AIR QUALITY CRITERIA
FOR
PHOTOCHEMICAL OXIDANTS
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PHOTOCHEMICAL OXIDANTS

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE

Public Health Service
Environmental Health Service
National Air Pollution Control Administration
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PREFACE

Air quality criteria tell us what science has thus far been able to measure of the obvious as well as the insidious effects of air pollution on man and his environment. Such criteria provide the most realistic basis that we presently have for determining to what point pollution levels must be reduced if we are to protect the public health and welfare.

The criteria we can issue at the present time do not tell us all that we would like to know; but taking all of man’s previous experience in evaluating environmental hazards as a guide, we can conclude that improved knowledge will show that there are identifiable health and welfare hazards associated with air pollution levels that were previously thought to be innocuous. As our scientific knowledge grows, air quality criteria will have to be reviewed and, in all probability, revised. The Congress has made it clear, however, that we are expected, without delay, to make the most effective use of the knowledge we now have.

The 1967 amendments to the Clean Air Act require that the Secretary of Health, Education, and Welfare “...from time to time, but as soon as practicable, develop and issue to the States such criteria of air quality as in his judgment may be requisite for the protection of the public health and welfare.... Such criteria shall...reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on health and welfare which may be expected from the presence of an air pollution agent....”

Under the Act, the issuance of air quality criteria is a vital step in a program designed to assist the States in taking responsible technological, social, and political action to protect the public from the adverse effects of air pollution.

Briefly, the Act calls for the Secretary of Health, Education, and Welfare to define the broad atmospheric areas of the Nation in which climate, meteorology, and topography, all of which influence the capacity of air to dilute and disperse pollution, are generally homogeneous.

Further, the Act requires the Secretary to define those geographical regions in the country where air pollution is a problem—whether interstate or intrastate. These air quality control regions will be designated on the basis of meteorological, social, and political factors which suggest that a group of communities should be treated as a unit for setting limitations on concentrations of atmospheric pollutants. Concurrently, the Secretary is required to issue air quality criteria for those pollutants he believes may be harmful to health or welfare, and to publish related information on the techniques which can be employed to control the sources of those pollutants.

Once these steps have been taken for any region, and for any pollutant or combination of pollutants, then the State or States responsible for the designated region are on notice to develop ambient air quality standards applicable to the region for the pollutants involved, and to develop plans of action for implementing the standards.

The Department of Health, Education, and Welfare will review, evaluate, and approve these standards and plans and, once they are approved, the States will be expected to take action to control pollution sources in the manner outlined in their plans.

At the direction of the Secretary, the
National Air Pollution Control Administration has established appropriate programs to carry out the several Federal responsibilities specified in the legislation. Previously, on February 11, 1969, air quality criteria and control techniques information were published for sulfur oxides and particulate matter.

This publication, *Air Quality Criteria for Photochemical Oxidants*, is the result of extensive and dedicated effort on the part of many persons—so many that it is not practical to name each of them.

In accordance with the Clean Air Act, a National Air Quality Criteria Advisory Committee was established, having a membership broadly representative of industry, universities, conservation interests, and all levels of government. The committee provided invaluable advice on policies and procedures under which to issue criteria, and provided major assistance in drafting this document.

With the help of the committee, expert consultants were retained to draft portions of this document, while other segments were drafted by staff members of the National Air Pollution Control Administration. After the initial drafting, there followed a sequence of review and revision by the committee, as well as by individual reviewers specially selected for their competence and expertise in the many fields of science and technology related to the problems of atmospheric pollution by photochemical oxidants. These efforts, without which this document could not have been completed successfully, are acknowledged individually on the following pages.

As also required by the 1967 amendments to the Clean Air Act, appropriate Federal departments and agencies, also listed on the following pages, were consulted prior to issuing this criteria document. A Federal consultation committee, comprising members designated by the heads of 17 departments and agencies, reviewed the document, and met with staff personnel of the National Air Pollution Control Administration to discuss their comments.

This Administration is pleased to acknowledge the efforts of each of the persons specifically named, as well as the many not named who have contributed to the publication of this volume. In the last analysis, however, the National Air Pollution Control Administration is responsible for its content.

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AIR QUALITY CRITERIA FOR PHOTOCHEMICAL OXIDANTS

Chapter 1.

INTRODUCTION

Pursuant to authority delegated to the Commissioner of the National Air Pollution Control Administration, Air Quality Criteria for Photochemical Oxidants is issued in accordance with Section 107 (b), of the Clean Air Act (42 U.S.C. 1857-18571).

Air quality criteria are an expression of the scientific knowledge of the relationship between various concentrations of pollutants in the air and their adverse effects on man and his environment. Criteria are issued to assist the states in developing air quality standards. Air quality criteria are descriptive: that is, they describe the effects that have been observed to occur when the concentration of a pollutant in the ambient air has reached or exceeded a specific level for a specific time period. In the development of criteria, many factors have to be considered. The chemical and physical characteristics of the pollutants and the techniques available for measuring these characteristics must be considered, along with exposure time and conditions of the environment. The criteria must also include consideration of the contributions of all such variables to the effects of air pollution on human health, agriculture, materials, visibility, and climate. Further, the individual characteristics of the receptor must be taken into account. Table 1–1 is a list of the major factors considered in developing criteria.

