential mutagenic effects of ozone on *Arabidopsis thaliana.*[49] This plant completes its life cycle in about 35 days and is an excellent test species for determining effects of ozone across life cycles. Plants were exposed to acute doses for 6 h/day, 3 days/week, through 4 weeks of the life cycle. Seeds were collected from control and exposed plants and planted over seven generations. Seed production and biomass were reduced within generations, but no factors studied were carried over into later generations. The results showed that, for this particular species and the concentrations used, no mutagenic effects were carried via seed to later generations. This was a preliminary study that also reported problems with variability in plant response. The author suggested that similar studies be initiated to verify these results.

Biomass and Yield

Photochemical oxidants, including ozone and PAN, reduce plant biomass and yields in many plant species. Although yield and growth reductions are normally associated with visible injury, the oxidants can cause growth reductions with little or no injury. This relationship is not understood for the oxidant pollutants and probably varies with species and cultivars, as well as with conditions of growth and exposure, including dose. Todd and Arnold[487] compared injury with biomass and chlorophyll content for pinto bean exposed to a synthetic oxidant (ozone plus hexene). They found a logarithmic relationship between injury and the two physiologic measurements and suggested that injury was not a reliable index of growth effects. Their data were from acute exposures and suggest effects on growth at low injury values. A group of Canadian workers[3,8] have used physiologic measures exclusively for acute exposures. Many investigators are using physiologic measures, instead of injury, because they are more objective. Although physiologic responses are often better measures of chronic effects, their variability is usually at least as great as that of a subjective injury measure, and they are sometimes not as sensitive in indicating acute effects. Runeckles and Resh[421] reported on a reflectance measure of chronic effects that was much less variable than chlorophyll extraction. They did not compare this with a visual injury estimate. This technique should be studied further.

Considerable information is available on growth and yield effects. However, because it comes from scattered sources and much of it is subjective, no one has developed the data for predictive use (relating dose or injury to yield reductions). This section is divided into a discussion of effects under ambient conditions and a discussion of effects with

controlled additions of oxidants, primarily ozone. The ambient-oxidant section includes observational information and chamber studies comparing plant response in filtered versus unfiltered ambient air.

AMBIENT-OXIDANT STUDIES

Since the first observations of oxidant injury,[329] there have been continual reports of effects of oxidants on vegetation. This section does not attempt to cover all those reports, but does give several descriptive examples. The closing sections of this chapter (on plants as monitors and on economics) have additional examples. Engle *et al.*[128] first identified ozone as the cause of onion tip burn. They associated the development of this condition with severe weather fronts and suggested that ozone, produced in thunderstorms, may be the culprit. They identified both resistant and sensitive cultivars where resistance was genetically controlled. Weaver and Jackson[514] first described bronzing in white bean and associated this with oxidant (ozone) episodes. They found that ozone produced similar symptoms and that 0.16-ppm ozone for 0.5 h was the threshold for effects in their studies. This disease is known to cause reductions in bean yields, Haas[159] did a growth analysis in field-plot studies. He selected five plots based on severity of bronzing symptoms and showed a relationship between severity of bronzing and growth. He found that the rate of growth influenced symptom severity and that the stage of growth determined the dose at which bronzing occurred. He showed that physical conditions within a field could cause a variable response. Brasher *et al.*[36] reported 10–95% injury to three cultivars and 16 seedling lines of potato after 3 days of ambient oxidant that reached a maximum of 0.15 ppm. No yield data were reported for this study. Rich *et al.*[409] reported crop damage from oxidants to many plants in Connecticut when oxidant concentration was above 0.05 ppm. There were 83 of these high-oxidant days in 355 days monitored over four growing seasons. Reinert *et al.*[400] found similar effects around Cincinnati, Ohio, when oxidant concentrations were high. In this study, a group of plants was set out to monitor effects. Generally, these studies have not included quantitative yield data. Oshima *et al.*[380] reported a severe episode in California where injury was greater than expected on the basis of total-oxidant measurements. The presence of PPN was suspected. This is indicative of the episodic potential in high-pollution areas throughout the country. Several studies have reported on the severe effects of oxidants on ponderosa pine in the San Bernardino Mountains,[67,337] and quantitative work (including growth-ring analyses) is reported elsewhere in this document. Several subjective observations have come from

Europe[28,504] and Australia,[520] but little quantitative work has come from outside the United States and Canada.

Several excellent studies have reported yield data obtained by use of filtered and nonfiltered greenhouse or field chambers. Selected summary data are shown in Table 11-2. The first field-chamber work was reported on citrus by Thompson and Taylor.[465] Their report summarized several years of detailed work on lemon and orange and considered many combinations of pollutants. Reductions in fruit size, number, and total yields were reported in these studies and are the basis for the projected 50% reductions in citrus yields from the Los Angeles basin. Thompson and Kats[464] showed that some of these effects on citrus were probably related to concentrations of PAN in the ambient air. Thompson and co-workers[462,463] used the same chambers to determine effects of oxidants on grape over 2 yr. They found nominal effects in the first year and 50–60% reductions in the next 2 yr. The differences were attributed to the effects of oxidants on floral initiation in the year before the first study year. This concept could hold for any plants when floral structures are initiated in the prior season. They also reported a 41–62% reduction in leaf chlorophyll and a 13–17% reduction in sugars. A wax emulsion spray gave a 20% increase in yield over unsprayed plants; this suggests that selected protectants may be useful in protecting grapes grown in areas of high oxidant concentration.

These chambers were also used in the San Joaquin Valley in California to determine effects of oxidants on cotton.[46] Yield reductions of 5–29% in the lint and seed were reported at three locations over 2 yr, but no effects on lint quality or seed grade was found. Heggestad[193] reported reduced yields in four potato cultivars grown in filtered versus nonfiltered greenhouses. The results cover a 3-yr study and show up to a 50% loss in yield from sensitive cultivars. He also presented an excellent description of oxidant and ozone symptoms on potato. Heagle *et al.*[172] reported preliminary results on yield reduction in tobacco grown as part of a test of an open-top field-chamber design. They found significant reductions in both top and root fresh weight that compared closely with injury ratings (Table 11-3). Howell and Koch[226] reported yield reductions of 16–40% in four soybean cultivars with these open-top chambers. They found no changes in the protein or oil of seed.

CONTROLLED-CHAMBER STUDIES

Many workers have used controlled additions of ozone in acute or chronic studies to determine effects on a variety of growth measures. These studies suggest results attributable to ambient oxidants in field

plantings. However, in the field, there are probably combinations of acute, chronic, and predisposition effects of oxidants on the susceptible species and cultivars. Most of these studies were of greenhouse and controlled-chamber exposures, but several have used chambers placed over field plantings. Several studies have shown correlations between injury and yield or biomass reductions (Table 11-3). These suggest that injury may be a good criterion of yield reductions. However, it is known that yield reductions occur with little or no visible injury and that injury may cause no measurable yield reductions. These studies have been divided according to whether they used short- or long-term exposures.

Growth Effects of Short Ozone Exposures Ordin and Propst[371] used high concentrations of either ozone or PAN and reported a 50% inhibition in indoleacetic acid-stimulated growth in *Avena* coleoptile. This suggested that the oxidant may have a direct effect on hormonal production. Adedipe and associates reported reduced biomass and floral production in four bedding plants[3] and in two radish cultivars[8] from acute ozone exposures (Table 11-4). They reported no effects on marigold, celosia, impatiens, and salvia, even at the high concentration of 0.80 ppm for a 2-h exposure. The radish study included effects of exposure temperatures on growth. One cultivar reacted the same across temperatures, but the response of the second was conditioned by temperature. Tingey *et al.*[475] exposed radish at 7, 14, or 21 days and all combinations of these ages to ozone at 0.40 ppm for 1.5 h. They found the greatest effect on root growth at 14 days for the single exposure and at 7 and 14 days for the double exposure. The greatest effect was found in the root growth from the triple exposure (7, 14, and 21 days). The root reductions for the multiple exposures were equal to the additive reductions for single exposures. It was concluded that reductions were a result of photosynthate going preferentially to top growth. Reduced root growth rate was still found in the second week after exposure.

The studies of acute effects resulted in interesting growth phenomena that require further study. Tingey *et al.*[477] related foliar sensitivity in the first trifoliate leaf of soybean to leaf growth, stomatal resistance, and several metabolites. Maximal sensitivity occurred during the end of the period of rapid leaf expansion, when stomatal resistance was low. However, reduced sensitivity was shown in older leaves when stomatal resistance was still low. They found no relationship between sensitivity and concentrations of protein, amino acids, or various carbohydrates. This suggested that metabolic pools were not directly related to foliar response. Tingey and Blum[474] reported a consistent reduction in root growth that was related to reduced nodule number in soybean. The ozone did

TABLE 11-2 Effects of Ambient Oxidants (Ozone) on Growth, Yield, and Foliar Injury in Selected Plants[a]

Plant Species	Oxidant Concentration, ppm	Exposure Time	Plant Response, of reduction from control in response listed or % injury	Reference
Lemon	>0.10	Over growing season	32, yield 52, yield (leaf drop and other effects)	465
Orange	>0.10	148 h/month av in March-Oct.; 254 h/month av in July-Sept.	54, yield (other reductions found)	465
Grape, cultivar Zinfandel	≥0.25	Often over May-Sept. growing season	12, yield (first year) 61, yield (second year) (increased sugar content) 47, yield (third year)	462 463 *b*
Corn, sweet	0.20-0.35	Hourly maximum for 3-4 days before injury	67, injury (10 cultivars, 5 unmarketable) 18, injury (13 cultivars) 1, injury (11 cultivars)	53

Bean, white	>0.08	9 h	(Bronze color, necrotic stipple, premature abscission)	514
Tobacco, cultivar White Gold	0.20 ppm-h[c]	—	(Threshold for sensitive plants)	285
Tobacco, cultivar Bel W_3[d]	0.02-0.03	6-8 h	(Minimal—threshold—injury)	185
Tobacco, cultivar Bel W_3	>0.05	Often over growing season	22 (fresh wt), top 27 (fresh wt), root	172
Soybean, 4 cultivars	≥0.05	465 h during growing season	28 (16-40), yield	226
Cotton, cultivar Acula	Ambient	Over growing season	7-20, lint + seed (3 locations, 1972) 5-29, lint + seed (3 locations, 1973)	46
Potato, 4 cultivars[d]	≥0.05	326-533 h (2 yr)	34-50, yield (2 yr for 2 cultivars) 20-26, yield (1 yr for 2 cultivars)	193
Potato, cultivar Haig	0.15	3 consecutive days	95, injury (leaf area covered)	36

[a] Many field observations of injury to vegetation have been reported in the literature since Middleton *et al.*[329] first reported effects in California Most were reported without quantitative data on pollutant dose or plant effects and gave descriptions of plant injury.[36,128,159,380,400,409,514] Good summaries are given in several reviews.[183,196,204]

[b] C. R. Thompson, personal communication.

[c] Dose = concentration × time.

[d] Greenhouse studies.

TABLE 11-3 Injury versus Growth Reduction in Selected Plants Exposed to Ozone

Plant Species	Ozone Concentration, ppm	Exposure Time, h	Plant Growth Response, % reduction from control	Foliar Injury Response, % increase over control	Reference
Bean, cultivar Pinto	0.30	0.5/day, 14 days	11, leaf dry wt	9	280
	0.30	1.0/day, 14 days	40, leaf dry wt	46	
	0.30	2/day, 14 days	70, leaf dry wt	69	
	0.30	3/day, 14 days	76, leaf dry wt	78	
Corn, sweet	0.05	6/day, 64 days	12, av 4 yield responses	14	174
	0.10	6/day, 64 days	35, av 4 yield responses	25	
Soybean, cultivar Dare	0.05	6/day, 133 days	22, plant fresh wt	19	173
	0.10	6/day, 133 days	65, plant fresh wt	37	
	0.1 (+ SO_2 at 0.1)	6/day, 133 days	75, plant fresh wt	46	
Tobacco, cultivar Bel W_3	>0.05[a]	(Often over growing season)	22, top fresh wt	17	172
Soybean (2 cultivars)	0.05	8/day, 5 days/week, 3 weeks	2, top fresh wt	8	483
	0.10	8/day, 5 days/week, 3 weeks	21, top fresh wt	19	

[a] A study in field chambers with ambient photochemical-oxidant (ozone) concentrations.

not affect nodular activity or size. Evans [129] exposed pinto bean to acute ozone doses and found a reduction in leaf expansion within 12 h (10%) and a 33% maximal reduction on the day after a 0.20-ppm exposure. Growth rate continued to decrease 3 days after exposure. He used the terminal leaflet of the first trifoliate for these studies. A stimulation of stem elongation in tomato within 3 days after a 2-h exposure to 0.30-ppm ozone[361] and a predisposition to increased sensitivity from acute ozone exposure after exposure to low ozone concentrations[419] have also been reported. These are only some of the effects seen. Similar results are reported elsewhere in this chapter.

Growth Effects of Long-Term Ozone Exposures Taylor *et al.*[455] first reported a 52% reduction in the fresh weight of avocado seedlings exposed to a synthetic "smog" (ozone plus hexene) for 280 h. Tingey *et al.*[483] found reduced growth in two soybean cultivars (Hood and Dare) after intermittent exposure to ozone at 0.10 ppm over 3 weeks. They found that root and top growth were equally affected, but that roots were most affected when soybean was exposed to a mixture of ozone and sulfur dioxide. They found similar results[479] with radish. Little injury was reported in either paper. Frey[148] reported a 55% reduction in seed yield of soybean with a 5% decrease in seed lipids and a 21% increase in free amino acids. This is one of the few reports on quality changes in seed as a result of ozone exposure. A 30% reduction in wheat yield occurred when wheat was exposed at anthesis to ozone at 0.2 ppm, 4 h/day for 7 days.[428] Oshima[379] reported a reduction in tomato yield at 0.35 ppm over a long exposure period, whereas injury effects were significant at both 0.20 and 0.35 ppm. He suggested an injury-tolerance threshold for tomato below which one would find no yield reduction. He also reported a decrease in kernel weight of sweet corn exposed to ozone at 0.20 or 0.35 ppm.[378] The reduced weight was associated with a shrivel ear condition (kernels) that might relate to the effects of ozone on pollen development.

Craker[75] reported a reduction in petunia flower weight after a 53-day exposure to ozone at 0.05–0.07 ppm, but an increase in petunia flower weight[80] was found in a 7-day exposure to three different concentrations. Carnations continuously exposed to 0.07–0.08 ppm produced a single deformed flower, but the controls had 24 normal flowers.[138] Poinsettia bract area was decreased by 39% after a 50-day exposure (6 h/day) to ozone at 0.10–0.12 ppm.

Bennett *et al.*[19] made a case for stimulation of growth at low concentrations of ozone. They exposed bean (cultivar Pure Gold Wax), barley (cultivar Brock), and smartweed to ozone at 0.03 ppm over some growth

TABLE 11-4 Effects of Acute Ozone Exposure on Growth and Yield of Selected Plants

Plant Species	Ozone Concentration, ppm	Exposure Time, h[a]	Plant Response, % reduction from control in response listed[b]	Reference
Begonia, cultivar White Tausendschon	0.10	2	5, av of 3 growth responses: shoot wt, flower wt, flower no.	3
	0.20	2	10, av of same responses	
	0.40	2	19, av of same responses	
	0.80	2	38, av of same responses	
Petunia, cultivar Capri	0.10	2	9, av of same responses	3
	0.20	2	11, av of same responses	
	0.40	2	21, av of same responses	
	0.80	2	31, av of same responses	
Coleus, cultivar Scarlet Rainbow	0.10	2	2, av of same responses	3
	0.20	2	17, av of same responses	
	0.40	2	24, av of same responses	
	0.80	2	39, av of same responses	
Snapdragon, cultivar Floral Carpet, mixture	0.10	2	0, av of same responses	3
	0.20	2	6, av of same responses	
	0.40	2	8, av of same responses	
	0.80	2	16, av of same responses	

Radish, cultivar Cavalier, Cherry Belle	0.25	3	36, top dry wt (Cavalier) 38, root dry wt (Cherry Belle)	8
Radish	0.40	1.5 (1)	37, root dry wt	475
		1.5 (2)	63, root dry wt	
		1.5 (3)	75, root dry wt	
Cucumber, cultivar Ohio Mosaic	1.00	1	19, top dry wt (1% injury)	374
	1.00	4	37, top dry wt (18% injury)	
Potato, cultivar Norland	1.00	4	0, tuber dry wt (no injury)	374
	1.00	4 (3)	30, tuber dry wt (injury severe)	
Tomato, cultivar Fireball	0.50	1	15, plant dry wt (grown in moist soil)	247
	1.00	1	20, plant dry wt (grown in moist soil)	
	0.50	1	15, increase in plant dry wt (grown in dry soil)	
	1.00	1	25, increase in plant dry wt (grown in dry soil)	
Onion, cultivar Spartan Era	0.20	24	0, effect	374
	1.00	1	19, plant dry wt (no injury)	
	1.00	4	49, plant dry wt	
Tobacco, cultivar Bel W_3	0.30	2	48, chlorophyll content	4

[a] Number of exposures in parentheses.
[b] Unless other noted.

stage and found instances of significant growth increases. The concept needs further study in light of the current concern over "normal" background ozone concentrations.

Heagle and associates found a reduction in yield of sweet corn[174] and soybean[173] after exposure to ozone at 0.10 ppm for 6 h/day over much of the growing season. These exposures were carried out in field chambers set over soybean plots in the field. They suggested that a threshold for measurable effects on these crops would lie between ozone (oxidant) concentrations of 0.05 and 0.10 ppm for 6 h/day. These values are realistic in terms of growing-season averages in the eastern United States. More of these studies could help to clarify dose-response relationships for economically important crops. Table 11-5 summarizes these long-term, chronic studies.

Harward and Treshow[168] exposed 15 species, representative of the aspen plant community, to ozone at 0, 0.05, 0.15, and 0.30 ppm and ambient air during the growing season and reported effects in all species at the highest pollution concentration (Table 11-6). There was considerable plant variability, and only six species reproduced. However, vigor was reduced and most species were sensitive. Price and Treshow[395] found major biomass reductions in six grass and two tree species exposed for 4 h/day to ozone at 0.15–0.33 ppm over a growing season. They also found a reduction in or loss of some reproductive components. These effects could result in subtle shifts in community composition after several years of ozone exposure.

The results of these experiments suggest that overall economic effects on agricultural production could be extensive, depending on the sensitivity of cultivars used in production, and should be considered in the attempt to arrive at valid economic-damage functions for vegetation.

Factors Affecting Plant Response

The sensitivity of plants to ozone and PAN is conditioned by many interrelated factors. Heck[180] reviewed these factors and found that our understanding of the importance of any given factor on plants was fragmentary. Since then, a new body of research has become available that permits a more concise discussion of many factors that affect the response of plants. Although the response of a given plant is not predetermined by the response of related plants, sufficient information to predict relative responses under given sets of conditions is available. Information to predict interactions between variables and to predict whether species respond the same way to environmental factors under different pollutant stresses is not available. Current research demonstrates our inadequacies in understanding interacting variables.

Before we can predict the response of a plant variety to a specific pollutant or group of pollutants, we must understand the following factors: genetic variability (both between and within species), climatic and edaphic factors, interactions with other pollutants, interactions with biotic pathogens and insects, and the growth and physiologic age of susceptible plant tissue. The overall conceptualization of relationships between pollutant exposure and ultimate effects is shown simplistically (Figure 11-1) in an adaptation from van Haut and Stratmann.[507]

GENETIC FACTORS

Knowledge concerning the influence of genetic variability on plant response to ozone has been obtained from both field observations and chamber experimentation since the mid-1960's. Similar information on PAN is scarce, but several published reports are available. Resistance

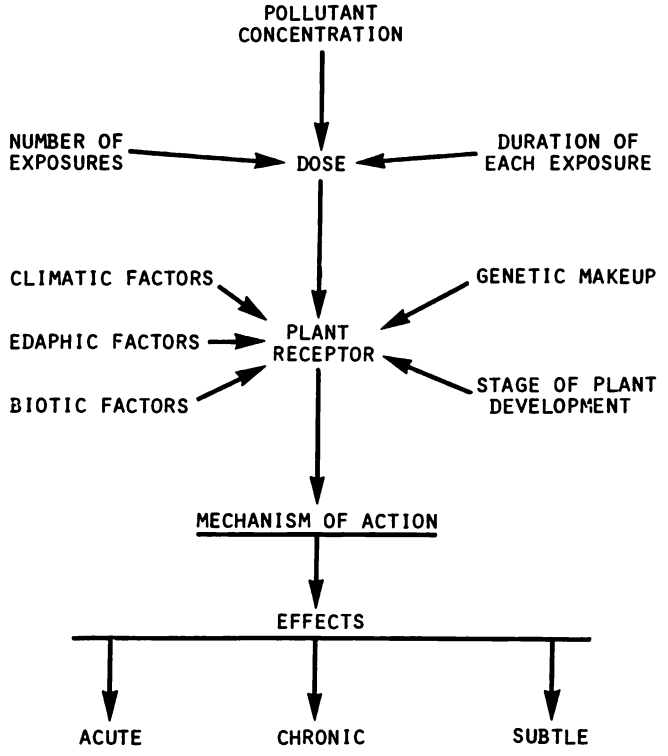


FIGURE 11-1 Conceptual model of factors involved in air pollution effects on vegetation. Modified from van Haut and Stratmann.[507]

TABLE 11-5 Effects of Long-Term Controlled Ozone Exposures on Growth, Yield, and Foliar Injury to Selected Plants

Plant Species	Ozone Concentration, ppm	Exposure Time, h	Plant Response, % reduction from control in response listed or % injury	Reference
Lemna, duckweed	0.10	5/day, 14 days	100, flowering; 36, flowering (1 week after exposure completed) 50, frond doubling rate	140
Carnation	0.05-0.09	24/day, 90 days	50, flowering (reduced vegetative growth)	136
Geranium	0.07-0.10	9.5/day, 90 days	50, flowering (shorter flower lasting time, reduced vegetative growth)	136
Petunia	0.05-0.07	24/day, 53 days	30, flower fresh wt	75
Poinsettia	0.10-0.12	6/day, 5 days/week, 10 weeks	39, bract size	80
Radish	0.05	8/day, 5 days/week	54, root fresh wt 20, leaf fresh wt	479
	0.05	8/day, 5 days/week (mixture of O_3 and SO_2 for same periods)	63, root fresh wt 22, leaf fresh wt	
Beet, garden	0.20	3/day, 38 days	50, top dry wt 40, storage root dry wt 67, fibrous root dry wt	369
Bean, cultivar Pinto	0.13	8/day, 28 days	79, top fresh wt 73, root fresh wt 70, height	294
Bean, cultivar Pinto	0.05 0.05	24/day, 3-5 days 24/day, 5 days	50, leaf senescence (chlorosis) (Fivefold increase in lateral bud elongation)	125, 127
Bean, cultivar Pinto	0.15 0.25 0.35	2/day, 63 days 2/day, 63 days 2/day, 63 days	33, plant dry wt; 46, pod fresh wt 95, plant dry wt; 99, pod fresh wt 97, plant dry wt; 100, pod fresh wt	215

Bean, cultivar Pinto	0.15	2/day, 14 days	8, leaf dry wt } (data available on	280
	0.15	3/day, 14 days	8, leaf dry wt } whole plants, roots,	
	0.15	4/day, 14 days	23, leaf dry wt } leaves, injury, and	
	0.15	6/day, 14 days	49, leaf dry wt } 3 levels of soil	
	0.225	2/day, 14 days	44, leaf dry wt } moisture stress)	
Bean, cultivar Pinto	0.225	4/day, 14 days	68, leaf dry wt } (data available on whole plants, roots, leaves, injury, and 3 levels of soil moisture stress)	280
	0.30	1/day, 14 days	40, leaf dry wt	
	0.30	3/day, 14 days	76, leaf dry wt	
Tomato, pole var	0.20	2.5/day, 3 days/week, 14 weeks	1, yield; 32, top dry wt; 11, root dry wt	379
	0.35	2.5/day, 3 days/week, 14 weeks	45, yield; 72, top dry wt; 59, root dry wt	
Corn, sweet, cultivar Golden Jubilee	0.20	3/day, 3 days/week, till harvest	13, kernel dry wt; 20, top dry wt; 24, root dry wt	378
	0.35	3/day, 3 days/week, till harvest	20, kernel dry wt; 48, top dry wt; 54, root dry wt	
Wheat, cultivar Arthur 71	0.20	4/day, 7 days (anthesis)	30, yield	428
Soybean	0.05	8/day, 5 days/week, 3 weeks (mixture of O_3 and SO_2 for same periods)	2, top fresh wt	483
	0.10		24, root fresh wt	
			21, top fresh wt	
	0.05		24, root fresh wt	483
			12, top fresh wt	
			30, increase in shoot: root ratio[a] (greatest effect on roots)	
Alfalfa	0.10	2/day, 21 days	16, top dry wt	433
	0.15	2/day, 21 days	26, top dry wt	
	0.20	2/day, 21 days	39, top dry wt	
Grass, brome	0.15–0.33 (varied)	4/day, 5 days/week, growing season	83, biomass	395
Pine, eastern white	0.10	4/day, 5 days/week, 4 weeks (mixture of O_3 and SO_2 for same periods)	3, needle mottle (over 2–3 days of exposure)	110
			16, needle mottle	

TABLE 11-5 (Cont.)

Plant Species	Ozone Concentration, ppm	Exposure Time, h	Plant Response, % reduction from control in response listed or % injury	Reference
Pine, ponderosa	0.15	9/day, 10 days	4, photosynthesis	338
	0.15	9/day, 20 days	25, photosynthesis	
	0.15	9/day, 30 days	25, photosynthesis	
	0.15	9/day, 60 days	34, photosynthesis	
	0.30	9/day, 10 days	12, photosynthesis	
	0.30	9/day, 20 days	50, photosynthesis	
	0.30	9/day, 30 days	72, photosynthesis	
	0.45	9/day, 30 days	85, photosynthesis	
Poplar, yellow	0.30	8/day, 5 days/week, 13 weeks	82, leaf drop; 0, height	233
Maple, silver	0.30	8/day, 5 days/week, 13 weeks	50, leaf drop; 78, height	233
Ash, white	0.30	8/day, 5 days/week, 13 weeks	66, leaf drop; 0, height	233
Sycamore	0.30	8/day, 5 days/week, 13 weeks	0, leaf drop; 22, height	233
Maple, sugar	0.30	8/day, 5 days/week, 13 weeks	28, leaf drop; 64, height	233
Corn, sweet, cultivar Golden Midget[b]	0.05	6/day, 64 days	9, kernel dry wt; 14, injury (12, av 4 yield responses)	174
	0.10	6/day, 64 days	45, 25, 35 for same responses	
Soybean, cultivar Dare[b]	0.05	6/day, 133 days	3, seed yield; 22, plant fresh wt; 19, injury	173
	0.10	6/day, 133 days (mixture of O_3 and SO_2 for same periods)	55, 65, 37 for same responses	
	0.10		63, 75, 46 for same responses	
Poplar, hybrid	0.15	8/day, 5 days/week, 6 weeks	50, shoot dry wt; 56, leaf dry wt; 47, root dry wt	234

[a] The only increase.
[b] Studies conducted under field conditions, except that plants were enclosed to ensure controlled pollutant doses.

TABLE 11-6 Effects of Ozone on Selected Understory Species from an Aspen Community[a]

Plant Species	Plant Response at Different Ozone Concentrations[b]								
	Foliar Injury, %			Plant Wt, % of control			Seed Wt, % of control		
	0.05 ppm	0.15 ppm	0.30 ppm	0.05 ppm	0.15 ppm	0.30 ppm	0.05 ppm	0.15 ppm	0.30 ppm
Chenopodium album L.	0	35	40	103	71*	83	87	99	87
C. fremontii L.	10	35	90	71	98	32	102	96	94
Descurainia sp.	0	15	55	179*	77	56*	—	—	—
Geranium fremontii L. Torr. *ex* A. Gray	7	50	90	93	94	53*	—	—	—
Lepidium virginicum L.	50	100	100	121	72	31*	94	69	47*
Madia glomerata	40	100	100	112	38*	13*	79	74	18
Polygonum aviculare L.	30	95	95	271	186*	29*	127	43	50
P. douglasii Greene	5	35	95	87	29*	5*	93	84	3*

[a] Exposures were 3 h/day, 5 days/week, through growing season. Data from Harward and Treshow.[168]
[b] Asterisk indicates significant effect.

to ozone and PAN varies between species of a given genus and between cultivars within a given species. Variations in response are functions of genetic variability and environmental stress as they affect morphologic, physiologic, and biochemical characteristics. In native populations and in breeding experiments, both ozone and PAN may act as selective pressure stresses.

Variations in species response to ozone and PAN are well documented.[204,206,238,265,452,457,505,525] A list of species susceptibilities to ozone is found in Table 11-24, which was generated from results of controlled exposures and the use of the sensitivity table, Table 11-23.

Heck[180] summarized variations in cultivar responses of several species. Reinert[397] developed a compendium of research papers covering horticultural cultivar responses to ozone, PAN, and other pollutants that is complete, but gives no summary of results. A number of papers have been published since Brennan *et al.*[44] first published their results on the separation of oat and potato cultivars exposed to ozone and stressed the importance of cultivar differences within species. Additional ozone studies are available for bean,[94] begonia,[3] morning glory,[358] chrysanthemum,[38,249] poinsettia,[298] spinach,[299] lettuce,[402] radish,[402] turfgrass,[37] forage legume,[45] alfalfa,[225] safflower,[229] soybean,[228,340,481] small grain (oat, rye, wheat, barley),[426] and English holly.[40] Petunia was studied with auto exhaust and PAN,[139] and both petunia and chrysanthemum have been studied with PAN.[112,526] Screens using ambient oxidant conditions, sometimes without pollutant concentration, are fairly common: bean,[218] potato,[36,218,274] tobacco,[194,218,313,323] petunia,[274] poinsettia,[274] sweet corn,[53] and grape.[242] Most of these studies used acute exposures, which may or may not relate to results from chronic exposures. Some reports of ambient oxidant exposures covered part or all of a growing season. These screens used injury as the response measure. We do not know whether foliar injury ranking relates to economic yield loss ranking.

Cultivar variations in three species have been intensively studied in relation to their sensitivity to ozone: petunia,[3,59,139] tomato,[65,66,151,403] and tobacco.[101,156,194,210,319,322,323] These studies have resulted in recommendations that some cultivars not be used in areas of high oxidant potential. The susceptible Bel W_3 tobacco is widely used as a biologic indicator of oxidant.

A summary of cultivar responses to oxidants, ozone, and PAN is given in Table 11-7. Summary data were also included in the development of the dose-response equations and Table 11-24 on plant sensitivities.

Hanson[162] presented a list of 160 woody species, from observations at the Los Angeles State and County Arboretum, that were sensitive or tolerant to oxidants. Several recent investigations have considered susceptibility of tree species to ozone. Jensen[233] studied 9 hardwood

species, Berry[22] looked at 3 pine species, Davis and Wood[93,99] reported on 18 coniferous species, and Miller[334] reported on 15 western conifers. In general, selection within natural tree species has not been studied.

Two studies have explored the mechanism of genetic resistance to ozone. Engle[125] and Engle and Gabelman[126] found that ozone sensitivity in onion is probably controlled by a single gene pair, with dominance of the resistant gene. In resistant plants, the membrane of the guard cells was more sensitive to ozone and lost its differential permeability, thus causing stomatal closure. This did not occur in the guard cells of the sensitive cultivar, and thus the stomata remained open. Taylor[446] crossed the sensitive Bel W_3 tobacco with a resistant line and found that the F_1 had intermediate sensitivity. The F_2 segregated into 40% resistant, 10% sensitive, and 50% intermediate. He postulated that sensitivity was controlled by at least two genes. Resistance mechanisms need to be studied in other species. Dean[100] reported a 50% increase in stomatal density of two sensitive tobacco cultivars over two resistant cultivars. Stomatal size was not a factor in this study.

Differential susceptibility of individual clones of eastern white pine to ozone and sulfur dioxide was shown by Berry and Heggestad[25] and Costonis.[69] When Dochinger *et al.*[109] determined that chlorotic dwarf could be caused by an interaction of ozone and sulfur dioxide, they used a chlorotic dwarf-susceptible clone to eliminate the genotype variable. Houston[220] tested the response of tolerant and susceptible clones of eastern white pine (on the basis of symptom expression under ambient conditions) to ozone or sulfur dioxide. Injury caused by sulfur dioxide or sulfur dioxide plus ozone correlated well with the earlier field responses, but ozone did not produce a consistent response. They also found that a 6-h exposure to a mixture of sulfur dioxide and ozone caused a difference in needle elongation between clones within tolerant and sensitive groups. This suggests that tolerance may function over a wide range of responses.

It is reasonable to suggest that a spectrum of genotypes represents various susceptibilities to oxidants, singly or in combination. Although environmental factors are important in conditioning pollutant susceptibility, the control of environment over injury to sensitive genotypes may be less pronounced, if specific biochemical or physiologic mechanisms that have weak interactions with the environment are involved.

CLIMATIC CONDITIONS

Plant response to ozone and PAN may be altered by climatic conditions before, during, and after exposure.[180] Plants may be sensitized to a given

TABLE 11-7 Response of Cultivars within Species to Ozone and PAN

Plant Species	No. Cultivars	Pollutant; Concentration, ppm; Time, h	Plant Response, % reduction from control in response listed				Sensitive Cultivars	Resistant Cultivars	Reference
			Min.	Mean	Max.	Remarks			
Oat	9	O_3; 0.22; 4	0	38	69	Injury	C.I. 7540	C.I. 7271	44
Potato	8	O_3; 0.23; 4	0	15	50	Injury	Chippewa	Avon	44
Bean	10	O_3; 0.25; 4	1	8	33	Injury	Sanilac	Tendercrop	94
Bean	10	PAN; 0.12; 2	9	37	64	Injury	Sanilac	Tempo	441
Potato	6	Oxidant; season[a]	Tolerant to susceptible			Injury	Norchip	Katahdin	218
Potato	24	Oxidant; 15; 3-day peak	10	54	98	Injury	Haig	Superior	36
Petunia	8	Oxidant; season[a]	0	15	21	Top fresh wt	Fiesta	Purple Plum	274
Begonia	3	O_3; 0.80; 2	7	39	50	Top dry wt	White Tausendschon	Scarletta	3
Petunia	3	O_3; 0.80; 2	9	19	40	Top dry wt	Capri	Canadian ALL Double Mix	3
Petunia	14	O_3; 1.00; 1	1	23	38	Injury	Festival	Lilac Time	139
Petunia	6	PAN; 0.25; 1	4	15	24	Injury	Blue Danube	Parti Pink	139
Petunia	15	Oxidant; 0.15; 1.5	2	12	45	Injury	White Cascade	Parti Pink	139
Petunia	28	PAN; 0.15; 1	0	9	44	Injury	Snow drift	Coral Magic	112
Chrysanthemum	45	O_3; 0.60; 3	0	23	70	Injury	Tranquility	Ann Ladygo	249
Chrysanthemum	8	PAN; 0.60; 4	0	0	5	Injury	None	All eight	526
Chrysanthemum	16	O_3; 0.40; 4	10	25	65	Injury	Golden Arrow	Indian Summer	38
Poinsettia	5	O_3; 0.35; 4	6	35	63	Injury	Eckespoint C-1	White Annette Hegg	298
Spinach	6	O_3; 0.15; 4	5	25	50	Injury	Virginia Blight Resistant Savoy	Bounty	299
Lettuce	8	O_3; 0.70; 1.5	10	24	46	Injury	Dark Green Boston	Great Lakes	402
Radish	9	O_3; 0.35; 1.5	17	25	35	Injury	Cherry Belle	Icicle	402
Tomato	12	O_3; 0.40; 1.5	10	35	89	Injury	Roma VF	VF 145 B	403
Tomato	24	O_3; 0.25; 1.5	5	50	75	Injury	Roma VF	Heinz 1439	66
Tomato	63	O_3; variable				Injury	L. esc. (247089)	L. pimp. (795.12)	151

Tomato	295	O_3; 0.25; 1.5	0	40	75	Injury	Various	Various	65
Turfgrass	11 (7 spp.)	O_3; 0.30; 6	0	60	90	Injury	Meyer Zoysia	Penncross	37
Forage legumes	10 (6 spp.)	O_3; 0.25; 4	0	10	50	Injury	Crownvetch	Kenland Red Clover	45
Alfalfa	14	O_3; 0.20; 4	12	29	52	Injury	Mesa-Sirsa	MSB-CW5A$_n$[2]	225
Safflower	12	O_3; 0.25; 2	32	65	73	Injury	Nebraska-4051	Frio	229
Soybean	21	O_3; 0.50; 2	45	64	79	Injury	Wye	PI 157474	340
Soybean	14	O_3; 0.70; 1.5	17	40	57	Injury	Hawkeye	Hood	481
Soybean	5	O_3; 0.50; 2	16	57	89	Injury	Kent	Cutler	228
Sweet corn	34	Oxidant; >0.30; 3-day peak	5	44	98	Injury	No names	No names	53
Tobacco, flue-cured	12	O_3; 0.20; 3	21	45	59	Injury	Speight G-5	Coker 187-Hicks	322
Tobacco, flue-cured	15	O_3; 0.36; 2.5	38	68	87	Injury	Speight G-36	Coker 187-Hicks	322
Tobacco, variable	16	Oxidant (Beltsville)[a]	1	16	65	Injury	Pa 60-19	Bell W_3	313
Tobacco, wrapper	5	O_3; 0.06; ?	10	20	30	Injury	Conn. 49	Dixie Shade	101
Tobacco, spp. & cultivars	5	O_3; 0.30; 1	6	24	52	Injury	*N. glutinosa*	Samson (NN)	156
Tobacco	6	O_3; 0.25; 6 (3 days)	4	9	42	Injury	Bel-C	Bel-B	194
Tobacco	5	Oxidant (Beltsville)[a]	4	51	73	Injury	Bel-C	Madole	194
Tobacco	4	O_3; 0.20; 1.5	17	32	37	Injury	Havana 142	Havana 501	210
Tobacco	6	O_3; 0.15; 3	27	35	38	Injury	Catterton	Wilson	323
Grape	134	Oxidant (N.Y. & Ontario)[a]	0 to severe				Ives	Delaware	242
Holly	2	O_3; 0.50; 7	No effects				—	English holly	

[a] Concentration was present at location but was not given.

set of conditions after 1-5 days. It appears that the major conditioning occurs within the first 3 days under a given set of environmental conditions. Conditions during exposure may be critical, and conditions after exposure are probably important, but less so than those before and during exposure. The responses of plants to ozone and PAN under varied climatic conditions are studied primarily under laboratory and greenhouse conditions, but field observations have often substantiated the results. Most studies have involved individual climatic factors and one or two response measures, usually including injury. Several have dealt with environmental interactions. Sufficient information exists for generalizations of plant response, but there is much uncertainty, because of the small number of species studied and the lack of information on the interactions of factors.

Light Quality The quality of light affects the growth and development of plants and plays some role in determining the response of pinto bean to PAN.[118,119] Injury to pinto bean from PAN was maximal from exposure at 420 and 480 nm and less than half that at 640 nm. The response is apparently associated with changes in carotenoid pigments. Shinohara *et al.*[433] reported an effect on tobacco (H-mutant) when exposed at 0.40-ppm ozone for 30 min. They found the least injury in far red, followed by blue, green, and white, with the greatest effect in red. This work was preliminary. More information is needed on the effects of light quality.

Photoperiod The effect of a given light period within a 24-h cycle exerts physiologic control over some aspects of plant development. Research has shown that plants are more sensitive to ambient oxidants and ozone when grown under an 8-h photoperiod than either a 12-h or a 16-h photoperiod[185,237,282] (Table 11-8).

Light Intensity The intensity of light affects many physiologic processes within plants and is known to affect the response of plants to oxidant pollutants. Studies have been reported on effects of intensity before, during, and after exposure (Table 11-9).

Dugger *et al.*[119] found a direct correlation between the sensitivity of pinto bean to PAN and increasing light intensity. Pinto bean required light just before, during, and after a PAN exposure for injury to occur.[119,456] This was not true with exposure to ozone, although some light period was necessary.

Generally, plants are more sensitive to ozone when grown at lower light intensities. This was shown for pinto bean[122,185] and tobacco,[122,185,433]

TABLE 11-8 Response of Plants to Ozone, as Conditioned by Photoperiod

Plant Species	Ozone Concentration, ppm; h	Notes	Response, % injury: Photoperiod, h 8	12	16	Reference
Tobacco, cultivar White Gold	0.67; 5	—	57	40	27	282
Tobacco, cultivar Bel W_3	0.30; 1	Control conditions, 2,000 ft-c (21,529 lx)	54	—	19	185
Bean, cultivar Pinto	0.30; 1	Control conditions, 2,000 ft-c (21,529 lx)	71	—	18	185

but was reversed in tobacco Bel W_3 (J. A. Dunning and W. W. Heck, personal communication). The reason for this reversal was unclear. In both pinto bean and tobacco, there was greater injury from ozone when plants were grown at lower light intensities when the humidity was 60% or 80%.[122] Dugger *et al.*[119] found greater injury in plants grown at 900 ft-c (about 9,690 lx) than at 2,220 ft-c (about 23,680 lx), but Dunning and Heck (personal communication) reported no difference between 1,000 and 2,000 ft-c (about 10,760 and 21,530 lx) for either pinto bean or tobacco exposed to ozone. Dugger *et al.*[119] found a relation between plant age and light intensity. Peak sensitivity to ozone was about the same at both intensities, but the span of plant age that was sensitive was increased at 900 ft-c (about 9,690 lx). This could have an important bearing on environmental work and has not been seriously considered by other workers.

Sensitivity to ozone as light changes during exposure generally increases with intensity. This was found for tobacco when grown at 4,000 ft-c (about 43,060 lx) and exposed between 1,000 and 4,000 ft-c (about 10,760 and 43,060 lx) (Dunning and Heck, personal communication). A slight decrease in response was found at 6,000 ft-c (about 64,580 lx). When pinto bean was grown at 4,000 ft-c (43,060 lx), there was an increase in sensitivity from 1,000 to 6,000 ft-c (10,760 to 64,580 lx) during ozone exposure; but, at lower intensity during growth, the 4,000- and

TABLE 11-9 Response of Plants to Ozone, as Conditioned by Growth and Exposure Light Intensity

Plant Species	Ozone Concentration, ppm, for 1 h	Notes[a]	Growth or Exposure[b]	Injury, %, at Light Intensity, ft-c (lx)				Reference
Bean, cultivar Pinto	0.35	Grown under control conditions; ET = 85°F (29°C)		0 (0)	100 (1,076)	1,800 (19,375)	2,600 (27,987)	186
			Exposure	2	10	73	89	
Bean, cultivar Pinto	0.30	Control conditions; PP = 8 h		2,000 (21,529)		3,000 (32,293)		185
			Growth	71		49		
Tobacco, cultivar Bel W_3	0.30	Control conditions; PP = 8 h	Growth	54		41		185
Bean, cultivar Pinto	0.40	Control conditions; PP = 8 h; EL = 2,000 ft-c (21,530 lx)		1,000 (10,764)	2,000 (21,529)	4,000 (43,057)	6,000 (64,585)	[c]
			Growth	80	84	28	—	
	0.40	Control conditions; PP = 8 h; GL = 4,000 ft-c (43,057 lx)	Exposure	16	28	57	67	

Tobacco, cultivar Bel W_3	0.40	Control conditions; PP = 8 h; EL = 4,000 ft-c (43,057 lx)	Growth	21	19	38	—		[c]
	0.40	Control conditions; PP = 8 h; GL = 4,000 ft-c (43,057 lx)	Exposure	8	27	38		32	
Bean, cultivar Pinto	0.40	Control conditions; PP = 8 h	Growth						
			at 80% RH	—	78	44	—		122
			at 60% RH	—	66	47	—		
	0.40	Control conditions; PP = 8 h	Exposure	—	50	68	—		
Tobacco, cultivar Bel W_3	0.40	Control conditions; PP = 8 h	Growth						
			at 80% RH	—	36	27	—		122
			at 60% RH	—	43	34	—		
	0.40	Control conditions; PP = 8 h	Exposure	—	33	35	—		

[a] PP = photoperiod; ET = exposure temperature; EL = exposure light; GL = growth light.
[b] Time of light treatment.
[c] Dunning and Heck, personal communication.

6,000-ft-c (43,060- and 64,580-lx) exposure intensity gave similar responses. Heck[122] reported that exposure light had no effect on the response of tobacco or pinto bean to ozone when the relative humidity was 80%, but the response increased with increasing exposure light at 60%. In an earlier study, Heck *et al.*[186] found an increasing response in pinto bean from darkness to 2,600 ft-c (about 27,980 lx) with a threshold response around 100 ft-c (about 1,080 lx).

Shinohara *et al.*[433] reported that up to 10 h of postexposure light did not affect the response of tobacco (H-mutant) to ozone. Davis and Wood[97] delayed symptom development in Virginia pine when the plants were held in the dark for extended periods after ozone exposure. However, the final severity of response was unchanged.

Temperature Variations in plant response to ozone or oxidants occur with increasing growth temperature, if a given temperature is maintained for 3 days or more before exposure. Macdowell[282] found that a low day or high night temperature increased the susceptibility of White Gold tobacco to ozone. He reported no interaction between day and night temperatures. Juhren *et al.*[237] used eight combinations of day and night growth temperatures with *Poa annua* and then exposed these plants to ambient oxidants for a day. The sensitivity varied with plant age, showed a partial reversal after 3 days of changed temperatures, and was greatest at the 26°–20° C day-night temperatures. Radish[8,373] and pinto bean (under some conditions),[123] were more sensitive if grown under cooler conditions, whereas snap bean,[5] soybean,[123] pinto bean (under other conditions),[123] Bel W_3 tobacco,[320,430] Virginia pine,[93,97] and white ash[521] were more sensitive if grown under warmer conditions.

Shinohara *et al.*[432] looked at growth, exposure, and postexposure temperatures and their effect on the response of several tobacco cultivars to ozone. Plants were grown at constant temperature (day/night) during growth and were kept in the dark for 48 h after exposure. These elements of design weaken the value of this report. However, generally they showed an interaction between growth and postexposure temperature, night temperatures were more important than day temperatures, and lower temperatures during exposure increased the sensitivity of tobacco to ozone.

Drummond[112] reported increased injury to petunia (cultivar White Cascade) with increasing temperature when exposed for 1 h to PAN at 0.15 ppm.

Early reports suggested a positive correlation between plant response to ozone and increasing exposure temperature, to about 30° C. These results were shown for pinto bean when exposures were under green-

house conditions,[186] but an inverse correlation was found when they were under controlled lighting in growth chambers. This suggested that, under greenhouse conditions, it was not possible to separate light and temperature effects. This inverse relationship has been shown for Bel W_3 tobacco,[55,56,186] *Lemna minor*,[141] Virginia pine,[93,97] and white ash.[521] Under some conditions, this does not seem to hold for pinto bean,[123] in which a direct correlation was found from 20° to 32° C.

It has been suggested[180] that higher postexposure temperatures will cause an increase in sensitivity. This was shown for Virginia pine[93,97] and for white ash,[521] but the reverse occurred in Bel W_3 tobacco[430] and in radish.[8]

Two papers have considered interactions between temperature and other variables. Dunning *et al.*[123] studied Dare soybean and pinto bean with respect to ozone dose, potassium nutrition, growth temperature, and exposure temperature. The two species did not respond equivalently, except that there were no interactions with potassium nutrition in either. Soybean showed no interactions between dose and temperature under either temperature design. Pinto bean showed strong interactions between dose and growth temperature and between exposure and growth temperatures and a mild interaction between dose and exposure temperature, with no higher-order interactions. The dose-growth-temperature interaction is shown graphically in Figure 11-2. Apparently, pinto bean is very sensitive to a variety of stresses, and these variations make pinto bean both a unique test plant and one that can be very difficult to handle. In later studies, Dunning and Heck (personal communication) looked at interactions among growth-temperature, exposure temperature, and plant species (Bel W_3 tobacco and pinto bean). They found that the two species responded differently to the temperature conditions. Pinto bean showed no interaction between growth and exposure temperatures, whereas tobacco showed a strong interaction. For tobacco, growth temperature had little effect at an exposure temperature of 16° or 32° C, but a marked effect at 21° or 27° C (sensitivity increased with increasing growth temperature). These types of interactions need further clarification as we attempt to understand the effects of a total environmental system on the response of plants to oxidant pollutants.

Crops that show greater injury under cooler conditions may be more severely injured in early spring (assuming that oxidant is present). Feder and Sullivan[141] used duckweed to remove the confounding effects of stomatal movement. They reported similar results in light and dark fumigations with temperature variations. Dunning *et al.*[123] found no changes in carbohydrate content associated with sensitivity of either pinto bean or Dare soybean. This was also reported for radish,[8] except that

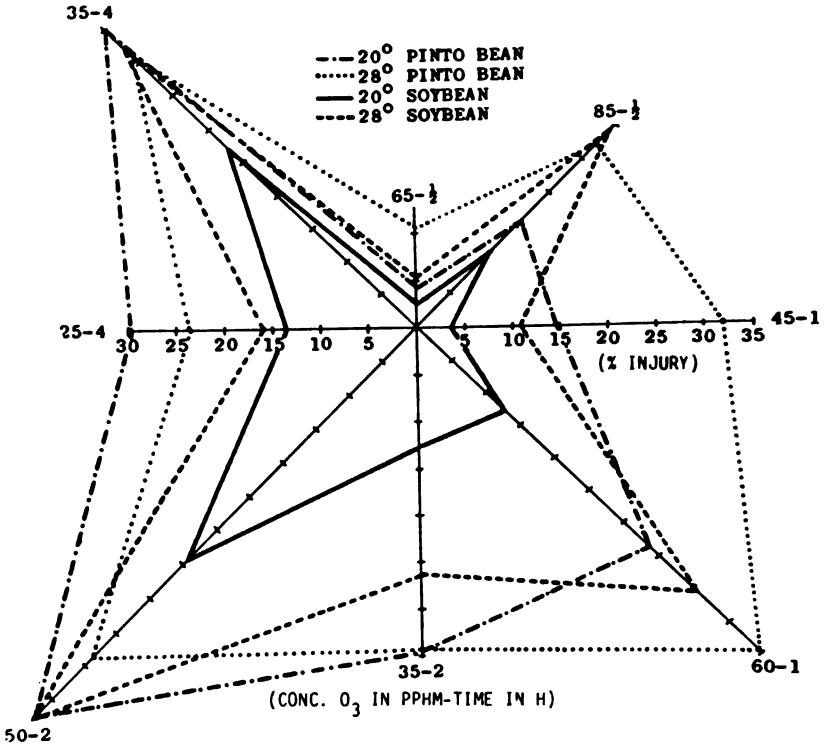


FIGURE 11-2 The significant interaction of growth temperature (°C), ozone dose (concentration × time), and species on foliar response (% injury) to ozone. Dose is shown at the end of each of the eight axes as concentration (pphm) and time (h). Percentage injury is shown in 5% increments along each axis. Each value is a mean of 15 observations. The LSD at the 1% level is 8.3 for the injury response. This value can be used to compare any two points in the figures. Reprinted with permission from Dunning *et al.*[123]

cultivar Cavalier showed an increase in carbohydrate after an ozone exposure in a single experiment.

A summary of these temperature effects is found in Table 11-10.

Relative Humidity Field observations suggest that plants are more sensitive to oxidants as relative humidity increases. Davis and Wood[93,97] found an increase in sensitivity of Virginia pine to ozone at a high humidity during exposure, but reported no effects from growth or postexposure humidity changes. Otto and Daines[384] found similar results over a wider humidity range and over several ozone concentrations during exposure of pinto bean and Bel W_3 tobacco. They did not study growth

or postexposure conditions. Dunning and Heck[122] showed a significant increase in the response of pinto bean, but not of tobacco, to increased exposure humidity. For growth humidity, tobacco sensitivity decreased with an increase in humidity; pinto bean response was unaffected by humidity at a growth light of 4,000 ft-c (43,060 lx), but was increased with an increased humidity at 2,000-ft-c (21,530-lx) growth light. In later work, Dunning and Heck (personal communication) found an increase in sensitivity of both tobacco and pinto bean to ozone with increasing exposure humidity from 45 to 90%, regardless of growth humidity. However, there was an interaction between growth humidity and exposure humidity such that the response of these two species to growth humidity was conditioned by the exposure humidity. Plants were always more sensitive when grown at 75% humidity, regardless of the exposure humidity. Table 11-11 shows some of these results.