Air quality standards are prescriptive. They prescribe pollutant exposures or levels of effect that a political jurisdiction determines should not be exceeded in a specified geographic area, and are used as one of several factors in designing legally enforceable pollutant emission standards.

This document focuses on photochemical oxidants as they are found in the ambient air. In general, discussions in the earlier chapters are oriented towards the physical and chemical nature of these oxidants, atmospheric concentrations and measurement of these oxidants, and possible natural sources of ozone. The presence of photochemical oxidants in the ambient air is then considered in later chapters in relation to (1) effects on vegetation, (2) effects on materials, (3) toxicological studies of effects on animals and man, and (4) epidemiological studies.

The National Air Pollution Control Administration is currently advocating the use of the metric system to express atmospheric concentrations of air pollutants, e.g., micrograms per cubic meter (μg/m³). In most instances, gaseous pollutants have hitherto been reported on a volume ratio basis, i.e., parts per million (ppm). In this document, whenever possible, both types of units are given. Conversion from volume (ppm) to mass (μg/m³) units requires a knowledge of the gas density at the temperature and pressure of measurement, since gas density varies with changes in these two parameters. In this document 25°C (77°F) has been taken as standard temperature and 760 mm Hg (atmospheric pressure at sea level) as standard pressure. Since the major oxidant in the atmosphere is ozone, the methods of oxidant measurement are calibrated in ozone equivalents. Therefore, in expressing ppm total oxidants in terms of
Table 1-1. FACTORS TO BE CONSIDERED IN DEVELOPING AIR QUALITY CRITERIA

Properties of pollution
- Concentration
- Chemical composition
- Mineralogical structure
- Adsorbed gases
- Coexisting pollutants
- Physical state of pollutant
  - Solid
  - Liquid
  - Gaseous
- Kinetics of formation
- Residence time

Measurement methods
- Colorimetric
- Coulometric
- Spectroscopic
- Chemical
- Chromatographic

Exposure parameters
- Duration
- Concomitant conditions
  - Temperature
  - Pressure
  - Humidity

Characteristics of receptor
- Physical characteristics
- Individual susceptibility
- State of health
- Rate and site of transfer to receptor

Responses
- Effects on health (diagnosable effects, latent effects, and effects predisposing the organism to diseases)
- Human health
- Animal health
- Plant health
- Effects on human comfort
- Corrosion of materials
- Deterioration of materials
- Effects on atmospheric properties
- Effects on radiation and temperature

\( \mu g/m^3 \), an ozone basis has also been used. Throughout the document, wherever mass concentrations of oxidants are mentioned, the reference is to equivalent amounts of ozone. It should be borne in mind that almost all data for the photochemical oxidants have originally been recorded in ppm. Factors for converting \( O_3 \) and PAN concentrations from volume (ppm) to mass (\( \mu g/m^3 \)) units are given in Chapter 2. The determination of these factors appears in the Appendix.

The terminology employed in this document generally follows usage recommended in the publications style guide of the American Chemical Society.

The scientific literature has been reviewed through January 1969, with additional sources from publications as recent as November 1969. The results and conclusions of foreign investigations have been evaluated for their possible application to the air pollution problem in the United States. This document is not intended as a complete, detailed literature review, and it does not cite every published article relating to the presence of photochemical oxidants in the ambient atmosphere. The literature has, however, been reviewed thoroughly for information related to the development of criteria, and the document not only summarizes the current scientific knowledge of photochemical air pollution, but also attempts to point up the major deficiencies in that knowledge and the presently recognized needs for further research.

Methods and techniques for controlling the sources of photochemical oxidants as well as the costs of applying these techniques are described in: AP-66, Control Techniques for Carbon Monoxide, Nitrogen Oxide, and Hydrocarbon Emissions from Mobile Sources; AP-67, Control Techniques for Nitrogen Oxide Emissions from Stationary Sources; and AP-68, Control Techniques for Hydrocarbon and Organic Solvent Emissions from Stationary Sources.

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*Adapted from S. Calvert's statement for air quality criteria hearings held by the Subcommittee on Air and Water Pollution of the U.S. Senate Committee on Public Works, July 30, 1968. Published in "Hearings before the Subcommittee on Air and Water Pollution of the Committee on Public Works, United States Senate (Air Pollution—1968, Part 2).”*
Chapter 2.

PHYSICAL AND CHEMICAL NATURE
OF PHOTOCHEMICAL OXIDANTS

A. INTRODUCTION

As initiated by sunlight, the series of atmospheric reactions between hydrocarbons and oxides of nitrogen, which lead to the formation of new products, is a most complex system. Among these products are substances termed “oxidants.” Oxidants are chemical entities detrimental to biological systems and destructive to certain materials. The purpose of this document is to examine and quantify the deleterious effects of oxidants on man and his environment. To accomplish this goal it is necessary to delineate the atmospheric physical and chemical parameters which cause oxidant formation, a subject which is difficult to discuss in a concise manner. Conciseness frequently leads to unwarranted, if not false, concepts and conclusions. The following discussion in the chapter is detailed, but it cannot be condensed further without increasing the chance of misinterpretations.

Because of the complexity of the subject matter, this discussion of photochemical oxidants has been divided into subsystems. The interrelationship of these subsystems, however, and their relation to the total air pollution system, should be kept clearly in mind. Too sharp a focus on any one photochemical subsystem can lead also to false conclusions.

The purpose then of this chapter is to detail the relationship of primary hydrocarbon emissions to oxidants, one of the secondary products. This involves the interaction of certain hydrocarbons with light-absorbing species, the subsequent oxidation of the hydrocarbons, and the accompanying formation of several new products, among which are found the oxidants, ozone and peroxyacetyl nitrates. This subject is treated in greater detail in AP-64, Air Quality Criteria for Hydrocarbons.

B. GENERAL DISCUSSION

In the original sense, the term “oxidation” describes chemical reactions in which certain atoms combine with oxygen (O₂) to form compounds known as oxides. The complete combustion of carbon in air to form carbon dioxide (CO₂) is a typical example of an oxidation reaction.

Today, the term “oxidation” is more generally used to describe the loss of one or more electrons by an atom, ion, or molecule. Conversely, a gain of electrons is referred to as “reduction.” Oxidation-reduction reactions cannot be separated since in any reaction, one of the reactants is oxidized while the other is reduced; for example, in the combustion of carbon in air, carbon is oxidized while oxygen is reduced.

The combustion of coal and petroleum products such as natural gas, gasoline, and fuel oil, termed “fossil fuels”, is an oxidation-reduction reaction which is responsible for most of the air pollution in urban atmospheres. Fossil fuels are composed principally of hydrocarbons (RH or HC)* which, upon complete combustion, produce two oxides, carbon dioxide (CO₂) and water (H₂O).

---

* A hydrocarbon is an organic compound containing carbon and hydrogen only. The hydrocarbons are classified as alicyclic, aliphatic, and aromatic, according to the arrangement of the atoms and the chemical properties of the compounds.
These two oxides are not considered to be air pollutants, since they are relatively non-toxic and are normal constituents of the atmosphere.

It is other by-products of combustion in exhaust gases and the products of incomplete combustion which lead to localized air pollution problems. Since combustion processes are usually less than 100 percent efficient, the exhaust gases contain minor amounts of the original fuel as well as minor amounts of partially oxidized fuel. Carbon monoxide (CO), aldehydes, and unsaturated hydrocarbons (those which contain less than the maximum number of hydrogen atoms as a result of the presence of double or triple bonds) are examples of partially oxidized fuel. Small amounts of nitrogen, a major constituent of air, are oxidized at the high temperatures characteristic of combustion processes, to nitric oxide (NO), and sulfur compounds, also found in varying quantities in fossil fuels, are oxidized to sulfur dioxide (SO₂). Therefore, the combustion processes produce emissions which contaminate the air with oxides of carbon, nitrogen, and sulfur, in addition to a large variety of hydrocarbons and partially oxidized hydrocarbon fragments.

When these contaminants have been released into the atmosphere, they may react chemically to produce other contaminants quite different from those originally released. Sunlight-induced oxidation processes, termed photo-oxidation, are especially important in some community air pollution problems. During daylight hours, for example, NO in polluted atmospheres is rapidly oxidized to nitrogen dioxide (NO₂). Similarly, the oxidation of SO₂ to sulfur trioxide (SO₃) is accelerated and olefins and alkylbenzenes are oxidized to form aldehydes and ketones. Formation of ozone (O₃) is also observed, as well as formation of a family of compounds identified as peroxyacyl nitrates [R'OONO₂]. Recent laboratory studies suggest the presence in such atmospheric mixtures of peroxybenzoyl nitrate, hydrogen peroxide, and alkyl hydroperoxides.

Several of the pollutants formed during the photochemical reaction process are termed oxidants. These are defined as atmospheric substances which will oxidize certain reagents not readily oxidized by oxygen. Because they are products of the photochemical air pollution process, these substances are often referred to as photochemical oxidants.

The reagent most frequently employed to measure the presence of photochemical oxidants in polluted atmospheres is a solution of neutral-phosphate-buffered potassium iodide. This reagent responds to ozone and, to some extent, to nitrogen dioxide, and to the peroxacyl nitrates. However, reducing agents such as SO₂ will negate the effect of oxidants on the reagent solution. The potassium iodide method may be used, therefore, to provide a measure of the net oxidizing properties of atmospheric pollutants without discrimination as to the species of the oxidants or reducing agents. Details of this method and its limitations are given in Chapter 5.

As has been noted, the recognized oxidants which have been measured in the atmosphere are ozone, the peroxacyl nitrates, and nitrogen dioxide. Nitrogen dioxide will be considered here only in relation to its involvement in the formation and the measurement of other oxidants. Although they are not oxidants but are involved in measurement and formation of oxidants, sulfur dioxide and hydrocarbons also will only be considered to this extent in this report. The roles of nitrogen dioxide and hydrocarbons in air pollution are discussed in other air quality criteria documents. The role of SO₂ in air pollution has been documented.¹

C. PHYSICAL PROPERTIES

1. Ozone

Some physical properties of ozone are listed in Table 2-1. The ultraviolet spectrum of ozone has been studied extensively.² The strong absorption of ozone between 2,000 and 3,000 angstroms (Å) is useful for analysis. Ozone also exhibits strong infrared absorption at about 1,050 cm⁻¹ (wave number) or 9.5 microns. Detection of this band, with its
characteristic shape, by long-path infrared spectroscopy, provides the most unambiguous demonstration of the presence of ozone in polluted air.