Carbon Dioxide Stomatal activity is affected by carbon dioxide, and it may affect plant sensitivity to oxidants. Heck and Dunning[185] reported a decrease in sensitivity of tobacco to ozone if the tobacco was exposed to added carbon dioxide at 500 ppm immediately before and during exposure to ozone (22% injury with added carbon dioxide, and 66% injury without). If this is a general plant response, the carbon dioxide now added to greenhouses to increase productivity may also increase resistance of the plants to oxidant pollutants.

Field Observations Canadian workers using ambient oxidant dose have correlated meteorologic variables with plant injury.[285,354] A correlation was discovered when an empirical relationship involving evapotranspiration (the coefficient of evaporation) was developed and used to modify the dose information. This empirical relationship has been used on a limited basis to predict damaging oxidant concentrations from monitored meteorologic conditions.

Linzon[277] reported SNB symptoms on white pine after several days of wet weather followed by a continuous sunny period. Symptoms were noted several times during the 1957–1964 growing seasons at Chalk River, Ontario, but time of occurrence did not correlate well with peak oxidant concentrations.[276] Berry and Ripperton[26] observed emergence tipburn on susceptible trees in West Virginia several days after oxidant peaks of 0.065 ppm. They found that container-grown susceptible pine clones were protected from injury if placed in a chamber supplied with charcoal-filtered air.

Such factors as windspeed and barometric pressure appear to play little or no role in affecting plant sensitivity to oxidant pollutants. Air

TABLE 11-10 Response of Plants to Ozone, as Conditioned by Growth or Exposure Temperature

Plant Species	Ozone Concentration, ppm; h	Notes[a]	Type of Response	Growth or Exposure[b]	Response, % Day or Day/Night Temperature, °C[c]					Reference
Tobacco, cultivar White Gold	0.67; 5	—			15.6	23.9	32.2			282
			% injury	Growth						
				day	—	44	27			
				night	28	33	44			
Radish, cultivar Cavalier	0.24; 4	PP = 16 h			20/15		30/25			373
			% growth reduction	Growth						
				60 mg of N/liter	50		43			
				300 mg of N/liter	55		47			
Bean, cultivar Blue Lake	0.50; 2	Exposed twice			20/15	25/20	30/25			5
			% chlorophyll reduction; % reduction of dry wt of 1st trifoliate leaf; % reduction of root dry wt		7	29	55			
					14	32	33			
					27	31	28			
Tobacco, cultivar Bel W_3	0.20; 4	PP = 12 h; 2,000 ft-c (21,529 lx); 85% RH			10	15.6	21.1	26.7	32.2	55, 56
			% injury	Exposure	66	34	13	11	1	
Lemna minor, L.	0.25; 4	PP = 8 h; 500 ft-c (5,382 lx)			7.2	18.3	25	30.6		141
			% chlorophyll reduction	Exposure	53	18	0	0		
Tobacco, cultivar Bel W_3	3 (ambient oxidant exposure; av = 0.69 ppm·h)	—			10	13.9	20	23.9		430
			% injury	Postexposure	50	33	10	0		

Tobacco, cultivar H-2	0.35; 2	Constant temp. 4 days before exposure (23°C); 48 h dark at 13°C after exposure	% injury (5 leaves/plant affected)	Exposure	13 66	20 50	27 9	32 0	432
Radish, cultivar Cherry Belle	0.25; 3	25°/20°C normal temp.; PP = 16 h; 1,800 ft-c (19,375 lx); 82% RH	% growth reduction of roots	15 days growth 15 days postexposure	20/15 37 52	30/25 25 38			8
Soybean, cultivar Dare	8 doses	PP = 12 h; 2,200 ft-c (23,681 lx); 1 week at special temp. for growth	% injury	Growth	20 14	28 28			123
Bean, cultivar Pinto	8 doses	PP = 12 h; 2,200 ft-c (23,681 lx); 1 week at special temp. for growth	% injury	Growth at 20 ET Growth at 28 ET Exposure at 28 GT	28 30 24	24 43 43			123
Poa annua	Ambient oxidant; 24	Controlled growth	% injury	Growth	20/14 14	23/17 11	26/20 20	30/24 1	237
Tobacco, cultivar Bel W_3	0.21; 2	2 weeks of conditioning	% injury	Growth	20/15 7	25/20 39			320
Bean, cultivar Pinto	0.55; 1	Greenhouse exposure (correlated with light intensity)	% injury	Exposure	22.2 25	26.7 54	32.2 77		186
Bean, cultivar Pinto	0.35; 1	Grown under control conditions; exposed under constant conditions	% injury	Exposure	18.3 —	23.9 77	29.4 68	35 51	186
	0.32; 1	Grown under control conditions; exposed under constant conditions	% injury	Exposure	77	68	56	41	

TABLE 11-10 (Cont.)

Plant Species	Ozone Concentration, ppm; h	Notes[a]	Type of Response	Growth or Exposure[b]	Response, % Day or Day/Night Temperature, °C[c]					Reference
Tobacco, cultivar Bel W_3	0.35; 1	Grown under control conditions; exposed under constant conditions	% injury	Exposure	—	61	44	29		186
Pine, Virginia	0.50; 1.5	Control conditions; 3-yr seedlings			10	15	21	27	32	97
			% injury	Exposure	52	33	27	15	3	
	0.25; 4	Control conditions; 3-yr seedlings	% injury	Growth	—	3	—	—	42	
				Postexposure	—	19	—	—	34	
Ash, white	0.25; 4	Control conditions; 1-yr seedlings			16	27			521	
			% injury	Growth	49	66				
				Exposure	67	51				
				Postexposure	54	63				
Bean, cultivar Pinto	0.40; 1	Control conditions; PP = 8 h; ET = 27° C			16	21	27	32		d
			% injury	Growth	—	39	57	76		
	0.40; 1	Control conditions; PP = 8 h; GT = 27° C	% injury	Exposure	41	32	57	89		
Tobacco, cultivar Bel W_3	0.40; 1	Control conditions; PP = 8 h; ET = 27° C			16	21	27	32		d
			% injury	Growth	—	31	42	61		
		Control conditions; PP = 8 h; GT = 27° C	% injury	Exposure	58	44	42	11		

[a] PP = photoperiod; ET = exposure temperature; GT = growth temperature.
[b] Time of temperature treatment.
[c] Temperatures are shown as day or as day/night values and are underlined.
[d] Dunning and Heck, personal communication.

movement would be expected to play a role under ambient conditions, because of its known effect on leaf boundary layers, but it probably has little effect in chamber work, unless wind velocities are greater than 1 mph (1.6 km/h).[41,176,202]

It is widely believed that vegetation growing in the humid eastern United States would be severely injured if oxidant concentrations reached the daily peak concentrations (0.20–0.40 ppm) commonly experienced in the less humid sections of California. An air pollution episode that occurred on July 27–30, 1970, in the Washington, D.C., area is indicative of what may happen. During this 4-day period, the peak oxidant concentrations ranged from 0.14 to 0.22 ppm and were accompanied by a low concentration of sulfur dioxide (0.04 ppm). Oxidant injury was observed on 31 tree, 15 shrub, and 18 herbaceous species in an area of 72 mi^2 (about 187 km^2). Increased emission of the precursors of photochemical-oxidant formation could result in repeated episodes of acute injury or even chronic injury to eastern vegetation.

Several workers have explored the relationships between ozone effects on vegetation and several climatic factors. Thus, we have some understanding of how these factors may affect a plant's response to ozone and probably to ambient oxidants. The few reports on these interactions[122,123] suggest that much remains to be learned about how these climatic factors interact in causing a plant response. Studies to date suggest that stresses applied during exposure have their greatest effect on stomatal activity, whereas those applied during growth affect a physiologic basis of resistance that is not related to stomatal function. Postexposure reactions would no doubt be physiologic. Interactions between growth and exposure conditions would combine these stress factors. A physiologic stress may be so pronounced that the normally responsive stomatal mechanism would not function during exposure.[122] More emphasis needs to be placed on the study of multiple-stress interactions.

EDAPHIC CONDITIONS

Soil-water stress during growth and exposure to oxidant is probably the most significant environmental factor affecting plant response. Khatamian *et al.*[247] found less reduction in chlorophyll content and an increase in dry-matter production in tomato exposed to ozone at 0.50 or 1.00 ppm for an hour, if plants were grown under a water stress that did not itself cause a reduction in growth. Plants in the three-leaf stage were most sensitive to ozone if grown in optimal soil moisture (Table 11-12). Markowski and Grzesiak[307] found protection from ozone in bean and barley grown under drought conditions. Several early field

TABLE 11-11 Response of Plants to Ozone, as Conditioned by Growth or Exposure Humidity

Plant Species	Ozone Concentration, ppm; h	Notes[a]	Growth or Exposure[b]	Response, % Injury at % RH[c]				Reference
Pine, Virginia	0.25; 4	Control conditions; 3-yr seedlings		60	85			97
			Exposure	4	25			
			Growth	50	58			
	0.25; 4	Juvenile	Exposure	1	35			
Bean, cultivar Pinto, and Tobacco, cultivar Bel W_3, averaged	0.40; 1	Control conditions; PP = 8 h		45	60	75	90	[d]
			Growth					
			45% EH	36	39	41	31	
			90% EH	73	67	81	80	
		Control conditions; PP = 8 h; 75% GH	Exposure	41	53	70	81	
Bean, cultivar Pinto	0.40; 1	Control conditions; PP = 8 h; 2,000 ft-c (21,529 lx)		60	80			122
			Growth	66	78			

		Control conditions; PP [a] = 8 h	Exposure	52	67		
Tobacco, cultivar Bel W_3	0.40; 1	Control conditions; PP = 8 h; 2,000 ft-c (21,529 lx)	Growth	42	36		122
		Control conditions; PP = 8 h	Exposure	33	36		
Tobacco, cultivar Bel W_3	0.30; 1.5	Control conditions; 31 °C		26	51	95	381
			Exposure	9	39	50	
Bean, cultivar Pinto	0.20; 1.5	Control conditions; 31 °C	Exposure	0	0	55	381
Ash, white	0.25; 4	Control conditions; 1-yr seedlings		60	80		521
			Growth	33	46		
			Exposure	38	41		
			Postexposure	36	41		

[a] PP = photoperiod; GH = growth humidity; EH = exposure humidity.
[b] Time of humidity treatment.
[c] Humidity values are underlined.
[d] Dunning and Heck, personal communication.

TABLE 11-12 Effects of Soil Moisture on Response of Selected Plants to Oxidant Stress

Plant Species	Oxidant Concentration, ppm; h	Type of Response	Response, % Moisture Conditions (high → low)*			Reference
Tobacco, cultivar Catterton			Irrigated	Normal		324
	Ambient oxidant	% injury	29	11		
Tomato, cultivar Fireball			90% turgid	80% turgid		
	1.00; 1.5	% reduction in chlorophyll	54	10		6
	1.00; 1.0	% reduction in chlorophyll	67	24		247
	0.50; 1.0	% reduction in chlorophyll	36	+3		247
	1.00; 1.0	% reduction leaf dry wt	48	+40		247
Beet, garden			−40 kPa	−440 kPa	−840 kPa	369
	Control	% reduction in dry wt of storage root from nonsaline control	0	24	68	
	0.20; 3 (daily for 38 days)		40	52	69	
Bean, cultivar Pinto			−40 kPa	−200 kPa	−400 kPa	215
	Control	% reduction in shoot dry wt from nonsaline control	0	18	78	
	0.15; 2/day (63 days)		27	42	87	
	0.25; 2/day (63 days)		93	91	88	
	Control	% reduction in root dry wt from nonsaline control	0	25	65	
	0.15; 2/day (63 days)		25	28	78	
	0.25; 2/day (63 days)		91	89	79	

*Special soil-moisture conditions are underlined. kPa = kilopascals (bars × 10^2).

studies[102,448,511] showed a close correlation between soil moisture and oxidant injury to several tobacco cultivars. Field observations generally suggest that sensitive plants may become resistant under drought conditions. It is possible that drought during growth causes physiologic changes within the plant that increase resistance, whereas similar conditions during exposure may reduce stomatal opening. Adedipe *et al.*[6] reported some stomatal closure in tomato exposed to ozone at 1.00 ppm for 1.5 h. However, closure may have been triggered by the low light intensity of exposure (50 ft-c, or about 540 lx). Rich and Turner[411] found rapid stomatal closure in pinto bean during a 30-min exposure to ozone at 0.20–0.25 ppm, if plants were grown under a soil-moisture stress. Closure under optimal water availability was slower. They also reported rapid closure in pinto bean and Bel W_3 tobacco if the plants were conditioned and exposed to ozone at a relative humidity of 37%; this was not found at 73%. Their evidence suggests that ozone may induce more rapid closure when plants are already under some type of water stress. Ting and Dugger[470] related activity of two tobacco cultivars to the size of the root systems; those with larger root systems were more sensitive. Several workers have recommended withholding water from greenhouse and irrigated crops during times of high pollution potential.

Starkey[441] found that a sensitive bean exposed to subacute concentrations of PAN was sensitive to postexposure drought, but more resistant plants were not. This was related to effects of PAN on membranes.

In perennial vegetation, adequate soil moisture in the early season interacts to allow open stomata, and thus oxidant injury occurs. When soil moisture becomes limiting, the nonfunctional stomata of injured leaves may remain open, thus increasing transpiration. The moisture stress induced in the late season is additive with oxidant injury, because both increase defoliation.

Oertli[368] found that both salinity and low soil moisture increased the resistance of sunflower to ambient oxidants. He used four different-strength salt solutions from 0.75 to 6 atm and two soil-moisture levels (0.3 and 0.8 atm). Ogata and Maas[369] exposed garden beet for 38 days to ozone at 0.20 ppm and varied exposure time from 0 to 3 h/day. The garden beet was protected from ozone injury and yield reductions at high soil salinity, but this protection was offset by growth reductions caused by the salinity (Table 11-12). The storage root was the most sensitive yield indicator for both salinity and ozone stress. They found that yield was not affected until the daily ozone exposure was 2 h (-40 kPa) or 3 h (-440 kPa). Ozone had no effect at -840 kPa. Hoffman *et al.*[215] reported similar results with pinto bean exposed to four ozone concentrations for 2 h/day from seed to harvest. They had three degrees of

TABLE 11-13 The Interaction of Ozone and Salinity on Biomass of Pinto Bean Exposed to Ozone[a]

Osmotic Potential, kPa	Ozone Concentration, ppm	Reduction from Control Weight of Plants Grown at −40 kPa and Not Exposed to Ozone, %							
		Dry Wt of Leaves				Dry Wt of Roots			
		Ozone Dose, ppm-h[b]				Ozone Dose, ppm-h[b]			
		0	0.30	0.60	0.90	0	0.30	0.60	0.90
−40	0.15	0	14	28	67	0	18	53	72
	0.30	0	36	68	75	0	64	83	87
−240	0.15	25	31	41	50	24	25	35	51
	0.30	25	39	70	76	24	50	81	85
−440	0.15	59	65	60	67	47	49	40	53
	0.30	59	55	63	75	47	42	56	74

[a] Data from Maas *et al.*[280]

[b] Specific dose was given daily for 14 days; the duration of exposure can be determined from ozone concentrations used.

nutrient osmotic potential and used sodium chloride to increase salinity. The 0.15-ppm treatment at −40 kPa gave a 46% reduction in bean fresh weight, whereas no yield was found at 0.25 ppm and above. Results for top and root dry weights were similar (Table 11-12).

Maas *et al.*[280] studied the response of pinto bean to ozone and salinity as conditioned by concentration and duration of exposure (Table 11-13). They found that the injury and growth reductions were sigmoidal at the low salinity (Tables 11-3, 11-5, and 11-13). Salinity suppressed plant growth, but extended the ozone tolerance for both growth and injury response. In this study, the roots were more severely affected than the tops. However, in contrast with their earlier study,[215] the pinto beans were exposed for only 14 days and harvested as young plants. Maas *et al.*[280] also looked at nutritional effects; although some redistribution of nutrient was found (with respect to calcium, magnesium, potassium, and nitrogen), no serious nutritional imbalances occurred. In the last of a series of excellent papers on salinity effects, Hoffman *et al.*[214] studied the effects of four ozone concentrations (2 h/day for 21 days) at four levels of salinity on biomass production in alfalfa (Table 11-14). As in the earlier studies, the ozone effect decreased with increasing salinity. However, in contrast, they found that −200 kPa gave protection from ozone with no effect of salinity on biomass production. This suggests that moderate salinity, in areas of high oxidant, could protect forage legumes (such as alfalfa) from yield reductions. They also found that both high salinity and ozone increased water-use efficiency of alfalfa and leaf diffusion resistance. These reports on salinity suggest that we are dealing with a moisture stress problem. Although this may involve stomatal response, it is more likely that we are dealing with a changed physiologic state that increases the resistance of the plants to ozone effects. At high salinity, both phenomena may play a role, but salinity itself can cause major yield reductions in the crops reported on here.

The importance of soil fertility in the response of plants to oxidants is not understood. No research has attempted to explore, in depth, the nutritional interactions with oxidants. Nitrogen nutrition has received the most attention. Menser and Street,[324] in an ambient-oxidant study, reported an increase in tobacco fleck with increasing application of soil nitrogen. This was also found in spinach exposed to ozone.[47] Leone *et al.*[273] reported that nitrogen concentrations optimal for growth produced the most sensitive tobacco, whereas either higher or lower nitrogen applications increased resistance to ozone. The opposite was reported for White Gold tobacco, in which the optimal nitrogen concentration for growth gave the greatest resistance to ozone.[282] Ormrod *et al.*[373] found no effect of two nitrogen concentrations on growth of radish exposed to

TABLE 11-14 Interaction of Ozone and Salinity on Top Growth of Alfalfa[a]

Ozone Concentration, ppm[b]	Reduction from Control Weight of Plants Grown at −40 kPa and Not Exposed to Ozone, %[c]			
	−40 kPa	−200 kPa	−400 kPa	−600 kPa
0	0	+2	24	35
10	16	11	29	41
15	26	14	33	49
20	39	23	31	48

[a] Data from Hoffman *et al.*[214]
[b] Concentration shown was given 2 h/day for 21 days.
[c] Values that differ by 10 are probably significantly different.

ozone. It is apparent that nitrogen concentrations and other conditions used in these experiments were not sufficiently critical for an evaluation of nitrogen–oxidant interactions. Craker[76] reported that increased nitrogen in the nutrient increased chlorophyll loss in duckweed exposed to ozone.

Several studies have explored the importance of phosphorus in controlling the sensitivity of plants to oxidants. Work by Ormrod *et al.*[373] suggested that radish grown at 20° C is more sensitive to ozone at a higher phosphorus concentration, but that phosphorus has no effect at a growth temperature of 30° C. Ripaldi and Brennan[416] reported an increase in tissue phosphorus for pinto bean after exposure to ozone. They did not determine nutrient phosphorus. Leone and Brennan[272] reported an increased ozone injury to tomato with increasing nutrient and tissue phosphorus (1.5–62 ppm). Brewer *et al.*[47] found an interaction between potassium and phosphorus in the response of spinach to ozone. They reported that, at low phosphorus, an increase in potassium tends to increase injury, but, at high phosphorus, the potassium increase tends to reduce injury. Craker[76] reported a similar effect on duckweed; chlorophyll loss increased with increasing potassium at a medium phosphorus content, but decreased with increasing potassium at zero or higher phosphorus content. There is an apparent interaction between these nutrients, but its importance is not known.

Dunning *et al.*[123] found that pinto bean and soybean were more sensitive to ozone at low nutrient potassium, whereas Adedipe *et al.*[5] reported that low nutrient sulfur increased the response of Blue Lake snapbeans to ozone. McIlveen *et al.*[311] reported increased injury with increasing soil zinc. Craker[76] found no change in the response of duckweed to ozone when total salt was varied from one-tenth to one-half

of a full-strength nutrient solution. The response in a full-strength nutrient was about one-third less. Soil nutrition probably plays a fairly important role in plant response to pollutants only in cases of nutrient imbalances, although total salts should affect response under some conditions. In soils with balanced fertilizer, plants may respond fairly uniformly to oxidant stress. Table 11-15 summarizes several nutritional studies.

Stolzy *et al.*[444] found that low oxygen partial pressures over soil (0–10 mm Hg) for 40 h effectively protected tomato from injury by ozone (0.35 ppm for 3 h). These anaerobic conditions also decreased water use and general vigor. In later work,[443] Stolzy *et al.* reported that, with anaerobic soil conditions, the photosynthetic rate stablized within 24 h, well below the control (32%). Exposure to ozone (at 0.17 ppm for 3 h) caused no injury, but caused a marked reduction in the photosynthetic rate of the control with a slight increase in photosynthetic rates in plants under anaerobic conditions. They also showed that a 3-h period of anaerobic treatment of the soil affected the plant response to an ozone exposure 22 h later. The anaerobic treatment showed less injury and the photosynthetic rate was reduced less than in the control plants. These changes were related to carbohydrate changes within the plants. These experiments suggest that even passing periods of low soil oxygen tension could reduce plant response to ozone stress.

Blum and Tingey[29] found that ozone reduction in soybean root growth and nodulation was a function of foliar impact. They found no direct effect of ozone on the roots or soil systems surrounding the roots.

POLLUTANT INTERACTIONS

Oxidant air pollutants exist as parts of a complex mixture of gases, many of which may be phytotoxic. However, expect for ambient-air studies and simulated photochemical-oxidant studies, little research was done with pollutant combinations until the classic work of Menser and Heggestad in 1966.[318] It is of interest that Thomas *et al.*[461] suggested that sulfur dioxide might lessen the effect of oxidants in causing foliar injury to pinto bean. Middleton *et al.*,[328] working with ratios of sulfur dioxide to ozone of from 4:1 to 6:1, did not observe an increase in injury, although they found that, at 4:1, ozone appeared to interfere with the expected sulfur dioxide injury. Heck[182] found that various combinations of ethylene, propylene, and acetylene, when mixed with products of irradiated propylene–nitrogen dioxide mixtures, did not decrease or increase the development of foliar injury.

Menser and Heggestad[318] first reported that exposure to mixtures of sulfur dioxide (0.50 ppm) and ozone (0.03 ppm) for 2 or 4 h caused 23–48% foliar injury to the sensitive tobacco cultivar Bel W_3, whereas the

TABLE 11-15 Effects of Various Nutrients on Response of Selected Plants to Ozone (Oxidant) Stress

Plant Species	Oxidant Concentration, ppm; h	Type of Response	Element	Response, % Nutritional Levels (low → high)[a]				Reference
Tobacco, cultivar White Gold	0.35; 48	% injury	Nitrogen	0.1 21	0.5 16	1.0 4	5.0[b] 22	282
Tobacco, cultivar Catterton	Ambient oxidant	% injury	Nitrogen	60 lb/acre 29		90 20	120 11	324
Tomato, cultivar Rutgers	0.18; 4	% injury	Nitrogen	28 mg/l 20	280 40	560 40	1120 20	273
Spinach, cultivar Viroflay	0.25; 9/day, 3 days	% injury	Nitrogen	10 ppm 3	30 ppm 18	90 ppm 22		47
Radish, cultivar Cavalier	0.25; 4	% growth reduction	Nitrogen	60 mg/l 50	300 mg/l 55			373
Lemna minor L. (duckweed)	0.35; 2	% chlorophyll reduction	Nitrogen	0 18	0.9-3.6 mM 28-42		4.5 mM 55	76

Tomato, cultivar Rutgers				1.5 ppm	15.5 ppm	62 ppm	273
	0.15; 3	% injury	Phosphorus	0	0	40	
	0.25; 3	% injury	Phosphorus	20	40	60	
	0.30; 3	% injury	Phosphorus	20	60	60	
Spinach, cultivar Viroflay	0.25; 9/day, 3 days			20 ppm	150 ppm		47
		% injury	Phosphorus				
			5 ppm K	14	13		
			40 ppm K	25	9		
Radish, cultivar Cavalier	0.25; 4			30 mg/l	150 mg/l		373
		% growth reduction	Phosphorus	35	43		
Lemna minor L. (duckweed)	0.35; 2			0	0.33	0.66[b]	76
		% chlorophyll reduction	Phosphorus	24	34	41	
Bean, cultivar Blue Lake	0.50; 2/day, 2 days			1.3 mg/l		32 mg/l	5
		% chlorophyll reduction	Sulfur	55		11	
Soybean, cultivar Dare	8 doses (acute)			105 mg/l		710 mg/l	123
		% injury	Potassium	40		23	
Bean, cultivar Pinto	8 doses (acute)	% injury	Potassium	26		18	123

[a] Nutritional values are underlined and show a number of different units.
[b] Relative to a full-strength nutrient solution.

concentrations of the individual gases produced no injury. This study stimulated plant scientists to develop research on pollutant combinations.

Reinert *et al.*[399] have suggested the use of the terms "simultaneous" (mixtures of pollutants), "sequential" (one pollutant followed by a second pollutant), and "intermittent" (when there is some period between sequential exposures) to refer to exposures in discussing studies of pollutant combinations. They also recommended terminology for use in describing plant response to pollutant combinations: less than additive, −; additive, 0; and greater than additive, +.

Ozone and sulfur dioxide mixtures are of special interest, because of their widespread occurrence and the greater than additive effect on Bel W_3 tobacco.[318] Concentrations of either or both that may cause foliar injury are found around major metropolitan areas throughout the world and are widespread throughout rural eastern United States. Macdowall and Cole[284] reported that the two-gas combination lowered the threshold for injury of tobacco (cultivar White Gold) by sulfur dioxide, but not the threshold for ozone injury. Macdowall *et al.*[285] defined the threshold in terms of dose when they reported the threshold at 20 pphm-h (0.20 ppm-h). This has not appeared true in several other reports,[318,482] nor within the results reported by Macdowall and Cole.[284] Symptoms reported, when sulfur dioxide was below the threshold for the specific plant, were similar to those reported for ozone.

Tingey *et al.*[482] exposed 11 species to different ratios of sulfur dioxide and ozone mixtures. There was no general trend in terms of how the ratios of pollutant concentrations influenced foliar injury; additive, greater than additive, and less than additive responses were noted (Table 11-16). They described an undersurface silvering and collapse of epidermal tissue, whereas the upper surface injury was generally an interveinal necrotic fleck or stipple (pigment accumulation). Menser and associates[156,210,321,323] determined the response of many *Nicotiana tabacum* types, several *Nicotiana* species, and various cultivars within *N. tabacum* to sulfur dioxide and ozone mixtures. They found, generally, that tobacco was more sensitive to the sulfur dioxide–ozone mixtures than to the individual pollutants, but the relative sensitivity was often similar to that shown to exposure to ozone, ambient oxidant, or both. A. S. Heagle and G. E. Neely (personal communication) compared the relative foliar injury among cotton and soybean cultivars exposed to sulfur dioxide–ozone mixtures and to the individual pollutants. The relative cultivar sensitivity to each pollutant was normally different. Jacobson and Colavito[231] found that sulfur dioxide at 0.04 ppm decreased the sensitivity of bean and increased that of vetch to ozone during a 4-h exposure. Mandl *et al.*[291] found that the response threshold of alfalfa to sulfur dioxide was increased by ozone

TABLE 11-16 Summary Effects of Sulfur Dioxide and Ozone Mixtures on Foliar Injury[a]

Plant Species	Response at Concentration Ratio, SO_2:O_3, ppm[b]			
	0.50:0.05	0.10:0.10	0.25:0.10	0.50:0.10
Alfalfa	−	+	+	+
Broccoli	+	+	0	0
Cabbage	0	0	0	+
Radish	0	+	+	+
Tomato	0	−	0	0
Tobacco, cultivar Bel W_3	+	0	+	+

[a] Data from Tingey *et al.* [482]
[b] + = greater than additive; 0 = additive; − = less than additive.

at 0.07 ppm during a 4-h exposure. Dochinger *et al.*,[109] Costonis,[70] and Houston[220] presented evidence that ozone-sulfur dioxide mixtures change the response of white pine to the individual pollutants. Dochinger *et al.*[109] and Houston[220] found that the mixture increased the amount of injury, whereas Costonis[70] reported less injury from the mixture than from sulfur dioxide alone. Both Costonis and Houston reported effects from sulfur dioxide and its mixture with ozone at concentrations of both gases well below those of other reports. It is possible that they used ultrasensitive clonal materials. Whatever the reason, this work needs verification. Applegate and Durrant[11] reported injury to peanut at sulfur dioxide-ozone concentrations and ozone concentrations well below those reported for other plants. Their work also requires substantiation. In the latter two cases, the concentrations reported were close to the detection limits of the monitors used.

Tingey *et al.*[479] found additive inhibition of top growth of radish and less than additive inhibition of root growth after exposure to sulfur dioxide-ozone mixtures. Tingey and Reinert[480] and Tingey *et al.*[483] exposed soybean, tobacco, and alfalfa to mixtures of sulfur dioxide and ozone and reported greater than additive inhibition of root growth of soybean, additive inhibition for tobacco, and less than additive inhibition for alfalfa. Heagle *et al.*[173] reported greater than additive effect on growth and yield in soybean, grown under field conditions, from a mixture of these gases, but the differences between the mixture and the ozone treatments were not significant. A. S. Heagle and L. L. Trent (personal communication) reported a less than additive effect on yield of peanut from a mixture of the two pollutants.

D. Weber (personal communication) reported reductions in plant growth and nematode populations from mixtures of ozone and sulfur dioxide, but these changes were similar to those caused by ozone alone.

Other combinations of pollutants with ozone, PAN, or both may be important, but have received little study. Matsushima[308] reported additive foliar effects on pinto bean and tomato from a mixture of sulfur dioxide and PAN and a less than additive effect on tomato from mixtures of ozone and nitrogen dioxide. Fujiwara[149] reported a greater than additive effect on pea from a mixture of ozone and nitrogen dioxide. Kress[256] and Kohut[253] studied the response of hybrid poplar to ozone–PAN mixtures. Kress used sequential exposures and found a greater than additive effect after most exposures; after others, he reported mixed responses. Kohut used simultaneous exposure and found all three responses in three replicates of a study. The reasons for these variations are unclear.

Fujiwara[149] and Reinert *et al.*[399] have recently reviewed the subject of pollutant interaction. Fujiwara gave a straightforward reporting of research that is fairly comprehensive, whereas Reinert *et al.* interpreted and analyzed results. Fujiwara also included some of his data on peas and spinach. His graph (Figure 11-3) showing the greater than additive response of pea to the mixtures of ozone and sulfur dioxide is of interest, because of the linear responses of the two ozone concentrations across sulfur dioxide concentration. Reinert *et al.*[399] developed some useful tabular material, some of which is shown in Tables 11-17 and 11-18.

Studies of pollutant interactions are preliminary. We are still not able to define adequately the total potential impact of pollutant combinations on the production of quality food, feed, and fiber. We do know that plant species respond differently to pollutant combinations and tive. We still do not understand variations in species or cultivar responses or the responses of plants grown or exposed under a variety of environmental stresses.

POLLUTANT-PATHOGEN INTERACTIONS

An important factor in the response of vegetation to oxidants (primarily ozone) is the presence of biotic pathogens. Such responses have been studied from several perspectives since Yarwood and Middleton[530] accidentally found that rust-infected bean leaves were less sensitive to photochemical oxidants (probably PAN). Several investigators have looked at the protection from ozone injury afforded to plants with active infections; others have noted that ozone injury increases the sensitivity of plants to

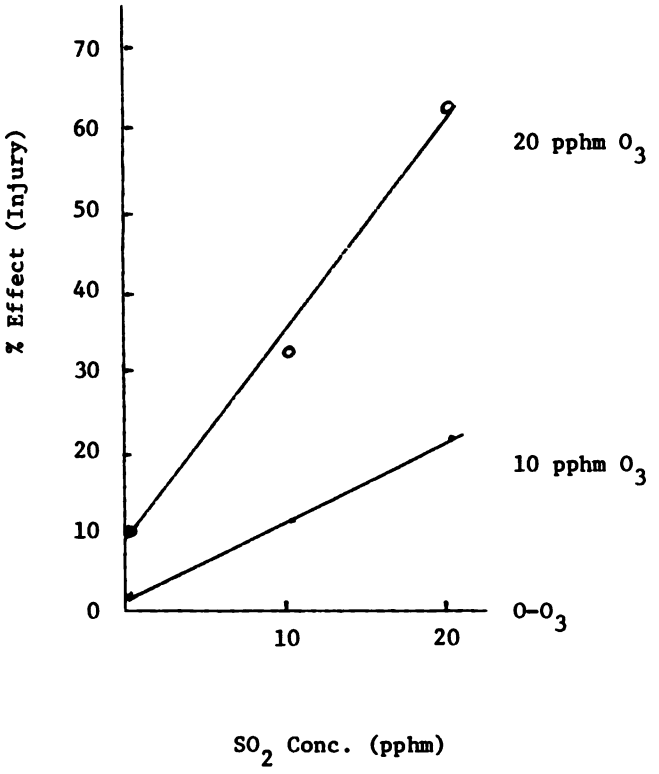


FIGURE 11-3 The effects of sulfur dioxide and ozone on percentage foliar injury to garden pea. Reprinted with permission from Fujiwara.[149]

infection; some have studied the effects of ozone on pathogens; and several have found no interacting effects.

Yarwood and Middleton[530] reported the first protective effects in 1954. Little more was done until about 1968. Protection of plants from ozone has been shown in several instances: rust infection of wheat;[178] *Botrytis cinerea* on broad bean;[288] *Pseudomonas phaseolicola* on pinto bean;[246] mosaic virus on tobacco;[39,344] three tobacco viruses (R. A. Reinert, personal communication); and mosaic virus on bean.[95,96] In the latter two cases, protection was reported without visible symptoms of the virus. This may be a general phenomenon, inasmuch as some protection was reported with very mild symptoms by Brennan and Leone.[39] This protective action has generally been ascribed to the production of a

TABLE 11-17 Foliar Response of Selected Plants to Sulfur Dioxide and Ozone Mixtures[a]

Plant Species	Concentration Ratio, SO_2:O_3, ppm	Exposure Duration, h	Foliar Injury, %	Response to Mixture[b]	Reference
Bean, garden	1.70:0.19	0.5	24	+	308
Bean, lima	0.25:0.05	4	0	0	482
Broccoli	0.50:0.05	4	17	+	482
Cabbage	1.00:0.10	4	28	0	482
Tomato	0.10:0.10	4	10	−	482
Radish	0.50:0.10	4	50	+	482
Alfalfa	0.50:0.10	4	60	+	482
Eastern white pine	0.025:0.05	6	26	+	220
Cotton (6 cultivars)	1.00:0.30	6	10-24	0	[c]
Tobacco, cultivar Bel W_3	0.25:0.03	4	41	+	318
Tobacco, cultivar Bel W_3	0.50:0.10	4	88	+	482
Tobacco, Md (6 cultivars)	0.50:0.10	2	20	+	323

[a] Data from Reinert *et al.*[309]
[b] + = greater than additive; 0 = additive; − = less than additive.
[c] Heagle and Neely, personal communication.

TABLE 11-18 Growth Response of Selected Plants to Sulfur Dioxide and Ozone Mixtures[a]

Plant Species	Concentration Ratio, SO_2:O_3, ppm	Exposure Duration, h	Plant Response, % reduction from control[b]	Response to Mixture[c]	Reference
Radish	0.05:0.05	8/day, 5 days/week, 5 weeks	10 TDW 55 RDW	0 –	479
Radish	0.45:0.45	4	16 TDW 70 RDW	0 0	399
Alfalfa	0.05:0.05	8/day, 5 days/week, 12 weeks	18 TDW 24 RDW	– –	399
Soybean	0.05:0.05	7/day, 5 days/week 3 weeks	24 RDW	+	483
Soybean	0.10:0.10	6/day, 5 days/week	52 TDW 63 seed wt	0 0	173
Tobacco	0.05:0.05	7/day, 5 days/week 4 weeks	32 TDW 49 RDW	0 0	399

[a] Data from Reinert *et al.*[399]
[b] TDW = top dry wt; RDW = root dry wt.
[c] + = greater than additive; 0 = additive; – = less than additive.

TABLE 11-19 Effects of Ozone on Plant Diseases[a]

Disease Affected	Effects	Ozone Concentration and Time	Reference
Botrytis on gladiolus and chrysanthemum petals	Fewer infections by conidia	Not measured	289, 290
Botrytis on geranium petals	Decreased pathogenesis	0.35 ppm; 4 h	296
Oat crown rust	Smaller pustules	0.10 ppm; 6 h/day, 10 days	169
Wheat stem rust	Fewer infections by urediospores	0.06 ppm; 6 h	177
Wheat stem rust	Decreased hyphal growth	0.06 ppm; 6 h/day, 3 days	177
Wheat stem rust	Decreased urediospore production	0.06 ppm; 6 h/day, 17 days	177
Barley powdery mildew	Fewer infections by conidia	0.10 ppm; 24 h after inoculation	179
Barley powdery mildew	Fewer infections by conidia	0.25 ppm; 8th through 12th h after inoculation	423
Botrytis on potato leaves	Increased incidence and pathogenesis	Ambient	300
Botrytis on broad bean leaves	More infections and pathogenesis	0.15 ppm; 8 h	287
Botrytis on potato leaves	More infections and pathogenesis	0.15–0.25 ppm; 6–8 h	300
Botrytis on geranium leaves	More infections and pathogenesis	0.07–0.10 ppm; 10 h/day, 15 days	297
Tobacco mosaic virus	More infections on pinto bean	0.30 ppm; 6 h	39

[a] Under ambient conditions, pollutants often exist in mixtures. Therefore, some of the effects observed in the field (ambient) may have been caused by interactions of more than one pollutant. Adapted from Heagle.[170] All work done in United States.

diffusible substance by the invading pathogen. This might be due to an alteration in plant metabolism caused by the viral infection.

Several investigators have reported that ozone injury increases the infectivity of some weak pathogens. This was originally investigated when a high incidence of *B. cinerea* was reported on potato that appeared to have severe ozone fleck.[300] The authors later reproduced the symptoms and disease incidence in greenhouse exposures. *B. cinerea* also invades ozone-injured geranium leaf tissue more rapidly.[297] It was suggested that the ozone lesions serve as infection sites for the weak pathogens.

Plant pathologists have used the possibility of pathogen-pollutant interactions to study the direct effects of ozone on the pathogen while it is active on plant tissue. This is the best way to study the effects of ozone and is the only way to study these effects on obligate parasites. The results have included reduced growth of crown rust uredia on oat[169] and bean,[405] reduced hyphal growth and number of urediospores of wheat stem rust,[177] reduced infectivity of barley by powdery mildew conidia,[179] and a small reduction in symptoms caused by *Fusarium oxysporium* on cabbage.[293] In general, it is felt that the results are due to changes in the host physiology, and not to direct effects on the pathogen. This may not be true for reduced infectivity of fungal spores.

There are several reports of the use of ozone with biotic pathogens, and the responses noted were independent of each other. These reports include *Lophodermium pinastri* infection of white pine,[73] *Fusarium* infection of cabbage,[293] *Botrytis* infection of poinsettia bracts,[295] rust on bean,[405] and brown root rot of tomato.[304] These results could reflect the conditions used.

Heagle[170] has developed an excellent review of this entire subject. He has also covered the direct effects of pollutants on pathogenic organisms. We have used the portion of this tabular material that covers the effects of ozone on plant diseases (Table 11-19).

OTHER FACTORS

Stark *et al.*[440] and Miller *et al.*[335] reported that oxidant (ozone) injury to ponderosa pine predisposed the trees to later invasion by pine bark beetles. The beetles increase the rate of decline and may be the final cause of tree mortality[67] (see Chapter 12). It is possible that oxidant stress in other parts of the country contributes to insect infestation in forest areas. Weber (personal communication) has shown that ozone and mixtures of ozone with sulfur dioxide (0.25 ppm, 4 h/day) can decrease the population of four nematodes associated with soybean. These

TABLE 11-20 Response of Most Sensitive Plant Tissue to Ozone Exposures

Plant Species	Ozone Concentration, ppm; h	Leaf Type	Plant Age, wk	Effect	Reference
Pine, Virginia	0.25; 4	Cotyledon	3-5	50% injury	98
		Primary	4-6	25% injury	
		Secondary	3-14	30% injury	
Pine, Virginia	0.25; 2	Primary	2	58% seedlings affected	20
Pine, shortleaf	0.25; 2	Primary	2	45% seedlings affected	
Pine, loblolly	0.25; 2	Primary	2	45% seedlings affected	
Pine, slash	0.25; 2	Primary	2	34% seedlings affected	
Maple, red	0.75; 7 h/day, 3 days	True	(90% expanded)	—	497
Cotton, cultivar Acala	0.70; 1	1*st* true	(2/3 expanded)	50% injury	469
Bean, cultivar Pinto	O_3-hexene	Primary	2-5	—	328
Soybean	0.50; 2	1*st* trifoliate	(3/4 expanded)	45% injury	477

types of interactions may be significant in areas of the country with significant oxidant pollution problems.

Heagle and Heck[175] found that Bel W_3 tobacco was predisposed to later oxidant injury by exposure to ambient pollutants. Macdowall[282] reported the same when the pre-exposure was to low oxidant and the later exposure was also low. Antagonism was noted when both doses were high.

Sensitivity of plants to ozone and PAN is conditioned by leaf maturity (Table 11-20). This has received its best documentation in the work by Bobrov.[31] Middleton *et al.*[328] found that pinto bean primary leaves were most sensitive at the age of 14–34 days when exposed to an ozone-hexene mixture. Under ambient oxidant conditions, leaves of White Gold tobacco did not fleck until a week or so after the logarithmic growth phase.[282] Ting and Dugger[469] found that cotton leaves were most sensitive at about 70% expansion and that sensitivity was rapidly lost. Stomatal resistance was low, even if plants were not resistant, all other things being equal. Ting and Mukerji[471] associated this with reduced amino acid and carbohydrate pools. There was a twofold increase in amino acids 24 h after a 1-h exposure to ozone at 0.80 ppm; it was a transient increase. This suggests that low pool compounds could reduce the speed of repair mechanisms. Tingey *et al.*[477] reported that the first trifoliate leaf of soybean was most sensitive during the latter stages of leaf expansion. However, their metabolite studies suggested that metabolic pools did not directly affect foliar sensitivity. Townsend and Dochinger[497] did definitive work on red maple seedlings exposed to ozone at 0.75 ppm, 7 h/day over 3 days. They reported that leaves of seedlings about 90% expanded were most sensitive, but that young leaves were tolerant.

Generally, studies have shown that plants are most sensitive to ozone at a physiologic age associated with nearly expanded leaves. Sensitivity is associated with functional stomata, intercellular spaces, and rate of cutin formation on cell walls.[31] This is not true in the case of PAN, in which leaves of a lower physiologic age, just before maximal leaf expansion, are most sensitive. Plants generally are more sensitive to oxidant pollutants during rapid growth stages and lose sensitivity as leaves mature. When oxidant episodes occur throughout the growing season, the older leaves, weakened during their stage of maximal physiologic growth, show early senescence.

Hanson *et al.*[164] reported an increased tolerance in petunia cultivars as they approach the flowering stage. This was true for both sensitive and tolerant cultivars, with the latter more strongly influenced. They suggested that bud development produced a diffusible substance that moved down the plant and acted as a protectant.

Adedipe *et al.*[4] found that Bel W_3 tobacco leaves were more sensitive to ozone when attached, rather than detached or used as leaf disks. Effects were seen as visual injury and change in chlorophyll content.

Davis and Wood[93,98] found that ages of Virginia pine needles influenced their response to a 4-h exposure to ozone at 0.25 ppm. Generally, cotyledons were more sensitive than primary needles, which were more sensitive than secondary needles. Secondary needles of seedlings were about as sensitive as those of 3-year-old trees. This suggests that seedlings may be good test plants for determining the sensitivity of mature trees. The cotyledons and secondary needles became resistant after 16 and 18 weeks, respectively, whereas the primary needles remained sensitive beyond 18 weeks. Berry[20] reported that Virginia, shortleaf, loblolly, and slash pines at 2-6 weeks from seed were most sensitive to ozone at 0.25 ppm for 2 h. Most species had peak sensitivities at about 2 weeks for the primary needles. The species were listed in order of sensitivity. These results may not be contradictory, inasmuch as the exposures and growth conditions varied greatly between the two reports.

Ozone is known to reduce nodulation in soybean[404,474] and in ladino clover.[252] The reduction in fixed nitrogen was related to a reduced nodule number, and not to nodule size. U. Blum (personal communication) found no direct effect of ozone on *Rhizobium* or on nodule formation. He attributed the reduction in nodule number to the reduction in available energy in the root tissue. This reduction in nitrogen fixation could affect total biomass and agricultural production, especially in areas of high oxidant pollution and low soil nitrogen. Kochhar[252] also reported an inhibition of plant growth and nodulation of *Trifolium repens* (clover) when the plants were treated with root exudates from fescue grass exposed to ozone. If these factors are widespread, they could change the competitive ability of plant species, with a resulting change in plant diversity and a possible decrease in agricultural productivity.

Although there has been considerable interest in understanding how various factors, including air pollutants, affect the response of plants to pesticides, especially herbicides, very little has been done with air pollutants. Hodgson and associates[211-213] first showed an effect of ozone on the metabolism of herbicides. They found that ozone inhibited the dealkylation of atrazine in corn and altered the pathway of diphenamid metabolism in tomato. The changes could be beneficial, if oxidants increase pesticide degradation, or harmful, if oxidants stop biologic breakdown at a toxic intermediate. Carney *et al.*[57] reported that the herbicide pebulate in combination with ozone gave a greater than additive response on White Gold tobacco and that chloramben did the same with Delhi 34 tobacco. They reported a less than additive response

of both tobacco cultivars to the combination of benefin and ozone. These and other herbicides acted independently of ozone exposure on tomato and white bean. Ordin *et al.*[370] found that *Avena* coleoptile growth was less inhibited by PAN when 2,4-D was used in amounts giving optimal growth. These interactions with herbicides need additional investigation, to determine whether the responses noted are of general importance. Research needs to be directed at possible interactions between atmospheric pesticides (vapors or fine particles) and oxidant air pollutants.

Another interaction has recently been reported between cadmium applied to soil and ozone exposure of cress.[85] If cadmium potentiates the ozone response of cress, maybe other heavy metals respond in a similar fashion.

DISCUSSION

A complete understanding of the many factors that affect the response of vegetation to oxidant pollutants is probably impossible. An understanding of the individual factors is possible, however, and much is already known; but the interactions between some of these many factors are unclear. It is possible that, as the mechanism of response becomes better understood, we can develop an expectation as to how various factors will interact. However, a well-trained and knowledgeable investigator can develop a subjective estimate of response that is repeatable. Inherent genetic resistance is probably the most important factor that affects the response of a plant to an oxidant dose. However, the factors discussed here will influence the severity of response of sensitive genotypes. Even normally resistant genotypes can be injured by appropriate combinations of other factors.

Genetic or other factors that induce stomatal closure will reduce plant sensitivity to oxidant pollutants. Generally, the sensitivity of plants at the time of exposure is controlled primarily by factors that affect the stomatal aperture. The internal resistance to gas flow may also influence leaf sensitivity. Factors that affect sensitivity during growth usually cause physiologic changes in the plant that tend to make it more resistant to the added stress of oxidant. Many of these stresses may alter membrane physiology and make the membranes either more or less sensitive to oxidant stress.

Dose–Response Relationships

The development of criteria for setting air quality standards requires a sufficient data base relating a given dose (concentration of pollutant × duration of exposure) of oxidant (e.g., ozone or PAN) to some mean-

ingful effect on plants. An understanding of dose response is also important for a basic understanding of the mechanism of oxidant effects on plants. Heck and Brandt[183] have suggested that an ideal criterion would be a set of standard equations that would relate response to concentration and duration of exposure and that would reflect the effects of all other factors that control the response of the plant. If these equations were developed, they would be different for acute and chronic exposures and perhaps specific for individual species or cultivars. Such a depth of coverage would have little practical value for overall understanding or for ambient air quality standards. However, some average equation involving groupings of plants expressed with confidence limits might permit a more reasonable interpretation of dose-response functions under average ambient conditions. Discussions of the relationship of time, concentration, and response are found in several publications.[183,279,505]

Data regarding the chronic effects of oxidants (including ozone and PAN) have not been gathered on a scale wide enough to permit the development of usable equations in regard to concentration and duration of exposure. Much of the information available was discussed and tabulated earlier in this chapter. The data suggest that chronic and acute-chronic effects with resulting yield and biomass reductions can occur when average concentrations of oxidant (ozone) are between 0.05 and 0.10 ppm for 2-6 h/day over a number of days during the growing season. This type of statement is not yet possible for PAN, but in the Los Angeles basin it is always a factor in ambient-oxidant studies.

Even that kind of information is not available for forest species. Other than chronic injury to white pine (associated with ozone, sulfur dioxide, and their mixtures), no clearly defined examples of chronic injury from ozone have been reported for eastern forests, and no information is available on PAN. It is of interest that both Virginia and jack pine appear more sensitive than white pine to acute ozone exposures,[22,99] but chronic symptoms have not been observed in either species. The relationship between oxidant dose and injury in the San Bernardino Mountains area[453] suggests that ponderosa pine is moderately to severely injured in areas that receive oxidant at above 0.08 ppm for 12-13 h each day (Chapter 12). Ponderosa pine seems to be the most sensitive western pine, but in some areas Jeffrey pine is about as sensitive. White fir, incense cedar, and sugar pine all appear more tolerant, even to the high oxidant concentrations in the San Bernardino Mountains.[334] PAN may play some role in the chronic responses noted in the western forest species, particularly by broadleaf deciduous trees and some shrubs.

The information available on exposures to PAN and its homologues is not sufficient to develop dose-response curves. Taylor[452] reported

comparative results from exposures to PAN, PPN, PBN, and PisoBN. He found increasing sensitivity in the first three homologues. Drummond[112] found a nonlinear response of petunia to PAN that was similar to those reported for ozone. Representative information for PAN is presented in Table 11-21. Davis[92] was unable to injure cotyledons or primary needles of ponderosa pine with PAN concentrations of 0.08, 0.20, or 0.40 ppm during 8-h exposures. A species-sensitivity table for PAN was developed in the *Atlas*.[457]

The information on acute exposures of forest species to ozone is limited.[22,93,99,110,201,334,521,524] The results of these reports suggest that many eastern deciduous species are sensitive to exposures to ozone at 0.20–0.30 ppm for 2–4 h. Generally, the eastern conifers are somewhat more sensitive and western conifers somewhat less. These results are included in the summary data presented in this section. Treshow and Stewart[501] developed an extensive list (70 plants) from two plant communities, but it is impossible to determine percentage effects from their data. They found some injury to several species at 0.15 ppm for a 2-h exposure. This list of plants should interest investigators concerned with plant communities.

OZONE SENSITIVITY

This section deals with published data related to short-term exposures of sensitive plants to ozone and the resulting responses. In most cases, the measure of response is a subjective estimate of visible injury. However, various growth measures have been reported in some research, and correlations between injury and growth measures are often possible (Table 11-3). These acute ozone effects have received sufficient study to permit the construction of preliminary models to relate time, ozone concentration, and plant response for a number of plant species.