### TABLE 2-1. PHYSICAL PROPERTIES OF OZONE

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<th>Physical state</th>
<th>Colorless gas</th>
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<td>Chemical formula</td>
<td>(O_3)</td>
</tr>
<tr>
<td>Molecular weight</td>
<td>48.0</td>
</tr>
<tr>
<td>Melting point</td>
<td>(-192.7 \pm 0.2^\circ C)</td>
</tr>
<tr>
<td>Boiling point</td>
<td>(-111.9 \pm 0.3^\circ C)</td>
</tr>
<tr>
<td>Specific gravity relative to air</td>
<td>1.658</td>
</tr>
<tr>
<td>Vapor density</td>
<td></td>
</tr>
<tr>
<td>At (0^\circ C, 760) mm Hg</td>
<td>2.14 g/liter</td>
</tr>
<tr>
<td>At (25^\circ C, 760) mm Hg</td>
<td>1.96 g/liter</td>
</tr>
<tr>
<td>Solubility at (0^\circ C) (Indicated volume of ozone at (0^\circ C, 760) mm Hg)</td>
<td>0.494 ml/100 ml water</td>
</tr>
<tr>
<td>Conversion factors</td>
<td></td>
</tr>
<tr>
<td>At (0^\circ C, 760) mm Hg</td>
<td>(1) ppm=2141 µg/m³</td>
</tr>
<tr>
<td></td>
<td>(1) µg/m³ =4.670 \times 10^{-4}</td>
</tr>
<tr>
<td>At (25^\circ C, 760) mm Hg</td>
<td>(1) ppm=1962 µg/m³</td>
</tr>
<tr>
<td></td>
<td>(1) µg/m³ =5.097 \times 10^{-4}</td>
</tr>
</tbody>
</table>

#### 2. Peroxyacetyl Nitrate

Known physical properties of peroxyacetyl nitrate (PAN) are listed in Table 2-2. In addition, the infrared, ultraviolet, mass, and nuclear magnetic resonance spectra of PAN are available.3-7

### TABLE 2-2. PHYSICAL PROPERTIES OF PEROXYACETYL NITRATE

<table>
<thead>
<tr>
<th>Physical state</th>
<th>Colorless liquid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chemical formula</td>
<td>(CH_3COONO_2)</td>
</tr>
<tr>
<td>Molecular weight</td>
<td>121</td>
</tr>
<tr>
<td>Boiling point</td>
<td>No true boiling point, compound decomposes before boiling</td>
</tr>
<tr>
<td>Vapor pressure at room temperature</td>
<td>About 15 mm Hg</td>
</tr>
<tr>
<td>Conversion factors</td>
<td></td>
</tr>
<tr>
<td>At (0^\circ C, 760) mm Hg</td>
<td>(1) ppm=5398 µg/m³</td>
</tr>
<tr>
<td></td>
<td>(1) µg/m³ =1.852 \times 10^{-4} ppm</td>
</tr>
<tr>
<td>At (25^\circ C, 760) mm Hg</td>
<td>(1) ppm=4945 µg/m³</td>
</tr>
<tr>
<td></td>
<td>(1) µg/m³ =2.022 \times 10^{-4} ppm</td>
</tr>
</tbody>
</table>

### D. OXIDANT FORMATION PROCESSES

#### 1. Nitrogen Dioxide Photolytic Cycle

The oxidant found in the largest quantity in polluted atmospheres is ozone. In order to photochemically generate these observed \(O_3\) concentrations, a process other than direct light absorption by \(O_2\) must be involved. Light absorption by \(O_2\) does occur in the atmosphere several miles above earth. The short ultraviolet wavelengths responsible for this reaction do not reach the earth’s surface, however, and, therefore, could not produce the amounts of ozone detected in the ambient air. As will be indicated in Chapter 4, only a small portion of the \(O_3\) observed near the earth’s surface could have been transported from the stratosphere, where high concentrations exist naturally. Consequently, some other generation process must be involved.

Comparison of the absorption characteristics of the major atmospheric pollutants indicates that nitrogen dioxide is the most efficient absorber of the portion of the sun’s ultraviolet light which reaches the earth’s surface. This absorption of ultraviolet light by \(NO_2\) leads to a complex series of reactions.

\(NO_2\) is broken down (photolyzed) by ultraviolet light energy into \(NO\) and \(O\); ozone is formed in the subsequent reactions of the \(O\) atoms and \(O_2\) (stable oxygen molecules in the air have two atoms); and new \(NO_2\) and \(O_2\) are generated by the reaction of \(NO\) and \(O_3\). Thus the balance is maintained and the cycle is perpetuated. This cyclic set of reactions is depicted in Figure 2-1.

As indicated, the absorption by \(NO_2\) of the ultraviolet portions of sunlight (3,000 to 4,000 Å) is followed by a set of three reactions:

\[
NO_2 \xrightarrow{U.V., light} NO + O \quad (1)
\]

\[
O + O_2 + M \rightarrow O_3 + M \quad (2)
\]

\[
O_3 + NO \rightarrow NO_2 + O_2 \quad (3)
\]
These reactions may be explained as follows. In reaction 1, the energy contained in the ultraviolet light acting upon the NO₂ molecule is sufficient to break its bond, yielding two chemical substances, NO and O. The most probable fate of the oxygen atom thus formed is reaction with O₂ in the atmosphere to form O₃.

Reaction 2 actually proceeds in two steps. An O atom reacts with O₂ to form an energy-rich O₃ molecule. In the absence of a means to remove this excess energy, the O₃ decomposes rapidly back to O₂ and an O atom. Collision with another molecule in the system, however, will result in the transfer of some of this excess energy, leading to formation of a stable O₃ molecule. This collisional molecule is indicated by the symbol M in reaction 2. It can be any molecule in the system. In air, the collision will probably occur with N₂ or O₂, since these account for most of the molecules present.

Reaction 3 shows that the O₃ reacts with NO to reform NO₂ and O₂. Because reaction 1 is very efficient, in the absence of reactions 2 and 3, the half-life of NO₂ in the atmosphere during periods of intense sunlight would be on the order of a few minutes. Reactions 2 and 3, which result in the reformation of NO₂ in air, are also very fast, however, and tend to maintain a constant level of NO₂. Reactions 1 to 3 can be visualized as a system (Figure 2-1) in which the ultraviolet energy is acting as a pump in the rapid destruction and reformation of NO₂.

Analysis of reactions 1, 2, and 3 indicates that ozone concentrations are constrained by the steady state relationship:

![Figure 2-1. Atmospheric nitrogen dioxide photolytic cycle.](image-url)
\[(O_3) = k_1 I \left( \frac{(NO_2)}{(NO)} \right) \] (4)

In this equation, I represents the intensity of light, and \(k_1\) is a constant whose value is determined by the absorption characteristics of NO\(_2\) and by the rate constant for reaction 3. Parentheses are used to designate concentrations.

In the analysis of reactions 1 to 3, Leighton concludes that equation 4 must play a dominant role in determining ambient O\(_3\) concentrations.\(^8\) This conclusion is based on the knowledge that reactions 1 to 3 are proceeding at a rate which is one to two orders of magnitude greater than any other reactions known to be occurring in the atmosphere. Thus, reactions 1 to 3 must play a dominant role in any scheme which attempts to account for ambient O\(_3\) concentrations. The upper limit on the numerical value of the \(k_1 I\) term in equation 4 is fixed by the maximum value of I. In the atmosphere, measurements show that \(k_1 I\) can be no greater than 13 to 26 \(\mu g/m^3\) (0.01 to 0.02 ppm). Therefore, to obtain O\(_3\) concentrations of the order observed in the atmosphere, e.g., greater than 200 \(\mu g/m^3\) (0.1 ppm), the ratio \((NO_2)/(NO)\) must be 10 or greater. In general, atmospheric measurements of O\(_3\), NO\(_2\), and NO during hours of sunlight confirm this prediction.

As indicated, nitric oxide and ozone cannot coexist for long because of their very fast interactions. Figure 2-2 shows, as an example of this, concentrations of certain pollutants measured in Los Angeles on July 19, 1965. The concentrations of ozone and nitric oxide

![Graph showing diurnal variation of NO, NO\(_2\), and O\(_3\) concentrations in Los Angeles, July 19, 1965.](image)

Figure 2-2. Diurnal variation of NO, NO\(_2\), and O\(_3\) concentrations in Los Angeles, July 19, 1965.

2-5
did not simultaneously exceed 0.01 ppm except between 7:00 a.m. and 10:00 a.m., when concentrations of nitric oxide were rapidly decreasing and ozone rapidly increasing. It should be noted that the Los Angeles Air Pollution Control District records concentrations of 0 to 0.01 ppm as 0.01 ppm. The noncoexistence of high concentrations of both ozone and nitric oxide cannot always be demonstrated when using 1-hour averages. It is possible, for example, that during the first half of an hour nitric oxide is present but ozone is not, while during the last half of the hour, ozone is present but nitric oxide is not. On the average for the hour, the presence of both will be indicated. Another factor is the relatively long response time, 15 minutes or more, which is characteristic of the nitric oxide analyzer.

The changes which do occur in the atmosphere show that reactions other than 1 to 3 must also be occurring. As indicated in Figure 2-2, early in the morning nitric oxide is in some instances the major oxide of nitrogen present. Typical ratios of NO₂ to NO usually range between 0.25 and 0.50, but can reach 1 and above. As the ultraviolet light intensity increases, a rapid and almost quantitative oxidation of NO to NO₂ is observed. This photochemical oxidation is unusual since, as Leighton⁸ points out, it is one of the few known instances in which the concentration of the light absorber, NO₂, increases with irradiation. In addition to the conversion of NO to NO₂, appreciable concentrations of O₃ are found after the NO concentration has been decreased as required by equation 4. Additional reactions must occur, therefore, which disrupt or modify the equilibrium concentrations dictated by reactions 1 to 3.