A graphic expression was developed for pinto bean and Bel W_3 tobacco exposure to ozone by Heck and Dunning.[187] Later work with a number of plants permitted the development of a simplistic model derived as an empirical relationship between ozone concentration, time, and response;[192] this gave a reasonable interpretation of acute response up through a single 8-h exposure. It also permitted the development of a reasonable acute threshold concentration for a number of species. The equation was a variant of the O'Gara equation for sulfur dioxide and is shown as

$$C = A_0 + A_1 I + A_2/T,$$

where C is concentration of ozone; I is the response measure, in percentage injury or percentage reduction from control; T is time, in hours;

TABLE 11-21 Effects of Time and Concentration on Foliar Response of Selected Plants to PAN

Plant Species	PAN Concentration, ppm	Time, h	Plant Response	Reference
Controlled Exposures:				
Petunia	0.10	5	Severe injury	442
Petunia	0.12	1	11% injury	139
Petunia, cultivar Rosy Morn	0.14	1	33% injury	452
Petunia, cultivar White Cascade	0.05	2	23% injury	112
	0.10	1	19% injury	
	0.20	0.5	15% injury	
Bean, cultivar Pinto	0.02	8	44% injury	452
	0.04	4	90% injury	
	0.14	1	55% injury	
Bean, cultivar Pinto	0.10	5	Moderate injury	442
Bean, cultivar Sanilac	0.12	2	64% injury	441
Acer, Fraxinus, Gleditsia, and *Quercus* spp.	0.20-0.30	8	Variable symptoms; sensitivity related to tissue age	111
Ambient Exposures:				
Petunia, tomato, Swiss chard, others	0.025-0.03	Several	Typical symptoms	491
Petunia, tomato	*ca.* 0.014	4	Acute injury	452
Petunia, tomato, romaine lettuce, others	0.015-0.02	4	Acute injury	457
Tomato, cultivar Pearson	0.10	5	Severe injury	444

and the constants A_0, A_1, and A_2 relate to inherent and external factors affecting sensitivity. Heck and Brandt[183] and Linzon *et al.*[279] critically reviewed these dose-response relationships. They summarized much of the available acute-response data and analyzed the data on the basis of the O'Gara equation. Heck and Brandt[183] reported the 95% confidence curves for 5% and 33% response; Linzon *et al.*[279] reported the curve for 5% response, along with data points. They constructed a threshold curve from the minimal data points shown. These reviews attempted to develop an average response across much of the useful information on acute plant response.

Larsen and Heck[262] analyzed data on the foliar response of 14 plant species to ozone (from Heck and Tingey[192] and Heck *et al.*[187]). They depicted the data using a mathematical model with two characteristics: a constant percentage of leaf surface is injured by an air pollutant concentration that is inversely proportional to exposure duration raised to an exponent (Figure 11-4); and, for a given exposure duration, the percentage leaf injury as a function of pollutant concentration fits a log-normal frequency distribution (Figure 11-5). The complete leaf-injury equation combines the equations shown in Figures 11-4 and 11-5. This equation expresses pollutant concentration as a function of the other variables:

$$c = m_{g\ \mathrm{hr}}\, s_g^{\,Z}\, t^{p}$$

where c is concentration, in parts per million; $m_{g\ \mathrm{hr}}$ is geometric mean concentration for 1-h exposure; s_g is standard geometric deviation; Z is number of standard deviations from the median (injury); t is time, in hours; and p is slope of injury line on logarithmic paper. From the data for pinto bean in Figures 11-4 and 11-5:

$$c = (0.31)1.44^{Z} t^{-0.57}.$$

Thus, c = 0.10 ppm to give 10% injury in a 3-h exposure. The summary table of Larsen and Heck for the exposure of 14 plant species (two cultivars of corn) appears as Table 11-22. From this analysis, they suggested that averaging times of 1, 3, and 8 h should be used in the development of oxidant standards for the protection of vegetation.

For the present report, the literature was critically reviewed for information on three measures: duration of exposure, ozone concentration, and plant response. From this review, 74 references were found to have usable data. The following criteria were established before the data were included:

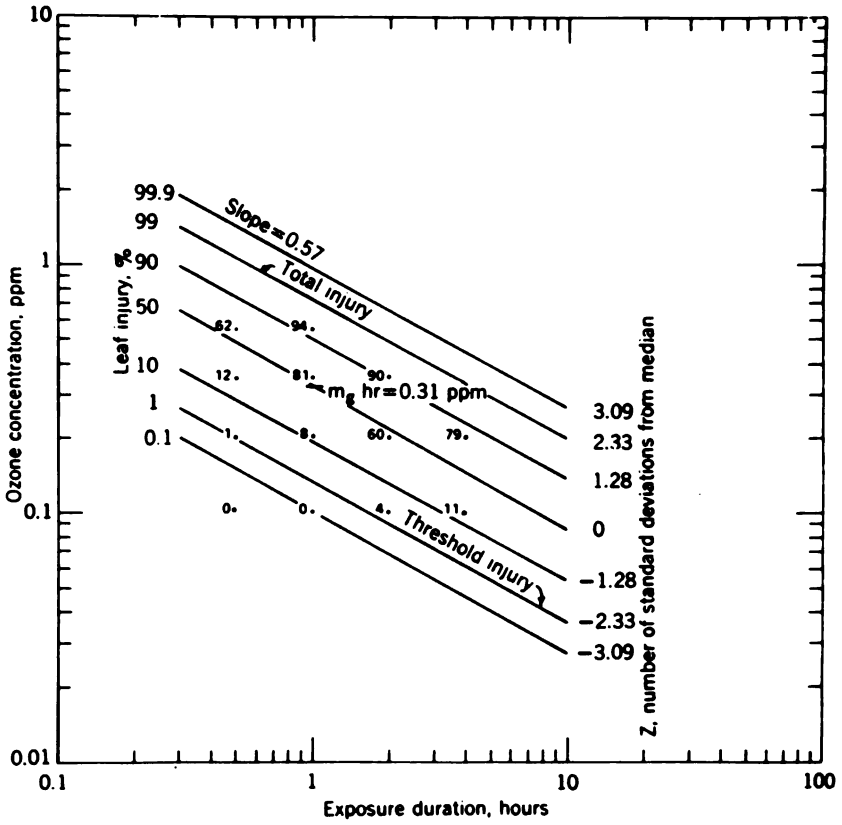


FIGURE 11-4 Percentage leaf injury in pinto bean plants exposed to various ozone concentrations for various durations—concentration plotted vs. exposure duration. $m_g = m_{g\,h}t^p$, where m_g = geometric mean concentration for a particular exposure duration; $m_{g\,h} = m_g$ for 1 h; t = time, in hours; and p = slope of injury line on logarithmic paper. Reprinted with permission from Larsen and Heck.[262]

1. The monitoring technique and method of calibration had to be defined. Of the references included, 40% reported use of a calibrated Mast instrument; 50% reported use of the Mast uncalibrated or the calibration procedure was impossible to determine; 4% reported using a 20% buffered potassium iodide wet chemistry; for 3%, the technique could not be determined, but other procedures suggested that the data were reliable; one paper reported an ultraviolet method; another reported an automated iodometric technique.

2. The plant response must have been subject to the development of

a percentage evaluation in comparison with the control plant. About 93% of the responses were foliar injury; for these we often made a subjective evaluation of scales used, on the basis of injury description given. Other responses included a reduction in chlorophyll, yield, leaf area, root dry weight, or top dry weight and increase in respiration.

3. The general experimental procedures used needed to conform to those generally reported around the country.

The data used came from a number of laboratories where growth and exposure conditions were highly variable and plants were exposed at different ages, at various times of year, and under various soil conditions.

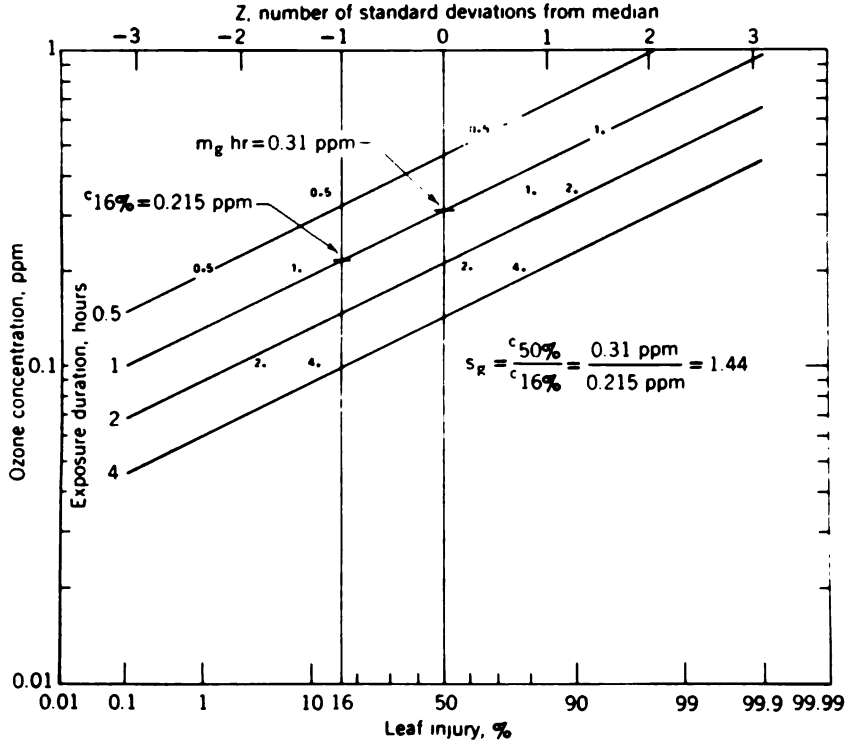


FIGURE 11-5 Percentage leaf injury in pinto bean exposed to various ozone concentrations for various durations—concentration plotted vs. percentage injury. $c = m_g s_g^Z$, where c = concentration, in ppm; m_g = geometric mean concentration for a particular exposure duration; s_g = standard geometric deviation; and Z = number of standard deviations that the percentage of leaf injury is from the median. (Z for 10% injury is −1.28.) Reprinted with permission from Larsen and Heck.[262]

TABLE 11-22 Calculated Injury Parameters for Plants Exposed to Ozone[a]

Pollutant and Plant	Calculated Injury Threshold[b], ppm, for Exposure of			Leaf Injury Equation[c] Parameters			Multiple Correl. Coeff.	Injury Conc. Ratio, median/ threshold
	1 hr	3 hr	8 hr	$m_{h\ \mathrm{hr}}$	s_g	p		
Ozone								
a. Bean, Pinto	0.13	0.07	0.04	0.31	1.44	−0.57	0.98	2.3
b. Tomato, Roma	0.09	0.06	0.04	0.24	1.53	−0.35	0.98	2.7
c. Clover, Penscott Red	0.20	0.12	0.08	0.65	1.68	−0.43	0.89	3.3
d. Tobacco, Bel W-3	0.11	0.05	0.03	0.34	1.60	−0.68	0.93	3.0
e. Spinach, Northland	0.24	0.13	0.08	0.72	1.60	−0.55	0.89	3.0
f. Chrysanthemum, Oregon	0.36	0.26	0.20	1.79	2.00	−0.27	0.76	5.0
g. Begonia, Thous. Wonders	0.18	0.13	0.10	0.76	1.85	−0.31	0.90	4.2
h. Corn, Pioneer 509	0.18	0.10	0.05	0.80	1.91	−0.57	0.94	4.5
i. Corn, Golden Cross	0.15	0.09	0.06	0.83	2.11	−0.41	0.99	5.7
j. Bromegrass, Sac Smooth	0.16	0.11	0.07	0.64	1.80	−0.38	0.95	3.9
k. Oats, Clintland 64	0.09	0.06	0.04	0.47	2.02	−0.41	0.91	5.1
l. Radish, Cherry Belle	0.08	0.06	0.04	0.29	1.72	−0.32	0.93	3.5
m. Periwinkle, Bright Eyes	0.48	0.31	0.21	1.37	1.57	−0.40	0.94	2.8
n. Wheat, Wells	0.39	0.23	0.14	0.83	1.3	−0.4	0.94	2.2
o. Squash, Summer	0.17	0.17	0.17	0.70	1.83	−0.01	0.96	4.1

[a] Reprinted with permission from Larsen and Heck.[262]

[b] The injury threshold is arbitrarily defined here as 1% leaf injury in the median plant of an exposed set.

[c] $c = m_{g\ \mathrm{hr}} s_g^{z_t} p$.

We have assumed that these variations have given us some average over environmental conditions.

The 1,135 data points selected for use here were divided into three susceptibility groupings on the basis of information from Table VIII in Heck and Brandt[183] and Table 6-6 in *Air Quality Criteria for Photochemical Oxidants.*[505] These tables were modified on the basis of the analysis of the data used, and are presented in Table 11-23. The three susceptibility groups were: sensitive (471 data points), intermediate (373 data points), and resistant (291 data points). The plants (and appropriate references) in each susceptibility group are listed in Table 11-24. In addition, see "Index of Plant Names and Reference Numbers," the last section of this chapter. Note that the *italicized* references in Table 11-24 were *not* used in compiling the data for the generation of Figure 11-6 and Table 11-25.

The data developed for each susceptibility group were then analyzed with the equation of Heck and Tingey[192] (Figure 11-6 and Table 11-25). This is not to say that the data generated could not be used to develop a different or more complex model. However, this model presents a better interpretation of data than other simple models that have been used. It should be emphasized that the data presented are pertinent only to pollutant concentrations that produce an acute response in 8 h or less (two data points in the sensitive group were for 12 h). None of the infor-

TABLE 11-23 Ozone Concentrations for Short-Term Exposures that Produce 5% or 20% Injury to Vegetation Grown under Sensitive Conditions[a]

Exposure Time, h	Ozone Concentrations that May Produce 5% (or 20%) Injury, ppm		
	Sensitive Plants	Intermediate Plants	Resistant Plants
0.5	0.35–0.50 (0.45–0.60)	0.55–0.70 (0.65–0.85)	≥0.70 (0.85)
1.0	0.15–0.25 (0.20–0.35)	0.25–0.40 (0.35–0.55)	≥0.40 (0.55)
2.0	0.09–0.15 (0.12–0.25)	0.15–0.25 (0.25–0.35)	≥0.30 (0.40)
4.0	0.04–0.09 (0.10–0.15)	0.10–0.15 (0.15–0.30)	≥0.25 (0.35)
8.0	0.02–0.04 (0.06–0.12)	0.07–0.12 (0.15–0.25)	≥0.20 (0.30)

[a] Data developed from analysis of acute responses shown in Table 11-25 and Figure 11-6.

TABLE 11-24 List of Plant Species and Cultivars by Susceptibility to Acute Ozone Exposures[a]

Plant	Reference
SENSITIVE	
Alfalfa	367
Dawson	225
Glacier	225
Mesa-Sirsa	225
Moapa	225
Vernal	225
Williamsburg	225
Aspen, quaking	501
Bean	
Pinto	77, 94, 119, 123, 129, 185-187, 328, 389, 391, 416, 438, 456, 490
Seeway	55, 91
White	144
Sanilac	94, 514
Broccoli	
Calabrese	192
Chrysanthemum	
King's Ransom	249
Mango	249
Minn White	249
Mt. Snow	249
Red Mischief	249
Tranquility	249
Clover, red	
Kenland	45
Crimson Giant	402
Safflower	
Arizona 14154	229
Biggs	229
Jordan	229
Nebraska 1-1-5-1	229
Nebraska-4051	229
Nebraska-6	229
Nebraska-8	229
Pacific-2	229
Utah 1421-9-16	229
Ute	229
Soybean	367
Clark	340
Clark 63	340
Cutler	340
Dare	123, 228, 340
Haberlandt	340
Kent	228, 340
Lee	340
Lincoln	340
Peking	340
Richland	340
Roanoke	340
S100	340
Semmes	340
Wye	340

TABLE 11-24 (Cont.)

Plant	Reference
Speight G-3	322
Speight G-7	322
Speight G-36	322
Va 115	322
White Gold	192, 322
Wilson	323
Xanthi	156
Tobacco (*N. glutinosa*)	156
Tobacco (*N. rustica*),	
Brasilia	156
Tomato	
Heinz 1350	403
Marglobe	403
Pearson	403, 443
Red Cherry	403
Roma	192
Roma VF	403
Rutgers	272, 273
INTERMEDIATE	
Alfalfa	
Cherokee	225
Iroquois	225
Kanza	225
MSA-CW3An2	225
MSB-CW5An2	225
MSHp6F-An2W2	225
Saranac	225
Team	225
Yellow Supreme	*249*
Clover, red	
Chesapeake	45
Pennscott	45, 192
Clover, ladino	45
Clover, white sweet	45
Coleus	
Pastel Rainbow	3
Corn	
Golden Cross	192
Pioneer 509	192
Cucumber	
Chicago Pickling	389
Long Marketer	192
Duckweed	76
Grass, bent	
Highland	37
Kingstown	37
Grass, Bermuda	
Kansas P-16	37
Grass, brome	
Smooth Sac	192
Grass, Kentucky blue	
Delta	37
Merion	37
Grass, perennial rye	
Lamora	37
Manhattan	37
Grass, red fescue	

TABLE 11-24 (Cont.)

Plant	Reference
Soybean	
Amsoy	481
Arksoy	340
Chippewa 64	481
Clark 63	481
CNS	340
Dare	123, 477, 481
Delmar	228
Dunfield	340
Hark	481
Hawkeye	481
Kent	481
Lee	481
Ogden	340
P. I. 157474	340
P. I. 181550	340
Scott	192, 481
Traverse	481
Wayne	489
York	340
Spinach	
Bounty	*299*
Northland	192
Winter Bloomsdale	*299*
Spinach, New Zealand	*299*
Squash, summer	192
Tobacco	
56-92B	316
Ast-C	316
Tender Crop	94
Begonia	
Christmas	192
Scarletta	3
Chard, Swiss	
Fordhook Giant	192
Chrysanthemum	192
Ann Ladygo	*249*
Bonnie Jean	*249*
Bright Yellow Tuneful	*249*
Cameo	38
Dark Yellow Tokyo	*249*
Distinctive	*249*
Dolli-ette	*249*
Flair	*249*
Fuji Jess Williams	*249*
Fuji-Mefo	*249*
Golden Cushion	*249*
Golden Peking	*249*
Golden Yellow Princess Ann	*249*
Indian Summer	38
Jessamine Williams	*249*
Larry	*249*
Lipstick	*249*
Mandalay	*249*
Mermaid	*249*
Muted Sunshine	38
Oregon	192
Pink Chief	38

TABLE 11-24 (Cont.)

Plant	Reference
Hemlock, Eastern	99
Holly, English	40
Larch, Japanese	99
Lettuce	
Big Boston	402
Butter Crunch	402
Dark Green Boston	192
Grand Rapids Forcing	402
Great Lakes	402
Imperial #456	402
Romaine	402
Simpson, Black Seeded	402
Maple, sugar	199
Oats	
Clintland 64	192
Onion	192
Periwinkle	
Bright Eyes	192
Petunia	
Blue Danube	139
Blue Jeans	139
Blue Sea	139
Bonanza	3
Calypso	139
Canadian-All Double Mix	3
Cherry Blossom	139
Festival	139
Pine, Scotch	99
Poinsettia	192
Annette Hegg	58
Dark Red Annette Hegg	298
Eckespoint C-1	58
Mikkelwhite	298
White Annette Hegg	298
Snapdragon	
Floral Carpet	3
Rocket Mixture	3
Sorghum	
Martin	192
Soybean	
Cutler	228
Harosay	481
Hood	481
Pickett	481
Scott	192
York	228
Spinach	
Viroflay	47
Spruce, black	99
Spruce, blue	99
Spruce, Norway	99
Spruce, white	99
Sultana	
White Imp	192

Lilac Time	139
Parti Pink (Pink)	139
Peach Blossom	139
Peaches & Cream	139
Red Magic (Red)	139
Roulette	139
Victory	139
Warrior	139
Pine, eastern white	99
Pine, pitch	99
Pine, red	99
Tobacco	
Bel C	4
Hicks	315
Tomato	
Fireball	247
Heinz 1439	403
VF 145B	403
Vetch, crown	
Chemung	45
Penngift	45

[a] Plants are listed by common generic names followed by species common name. Cultivars are listed under the common names. Data from the references whose numbers are in italics were not used in developing Figure 11-6 or Table 11-25.

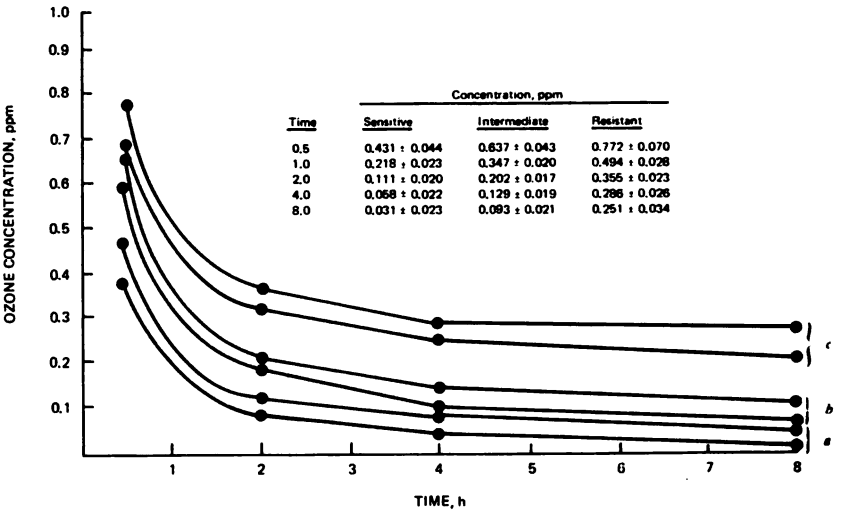


FIGURE 11-6 Ozone concentration vs. duration of exposure required to produce a 5% response in three different plant susceptibility groupings. The curves were generated by developing 95% confidence limits around the equations for "all plants" in each susceptibility grouping from Table 11-25. Curves: *a* = sensitive plants, *b* = intermediate plants, *c* = resistant plants.

mation should be extrapolated beyond this period. The data from exposure of the plants in Table 11-24 (except for italicized references) were used in the model. Several rather extensive lists of species and cultivar sensitivities to oxidants, ozone, and PAN are found in a number of references.[59,204,206,238,425,457,459,501,505]

The equations developed and the figures shown reflect the fact that concentration plays a greater role than time in the response of plants to ozone. Although the concept has long been accepted, it is often forgotten that equal doses do not necessarily give equal responses.[187,338] That is, a given dose applied over a short period produces a much greater plant response than an equal dose applied over a longer period. The concept of a concentration threshold actually ensures this basic dose concept. Although some disagree with the threshold concept with respect to the oxidant pollutants, the concept must have mechanistic validity, inasmuch as organisms have some inherent mechanism for detoxifying oxidants. This might not be true in the case of some individuals of a given species that might have lost the inherent protective mechanism. However, when we are dealing with a large number of species of vegetation and are attempting to develop some type of realistic oxidant dose response, we

must use the species as a whole, or at least a major subdivision (cultivar) of it. In these cases, in the work that has been done, there is a rational body of data to support a threshold concept.

The Plant as an Oxidant Sink

Heck and Brandt[183] discussed the effectiveness of green belts in relation to vegetation as a pollutant sink and concluded that vegetation probably acts as a major sink for air pollutants, including oxidants and ozone, over time, but has a relatively minor effect on oxidant concentrations during high-pollution episodes; is more effective in some seasons than others or with some cultural and management practices than others; and should not be considered an important contributor to short-term reductions in oxidant or ozone concentrations.

The concept of vegetation as a pollutant sink is significant to the atmospheric chemist and meteorologist who is attempting to develop air pollution material budgets. There is a need to know the ultimate sinks of air pollutants released by the activities of man. The preliminary research results available show attempts to understand the parts played by plants, soils, and soil organisms.

Turner *et al.*[502] reported that freshly turned soil exposed to air was a significant sink for ozone. They found that soil was about 50% as active as charcoal in removing ozone from air. Turner *et al.*[503] reported ozone flux within a maize field and a forest. They determined that vegetation could reduce ozone loss to soil when plants were under stress, but supported the view that vegetation and soil could be the primary sinks for ozone. Macdowall[281] found that soil absorbed twice as much ozone as mature field-grown tobacco growing on the soil. He concluded that soil is an effective sink and may play a protective role, in that it tends to keep the oxidant concentration within the canopy low. Abeles[2] suggested that most soil activity, in relation to reduced pollutant concentration, is associated with the metabolic activity of soil microorganisms. This may not be true with ozone, because it is such a reactive gas. Soils may play a more important role than has been suggested, but they are usually covered with vegetation and in general are probably less important than vegetation in acting as pollutant sinks.

Two reports[412,468] based on rather gross measuring techniques found a close correlation between ozone uptake and transpiration. These studies indicated that stomatal control is the prime factor in controlling pollutant uptake and that cuticular sorption is negligible in relation to stomatal absorption. These findings are generally supported by past work that indicates that stomata are the prime sites of pollutant entry

TABLE 11-25 Concentration, Time, Response Equations for Three Susceptibility Groups and for Selected Plants or Plant Types with Respect to Ozone[a]

Plants	[b] $C = A_0 + A_1 I + A_2/T$	R^2[c]	Threshold Concentration, ppm[d]	No. Data Points	Mean Values[e]			
					Conc., ppm	Time, h	Response, %	Dose, ppm-h
Sensitive								
All plants	$C = (-1.52 + 0.40)I + 21.3/T$	0.57	0.03	471	0.29	1.74	45.4	0.503
Grasses	$C = (-5.65 + 0.48)I + 29.1/T$	0.74	0.01	71	0.37	1.66	50.9	0.608
Legumes	$C = (4.52 + 0.36)I + 17.2/T$	0.46	0.09	100	0.34	1.42	48.1	0.480
Tomato	$C = (-8.23 + 0.43)I + 24.3/T$	0.58	None	28	0.31	1.58	56.5	0.493
Oat	$C = (-4.27 + 0.51)I + 27.3/T$	0.76	0.02	30	0.37	1.66	48.2	0.611
Bean	$C = (-0.98 + 0.38)I + 16.4/T$	0.58	0.03	62	0.30	1.23	47.2	0.370
Tobacco	$C = (2.45 + 0.34)I + 13.7/T$	0.52	0.06	197	0.23	1.98	38.9	0.448

Intermediate									
All plants	$C = (2.44 + 0.65)I + 29.0/T$	0.74	0.09	373	0.37	1.67	27.0	0.625
Vegetables	$C = (-0.79 + 0.64)I + 26.3/T$	0.79	0.06	25	0.41	1.29	33.5	0.532
Grasses	$C = (1.87 + 0.59)I + 29.2/T$	0.82	0.09	68	0.39	1.61	31.8	0.625
Legumes	$C = (1.16 + 0.74)I + 32.9/T$	0.81	0.09	104	0.40	1.59	25.0	0.642
Perennial	$C = (7.48 + 0.70)I + 23.7/T$	0.77	0.14	27	0.36	1.91	22.9	0.687
Clover	$C = (-0.99 + 0.71)I + 26.8/T$	0.95	0.06	24	0.28	2.13	23.0	0.595
Wheat	$C = (-0.36 + 0.81)I + 30.2/T$	0.88	0.08	15	0.47	1.25	28.9	0.588
Tobacco	$C = (6.31 + 0.87)I + 15.2/T$	0.78	0.13	59	0.28	1.99	15.7	0.551
Resistant								
All plants	$C = (16.89 + 0.95)I + 27.8/T$	0.51	0.25	291	0.45	1.55	10.6	0.696
Legumes	$C = (8.98 + 1.08)I + 30.4/T$	0.82	0.18	36	0.38	1.89	12.2	0.722
Grasses	$C = (19.06 + 1.17)I + 26.3/T$	0.55	0.28	13	0.45	1.47	6.5	0.655
Vegetables	$C = (19.79 + 1.26)I + 18.7/T$	0.70	0.28	16	0.55	1.50	17.8	0.819
Woody plants	$C = (23.12 + 0.61)I + 28.8/T$	0.45	0.30	46	0.39	2.50	7.8	0.985
Cucumber	$C = (15.05 + 1.41)I + 10.6/T$	0.83	0.23	18	0.41	1.41	13.3	0.581
Chrysanthemum	$C = (20.68 + 0.52)I + 25.6/T$	0.48	0.27	45	0.39	2.17	12.6	0.847

[a] Equations were developed from exposures limited in time (0.5–8 h, except for 2–12-h points in the sensitive group) and denote acute responses of the plants. Concentrations range from 0.05 to 0.99 (1.0) ppm and responses from 0 to 99 (100)% of control.

[b] C is ozone concentration in pphm; I is percent response; T is time in hours; and A_0, A_1, and A_2 are constants (partial regression coefficients) that are specific for pollutant, plant species or group of species, and environmental conditions used.

[c] Multiple correlation coefficient squared, which represents the percent variation explained by the model.

[d] For 5% response in an 8-h period.

[e] From the computer analysis.

into plant tissues. However, cuticular sorption may be a small but effective sink, over time. Townsend[496] used similar techniques and reported ozone sorption by nine tree species. He did not attempt to separate stomatal and cuticular sorption. White birch showed a linear uptake from 0.10 to 0.80 ppm (Figure 11-7) and only a 5% decrease in uptake rate over an 8-h exposure at 0.20 ppm. Uptake in red maple was linear to 0.60 ppm and was at only 40% of the maximal rate after an 8-h exposure at 0.20 ppm. Townsend presented values of actual uptake rates that may be useful. Several of these are given in Table 11-26. All these studies determined uptake on the basis of the time needed to deplete the ozone in a closed system. This does not permit a high reliability in the data given, but they are among the best available.

Hill[203] reported pollutant uptake values for a number of gaseous pollutants, including ozone and PAN, with alfalfa as his test organism (Table 11-26). These values were obtained with a dynamic, but closed, exposure facility. Uptake was determined by the amount of pollutant needed to maintain a constant chamber concentration over an alfalfa bed. Uptake values, expressed on the basis of leaf area, reflect the effect of the plant canopy on the exchange of gases within the canopy and do not

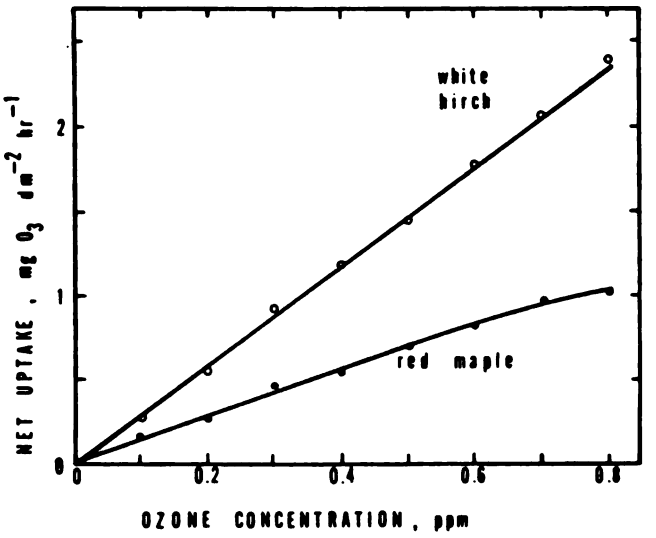


FIGURE 11-7 Net ozone uptake by foliage of red maple and white birch seedlings in relation to ozone concentrations. Reprinted with permission from Townsend.[496]

TABLE 11-26 Rates of Oxidant Uptake by Selected Plant Species

Plant Species	Pollutant	Uptake Rate, $\mu g \cdot dm^{-2} \cdot h^{-1} \cdot pphm^{-1}$	Reference
White oak	O_3	32	496
White birch	O_3	27	496
Sugar maple	O_3	19	496
Sweet gum	O_3	14	496
Red maple	O_3	14	496
Alfalfa	O_3	1.1	203
Alfalfa	PAN	1.2	203

give the maximal capability of plants for pollution sorption. They are, however, representative of the agricultural situation. The values shown are an order of magnitude below those reported by Townsend[496] and reflect the experimental conditions used, as well as the test species. Hill's use of the alfalfa canopy partially explains his lower values, but the technique used by Townsend could have resulted in inflated values.

Bennett *et al.*[18] have presented a model for gaseous pollution sorption by plants. The model includes all the known factors that might have a significant effect on pollution sorption by plant leaves, including gas concentration (ambient air and internal leaf), gas fluxes (external and internal), resistance to flow (leaf boundary layer, stomatal, and internal), nature of leaf surfaces (stomatal presence, cutin, and surface properties), importance of gas solubility and thus solute concentration within the leaf, and ability of the plant to metabolize pollutants (decontaminate itself). They mentioned the reactivity of ozone as another factor to consider. They believe that surface sorption may be important, at least over short periods. They presented a possible mathematical representation of these factors, which they suggested is equivalent to the mathematical statement of Ohm's law. This material is well integrated in the review by Bennett and Hill.[17]

Rogers[418] used a constantly stirred tank reactor that permitted instantaneous mixing of incoming air with air already in the chamber. The system was based on a dynamic exposure design with a single-path airflow system. This system should permit the development of maximal uptake rates under given oxidant loads and a more accurate appraisal of the effects of oxidants on stomatal activity. The chamber design permits a constant turbulence within the chamber and an average airflow over leaf surfaces. Within bounds, one can vary this turbulence, while maintaining a uniform mixture of pollutants in the chamber. Preliminary

work with this chamber has produced uptake rates essentially doubling those reported by Hill[203] for nitrogen dioxide. However, Rogers has not yet used the design to study other oxidants (ozone and PAN).

Oxidant uptake rates are controlled by inherent variations within plant species, the effect of the oxidant on the uptake potential of the plant, the effect of environmental stresses on the uptake potential, and meteorologic factors that affect pollutant distribution. It is important to know whether both resistant and sensitive plants act as oxidant sinks or whether only sensitive plants are major sinks. This knowledge requires some understanding of the mechanism of plant resistance to oxidants. If resistance is associated with stomatal closure due to the oxidants,[126] then resistant plants will not act as effective sinks. However, if resistance is physiologic, these plants could act as effective oxidant sinks, with little adverse effect on the plants. Neither resistant nor sensitive plants would be effective sinks under environmental stress that caused stomatal closure.

Plants probably act as major oxidant sinks over both time and distance. However, it is important to remember that plants do not respond in a predictable way over time and are not active over the greater portion of the year. The total capacity of plants as sinks with or without harm to the receptors is not known.

Plant Protection

Air pollution research in effects on vegetation has focused largely on symptom identification, dose response, and mechanisms. The purpose has been to develop criteria necessary to understand effects and to form a basis for the promulgation of ambient air quality standards. Environmentalists were not concerned with protecting plants *per se*, but in controlling emission. Thus, those interested in protection of plants received little encouragement. In addition, no economical ways were suggested for control, and research leaders were not convinced of the importance of adding air pollution stress to breeding programs. It was the hope of most research workers that air quality standards would protect vegetation from ozone and other oxidants.

Most research workers are now convinced that pollution abatement will have little impact on overall pollution concentrations until clean energy forms are developed and in widespread use. Because phytotoxic concentrations of ozone and other oxidants are inevitable for the foreseeable future, researchers are seriously considering other means of protecting plants from injurious effects of oxidants.

For managed agricultural crops, including forest species and other

woody perennials, the addition of air pollution stress into standard breeding programs has begun or is being discussed. Some workers are trying to develop protective sprays for the more sensitive species and cultivars. Some consideration is also being given to the management of cultural practices and to general land management. Ormrod and Adedipe[372] have presented an excellent review of the concepts and work to date regarding the protection of horticultural plants from atmospheric pollutants.

BREEDING FOR RESISTANCE

Varietal studies define the genetic variability in resistance to oxidants within different species. These types of studies are necessary to select genetic lines or accessions for breeding programs. Although plant-breeders have not selected for oxidant resistance, selection probably has occurred, for two reasons: breeders normally select plants with the highest yield and least injury, regardless of cause; and many breeding programs are carried out in areas of relatively high oxidant content. In this regard, natural selection pressures should increase the tolerance of populations of native species near urban industrial areas, although this would tend to reduce the genetic plasticity within sensitive species.

Oxidant problems are starting to be of concern to plant-breeders. Extensive varietal screening of tomato, petunia, and other plants has permitted some resistant cultivars to be recommended for use in high-oxidant areas. None of the varietal screens have involved breeding experiments in which resistant lines are developed and used in the development of resistant varieties for new introductions.

Growers in the Connecticut valley have probably selected wrapper tobacco for oxidant resistance since the 1950's. Similar selection is probably taking place in other tobacco-growing areas. Sand[422] worked with a number of wrapper selections and suggested a partial dominance of genes for fleck resistance. He found that the F_1 hybrid between a sensitive and a resistant selection was more resistant than the parental average. Povilaitis[394] used five tobacco varieties and made six crosses with six genetic populations from each cross, for 36 different populations. He used injury ratings and, unlike Sand, suggested that susceptibility may be dominant over tolerance (dominance was important in five of six crosses).

Johnson *et al.*[235] determined the variation in response of 16 sweet corn hybrids to ambient oxidants in California. From these observations, Cameron and Taylor[54] selected five inbreds and three of their F_1 hybrids for further field studies and four inbreds and two hybrids

for use in ozone exposures. The relative effects of the polluted environments were the same. The final data suggested an inheritance for partial dominance to ozone susceptibility.

Townsend and Dochinger[497] worked with four red maple selections and found sensitive and tolerant selections that showed the same relative susceptibility over four growth stages and four leaf developmental phases. Symptoms were similar in the four selections, but the design should have used lower ozone dosages. These results suggest a strong genetic control that will facilitate selection of ozone-tolerant seedlings for urban use.

Houston[219] and Houston and Stairs[221] did clonal repeatability analyses to determine genetic control of tolerance in white pine with an ozone-sulfur dioxide mixture and a 6-h exposure. They used needle elongation and two injury estimates in assessing effects. The repeatability estimates indicated that tolerance to the pollutant mixture is under genetic control. The nature of the inheritance of tolerance is still not understood, but field selection of tolerant or susceptible individuals is possible. Demeritt *et al.*[107] reported an evaluation system that used visible needle injury for determining resistance of Scotch pine to ozone. This permitted the discrimination of phenotypic differences in a quantitative way. Results suggested that a few genes were responsible for resistance in Scotch pine. A program was initiated to select for ozone resistance in loblolly pine, but no definitive results are available (R. Weir, personal communication).

As genetic understanding develops, it will be incorporated into basic breeding programs that are concerned with such characteristics as yield, growth habit, insect and disease resistance, flavor, and texture. These programs will be most effective if present ambient ozone and other oxidant concentrations do not increase.

PROTECTANT SPRAYS

Kendrick *et al.*[245] first reported protection of pinto bean foliage from sprays or dusts of four fungicides. They later reported rather extensive studies that used a number of fungicides and antioxidants on selected test plants.[244] These tests were in general agreement with the earlier studies and suggested that the response was due to a surface deactivation of ozone and other oxidants. Jones[236] found protection of tobacco with various particulate substances, including charcoal. Freebairn and Taylor[147] found protection from use of ascorbate sprays as antioxidants. They reported partial protection in pinto bean, petunia, celery, lettuce, and grapefruit from oxidant injury in the Los Angeles basin. Several other

early studies reported various degrees of chemical protection from fleck injury to tobacco.[27,510] Taylor and Rich[449] found that an antiozonant-treated cloth protected wrapper tobacco from oxidant injury.

These early studies, along with the recognition that oxidant pollution will continue as a significant problem, encouraged a number of studies directed at finding chemical treatments that might serve a dual function with a minimum of applications. Some selected results of these studies are summarized in Table 11-27.

The fungicides, as a group, have received the most attention, and Benomyl (methyl-1-butylcarbamoyl-2-benzimidazolecarbamate) has been most widely studied, owing to its protective properties. Benomyl has been used as a foliar spray, a soil drench, and a soil amendment. Mixed results have been reported when Benomyl was used as a spray. Protection was reported on tobacco,[401] pinto bean,[303,305,427] white bean,[84] and grape;[243] no protection was found for pinto bean,[389,390] and cucumber.[389] It was not possible to equate the Benomyl concentrations used, but most were 20–4,000 ppm. Most reports on soil applications showed protection against oxidants. Soil drenches at 10–1,000 ppm gave complete to partial protection for poinsettia,[302] pinto bean,[390,490] chrysanthemum,[249] and annual bluegrass.[342] Soil amendments at 2–160 ppm were effective for pinto bean,[301,389,390,420,427] annual bluegrass,[342] and cucumber.[389] Taylor and Rich[450] reported that a single soil amendment at about 25 ppm protected the first eight leaves of tobacco, but not the younger leaves. They also found fewer tobacco root cyst nematodes from Benomyl applications, and the fungicide was not toxic to the plants at the rates used. Benomyl does not induce stomatal closure,[389,450] so an internal physiologic mechanism must be involved. The active ingredient is the benzimidazole moiety,[390,490] which inhibits senescence in bean and also inhibits the loss of free sterols in plant membranes.[490] Sterols themselves are effective inhibitors,[439] and the mechanism of action may involve effects on membrane sterols and thus membrane permeability. Spotts *et al.*[438] studied the effects of benzimidazole and ozone on several water relation effects that suggest that the protection is due to the effect of benzimidazole on the cell membrane.

The fungicide Carboxin has received some study, but with varied results. A 0.43 mM solution gave protection at 5–10 days in white bean,[83] a 0.36% spray gave no protection to pinto or tempo bean,[305] and a 0.01% spray increased injury to azalea.[343] Soil applications of Carboxin were generally more beneficial, but results were still varied. A Carboxin soil amendment was phytotoxic to tobacco[450] and pinto bean,[407] although protection was noted in pinto bean. Pinto and tempo bean were protected from oxidant for 36–40 days after a soil amendment with Carboxin

TABLE 11-27 Protection of Plants from Oxidant Injury by Application of Protective Chemicals[a]

Plant Species	Pollutant Protected from	Chemical (Concentration)[b]	Type of Protectant	Degree of Protection, %[c]	Reference
Bean, cultivar Pinto	Oxidant	K-Ascorbate (0.01 M)	Antioxidant	52	147
Petunia	Oxidant	K-Ascorbate (0.01 M)	Antioxidant	39	147
Tobacco	Oxidant	Zn-ethylenebisdithiocarbamate dust (variable)	Fungicide	44	27
Tobacco, cultivar White Gold	Oxidant	Phygon XL (variable)	Antioxidant	89	510
Tobacco, cultivar White Gold	Oxidant	Phygon XL (variable)	Antioxidant	78	510
Tobacco, cultivar White Gold	Oxidant	4,4-Dioctyldiphenylamine in butyl latex	Antioxidant	100	449
Bean, cultivar Pinto	Oxidant	Zineb (normal use)	Fungicide	91	244
Bean, cultivar Pinto	Ozone	Zineb (normal use)	Fungicide	97	244
Azalea	Oxidant	Benomyl (60-ppm drench)	Fungicide	96	343
Bean, cultivar Pinto	Ozone	Carboxin (2.3 ppm in soil)	Fungicide	95	407
Radish	Ozone	*N*-6-Benzyladenine (30-ppm spray)	Growth substance	100	7
Poinsettia	Ozone	Ancymidol (100-ppm spray)	Growth retardant	100	58
Poinsettia	Ozone (chronic)	Benomyl (500-ppm drench)	Fungicide	57	302
Bean, cultivar Pinto	Ozone	Folicote (0.5% spray)	Wax emulsion	92	391
Bean, cultivar Pinto	Ozone	Benomyl (5 ppm in nutrient solution)	Fungicide	97	389
Bean and Cucumber	Ozone	Benomyl (80 ppm in soil)	Fungicide	94	389

Grape	Ozone	Benomyl (6.7 kg/ha 6 times)	Fungicide	53	243
Bean, cultivars Tempo and Pinto	Ozone	Benomyl (0.25–0.36%, 4 weekly sprays)	Fungicide	75	305
Bean, cultivars Tempo and Pinto	Oxidant	Carboxin (10% granular as soil amendment, 8 g/5-m row)	Fungicide	100	305
Tobacco	Ozone (0.50 ppm, 2 h)	Piperonylbutoxide (2 mM solution)	Insecticide	99	254
Tobacco	Ozone (0.35 ppm, 2 h)	Safroxane	Insecticide	76	254
Bean, cultivar Tempo	Oxidant	Benomyl (0.24% spray)	Fungicide	32–41[d]	303
Grass, annual blue	Ozone (0.25 ppm, 2 h)	Benomyl (60-ppm amendment)	Fungicide	85	342
Bean, cultivar Pinto	Ozone (0.30 ppm 4 h)	Triarimol	Fungicide	81	427
Bean, cultivar Pinto	Ozone (0.25 ppm, 4 h)	Benomyl (1.60-μg/g soil amendment)	Fungicide	98	390
Bean, cultivar White	Ozone (0.13–0.50 ppm, 0.5 h)	Ascorbic acid	Antioxidant	75	91
Petunia	Oxidant	SADH (0.5% spray)	Growth retardant	82	59
Tobacco	Oxidant	Benomyl (25-ppm drench)	Fungicide	68	450
Tobacco	Oxidant	Benomyl (0.18% spray)	Fungicide	59	401
Tobacco	Ozone	Peroxidase (0.10 ppm injected)	Enzyme	89	33

[a] Selected data from the references cited.
[b] These are applied as sprays unless otherwise noted.
[c] Percent reduction in plant injury from ozone as a result of the protectant treatment.
[d] Increase in yield by protectant application.

at 8 g per 4.6-m row,[305] and the sulfoxide analogue gave protection to white bean, with a 13% yield increase.[84] A soil drench gave protection to soybean, cotton, tomato, and tobacco at 9.5 ppm,[407] but a soil drench at 1-100 ppm increased azalea senescence that was due to oxidants.[343] These results could reflect differences in species or conditions of application, but they do call for clarification of the possible role of Carboxin in protection against oxidants. Carboxin had no effect on stomatal opening,[407] and its protective action appears due to the sulfoxide breakdown product,[84,407] which is not fungistatic. Thus, although Carboxin can serve a dual role, the modes of action are dissimilar.

In addition to the compounds named, several others have been studied briefly. Cathey and Heggestad[58] found protection to eight cultivars of poinsettia after treatment with the growth retardants Ancymidol and Chlormequate. They also reported a 75% protection in petunia treated with the growth retardant succinic acid 2,2-dimethylhydrazide (SADH) as a 0.5% spray.[59] This was part of a screen to ozone of 65 petunia cultivars. These workers found that the resistant cultivars did not respond to SADH; this suggested that protection may be related to modified leaf structure (reduced cell size, intercellular space and stomata, and thicker cell walls). The SADH treatment was not effective above an ozone concentration of 0.45 ppm for 3 h. Adedipe and Ormrod[7] found that a 30-ppm spray of the growth hormone *N*-6-benzyladenine protected radish from ozone at 0.25 ppm (4 h), and Fletcher *etal.*[144] protected white bean by use of a 1-ppm abscisic acid (ABA) solution. The ABA caused partial stomatal closure, which the authors suggested is the mechanism of protection. Studies with ascorbic acid on white bean,[91] the insecticide piperonylbutoxide on tobacco,[254] road dust on pinto bean,[508] and a wax emulsion (Folicote) on pinto bean[391] all stressed that many compounds can act as protectants to vegetation against oxidant (ozone) effects.

Larkin[261] injected several peroxidases at 0.1-10 ppm into one-half of a tobacco leaf and found some protection. He suggested that peroxidase, which is often associated with plant stress conditions, may be important in physiologic resistance. It is doubtful that any one mechanism of action exists. It is important that we understand the mechanism of ozone injury and resistance in plants, so that we can determine better what chemicals may play a role in protecting plants against oxidants.

We do not yet have good answers to four basic questions that have kept protective chemicals from practical use: we do not know the frequency or rate of application needed for continued resistance, or, therefore, the total cost of application. We have little idea of the specificity of selected chemicals on different plants. Little is known of possible

undesirable residues or side effects. We do not have sufficient prediction accuracy for high-oxidant days for chemicals that are not long-lived. These basic questions need to be answered as chemicals are tested and promoted for use. The ideal chemical would be effective on several species (cultivars), have multiple uses (e.g., fungicides), have a long life (a single pre-emergence soil application would be best), and leave no toxic residues. Such chemicals would have value, at least until breeding programs can incorporate resistance to pollution stress into new cultivar introductions.

CULTURAL PRACTICES AND LAND USE

Ormrod and Adedipe[372] have developed an excellent presentation on edaphic and climatic factors that play a role in making plants more resistant to ozone and other oxidant stress. They suggest how these environmental factors may be used in cultural and management practices to help to alleviate the effects of oxidant pollutants. These practices may be of help in greenhouse management and to some extent in field irrigation systems. The association of water stress with resistance to oxidant has long been recognized, and growers have been urged to keep this in mind during periods of high air pollution potential. The increased use of carbon dioxide in greenhouse management would have a positive effect, but the additions are made during cooler months, when oxidant concentrations are not high. These practices are worth considering, but are as yet of no more than supplemental help.

L. S. Dochinger (personal communication) was able to predict chlorotic dwarf on seedbed white pine with greater than 90% accuracy. Taylor[447] found that susceptible and resistant tobacco could be identified in seedbeds. Both suggested that, for some sensitive plants, time could be saved by visual screening of transplant beds and the use of only the more resistant members of the population as transplants.

Interest in land use planning—with respect to areas to be set aside for agricultural use, as opposed to industrial use—needs to be considered as a potential ameliorating factor in the control of air pollution effects on vegetation. Land use might not be as effective for the oxidant pollutants, because of their ubiquitous nature. Heck and Brandt[183] have developed a brief but acceptable point of view in terms of the need for land use planning and protection of agricultural commodities from air pollution.

A rational approach to land use planning for air quality maintenance may be built on the basis of diffusion modeling. Although the photochemical versions of the models that relate air quality to emission are

only beginning to be applied, there has been sufficient validation to permit their use at least to obtain relative assessments (in contrast with prediction of absolute pollutant concentrations). Chapter 5 discusses the principles and performance of models that can be used for studying the distribution of photochemical ozone and other oxidant concentrations. The application of these techniques requires a succession of forecasts, beginning with land use patterns and continuing with transportation network use, vehicle emission intensities, stationary-source contributions, and, finally, patterns (in space and time) of pollutant input to the atmosphere. Meteorologic characterizations based on detailed weather data provide the transport measures needed. For various scenarios, the model generates statistics on the air quality that should be expected. The loop is closed by adjusting the land use to achieve the desired atmospheric pollutant concentrations.

RESPONSES OF NONVASCULAR GREEN PLANTS

Lichenologists have long used the presence and abundance of lichen and moss species to map the biologic impact of large urban and industrial areas.[143] Early workers considered changes in the presence and abundance of these organisms to be related to such factors as temperature and humidity. More recently, most researchers have tended to relate changes in lichen population more to industrial air pollution than to other environmental changes. Although most of the air pollution work lacks ambient air quality measurements, there is strong indication that both the presence and the abundance of lichen and moss species are correlated with sulfur dioxide concentrations in large urban and industrial areas. Such studies have not been done in areas where oxidants are the primary air pollutants, nor in rural areas with relatively high oxidant and mixtures of sulfur dioxide and nitrogen dioxide. Thus, little is known about the direct effects of ozone or other oxidants on lichen and moss morphology or physiology.

Comeau and Le Blanc[68] found that a 4-h exposure of *Funaria hygrometrica* to ozone at 0.25–1.00 ppm stimulated the regenerative capacity of the moss leaves. This was not true for 6- and 8-h exposures.

Glater[152] reported oxidant injury to several species of fern in the Los Angeles area. Initially, tan lesions appeared near the smaller veins, but in no special pattern. Later, the entire leaf became necrotic. Symptom development and sensitivity of leaves were different from those noted in vascular plants. All leaves appeared to be equally sensitive, except for

the growing tip and the youngest uncoiling leaves. Occasionally, a young plant was killed.

We know little about the overall effects of ozone or other oxidants in the nonvascular green plants. Although they may not be economically important, such effects may have an adverse ecologic impact.

RESPONSES OF MICROORGANISMS

Heagle[170] reviewed the effects of ozone on fungus growth, sporulation, and germination. Ozone may inhibit colony growth on artificial media, but rarely causes death, even at high concentrations. Differences in species susceptibility are known. Exposure to ozone at 0.10 ppm for 4 h stopped conidiophore elongation and spore production in *Alternaria solani*.[410] In several fungi, exposure to ozone at 0.10 or 0.40 ppm for 4 h caused a 10-fold to 25-fold increase in sporulation.[170] Heagle[171] reported effects on three obligate fungi from low ozone exposures. Spore germination was not affected in any of these studies. Kuss[257] grew 30 representative fungi on agar and often found increased spore production after exposure to ozone. Spore germination was decreased in most species, but increased in others.

Rabotonova[396] exposed two species of yeast to ozone: *Candida lipolytica* was sensitive, and *C. auilliermondii* was resistant. The biocidal activity of ozone was determined under various cultural conditions with airstreams of about 150 or 5,500 ppm (v/v for 10–30 min). Ozone was an effective biocide under most conditions, and effectiveness increased with decreasing pH. Kanoh[239] found that exposure to ozone at 30 ppm for 30 min increased oxygen uptake in slime mold (*Physarum polycephalum*) homogenate. The ozone also increased succinoxidase activity and inhibited part of glycolysis. deKoning and Jegier[103–106] reported effects of ozone on *Euglena gracilis* that included reduction of net photosynthesis, increase in respiration, and effects on pyridine nucleotide reduction and phosphorylation. They reported that reduction of net photosynthesis was a logarithmic function of ozone concentrations in 1-h exposures.[106] They also found a 5% reduction in oxygen evolution after a 1-h exposure to 0.5-ppm ozone bubbled into 5 ml of solution and an additive effect with a mixture of sulfur dioxide and ozone.[105] Verkroost[509] carried out a detailed study of the effects of ozone on *Scenedesmus obtusiusculus* Chod., with special concern over the effects on photosynthesis and respiration. A major weakness in this study was the use of an airstream containing ozone at 150 ppm. The report sug-

gested that the primary site of ozone action is the membrane structure, which produces changes in photosynthesis and respiration.

Ozone at high concentrations has been used in a variety of applications for the control and suppression of fungi and bacteria. These applications have included food protection, drinking-water purification,[157] and treatment of sewage.[248,309] It is generally accepted that ozone is not an effective germicide at concentrations below the point of human sensitivity—0.04 ppm. The germicidal effectiveness depends on concentration, relative humidity, and the specific organism. In many cases, even a concentration of 3-5 ppm was not sufficient to kill some bacteria. Burleson *et al.*[50] showed inactivation of several viruses and bacteria after ozone exposure and a greater inactivation with simultaneous sonication. Zobnina and Morkovina[531] related the tolerance of a carotenoid strain of *Mycobacterium carothenum* with the presence of the pigment. The dwarf mutant was much more sensitive.

Large doses of ozone may inhibit growth and sporulation of fungi on fruit, although most fungi tested were resistant to ozone. Spaulding[436] reported that ozone acted as a surface biocide. Above 0.5 ppm, ozone inhibited surface growth of fungi on strawberry and peach. At 0.06 ppm, it severely injured leaves on head lettuce after 8 days. He also reported some direct injury on peach fruits above 0.5 ppm. Ridley and Sims[415] extended the shelf-life of strawberry and peach by exposing them to ozone, but stated no concentrations. Ozone at 1-2 ppm for 1-2 h/day controlled the surface growth of fungi and sporulation on apple, reduced offensive odors, and decreased the ripening rate.[435] Watson[513] found that ozone at 0.4-2.0 ppm acted as a surface fungicide, but did not penetrate. Thus, there was no effect on fungal growth within the fruit. Sporulation and some decay control of *Penicillium digitatum* and *P. italicum* were noted in open storage boxes of lemon and orange exposed to ozone at 1 ppm.[78] Ozone was more effective than a fungicide dip in controlling *Botrytis* bud rot of gladiolus, but no concentrations were determined.[290] In general, researchers have suggested that rather large dosages of ozone are required to protect storage fruits from fungal infection. These concentrations may be so high as to preclude the use of ozone in storage facilities. The ability of ozone to reduce spore germination in fungi apparently depends on species, spore morphology, moisture, and substrate.[170] Single-celled spores and those with thin cell walls are most sensitive. Wet spores are more sensitive than dry spores.

Haines[160] reported that ozone at 4 ppm retarded growth of *Escherichia coli*, whereas 10 ppm prevented growth. Scott and Lesher[424] found that approximately 2×10^7 molecules of ozone per bacterium killed 50% of the cells of *E. coli* and that the primary effect was on the cell membrane.

Only metabolites leached from the cell were affected. Elford and van den Ende[124] reported that ozone at 20 ppm had a lethal effect on some bacteria deposited from aerosol mists on various surfaces. Relative humidity is an important factor, particularly when ozone concentration is low. They found little death at a humidity below 45%, at concentrations of 1 ppm, as opposed to a 90% kill in 30 min at 0.025 ppm with a humidity of around 70%. A 5-min exposure of *Bacillus cereus* to ozone at 0.12 mg/liter was the minimal lethal dose, whereas 0.10 mg/liter was effective for *B. megaterium* and *E. coli.*[48] Spores of the *Bacillus* sp. were killed by ozone at 2.29 mg/liter. These responses were of the all-or-none type with ozone between 0.4 and 0.5 mg/liter of water. Time of exposure, from 1 to 32 min, was not important. Chlorine was effective at 0.27–0.30 mg/liter, with time an important consideration. These two gases did not affect *E. coli* in the same way.

In most research on lower organisms, there has been an attempt to use ozone as a germicide or to understand the interactions of pollutants and pathogens on the responses of higher plants. In few studies has the interest been on the effects of ozone on the organisms themselves, except in the studies of effects on algae.

BIOLOGIC MONITORS

The use of biologic indicators as an early detection system for severe air pollution episodes or for chronic air pollution problems is of interest to many urban and industrial control officials. Plants served as air pollution detection systems long before pollution was acknowledged a problem by industry or government. Higher plants serve as useful detection tools, because they develop characteristic symptoms from acute exposure, even though the symptoms are not necessarily specific to cause. Cause and effect can be reliably ascertained, when an air pollution source is identified and symptom patterns are identified within given sensitive species.

Plants have been used effectively in field surveys to determine the extent and magnitude of pollution problems and in bioassay techniques in conjunction with field surveys. Most general review articles (Table 11-1) treat, to some extent, the use of plants as indicators of air pollution. Went[517] covered plant sensitivities to pollutants and the use of plants as indicators. He stressed the photochemical oxidants and recommended charcoal filtration for greenhouse use. Heck[182] presented a detailed discussion and review of plants as indicators in field surveys and in the bioassay of photochemical problems. Heggestad and

Darley[195] reviewed plants as indicators of ozone and PAN and recommended pinto bean as the best indicator of both oxidants. They also suggested three variably resistant tobacco cultivars for use in monitoring the severity of ozone episodes.

It may be of value to differentiate the indicating and monitoring uses of plants. Most reported work has used the indicator concept, with plant injury (symptoms) as indicative of a problem. Monitoring implies some degree of reliability. Several studies have attempted to use plant response as a monitor of pollution concentrations or doses, with variable but uncertain success. Plants could also be considered monitors if they gave a reliable index of the biologic effects on biologic systems of concern to man (crops, forests, animals, and man himself). The latter has not been seriously discussed.

This section is divided into reviews of plants in field surveys and plants as a bioassay technique and a brief discussion of the possible value of biologic indicators.

Field Surveys

Field surveys have played a significant role in the assessment of air pollution problems. Basic techniques were developed in surveys around point sources of sulfur dioxide and fluoride. Similar techniques have been developed regarding photochemical pollutants, starting with the early report by Middleton *et al.*[329]

Treshow[499] has developed the thesis that foliar injury is a useful criterion in the identification and analysis of air pollution effects on vegetation and presented some basic concepts for use in field evaluation.

Sharma and Butler[429] found that white clover in a highly polluted area (Nashville, Tenn.) had a lower stomatal frequency, a higher trichome frequency, and a greater trichome length than the same species growing in an area of lower pollution. They suggested oxidants as one of the pollutant stressors. It is possible that pollutant stress (among other things) is causing selective pressures that favor these changes; thus, a separate race may be evolving. This type of work needs further evaluation and exploration.

Most researchers have felt that biochemical changes in plant tissues are associated with so many normal growth and stress phenomena that they would have no relevance in field evaluations of air pollution effects. Keller[241] has associated changes in peroxidase activity with different pollution stresses (fluoride and oxidants) in apricot and white ash growing in areas of high pollution, as opposed to those of low pollution. This study suffers from lack of pollution monitoring (although leaves were

analyzed for fluoride) and consideration of other stress factors. This is the same type of criticism that has long been made of lichen studies in industrialized areas.[143] Lichens may respond to the photochemical complex, although such work has not been reported. The larger question of the value of these studies persists. If, in fact, changes in some biochemical entity could be clearly associated with one or more pollutant and if this could be separated from other stresses, what would it tell an interested party? Can it be related to adverse yield, quality, or genetic changes? These approaches to understanding air pollution effects through field surveys appear to be of doubtful value and must rely on statistical methodology involving many variables.

A technique that offers greater potential is the use of remote sensing of injury to vegetation. This technique was first tried with ponderosa pine as the species of concern.[518] Wert *et al.*[519] and Larsh *et al.*[263] found that injury to ponderosa pine in the San Bernardino Mountains was severe enough for the technique to have real potential. They were able to identify the severity of diseased areas through color photography with a high degree of correlation with ground plots. In severely affected areas where air pollution is the principal stressor, this approach offers a rapid and inexpensive technique for surveying large tracts. It is not yet possible to use remote sensing to identify pollutant stress amid a variety of other stressors. However, as these techniques are improved, they should be applied to air pollution problems.

Early field surveys depended on identification of a syndrome of responses that included symptoms on both native and cultivated plant species. Middleton and Paulus[330] directed the first large-scale survey to determine the extent and severity of photochemical-oxidant effects in California on crops of agronomic importance. They delineated four categories of crops (field, flower, fruit, and vegetable) and one of weeds. This was the most extensive survey of oxidant effects until the late 1960's. The information was later used as a basis for subjective estimates of economic losses. This type of visual assessment of foliar injury has been attempted in many states and has been purposefully developed in some for use in economic estimates of damage to vegetation.

Plant Bioassay of Oxidant Effects

During the 1950's, when the phytotoxic components of the photochemical-oxidant complex were not known, plants were used extensively as a bioassay of many simulated reaction mixtures to indicate toxicity.[158,182,209,364,365,442] Plants were also used to help in identifying specific components of photochemical reaction products, such as PAN and its analogues.[442]

This bioassay technique was also used to determine the phytotoxicity of the ambient photochemical complex and to attempt to standardize plants, so that oxidant concentrations could be determined from severity of injury. Two such studies were initiated at about the same time in the Los Angeles basin. Middleton *et al.*[328] established five stations where young pinto bean plants, grown under uniform conditions, were set out daily and foliar injury intensity was compared with results from oxidant instruments. Results were recorded as the percentage of days showing plant injury and the average injury index per day per station. There was a relationship between injury and oxidant values, but the correlations were not significant and the association was obscure. The most detailed study was carried out over a period of about 4 yr with annual bluegrass.[366] The plants were grown under standardized conditions,[237] and injury development was concisely defined.[33] The plants were transported daily to locations around Los Angeles County, California. They were exposed for 24 h and returned for a control posttreatment period before injury assessment. The injured leaf areas were measured and correlated with the severity of oxidant episodes. The procedures were time-consuming, and the results were poorly correlated with oxidant concentrations. Thus, this procedure was discontinued as automatic instrumentation became available.

The bioassay technique was developed to reduce the uncertainties associated with the use of native vegetation or cultivated crops. Plants can be started under controlled conditions and exposed under standardized conditions. Species and cultivars can be selected for oxidant sensitivity and symptom characteristics. The two studies just noted were the most closely controlled. Similar work has not been repeated. However, many investigators have grown plants under known cultural conditions and then transplanted them to field sites where they received special care. These plants can then be read for foliar symptoms throughout a given period, and the symptoms related to oxidant concentrations. The lack of apparent correlation in the two early studies could be due to the lack of specificity for the monitored oxidants, the presence of different concentrations of interacting oxidants at different times, or variations in cultural conditions between exposure times.

Brennan *et al.*,[42] using petunia, found a better correlation with atmospheric aldehydes than with total oxidants. This appears spurious, because researchers do not regard aldehydes themselves as important atmospheric phytotoxicants. Macdowall *et al.*[285] were able to correlate tobacco injury with oxidant concentrations by considering ozone flux into plant leaves. They then predicted fleck attacks with fair consistency on the basis of meteorologic considerations. Several investigators have used acknowledged plant sensitivity and emphasized the importance of

accurate monitoring for pollutants[71] and meteorologic factors.[108] These aspects must concern any investigator who is seriously considering a plant monitoring system. Berry[23] developed clonal white pine material for use with specific atmospheric pollutants. Oshima[375] has recommended pinto bean as a reliable monitor of oxidant intensity with a subjective injury evaluation (0-5) that has been standardized against colored photographs. This is not as sensitive as a percentage separation, but is no doubt easier for an untrained observer to use. He did show good correlation between weekly oxidant concentrations and plant response. This is a relatively simple system, but the monitoring plant is not as sensitive as Bel W_3 tobacco to low concentrations of oxidant.

The most extensively used monitoring system for photochemical oxidants has used the ultrasensitive Bel W_3 tobacco.[197] The technique was first developed by Heck and associates.[189,190] The response of this tobacco variety was used to determine the distribution, frequency of occurrence, and approximate average concentrations of photochemical oxidants in and around Cincinnati, Ohio, over four growing seasons. Monitoring sites were set up at various distances from Cincinnati, up to 75 miles (120 km) due east. Injury responses from this monitoring network showed that the average effective concentration of photochemical oxidants was as high in rural Ohio as in Cincinnati. Although oxidant concentrations did not correlate well with plant injury on a weekly basis, the seasonal ratio of injury to oxidants was similar over a 3-yr period. It was suggested that all areas east of the Mississippi have sufficient photochemical oxidants to cause injury to sensitive plants at some stage of their development. This monitoring system has since been used as part of a regional study to determine the importance of oxidant pollutants with respect to sensitive crops in the northeastern United States,[232] and similar systems have been used as oxidant indicators in South Dakota,[150] Germany,[250] the United Kingdom,[15] and other locations—including North Carolina, Washington, and Arizona. This type of monitoring system was designed to provide communities with estimates of the frequency of occurrence of phytotoxic concentrations of oxidants, of the relative severity of each episode, and of the regional distribution of oxidant pollutants. The system will not give a reliable estimate of oxidant concentrations. Thus, it is an indicator of oxidant phytotoxicity, instead of a monitor for oxidant concentrations.

Craker *et al.*[79] used Bel W_3 tobacco as a relatively simple monitoring device whereby interested citizens could develop some idea of the biologic severity of oxidant pollution in their community. There are problems with their study, but the concept is useful and might alert a community to potential pollution problems.

Several investigators have used systems ranging in complexity from

simple antioxidant-impregnated cloth covers[512] to fairly elaborate filtered and unfiltered chambers[172,200,207,506] to determine the effects of ambient oxidants on sensitive indicator (monitoring) plants. Bel W_3 tobacco was used in all these tests. Such tests can be used to determine growth and yield reductions, as well as to identify possible problems.

Discussion

Many investigators have discussed the use of biologic indicators as early detection systems for air pollution problems. Some have viewed the possibility of a nationwide monitoring system for air pollutants and other environmental contaminants. Other have felt that the increased collection of chemical data at many sites obviates biologic indicators or monitors. However, the chemical and meteorologic data do not yet give a clear picture of the biotoxicity of air pollution episodes. Thus, agencies should give serious consideration to the development of national networks. If such interest becomes widespread, it should be possible to correlate the biologic response of the monitors with the response of important economic species, ornamental species, or some human health measures. Without the ability to correlate these factors, bioindicators are merely another index of pollution that carries no rational value. Without the use of the bioindicator, we are back to a direct assessment of the effects of a given pollution episode on the biologic organisms of interest to man.

ECONOMIC ASSESSMENT

This section will bring into focus our present understanding of the economic effects of oxidant air pollution on vegetation. Heck and Brandt[183] presented in depth the problems inherent in making an economic evaluation of the response of plants to air pollutants, including ozone and other oxidants. They used the distinction between injury and damage that was proposed by German workers.[35] *Injury* is defined as any identifiable and measurable response of a plant to air pollution; *damage* is any measurable adverse effect on the desired or intended use of the plant. Thus, before an effect on a plant can be evaluated in terms of economics, the plant must have been altered either quantitatively or qualitatively in such a way as to reduce its use value. In this context, visible symptoms or transient changes in physiologic responses may not result in an economic loss. Thus, leaf necrosis in soybean is injury; to be classified as damage, the injury must affect yield. In the same way, a severe oxidant episode that bronzes the leaves of romaine lettuce may

not affect biomass, but will affect use and may result in complete economic loss. Ornamental plants, whose major use value depends on appearance, may be both injured and damaged.

Emergence tipburn of white pine[24] and other physiogenic diseases of white pine associated with air pollution occur throughout the natural range of the species. Insular stands containing ponderosa pine are frequently injured in the Southwest. The significance of these physiogenic diseases is not understood because they affect only sensitive genotypes. The disease occurs randomly in the forest, and its most obvious effect is the slow selection and gradual elimination of genotypes that may have otherwise superior silvicultural characteristics. This could be a serious loss to future tree improvement efforts and may be occurring in other forest species. Economic considerations have not been addressed in any reasonable way.

Early work with sulfur dioxide showed a linear relationship between visible injury and reduction in yield for many crop species. The assessment was made that no reduction in yield would be found unless visible injury were noted. Definitive research with ozone, other oxidants, or mixtures of these pollutants with other gases has not been done. Thus, we do not know whether such relationships between visible injury and yield hold for the oxidants, but data in Table 11-3 suggest that for acute exposures there may be good correlations between injury and yield reductions. Many researchers have hypothesized that the oxidants may have an effect on plants that will produce a yield reduction with little or no visible injury. Such studies need to be designed in a more definitive manner before it is concluded that yield reductions without visible symptoms are clearly acceptable. Projections of yield losses have made use of some of the data reported earlier.[16,331]

Oshima[376,377] has developed a methodology for evaluating and reporting economic crop losses that involves continuous air monitoring, chamber exposures, and monitoring plant species. He has attempted to weld these into a comprehensive method of determining yield reductions. He has reported yield reductions for some species, but has not fully clarified the procedure. He presented no economic values in either report. This type of approach needs to be considered further.

The reports of specific effects related to sensitive growth stages,[475,477-479] especially to reproductive growth stages in which the pollutant may be affecting reproductive structures and not leaf tissues,[53,54,137] are probably of greater importance to the understanding of reduced yield without visible symptoms than to the potential of a debilitating effect caused by chronic continuous exposure to ozone or other oxidants. These economic considerations have not been addressed.

Any attempt to assess oxidant damage to agricultural crops requires

TABLE 11-28 Economic Estimates of Crop and Vegetation Losses Owing to Oxidant Air Pollutants, United States[a]

Area	Year	Cost, × $1,000	Comments	Reference
United States	1963	65,000	First approximation for commercial crops (SRI)	16
		121,400	Revised SRI report to include ornamentals	16
California	1963	33,700	Revised SRI report	16
	1970	17,500	Does not include ornamentals or indirect costs	331
Pennsylvania	1963	6,300	Revised SRI report	16
	1969	9,600	Pa. survey; includes indirect costs	259
	1970	60	Pa. survey, as above	258
New Jersey	1971	960	N.J. survey of a limited number of crops, based on visible injury	142
	1972	60	N.J. survey, as above	386
New England	1971	1,100	Mass. survey; primarily crops and ornamentals	357

[a] Some of variation reflects methodology; other variation reflects differences in plant susceptibility and pollution over years.

judgment by a competent investigator. Landau and Brandt[260] believe that the success of crop surveys is directly related to the number of subjective decisions required in data collection. The first surveys were conducted in California[329,330] and were used, with a general survey of conditions on the East Coast, to develop some of the early economic estimates of $8–10 million in California and $18 million on the East Coast. These estimates were raised to $500 million on the basis of increased awareness of pollution effects and increased recognition of additional sensitive species. However, all early estimates were subjective, with no substantial backup data.

Since 1969, a number of states have instituted an intensive training program for county agricultural agents and made detailed reports of crop injury and damage[142,257,259,331,357,386] (Table 11-28). The first such report came from Pennsylvania[259] in 1969 and gave an estimate of a $9.6-million loss to agronomic commodities from oxidant pollutants. This survey included direct and some indirect cost. Similar surveys have been conducted in New England,[357] New Jersey,[142,386] California,[331] and Michigan (no report is currently available). These surveys considered yield reductions on the basis of injury and made no direct growth or yield assessments, although subjective estimates of damage were obtained. Pell and Brennan[388] presented a well-developed thesis on the rationale for the difference in estimated losses to agriculture in New Jersey between 1971 and 1972. They discussed this in relation to the overall problem of assessing agricultural losses.

A survey was initiated in 1969[16] by Stanford Research Institute (SRI) to develop an empirical model for assessing damage to vegetation. This program made use of laboratory and field data from controlled exposures on various crops and chemical data from simulated reaction chambers, so estimates of ozone and other oxidants could be made on the basis of concentrations of primary pollutants. Hydrocarbon was chosen as the basic pollutant from which to develop the model for prediction of expected oxidant values and, therefore, effects on various crops. From the oxidant value, injury and damage for specific crops were calculated for over 100 statistical reporting areas in the United States. The report used many subjective assumptions and was related primarily to visible injury symptoms. The results were preliminary, and the approach had many deficiencies; but similar approaches should be contemplated in the development of damage functions that will give some reliable estimates of economic losses. On the basis of the SRI model, the 1969 estimated loss to vegetation from oxidants in the United States was approximately $125 million. Considering the increase in crop values and assuming that oxidant concentrations have not been signifi-

cantly reduced during the last few years, the loss in 1974 from oxidants could approach $300 million, on the basis of the SRI study.

A summary of estimates derived from various surveys and assessment techniques is shown in Table 11-28. These values are suggestive at best. As with all values developed for agricultural losses, these have been directed at losses to the producer—not to the consumer.

Whereas a reduction in production may actually increase the aggregate farm income and produce serious income distribution problems, the consequent reduction in marketable surplus would cause a significant rise in the cost to consumers, because of the inelastic consumer demand for most agricultural crops. Therefore, at the consumer level, losses based on farm prices are not appropriate and are likely to be conservative. Because of percentage markups and fixed wholesale and retail marketing costs, the cost to the consumer of agricultural losses could be twice as great as that observed at the farm level—i.e., a $300 million loss at the farm level in 1974 could represent a $600 million loss to the consumer.

INDEX OF PLANT NAMES AND REFERENCE NUMBERS

This index is a compendium of the literature on which the content of this chapter is based. References are *in addition* to those in Table 11-24 for the same plants. For complete references on a single species, the reader should check both the following list and Table 11-24.

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12

Ecosystems

The effects of ozone and other photochemical oxidants on individual species of green plants and microorganisms are discussed in detail in Chapter 11. The purpose of this chapter is to examine the effects of oxidant-pollutant stress on both simple and complex communities of organisms. The human population is an integral and dependent component of these biotic communities, or ecosystems.[49]

It is usually not possible to interpret the meaning of a stable ecosystem for man's welfare in the terms of conventional economics, for example, as a cost-benefit analysis. In the case of some natural ecosystems, there may be only social and psychologic values to be considered,[77] which are only remotely related to monetary values through the recreational opportunities that they offer.[79] One of the most important justifications for the examination of the effects of oxidant stress on an ecosystem is the concern that it will not revert to its prior condition even after removal of the stress. For example, it has been suggested that the arid lands of India are the result of defoliation and elimination of vegetation, which induced local climatic changes that were not conducive to the re-establishment of the original vegetation.[7]

A natural ecosystem is a distinct association of plants and animals with the physical environment that controls them. Spatial boundaries of ecosystems are defined when the physical and biologic parts form an in-

tegrated unit in which there are defined paths for energy flow and material transport or cycling. Classes of terrestrial ecosystems called "biomes" are distinguished by their dominant vegetation form—e.g., grasslands and deciduous and coniferous forests. Additional characteristics are emphasized in Odum's more formal definition: "Any unit including all of the organisms (i.e., the community) in a given area interacting with the physical environment so that a flow of energy leads to a clearly defined trophic structure, biotic diversity, and material cycles (i.e., exchange of materials between living and non-living parts) within the system is an ecological system or ecosystem."[50] The agroecosystem is defined as "a unit composed of the total complex of organisms in the crop area together with the overall conditioning environment and as further modified by the various agricultural, industrial, recreational, and social activities of man."[62] The important components and processes of natural ecosystems are summarized in the following list:

Components	
Inorganic substances (C, N, H_2O, etc.)	Autotrophs (producers)
Organic substances	Phagotrophs (macroconsumers)
Climatic regime	Saprotrophs (microconsumers)
Processes	
Energy-flow circuits	Nutrient (biogeochemical) cycles
Food chains (trophic relationships)	Development and evolution
Diversity patterns in time and space	Control

Ecosystems may be described developmentally as young (seral, successional) or mature (climatic). In a young ecosystem, developmental stages or communities are rapidly replaced by other communities. This succession leads ultimately to the mature stage. The distinguishing characteristic of the mature, or climax, stage is that the dominant species that form the community can replace themselves; thus, the community is in equilibrium with its normal environment.

There are important differences among the components and processes of young and mature ecosystems that result in different degrees of response to environmental stresses or new perturbations, such as the presence of oxidant air pollutants (Table 12-1). The greater diversity of a mature ecosystem slows down the disruption of normal structure and function. For example, a forest ecosystem in which many species make up the producer community would show less immediate visible damage than a successional stage with only a few species. Even greater damage would be anticipated in an agroecosystem (which might be considered the simplest of successional stages, inasmuch as often only a single producer species is present).

TABLE 12-1 Characteristics of Ecosystem Development

Agroecosystems and Successional (Young) Stages	Climax (Mature) Stages
High production:respiration ratio	High biomass: respiration ratio
High net production	Low net production
Short food chains	Complex food webs
Small organisms	Great diversity in size and numbers of organisms
Open nutrient cycles	Closed nutrient cycles
Lack of stability	High stability
Maintenance cost reduced by energy subsidies (by man); production increased	Maintenance cost high, but rarely subsidized

GENERAL RESPONSES OF NATURAL ECOSYSTEMS AND AGROECOSYSTEMS TO STRESS BY OXIDANTS

Ecosystems subjected to oxidant air pollutants must be carefully observed and described individually if we are to understand and predict the complex consequences of chronic injury. Woodwell[83] has summarized some of the expected effects of air pollutants on ecosystems: elimination of sensitive species and reduction of diversity in numbers of species; selective removal of larger overstory plants and a favoring of small plants; reduction of the standing crop of organic matter, which leads to a reduction of nutrient elements held within the living system; and increase in the activity of insect pests and in some diseases that hasten producer mortality. Many other effects can be suggested.

Photochemical-oxidant air pollutants have constituted a chronic problem only during the last 20–25 yr, at first principally in southern California, where both natural ecosystems and agroecosystems have been subjected to these pollutants for the longest period. Citrus groves and vineyards in the inland valleys of southern California are prime examples of agroecosystems stressed by chronic exposure to oxidants. Studies were initiated in 1960 on lemon and navel orange trees[71] and in 1968 on wine grapes[72] to determine the economic losses due to oxidant pollutants. Both studies were performed under field conditions for several years. The lemon and orange studies provided data that may be interpreted in an agroecosystem context, although they were highly oriented toward the primary-producer components.[73-76] There was no effort to examine the effects on consumer and decomposer components.

At two lemon groves and one orange grove near Ontario and Upland, California, 24 trees were selected at each grove and divided according to

a randomized block design into six treatments with four replications each. The most important treatments for this discussion were greenhouse-enclosed ambient air, enclosed carbon-filtered air, and unenclosed ambient air. The first difference noted was a lower rate of water use by oxidant-stressed trees. The number of irrigations required by trees in filtered air was always significantly greater than that in ambient-air treatments; this behavior could not be correlated well with transpiration rates.[76] From a systems view, one of the major input variables, irrigation water needed, would be lower for this stressed ecosystem. Comparisons of the apparent photosynthesis of single branches of trees in filtered and ambient air yielded mixed results, but with a trend toward reduced apparent photosynthesis in ambient-air treatments.[76] After five consecutive seasons of treatment at the two lemon groves and 4 yr at the orange grove, there were no significant differences in tree circumference among the treatments.[74] The lemon trees showed significantly greater leaf drop in both ambient-air treatments; the orange trees also followed this trend, but the differences were not significant. The drop of small, unripened fruit was a severe problem in ambient-air treatments with orange trees, but insignificant for lemons. The average annual yields at both lemon groves and the orange grove were significantly reduced when filtered versus unfiltered treatments were compared; yield was sometimes reduced by as much as 50%.[74] Additional studies with navel oranges exposed to ambient air, carbon-filtered air, and carbon-filtered air containing either ambient or half-ambient concentrations of ozone again showed increases in leaf and fruit drop and decreases in yield of marketable fruit in accordance with increasing ozone or oxidant dose.[73] None of the above studies managed to separate the different effects of ozone and peroxyacetylnitrate or its homologues in the photochemical-oxidant mixture.

In summary, oxidant stress reduced water use and photosynthesis, increased leaf drop and fruit drop, and resulted in a severe reduction in yield of marketable fruit. All these effects occurred without the development of plainly visible leaf symptoms.

Several inferences can be drawn from these data that may suggest the impacts to be expected at the consumer and decomposer levels. Accelerated leaf drop may influence the development of pests—namely, aphids, scale insects, and red citrus mites. Pest populations might be increased if injured leaves had higher concentrations of amino acids or free sugars before abscission (see Chapter 11) or diminished if leaves fell too rapidly. Leaf and fruit drop would provide a larger substrate for populations of decomposer organisms at the soil surface.

In southern California, the coastal chaparral ecosystem, dominated by

chamise and manzanita or woodland species (including the live oaks and big-cone Douglas fir), and the coniferous forest ecosystem have received severe exposure; and the desert ecosystems in the vicinity of mountain passes connecting the coastal and desert regions have undoubtedly been exposed. Injury has been well documented only in the mixed-conifer forest ecosystem of the San Bernardino Mountains. Early symptoms of injury in conifer species were reported in a number of California national forests in 1970.[46] In the southern Sierra Nevada, Forest Service surveys in 1974[82] detected increased injury in ponderosa pine since 1970 at many locations in the Sequoia National Forest, Sequoia National Park, and Kings Canyon National Park. Particular stands of mixed-conifer forest on the western slopes of the southern Sierra Nevada now appear to be affected by oxidants from the San Joaquin Valley.[44] The potential loss of timber growth alone in this area is a very serious prospect.

Injury to important primary-producer species constituting forest ecosystems is not limited to California. In the eastern United States, a disease called emergence tipburn of eastern white pine was related to ozone by Berry and Ripperton.[4] Occurrence of similar symptoms on the same species in eastern Canada could not be definitely related to ozone by Linzon.[39] The disease is characterized by bands of necrosis initiated in the semimature tissue of elongating needles; the necrosis spreads to the needle tip. In other studies with ozone fumigations at 0.07 ppm for 4 h or 0.03 ppm for 48 h, the tipburn appeared; additional symptoms were silvery or chlorotic flecks and chlorotic mottling.[13]

Under forest conditions, the affected trees occur randomly in the stand; the same trees are injured successively in a single season or in successive years.[3] Eastern white pine either forms pure stands or occurs in mixtures with other species in abandoned fields; under these conditions, it is an important pioneer tree.[80] In established stands, it is a major component of 4 forest types and an associate in 14 other types with a range extending over 7 million acres from the Lake States to the Applachian Mountains.[80] Berry[3] reported that emergence tipburn occurs throughout the natural range of this species; there is also evidence of a slow decline in tree vigor due to the deterioration of feeder rootlets.

Higher concentrations of ozone in the forested areas of the eastern United States would undoubtedly cause greater injury to eastern white pine and other forest species. Chapter 11 reports additional studies that suggested that other conifer species, in particular Virginia pine and jack pine, may be more sensitive to ozone than eastern white pine.[16] In addition, there is a synergistic interaction between low concentrations of ozone and sulfur dioxide that is the cause of the chlorotic dwarf disease

of eastern white pine.[17] A study by Ellertsen *et al.*[21] showed 10% mortality between 1956 and 1965 of dominant and codominant eastern white pines near an industrialized area including several hundred square miles on the Cumberland Plateau. Both ozone and sulfur dioxide were considered responsible for tree decline. Because eastern white pine represented only 5% of the total wood volume available for harvest, the economic impact was slight. There was no effort to interpret pollutant effects in an ecosystem context. An air monitoring network was operated by Virginia Polytechnic Institute in 1975 at three locations—the Blue Ridge Mountains, the Shenandoah Valley, and the southern Appalachians. According to J. M. Skelly (personal communication), a pollution episode occurred during early July 1975 during which total-oxidant peaks as high as 0.13 ppm were observed, along with 43 h when concentrations were 0.08 ppm or higher. After this episode, significant increases in oxidant injury were observed, particularly in the Blue Ridge Mountains, in three categories of eastern white pine—those previously without symptoms, those with chlorotic mottle, and those exhibiting chlorotic dwarf symptoms. Such incidences suggest the need for more comprehensive studies of oxidant (and sulfur dioxide) effects in forests of the eastern United States. In the long run, the broader question regarding effects of pollutant stress on all ecosystem components—primary producers, consumers, and decomposers—should be addressed. An analysis of the multiple effects of oxidants on eastern forest ecosystems will be a much more adequate measure of their future usefulness to man than the small amount of information now available related mainly to single primary-producer species.

The purpose of this chapter is to examine in the greatest detail possible the effects of oxidant air pollutants on ecosystems. A project is now going on to study the effects on a mixed-conifer forest ecosystem in southern California,[67,68] and the planning documents and early results from this study constitute the major source of information for the remainder of this chapter. Other examples of damage to agroecosystems and natural ecosystems are included.

ORIGIN OF INJURIOUS CONCENTRATIONS OF OZONE AND OXIDANTS AFFECTING NATURAL ECOSYSTEMS

Advection from Urban Centers to Remote Areas

SOUTHERN CALIFORNIA

Descriptions of the vertical and horizontal distributions of photochemical smog in the Los Angeles basin (southern coastal air basin) during

typical summer days have recently been provided by Blumenthal *et al.*,[5] Edinger,[19] Edinger *et al.*,[20] and Miller *et al.*[45] Important observations to be drawn from their reports are the interactions of basin and mountain topography and local meteorology in determining the transport and concentrations of oxidant air pollutants in relation to elevational zones of vegetation.

The marine temperature inversion layer that frequently forms above the heavily urbanized Los Angeles metropolitan area often extends inland as far as 90 miles (144 km), depending on season and time of day. Surface heating of air under the inversion increases with distance eastward in the basin and often disrupts the inversion by midmorning at its eastern edge. The northern rim of the basin is formed by the San Gabriel and San Bernardino Mountains, interrupted only by the Cajon Pass about 55 miles (88 km) inland (see Figure 12-1). The marine temperature inversion layer encounters the mountain slopes usually below 4,000 ft (1,200 m). In the morning, the temperature inversion often remains intact at this juncture, and air pollutants are confined beneath it. Studies by Edinger *et al.*[20] and Edinger[19] have described how the heated mountain slopes act to vent oxidant air pollutants over the crest of the mountains and cause the injection of pollutants into the stable inversion layer horizontally away from the slope. Oxidant concentration within the inversion is nonuniform, containing multiple layers and strong vertical gradients. In some cases, the inversion may serve as a reservoir for oxidants, principally ozone, which may arrive at downwind locations along the mountain slopes relatively undiluted, because of a lack of vertical mixing within the inversion layer and a lack of contact with ozone-destroying material generated at the ground. The important result of the oxidant's being trapped in these layers is its prolonged contact with high terrain at night.

The most important effect of the interaction of pollutant and inversion layer at the heated mountain slope is the vertical venting of oxidants over the mountain crest by upslope flow. A gradient of oxidant concentrations or doses is established across distinct vegetation zones ordered along an elevational gradient—i.e., the chaparral and conifer forest ecosystems that occupy the slopes and mountain terrain beyond the crest, respectively. The altitudinal sequence of these ecosystems is illustrated in Figure 12-2. According to Horton,[27] the chaparral zone is subdivided from lower to higher elevations into three subzones called the chamise, manzanita, and woodland chaparral; the mixed-conifer forest occupies the mountain crest.

The daytime changes in oxidant concentrations at several stations (A, B, C, and D in Figure 12-1) along the southern slope and at the crest of the San Bernardino Mountains are illustrated in Figure 12-3. In

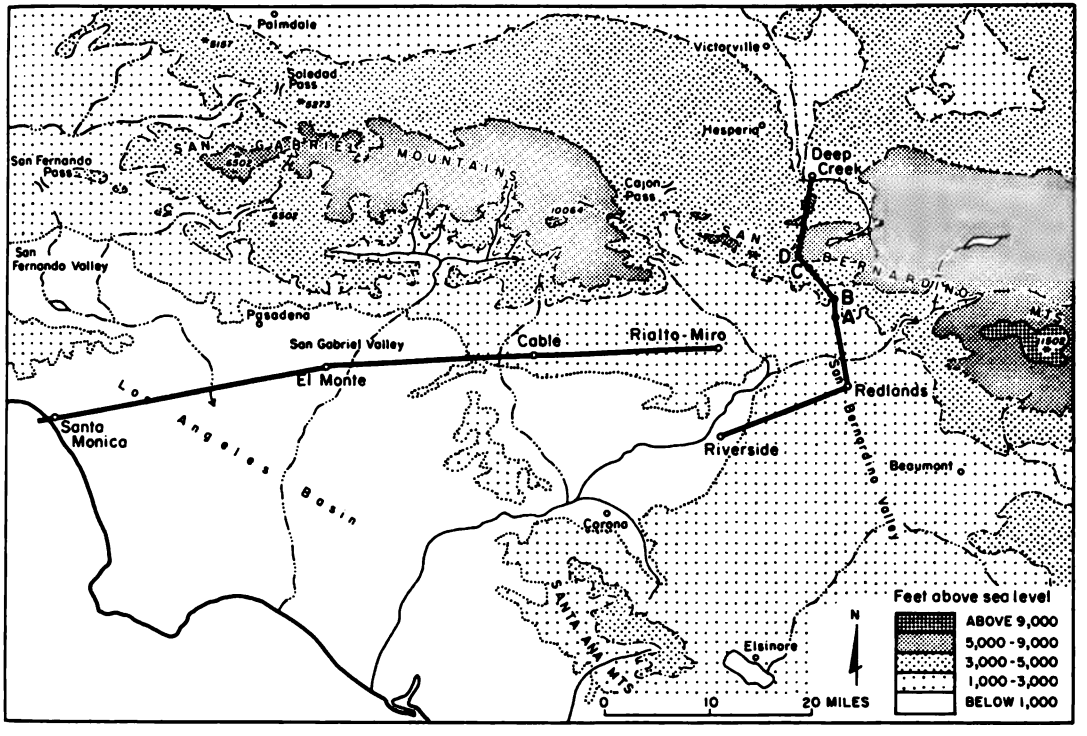

FIGURE 12-1 Major topographic features of the Los Angeles basin with inland valleys and mountains. Station locations: A, Highland; B, City Creek; C, Mud Flat; D, Rim Forest. Aircraft flight paths for the study area are also shown. Reprinted with permission from Edinger *et al.*[20]

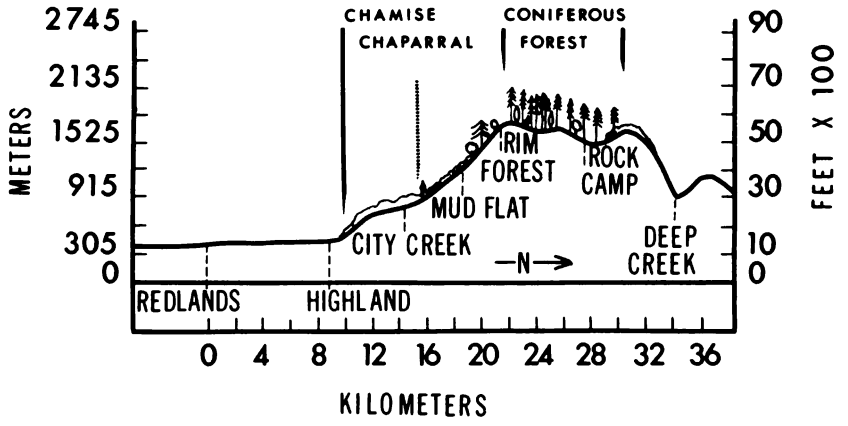

FIGURE 12-2 Altitudinal sequence of ecosystems in the San Bernardino Mountains. Reprinted with permission from Miller *et al.*[45]

the late afternoon, the highest concentrations in this profile were at 3,000-4,000 ft (900-1,200 m) and adjacent to the mountain slope. The oxidant was not always confined below the inversion layer, but was present in the inversion layer and above the mountain crest.[20] An instrumented aircraft measured concentrations of oxidant ranging from 0.05 to 0.11 ppm as high as 2,432 m (approximately 1,033 m above the ridge crest) during several noon and 4 p.m. flights. At these times, downwind diffusion of oxidant beyond the crest—on the basis of aircraft flights approximately 500 ft (150 m) above the terrain—suggested only slight dilution in the first 10 km north of the crest.[20] Observations on other days[45] suggested a 50-60% reduction in oxidant 10 km north of the crest. There was no ground station at this point for comparison with the aircraft measurements at either time.

Total oxidant, temperature, and vapor-pressure gradient were measured continuously during 16 days in July and August at mountain slope and crest stations (A, B, C, and D in Figure 12-1). In Figure 12-4, the time of the daily peak oxidant concentration was progressively later at stations of higher elevation. Temperatures and vapor-pressure gradients were also progressively lower at higher elevations at the time of oxidant peak. The duration of oxidant concentrations exceeding 0.10 ppm was 9, 13, 9, and 8 h/day going from lower to higher stations. The longer duration at City Creek (elevation, 817 m) probably coincides with the point where the inversion layer most often contacts the mountain slope. The oxidant concentrations rarely decreased below 0.05 ppm at night

on the slope of the mountain crest, whereas it usually decayed to near zero at the basin station (Highland).

The vegetation zones along the slope and at the crest are subjected to oxidant exposure differently. Even though the lower chaparral receives longer exposure, the peak concentrations coincide more closely with the maximal evaporative stress for the day. There is some support for the hypothesis that stomates would be closed during this period and that pollutant uptake would be lower. There is in fact very little visible injury to the species in this zone. In contrast, the daily oxidant peak occurs well after the temperature and vapor-pressure gradient maximums in

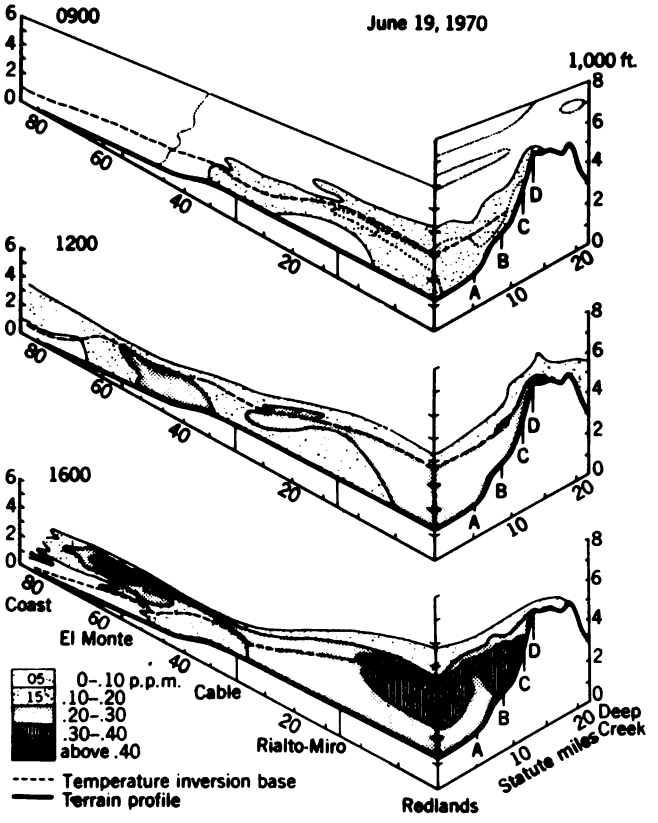

FIGURE 12-3 Daytime changes in oxidant concentrations along a west-to-east transect in the southern coastal air basin, including the slopes of the San Bernardino Mountains (see Figure 12-1). Reprinted with permission from Edinger *et al.*[20]

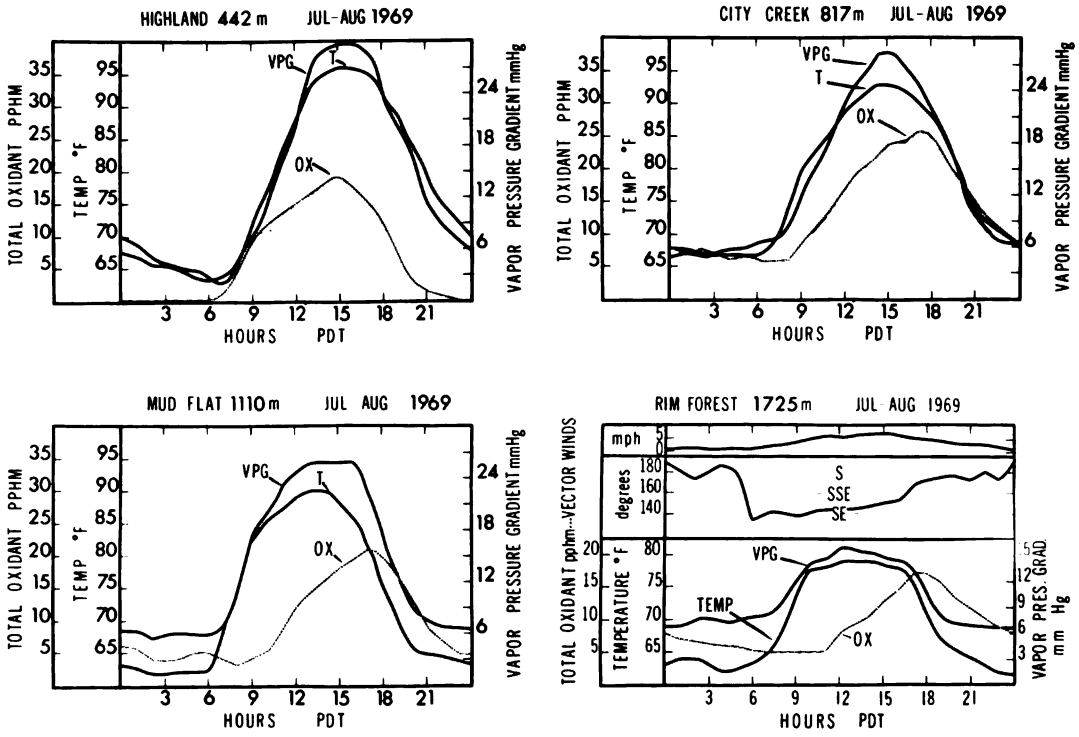

FIGURE 12-4 The relationship of times of occurrence of the daily oxidant peak concentration to temperatures and vapor-pressure gradients in an elevational sequence (see Figures 12-1 and 12-2) on the slopes of the San Bernardino Mountains, July-August 1969. Reprinted with permission from Miller *et al.*[45]

the conifer forest at the mountain crest; the oxidant injury to plant species here is severe. These suggestions of strong microclimatic control of the sensitivity of these native species to ozone form a working hypothesis that needs further investigation.

THE SAN JOAQUIN VALLEY AND ADJACENT SIERRA NEVADA MOUNTAINS

Field surveys[46,82] have confirmed oxidant injury to ponderosa pine and associated species at numerous locations in the Sierra Nevada foothills east and southeast of Fresno. Oxidant measurements at ground stations and by instrumented aircraft show late-afternoon peaks of "transported" oxidant on the western slopes of the Sierras. Limited measurements by instrumented aircraft suggest the development of a layer of oxidant approaching the forested mountain slopes between 610 and 1,829 m during the late afternoon.[44] A very weak inversion or isothermal layer may serve as a reservoir of oxidant, which is advected to the mountain slope in the southern coastal air basin, as suggested by Edinger.[19]

Considerable concern has been registered about air quality in the Lake Tahoe basin, where local development may cause adverse oxidant concentrations.[9]

Seasonal and Daily Variations of Injurious Concentrations

SYNOPTIC WEATHER PATTERNS ASSOCIATED WITH EPISODES OF HIGH POLLUTION

McCutchan and Schroeder[41] classified 145 days during May through September 1970 with data collected on the southern slopes of the San Bernardino Mountains. Five general weather patterns were described, with the associated synoptic patterns at the surface and at 500 mb (Table 12-2). An analysis of eight meteorologic variables was used to classify days during May through September into the five general weather categories. Of 123 days classified by stepwise discriminant analysis, 10, 13, 44, 23, and 24 were correctly categorized into classes 1-5, respectively; the remainder could not be clearly placed in the five classes. The oxidant concentrations associated with these classes were: 1, low; 2, low and high; 3, high; 4, low; and 5, low. In this sample, the sum of days in classes 2 and 3 (57 days) suggests that about 46% of the sample days had high concentrations of oxidant air pollutants.

Class 2 and 3 days are both characterized by high pressure (500-mb

TABLE 12-2 Descriptions of Meteorologic Patterns for Five Classes of Spring and Summer Days in Southern California[a]

Class	General Weather	Associated Synoptic Pattern	
		At Surface	At 500 mb
1	Hot, dry continental air throughout the day (Santa Ana)	Large high pressure over Great Basin	Strong northerly winds over area with trough east of the area
2	Relatively dry forenoon; modified marine air in afternoon; very hot (heat wave)	High pressure over Great Basin and thermal trough over desert	Subtropical closed high over area
3	Moist, modified marine air; hot in afternoon	Thermal trough over desert	Ridge over area
4	Moist, modified marine air; warm in afternoon	Thermal trough over desert	Trough over area
5	Cool, moist, deep marine air throughout the day	Synoptic low over desert	Deep trough or closed low over area

[a] Derived from McCutchan and Schroeder.[41]

ridge over the area). Descriptions of the synoptic weather patterns (contained in the U.S. Forest Service "California Fire Weather Severity, 10-Day Summaries") for May through August 1972-1974 were compared with episodes of severe oxidant concentration at Rim Forest and nearby Sky Forest. For the purposes of this comparison, all days having maximal hourly concentrations of 0.33 ppm or higher were compared with the synoptic patterns for those days. In all cases, a persistent 500-mb high pressure over the area was the most common synoptic feature. For 8 qualifying days in 1972, the mean of the maximal hourly concentrations for those days was 0.37 ppm; for 16 days in 1973, 0.41 ppm; and for 46 days in 1974, 0.38 ppm. The highest hourly concentration ever obtained was 0.60 ppm, on June 28, 1974.

During episodes of severe oxidant pollution, the weather is generally very hot (85-100° F or about 29-38° C) and the relative humidity may be either low or moderately high on class 2 and 3 days, depending on the behavior of the marine layer. The small difference between means of maximal hourly concentrations on high-pollution days in 1972-1974 suggests continuation of heavy primary-pollutant emission in spite of current control strategies.

ANNUAL TRENDS OF TOTAL-OXIDANT CONCENTRATIONS AT A SAN BERNARDINO MOUNTAIN STATION AND THE NEARBY CITY OF SAN BERNARDINO

Since 1968, total-oxidant concentrations have been measured continuously with a Mast ozone meter (calibrated by the California Air Resources Board method) from May through September at Rim Forest-Sky Forest.[32] The fall, winter, and early spring months have generally been omitted until recently, because synoptic patterns are usually not conducive to oxidant accumulation and transport. For example, average maximal hourly oxidant concentrations from October through April 1973 and 1974 stayed below 0.10 ppm; those for April were 0.10-0.15 ppm.[68] The main data-collection period coincides with the growing season and thus permits a reasonable estimate of the total annual dose of oxidant air pollution received by vegetation.