At steady-state conditions, the circulation scheme illustrated in Figure 2-1 indicates that O₃ and NO are formed and destroyed in equal quantities. If the amount of O₃ consumed was slightly less than the amount of NO converted to NO₂, O₃ and NO₂ would accumulate while NO would be depleted. A process slower than that represented by reactions 1 to 3 can produce such an effect if it converts NO to NO₂ without destroying an equivalent quantity of O₃.

2. Hydrocarbon Interaction with Nitrogen Dioxide Photolysis

The NO₂ photolytic cycle explains the initial formation of ozone in polluted atmospheres, but it does not explain how concentrations can develop as large as those which have been measured. If no additional mechanism were involved, most of the O₃ would quickly break down as it reacted with the NO created in NO₂ photolysis. In other words, at steady state conditions, O₃ and NO would be formed and destroyed in equal quantities.

Laboratory experimentation and atmospheric measurements indicate that hydrocarbons provide the necessary added reactants. Certain types of hydrocarbons which are also emitted into the atmosphere in fossil fuel exhausts, notably olefins and substituted aromatics, enter the NO₂ circulation scheme. Studies suggest that oxygen atoms attack the hydrocarbons and the resultant oxidized compounds and free radicals react with NO to form more NO₂. Thus the balance of O₃ consumption by NO is upset so that O₃ and NO₂ levels build up while NO levels are depleted.

Figure 2-3 shows schematically how hydrocarbons are involved in photochemical oxidation reactions. Both O atoms and O₃ molecules have the ability to oxidize hydrocarbons; but, by examining rates of reactions, it has been determined that the more probable initial reaction is O atom oxidation since it is 10⁸ times as fast as oxidation by O₃. Although very fast, O atom oxidation is still one to two orders of magnitude slower than reaction 2. As indicated, however, processes very much slower than reaction 1 to 3 can disturb the delicate balance of the NO₂ circulation scheme illustrated in Figure 2-1.

Oxidation of hydrocarbons by O atoms is not by itself an explanation of the O₃ accumulation, nor the oxidation of NO. The intermediate free radical formed from the O atom attack on hydrocarbons is very reactive and apparently undergoes a series of changes
Figure 2-3. Interaction of hydrocarbons with atmospheric nitrogen dioxide photolytic cycle.
in which it readily reacts with O₂ and oxidizes NO to NO₂. The effect of the hydrocarbon involvement, which adds certain postulated reactions to the NO₂ circulation scheme, is shown by the dashed lines in Figure 2-3. Reflection on the indicated alternate path for oxidation of NO to NO₂ shows that the intermediates produced by the O + RH reaction must oxidize more than one molecule of NO to NO₂. Figure 2-3 thus represents schematically the manner in which hydrocarbons enter the NO₂ circulation scheme and permit a rapid buildup of NO₂ and the accumulation of O₃.

It is important to differentiate between what is known and what is uncertain concerning the mechanism outlined in Figure 2-3. Reactions 1 to 3 have been investigated extensively and are, therefore, reasonably well understood.⁹ The importance of O atom and O₃ oxidation of RH also has been investigated extensively and the rate constants of these reactions are reasonably well-known for a variety of hydrocarbons.⁸,¹⁰,¹¹ However, the reactions of oxygen atoms and ozone⁹,¹²,¹³ do not always account for the observed disappearance of hydrocarbons. Such discrepancies are probably due to the additional reaction of free-radical intermediates with the hydrocarbons.

The foregoing discussion of the interaction of hydrocarbons with the NO₂ photolytic cycle should not be interpreted as invalidating the applicability of equation 4. At atmospheric O₃ concentrations below 200 μg/m³ (0.1 ppm), however, the concurrent present of nitric oxide and ozone occasionally can be found.¹⁴ The time-averaging period for the aerometric measurements, therefore, may be significant with respect to the reaction on a real-time basis.

In the atmosphere, the major component of the peroxyacyl nitrate series is peroxyacetyl nitrate. A small amount of peroxypropionyl nitrate [CH₃CH₂COO NO₂] has also been observed. In the laboratory, these compounds can be formed conveniently by irradiation of the parent nitrite (RONO) in oxygen or air.¹⁵ This has not been proven to be a mode of formation in the atmosphere, however, since the presence of the nitrites has not been demonstrated conclusively either in the atmosphere or in laboratory systems designed to simulate photochemical air pollution systems. On the other hand, this lack of direct evidence does not eliminate nitrites as a potential source of peroxyacyl nitrates. Nitrites are rapidly photolized, and their concentrations as intermediates may not be observable. An alternate possibility is that a free radical arising from an O atom attack on hydrocarbon reacts with O₂ to form a peroxyacyl radical [RCO₂]⁹. The peroxyacyl radical then reacts with NO₂ to form a peroxyacyl nitrate. This radical can be formed also by the reaction of O atoms with aldehydes.