Two methods of documenting trends of oxidant concentrations during the 1968-1974 period at Rim Forest-Sky Forest express the accumulated dose as a sum of all hourly values in micrograms per cubic meter and as number of hours with a concentration of 0.08 ppm (the federal standard) or more for each month separately, June through September (Figure 12-5). The dose excludes background concentrations—those less than 59

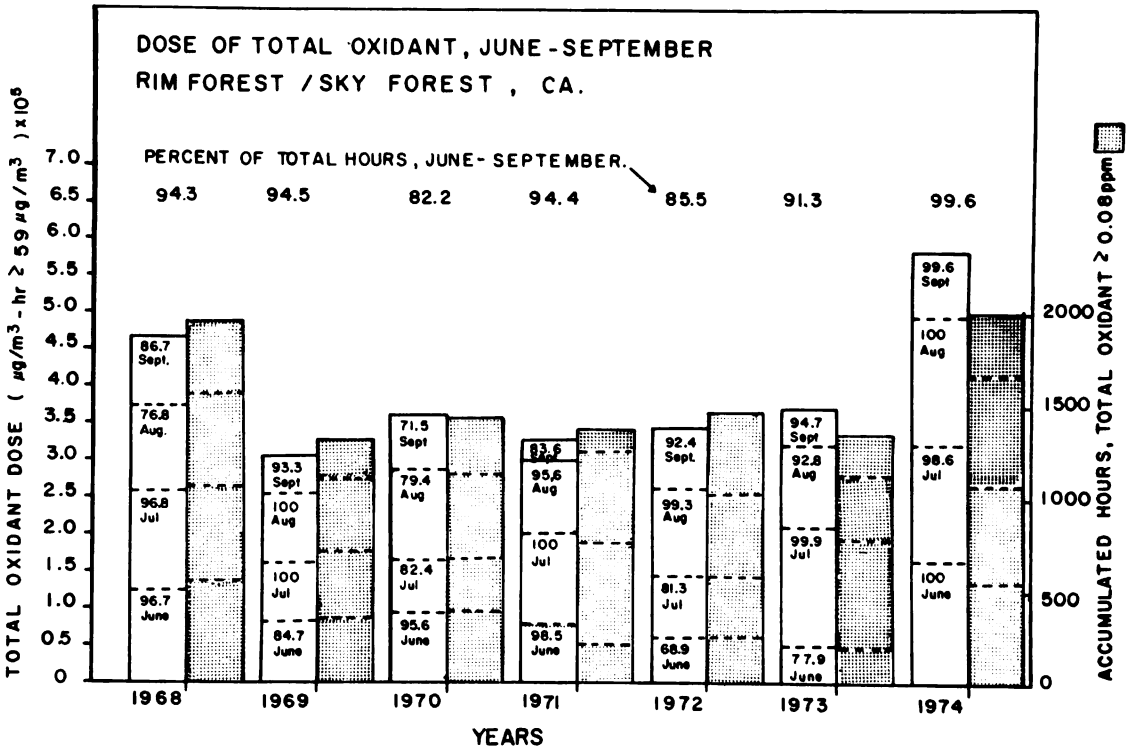

FIGURE 12-5 Monthly summation, June-September 1968–1974, of total-oxidant dose and total number of hours at a dose ≥ 157 $\mu g/m^3$ (0.08 ppm) at Rim Forest-Sky Forest, Calif., in the San Bernardino Mountains. Numbers in open bars indicate percentage of total possible hours for which data were available. Reprinted with permission from Kickert *et al.*[32]

$\mu g/m^3$ (0.03 ppm). The percentage of valid data recovered is also indicated. The absence of some data—ranging up to 17.8% in 1970, but averaging 8.3% during the 7 yr—represents an underestimate that cannot be adjusted with any certainty. The total number of hours with concentrations of 157 $\mu g/m^3$ (0.08 ppm) or more during June through September was never less than 1,300 during each of the 7 yr. These measures of oxidant trends indicate no improvement in air quality.

Data are available back to 1963 from the downtown San Bernardino station operated by the county Air Pollution Control District (APCD). The colorimetric potassium iodide method used to measure total oxidants was calibrated according to the method of the California Air Resources Board. A positive correction factor of 1.22 was used to adjust mountain data for the decreased air pressure at the higher elevation.

The data from the Rim Forest-Sky Forest station were compared with published data from the San Bernardino County APCD. The numbers of hours with concentrations exceeding 392 $\mu g/m^3$ (0.20 ppm) during July, August, and September in 1969-1974 were compared (Figure 12-6). For 1963-1968, data are shown only from the San Bernardino APCD. A large part of the year-to-year differences at the same station and between stations can be attributed to differences in synoptic and mesoscale (local) meteorologic patterns. For example, the increases in 1972, 1973, and 1974 at Rim Forest-Sky Forest are associated with 6, 16, and 46 days, respectively, when a persistent 500-mb ridge occurred over the Southwest, particularly southern California. The difference between stations in the same year is probably influenced most by inversion height. Lower inversions partially restrain transport upslope to shorter periods each day. Higher inversions would have the opposite effect and also allow a

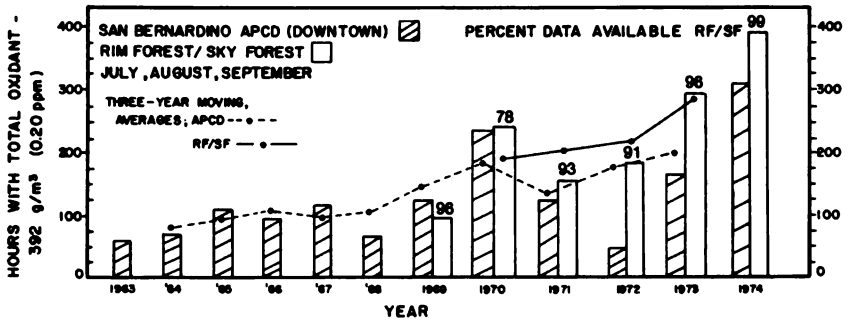

FIGURE 12-6 Number of hours of total oxidant, July-September, 392 $\mu g/m^3$/0.20 ppm, at downtown San Bernardino County on Pollution Control District Station, 1963-1964, and Rim Forest-Sky Forest, 1968-1974. After Kickert *et al.*[32]

greater air volume below to dilute oxidants. The index for comparison chosen in Figure 12-6 (i.e., hours $\geqslant 392\ \mu g/m^3$) would be sensitive to inversion height. The 3-yr moving averages for each station tend to remove some of the variation due to meteorology. In terms of hours equal to or exceeding $392\ \mu g/m^3$, the moving average between 1970 and 1973 at Sky Forest-Rim Forest has increased from 175 to 290. The increased oxidant concentrations at both these stations are the reverse of those in upwind, urban Los Angeles County, where increased emission of NO_x tends to shift the chemical equilibrium to the left, toward the ozone precursors. The most recent data[11] firmly indicate that oxidant concentrations will either increase annually or continue to oscillate around the mean of present high concentrations in the foreseeable future at these distant locations.

The tropospheric ozone cycle[11] describing rural upwind, urban, and rural downwind variations in concentration can be easily demonstrated in the Los Angeles and connected inland basins. The effects on both natural ecosystems and agroecosystems also become apparent. For example, oxidant doses during August 1972 at six oxidant stations—Costa Mesa, La Habra, Corona, Riverside, City Creek, and Rim Forest—extending in a line northeastward from the coast to the mountains show low doses at the coast increasing to a maximum on the chaparral-covered mountains and decreasing beyond the mountain crest (Figure 12-7). The oxidant dose is indicated by the dashed line. A crude estimate of the relative economic value of the products from ecosystems encountered along this transect is expressed in a very general way by the solid line and the nomenclature on the abscissa. Finally, the relative complexity of the ecosystems involved is shown below the abscissa. This conceptualization emphasizes the enormously greater dosage received by the natural ecosystems during this month. This pattern of dosages is very typical of June, July, and August, but the offshore flow typified by the Santa Ana winds may reverse the situation in September or October; susceptible crops growing on the coastal plain may be seriously damaged.

In general, the permanent vegetation constituting natural ecosystems receives much greater chronic exposure, whereas the short-lived higher-value vegetation constituting the agroecosystems of the coastal plain can be subject to injurious doses, but in intermittent short-term fumigations. Each situation has measurable economic and aesthetic effects, but on different time scales. The simple agroecosystem has little resiliency to pollutant stress; losses are immediate and may be catastrophic. The complex natural ecosystem is initially more resistant to pollutant stress, but the longer chronic exposures cause the disruption of both structure and function in the system. Damage may not be reversible.

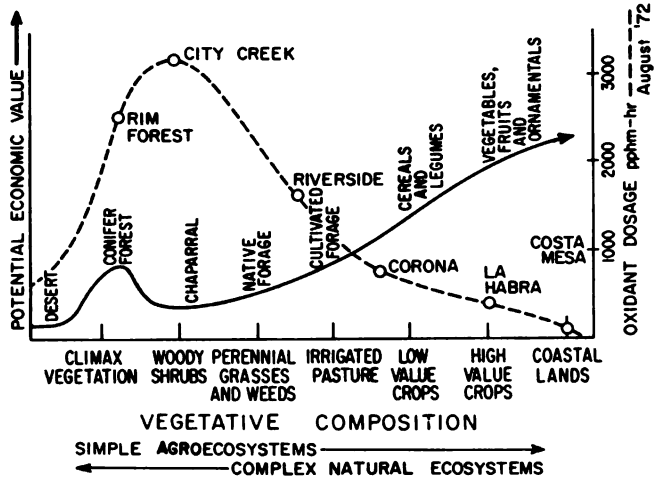

FIGURE 12-7 A hypothetical expression of the relationships of oxidant dose, natural ecosystems, agroecosystems, and the relative values of their vegetative components along a transect in the southern coastal air basin of California. The only real data are oxidant doses, which define the pollutant concentration gradient from the coast to the interior.

AN EXPERIMENTAL DESIGN FOR INVESTIGATING THE EFFECTS OF OXIDANT STRESS ON ECOSYSTEMS

Techniques for Ecosystem Modeling

The steps needed to understand the effects of oxidant air pollutants on an ecosystem are description of ecosystem components or subsystems and identification of the most important processes within organisms or interactions between organisms or communities of organisms that would be most vulnerable to pollutant stress, including an evaluation of factors of the physical environment that may limit ecosystem processes. This information can be conveniently arrayed in terms of interaction tables that show which variables are involved in various subsystems and whether the interaction between any two variables is positive or negative. The next step involves the selection of the most appropriate paradigm for the substance or "unifying thread" that flows through the various subsystems. Examples of possible "flows" are energy, biomass, mineral nutrients, water, numbers of species, population densities, or area occupied per biotic unit.

The state of the art of ecosystem modeling has evolved rapidly in re-

cent years. Discussions of various ecologic subsystem simulators are published.[30,35,52] Recently, discussions of the utility of computer simulation modeling in ecologic research have been published by the National Academy of Sciences[48] and the National Science Foundation.[70]

Depending on the "flows" through the ecosystem that one chooses to study, the modeling procedures may be either deterministic or stochastic. Rochow[58] has described the step-by-step procedure for studying the flow of nitrogen through a forest ecosystem. In this case, nitrogen can be found in a number of well-defined storage compartments. The transfers of nitrogen between compartments are described by transfer coefficients arrayed in a symmetric transfer matrix. Differential equations are written that represent the rate of change (or flow) of nitrogen through time for each of the various compartments. Computer programs already exist to solve these equations. Once the model is running smoothly on the computer, it is possible to impose various experimental conditions—e.g., clear-cutting or selective cutting—and observe the effects on nitrogen flow. Sensitivity analysis can then be performed to study which transfer coefficients are sensitive to small changes and which only to large changes. The useful outputs of these manipulations include a determination of how much particular transfers in the system can be changed or stressed before the ecosystem deteriorates. This example uses a deterministic model; however, the flow of water through an ecosystem may have both deterministic and stochastic mathematical properties.[15]

Models of energy flow or biomass flow have been more successful when uniform plant canopies were involved—e.g., a field of corn. A recent development from the field of integrated pest management has been described in which an alfalfa growth model has been coupled with an alfalfa weevil population dynamics model.[24] The plant growth model in this example might be adaptable to air pollutant injury studies in agroecosystems. Because the model is built to incorporate current weather data that have direct influences on pollutant concentration and plant sensitivity, this type of model seems to be a worthwhile departure point for simulating dose responses.

The simulation of the population structure and dynamics of autotrophs and phagotrophs is another important interaction that can be modeled to test for effects of pollutant stress. A standard approach is the use of a finite-population-difference model.[36] The model assumes that the population change of a species in a specific period is equal to the species population multiplied by an intrinsic coefficient of rate of change. The rate coefficients are difficult to define without extensive data. The task is further complicated because "a consistent feature of

communities is that they contain *a comparatively few species that are common*—that is, represented by large numbers of individuals or a large biomass—and *a comparatively large number of species that are rare at any given locus in time and space*."[49] This statement implies that adequate sampling procedures may be difficult to design if only the spatial aspect of the problem is considered. The constraints of short-term research funding in the form of grants or contracts generally preclude adequate sampling of the time dimension. Ewing *et al.*[22] have considered a related modeling constraint: the simulation of the hierarchic structure inherent in a biologic population. Through exploitation of the structure of the biologic, mathematical, and computer systems, a Monte Carlo simulation of a structured population is possible. With a nonhomogeneous Poisson process, events are defined and a suitable risk structure is developed that satisfies mathematical and biologic requirements. A structured computer language allows one to mimic the structure and organization of a population—different levels of organization ranging from the individual to the complete population.

A description of another "flow," the area occupied per biotic unit over time, is also very important for defining and predicting long-term successional changes due to pollutant stress in plant communities. Shugart *et al.*[61] have described a model for 250 yr of succession in the western Great Lakes region in the absence of fire, epidemics, and forest management. Botkin and Miller[6] predicted successional changes during a 100-yr period after logging in a northern hardwood forest.

There are no examples of models dealing directly with oxidant stress on communities or ecosystems. However, the types of models just described may be applicable to this problem. In view of the trend of increasing oxidant concentrations downwind from urban areas,[5] a much more precise understanding of chronic effects on both natural ecosystems and agroecosystems is required. Descriptive and predictive models dealing with the responses of biologic systems can provide the best input to the decision models needed for standard-setting, land-use planning, and resource management.

Modeling the Effects of Oxidant Stress on a Western Mixed-Conifer Forest Ecosystem

The recent history of the mixed-conifer forest of the San Bernardino Mountains has been analyzed.[67] This analysis included an initial inventory of ecosystem components and processes, as indicated in Figures 12-8 through 12-12. The inventory emphasizes ponderosa and Jeffrey pines, the most dominant species in the climax community, and is

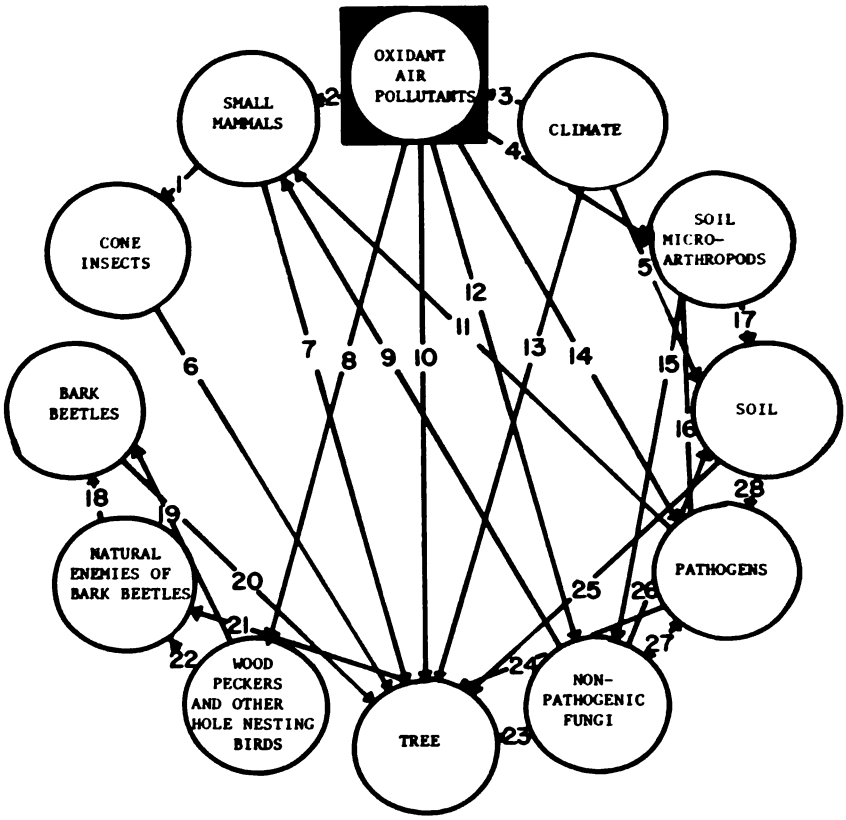

FIGURE 12-8 Organism-level interactions in a mixed-conifier forest ecosystem. Reprinted with permission from Taylor.[66] Types of interaction:

1 Competition for food supply
2 Direct effect of ozone on small-mammal physiology
3 Climatic control of oxidant concentration in forest
4 Direct effect of ozone on soil microarthropods
5 Effect of precipitation and temperature on soil moisture and soil temperature
6 Insect damage to developing cones
7 Small-mammal damage to developing cones and importance of cone crop as animal food supply
8 Direct effect of ozone on physiology of cavity-nesting birds
9 Role of fruiting bodies of nonpathogenic fungi in nutrition of small mammals
10 Direct effect of ozone on tree physiology
11 Role of fruiting bodies of pathogens in nutrition of small mammals
12 Direct effect of ozone on nonpathogenic fungi
13 Effect of temperature and evaporative stress on tree growth
14 Direct effect of ozone on pathogenic fungi
15 Interaction of nonpathogenic fungi and soil microarthropods
16 Interaction of pathogenic fungi and soil microarthropods

stratified according to organizational level—namely, organism or tree (Figures 12-8 and 12-9), community or stand (Figures 12-10 and 12-11), and finally a time-oriented analysis of plant succession in the aggregation of relatively distinct communities or stands that collectively make up the mixed-conifer forest ecosystem (Figure 12-12). This analysis was assembled by nearly a dozen coinvestigators representing many disciplines.[68] These detailed diagrams are presented here to emphasize the complexity of ecosystem interactions at each level of biologic organization. Only the interactions considered to be the most important ones guiding the course of plant succession in the several forest plant communities now constituting the mixed-conifer forest have been selected for immediate investigation.[69] In the case of each interaction, it is important to decide the time frequency at which the selected state variables and driving variables will be measured—i.e., hourly, weekly, or annually. A data capture, storage, and retrieval system is being devised to provide modelers with immediate access to all subproject data.

Because this project is currently the most active effort directed toward understanding the effects of ozone and other oxidants on a natural ecosystem, the remainder of this chapter refers frequently to its progress.

Other researchers at the University of Utah[26,56,78] have completed studies to determine the ozone susceptibility of the most prevalent species in some major vegetation associations in the intermountain area. These data are intended to be combined with background information on community stability and to be integrated into mathematical equations. The equations may be able to predict the community changes that occur after stress of various degrees. The ozone concentrations in grassland and aspen plant communities in the Wasatch Mountains above Salt Lake City are much lower than those in the San Bernardino Mountains. According to Treshow and Stewart,[78] "background ozone concentrations in the grassland communities geographically contiguous to the

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17 Effect of soil microarthropods on litter reduction
18 Impact of predation and parasitism on bark beetles
19 Predation of bark beetles by woodpeckers
20 Effect of bark beetles on tree mortality and vigor and effect of phloem thickness and moisture on bark beetles
21 Effect of phloem moisture and thickness on natural enemies of bark beetles
22 Influence of woodpeckers on rate of parasitism
23 Effect of nonpathogenic fungi on tree growth
24 Effect of pathogens on tree vigor and mortality
25 Effect of soil moisture and soil temperature on tree growth
26 Effect of soil moisture and temperature on occurrence of nonpathogenic fungi
27 Interaction of pathogenic and nonpathogenic fungi
28 Effect of soil moisture and temperature on occurrence of pathogenic fungi.

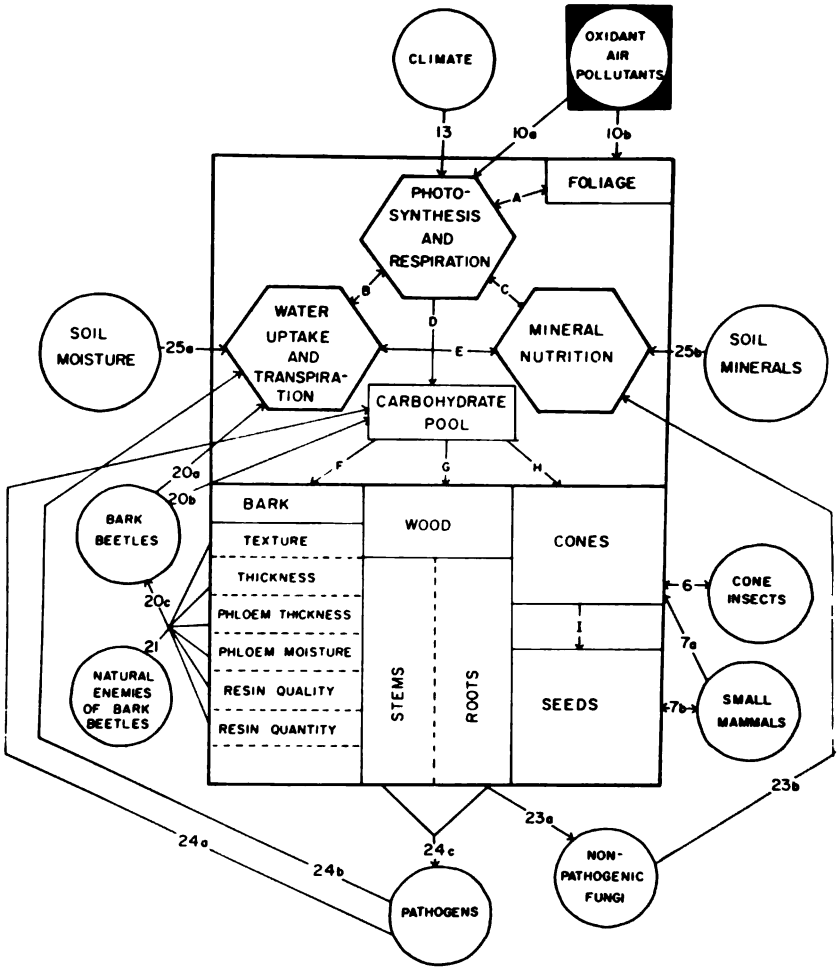

FIGURE 12-9 Tree-level interactions in a mixed-conifer forest ecosystem. a indicates direct influence of external factor on trees; b and c indicate influence of tree condition on external factor. Reprinted with permission from Taylor.[68] Types of interaction:

- A Relationship between net photosynthesis, foliage age, and foliage retention
- B Effect of plant moisture stress on net photosynthesis
- C Relationship between mineral uptake and net photosynthesis
- D Relationship between net photosynthesis and carbohydrate storage
- E Relationship between water uptake and mineral nutrition
- F Relationship between carbohydrate storage and bark characteristics
- G Relationship between carbohydrate storage and wood
- H Relationship between carbohydrate storage and cone production

- 1 Relationship between cone crop and seed crop
- 6 Cone insect damage to developing cones
- 7a Small-mammal damage to developing cones

Salt Lake Valley occasionally reached peaks at 15 pphm, but the average concentration over a 1-h period never exceeded 6 pphm at any of the plots. Concentrations measured in the aspen community during the study reached 9 pphm but never for more than a few minutes at a time." The present low concentrations may be important to some species or cultivars[26,56] and the plant communities of which they are members.

EFFECTS ON PRIMARY PRODUCERS (GREEN PLANTS)

The ozone dose responses and the specific effects on the photosynthetic activity of both herbaceous and woody plants, principally in controlled short exposures, are discussed in Chapter 11. The main aim of this section is to evaluate the effects of the chronic exposure of vegetation in natural ecosystems to total oxidants (more than 90% ozone) under field conditions or simulated field conditions. The effects of chronic exposure on agroecosystems are also discussed to a limited extent in Chapter 11.

W. H. Smith[63] has suggested three classes of ecosystem response to air pollution: those in which vegetation and soils serve only as a sink for pollutants, with no visible injury; those in which some species or sensitive individuals within species are injured and are more subject to other

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7b Small-mammal predation of seeds
10a Effect of ozone on photosynthesis and respiration
10b Effect of ozone on needle retention
13 Effect of temperature, light intensity, and evaporative stress on photosynthesis and respiration
20a Effect of bark beetles on water uptake and transpiration and relationships of tree moisture stress to bark beetle attack
20b Effect of bark beetles on tree carbohydrate concentration and relationships of carbohydrate concentration to bark beetle population in tree
20c Relationships between bark characteristics and bark beetle attack and population
21 Relationship between bark characteristics and natural enemies of bark beetles
23a Relationship between root characteristics and mycorrhizal-forming nonpathogenic fungi
23b Effect of nonpathogenic fungi on mineral uptake
24a Effect of pathogens on tree carbohydrate concentration and relationship of carbohydrate concentrations to pathogen attack
24b Effect of pathogens on water uptake and transpiration
24c Relationship between stem and root characteristics and pathogen attack
25a Effect of soil moisture and temperature on water uptake and transpiration
25b Effect of soil mineral concentration and temperature on mineral nutrition of tree

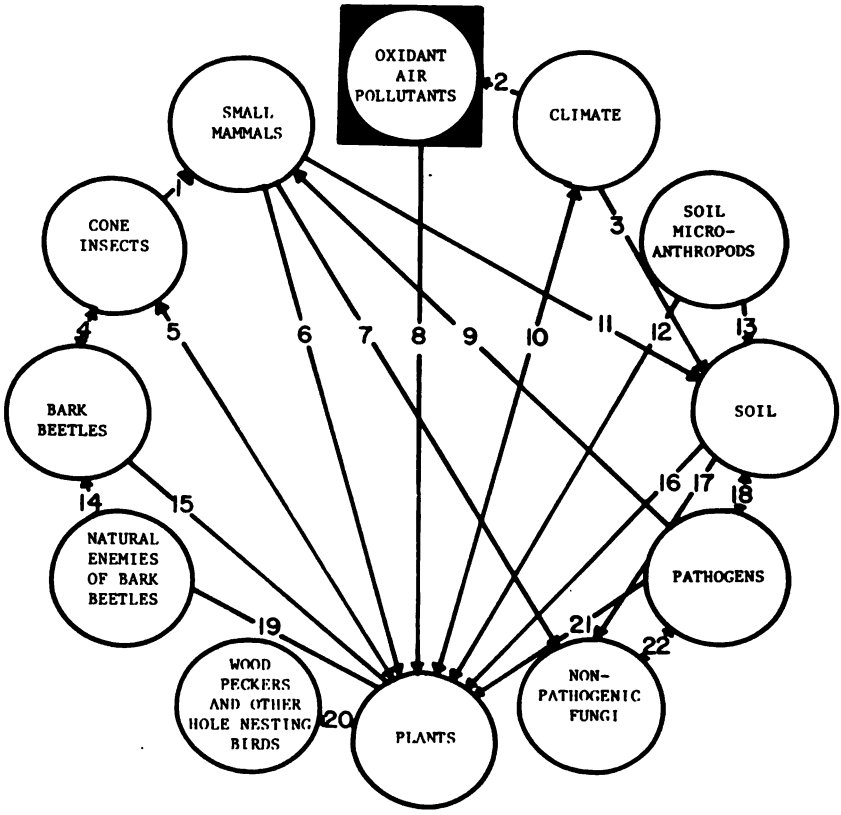

FIGURE 12-10 Community-level interactions in a mixed-conifer forest ecosystem. Reprinted with permission from Taylor.[68] Types of interaction:

1 Competition between woodpeckers and small mammals

2 Climate control of oxidant concentration in different forest communities

3 Effect of precipitation and temperature on soil moisture and soil temperature in different forest communities

4 Predation of bark beetles by woodpeckers in different forest communities

5 Effect of cone crop abundance on cone insect populations in different forest communities

6 Effect of cone crop abundance on small-mammal populations in different forest communities

7 Fruiting bodies of nonpathogenic fungi as food for small mammals in different forest communities

8 Smog-caused mortality and morbidity in different forest communities

9 Fruiting bodies of pathogens as food for small mammals in different forest communities

10 Effect of temperature and evaporative stress on species composition in different forest communities

11 Relationship between soil characteristics and population density of burrowing small mammals in different forest communities

stresses; and those in which high dosages cause acute morbidity or mortality of some species. Although these classes are convenient for discussion, in most situations they are not clearly separated in space. More often, these classes of response occur along a gradient of decreasing pollutant exposure, as in the San Bernardino Mountains.

Extent and Intensity of Injury to Overstory Trees in the San Bernardino National Forest

Wert[81] used aerial photography to determine the extent of oxidant injury to ponderosa and Jeffrey pines in diameter classes larger than 30 cm. Injury was categorized as heavy, moderate, light, or negligible, and it generally decreased with distance from the source area. Of the 160,950 acres (64,380 ha) of ponderosa-Jeffrey type within the forest boundaries, 46,230 acres (18,492 ha) had heavy damage, 53,920 acres (21,568 ha) had moderate damage, and 60,800 acres (24,320 ha) had light or negligible damage. An estimated 1,298,000 trees were affected; of them, 82% were moderately affected, 15% were severely affected, and 3% were already dead.

The term "ponderosa-Jeffrey type" is a general term that includes a mosaic of five subtypes described by McBride on the basis of species dominance.[32] These subtypes are: ponderosa pine forest, ponderosa pine-white fir forest, ponderosa pine-Jeffrey pine forest, Jeffrey pine forest, and Jeffrey pine-white fir forest. The injury by oxidant air pollutants is most intense in the types dominated by ponderosa pine and less intense in the Jeffrey pine types. In the field plots of these various types, the average area covered by shrubs is only 3.8% in the ponderosa types, but 26% in the Jeffrey pine types.[32]

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13 Relationship between soil characteristics and microarthropod population

14 Bark beetle mortality caused by natural enemies in different forest communities

15 Effect of bark beetles on tree mortality and vigor in different forest communities

16 Relationship between soil characteristics and forest community composition and growth

17 Relationship between soil characteristics and species distribution and behavior of nonpathogenic fungi

18 Relationship between soil characteristics and species distribution and behavior of pathogens

19 Influence of forest community type on populations of natural enemies of bark beetles

20 Woodpecker distribution and density in different forest communities

21 Effect of pathogens on tree vigor and mortality in different forest communities

22 Relationship between nonpathogenic fungi and forest community composition and growth

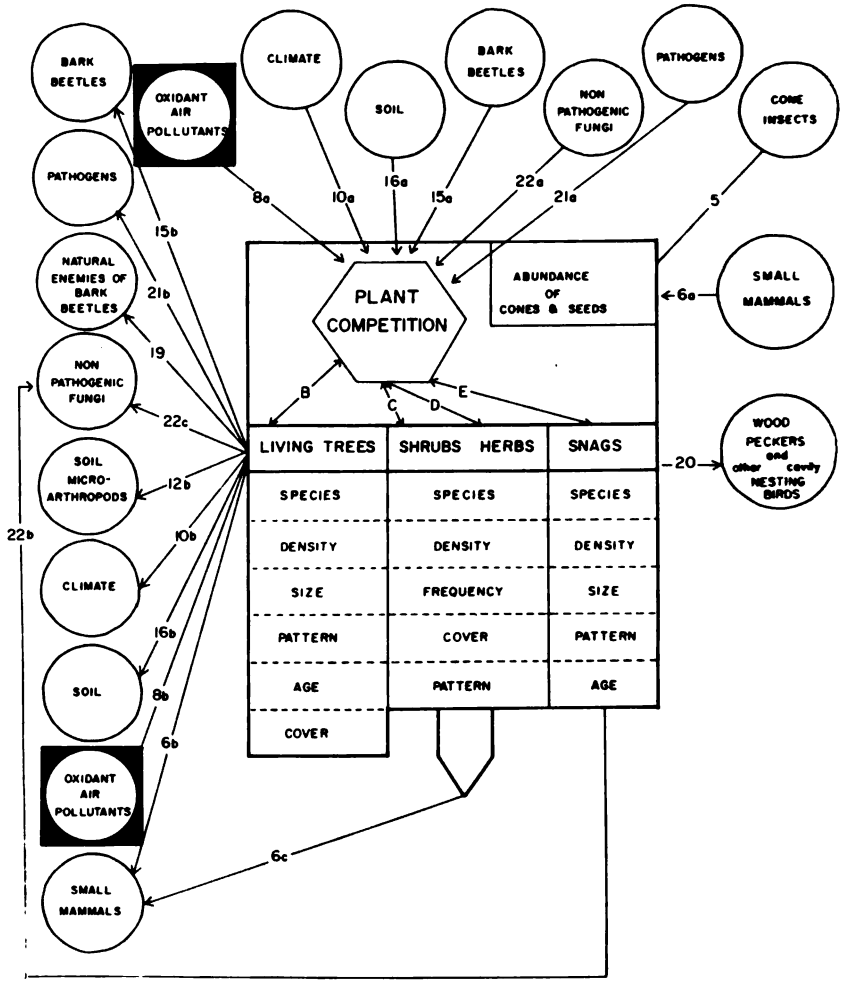

FIGURE 12-11 Stand-level interactions in a mixed-conifer forest ecosystem. a indicates direct influence of external factor on trees in stands; b and c indicate influence of stand condition on nonpathogenic fungi. Reprinted with permission from Taylor.[68] Types of interaction:

A Effect of plant competition on abundance of cone and seeds
B Effect of plant competition on characteristics of living trees
C Effect of plant competition on characteristics of shrubs
D Effect of plant competition on characteristics of herbs
E Effect of plant competition on tree mortality

5 Abundance of cones and seeds and predation by cone insects
6a Abundance of cones and small-mammal predation of cones
6b Characteristics of forest stands and small-mammal populations

Later studies[43,67,68] have expressed the amount of injury to ponderosa and Jeffrey pines and associated tree species in permanent study plots as a numerical score. The range of scores is subdivided into seven categories ranging from dead to no visible symptoms.[43]

In 1974, all tree species at 19 of these permanent study plots were scored individually by binocular inspection. The data can be obtained from conifers early in the fall, but the most important deciduous species, black oak, was evaluated during 3 days, August 28–31, to prevent confusion of oxidant-injury symptoms with natural autumn senescence of leaves. The injury to black oak as of August 31, 1974, at several representative study sites and the June–August accumulated dose at nearby monitoring stations are shown in Figure 12-13 in relation to the topographic projection of the San Bernardino Mountains. Lower scores mean greater injury. The darkened portion of the bar representing oak injury is for leaf chlorotic mottle and interveinal necrosis. A score of 8 means no injury. The remaining portion of the score is the sum of scores for leaf complement, leaf size, and twig mortality, not shown separately. These data suggest that oak shows no visible injury where the accumulated June–August dose does not exceed about 2.5×10^5 $\mu g/m^3$-h from about Snow Valley eastward.

The distribution of ponderosa and Jeffrey pines into various injury classes with respect to the distance of the study site along the gradient of oxidant dose (June–September) is illustrated above the topographic projection in Figure 12-14. It is important to realize that the 1974 distribution into injury classes is also a product of earlier years, when the

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6c Influence of shrub and herb layer vegetation on small-mammal populations
8a Smog-caused mortality and morbidity in different tree species with forest stands
8b Influence of stand conditions on concentration of oxidants
10a Influence of temperature and evaporative stress on stand structure and composition
10b Influence of stand condition on temperature and evaporative stress
12b Influence of stand condition on soil microarthropod population
15a Influence of bark-beetle-caused tree mortality on stand condition
15b Influence of stand condition on bark beetle population
16a Influence of soil moisture and temperature on stand characteristics
16b Influence of stand condition on soil characteristics
19 Influence of stand condition on population dynamics of natural enemies of bark beetles
20 Relationship between smog occurrence and woodpecker population
21a Influence of pathogen-caused mortality on stand condition
21b Influence of stand condition on pathogen population
22a Influence of mycorrhiza fungi
22b Influence of snags (and downed trees) on nonpathogenic fungi
22c Influence of stand condition on nonpathogenic fungi

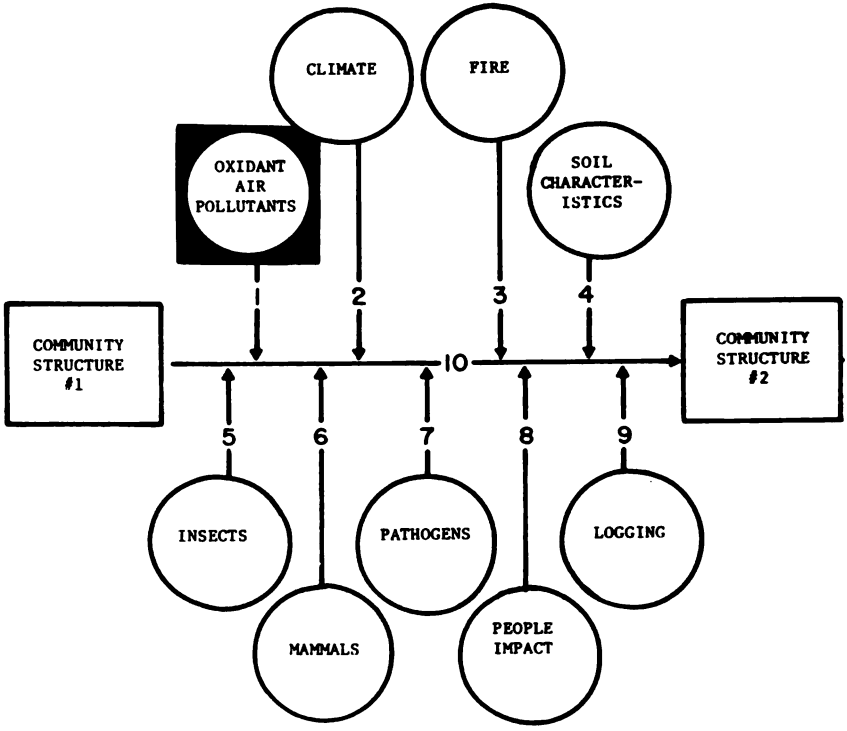

FIGURE 12-12 Community-succession interactions in a mixed-conifer forest ecosystem. Reprinted with permission from Taylor.[68] Types of interaction:

1 Oxidant modification of community structure
2 Climate modification of community structure
3 Fire modification of community structure
4 Soil characteristics and their influence on community structure
5 Insect modification of community structure
6 Mammal modification of community structure
7 Pathogen modification of community structure
8 People impact on community structure
9 Logging modification of community structure
10 Predictive capability through integration of submodels for items 1-9

oxidant concentrations were not always as high as in 1974 (see Figure 12-5). The trend toward greater numbers in the very slight (29-35) and no visible injury (36 and greater) categories is quite evident in the eastern plots receiving lower doses—e.g., Holcomb Valley (HV). The assumption has been made that ponderosa and Jeffrey pine respond similarly to oxidant. Ponderosa pine is replaced by Jeffrey pine in the natural stands

east of Camp O'ongo, and they intermix at Barton Flats (BF). The validity of this assumption can be partially verified by examining the distributions of the two species into injury classes at Barton Flats (Figure 12-14). These data indicate reasonable similarity at a common site; but the influence of other environmental variables that change continuously along the oxidant gradient—such as soil moisture availability, temperature, and humidity—must be examined more intensively to understand the degree to which they influence oxidant susceptibility (see Figures 12-8 through 12-12).

Trends in Oxidant Damage to Conifer Species in the 19 Major Study Plots from 1973 to 1974

The first evaluation of oxidant injury to all tree species in the new study plots was completed in September and October 1973. The second evaluation, in 1974, offered the first opportunity to assess trends of tree injury and mortality. In Table 12-3, the plots are listed in the order of severe injury (first) to no visible injury (last), according to the 1974 average injury score for all ponderosa or Jeffrey pine in each plot. Of the 13 plots categorized as having severe and moderate injury, all but 6 showed significantly lower scores (increased injury) in 1974 than in 1973, with a probability of 0.05. Among the remaining six plots classified as having slight, very slight, or no visible injury, there were three significant increases, one insignificant increase, and two decreases (one significant and one insignificant), all at a probability of 0.05.

The general increase in injury in the severe and moderate plots is probably related to the 1974 increase in June–September dose (Figure 12-5). Tree mortality among ponderosa and Jeffrey pines was about the same in 1973 and 1974. The largest mortality was at permanent study plots in the moderate injury category. Perhaps the populations in these plots still retain greater numbers of the more susceptible genotypes. In earlier years, tree mortality rates for ponderosa or Jeffrey pines in several stands suffering moderate to severe injury were 8% and 10%, respectively, from 1968 to 1972,[67] 8% from 1969 to 1971,[43] and 24% from 1966 to 1969.[10] The final cause of death of weakened trees is usually the pine bark beetle.[65] Mortality has not been observed in tree species other than ponderosa and Jeffrey pine.

Data for the longest observation period of tree decline extend from 1952 to 1972 in two 5-acre (2-ha) control plots in the vicinity of Barton Flats in the San Bernardino National Forest.[80] These plots are in the Jeffrey pine–white fir subtype and are now considered to be in an area of

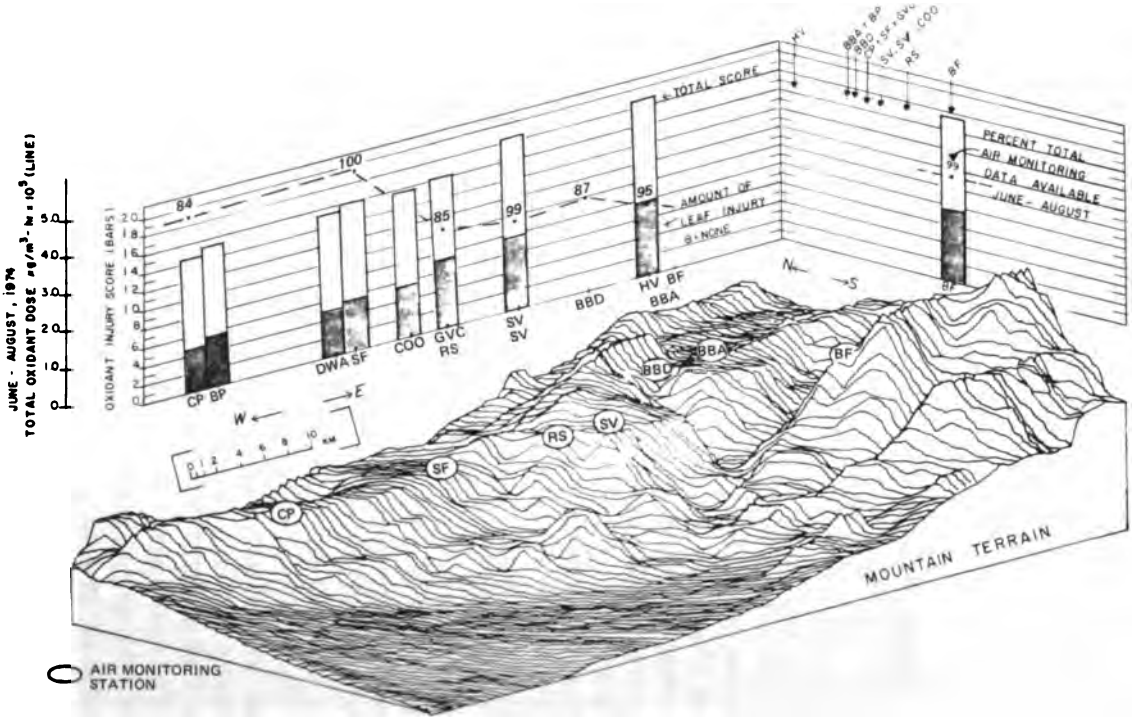

FIGURE 12-13 Topographic projection, San Bernardino Mountains, with comparison of oxidant injury to black oaks at major study sites, August 31, 1974, with accumulated total-oxidant dose for June–August measured at nearby monitoring stations. Higher score means less injury. Reprinted with permission from Kickert *et al.*[32]

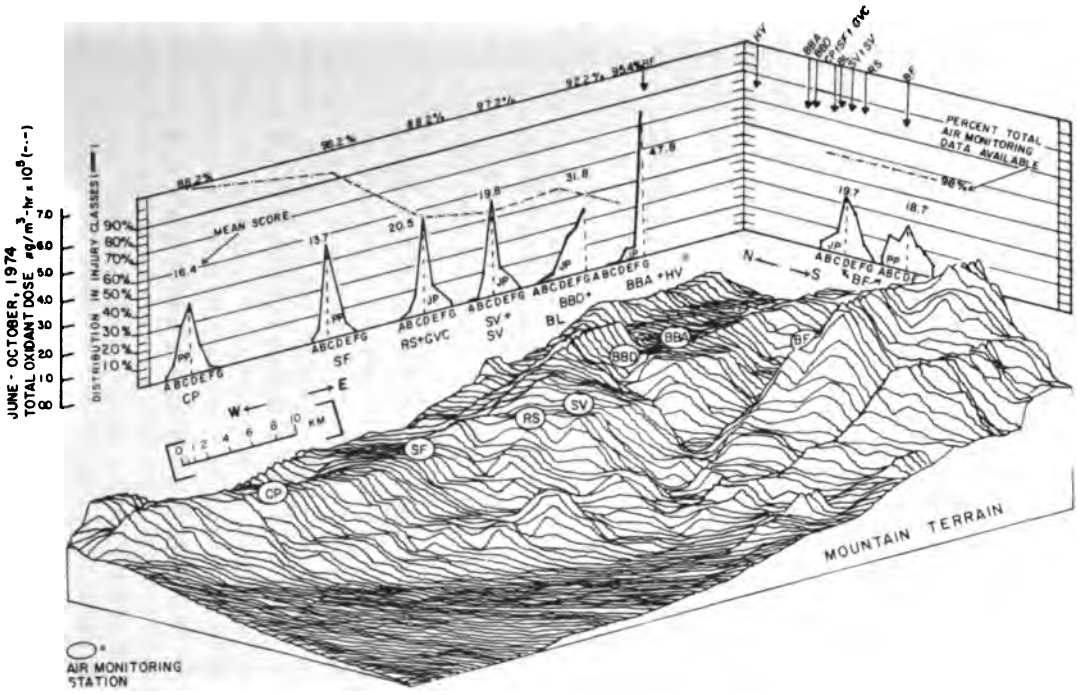

FIGURE 12-14 Topographic projection, San Bernardino Mountains, showing how ponderosa pine (PP) and Jeffrey pine (JP) in major study sites are distributed in six injury classes according to seasonal dose of total oxidant. *A*, dead, 0; *B*, very severe, 1-8; *C*, severe, 9-14; *D*, moderate, 15-21; *E*, slight, 22-28; *F*, very slight, 29-35; *G*, no visible damage, 36+. Reprinted with permission from Kickert *et al.*[32]

TABLE 12-3 Changes in Oxidant-Injury Scores and Mortality Rates of Ponderosa and Jeffrey Pines at 18 Major Study Plots, 1973-1974

Plot	Species	Tree Density,[a] 1973	Average Injury Score[b]		Mortality Rate, %		Accumulated Mortality, %	Injury Description
			1973	1974	1973	1974	1974	
Schneider Creek (SCR)	JP	28	12.4	11.7	0.0	0.0	0.0	
Camp O'ongo (COO)	PP	90	15.1	12.9*	0.0	0.0	0.0	Severe
Sky Forest (SF)	PP	144	13.3	13.7	0.8	0.8	1.6	
University Conf. Center (UCC)	PP	309	15.5	15.6	0.0	1.5	1.5	
Breezy Point (BP)	PP	236	16.3	16.0	2.7	2.7	5.5	
Camp Paivika (CP)	PP	217	17.1	16.4	0.0	3.1	3.1	
Dogwood A (DWA)	PP	168	19.9	16.5*	1.2	0.0	1.2	
Tunnel Two Ridge (TUN 2)	PP	122	19.5	16.7*	0.0	1.4	1.4	
Camp Angeles (CA)	PP	112	25.6	16.8*	0.0	1.5	1.5	Moderate
Barton Flats (BF)	PP	200	21.4	18.7*	3.7	3.7	7.4	
Barton Flats (BF)	JP	124	21.0	19.7	3.6	3.6	7.3	
Snow Valley 2 (SV 2)	JP	129	22.1	19.7*	0.0	1.0	1.0	
Green Valley Creek (GVC)	JP	43	21.8	20.5*	0.0	1.5	1.5	
Camp Oceola (CAO)	JP	192	21.7	22.8*	0.8	7.2	8.0	Slight
Bluff Lake (BL)	JP	186	29.4	31.8*	0.0	0.0	0.0	Very slight
N.E. Green Valley (NEGV)	JP	120	33.1	32.1	0.0	0.0	0.0	
Heart Bar (HB)	JP	130	44.0	39.2*	0.0	0.9	0.9	No visible symptoms
Sand Canyon (SC)	JP	56	41.3	47.3*	0.0	0.0	0.0	
Holcomb Valley (HV)	JP	193	46.4	47.7	0.0	0.0	0.0	

[a] Number of trees per hectare.
[b] Asterisk indicates difference significant at probability of 0.05 (comparisons valid only between years at a single plot).

moderate injury (see Barton Flats and Camp Oceola plots in Table 12-3). All Jeffrey pines with a diameter breast height (dbh) of 12 in. (30.5 cm) and larger were measured, and their vigor was described by judging the risk or the probability that they would be susceptible to attack and kill by bark beetles (*Dendroctonus* sp.) Risk classes 1 and 2 indicate low-risk trees that would definitely be preserved if trees were being marked for a timber sale. Classes 3 and 4 are high-risk trees that would be marked for removal in a timber sale. In Table 12-4, the changes in merchantable volume in board feet (bd ft) in all four classes are recorded for two control plots in 1952, 1963, and 1972. The increases in volume of high-risk trees since 1952 are remarkable; decreases in volume of low-risk trees and total volume in the plots are very large. The total volume decrease is related, first, to one-by-one removal of bark-beetle-killed trees inside the plots indicated for certain by the increase in snags and current stumps and, second, possibly to suppressed radial growth.

One of the objectives of the two 5-acre (2-ha) control plots was to show what would have happened in the absence of sanitation salvage logging. The distributions of trees in all categories in 1952, 1963, and 1972 (Table 12-4) suggest that by 1972 in plots 1 and 2 the sanitation salvage logging or timber sales would have removed 45% and 68% of overstory trees, respectively. The more selective tree-by-tree salvage would have removed only 8% and 10% of the overstory trees in plots 1 and 2, respectively. These results must be treated as suggestions, not firm conclusions, because of the lack of a more adequate statistical design, which should have included more plots in additional stand types and sampling of several kinds of sites at various distances from the pollution source. However, because of the suggested impact, the sanitation salvage logging practice needs careful scrutiny.

Other information is avialable from the ponderosa pine subtype that suggests that biomass production of overstory species is diminished in proportion to the oxidant dose received. Parmeter *et al.*[54] observed decreases in height growth of ponderosa pine that showed injury symptoms. Injured trees did not respond with greater growth during years with more favorable soil moisture content; uninjured trees (often side by side with injured trees) did have increased height growth in these years. McBride (in Kickert *et al.*[32]) studied two populations, each including 19 ponderosa pines dominant in their stands. One population ranged in age from 52 to 71 yr in 1972, and the other from 20 to 39 yr. The influence of tree age on ring width growth was minimized by comparing rings of equivalent age in each population. The measured rings in the older group were produced from 1910 to 1940, before the advent of "Los

TABLE 12-4 Changes of Timber Volume and Percentage of Total Jeffrey Pines in Four Insect Risk Classes at Two Control Plots Excluded from Sanitation Salvage Logging between 1952 and 1972 at Barton Flats, San Bernardino National Forest

Risk Classes	1952		1963		1972	
	Timber Volume, bd ft	Percentage of Trees	Timber Volume, bd ft	Percentage of Trees	Timber Volume, bd ft	Percentage of Trees
	Control Plot 1 (JCA Camp, Highway 38)					
Total, all classes	73,040	100	63,530	100	52,730	100
Risks 1 and 2	58,520	87	38,700	73	23,780	55
Risk 3	6,740	7	14,630	13	14,140	16
Risk 4	7,780	5	10,200	7	14,810	20
Snags and current stumps*	1	1	11	7	13	8
	Control Plot 2 (Camp Oceola Road)					
Total, all classes	120,130	100	112,660	100	112,930	100
Risks 1 and 2	110,830	93	98,080	82	45,670	32
Risk 3	5,990	3	10,170	6	37,420	30
Risk 4	3,310	2	4,410	6	29,840	28
Snags and current stumps*	3	2	13	6	18	10

* Accumulation during 10-yr period. Data obtained from the Supervisor's Office, San Bernardino National Forest.

Angeles smog," and in the younger group from 1941 to 1971, when smog was present. After the influence of precipitation on growth was evaluated, there was a difference of 0.20 cm in average annual growth attributable to oxidant air pollutant injury. In this sample, a 30-yr-old tree subjected to air pollution would be 24 ft (7.2 m) tall and 7.5 in. (19 cm) in dbh, with one 16-ft (4.8-m) log having a volume of 30 bd ft. In comparison, a 30-yr-old tree growing before the injury by oxidant air pollutants (1910–1940) would be 30 ft (9 m) tall and 12 in. (30.5 cm) in dbh and could yield a 4.8-m log with a volume of 80 bd ft. The impact of oxidant stress on forest growth is apparent without further elaboration.

The growth suppression of ponderosa pine saplings has been demonstrated by enclosing a group of 10 naturally seeded trees in the plastic-covered greenhouses used by Thompson;[71] enclosed trees were provided with activated-charcoal-filtered air (FAH) from April through October 1968–1973 by Miller and McBride (in Kickert *et al.*[32]). The controls consisted of two similar groups of trees, one in a greenhouse receiving unfiltered or ambient air (AAH) and another group not enclosed (AAO). Precipitation was rare during the summer enclosure period, and all three groups received the same amount of winter precipitation. At the end of the 1973 growing season, most of the saplings were harvested from each treatment for growth analysis. One indicator of the response to these treatments was the length of annual growth of terminal shoots and first-order branches in the upper half of the saplings (Figure 12-15). After an initial lag in 1969 and 1970, the growth of shoots and branches on the FAH trees increased dramatically. The retention of annual needle whorls was remarkably different by 1973; needles in internodes older than 1972 were completely absent from the AAH and AAO trees. The decrease in needle biomass on the AAH and AAO trees for the 1973 whorl was 32% and 37%, compared with that on FAH trees (Figure 12-16).

Needle length and litter production furnish another important measure of biomass decrease. In field studies, it was found that the average amount of litter production increased with decreasing ozone injury of selected ponderosa and Jeffrey pines ($r = 0.55$) and that the average mass of needles per fascicle increased with decreasing ozone injury ($r = 0.96$).[68]

Early studies[65] suggested that injury to ponderosa pine was similar in all size classes, but Cobb and Stark[10] later reported higher mortality rates in understory ponderosa pines (9–12 in., or 22.9–30.5 cm, in diameter) than in larger size classes. The probable effect of tree mortality on stand composition can be anticipated to some extent from an example of the present species and size-class composition, as shown in Table 12-5.[43] In this severely damaged stand in the ponderosa pine–white fir subtype, nearly 50% of the overstory and about 22% of the understory is ponderosa pine.

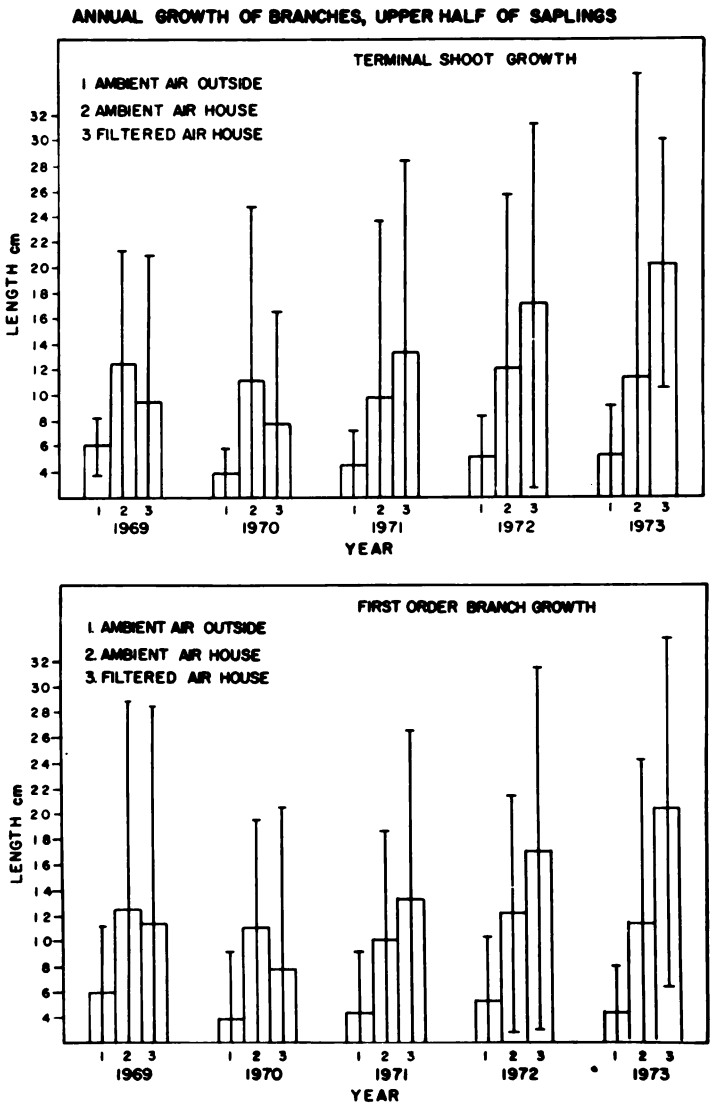

FIGURE 12-15 Annual growth of the terminal shoot (upper) and first-order branches (lower) in upper half of ponderosa pine saplings maintained in filtered or unfiltered (ambient) air greenhouses, and an outside ambient-air treatment from 1968 through 1973. Reprinted with permission from Kickert *et al.*[32]

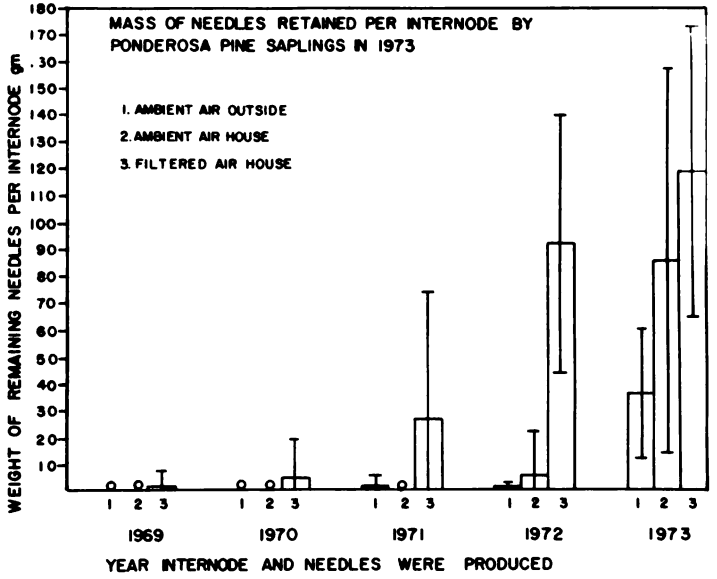

FIGURE 12-16 Average weight of all needle fascicles retained in 1973 per internode in filtered or unfiltered (ambient) air greenhouses, and an outside ambient-air treatment after treatment since 1968. Reprinted with permission from Kickert *et al.*[32]

With all size classes considered, 16.1% had slight, 33.3% moderate, 31.2% severe, and 19.4% very severe injury. White fir and incense cedar are well established in the understory, but their poorer survivorship to 12.00-23.99 in. (30.5-60.9 cm) in dbh compared with ponderosa pine suggests that other mortality factors have acted heavily on them in the past. Incense cedar and sugar pine are the most tolerant to oxidant, but sugar pine is present in very low numbers. The accelerated mortality of ponderosa pines has particular significance in this ecosystem, because it is the dominant member of the climax community.

The direct effects of ozone on plant species constituting the shrub layer in the conifer forest are not yet sufficiently understood to permit any conclusion to be drawn. In many sites where the conifer overstory is well developed, the shrub species are excluded completely. In more open pine stands, some shrubs are very common, such as skunk bush (*Amorpha californica*) and white horn (*Ceanothus cordulatus*). Of these two species, only skunk bush shows highly visible chlorotic mottle of leaflets and premature defoliation where ozone dosages are high. A shrub species common to the lower chaparral zone, squaw bush (*Rhus trilobata*), is even more susceptible to ozone injury.[57] In the San Bernardino Moun-

TABLE 12-5 Tree Species and Size Composition on a Study Site Affected by Oxidant Air Pollution*

Tree Size Class	Number per acre			
	Ponderosa Pine	Incense Cedar	White Fir	Sugar Pine
Understory				
Seedlings (up to 3.00 ft tall)	1,507	2,381	1,043	302
Saplings (more than 3.00 ft tall, less than 4.00 in. dbh)	33	33	57	10
Poles (4.00–11.99 in. dbh)	21	12	38	3
Percent	22.2	48.6	22.8	6.3
Overstory				
Standard (12.00–23.99 in. dbh)	18	9	8	3
Veteran (24 in. dbh and larger)	12	5	4	2
Percent	49.6	22.7	19.7	8.0

* Adapted from Miller.[43] Trees from 575-acre study area, San Bernardino National Forest, Calif.

tains, 166 herb-layer species have been identified as common to the conifer forest.[68] The phenology of these species is being studied so that growth and flowering can be correlated with soil moisture availability and seasonal changes of ozone dose. Initial field observations suggest that at least 10 species are obviously injured by total oxidant in areas receiving high dosages. These areas have been subject to exposure for at least 20 yr, so some species or subspecies may have been completely eliminated. Dunn[18] reported that smog may have acted as a selective agent to remove oxidant-sensitive subspecies of *Lupinus bicolor* from the Los Angeles area. Furthermore, *L. latifolius* appeared to have floral ontogeny inhibited without damage to leaves, and *L. densiflorus* was severely damaged by ambient total oxidant at Claremont, California.

Price [56] selected six important grass species found in either the Gambel oak or quaking aspen communities in the Wasatch Mountains of Utah. The selected species were fumigated under greenhouse conditions with ozone varying from 0.15 to 0.30 ppm during each experiment. These treatments caused a 53% reduction in top-growth biomass of the six species. The initiation of reproductive structures of all species was inhibited, and the reproductive potential of *Bromus tectorum* and *B. carinatus* declined to zero. These results suggest that the species composition of affected natural communities could be altered. In other studies, 17 representative species from the quaking aspen community were fumi-

gated with ozone 3 h each day, 5 days/week, with ozone at 0.30, 0.15, and 0.05–0.07 ppm in the first two cases and with ambient total oxidant in the last case. Experiments extended through three seasons. At concentrations of 0.15 ppm or below, growth reduction and stimulation were both observed in different species; growth was reduced in all but two species at 0.30 ppm. Seed production was reduced in two species at 0.15 ppm and in three species at 0.30 ppm, of the total of six species that produced fruits or seeds.[25]

In a third series of experiments, Treshow and Stewart[78] fumigated native plants growing naturally under field conditions. The threshold ozone concentrations required to injure important species in the grassland-oak and aspen-conifer communities during 2-h exposures were determined (Table 12-6). Three important species—*Bromus tectorum*, *Quercus gambelli*, and *Populus tremuloides*—were injured by a single 2-h exposure to ozone at 0.15, 0.25, and 0.15 ppm, respectively; over half the remaining species showed visible injury after exposure to 0.30 ppm or less. The implications for possible imbalances in community stability are readily apparent if these plant communities receive dosages similar to those typical in the San Bernardino Mountains.

Yonkers *et al.*[84] have tested the ozone susceptibility of 15 species of annuals common to the Mojave Desert just north and east of the Los Angeles basin (and San Bernardino Mountains). Compared with the susceptible pinto bean plants included in the experiment with ozone at 0.35 ppm, *Plantago* sp., *Cercidium* sp., and *Prosopsis* sp. were also sensitive. Further interpretation of these results is complicated by the influences of plant age and pre-exposure conditions.

Dose–Injury Observations with Ambient Oxidant and Native Ponderosa Pine Saplings

During 1969, 1970, and 1971, 10 sapling-size ponderosa pines that received the ambient-air-outside (AAO) treatment for comparison with trees enclosed in filtered- or ambient-air greenhouses were observed to determine the rate of current-year needle growth and appearance of oxidant-injury symptoms on both current and 1-yr-old needles.[32] Forty new needles on each tree were measured monthly (Figure 12-17, left); growth leveled off in early September. Current-year and 1-yr-old needles were inspected monthly for amount of chlorotic mottle, necrosis, and abscission. Each symptom was given a descriptive rating: O, none; 1, very slight; 2, slight; 3, moderate; and 4, severe. In Figure 12-17, an average of the 3 yr shows that, by Julian day 250 (September 7), the current-year needles had a visible-injury score of 2.2, indicating slight

TABLE 12-6 Injury Thresholds for Two-Hour Exposures to Ozone

Species	Injury threshold (pphm ozone)	Species	Injury threshold (pphm ozone)
Grassland–Oak Community Species		Perennial Forbs	
		Allium acuminatum Hook.	25
Trees & Shrubs		*Angelica pinnata* S.Wats.	under 25
Acer grandidentatum Nutt.	over 40	*Aster engelmanni* (Eat.) A.Gray	15
Acer negundo L.	over 25	*Carex siccata* Dewey	30
Artemesia tridentata Nutt.	40	*Cichorium intybus* L.	25
Mahonia repens G.Don	over 40	*Cirsium arvense* (L.) Scop.	under 40
Potentilla fruticosa L.	30	*Epilobium angustifolium* L.	30
Quercus gambelii Nutt.	25	*Epilobium watsoni* Barbey	30
Toxicodendron radicans (L.) Kuntze	over 30	*Eriogonum heracleoides* Nutt.	30
		Fragaria ovalis (Lehm.) Rydb.	30
Perennial Forbs		*Gentiana amarella* L.	over 15
Achillea millefolium L.	over 30	*Geranium fremontii* Torr.	under 25
Ambrosia psilostachya DC.	over 40	*Geranium richardsonii* Fisch. & Traut.	15
Calochortus nuttallii Torr.	over 40	*Juncus* sp.	over 25
Cirsium arvense (L.) Scop.	40	*Lathyrus lanzwertii* Kell.	over 25
Conium maculatum L.	over 25	*Lathyrus pauciflorus* Fern.	25
Hedysarum boreale Nutt.	15	*Mertensia arizonica* Greene	30
Helianthus annus L.	over 30	*Mimulus guttatus* DC.	over 25
Medicago sativa L.	25	*Mimulus moschatus* Dougl.	under 40
Rumex crispus L.	25	*Mitella stenopetala* Piper	over 30
Urtica gracilis Ait.	30	*Osmorhiza occidentalis* Torr.	25
Vicia americana Muhl.	over 40	*Phacelia heterophylla* Pursh	under 25
		Polemonium foliosissimum A.Gray	30
Grasses		*Rudbeckia occidentalis* Nutt.	30
Bromus brizaeformis Fisch. & Mey.	30	*Saxifraga arguta* D.Don	under 30
Bromus tectorum L.	15	*Senecio serra* Hook.	15
Poa pratensis L.	25	*Taraxacum officinale* Wiggers	over 25
		Thalictrum fendleri Engelm.	over 25
Aspen and Conifer Community Species		*Veronica anagallis-aquatica* L.	25
		Vicia americana Muhl.	over 25
Trees & Shrubs		*Viola adunca* Sm.	over 30
Abies concolor (Gord. & Glend.) Lindl.	25		
Amelanchier alnifolia Nutt.	20	Annual Forbs	
Pachystima myrsinites (Pursh) Raf.	over 30	*Chenopodium fremontii* Wats.	under 25
Populus tremuloides Michx.	15	*Collomia linearis* Nutt.	under 25
Ribes hudsonianum Richards.	30	*Descurainia californica* (Gray) O.E.Schulz	25
Rosa woodsii Lindl.	over 30	*Galium bifolium* Wats.	over 30
Sambucus melanocarpa A.Gray	over 25	*Gayophytum racemosum* T. & G.	30
Symphoricarpos vaccinioides Rydb.	30	*Polygonum douglasii* Greene	over 25
Perennial Forbs		Grasses	
Actaea arguta Nutt.	25	*Agropyron caninum* (L.) Beauv.	over 25
Agastache urticifolia (Benth.) Kuntze	20	*Bromus carinatus* Hook. & Arn.	under 25

[a] Reprinted with permission from Treshow and Stewart.[78]

chlorotic mottle (*signifying severe injury to the trees*); there was usually no necrosis and almost always no abscission of current-year needles. Symptoms developed more rapidly on 1-yr-old needles; the combined score of the current-year and 1-yr-old needles was about 9.

The accumulated ozone dose since June 1, which is associated with the current-year and 1-yr-old needle injury expressed on the left of Figure 12-17, can be roughly estimated by transferring the injury score to the right. For example, the current-year needle score of 2.2 on September 7 is associated with a total-oxidant dose of 2.75×10^5 $\mu g/m^3$-h. These results assume that all the air monitoring data were available, but

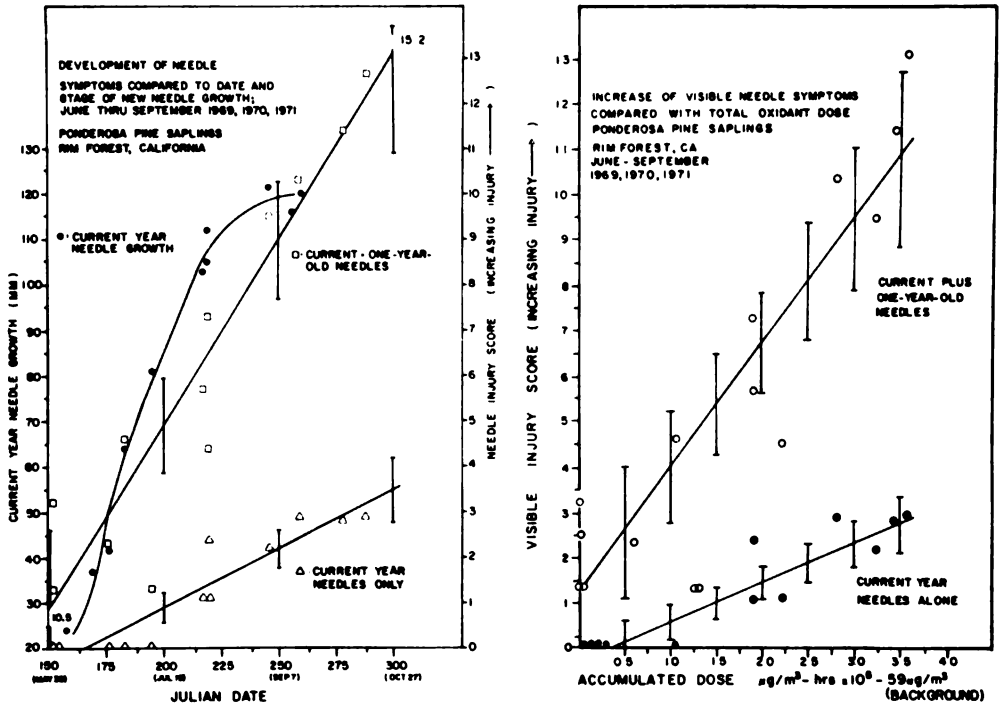

FIGURE 12-17 Development of oxidant injury symptoms on current and current-plus-1-yr-old needles of ponderosa pine saplings, in relation to stage of current-year needle growth and time during the summer season (left) and in relation to total dose of oxidant (right). Reprinted with permission from Kickert *et al.*[32]

on the average about 90% were available. The dose-injury curve is apparently not linear as assumed, and interpretation is further confounded by the random occurrence of injurious doses in the field. Additional observations of ponderosa and Jeffrey pine are required to define more completely the ozone or total-oxidant dose response. The development of a model relating injury to dose and important controlling environmental variables is a high-priority need for understanding chronic-exposure effects.

EFFECTS ON REPRODUCTION

The effect of ozone injury on herbaceous plant reproduction has been mentioned earlier in this chapter and in Chapter 11. Seed production by annuals is influenced mainly by the environmental conditions of the current year, but perennial woody plants—particularly conifers—are erratic seed-producers. Intrinsic factors affecting cone production include age and vigor; seasonal temperature and soil moisture are important environmental factors.[23]

The effects of sulfur dioxide on cone production have been described by Scheffer and Hedgcock[60] and Pelz.[55] Generally, the decrease in tree vigor caused by the pollutant may eliminate or lower the frequency of cone production and diminish the size, weight, and germination of seed. The effects of chronic ozone injury on conifer seed production may be similar, in that tree vigor is drastically reduced. The effects of chronic ozone injury on ponderosa and Jeffrey pine seed production are under investigation by Luck (in Kickert *et al.*[32]).

Seeds from individual species may constitute the bulk of the diet of small vertebrates, such as the deer mouse (*Peromyscus* sp.) and the western gray squirrel (*Sciurus griseus anthonyi*). During periods of low seed production due to diminished tree vigor, squirrels converge on the few remaining vigorous ponderosa pines and consume about half the seed crop before it matures and reaches the ground. In the areas severely affected by ozone, squirrels return to the same trees year after year. After the seed reaches the ground, other small vertebrates, such as mice, seek it out. The habit of preferential seed use by small vertebrates may be a stress acting additively with ozone injury to decrease seriously the regeneration potential of ponderosa pine.

Indirect Effects

Most indirect effects of oxidant air pollutants on primary production and reproduction would be mediated through changes in the physical

environment resulting from disruption of stand structure. For example, Hursh[28] described three microclimates in the area surrounding the ore smelter at Copper Basin, Tennessee. These zones were distinguished by changes in air and soil temperatures, winds, evaporation, air moisture, and rainfall. Because sulfur dioxide was the pollutant in this case, there were also changes in soil pH and sulfur content, which would not accompany oxidant air pollution damage. Soil erosion increased dramatically.

Changes in such physical factors as light, temperature (particularly maximums and minimums), relative humidity, and wind speed in forest communities subject to structural alteration by mortality of susceptible species could change the suitability of some sites for growth, reproduction, and re-establishment of survivor species. We can only speculate on some of the possible secondary effects until more data are gathered.[43]

Wildfire is a very important factor in western forest ecosystems. In the San Bernardino Mountains, the fire frequencies were determined by McBride and Laven[40] in two stand types before and after 1893, when the area was first set aside as a forest preserve and fire protection began. Before 1893, the average interval between fires in ponderosa stands was 12 yr; after 1893, it was 24 yr. The comparable numbers for Jeffrey pine stands were 16 and 38 yr. The buildup of heavy fuels due to ozone-caused mortality and fire protection results in hotter fires, and the thinning of the tree canopy results in increased rates of fire spread.[14] Hotter fires decrease tree survival. Moisture interception by condensation in living tree crowns would decrease as the stands became thinner, thus causing some sites to be drier.[33]

EFFECTS ON CONSUMER POPULATIONS

Vertebrate Populations

The effects of oxidant air pollutants on vertebrates can be segregated into direct and indirect categories. Direct effects are clinical and pathologic alterations of tissues that result from exposure to ambient air. Indirect effects result from alterations in numbers or distribution of the plant and animal populations exposed to ambient air. For example, if air pollution eliminates or thins numbers of a susceptible plant species, the food chain of the consumers that feed on it may break down. The result could be a simpler and less stable ecosystem, with fewer plants and animals in species and numbers.

The clinical and pathologic effects of oxidant air pollutants on domesticated vertebrates have received little study in the laboratory. We have

found no major references to studies of these effects on free-ranging native species. It is therefore necessary to extrapolate from laboratory results to probable effects on wildlife in the field and forest.

Chapter 8 indicates that oxidant air pollutants may adversely affect the olfactory and sight senses, degree of activity, general health and vigor, reproductive rate, heart and kidney function, protein synthesis, respiratory function, and disease resistance in domestic vertebrates under laboratory conditions. Many of these adverse physical responses develop at or near the concentrations of ozone currently experienced daily in ambient air in parts of the San Bernardino Mountains.

Although it is risky to extrapolate from domestic species in a laboratory to free-ranging species in the forest, it appears likely that some wild vertebrates in the San Bernardino Mountains could be affected adversely by their prolonged exposure to current concentrations of ozone. Nitrogen dioxide, PAN, ozone, and environmental factors may act additively or synergistically to increase the overall stress on the individual. Potentially, the direct effects of oxidant air pollution on wild vertebrates within a forest system could be great. A reduction in visual or olfactory acuity or other loss of health or condition could be a serious handicap for a predator whose survival depends on overcoming prey, and equally serious for the prey that survives by staying one jump ahead of a predator.

In the San Bernardino Mountains, our goals have been to describe the terrestrial vertebrate community within this mixed-conifer forest, particularly in relation to ponderosa and Jeffrey pine stands, and to determine the effects of oxidant air pollutants on this community. The possible interactions of vertebrates in this system are shown in Figures 12-8 through 12-12.

The most abundant species of small mammals were selected to exemplify vertebrate interactions, because of the importance of this group as seed-eaters. The results from trapping of abundant species show that the deer mouse (*Peromyscus* spp.) is the most numerous. Chipmunks (*Eutamias* spp.), the golden-mantled ground squirrel (*Callospermophilus lateralis*), the dusky-footed woodrat (*Neotoma fuscipes*), the meadow mouse (*Microtus californicus*), and the harvest mouse (*Reithrodontomys megalotus*) are the other common small mammals.[34] Numbers have fluctuated widely from year to year and from plot to plot, as is characteristic of small-mammal populations. Deer mouse numbers fluctuate the most. Preliminary analysis indicates that the same species of small mammals are present in this forest as were reported from similar trappings 70 yr ago.[34] Thus, it appears that oxidant air pollution has not yet resulted in a reduction in the diversity of this component of the vertebrate fauna. Population numbers of the com-

mon small-mammal species do, however, appear to be low, in comparison with those in other similar forest regions.[32] This is particularly true for the study plots in areas of heavy oxidant air pollution. We have not yet determined whether these low population densities of small mammals are directly or indirectly related in any measure to the oxidant air pollution. These differences may result from variations in other aspects of habitat quality. For example, the study plots in the areas of heavy oxidant air pollution are dominated by ponderosa pine and have sparse shrub cover, whereas the study plots in areas of light oxidant air pollution are dominated by Jeffrey pine and have much more shrub cover.[32]

When changes occur in one part of an ecosystem, the intimate nature of the interrelationships results in changes in many other parts. Any factor that causes change in one component of a system potentially affects all subsystems of that ecosystem. The most important indirect effects of oxidant air pollutants on vertebrates are those resulting in changes in the habitat. Foremost among these effects are those on the vegetation and the successional patterns of the plant community. Because of the high degree of interrelationship and interaction between the vegetation, the fauna, and the inorganic matrix of an ecosystem, effects of air pollution on the vegetation potentially can result in changes throughout the ecosystem. Damage to vegetation is probably the most important effect of chronic, low-concentration air pollution on wildlife. Ponderosa pine, Jeffrey pine, and black oak are all susceptible to damage, and these are the most important trees within the forest as providers of food and habitat for wildlife. A similar selectivity by species doubtless occurs within the shrub and herb layers of the vegetation. The long-term effects will be reduced production of fruits and seeds and elimination of the sensitive plant species and, therefore, reduction in the diversity of the vegetation. In turn, this will lead to a reduction in abundance and diversity of the vertebrate fauna.

Likewise, Woodwell's prediction[83] of enhancement of the activity of insect pests and some disease agents (which has been demonstrated in the San Bernardino Mountains forest) could lead to an increase in vertebrate species that feed on invertebrates or utilize dead plants for cover. Birds would be the most likely to increase and, to a lesser extent, such small mammals as deer mice, which are partially insectivorous.

Fruit and seeds make up the largest part of the diet of most of the common small mammals on our study sites, particularly the deer mouse, harvest mouse, chipmunk, ground squirrel, and western gray squirrel (*Sciurus griseus anthonyi*). The gray squirrel is an excellent example of the interactions within this forest and of the potential effects of oxidant air pollution. It is abundant throughout the mixed-conifer type, depend-

ing specifically on the pines and oaks for the majority of its food, cover, and nest sites. This squirrel eats or stores a major portion of the pine and oak seed crops each year. On some yellow pine trees on our study plots, gray squirrels cut more than 2,000 cones per tree.

Thus, tree squirrels are a major source of loss of seeds of ponderosa, Jeffrey, and sugar pine and of black oak acorns. Vertebrates, then, can have a major effect on the reproduction of these species, particularly because the gray squirrel is only one of numerous species in this forest that feed on conifer seeds and acorns.

An alteration of the balance between pine and oak, a change in squirrel populations, or a reduction in any of the mast crops brought about by oxidant air pollution obviously could have a major impact on this forest, particularly on tree reproduction and successional patterns. Reduction in conifer seed crops as a result of air pollution has been suggested in other forests, where cone production was lowered and size, weight, and germination ability of the seeds were reduced.[55,60]

Another way that oxidant air pollution could affect this subsystem is through an alteration in forest moisture. Elimination of vegetation cover allows the exposed soil to dry more rapidly, which would affect soil-burrowing and soil-inhabiting vertebrates. The lower moisture content may reduce or inhibit fruiting-body formation of fleshy fungi. These fleshy fungi are an important food source for tree squirrels, making up a third or more of their diet in some seasons. A reduction in this food source doubtless would result in an even greater utilization of conifer seeds and acorns, reducing further the reproductive capabilities of these trees and eventually limiting future food supplies for the squirrel population.

In summary, subtle and simple initial changes may radiate and magnify throughout all trophic levels of the ecosystem. Restoration of the system may be impossible.

Macroarthropods

Laboratory studies have indicated that ozone at 196 $\mu g/m^3$ (0.10 ppm) was lethal to adult houseflies (*Musca domestica* L.) and caused them to lay fewer eggs.[2] Two cockroach species (*Paraplaneta americana* L. and *Nauphoeta cinerea* Oliver) and the red fire ant (*Solenopsis invicta* Buren) were exposed to ozone at 588 $\mu g/m^3$ (0.30 ppm) for up to 10 days. There was no unusual mortality or evidence of direct injury to individual insects. The fire ant workers were stimulated to migrate inside their nest initially, but further observations indicated no disruption of social behavior.[37] These reports do not suggest that free-ranging insects would be directly affected by ambient concentrations of ozone in natural ecosystems or agroecosystems.

The indirect effects of ozone through modification of the availability of food for insects, particularly in a conifer-forest ecosystem, have received some investigation.[64,65] The weakening of ponderosa pines by chronic exposure to ozone makes them more vulnerable to successful infestation by pine bark beetles (*Dendroctonus brevicomis* and *D. ponderosae*). Figure 12-18 shows a positive relationship between degree of tree injury and frequency of bark-beetle infestation and the relative frequency of attack by the two species of bark beetles.[64] The relationship (see Figures 12-8 through 12-12) between tree health and brood productivity and the population dynamics of bark beetles and their insect associates in infested trees are being investigated.[32,68] Pine bark beetles have been a constant threat to ponderosa pines in the San Bernardino Mountains for many years, since before the inception of oxidant air pollution injury.[66] Bark beetles are a key element responsible for accelerating the modification of stand structure.

Plant Parasites and Symbionts

PARASITIC PHANEROGAMS

Both true mistletoe and dwarf mistletoe are common parasites of forest tree species. The true mistletoes (*Phoradendron* spp.) occur commonly on

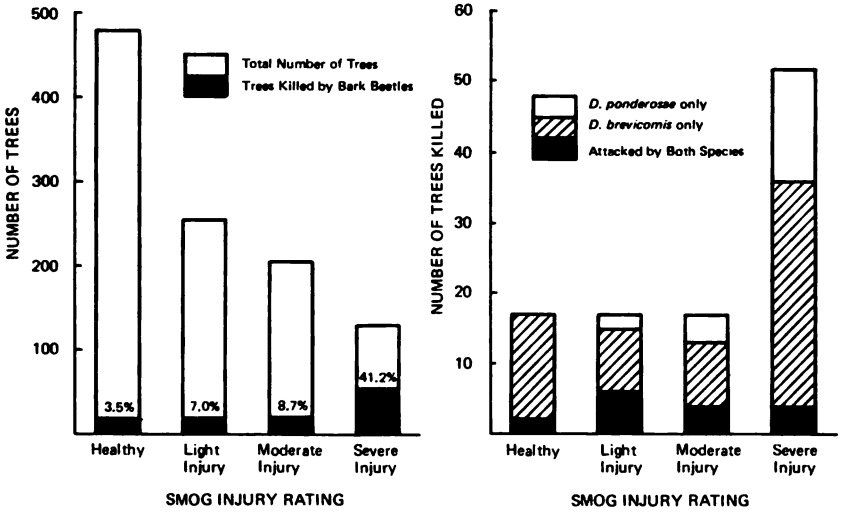

FIGURE 12-18 Relationship between degree of oxidant injury to ponderosa pines and bark-beetle attack (left) and numbers of trees killed by western pine beetle, mountain pine beetle, and the two species together (right). Reprinted with permission from Stark and Cobb.[64]

California black oak and white fir and less often on incense cedar in the San Bernardino Mountains. No direct effects of oxidants have been noted on the mistletoe plant itself under field conditions. The true mistletoe obtains mainly water from its host and would be indirectly affected by debilitation of the host tree. The dwarf mistletoes (*Arceuthobium* spp.) are common on ponderosa, Jeffrey, and sugar pines in the San Bernardino National Forest. They depend on their host for both water and carbohydrates. Heavily infected or "broomed" branches on ponderosa or Jeffrey pines severely injured by ozone often have more annual needle whorls retained than do uninfected branches on the remainder of the tree. The needles are also greener. It can be hypothesized that the infected branch is a carbohydrate sink where a pooling of carbohydrates occurs; higher carbohydrate concentrations may be instrumental in either preventing or helping to repair ozone injury to needles on the broomed branches. In the long term, stresses from mistletoe and ozone are probably additive and hasten tree death.

PARASITIC FUNGI

Fungi that infect above-ground plant parts causing leaf or stem diseases may be directly affected by ozone, as discussed in Chapter 11. Ozone injury to needles of eastern white pine increased infection by *Lophodermium pinastri* and *Pullularia pullulans*.[12] These results suggest that leaf tissue of many species may be made susceptible to fungi that are normally saprophytes but may be low-grade parasites when circumstances permit.

In the San Bernardino National Forest, the needle- and twig-infecting fungus *Elytroderma deformans* is a common disease of ponderosa and Jeffrey pines. The additive stresses of ozone and fungus on the host may hasten tree decline in severe cases. In some situations, it appears that infected or broomed twigs may die sooner than uninfected twigs at the same position in the tree. Observations are continuing.[32]

Root-infecting fungi, such as *Armillaria mellea* and *Fomes annosus*, are generally more virulent pathogens when they encounter trees already weakened by other stresses. This observation has been made mostly in Europe, where sulfur dioxide was the principal pollutant; the spread and virulence of *F. annosus* across the gradient of decreasing oxidant dosage are being studied in the San Bernardino Mountains.[32]

Significant increases have been reported in length of root tissue colonized by *F. annosus* when artificially inoculated ponderosa and Jeffrey pine seedlings were fumigated with ozone at 431 $\mu g/m^3$ (0.22 ppm) and 888 $\mu g/m^3$ (0.45 ppm) 12 h/day for 58 and 87 days.[32]

Beneficial mycorrhizal fungi infect the small feeder roots of trees and other plants. The resulting relationship is symbiotic and involves an intimate exchange of minerals and essential metabolites. The host tree benefits through increased efficiency of nutrient uptake from the soil. Any interruption or imbalance of the exchange of materials between the host root tissue and the fungous mantle surrounding it can have deleterious effects on the fungus and the host. Such stresses as air pollution injury to the host undoubtedly disrupt this balance.[25] The feeder rootlet system of ponderosa pines in the San Bernardino Mountains and those of eastern white pine have shown remarkable deterioration involving diminished numbers of mycorrhizal rootlets, which are replaced by saprophytic fungi that decay smaller rootlets.[54]

EFFECTS ON DECOMPOSERS

Although some of the solar energy fixed by producing plants is released by the respiration of these plants and of animals, much of it is stored in dead organic matter until released by decomposer organisms at rates that vary greatly with place, season, and kind of organic matter. Generally, one-third or more of the energy and carbon annually fixed in forests is contributed to the forest floor as litter, mostly leaves.[53] Because litter is generally related to the quantity of photosynthetic tissue in the ecosystem, it is a useful index of ecosystem productivity.

One of the predicted effects of pollutants on ecosystems suggested by Woodwell[83] is a reduction in the standing crop of organic matter, which would lead to a reduction in nutrient elements held within the living system. The evidence discussed earlier definitely shows that primary production is much lower in an ozone-stressed conifer-forest ecosystem. This result would be anticipated in all similarly stressed natural ecosystems or agroecosystems.

The reservoir of energy and mineral nutrients represented by litter is a very important resource in natural ecosystems with closed nutrient cycles. The growth of new green plant tissue depends on the slow release of nutrients by decomposer organisms. In agroecosystems geared for high production, litter is often removed or burned, and fertilizer is added to the soil; the nutrient cycle is open and subsidized.

In a conifer forest, litter production and decomposition release about 80% of the total minerals in the biomass of the stand; the remainder is retained in the living parts of the tree.[42] Standing dead material is not considered litter.

In terrestrial ecosystems, most decomposers occupy the mantle of

litter or the surface layers of the soil, where they supply the necessary recycling mechanisms to convert dead plant or animal material into humus and eventually into minerals, gases, and water. Small animals, arthropods, fungi, and bacteria exist as a complex in intricate food chains in which they feed not only on dead material, but also on one another, ultimately releasing the mineral nutrients needed by the producer populations. Without the decomposers, some essential elements—such as calcium, phosphorus, and magnesium—would concentrate in the litter until the supply in the soil was depleted; growth of green plants would then be seriously limited.

Direct Effects

The decomposition of tree leaves is not entirely confined to the litter layer on the forest floor. Leaves and needles are invaded by bacteria and fungi even as they grow; these microorganisms may be either pathogens or saprophytes.[29]

The first possible interaction between ozone and decomposer organisms might be with the bacteria and fungi that occupy the surface of the living green leaf. The direct effects of ozone on microorganisms are discussed in Chapter 11, but very little conclusive information is available that can be applied to natural conditions.

It is not known whether ozone, PAN, or other oxidants could have any direct influence on decomposer organisms in the litter layer; however, there does appear to be a rapid flux of ozone to soil surfaces. The ozone flux to some kinds of surfaces constituting ecosystems—e.g., vegetation, soil, and water—has been determined by Aldaz;[1] he expressed the flux as molecules per square centimeter per second times 10^{11}. The relative fluxes into different surfaces, assuming an ozone concentration of 40 $\mu g/m^3$, were: fresh water, 0.5; snow, 0.9; grass, 1.1; sand or dry grass, 5; and juniper bush, 10. Furthermore, it was found that bare soil destroyed about 75% more ozone when dry than when moist. The determination of ozone flux to surfaces may be a far more realistic measure of dose to living organisms than atmospheric concentration of ozone, according to Munn.[47]

Indirect Effects

The concentration of plant nutrients in litter influences both the rate of decomposition and the amount of nutrients released after decomposition. Ozone-injured foliage may be deficient in inorganic nutrients, because of the concomitant decay of the root systems of chronically injured trees.[54]

Coniferous leaf litter is more resistant to decomposition than broad-leaved litter.[42] Both low inorganic nutrient status and the presence of organic compounds that are toxic to microorganisms may account for slower decay. Ozone injury to needles or broad leaves might result in an accumulation of phenolic compounds that are toxic to bacteria and fungi. The rate of litter decomposition might be decreased. A high carbon:nitrogen ratio related to nitrogen deficiency may also slow decomposition.

Katz and Lieth[31] have categorized the roles of both microflora and microfauna groups in litter decomposition. They have discussed the successional trends of microflora and microfauna populations during the warm season. A typical problem emerges when population sampling is attempted: "a consistent feature of communities is that *they contain a comparatively large number of species that are rare at any given locus in time and space.*"[49] The task of identifying interactions (see Figures 12-8 through 12-12) of decomposer populations with pollutants, particularly oxidants, is formidable.

In the San Bernardino Mountains, studies are going on to describe the effects of oxidant injury to ponderosa and Jeffrey pines on the microarthropods and fungi of the litter layer under trees with various degrees of injury. Initial observations suggested lower population densities of microarthropods in the classes Insecta, Arachnida, and Myriapoda under some severely injured trees.[32]

A coincident effect on decomposers may be from the accumulation of heavy metals, such as lead, which is entrained in the photochemical-oxidant complex. In Sweden, Ruhling and Tyler[59] have preliminary evidence that litter decomposition rate in a spruce forest was limited by increased concentration of heavy-metal ions, but only during times of the year when water and temperature were not limiting factors.

The difficulties of understanding population changes of litter-decomposing microflora and microfauna as an index of pollutant effects suggest that a more simple approach should be used at the outset. The least sophisticated expression of accepted procedure[51] is:

$$\Delta x/\Delta t = \text{Income for interval} - \text{Loss for interval},$$

where Δx is the change in amount of litter over the interval t. This expression describes energy storage in litter and indicates the balance between producers and decomposers. Typical annual litter production, steady-state accumulation on the forest floor, and associated decomposition rate factors have been estimated for several locations and types of forest litter.[51] Comparisons of litter decomposition rate factors under similar stands of trees subjected to various doses of oxidant air pollu-

tants could be useful for quantifying the dynamics of ozone-induced tree decline. This approach is being investigated by Arkeley (in Kickert *et al.*[32]) in the San Bernardino Mountains.

Sophisticated decomposition models are being developed. A simulation model developed by Bunnell and Dowding[8] for tundra sites is a nine-compartment model with 23 transfers between compartments. This type of model may provide the only method for understanding the extremely complex litter decomposition process.

In summary, it is anticipated that decreasing litter production by green plants experiencing pollutant stress would result in a similar reduction in the inventory of nutrient elements held within the system, owing to the interruption of cycling pathways and mechanisms of nutrient conservation.[38]

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13

Effects of Photochemical Oxidants on Materials

The effects of photochemical oxidants on materials have been investigated by exposure of materials to ambient air containing photochemical oxidants, including ozone, PAN, peroxybenzoylnitrate (PBzN), and all other molecules in ambient air that oxidize iodide ion (but not nitrogen dioxide), as prescribed in the photochemical-oxidant ambient air quality standard.[48] Laboratory experiments for corroboration of field tests always use ozonized air that contains no NO_x if photochemical generation is used and may contain NO_x if electric discharge is the method of ozone generation. The reactivity of oxidizing species, such as atomic oxygen and excited electronic states of molecular oxygen, must be assessed both in test chambers and in ambient air before blame for damage is assigned to ozone. On the presumption that ozone is the damaging species in test-chamber experiments in which PAN and similar oxidants are absent, photochemical-oxidant damage is often referred to in this discussion as "ozone" damage. The subject has been reviewed in *Air Quality Criteria for Photochemical Oxidants*[47] and in a systems study by Salmon[35] and more recently by Sanderson.[40] Other, rather comprehensive, although specialized, reviews have appeared on the effects of air pollutants on textiles and dyes,[49] on rubber,[28] and on paint,[8,42] and on their economic effects.[50] In preparing this chapter, the literature has been reviewed with special emphasis on papers published since 1970, when the EPA criteria document appeared.[47]

TEST-CHAMBER STUDIES

Experimental studies on the effects of ozone on materials usually involve the laboratory generation[1,2,43] of ozone by photolysis of air with a mercury resonance lamp or by some form of electric discharge through air or oxygen. Several oxidizing species result, among which are the first singlet state of atomic oxygen, $O(^1D)$; ground-state atomic oxygen, $O(^3P)$; the lowest-lying singlet molecular oxygen, $O_2(^1\Delta_g)$; and ozone. To identify the species responsible for the effect, one must know the rate constants characteristic of the interactions of these species with the material in question and the relative concentrations of the species at the contact surface between the ozonized air and the damaged material.

The Photochemical Ozone Generator

The standard ASTM test method[43] (D-1149-64) for rubber damage includes a test chamber (volume, 0.11–0.14 m^3) through which ozonized air flows at a rate greater than 0.6 m/s. Because the residence time of the ozonized air in the test chamber is about 1 s, the ozone may be expected to reach the material in about 0.1 s. A somewhat similar test procedure[1,2] (AATCC test method 109-1972; ANSI L14, 174-1973) is used in testing colorfastness. The ozone generator is usually (but not necessarily) a mercury-vapor resonance lamp with emission lines at 184.9 and 253.7 nm. The 184.9-nm line is absorbed, and two ground-state oxygen atoms are produced:

$$O_2 + h\nu\ (184.9\ \text{nm}) \rightarrow 2O(^3P). \qquad (13\text{-}1)$$

Each oxygen atom then reacts with diatomic oxygen to form ozone:

$$O + O_2 + M \rightarrow O_3 + M, \qquad (13\text{-}2)$$

where M signifies any gaseous molecule, such as O_2 or N_2. Usually, the 253.7-nm line is more intense than the 184.9-nm line and falls in a very strong absorption band of ozone:

$$O_3 + h\nu\ (253.7\ \text{nm}) \rightarrow O(^1D) + O_2(^1\Delta_g). \qquad (13\text{-}3)$$

The species produced by this photochemical reaction can be quenched to their ground electronic states by collisional processes:

$$O(^1D) + M \rightarrow O(^3P) + M; \qquad (13\text{-}4)$$

$$O_2(^1\Delta_g) + O_2(^3\Sigma_g^-) \rightarrow 2O_2(^3\Sigma_g^-). \quad (13\text{-}5)$$

In addition, the ground-state oxygen atom reacts with ozone:

$$O + O_3 \rightarrow 2O_2. \quad (13\text{-}6)$$

Rate constants of these reactions are summarized in Table 13-1.

CONCENTRATIONS IN THE GENERATOR

The absorbed intensity, I_a, is obtained from the Beer-Lambert law, which relates the incident intensity, I^0, and the transmitted intensity, I_t,

$$I_t/I^0 = \exp(-\epsilon cl), \quad (13\text{-}7)$$

where ϵ is the absorption coefficient, c is the concentration of the absorbing species, and l is the path length. It follows that

$$\frac{I^0 - I_t}{I^0} = \frac{I_a}{I^0} = 1 - \exp(-\epsilon cl). \quad (13\text{-}8)$$

For $\epsilon cl \ll 1$,

$$I_a = I^0 \epsilon cl. \quad (13\text{-}9)$$

The stationary-state expressions are:

$$d[O]/dt = 2I_1 - k_2[O][O_2][M] + [O(^1D)][M] - k_6[O][O_3] = 0; \quad (13\text{-}10)$$

TABLE 13-1 Rate Constants at a Temperature of 300 K ($M \equiv O_2$ or Air)

Reaction	Rate Constant (k)	Reference
(13-1) $O_2 + h\nu$ (184.9 nm) $\rightarrow 2O(^3P)$	$\epsilon_1 = 2.0 \times 10^{-20}$ molec^{-1} cm^2 (base e)	51
(13-2) $O + O_2 + M \rightarrow O_3 + M$	6.0×10^{-34} molec^{-2} cm^6 s^{-1}	16
(13-3) $O_3 + h\nu$ (253.7 nm) $\rightarrow O(^1D) + O_2(^1\Delta_g)$	$\epsilon_3 = 1.27 \times 10^{-17}$ molec^{-1} cm^2 (base e)	18
(13-4) $O(^1D) + M \rightarrow O(^3P) + M$	7.5×10^{-11} molec^{-1} cm^3 s^{-1}	16
(13-5) $O_2(^1\Delta_g) + O_2(^3\Sigma_g^-) \rightarrow 2O_2(^3\Sigma_g^-)$	2.2×10^{-18} molec^{-1} cm^3 s^{-1}	16
(13-6) $O + O_3 \rightarrow 2O_2$	8.6×10^{-15} molec^{-1} cm^3 s^{-1}	16

$$d[O(^1D)]/dt = I_3 - k_4[O(^1D)][M] = 0; \quad (13\text{-}11)$$

$$d[O_3]/dt = k_2[O][O_2][M] - I_3 - k_6[O][O_3] = 0. \quad (13\text{-}12)$$

The values of I_1 and I_3—the absorbed light intensities at 184.9 nm and 253.7 nm, respectively—are related to incident intensities by expressions like Equation 13-8. The incident intensities depend heavily on lamp design and construction. For the purpose of the stationary-state computation, we use (on the basis of experience) $I_1 = 10^{17}$ photons/cm^3 and $I_3 = 3 \times 10^{17}$ photons/cm^3-s. In the calculations, a 1-cm path length is assumed. Summing Equations 13-10 through 13-12 and referring to Table 13-1:

$$I_1 = k_6[O][O_3] = I_1^0 \epsilon[O_2]. \quad (13\text{-}13)$$

The oxygen concentration in air is taken to be 5×10^{18} molecules/cm^3:

$$[O] = 1.16 \times 10^{30}/[O_3]. \quad (13\text{-}14)$$

The concentration of $O(^1D)$ is obtained from Equation 13-11:

$$[O(^1D)] = \frac{I_3}{k_4[M]} = \frac{I_3^0 \epsilon_3[O_3]}{k_4[M]} = 2.1 \times 10^{-9}\,[O_3]. \quad (13\text{-}15)$$

The concentration of $O_2(^1\Delta_g)$ is obtained by means of its stationary-state expression:

$$\frac{d[O_2(^1\Delta)]}{dt} = I_3 - k_5[O_2(^1\Delta)]M = 0; \quad (13\text{-}16)$$

$$[O_2(^1\Delta)] = \frac{I_3^0 \epsilon_3[O_3]}{k_5 M} = 7.1 \times 10^{-2}[O_3]. \quad (13\text{-}17)$$

Because $I_1 = k_6[O][O_3]$ and $I_3 = k_4[O(^1D)][M]$, Equation 13-10 yields

$$I_1 + I_3 = k_2[O][O_2][M]. \quad (13\text{-}18)$$

Substituting for [O] and solving with the quadratic formula:

$$I_1 + I_3 = k_2[O_2][M] \times 1.16 \times 10^{30} = I_1^0 \epsilon_1[O_2] + I_3^0 \epsilon_3[O_3]; \quad (13\text{-}19)$$

$$\underbrace{I_3^0 \epsilon_3[O_3]^2}_{a} + \underbrace{I_1^0 \epsilon_1[O_2][O_3]}_{b} - \underbrace{k_2[O_2][M] \times 1.16 \times 10^{30}}_{c} = 0; \quad (13\text{-}20)$$

$$[O_3] = \frac{-b + \sqrt{b^2 - 4ac}}{2a}. \tag{13-21}$$

Evaluating a, b, and c,

$a = 3.3$,
$b = 3.7 \times 10^{15}$, and
$c = 8.5 \times 10^{34}$.

The solution is $[O_3] = 1.6 \times 10^{17}$ molecules/cm^3. The concentrations of the oxidizing species for $[O_3] = 1.6 \times 10^{17}$ molecules/cm^3 are given in Table 13-2. The ratio of rates of attack of $O(^3P)$ and ozone on a material in the ozone generator is given by

$$R_O/R_{O_3} = \frac{[O(^3P)]}{[O_3]} \cdot \frac{k_O}{k_{O_3}}, \tag{13-22}$$

where k_O and k_{O_3} are the rate constants. As a hypothetical example of a material undergoing a damage test, we choose an olefinic polymer. In the absence of data on appropriate material, we may presume that damage done to an olefinic material by oxygen atoms and ozone would proceed at the same relative rates as attack on a typical olefin, e.g., butene-2. Rate constants for attack of various oxygen species on *trans*-2-butene are presented in Table 13-3. Thus, we estimate

$$k_O/k_{O_3} = 3.5 \times 10^5 \tag{13-23}$$

and

$$R_O/R_{O_3} = (4.5 \times 10^{-3})(3.5 \times 10^5) = 15.8. \tag{13-24}$$

TABLE 13-2 Stationary-State Concentration of Oxidants in Photochemical Ozone Generator[a]

Oxidant	Concentration, molecules/cm^3
O_3	1.6×10^{17}
$O(^3P)$	7.3×10^{12}
$O(^1D)$	3.4×10^{8}
$O_2(^1\Delta_g)$	1.1×10^{16}

[a] l = 1 cm; $I_1^0 = 10^{17}$ photons/cm^3-s; $I_3^0 = 3 \times 10^{17}$ photons/cm^3-s.