3. Hydrocarbon Reactivity

"Hydrocarbon reactivity" is the term used to denote the relative ability of a specific hydrocarbon to exert particular effects on the photochemical reaction process. For instance, a specific hydrocarbon may be involved in several reactions in the photochemical process, depending on its concentration, structure, and oxidation state. The end products of these reactions and the consequent intensity of the symptoms generated, such as eye irritation or plant damage, are largely dependent on the nature of the hydrocarbon involved. Any "rating of reactivity" for specific hydrocarbons will, of necessity, show marked differences, depending on the effect being measured. For example, a rating based on the ability to react with O atoms will be quite different from a rating based on the capacity to generate eye irritation.

A number of schemes have been proposed for rating the reactivity of hydrocarbons.¹⁶⁻²⁰ Some of these have been based on the rate of oxidation of nitric oxide to nitrogen dioxide, when the nitric oxide is irradiated with a specific hydrocarbon; the rate of disappearance of the hydrocarbon; the yield of products or effects; and, most simply, whether a hydrocarbon reacts or does not
react. While there are differences in the rankings, depending on which set of criteria is used, there is general agreement that unsaturated hydrocarbons are of more importance to photochemical air pollution symptoms than are saturated hydrocarbons.

Unsaturated hydrocarbons are also of great importance in the formation of oxidants. Table 2-3 presents a partial list of yields of ozone and oxidant from the photooxidation of various mixtures of organic substances and nitrogen oxides, as reported by several investigators.

Haagen-Smit\textsuperscript{21} used the rubber cracking method to measure the ozone produced by organic compounds and nitrogen dioxide irradiated during a 10-hour period. Schuck and Doyle\textsuperscript{16} measured ozone with a long-path infrared spectrophotometer. The initial concentrations of the reactants irradiated were 1230 \( \mu g/m^3 \) (1 ppm) of nitric oxide and 3 ppm of a selected hydrocarbon (except in the case of ethylene, in which 6 ppm was irradiated). Soon after the start of irradiation, ozone became detectable and continued to increase until a stable level was reached at the concentrations listed in Table 2-3. Altshuller and Cohen\textsuperscript{22} and Altshuller et al.\textsuperscript{24} used the colorimetric and coulometric potassium iodide methods for measuring oxidants.

Because of the differences in methods, concentrations of reactants, and physical properties of the environmental test chambers, the results obtained by one set of authors are not quantitatively the same as those obtained by another, nor can they necessarily be extrapolated to atmospheric pollution conditions. They do have applicability, however, on a relative basis. Thus, in general, the internally double-bonded olefins, the diolefins, and highly substituted aromatic compounds produce the greatest yields of oxidants or ozone. Acetylene, benzene, and the paraffins having less than five carbon atoms produce the least oxidant except at very high hydrocarbon-to-\( NO_x \) reactant ratios.\textsuperscript{16, 19, 21-24}

Several studies of peroxyacyl nitrate yields have been reported.\textsuperscript{16, 23, 15-27} Table 2-4 tabulates the reported peroxyacetyl nitrate yields from the photooxidation of various mixtures of hydrocarbons and nitrogen

<table>
<thead>
<tr>
<th>Organic substance</th>
<th>Cracking depth, mm</th>
<th>Static irradiation yields</th>
<th>Dynamic irradiation yields</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Haagen-Smit\textsuperscript{21}</td>
<td>Schuck and Doyle\textsuperscript{16}</td>
<td>Altshuller and Cohen\textsuperscript{22}</td>
</tr>
<tr>
<td>1,3-Butadiene</td>
<td>12</td>
<td>0.65</td>
<td>—</td>
</tr>
<tr>
<td>2-Alkenes</td>
<td>8</td>
<td>0.55-0.73</td>
<td>—</td>
</tr>
<tr>
<td>1,3,5-Trimethylbenzene</td>
<td>7</td>
<td>—</td>
<td>1.1</td>
</tr>
<tr>
<td>Xylenes</td>
<td>6-7</td>
<td>0.18</td>
<td>0.65-1.0</td>
</tr>
<tr>
<td>1-Alkenes</td>
<td>5</td>
<td>0.58-1.00</td>
<td>—</td>
</tr>
<tr>
<td>Methanol, ethanol</td>
<td>5</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Formaldehyde</td>
<td>4</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Propionaldehyde</td>
<td>4</td>
<td>—</td>
<td>1.0</td>
</tr>
<tr>
<td>3-Methylheptane</td>
<td>3</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>n-Nonane</td>
<td>3</td>
<td>—</td>
<td>0.2</td>
</tr>
<tr>
<td>Ethylene</td>
<td>2</td>
<td>1.1</td>
<td>—</td>
</tr>
<tr>
<td>Hexanes, heptanes, iso-octane</td>
<td>1</td>
<td>0.2</td>
<td>—</td>
</tr>
<tr>
<td>Toluene</td>
<td>0.6</td>
<td>—</td>
<td>0.5</td>
</tr>
<tr>
<td>Acetylene</td>
<td>0.5</td>
<td>—</td>
<td>0.0</td>
</tr>
<tr>
<td>( C_1 - C_5 ) paraffins</td>
<td>&lt; 0.2</td>
<td>0.0-0.2</td>
<td>—</td>
</tr>
</tbody>
</table>
Table 2-4. Peroxyacetyl Nitrate Yields from Photooxidation of Hydrocarbon-Nitrogen Oxide Mixtures in Air \(^6, 16, 23, 25\)