TABLE 13-3 Rate Constants for Reaction of Oxygen Species with *trans*-2-Butene[a]

Oxidant	Rate Constant (k), molec^{-1} cm^3 s^{-1}
$O_2(^1\Delta_g)$	4.6×10^{-18}
O_3	3.5×10^{-17}
$O(^3P)$	2.3×10^{-11}

[a] Data from Demerjian *et al.*[12]

It follows that, although the ozone concentration is more than 10,000 times that of the oxygen atom, the latter reacts so rapidly with olefins that oxygen atoms, rather than ozone, will be the species responsible for material damage, if the material is placed in the ozone generator. Similarly, for $O_2(^1\Delta_g)$:

$$\frac{RO_2(^1\Delta_g)}{RO_3} = \frac{k_\Delta}{k_{O_3}} \cdot \frac{[O_2(^1\Delta)]}{[O_3]}$$

$$= \frac{2.3 \times 10^{-18}}{1.1 \times 10^{-17}} \cdot \frac{1.1 \times 10^{16}}{1.6 \times 10^{17}} = 0.014. \quad (13\text{-}25)$$

Therefore, $O_2(^1\Delta_g)$ could be a significant problem in ozone damage tests on olefinlike materials, if the test were done in the ozone generator. The results in Table 13-2, although only very approximate because of the assumptions made, show that in a photochemical ozone generator we may expect $O_2(^1\Delta_g)$ concentrations to be an appreciable fraction of the ozone concentration. The $O(^1D)$ concentration is negligibly small, because it is removed in Reaction 13-4 on almost every collision.

CONCENTRATION DOWNSTREAM FROM THE GENERATOR

The ASTM method requires that the ozone generator be outside the test chamber, and we must ask how long $O_2(^1\Delta_g)$ and $O(^3P)$ survive after the ozonized air leaves the generator on its way to the material under test. About 0.1 s is required for the ozone to reach the material after leaving the generator. As the light source is extinguished ($t = 0$) or as the ozonized air leaves the photolysis chamber, the rate at which $O_2(^1\Delta_g)$ is removed is—using S to signify $O_2(^1\Delta_g)$—

$$-\frac{d[S]}{dt} = k_5[S][M]; \quad (13\text{-}26)$$

$$-d \ln [S] = k_5[M]\, dt. \tag{13-27}$$

Thus 0.1 s after the light is turned off, $[S]_0$ decays to $[S]$ (by Reaction 13-5, whose rate constant is given in Table 13-1). The concentration, $[M]$, is 2.7×10^{19} molecules/cm^3. Solving Equation 13-27, we obtain

$$-\int_{[S]_0}^{[S]} d \ln S = 6; \tag{13-28}$$

$$\ln \frac{[S]}{[S]_0} = -6; \tag{13-29}$$

$$\frac{[S]}{[S]_0} = 10^{-2.6}. \tag{13-30}$$

Therefore, although the $O_2(^1\Delta_g)$ is quenched very inefficiently by collision, at a pressure of 1 atm quenching is sufficiently rapid that only a few tenths of 1% of the original $O_2(^1\Delta_g)$ can reach the test material. However, when the pressure is smaller—say, 76 torr or 2.5×10^{18} molecules/cm^3—$1n\ ([S]/[S]_0) = -0.6$ after 0.1 s and more than half the original $O_2(^1\Delta_g)$ molecules may reach the test material.

Finally, we must ask whether the ASTM procedure is likely to result in removal of oxygen atoms with sufficient rapidity for the observed damage to be attributed to ozone. The rate of removal of oxygen atoms by Reaction 13-2, which is the major sink, is given by:

$$-d[O]/dt = k_2[O][O_2][M]; \tag{13-31}$$

$$-\int_{[O]_0}^{[O]} d \ln [O] = k_2[O_2][M] \int_0^{0.1} dt; \tag{13-32}$$

$$\ln \frac{[O]}{[O]_0} = -8.1 \times 10^3; \tag{13-33}$$

$$[O]/[O]_0 = 10^{-3500}. \tag{13-34}$$

Therefore, the ASTM test is valid for ozone damage, inasmuch as both the oxygen atoms and the $O_2(^1\Delta_g)$ are rapidly removed downstream from the generator. The importance of having the ozone generator outside the test chamber cannot be overemphasized. The validity of any ozone damage test using other sources depends very heavily on the test procedure. If an electric discharge is used as the ozone generator, the test

chamber should be designed in accord with such chemical kinetic considerations as indicated in the model discussed here. The relative concentrations of oxidizing species in a radiofrequency or microwave discharge or a photochemical generator are subject to conditions and to some speculation. However, equal concentrations of $O(^3P)$ and $O(^1\Delta_g)$ have been suggested as emerging from discharged air.[15] The ratio of oxygen atoms to ozone depends critically on the pressure. In a microwave discharge where the pressure may be 1 torr, 0.1 s after leaving the discharge a substantial fraction of the oxygen atoms remains.

Because oxygen-atom reaction rate constants can be orders of magnitude greater than those for ozone, an experiment done on material subject to a reduced-pressure discharge is likely to signify damage done by oxygen atoms, rather than ozone.

Oxidant Species Concentrations in Polluted Air

We have shown that, in a properly designed laboratory experiment, ozone is likely to be the only oxidant species producing damage in the test material. In a real atmosphere, such photochemical oxidants as PAN and PBzN are formed in complex atmospheric reactions. Because there is no information on the effects of these oxidants on materials, no consideration is given to them in assessing material damage in this discussion. In a smoggy atmosphere, sunlight and energy transfer processes result in generation of $O_2(^1\Delta_g)$, $O(^1D)$, $O(^3P)$, and such free radicals as OH and HO_2. Therefore, it is necessary to consider the concentrations of these species relative to that of ozone in a real atmosphere. This has been done, notably by Demerjian *et al.*,[12] one of whose estimates, calculated from smog-chamber considerations, is shown in Table 13-4. The ozone

TABLE 13-4 Calculated Concentrations of Oxidant Species in Smog Chambers*

Oxidant	Concentration, molecules/cm^3
$O_2(^1\Delta_g)$	1.4×10^8
O_3	3.8×10^{12}
$O(^3P)$	2.6×10^5
OH	1.9×10^6
HO_2	1.0×10^{10}

*Calculated concentrations for oxidants in smog chamber under conditions outlined in Chapter 2 for 60-min irradiation (see Table 2-2).

concentration assumed in Table 13-4 is typical of a fairly polluted atmosphere. To estimate the relative contributions of each oxidizing species to oxidant damage, it is necessary to multiply the concentration by the rate constant for each individual material. Unfortunately, there is almost no information on rate constants. It has been assumed in nearly all cases that the oxidizing species is ozone.

EFFECTS OF OXIDANTS ON INDIVIDUAL MATERIALS

According to the Midwest Research Institute (MRI) study of 1970,[35] both ferrous and nonferrous metals have excellent resistance to ozone. Such materials as building stone, building brick, cement, glass, and graphite also have excellent resistance. Some synthetic rubbers have good ozone resistance, but natural rubber has notoriously poor resistance. Polyethylene, polystyrene, and polypropylene are believed to have only fair resistance to ozone, and acetate, nylon, and fibers of cotton, rayon, and cellulose esters are also in the "fair" category. Wood is considered fairly resistant, but little information is available. The approach used by MRI in assessing economic effects is that the value of material exposed to air pollution, Q, is the product of four factors: the annual production, P; the average economic life of the material, N; the fraction of material exposed to air pollution, F; and the labor factor, R, which reflects the cost of putting the material in place where it is used. Hence,

$$Q = P \cdot N \cdot F \cdot R. \tag{13-35}$$

Having defined Q, the MRI report defines the damage costs as the product of Q and ν, the "interaction" value per year. For example, if Q = \$1 billion and a pollutant causes a 1% reduction in economic life per year of exposure, then $\nu = 0.01$, and the economic loss, L, due to the pollutant is $Q\nu$, or \$10 million/yr. The values of ν for pollutant damage generally are difficult to obtain, because few data are available. For the particular pollutant, ozone, the values of ν are almost impossible to assess. The MRI report points out that "intuitive feelings" are sometimes used to assess ν. The materials that have been classified poor or fair in ozone resistance are listed in Table 13-5 in order of decreasing in-place value. The chemical interaction value, ν, and the economic loss, L, are also tabulated. Because it is difficult at best to estimate overall pollutant interaction, ozone damage has been assessed in the last column of Table 13-5 by estimating L', the ozone damage, as follows: If chemical resist-

TABLE 13-5 Economic Value of Ozone-Sensitive Materials Exposed to Pollution[a]

Material	P, $ million	N, yr	F	R	Q, $ million	ν, per yr	L, $ million	L', $ million
Paint	2,587	4	0.7	3.3	23,900	0.05	1,195	600
Wood	10,875	30	0.03	1.8	17,620	0.001	18	9
Cotton	3,336	6	0.1	1.9	3,800	0.04	152	76
Polyethylene	975	6	0.2	1.0	1,170	0.01	12	12
Nylon fiber	830	6	0.1	1.9	950	0.04	38	13
Polystyrene	850	5	0.2	1.0	850	0.01	9	9
Polypropylene	403	8	0.2	1.0	640	0.01	6	6
Natural rubber	1,790	6	0.05	1.0	540	0.10	54	27
Rayon fiber	288	6	0.1	1.9	330	0.04	13	7
Nylon plastics	106	8	0.2	1.0	170	0.01	2	1
Acetate plastic	58	10	0.2	1.0	120	0.01	1	0.5

[a] L' refers to ozone damage; ν and L refer to total gaseous-pollutant damage. See text for explanation of other column headings.

ance has been estimated by MRI as either poor or fair for one pollutant other than ozone for which a national ambient air quality standard has been set, $L' = L/2$; if two other pollutants are in the poor or fair category, $L' = L/3$. The data in Table 13-5 should be regarded as very rough and indicative only of broad-brush outlines. For example, paint damage is undoubtedly a very important economic consequence of ozone pollution, but, as we shall see, the estimate of rubber damage in Table 13-5 is much too low.

The most studied materials, as well as the most important economically, with respect to ozone damage are paint and elastomers (e.g., rubber). Other materials are reviewed individually where scientific data are available.

Paint

FAILURE OF PAINT FILMS

Paint formulations consist of a binder (a natural or synthetic polymer or drying oil), a solvent, and a pigment or colorant, including an extender, typically calcium carbonate or a silicate. Because of the reactivity of organic polymers toward ozone, it is not surprising that ozone damage has been observed, at least in laboratory experiments. In 1968,

paint sales of over $2.5 billion were recorded in the United States. Table 13-6 outlines these sales.[42] The failure of paint films takes on a variety of forms, as shown in Table 13-7, and methods for testing the various forms of failure have been delineated by the ASTM.[3] These methods are listed in Table 13-8. The paint industry has been concerned mainly with "weatherability" of paints, rather than specifically with the effect of air pollutants.

Probably the most definitive study of the effect of air pollutants on paint is contained in a 1972 report of the Sherwin-Williams Research Center.[8] Five commercially important coating systems were selected for study:

1. House paint—lead-titanium-zinc extender in oil with 100% rutile titanium dioxide.
2. House paint—titanium extender in acrylic latex with 100% rutile titanium dioxide.
3. Industrial maintenance coatings—titanium in alkyl with 100% rutile titanium dioxide.
4. Coil coating finishes—titanium extender in urea-alkyl with 75% rutile and 25% anatase titanium dioxide.
5. Automotive refinish lacquer—titanium in nitrocellulose-acrylic with 100% rutile titanium dioxide.

For each coating system, three tasks were performed:

- Existing exterior exposure records were reviewed with respect to visual erosion ratios.
- Short-term exposures were studied at Leeds, North Dakota; Los Angeles, California; Chicago, Illinois; and Valparaiso, Indiana. The North Dakota location was a clean, rural site; Los Angeles represented a high-oxidant urban site; Chicago was a high-sulfur dioxide location; and Valparaiso was considered to have moderate sulfur dioxide pollution and low oxidant concentrations. Tests with commercial instruments included those for erosion, gloss, sheen, surface roughness, and tensile strength.
- Accelerated laboratory exposures were made at various pollutant concentrations: no pollutant, 0.1-ppm sulfur dioxide, 1-ppm sulfur dioxide, 0.1-ppm ozone, and 1-ppm ozone.

As measured by erosion rates (linearly related to pollutant concentration), it was found that in all cases coatings were affected more by sulfur dioxide than by ozone. Sulfur dioxide at 1 ppm had the following effects on the five paint systems: system 1, "considerable"; systems 2 and 4, "moderate"; and systems 3 and 5, "no effect." Both attenuated total reflection and scanning electron photomicrographs showed that the damage was greater in Chicago and Valparaiso than in Los Angeles and Leeds.

TABLE 13-6 Trade and Industrial Paint Sales, United States, 1968[a]

Type of Sale	Amount, millions of gallons	Cost, $ millions	Unit Cost, $/gal
Trade	424	1,428	3.37
Interior house paints:			
Latex emulsion	145	425	2.93
Oil and alkyd	45	170	3.78
Primer, sealers, etc.	10	30	3.00
Miscellaneous	25	100	4.00
Exterior house paints:			
Latex emulsion	55	190	3.45
Oil and alkyd	40	155	3.88
Enamels	15	55	3.67
Primers, sealers, etc.	10	35	3.50
Miscellaneous	20	50	2.50
Automotive refinishing	30	150	5.00
Traffic	25	45	1.80
Other	4	23	5.75
Industrial	419	1,159	2.77
Automotive, new	55	170	3.09
Marine	20	85	4.25
Railroad, aircraft, etc.	15	40	2.67
Coil coating	20	80	4.00
Prefinished wood	15	40	2.67
Industrial maintenance	45	160	3.56
Machinery and equipment	30	75	2.50
Miscellaneous	219	509	2.32

[a] Data from Spence and Haynie.[42]

Studies have been conducted on creep-compliance tests[42] in which paint films were subjected to tensile loads of 4–7 psi ($27.2–47.6 \times 10^3$ N/m^2) and to 6% ozone for 505 h. A typical result for a high-quality emulsion-base paint is shown in Figure 13-1. Creep compliance is reduced by exposure to 6% ozone. If the effect is linearly related to ozone concentration, we might expect the same reduction in creep compliance at 0.1-ppm ozone in 3×10^8 h, or some 30,000 yr. Thus, reduction in creep compliance is not viewed as having a serious ozone contribution.

ECONOMIC EFFECTS

The MRI report (pp. 41 ff.) computes L, the increased cost of cleaning or repainting per year, as the product of Q, the consumer purchase price

TABLE 13-7 Types of Film Failure[a]

Type Failure	Description
Chalking	Formation of powdery layer on surface of coating that is being eroded away
Cracking or checking	Shrinkage of the coating resulting in film rupture
Alligatoring	Film rupture resulting from the application of a brittle film over a more flexible coating
Peeling	Poor adhesion of the coat to substrate
Color fading	Reaction of binder or pigment in presence of sunlight or environment
Blistering	Projections or pimples on film that result from trapping of solvent or moisture between substrate and film
Rusting	Oxidation of metallic substrates, such as iron or steel, when film permits moisture or chemicals to attack substrates

[a] Data from Spence and Haynie.[42]

TABLE 13-8 ASTM Test Methods for Exterior Paint Films

Test	Purpose
D 659-44	Evaluating degree of resistance to chalking
D 660-44	Evaluating degree of resistance to checking
D 661-44	Evaluating degree of resistance to cracking
D 662-44	Evaluating degree of resistance to erosion
D 772-47	Evaluating degree of resistance to flaking (scaling)
D 1641-59	Measuring exterior durability of varnishes
D 1543-63	Measuring color change of white architectural enamels
D 1654-61	Evaluating painted or coated specimens subjected to corrosive environments
D 2197-68	Measuring adhesion of organic coatings
D 2370-68	Measuring elongation and tensile strength of free films of paint, varnish, lacquer, and related products with tensile testing apparatus

of a unit of material; γ, the cost of cleaning or repainting one such unit; and Δf, the fractional increase in cleaning frequency resulting from particulate pollution:

$$L = Q \cdot \gamma \cdot \Delta f, \text{ dollars/(yr} \times \text{unit)}. \tag{13-36}$$

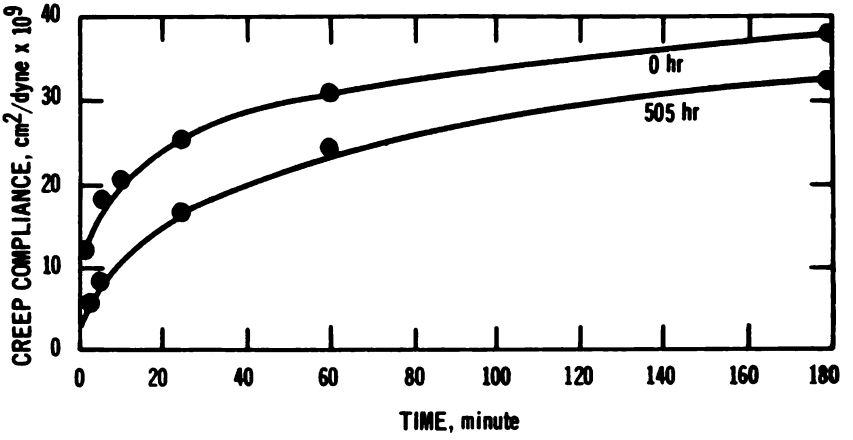


FIGURE 13-1 Initial creep of EM 7338 first-quality emulsion-base paint, before and after 505-h exposure to ozone at 60°C. Reprinted from Spence and Haynie.[42]

The cost, γ, of cleaning one unit is determined for two classes of material, fibers and nonfibers. For fibers, \$1.10/lb is taken as the cost of cleaning; for nonfibers, \$0.10/ft^2 is taken as the cost of cleaning (removal of particulate material). The purchase price is defined as the material cost times a factor that gives the consumer purchase price including labor (painters, outlet salesmen, and their overheads, etc.). The quantity $\gamma\Delta f$ is given by $\nu = \gamma\Delta f = (L/Q)$-yr and is called the soiling interaction:

$$L = Q\nu. \tag{13-37}$$

The determination of the quantity Δf, the fractional increase in cleaning frequency due to particulate matter, is the difficult part of the problem. The data of Michelson and Tourin (cited in Spence and Haynie[42]) suggest a value of ν for paint of 1.5/yr. The value of Q is obtained as follows: According to Noble,[30] the annual production of paint in 1968 was \$2.59 billion. Because the economic life of paint is considered to be 4 yr, \$10.36 billion worth of paint is in place, of which only 70% is exposed to atmospheric pollutants. Thus, \$7.25 billion is the manufacturers' value of in-place paint exposed to air pollution. Using a labor factor of 3.3, the total in-place value of paint is \$23.9 billion. Because $\nu = 1.5$ for particulate soiling, the cost of pollution damage (cleaning costs) is some \$36 billion/yr.

The costs associated with chemical effects of gaseous pollutants could

not be ascertained for each pollutant, because of a paucity of information. The value of ν for chemical deterioration was obtained by an educated guess that repainting is needed every 4 yr in a polluted environment and every 5 yr in a pristine air environment. Thus, ν (chemical) = 0.05. The corresponding figure for chemical deterioration costs is 0.05 ($23.9 billion) = $1.195 billion/yr.

Spence and Haynie have attempted to assess costs of air pollution damage to paints without special regard to the identity of the pollutant doing the damage. However, repainting frequencies have been determined in five American cities whose particulate concentrations have been measured. There appears to be a linear (although not necessarily causal) relationship between the maintenance frequency and particulate concentration.[42] Economic loss due to pollution was estimated by the following procedure: The average service life of exterior household paint in urban areas is 3 yr, and in rural areas, 6 yr. Thus, 0.33 and 0.17 are the replacement probabilities per year. Because 60% of the surface painted is urban and 40% rural, it may be calculated that some 74% of the paint used over a long period is consumed in urban areas. The total value of manufactured paint is $485 million, of which 74%, or $359 million/yr, is the cost of paint consumed in urban areas. Its service life is 50% less than in rural areas, so 0.50 ($359 million) = $180 million is the annual cost of exterior household paint deterioration due to pollution. The value of loss of paint in place was computed to be 3.0 ($180 million) = $540 million. It is noteworthy that the MRI report estimates $1.195 billion repainting costs due to chemical pollution.

The economic assessment of air pollution damage to exterior paints is

TABLE 13-9 Economic Assessment of Air Pollution Deterioration of Exterior Paints (1968 Figures—Manufacturers' Costs)[a]

Exterior Paint Class	Value of Paint Exposed in Urban Areas, $ million	Loss due to Air Pollution, $ million
Coil coating[b]	31	8
Automotive refinishing	111	22
Industrial maintenance	74	15
Household[b]	359	180
Total	575	225

[a] Data from Spence and Haynie.[42]
[b] Extender paints.

summarized in Table 13-9. There appears to be no definitive way to determine the fraction of the $225 million loss for all exterior paints that is due to photochemical oxidants. According to the convention introduced by MRI—that repainting costs may be considered to be due to chemical pollution—only NO_x, sulfur dioxide, and photochemical oxidants seem likely to be involved in this kind of paint damage. Sulfur dioxide seems to attack only the extender (e.g., calcium carbonate) component. It might also be expected that nitrogen dioxide would attack only extender paints. Sulfur dioxide has no effect on the nonextender types—industrial maintenance and automotive refinishing paints. Therefore, the $37 million worth of damage done to the nonextender types each year may be attributed to the effects of oxidants. Of the $188 million worth of damage to extender paints, we may expect some 80% to be caused by sulfur dioxide and nitrogen dioxide, with $38 million worth of damage (20%) due to oxidants. Thus, a total of $75 million worth of paint damage per year is caused by oxidants. The figure of $75 million includes only the cost at the manufacturers' level. The cost at the consumer level—i.e., the total cost of the paint applied to a surface—is obtained by multiplying by a factor of 3.0 (according to Spence and Haynie[42]) or 3.3 (according to Salmon[35]). Thus, the oxidant damage to paint (using a 3.3 factor) is $248 million/yr.

Elastomers

FAILURE OF ELASTOMERS

Elastomers include both synthetic elastomers and natural rubber. The most extensive study of the effects of ozone on elastomers (including economic effects) was done by Mueller and Stickney[28] and Stickney *et al.*,[44] and much of what follows is taken from the latter report. Table 13-10 presents the estimated elastomer production in 1975. The effect of ozone on cracking of natural rubber was proved by Newton[29] and by Crabtree and Kemp.[9] In general, highly unsaturated elastomers are more subject to ozone attack than are saturated elastomers, because ozone attacks aliphatic double bonds. The more highly unsaturated elastomers are listed in Table 13-11. Of these, neoprene is particularly resistant to ozone, owing to the presence of the electronegative chlorine atom in its structure. Elastomers crack under the influence of ozone when stressed, and the crack propagates in a direction normal to the stress direction. The chemical mechanism may be written:[10]

TABLE 13-10 Polymer Usage Projected to 1975[a]

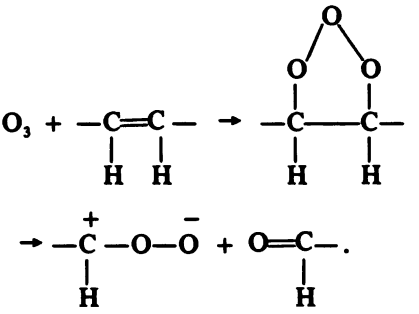
Polymer	Estimated Production, 1,000 tons[b]	Estimated Average Price, cents/lb[c]	Value, $ million	Portion of Total Polymer Value, %
SBR—copolymer of butadiene and styrene	1,748	23	804.1	39.8
Natural—natural polyisoprene	689	22	303.2	15.0
Polybutadiene—polymerized butadiene	375	27	202.5	10.0
Neoprene—polymerized chloroprine	146	40	116.8	5.8
Nitrile—copolymer of butadiene and acrylonitrile	95	50	95.0	4.7
Urethane—polymers containing urethane linkage	45	125	112.5	5.6
Butyl—copolymer of isobutylene and isoprene	101	27.5	55.5	2.7
Silicone—polydimethylsiloxane	13	250	65.0	3.2
Polyisoprene—synthetic polyisoprene	134	26	69.7	3.4
Polyacrylate—polymer or copolymer based on an acrylate	3	80	4.8	0.2
EPDM—copolymer of ethylene, propylene, and a diene	168	25	84.0	4.2
Polysulfide—polysulfide	17	135	45.9	2.3
Fluorocarbon—fluorinated polymers	2	1,250	50.0	2.5
Chlorohydrin—polymer of epichlorohydrin or copolymer of epichlorohydrin and ethylene oxide	8	85	13.6	0.7
Total	3,544		2,022.6	100.0

[a] Derived from Stickney *et al.*[44]
[b] Data from Dworkin.[14]
[c] Battelle and industry estimates.

TABLE 13-11 Highly Unsaturated Elastomers[a]

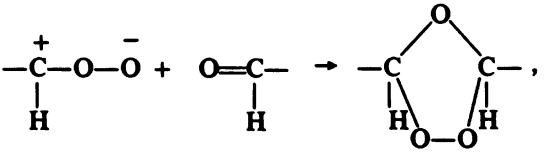
Polymer	Use	Crack Growth Rate, mm/min[b]	1970 Cost, cents/lb
SBR	General	0.37	23
Natural	General	0.22	25
Polybutadiene	Wear-resistant	ND	25
Neoprene	Oil-resistant	0.01	40
Nitrile	Oil-resistant	ND	50
Polyisoprene	General	ND	24

[a] Derived from Mueller and Stickney.[28]
[b] Data from Braden and Gent.[7] ND = no data.



(13-38)

The zwitterion and aldehydic group are thought to combine:



(13-39)

to form a highly oxidized structure of much lower extensibility than the unoxidized elastomer. Cracking is the result.

There are two main approaches to avoidance of ozone damage. One is the addition of antiozonants. The more extensively used antiozonants are listed in Table 13-12. These antiozonants have limited solubility in elastomers and tend to "bloom" to the surface. The antiozonant action is not well understood chemically, but probably involves formation of a protective film. Crack initiation occurs at critical stress, which is strongly

TABLE 13-12 Elastomer Antiozonants

Antiozonants	1970 Price, cents/lb
N,N'-Diphenyl-*p*-phenylenediamine	99
N,N'-Di-(2-Octyl)-*p*-phenylenediamine	90
N,N'-Di-3-(5-Methylheptyl)-*p*-phenylene-diamine	90
*N,N'*bis(1,4-Dimethylphentyl)-*p*-phenylene-diamine	86
*N,N'*bis(1-Ethyl-3-methylpentyl)-*p*-phenylenediamine	90
*N,N'*bis(1-Methylheptyl)-*p*-phenylenediamine	90

influenced by the presence of a few percent of the *N,N'*-dialkyl-*p*-phenylenediamines. Dithiocarbamates are a second class of antiozonants that act by slowing crack growth rates. The crack growth rates depend linearly on the concentration of ozone, as shown in Figure 13-2. In addition to antiozonants and special polymeric structures, protection against ozone damage during storage is afforded by the use of waxes, protective coatings, and paper wrapping.

IDENTITY OF OXIDIZING SPECIES

It is known that atomic oxygen, $O(^3P)$, reacts with double bonds at room temperature to produce a rupture of the carbon-carbon chain. Because elastomers are known to be very stable to ground-state ($^3\Sigma_g^-$) molecular oxygen, the possible oxidants responsible for rubber-cracking in polluted air are atomic oxygen, $O_2(^1\Delta_g)$, ozone, OH, and HO_2. Relative rates of reaction are products of rate constants and concentrations of oxidizing species.

One elastomer, *cis*-polybutadiene, has been exposed specifically to $O_2(^1\Delta_g)$ in the absence of ozone or oxygen atoms.[22] *cis*-Polybutadiene forms hydroperoxides (—OOH) when exposed to oxygen at 5 torr that has been subjected to an electrodeless discharge (2,450 MHz). Oxygen atoms and ozone were removed by mercuric oxide in these experiments, leaving only $O_2(^1\Delta_g)$. As we have seen, $O_2(^1\Delta_g)$ survives for a long time at the low pressures used, nearly half of it surviving 1 s after leaving the discharge zone. No measurements have been made of relative rates of reaction of $O_2(^1\Delta_g)$, ozone, atomic oxygen, OH, and HO_2 with individual materials, and it is therefore not possible to know the relative importance

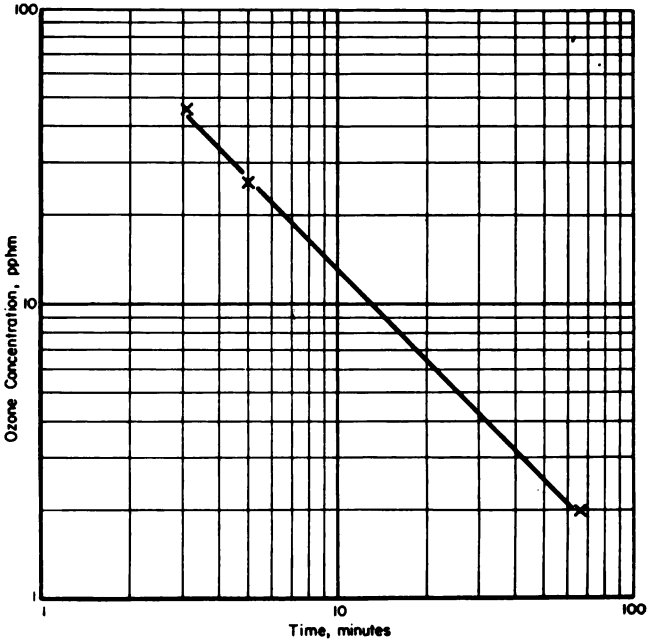


FIGURE 13-2 Effect of ozone concentration on cracking time. Reprinted from Mueller and Stickney.[28]

of attack by these species. The results in Table 13-13 would lead to the speculation that ozone is more important than $O_2(^1\Delta_g)$ in *cis*-polybutadiene damage. Because we do not have information on the relative rates of reaction of the five species with elastomers, we can assume that the rate constants parallel those of the reactions with *trans*-2-butene (see Table 13-3), as was done in estimating the rates of reaction with an olefinic polymer in the laboratory ozone generator. Using the concentrations in Table 13-4 with the rate constants in Table 13-3, we arrive at the attack rates shown in Table 13-13. These results suggest that HO_2 is the most important oxidant in attack on elastomers in ambient air.

ECONOMIC EFFECTS

Mueller and Stickney at Battelle[28] revealed very substantial compounding costs for protection against photochemical oxidants. Table 13-14 summarizes the results of an analysis based on responses to a questionnaire. These costs include the introduction of such ozone-resistant polymers as butyl, neoprene, EPDM, Hypalon, and polysulfide. Fair

TABLE 13-13 Rates of Attack of Oxidants on *trans*-2-Butene in Simulated Polluted Air

Oxidant	Rate constant (k), molec^{-1} cm^3 s^{-1} [a]	Concentration, molec/cm^3 [b]	Relative Rate
$O_2(^1\Delta_g)$	4.6×10^{-18}	1.4×10^8	6.4×10^{-10}
O_3	3.5×10^{-17}	3.8×10^{12}	1.3×10^{-4}
$O(^3P)$	2.3×10^{-11}	2.6×10^5	6.0×10^{-6}
OH	2.5×10^{-11}	1.9×10^8	4.8×10^{-5}
HO_2	2.4×10^{-11}	1.0×10^{10}	0.24

[a] Data from Demerjian *et al.*[12]
[b] Data from Table 13-4.

TABLE 13-14 Estimate of Added Elastomer Manufacturing Costs[a] on National Level[b]

Product	Estimated Value of 1970 Shipments, $ million	Estimated Added Cost per Dollar of Production, mils	Total Added Cost, $ million
Passenger tires	2,352	12	28.2
Truck and bus tires	1,037	12	12.4
Other tires	679	10	6.8
Rubber belts and belting	300	4	1.2
Rubber hose and tubing	480	2	1.0
Sponge and foam-rubber goods[c]	325	—	—
Rubber floor and wall covering[c]	66	—	—
Mechanical rubber goods	1,295	2	2.6
Rubber soles and heels[c]	165	—	—
Drug and medical	100	8	0.8
Footwear	400	1	0.4
Other rubber products	650	1	0.7
Total	7,849		54.1

[a] Extra costs due to special compounding or formulation to protect against oxidant damage.
[b] Derived from Mueller and Stickney.[28]
[c] No protective compounding or formulation used.

agreement was obtained between costs obtained from the questionnaire and those from estimates of individual compounding costs as summarized in Table 13-15. In addition to the costs outlined in Table 13-15, other costs are associated with replacement of entire assemblies when a part fails, labor costs for replacement, etc. These costs at the retail level have been estimated by Battelle to total $226 million/yr, of which $100 million is attributed to damage of medical goods. In 1969, the cost of antiozonants in automobile tires[47] alone was about $100 million, which represents the cost of avoiding ozone damage to tires. The $226 million estimate does not include labor costs for actual replacement. Because antiozonants tend to discolor, they cannot be used in white-sidewall tires, which use, instead, such costly elastomers as Hypalon and EPDM. It is of interest that tires, hoses, and belts do not represent the only important use of rubber in automobiles. Some $50 per car, or $500 million/yr, in other rubber parts represents the value at the manufacturer level. The cost of ozone damage is summarized in Table 13-16. An ap-

TABLE 13-15 Summary of Added Costs at Manufacturers' Level[a]

Type of Cost	Cost, $ million
Special polymers	20.6
Antiozonant	34.1
Wax	5.0
Protective finishes	
Wrapping	26.0[b]
Compound development	
Total	85.7

[a] Derived from Mueller and Stickney.[28]
[b] Rough estimate.

TABLE 13-16 Summary of Ozone Damage to Elastomers, Cost per Year (1969)

Type of Cost	Mfr. Level Cost, $ million	Labor Factor	Retail Cost, $ million
Special formulations	85.7	3.0	257
Early replacements	225.7	1.5	339
Total			596

proximate 25% rise in rubber production was expected between 1969 and 1975. Therefore, the cost of ozone damage to elastomers in 1975 should be 1.25 × 596 = ~$750 million.

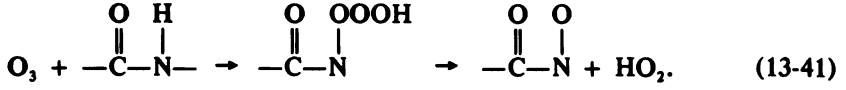
Textile Fibers

It is difficult to find any definitive information that indicates ozone damage as extensive as that suggested in the MRI report.[35] The latter identified cotton, nylon, and rayon as particularly susceptible to ozone. The oxidation of cellulose fibers by ozone was the subject of a study[6] in 1952 that showed that dry cotton was not seriously degraded by ambient ozone. In more recent studies, these conclusions were confirmed.[25,27] The only information available on fibers other than cotton addressed the effect of ozone on modacrylic, acrylic, Nylon 66, and polyester fabrics.[52] The results indicated minimal effects on these fibers.

However, Jellinek and Chaudhuri[21] exposed Nylon 66 films to nitrogen dioxide, ozone, and ultraviolet radiation. The degree of degradation of polymer α was measured by

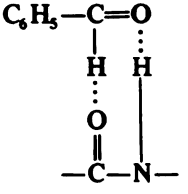
$$\alpha = (1/L_t) - (1/L_0), \tag{13-40}$$

where L_0 and L_t are the number-average chain lengths at times 0 and t, respectively. Degradation proceeds rapidly at 19.2 ppm and stops at around $\alpha = 0.002$ after 1 h. Lower concentrations produce lower ultimate degrees of degradation after longer times (see Figure 13-3). The mechanism of the degradation is believed to be attack at the N-H bond:



(13-41)

Protection of the peptide link is afforded by the addition of hydrogen-bonding compounds, such as benzaldehyde:



The overall evidence of ozone damage to fibers is not compelling.

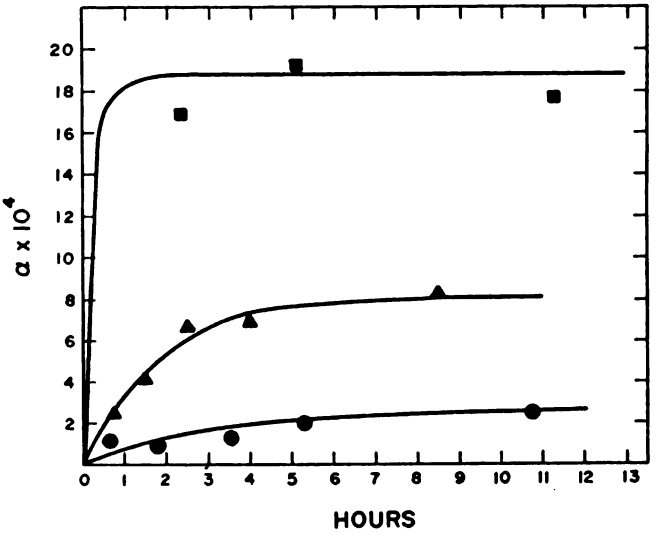


FIGURE 13-3 Plots of α vs. time for BI films at 35° C in presence of 1 atm of air containing ozone. ■, 19.2 ppm; ▲, 11 ppm; ●, 5.2 ppm. Reprinted with permission from Jellinek and Chaudhuri.[21]

Dye Fading

O-FADING

The discovery that dyes faded under the influence of ambient ozone was made by accident in 1955.[39] The dye molecule is converted by oxidation to much less deeply colored molecules. The phenomenon became known as "O-fading." Laboratory tests had shown a new dye, Disperse Blue 27, to be resistant to attack ("gas fading") by nitrogen dioxide. Field tests to confirm the laboratory findings were run on acetate draperies containing this dye in a high-nitrogen dioxide area (Pittsburgh) and a low-nitrogen dioxide area (Ames, Iowa). The draperies in both locations were protected from sunlight. The result of 12 months of exposure was opposite to that expected—the Ames draperies had faded more than the Pittsburgh draperies. Ozone was identified as the damaging pollutant, and O-fading was found to occur only when Disperse Blue 27 was used on cellulose triacetate and polyester, but not with acrylic and nylon. The dyes most susceptible to O-fading were found to be anthraquinone blues, some reds, and some azo reds. Gas-fading inhibitors (such antioxidants as diphenylethylenediamine and *tert*-butylhydro-

quinone) are used to retard fading. The Committee on Colorfastness of Textiles to Atmospheric Contaminants (RA-33) of the American Association of Textile Chemists and Colorists later conducted extensive tests,[38] and these tests eventually led to the discovery of fading problems due to the effect of ozone on permanent-press fabrics and on nylon carpets.[49] Other very extensive tests on dye fading by air pollutants have been carried out under the auspices of the AATCC Committee RA-33, the EPA,[4,5] and others.[13]

Test procedures established by Committee RA-33 include a test ribbon intended to characterize the ozone content of air to which fabrics are exposed. The test ribbon is dyed to the tertiary gray shade with CI Disperse Blue 27. Committee RA-33 has also established a test ribbon for nitrogen dioxide gas fading.

FADING OF PERMANENT-PRESS FABRICS

Permanent-press garments are blends of polyester and cotton in a ratio of 50:50 or 65:35. The permanent-press formulation includes a catalyst (zinc nitrate or magnesium chloride), a softener (polyethylene), a nonionic emulsifying agent, and a wetting agent. The anthraquinone blues react with the magnesium chloride catalyst to form a chelate that is soluble in the polyethylene softener and in the emulsifying agent, both of which are contained in the "finish." In the curing process (at 320–340° F, or 160–171° C), disperse dyes (e.g., anthraquinone blues) migrate to the "finish"—i.e., migrate preferentially to the folds and creases. The dye in this substrate, at folds, fades under the influence of ozone. Storage of garments in the summer in warehouses with open windows has resulted in fading in as few as 10 days.[37] Remedial measures include replacing anthraquinone dyes with azo disperse dyes, avoiding the use of magnesium chloride catalyst, and using different surfactants and softeners.

NYLON CARPETING

Consumer complaints of fading nylon carpeting in the warm, humid areas of Texas and Florida gave rise to the term "Gulf Coast fading."[36,37] Laboratory experiments showed the fading to be due to a combination of ozone and high relative humidity (above 65%). It was found that fading was reduced on nylon fibers textured by dry heat, rather than by steam, which produces a moisture-absorbing fiber structure. Such a structure encourages diffusion of ozone throughout the fiber. Measures have been taken to mitigate Gulf Coast fading, and it is no longer considered to be a serious problem.

ECONOMIC EFFECTS

Although measures can be and have been introduced to eliminate dye fading due to ambient ozone, these measures are expensive and represent a minimal cost of pollution damage that would otherwise be sustained.

Oppenheimer[31] and Waddell[50] attribute the preliminary economic summary in Table 13-17 to Salvin. These figures are based on increased costs of fade-resistant dyes, inhibitors, research, and quality control. Also included are costs to consumers of decreased product life. Waddell concluded that the "best" estimate of damage to materials for *all* air pollutants is about $2.2 billion per year.

Other Materials

A number of rather tenuous arguments suggest ozone damage to such materials as recording tape,[41] asphalt,[45] and dried milk.[26] However, studies on such subjects are scattered and generally uncorroborated. Some studies even discuss *beneficial* effects of ozone, such as reduction of corrosion rates in steel[20] and improvement in adhesion of ink to polyethylene films.

Polyethylene is a major electric insulating material, and the suggestion that ozone may "disastrously" affect its insulating properties[17] bears examination.

Laboratory studies carried out by Priest and his co-workers[23,24,32] have demonstrated by means of infrared and other techniques that terminal double bonds in polyethylene end groups are attacked by "ozonized" oxygen to form carboxylic acid groups and by rupture of

TABLE 13-17 Cost of Dye Fading, 1970[a]

Pollutant	Material	Cost, $ million
NO_x	Acetates	73
	Rayon	22
	Cotton	22
	Spandex	5
	Subtotal	122
Ozone	Acetates	25
	Nylon carpets	42
	Permanent press	17
	Subtotal	84

[a] Derived from Waddell.[50]

the polymer chain to produce short-chain dicarboxylic acids. A net gain in weight results.

Razumovskii and his colleagues[33] appear not to have been aware of the work of Priest *et al.*[23,32] and prefer to interpret their own results as supporting a mechanism that involves attack of ozone on the CH_2—CH_2 unit in polyethylene. Rate constants for the reaction have not been measured, and no assessment of the role of other oxidizing species has been made. However, it is presumed that a high-pressure ozonizer was used and that ozone was the active species.

It is known that atomic oxygen reacts with polyethylene at room temperature[34] to produce a loss in weight and some morphologic changes. The work of Trozzolo and Winslow[46] and of Kaplan and Kelleher[22] suggests that $O_2(^1\Delta_g)$ also interacts with polyethylene to form hydroperoxides. Because polyethylene is known to be very stable to ground-state ($^3\Sigma_g^-$) molecular oxygen, the possible oxidants responsible for polyethylene damage in polluted air are atomic oxygen, $O_2(^1\Delta_g)$, O_3, OH, and HO_2. Relative rates of reaction are products of rate constants and concentrations of oxidizing species in the real atmosphere. Thus, the relative rates of reaction of these species with polyethylene could be assessed if we had rate constants for their reactions with polyethylene. Because we do not, we can assume that the rate constants parallel those of the reactions with *trans*-2-butene (see Table 13-13). We arrived at the attack rates shown in Table 13-13.

These results suggest that HO_2 may be the dominant oxidant that attacks polyethylene or other materials in ambient air.

However, despite the known interactions of oxidants with polyethylene and other polyolefins to form intermediate peroxy radicals,[33] there is no evidence that the chemical reactions go far beyond the surface. In fact, polyethylene is specially treated with the products of a corona discharge to improve the surface adhesion of printing ink.[19] F. H. Winslow (personal communication) believes that the effects of atmospheric ozone on polyethylene insulation and other polyethylene products are negligible, compared with the embrittlement of polyethylene by a combination of oxygen and solar ultraviolet radiation. The mechanisms by which this embrittlement occurs probably involve sensitization to oxidation by absorption of ultraviolet radiation, by residual hydroperoxy and carbonyl groups in the polymer, and by surface deposits of aromatic sensitizers from polluted air. Deterioration of the electric insulating properties of polyethylene[17] by oxidation in some environments cannot be attributed to ozone. Damage to polyethylene by ozone suggested by the numbers in Table 13-5 is undoubtedly overestimated.

In view of the relative stability of polyethylene toward ozone, it is

surprising that the perfluorinated analogue (Teflon) reacts with ozone to produce perfluoroformaldehyde and carbon dioxide.[11] Because Teflon is widely used to contain ozone-air mixtures, researchers are cautioned to be aware of this reaction.

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14

General Summary and Conclusions

CHEMICAL ORIGIN

The major primary pollutants of importance to oxidant formation are nitric oxide, hydrocarbons, aldehydes, and carbon monoxide. A few free radicals are formed by photolysis of aldehydes and nitrous acid by sunlight or by the reaction of traces of ozone with reactive hydrocarbons. These free radicals initiate chain reactions involving hydroperoxy and alkylperoxy radicals. During these chain reactions, the nitric oxide is converted to nitrogen dioxide, and the hydrocarbons and aldehydes are degraded. The photolysis of nitrogen dioxide by sunlight forms a free oxygen atom, which combines with an oxygen molecule to form ozone. Because of the NO-NO_2-O_3 cycle (Reactions 2-1 through 2-3), the ozone concentration is determined primarily by the ratio $[NO_2]$:$[NO]$ and so does not become large until most of the nitric oxide has been converted to nitrogen dioxide. The total amount of oxidant formed depends, in a nonlinear fashion, on the amount of hydrocarbons available to continue pumping the nitric oxide to nitrogen dioxide. Aldehydes and even carbon monoxide can also serve this pumping function. When some of the peroxy radicals recombine or react with the nitrogen oxides, many secondary products, such as hydrogen peroxide and PAN, are formed.

Recent chemical modeling studies have been reasonably successful in reproducing the concentration-time histories of smog-chamber experiments. An examination of these models shows a need for much more detailed chemical knowledge. Modeling studies also point out the necessity of carefully defining the initial conditions of smog-chamber experiments. Some observations that have been made with these models are:

- Even if hydrocarbons are completely removed from the air, aldehydes and NO_x can generate high concentrations of photochemical oxidants.
- If both hydrocarbons and aldehydes are eliminated, carbon monoxide and NO_x alone can generate significant concentrations of ozone.
- The concentration of ozone generated photochemically goes through a maximum as the NO_x concentration is increased.
- The steady-state concentration of free radicals in smog is approximately 0.3 ppb and is rather insensitive to primary-pollutant concentration.

The concentrations, average lifetimes, and rates of attack of the reactive intermediates can be calculated with chemical models.

The effects of free radicals on biologic surfaces cannot be ignored.

The development of lasers has opened up several new techniques for monitoring pollutants in the atmosphere. Sensitivities down to the parts-per-billion range are claimed, and continuous monitoring is possible. The photoionization mass spectrometer has been developed as a sensitive detector for free radicals in the gas phase. A high-resolution mass spectrometer coupled to a computer is capable of detecting up to 300 compounds in air, both in particulate form and in the gas phase.

AEROSOLS

Review of the literature provides ample evidence that aerosol formation is an important part of the atmospheric chemistry linked with photochemical-oxidant production. The important chemical constituents of concern include sulfate, nitrate, and secondary organic material.

Secondary organic aerosols—formed by gas-phase reaction between nitrogen oxide, ozone, and hydrocarbons—constitute an important fraction of urban photochemical smog. Data obtained at high ozone concentrations (0.67 ppm) can be taken as an upper limit of the contribution of secondary organic aerosols to the organic aerosol fraction and total

suspended particulate material (95% and 65%, respectively). Most of the identified ambient secondary organic aerosols are difunctional compounds that bear carboxylic, nitrate, aldehyde, and alcohol groups. The same compounds have been identified in smog chambers from C_{5+} cyclic olefins and diolefins, with gas-to-aerosol conversion factors exceeding by more than an order of magnitude those measured for the ambient average conversion of all reactive hydrocarbons. The formation of such species in the gas phase in excess of their saturation concentration followed by condensation on pre-existing particles and further growth in the light-scattering range is the predominant physical mechanism that controls the gas-to-aerosol conversion process.

Because of their very low vapor pressures, difunctional compounds are readily converted to the aerosol phase, whereas more volatile monofunctional compounds require much higher precursor and ozone concentrations to reach their saturation concentration. This explains why most of the compounds formed from alkenes remain in the gas phase, whereas C_{5+} cyclic olefins and diolefins are efficient aerosol precursors. However, there is no known source of the latter class, so cyclic olefins, identified in both gasolines and auto exhaust, can be regarded as the most important source of secondary organic aerosols. The role of aromatics as aerosol precursors is essentially unknown. Because of their accumulation in the submicrometer range, all secondary organics are potentially dangerous. However, there is almost no information on health effects associated with the presence of such compounds in the atmosphere.

Because the conclusions on aerosols rely heavily on a few recent studies, it is extremely difficult to relate the urban concentrations of secondary aerosols to the concentration of their gas-phase precursors. Simple relations of the type $d(\text{secondary aerosol})/dt = \alpha(\text{precursor})(\text{ozone})$ have been derived from smog-chamber data for organic aerosol formation in mixtures of cyclic olefins and NO_x, and for sulfate aerosol formation in mixtures of NO_x, sulfur dioxide, and C_{5-} alkenes. Such kinetic data are consistent with the organic (a few micrograms per cubic meter per hour) and sulfate (up to 13%/h) aerosol formation rates observed in photochemically polluted urban areas. More complex kinetic relations reflect certainly all the possible variations between these extreme and rather simple systems. Although control of ozone, through control of NO_x and *total* hydrocarbon emission, would obviously have a roughly proportional effect on the formation of organic aerosols, present data suggest the identification and control of a few *specific* hydrocarbon precursors as an alternative approach. The contribution of photochemical reactions involving hydrocarbons to inorganic nitrate and

sulfate aerosol formation remains to be determined. More data on the identification of hydrocarbon precursors and on the kinetics of formation, physical characteristics, and health effects of their products would ultimately permit quantifying the complex relations between secondary aerosols and ozone concentrations in urban atmospheres.

ATMOSPHERIC CONCENTRATIONS OF PHOTOCHEMICAL OXIDANTS

In comparison with previously available material on atmospheric concentrations of photochemical oxidants, we now have a far richer data base and a deeper understanding of how to interpret the reported concentrations. The recent information on hydrogen peroxide and the broader geographic coverage of measurements abroad are examples of new data that have come to light.

Subtleties in future standard-setting must consider receptor damage in terms of exposure location and time and receptor distributions and response functions. The formula for damage function points up the need for improved knowledge of spatial and temporal distribution. The use of second-to-worst hourly readings for an ambient air quality standard must give way to a specification stated in terms of a statistically defensible higher-frequency event. This will reduce substantially the uncertainty inherent in confining one's attention to the "worst case."

Long-term trends in oxidant concentration cannot be identified with nearly the degree of certainty that we might like. The data suggest a decrease in oxidant concentrations in central-city areas and an increase in downwind areas. Measurements of nonurban oxidant are exhibiting a higher frequency of violations of the ambient standard than was once believed to occur. A model of the tropospheric ozone cycle (Figure 14-1) shows typical ranges of concentrations in the upwind and downwind regions of urban complexes.

Probably the most critical question today regarding atmospheric concentrations of ozone and other photochemical oxidants is: "What fraction of the observed values in each locale is susceptible to control by anthropogenic-emission reduction?" As brought out in Chapter 4, there is one school of thought embracing the idea that nature frequently presents us with concentrations that exceed the U.S. national ambient air quality standards. The other point of view is that global background ozone concentrations do not exceed 0.05–0.06 ppm at the surface and that higher concentrations than this have anthropogenic sources.

The data presented in the literature reviewed in Chapter 4 support the

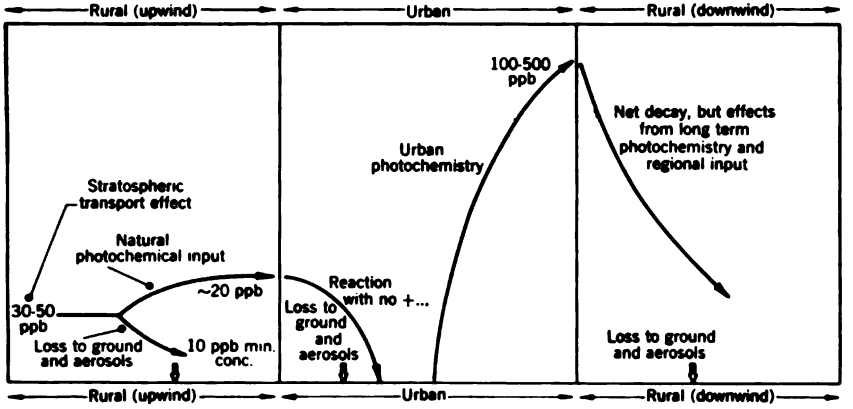


FIGURE 14-1 The tropospheric ozone cycle. Reprinted with permission from Corn *et al.*, Photochemical oxidants: Sources, sinks and strategies, J. Air Pollut. Control Assoc. 25:16–18, 1975.

second point of view. The observation of ozone concentrations exceeding the ambient standard in nonurban areas does not demonstrate that this is of natural origin. However, the measurements in remote areas of the Northern Hemisphere, compared with those in the continental United States, do support the thesis that anthropogenic sources are involved in cases where the standard is exceeded.

This is a very broad conclusion, and additional measurements must be made. Some of this effort (which is current) should address the problem of other pollutants and condensation nuclei that accompany the nonurban oxidant. Interpretation of these measurements will increase the specificity of separating anthropogenic sources from natural background sources. Theoretical assessments of the existing observations will shed light on the relative roles played by stratospheric injection, plant emission, background methane, and dry deposition on surfaces in the natural portion of the tropospheric ozone cycle.

Geographically, our best measurements have focused on the Los Angeles, California, region because of the severity of the problem there. The Regional Air Pollution Study and its extensions will, it is hoped, supply an additional rich data base for the St. Louis, Missouri, region. Airborne-pollutant measurements aimed at specific experimental objectives are needed in the central and eastern areas of the United States to broaden the foundations of a national control strategy. Existing ground-based continuous monitoring networks will not provide an adequate basis for the regional control of oxidant. *Ad hoc*, one-shot aircraft

measurements have led mainly to speculation that can establish incorrect attitudes on the origin and fate of nonurban ozone. In the vigor of environmental control efforts, incomplete data sets have stimulated hasty targeting on specific sources (for example, rural vehicular emission, power stations, trees, and frontal passages).

Another subject of recent interest has been the question of indoor-outdoor oxidant concentrations. Available measurements and models suggest that indoor exposures may be substantially reduced by appropriate choices of ventilation systems, air filters, and interior surface materials. The cost-benefit relationships coming from these studies may well have a great impact on future decisions based on atmospheric concentrations of oxidant pollutants.

Much care must be exercised in comparing atmospheric concentrations between one place and another, because of differences in primary calibration techniques or in instrumentation. Chapter 6 summarizes these problems in detail.

MODELS FOR PREDICTING AIR QUALITY

The literature contains reviews of air quality modeling that stress special purposes. Some concentrate on meteorologic aspects, and others combine this with air chemistry. Proceedings of several conferences are another information resource. Recent surveys have been addressed specifically to photochemical modeling problems. It may be concluded that, although they are relatively complex, the photochemical-diffusion models perform as well as, if not better than, available inert-species models.

A variety of goals and objectives may be met with air quality modeling:

- Scientific understanding of atmospheric phenomenology.
- Rational application of the regulatory process.
- Land-use planning within environmental constraints.
- Real-time control of episodes.

The fundamental elements of deterministic models involve a combination of chemical and meteorologic input, preprocessing with data transmission, logic that describes atmospheric processes, and concentration-field output tables or displays. In addition to deterministic models, there are statistical schemes that relate precursors (or emission) to photochemical-oxidant concentrations. Models may be classified according to time and space scales, depending on the purposes for which they are designed.

Specific model applications to the oxidant problem include both the simple rollback (with modifications) and the photochemical-diffusion techniques. Very little modeling of intermediate complexity seems to have been attempted for the oxidant system.

Model performance is now receiving critical attention because of the need for cost-effective control measures. Standard statistical performance descriptors can sometimes mislead a prospective user; therefore, more specialized tests are being devised. Various model types are being compared for a specified set of initial and boundary conditions. It is apparent from these studies that added fidelity is purchased at the expense of added complexity of a logical structure that must represent the controlling phenomenology.

MEASUREMENT METHODS

With the exception of calibration, the measurement problems that were apparent in 1970, at the time of publication of the first air quality criteria document on photochemical oxidants, have essentially been solved for ozone. This remarkable achievement is the result of unstinting efforts by people working at EPA's National Environmental Research Center, North Carolina; the National Bureau of Standards; private research contractors sponsored primarily by EPA; private instrument manufacturers; the Jet Propulsion Laboratory of the California Institute of Technology; the Air and Industrial Hygiene Laboratory, California Department of Health; the Air Pollution Research Center of the University of California at Riverside; and the California Air Resources Board (CARB).

The focus on the problem brought about by the CARB has caused a significant advance in the accurate calibration of instruments for monitoring ozone in ambient air; this was achieved during 1975. As a result, this agency adopted the measurement of ozone in the ultraviolet region at 254 nm as a primary calibration reference standard. It has also adopted statewide, as a transfer standard for calibrating ozone and oxidant monitoring instruments at air monitoring stations, a commercially available instrument (coupled with the precise controlled generation of ozone in air), which measures the differential absorption of ultraviolet radiation.

It is important to separate conceptually, and in practice, the calibration process from the monitoring process. Photochemical oxidants consisting primarily of ozone were first continuously measured in southern California by measuring the color change of potassium iodide solutions brought into contact with the ambient air. This measurement continues to yield valid photochemical-oxidant data in California. However, it has yielded questionable data at ambient air monitoring sites elsewhere in

the United States. For this reason, at the end of 1971, EPA officially adopted a continuous monitoring process that measures the chemiluminescence produced when ozone in air is brought into contact with the gas ethylene. This reference procedure when calibrated with the primary reference procedure using ultraviolet absorption is widely accepted.

Instruments based on differential ultraviolet absorption still need to be evaluated, and possibly modified, before their acceptance for *monitoring* ozone in polluted atmospheres on a nationwide scale. The CARB and other air pollution control agencies are currently conducting multiyear programs for evaluating ultraviolet absorption side by side with chemiluminescent and potassium iodide-based instruments, to determine their applicability and needed modification, as well as to ensure continuity in the data base while the older monitoring instruments are being replaced.

Thus, despite the remarkable progress in the monitoring for ozone, nitrogen oxides, and nonmethane hydrocarbons, which has strengthened the implementation and evaluation of control programs, substantial research and development are still required to help resolve the uncertainties that are inhibiting the actual achievement of desired air quality standards.

RESPIRATORY TRANSPORT AND ABSORPTION

Chapter 7 discusses the general approach required to model the transport and absorption of ozone and other pollutant gases in the respiratory tract. For unreactive or very weakly reactive gases, there are a few models that are qualitative descriptions for assessing total dosage and dosage in major regions. However, there is no adequate model for gases like ozone, which are strongly reactive within the mucus and tissue layers.

TOXICOLOGY

The acute lethal action of ozone is due to its capacity to produce pulmonary edema. The LC_{50} for rats and mice exposed for a single 4-h period is approximately 6 ppm, and cats, rabbits, guinea pigs, and dogs (in that order) are decreasingly susceptible to the lethal action. Numerous reviews have considered the toxicity of ozone at lethal and lower concentrations.

In Chapter 8 and in Table 14-1, attention is focused on studies of laboratory animals that have been exposed to ozone concentrations of

TABLE 14-1 Summary of Effects of Exposure of Laboratory Animals to Ozone at Low Concentrations*

Ozone Concentration, ppm	Duration of Exposure	Observed Effects	Animal
0.08	3 h	Increased mortality from pulmonary infection with *Streptococcus*	Mouse
0.1	7 h/day, 5 days/week for 3 weeks	Increased incidence of neonatal mortality in litters of exposed parents (related effects and reduced fertility noted in synthetic oxidant smog)	Mouse
	7 days	Increased succinate-dependent lung mitochondrial oxygen consumption in rats on diet relatively low in vitamin E; no significant effect in vitamin E-replete rats at this concentration of ozone	Rat
0.2	3 h	Degenerative changes in Type I alveolar cells; later replaced by Type II cells	Rat
	Continuous for 28–32 days	16% increase in lung volumes; overdistention at high lung volumes, suggesting some change in elasticity; no change in respiratory frequency, tail length, or external appearance	Young rat
	6 h or continuous for 7 days	Decreased voluntary runway activity during exposure; no-effect concentration not reported; reduced gross motor activity	Mouse
	7 h/day, 5 days/week for 3 weeks	Increased incidence of blepharophimosis and jaw abnormalities in neonates	Mouse
	7 days	Increased succinate-dependent lung mitochondrial oxygen consumption	Rat
	Continuous for 8 days	Dose-related increased activity of lung glutathione peroxidase and glutathione reductase (glucose-6-phosphate dehydrogenase activity significantly increased at 0.5 ppm)	Rat
	5 h	Lymphocyte chromosomal breaks	Hamster

TABLE 14-1 (Cont.)

Ozone Concentration, ppm	Duration of Exposure	Observed Effects	Animal
0.2–0.25	0.5–2 h	Increased red-cell spherocytosis after *in vitro* radiation	Mouse, rat, rabbit, man
0.25	4–6 h	Morphologic changes in medium-sized airways	Cat
0.25–0.5	6 h	Threshold for lung edema formation with ^{131}I albumin test	Rat
	3 h	Decreased lysozyme, acid phosphatase, and β-glucuronidase activity in alveolar macrophages (appears to be linearly related to dose up to 1 ppm)	Rabbit
0.26–0.5	4.6 h	Increased lung-flow resistance in 2 animals at 0.26 ppm; effect in all at 0.5 ppm	Cat
0.34	2 h	30% increase in frequency of breathing; 20% decrease in tidal volume	Guinea pig
0.37–0.5	2 h	Decreased red-cell acetylcholinesterase and increased osmotic fragility (no effect at 0.25 ppm)	Man
0.4	6 h/day, 5 days/week for 10 months	Increased serum trypsin protein esterase	Rabbit
0.4–0.7	4 h	Conjugated diene bonds, suggesting lung lipid peroxidation	Mouse
0.5	6–10 h	Minor chromosomal abnormalities	Man
	3 h	Inhibition of intracellular hydrolytic enzymes of alveolar macrophages; increased fraction of polymorphonuclear leukocytes	Rabbit
	165 min	Alterations in blood, including red-cell membrane and enzyme changes and increased serum vitamin E and lipid peroxides	Man
	6 h	Decreased lung DNA synthesis	Mouse
0.5–1	1 h	Decreased electric response of specific areas of brain with evoked-response technique	Rat

TABLE 14-1 (Cont.)

Ozone Concentration, ppm	Duration of Exposure	Observed Effects	Animal
0.54–0.88	Continuous for up to 3 weeks	Morphologic changes in distal and respiratory bronchioles, alveolar ducts, and associated alveoli	Young rat
0.6	Not applicable	Avoidance of cage ventilated with ozone	Mouse
	93 days	No change in behavior of chronaxial muscle ratios	Rat
0.68	4 h	Decreased rate of bacterial killing in lungs *in vivo*	Mouse
	2 h	No significant increase in respiratory flow resistance	Guinea pig
0.7–0.8	Continuous for 7 days	Increased acid phosphatase in specific lung areas determined histochemically	Rat
	Continuous for 5–7 days	Increased activity of lysosomal hydrolases in whole-lung homogenates	Rat
0.75	3 h	Decreased benzopyrene hydroxylase in lung and tracheobronchial mucosa	Hamster
	4–8 h	Histologic changes in parathyroid glands	Rat
0.8	Continuous for 7 days	Histochemically determined alteration in several lung enzyme activities	Rat
	Continuous for 8 days	Increased activity of lung and plasma lysozyme (no effect at 0.2 and 0.5 ppm)	Rat
	7 days	Increased activity of lung pentose shunt and glycolytic enzymes; decreased lactic dehydrogenase	Rat
0.84	4 h/day, 5 days/week for 2 weeks	Increased susceptibility to respiratory infection with *Klebsiella pneumoniae*	Mouse, hamster
0.85	4 h	Heinz bodies in circulating red cells; further exposure led to decrease in Heinz body formation	Mouse
0.9	Continuous	Lungs 38% heavier than those of normals; 50% dead in 3 weeks	Young rat

TABLE 14-1 (Cont.)

Ozone Concentration, ppm	Duration of Exposure	Observed Effects	Animal
1	1 h	Chemical changes in ground substance and lung protein	Rabbit
	4 h	Engorged blood vessels and excess leukocytes in lung capillaries	Mouse
	90 min	Decreased lung cytochrome P-450	Rabbit
	1 h	Formation of carbonyl compounds and alterations of hyaluronic acid in lung	Rabbit
	8–24 h/day for 18 months	Alteration in catechol-*O*-methyltransferase and monoamine oxidase of brain tissue	Dog
	3 h/day for 2–3 successive days	Prolongation of phenobarbital sleeping time (no effect after 1 or 4–7 days)	Mouse
	Continuous for up to 18 months	Bronchitis; bronchiolitis; emphysematous and fibrotic changes; acceleration of lung-tumor development	Mouse
	Continuous for 1 week	Decreased voluntary running activity	Rat
1–3	Continuous for 18 months	Thickening of bronchioles and respiratory bronchioles; barely noticeable at 1 ppm; at 3 ppm, formation of peribronchiolar collars with resulting narrowing of small airways	Dog
1.08	2 h	47% increase in respiratory flow resistance	Guinea pig
1.3	3 h	Increased susceptibility to *Klebsiella pneumoniae*	Mouse, hamster

*Concentrations listed are lowest for which observed effects have been reported.

about 1 ppm or less, because results of studies conducted with such concentrations are thought to be more directly relevant to ambient oxidant air pollution, which is the source of exposure for large human populations. In Table 14-1, no attempt has been made to list all studies; more comprehensive summaries can be found in some of the monographs cited

in the reference list at the end of Chapter 8. Instead, the table cites studies thought to be most useful for evaluating the health implications of exposure to low concentrations of ozone, and the concentrations listed are the lowest at which the described effects have been observed. Unfortunately, many studies have not included a sufficient range of experimental concentrations to permit construction of reliable dose-response curves or to determine what, if any, would be a "no-observed-effect" concentration for the experimental conditions used. Although it is understandable that research scientists find little stimulation in conducting exposure experiments at concentrations that fail to produce changes in the biologic system in which they are interested, the conduct (and reporting) of such experiments is extremely important for a pragmatic evaluation of the implications of positive findings for human health.

Table 14-1 illustrates the wide variety of biologic effects produced in laboratory animals exposed to relatively low concentrations of ozone. Obviously, some effects have more serious health implications than others.

CONTROLLED STUDIES ON HUMANS

Convincing new information on the health effects of oxidant exposure has emerged from controlled studies on humans, and tentative dose-response curves have been constructed. The new data show statistically significant reduction in the pulmonary function in healthy smokers and nonsmokers at ozone concentrations at and above 0.37 ppm for 2-h exposures. Other gases and aerosols found in an urban atmosphere were not present in these experiments. Some studies suggest that mixtures of sulfur dioxide and ozone at a concentration of 0.37 ppm are more active physiologically than would be expected from the behavior of the gases acting separately.

Wide variation in response among different individuals is a general finding in studies of oxidants, as well as other pollutants.

Undesirable health effects of oxidant air pollution exposure are increased by exercise, and many people apparently limit strenuous exercise voluntarily when oxidant pollution is high.

Safety, ethical, and legal considerations require that the utmost care be exercised in human experimentation. The risk inherent in this work can be minimized by taking reasonable precautions while ensuring the satisfactory performance of the study.

PLANTS AND MICROORGANISMS

Oxidant injury to vegetation was first identified in 1944 in the Los Angeles basin. Our understanding of oxidant effects and of the widespread nature of their occurrence has increased steadily since then. Although the major phytotoxic components of the oxidant (photochemical) complex are ozone and peroxyacetylnitrate (PAN), indirect data support the contention that other phytotoxicants are present in the photochemical complex. Ozone is considered the most important phytotoxic component and was first identified as the specific cause of weather fleck on tobacco and stipple on grape. PAN is associated with the undersurface glazing and bronzing associated with many of the vegetable crops.

Plant response to oxidants (including ozone and PAN) is often divided into visible and subtle effects. Visible effects are identifiable pigmented, chlorotic, and necrotic foliar patterns resulting from major physiologic disturbances. Subtle effects produce no visible injury, but include metabolic disturbances and may be measured on the basis of growth and long-term biochemical changes. These effects may influence plant populations and communities and could have an adverse influence on ecosystems. Visible injury may be acute or chronic. Acute injury breaks down the cell membrane and causes cell death, with leaf necrotic patterns that may be characteristic for a given oxidant, but can be confused with other stress factors. Classic injury from ozone is the upper-surface fleck on tobacco and the stipple of grape. Many plants show an upper-surface bleach with no lower-surface injury. Bifacial necrotic spotting is common and may appear flecklike. Classic injury from PAN in many plants appears as a glaze followed by bronzing of the lower leaf surface. Complete collapse of leaf tissue can occur, if concentrations are high. Chronic injury is associated with disruption of normal cellular activity followed by chlorosis or other color or pigment changes that may lead to cell death. Chronic injury patterns are generally not characteristic and may be confused with symptoms caused by biotic diseases, insects, nutritional disorders, or other environmental stresses. Early leaf senescence and abscission may result from chronic exposure.

Leaf stomata are the principal entry sites for ozone and PAN. Stomata, when closed by any of a number of factors, will protect plants. Ozone and PAN may interfere with various oxidative reactions in plant cells. Membrane sulfhydryl groups and unsaturated lipid components may be primary targets of oxidants. Physiologic leaf age is an important consideration in the response of the leaf to oxidants. Young leaf tissue is more sensitive to PAN, whereas newly expanding and maturing tissue is most sensitive to ozone. Light is required before plant tissue will respond to PAN, but not to ozone. Oxidants affect such physiologic

processes as photosynthesis, respiration, transpiration, stomatal opening, metabolic pools, biochemical pathways, and enzyme systems. The acute response of plants to ozone and PAN may result from a saturation of sensitive cell sites and a disruption of normal cellular repair mechanisms. Chronic injury probably results from secondary reactions involving membrane injury.

There is evidence that ozone is a radiomimetic gas. Ozone affects pollen germination in some species and thus may directly affect yield. A study with *Arabidopsis thaliana* suggested no mutagenic effects from ozone on this plant over five generations.

Ambient-oxidant studies in filtered versus nonfiltered field chambers have reported up to 50% reduction in citrus yield (orange and lemon), a 10–15% reduction in grape yield in the first year and 50–60% reductions over the following 2 yr, and a 5–29% reduction in yield of cotton lint and seed in California. Losses of 50% in some sensitive potato, tobacco, and soybean cultivars have been reported from the eastern United States. It is apparent that ambient oxidants do reduce yields of many sensitive plant cultivars. Growth reductions associated with acute exposure to ozone are often associated with injury; sometimes the correlations are high. Even multiple exposures and sometimes chronic exposures have shown fair to good correlation between injury and growth (biomass) reductions. The greater reductions in root growth than in top growth reported in several species are related to solute transport and may be fairly common under some conditions. Ozone affects nodule number, but not nodule efficiency in clover, soybean, and pinto bean. This causes a reduction in nitrogen fixation associated with legumes and, if widespread, could have a major impact on plant communities and affect fertilizer needs. The effect on nodulation is related to carbohydrate supply. Yield reductions with little injury after chronic exposure are known for several crops. Severe injury in tomato was required before a yield reduction was found. Chronic exposures to ozone at 0.05–0.15 ppm for 4–6 h/day will produce yield reductions in soybean and corn grown under field conditions. The threshold appears to be 0.05–0.10 ppm for some sensitive cultivars and is well within values monitored in the eastern United States. Growth or flowering effects, at chronic exposures to ozone at 0.05–0.15 ppm for 2–24 h/day, are reported for carnation, geranium, radish, and pinto bean grown in greenhouse chambers.

Plant sensitivity to ozone, PAN, and other oxidants is conditioned by many factors. Genetic diversity between species and between cultivars within a species is well documented. The mechanism of genetic resistance is known for only one onion cultivar and is related to the effect of ozone on stomatal closure. Variants within a natural species are well known for several pine species, including white, loblolly, and ponderosa.

Plant sensitivity to oxidants can be changed by both climatic and edaphic factors. A change in environmental conditions will initiate a change in sensitivity at once, but it will be 3–5 days before the response of the plant is completely changed. Plants generally are more sensitive when grown under short photoperiods, medium light conditions, medium temperature, high humidity, and high soil moisture. Injury due to PAN may increase with increasing light intensity. Conditions during exposure and growth affect the response of plants to oxidants in similar ways. However, plants exposed to ozone are more sensitive to increasing light intensity and, in some cases, to decreasing temperature during the exposure period. In general, growth factors that tend to cause a physiologic hardening of plant tissue make the plants more tolerant to ozone. At the time of exposure, factors that increase water stress tend to make plants more tolerant to ozone. Soil moisture is probably the most important environmental factor that affects response during the normal growing season.

Plants respond in different ways to pollutant mixtures; less than additive, additive, and greater than additive effects have been reported. Mixtures of ozone with sulfur dioxide and of nitrogen dioxide with sulfur dioxide can cause oxidantlike symptoms in some sensitive plants. Mixtures can cause effects below the threshold for either gas, although there is some disagreement here in regard to ozone. Ratios of mixtures, intermittent exposures, sequential exposures to pollutants, and predisposition by one pollutant to the effects of a second pollutant may all be important in nature, but little research has been done.

The response of some plants to oxidants is conditioned by the presence or absence of biotic pathogens. Depending on the plant and the pathogen, oxidants may cause more or less injury to a given species. Pathogens may protect their host or make it more sensitive. The pathogens themselves may be injured or may be protected by the host plant. This subject is just beginning to be understood.

Oxidant injury to ponderosa pine predisposes the trees to later invasion by pine bark beetles. Ozone and ozone–sulfur dioxide mixtures may decrease the population of soybean nematodes. Both greater and smaller effects have been noted when herbicides have been used in the presence of high oxidant concentrations.

The two most critical factors in terms of air quality standards are duration of exposure and concentration. These two factors determine the exposure dose for a plant. In determining the response of vegetation, concentration is more important than time. A given dose presented to a plant in a short period has a greater effect than the same dose applied over a longer period. This suggests a threshold effect for plant populations and is probably related to the repair mechanisms inherent in bio-

logic systems. Sufficient information is not available from long-term chronic studies, but a threshold between 0.05 and 0.10 ppm is probable for ozone (oxidant). For acute effects, an overall threshold concentration with respect to time can be determined from Figure 11-6 and Table 11-25. For pinto bean (Table 11-22), this threshold for injury is about 0.03 ppm for 8 h and about 0.10 ppm for 1 h. This suggests that oxidant standards may be needed for periods up to 8 h.

Vegetation can act as a major sink for oxidants over time, but has a relatively minor effect on oxidant concentrations during episodes of high air pollution, is more effective at some seasons or under some cultural and management practices than others, and should not be considered an important contributor to short-term reductions in oxidant or ozone concentrations.

Plant protection from air pollution stress has involved three types of programs. Several researchers are including pollutant stress in standard breeding programs with the aim of developing resistant cultivars. Our present concepts of pollution effects suggest that the gene pool of all species is large enough to permit the development of more tolerant cultivars. Natural selection will slowly do this for native vegetation. If pollution concentrations go no higher, this should be an effective protection device. Interim measures involve the use of chemical sprays. Such sprays are not yet economically feasible, but several do give adequate protection against oxidants. Fungicides, such as Benomyl, may serve a dual function. Cultural and land-use practices may also play important roles, especially on a short-term basis.

Little research on the effects of oxidants on nonvascular green plants and microorganisms has been reported. Lichens and mosses are responsive to acid gases, but there is no definite evidence that they respond to oxidants. Ferns may be especially sensitive, but their injury response is much different from that of higher plants. Growth and sporulation of fungi on surfaces are usually, but not always, affected. Ozone does not penetrate the leaf tissue or the colony and thus does not cause death of colonies. Ozone from 0.1 to several milligrams per liter of solution is required to kill many microorganisms in liquid media. Most work with microorganisms has been done to study the effectiveness of ozone as a biocide in the storage of vegetation or treatment of water or sewage supplies.

Plants have been used as biologic indicators of oxidant pollutants for many years. Attempts have been made to use plants as monitors, but too many unknown variables are involved. Plants may be capable of monitoring the total biologic potential for adverse effects, but no research has been developed along these lines.

Losses based on farm prices are not appropriate at the consumer level

and are likely to be conservative. Because of percentage markups and fixed wholesale and retail marketing costs, the cost to the consumer from agricultural losses to oxidant pollutants could be as much as $600 million per year.

ECOSYSTEMS

The transport of injurious concentrations of ozone and other oxidants to rural areas downwind from urban centers at numerous locations in the United States appears to be on the increase. Blumenthal *et al.* conservatively estimated that the urban plume from the Los Angeles area "could cause ozone concentrations to exceed the Federal standard of 0.08 ppm at locations as far as 260 km." Other areas where significant rural concentrations of oxidant have been observed are Salt Lake City, Denver, and the Blue Ridge Mountains.

In general, the permanent vegetation constituting natural ecosystems receives much greater chronic exposure, and the short-lived, higher-value vegetation constituting the agroecosystem of the Los Angeles coastal plain can be subject to injurious doses, but in intermittent short-term fumigations. Each situation has measurable economic and aesthetic effects, but on different time scales. The simple agroecosystem has little resilience to pollutant stress; losses are immediate and sometimes catastrophic. The complex natural ecosystem is initially more resistant to pollutant stress, but the longer chronic exposures cause disruption of both structure and function in the system that may be irreversible.

Simulation models of ecosystem subsystems are developing rapidly. They deal with flows of energy, biomass, mineral nutrients, water, numbers of species, population densities, and area occupied per biotic unit. Interactions between ecosystem components must be understood before prediction can be attempted. Simulation models offer a bright opportunity for determining the long-term effects on natural ecosystems and agroecosystems. New knowledge of biologic effects should suggest the importance of prevention and some means for ameliorating damage.

Oxidant injury to the mixed-conifer stands of the San Bernardino Mountains began in the early 1940's and is well advanced. A similar problem is developing in the forests of the southern Sierra Nevada. Both places show both direct and indirect effects on all subsystems of the forest ecosystem—producers, consumers, and decomposers. For example:

- Ozone injury limits biomass production by the primary producers and their capacity to reproduce.

- The decrease of biomass or energy flow to consumer and decomposer in the ecosystem affects the populations of these organisms.
- Essential recycling processes, such as recycling of nutrients, may be interrupted, further limiting primary production.
- Stand structure is altered rapidly in some areas by salvage logging of high-risk trees; as a result, species composition is changing, and wildlife habitat is being altered.

Oxidant injury to eastern white pine in some forest stands in the eastern United States is a significant problem. There is an important concern about injury caused by a synergistic reaction between ozone and sulfur dioxide at low concentrations.

The relationship between man's welfare and stable natural ecosystems and agroecosystems can be established in terms of the economic and aesthetic values derived from them. In some situations, where ecosystems are stressed by oxidant pollutants, the benefits realized by present and future generations may soon diminish. Considerable research is required to find alternatives that prevent stress or that may salvage some of these benefits. New management strategies should be instituted only when their consequences are predictable within reasonable limits.

EFFECTS OF PHOTOCHEMICAL OXIDANTS ON MATERIALS

In the context of this review, the term "photochemical oxidants" is considered to be synonymous with "ozone." In test chambers that have external ozone generators and that operate at or near atmospheric pressure, ozone is the only likely oxidizing species. In ambient air, however, ground-state atomic oxygen, hydroxyl radicals, and especially hydroperoxy radicals can compete with ozone and may even dominate in attacking materials, such as rubber, that contain olefinic bonds. The most economically important materials with respect to ozone damage are paint, elastomers (rubber), and textile fiber–dye systems. Damage to polyethylene by ozone is considered to be negligible. The ozone-specific damage to materials in 1970 has been assessed in economic terms and is approximately as follows: paint, $540 million; elastomers, $569 million; and textile fibers and dyes, $84 million. Total material damage attributable to ozone is, therefore, $1.22 billion. This is to be compared with Waddell's estimate of total air-pollutant material damage of $2.2 billion. It is clear that oxidants are very important molecules in pollutant damage to materials.

15

Recommendations for Future Research

CHEMICAL ORIGIN

- Rate constants are needed for almost all the reactions of hydroperoxy, HO_2, and alkylperoxy, RO_2, radicals.
- The homogeneous and heterogeneous reactions of the oxides of nitrogen with water vapor need study.
- The yields of free radicals from the photolysis of nitrous acid and of aldehydes should be established.
- Equation 2-4, $[O_3] = k_1[NO_2]/K_3\ [NO]$, should be tested in the real atmosphere, as well as in laboratory experiments. Simultaneous measurements of the concentrations of ozone, nitric oxide, and nitrogen dioxide and of the intensity of sunlight for a variety of conditions will provide a much-needed check on this dynamic equilibrium.
- A quantitative measure of the concentration of free radicals in smog (probably hydroxyl, OH, or hydroperoxy, HO_2) under well-defined conditions will provide an important test of present chemical models.
- Strong support for fundamental gas-phase kinetics is needed. Most of the reaction mechanisms and rate constants that are needed to construct realistic and detailed models of the polluted atmosphere are determined in laboratory studies under very special conditions, not in smog simulations at a pressure of 1 atm. Because there are still very

serious gaps in the present models, further research should be supported.

- Smog-chamber studies are needed for validating both the detailed chemical models and the lumped models. Many of the past chamber studies have not used sufficiently well-defined initial conditions. Measurements of more products and of the reactive intermediates will provide more stringent tests for models.
- Modeling studies are very useful in pointing out the important kinetic data that are lacking, in clarifying some of the past smog-chamber studies, and generally in making the very complex chemistry more comprehensible. Accurate models can make unique predictions about the polluted atmosphere. There are very useful interactions among the modeling studies, the smog-chamber experiments, and fundamental chemical kinetics; it is not possible to ignore one without hindering progress in the others.
- It seems probable that many new and unstable compounds are present in the polluted atmosphere or in smog chambers. A careful search for some of these compounds may provide some surprises.
- Promising new instrumental techniques should be supported, both for monitoring pollutants and for following reactive intermediates in kinetic studies. A reliable and accurate method of standardizing concentrations in the parts-per-billion range is needed.
- The possibility that free radicals, particularly hydroperoxy, have significant effects on biologic surfaces exposed to the irradiated atmosphere should be investigated. Sticking coefficients are needed. In experiments in which the observed biologic effects cannot be attributed to the measured ozone and PAN concentrations, the possibility of damage by the steady-state concentrations of free radicals in the atmosphere should be considered.

AEROSOLS

Our present knowledge of the chemical and physical processes that govern aerosol formation in the atmosphere is rather limited, and further studies are needed in most of the relevant areas of research. This may leave the reader—and the decision-maker—with a feeling of endlessness. However, substantial improvements could be made in a reasonable period by focusing research efforts on the subjects most directly involved:

- *Laboratory (smog-chamber) studies of aerosol formation from aromatic hydrocarbons:* gas-phase reaction mechanism, physical processes controlling gas-to-aerosol conversion, kinetic data on aerosol formation

and aerosol growth, identification of the aerosol products, and effect of hydrocarbon concentration on aerosol formation (threshold).

- *Careful search, in the atmosphere, for aerosol precursors,* such as cyclic olefins and C_{6+} alkenes.
- Study of the possible *health effects of exposure to difunctional oxygenated organics* (such as dicarboxylic acids) that are present in urban aerosols.
- *Identification of organic components of ambient aerosols,* to permit estimation of the relative importance of olefinic and aromatic hydrocarbons as *aerosol precursors.*
- Estimation of the relative *contributions* of *photochemical* and *nonphotochemical pathways* to the formation of *inorganic* nitrate and sulfate aerosols.

Identification of organic components of ambient aerosols and estimation of the contributions of various pathways are of immediate interest for control strategies and could by achieved by using the existing monitoring networks so as to provide more information on aerosol chemical composition. In view of the adverse effects (e.g., on health and visibility) associated with submicrometer aerosols, an air quality standard for submicrometer particles might be more adequate than the present standard for total suspended particles.

ATMOSPHERIC CONCENTRATIONS OF PHOTOCHEMICAL OXIDANTS

- Nonmethane hydrocarbons and both oxides of nitrogen should be monitored concurrently whenever photochemical oxidant or ozone is monitored.
- Photochemical-oxidant monitoring stations should be sited upwind and downwind from urban areas, as well as within those urban areas, wherever possible.
- A common primary calibration standard should be established for all monitoring networks.
- Documentation should be provided in each case to outline the rationale for location and design of monitoring stations and the rationale for data validation for photochemical oxidants.
- A clear indication of what constitutes background concentrations of photochemical oxidants and ozone must be made, in order to form the basis of emission control programs.

- The results of monitoring data must be generalized, in order to relate air quality to emission in a stochastic fashion.

MODELS FOR PREDICTING AIR QUALITY

Internal improvements in deterministic methods will be based on accounting for more physicochemical effects in the logical structure. One challenge to the researcher is to do this without making something that is already complex still more difficult to understand, and another challenge is to avoid needless elaboration of detail. Both pitfalls will be avoided, first, by asking how accurate a modeling job is demanded and, second, by carrying out order-of-magnitude analytic appraisals of the omitted phenomenology.

Perhaps the most important thing that research will contribute is a set of criteria delineating the fidelity of existing models, rather than a single supermodel that will consider all effects. Much remains to be done in statistical modeling. The scientific community is on the threshold of potentially great strides with these methods, because of the veritable explosion of data from measurement programs. It is absolutely essential for all agencies interested in environmental management to begin mounting analysis programs that are carefully designed to capitalize on the data base. Traditionally, support has been more readily obtained for making additional measurements in the hope that useful information would emerge directly or that someone would spontaneously dig out the useful information. Seldom has either been the case.

Specific research subjects have emerged with respect to improved descriptions of specific phenomena. Some time ago, it was speculated that gas-solid interactions and turbulence effects on reaction kinetics would be important areas of advance in the modeling art. Gas-solid interactions include both chemical formation of aerosols and reactions on surfaces of pre-existing suspended particulate matter. Because of differing effects of a material in the gas phase and in some condensed phase, it will be important to characterize transformation processes. The ACHEX (*A*erosol *CH*aracterization *EX*periment) program recently carried out under the direction of Hidy will provide an extensive data base with which to test new ways of treating the gas-solid interaction problem.

The turbulent mixing of emitted reactant gas (such as nitric oxide) with atmospherically formed reactant gas (such as ozone) results in macroscopic heterogeneities, which under some circumstances can significantly change the reaction rate from the value that the mean concen-

trations used in a rate equation would predict. Airborne measurement from some 40 operational days from the LARPP (*Los Angeles Reactive Pollutant Program*) study gives 6-s-interval gas-phase data for six gas-phase species simultaneously. This program (under the field management of W. Perkins and under the direction of the Coordinating Research Council's CAPA-12 committee, chaired by J. Black) has produced archives of these data that can serve as a test bed for theories of turbulent interactions with kinetics.

In a broader sense, the data obtained from the Regional Air Pollution Study (RAPS) and the California Three-Dimensional Pollutant Gradient Study Program should also serve as bases of further model development. It is incumbent on the agencies responsible for air quality control to identify resources specifically aimed at using these data for improving techniques for designing pollution-abatement strategies.

Without doubt, the top-priority application of air quality models is the determination of emission controls needed to achieve ambient air quality standards. With the re-examination of transportation control strategies and with the pressures of fuel substitutions, refinements well beyond the traditional proportional models are imperative. Where validated diffusion models are available, they should be used to recalculate the emission requirements that came from initial hasty efforts to implement the Clean Air Act Amendments of 1970. This is the greatest national service that could be performed by the air quality modelers at present. Before this can be achieved, however, the institutional apparatus must provide the impetus and resources called for in a recent National Academy of Sciences report to the U.S. Senate.

Much of the research work will add content to the model structures, but future applications demand simplifications that are oriented toward the nonspecialist user. One of the largest obstacles to the effective use of air quality prediction schemes is the resolution of this apparent conflict. At least two steps can be taken by those who produce models to encourage applications and to aid the user:

- Compile a catalog of air quality models that describes their capabilities in terms of a common set of performance standards.
- Clarify data communication in the input-output interfaces between user and model.

To accomplish the first step, model standards will be evolved on the basis of legislative mandates and regulatory needs. Each of the various types of model has undergone performance evaluation through the application of a set of tests peculiar to its own structure or output. For

example, Gaussian models that predict long-term averages are often evaluted by computing only the correlation coefficient between measured and computed concentrations. Early evaluations of species-mass-balance models stressed hour-by-hour comparison of the predicted and observed concentrations. Recently, a broader range of descriptors has evolved, as evidenced by the work of Nappo and Whitney.

The performance indexes must be designed with the model applications in mind. Will the model be used to predict local effects around a highway or a smelter where short-term high doses are as important as long-term averages? Will the model be called on to compare trends in air quality between two different scenarios of urban population growth? Will the model be used to select a control plan that will result in a given hourly air concentration's being exceeded only once a year? A properly designed set of performance standards will allow a potential user to compare models with respect to suitability for any specific application. The particular performance characteristic of interest influences strongly the rank-ordering of models on a scale of goodness.

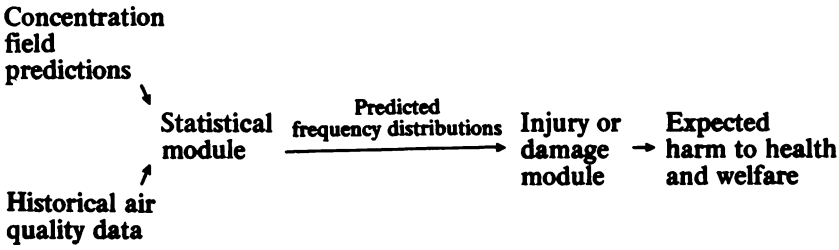
Fundamental to the definition of an optimal set of performance measures will be the relationship of risk (of health, property, or aesthetic attributes) to exposure (average pollutant concentration, time-integrated pollutant concentration, synergistic combination of pollutant dosages, or dosages integrated with respect to space, time, and population). Derived from the risk factor will be, not a single number, but a distribution of effects for each degree of exposure. For example, a range of pulmonary effects can be expected in a sample population in which each individual has been exposed to ozone at an average concentration of $100\ \mu g/m^3$ for 5 yr. The expectation value of the effect will be the risk factor that is the function of exposure described above. The model performance index will utilize these relationships to connect a probability density distribution output from the model (associated with imperfect knowledge) to a probability density distribution of the threat to public health and welfare. Stated in a different way, each model will be assessed on the basis of the uncertainty of damage estimate that arises from its imperfections. This must be done in an unambiguous way for the user, who may not be a specialist.

The second step that will be needed to ensure ready application of air quality models is largely a question of packaging and presentation. User-oriented documentation will be needed at data-processing centers for personnel who may not be specialists in chemistry, mathematics, or meteorology. Experience has shown that the user desires to operate the model in his own data center and wishes to understand enough about the model structure to explain it to others in his field. Models that can-

not be adapted to these requirements have not been widely applied. In some cases, an operating manual intended for persons with some knowledge of programing will need to be rewritten to allow the user to supply completed data forms to a computer center and routinely receive output in return. Other adaptations may require a user to punch data in on a teletypewriter and receive output on the same machine in an interactive mode. This involves a network of remote terminals served by the computer center, such as that under development in UNAMAP.

Output displays will be required to bring the abstract aspects of voluminous output data into some form that appeals to the experience of the user. Isopleth maps are useful, as are three-dimensional isometric plots like SYMVU, produced by Harvard University. Printer plots of concentration maps will undoubtedly enjoy an even greater application, because of the common availability of line printers or teletypewriters as output devices. Examples of these techniques are SYMAP and GRID, both produced by Harvard University.

Another aspect of matching output to user needs involves presentation of results in a statistical framework—namely, as frequency distributions of concentrations. The output of deterministic models is not directly suited to this task, because it provides a single sample "point" for each run. Analytic linkages can be made between observed frequency distributions and computed model results. The model output for a particular set of meteorologic conditions can be on the frequency distribution of each station for which observations are available in sufficient sample size. If the model is validated for several different points on the frequency distribution based on today's estimated emission, it can be used to fit a distribution for cases of forecast emission. The fit can be made by relating characteristics of the distribution with a specific set of model predictions. For example, the distribution could be assumed to be log-normal, with a mean and standard deviation each determined by its own function of output concentrations computed for a standardized set of meteorologic conditions. This, in turn, can be linked to some effect on people or property that is defined in terms of the predicted concentration statistics. The diagram below illustrates this process:



We have seen the wide variety of methods now available to calculate air quality. The priority for adapting these methods to current needs is clearly established. Only through clear expositions of model performance and simple implementation procedures will the present techniques have a favorable impact on air quality management. A growing appreciation by the specialist community of the policy requirements will be essential for the successful fulfillment of these goals.

MEASUREMENT METHODS

Instruments based on differential ultraviolet absorption still need to be evaluated, and possibly modified, before their acceptance for *monitoring* ozone in polluted atmospheres on a nationwide scale. The California Air Resources Board and other air pollution control agencies are currently conducting multiyear programs for evaluating ultraviolet absorption side by side with chemiluminescent and potassium iodide-based instruments, to determine their applicability and needed modification, as well as to ensure continuity in the data base while the older monitoring instruments are being replaced.

Thus, despite the remarkable progress in monitoring for ozone, nitrogen oxides, and nonmethane hydrocarbons, which has strengthened the implementation and evaluation of control programs, substantial research and development are still required to help resolve the uncertainties in our knowledge that are inhibiting the actual achievement of desired air quality standards.

The areas in which further research and development are needed, in sequence of priority, are:

- Evaluation of primary calibration procedures applicable nationwide for ozone measurement.
- Development principles and instruments that can easily track the sources of hydrocarbons that are reactive in the production of ozone and hydrocarbons that are reactive in the production of particles.
- Chemical identification of both gas- and particle-phase compounds occurring in the atmosphere that cause eye irritation and respiratory difficulties.
- Methods for the direct and continual measurement of chemicals in the particles of the atmospheric haze that are known to be formed during photochemical-pollution episodes and are already suspect as respiratory irritants. By implementing such measurements, it will be possible to find out to what extent the occurrence of such substances can be reduced by various emission controls. To assess actual population expo-

sures, it is also necessary that these measurement methods be easily carried out indoors and in vehicles.

- Improved measurement methods suitable for observations from airborne platforms, so that the regional-scale impacts of urban emission can be accurately assessed. This is needed because some control options for solving the urban-scale problem have the potential of transferring pollution from one geographic area to others.

RESPIRATORY TRANSPORT AND ABSORPTION

The development of models requires more knowledge about the chemical, physical, morphologic, and flow properties of the mucus layer; the kinetics of the reactions of ozone in the mucus and tissue layers; and the molecular diffusivity of ozone in these layers. Similar information is needed for the hydroperoxy and singlet oxygen, O_2 ($a^1\Delta$), free radicals, which are reactive intermediates in photochemical smog.

Furthermore, a realistic model based on such knowledge needs to be verified by measurements of uptake and tissue dosage in the various regions of the respiratory tract. These are currently difficult to make, but are required to establish accuracy and reliability. New methods of sampling and techniques using tagged gases should be developed, so that local uptake can be measured.

An extensive effort is needed in studies of pollutant-gas transfer, absorption, and reaction in the respiratory tract. After some of the experimental questions about behavior of ozone in the mucus layer and adjacent tissue are answered, available methods for calculating the local dosage to critical airway sites can be used in new uptake models. Gas-absorption and particle-deposition models for the upper respiratory tract (nose, mouth, pharynx, larynx) also need to be improved. Experimental data now available can be used to develop semiempirical relations for gas uptake in the nose in a procedure analogous to that used to model particle deposition. Development of a more refined model for nonreactive gases requires data on gas diffusivities in the mucus and tissue, local blood perfusion rates in the nasal epithelium, and physiologic and pharmacologic factors affecting the mucosa and local blood flow rates. Models need to be developed for mixtures of gases that may interact chemically in the gas phase, in the mucus, or in aerosol droplets to form other species. This requires theoretical and experimental studies of dissolution, absorption, adsorption, and desorption of gases in or on aerosols in the respiratory tract.

Improved modeling is needed for the design and interpretation of

animal experiments and controlled human studies, and for the collation of diverse data from animal and human exposures to ozone. Calculations of local dose at reactive tissue sites can help to explain the mechanisms of toxicity and are needed to extrapolate animal and human data for assessing population risks under different environmental conditions.

TOXICOLOGY

Enhanced susceptibility to respiratory exposure to infectious agents is of considerable potential public-health significance. This has been reported to occur in mice exposed to ozone at as low as 0.08 ppm, the lowest reported "effect" concentration in laboratory animal studies. It seems essential, therefore, to continue and expand research on the effect of ozone and other photochemical oxidants on physiologic protective mechanisms of the lung. Appropriate dose-response studies should be included to confirm or establish the exposure concentration-time relationships that result in increased susceptibility to inhaled microorganisms, and studies of the cellular responses and mechanisms should be conducted with a view to providing methods that are applicable to epidemiologic studies in oxidant-exposed human populations.

Research designed to elucidate the pathophysiologic implications of reversible changes in lung function, histology, and biochemistry that have been observed at concentrations of 0.2-0.5 ppm would be especially useful in evaluating the significance of these changes for health. In particular, it would be useful to determine what, if any, causal or correlative relationships exist between different effects detected by various experimental assay systems and between reversible changes and more chronic effects, such as reduced lung elasticity, fibrosis, and adenoma formation. Studies on laboratory animals are particularly suited to this type of mechanism-correlative research; but, with appropriate experimental designs, such research should be of value in determining which methods or biologic changes can be usefully applied to clinical or epidemiologic studies. At the same time, such research could determine whether some of the changes (e.g., increased activity of enzymes involved in cell redox systems) are indicative of injury or are homeostatic adaptive responses. This information will have value in predicting potential injury in the "average" population and possibly help to identify people who are hypersusceptible by virtue of some deficiency in adaptive protective mechanisms. Related to these research efforts, further investigation of the influence of oxidant exposure on enzymes that biotransform other inhaled chemicals (e.g., aromatic hydrocarbons) and the interaction effect of dietary anti-

oxidants (e.g., vitamin E) should be pursued, particularly in relation to oxidant-induced oxidation of membrane lipids and free-radical formation.

There are several scattered reports of extrapulmonary effects of ozone exposure in laboratory animals. Some (e.g., chromosomal aberrations in hamster lymphocytes) occur at or near concentrations that cause local effects in the lung and portend serious and long-term health implications. Others (e.g., reduction of voluntary activity) may be transient or reversible, but nevertheless contribute to decrements in performance or well-being. It is particularly important that further research be conducted to confirm (or refute) these reported extrapulmonary actions of ozone and the exposure concentrations at which they occur. If confirmation of their occurrence is obtained at or below the currently reported effective exposure concentrations, useful research efforts could be directed toward determining whether they are direct or secondary effects and toward identifying the chemical species responsible. More importantly, confirmation of an isolated effect on cell genetic material would demand a thorough expert evaluation of its significance on the basis of existing knowledge of the long-term implications of the effect and, accordingly, intensified laboratory and epidemiologic research.

CONTROLLED STUDIES ON HUMANS

Further studies are needed to give better dose-response information and to provide a frequency distribution of the population response to oxidants alone and in combination with other pollutants at various concentrations. Such studies should include the effects of mixed pollutants over ranges corresponding to the ambient atmosphere. With combinations of ozone and sulfur dioxide, the mixture should be carefully characterized to be sure of the effects of trace pollutants on sulfate aerosol formation. The design of such studies should consider the nced to use the information for cost-benefit analysis and for extrapolation from animals to humans and from small groups of humans to populations. Recent research has indicated the possibility of human adaptation to chronic exposure to oxidants. Further study is desirable.

Studies are needed to clarify the importance of age, sex, ethnicity, familial elements, nutritional factors, and pharmacologic agents in determining response to oxidants. Because people with lung disease are thought to be more susceptible to oxidant pollutants, exposure studies are needed to quantify this. Better methods for measuring or estimating the actual dose of oxidants absorbed by each subject are needed. The usual time variation in measures of human response should be evaluated

per se, because this information is needed to optimize experimental design.

More information is needed before rational guidance can be given about limiting exercise during periods of high-oxidant pollution.

Standards for the exposures of humans to controlled atmospheres should be discussed by national groups and agencies, such as the American Medical Association and the National Institutes of Health.

EPIDEMIOLOGIC STUDIES

Modification of the methods in which the CHESS studies are designed and in which the data are displayed would add considerably to their value. The scientific data collected in all the CHESS studies should be made available through accepted scientific publications to the scientific community as a whole.

The continuation of epidemiologic studies, including those of the CHESS program, is vital to our understanding of the effects of air pollution on health. There is no other way to determine the needed dose-response (exposure-response) relationships between the complex urban atmospheres and specific health effects. No animal or clinical experiment can duplicate the full range of variables to be found in ambient urban air. For this reason, all necessary support should be given to qualified scientists to conduct epidemiologic studies designed to answer these questions. Although such studies are expensive and time-consuming, the data they produce can be produced in no other way and are essential in the development of useful air quality standards.

It is important to know whether such phenomena as tolerance and cross-protection, well demonstrated or suggested in animals, occur in man. Analogues for all these phenomena should be sought in human populations, and methods should be devised for assessing their significance for human health.

PLANTS AND MICROORGANISMS

These recommendations are not listed in a priority order, but many could be followed in parallel or simultaneously. In general, the recommendations presented by Heck *et al.* are still germane to research needs with respect to oxidant pollutants.

A few definitive experimental designs are needed to further our knowledge of acute dose-response information on ozone. Much of this

type of information is still needed for PAN and its analogues. All experimental designs should incorporate dose-response.

Studies to develop dose-response curves for chronic exposures of crop and native species over growing seasons and under field conditions are needed for ozone, PAN, and other oxidants.

Research should be continued with filtered and nonfiltered field chambers to study effects of ambient oxidants on important agronomic, horticultural, and native species. We know that there is a problem, but its significance and magnitude are matters of conjecture.

There is a critical need to understand the interaction of multiple pollutants on individual plant species and ecosystems. Multiple-pollutant effects are generally important, but little is known of their effects on most plants. Variable concentrations, ratios of pollutants, and age of plants all affect response.

Models should be developed to understand the relative importance of other variables as they affect plant dose-response. These include, but are not limited to, climatic, edaphic, biotic, and genetic factors. Considerable information is available, but there are many gaps, and no comprehensive programs are in progress to determine how these factors act and interact to affect a plant's response.

The mechanism of response and the biochemical systems affected are not understood. Although plant membranes are considered the primary sites of action for the oxidant pollutants, there is no definitive work on this. An understanding of these responses would be supportive of breeding and spray protective programs. Both breeding and spray protective programs need to be developed, so that better-yielding and better-quality cultivars will be protected against oxidant pollutants.

Some effort is needed to explore the feasibility of using plants to monitor the overall biologic activity (or biomass reductions) caused by photochemical oxidants in specific air basins or regions. The response of sensitive plants should be correlated with the response of plants of economic and aesthetic importance. Additional monitoring of multiple pollutants is needed in rural areas.

Whenever possible, measures that vary on a continuous scale (e.g., biomass) should be used with subjective estimates of injury (e.g., indexes of visible injury).

ECOSYSTEMS

The present knowledge of the biologic consequences of chronic oxidant injury to both natural ecosystems and agroecosystems must be communi-

cated to groups in the public sector that work in planning and enforcement. The indirect effects on man's health and the direct effects on his welfare resulting from ecosystem deterioration due to oxidant injury are serious enough to be given more thorough consideration in all decisions related to abatement of air pollution from both mobile and stationary sources. Land-use planning and proper airshed classification should be used to prevent further deterioration of air quality, particularly in prime timber-producing areas and in remote, pristine areas, regardless of their present use designation.

The most important research needs are related to the determination of the responses of natural ecosystems and agroecosystems to chronic exposure to oxidant pollutants. In particular, chronic-dose-response models are needed to understand the responses of the dominant primary-producer species constituting forest ecosystems in both the eastern and the western United States. The resulting alteration of interactions with other subsystems—e.g., consumers and decomposers—must also be investigated.

EFFECTS OF PHOTOCHEMICAL OXIDANTS ON MATERIALS

- Laboratory studies of effects of photochemical oxidants other than ozone—e.g., PAN, peroxybenzoylnitrate, atomic oxygen, excited molecular oxygen, $O_2(^1\Delta_g)$, and hydroperoxy and hydroxyl radicals—on specific materials should be conducted.
- Methods to measure concentrations of such transient oxidants as hydroxyl and hydroperoxy radicals and $O_2(^1O_g)$ in real atmospheres should be developed.
- Mechanisms of attack of oxidants on materials should be investigated, so that distinctions can be made between effects on a given material due to various pollutants.
- An integrated study should be conducted in which relative effects of all major air pollutants on materials are assessed.

Index

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