<table>
<thead>
<tr>
<th>Hydrocarbon</th>
<th>Peroxyacetyl nitrate concentration, ppm by volume</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Stephens(^a)</td>
</tr>
<tr>
<td>n-Butane</td>
<td>-</td>
</tr>
<tr>
<td>Isopentane</td>
<td>-</td>
</tr>
<tr>
<td>n-Hexane</td>
<td>-</td>
</tr>
<tr>
<td>iso-octane</td>
<td>-</td>
</tr>
<tr>
<td>Ethylene</td>
<td>0</td>
</tr>
<tr>
<td>Propylene</td>
<td>&gt;0.55</td>
</tr>
<tr>
<td>1-Butene</td>
<td>0.55</td>
</tr>
<tr>
<td>iso-butene</td>
<td>0.15</td>
</tr>
<tr>
<td>1-Hexene</td>
<td>-</td>
</tr>
<tr>
<td>trans-2-Butene</td>
<td>-</td>
</tr>
<tr>
<td>cis-2-Butene</td>
<td>0.7</td>
</tr>
<tr>
<td>2-Methyl-2-butene</td>
<td>-</td>
</tr>
<tr>
<td>cis-3-Hexene</td>
<td>0.8</td>
</tr>
<tr>
<td>trans-3-Hexene</td>
<td>1.0</td>
</tr>
<tr>
<td>Tetramethylethylene</td>
<td>-</td>
</tr>
<tr>
<td>1,3-Butadiene</td>
<td>-</td>
</tr>
<tr>
<td>Benzene</td>
<td>-</td>
</tr>
<tr>
<td>Toluene</td>
<td>-</td>
</tr>
<tr>
<td>C(_g^+) Monoalkylbenzenes</td>
<td>-</td>
</tr>
<tr>
<td>o-Xylene</td>
<td>0.4</td>
</tr>
<tr>
<td>p-Xylene</td>
<td>0.4</td>
</tr>
<tr>
<td>m-Xylene</td>
<td>0.55</td>
</tr>
<tr>
<td>1,3,5-Trimethylbenzene</td>
<td>0.8</td>
</tr>
<tr>
<td>1,2,4,5-Tetramethylbenzene</td>
<td>0.7</td>
</tr>
</tbody>
</table>

\(^a\) 5 ppm hydrocarbon and 5 ppm NO or NO\(_2\) (References 6 and 25).
\(^b\) 2 ppm hydrocarbon and 1 ppm NO (Reference 23).
\(^c\) 3 ppm hydrocarbon and 1 ppm NO or NO\(_2\) (Reference 16).

Oxides. Altshuller et al.\(^{19,28}\) have been able to detect very low concentrations of peroxyacetyl nitrates in the 250- to 500-\(\mu\)g/m\(^3\) (0.05 to 0.10 ppm) range from the irradiation of n-butane, iso-pentane, 2,4-dimethylhexane, and toluene with nitrogen oxides. These results indicate, as do the results of Heuss and Glasson,\(^{23}\) that paraffinic hydrocarbons, together with benzene and monoalkylbenzenes, contribute very little to PAN yields in atmospheric reactions. The production of PAN-type compounds is associated with the presence of propylene and higher-molecular-weight olefins, and with dialkyl- and trialkylbenzenes.

Heuss and Glasson have reported peroxybenzoyl nitrate as a minor product of the irradiation of toluene and other benzyl-type monoalkylbenzenes with nitric oxide.\(^{23}\) Styrene and other aromatic olefins produce somewhat greater yields of peroxybenzoyl nitrate when irradiated with nitric oxide. Peroxybenzoyl nitrate was found to be a very potent eye irritant, since only 75 to 150 \(\mu\)g/m\(^3\) (0.01 to 0.02 ppm) is capable of causing from light to severe eye irritation.\(^{23}\) This lacrimator does not appear to be formed from other types of hydrocarbons.

### 4. Nitric Oxide and Nitrogen Dioxide

One of the consequences of high-temperature combustion of fossil fuels in air is the formation of NO by oxidation of a portion of the \(N_2\) content of the air. As previously
discussed, the major process by which atmospheric nitric oxide is oxidized to nitrogen dioxide is photochemical, involving hydrocarbon interaction. There are, in addition, two other reaction pathways which contribute to atmospheric nitrogen dioxide concentrations.

The first of these, the reaction of ozone with nitric oxide, has already been considered briefly in the discussion of the nitrogen dioxide photolytic cycle. At or near sunset, when ozone photochemical formation ceases, a polluted atmosphere will generally contain an excess of ozone and a minimum of nitric oxide. While ozone formation stops at sunset, however, exhaust emissions of nitric oxide usually do not. From a rate-of-reaction viewpoint, the most probable fate of the remaining ozone is reaction with the continuing nitric oxide emissions. During the early evening hours, therefore, some fraction of an observed nitrogen dioxide concentration is, and would be expected to be, contributed by this reaction.

Nevertheless, a second reaction, the oxidation of nitric oxide by oxygen in the air during the early phases of atmospheric exhaust gas dilution, is the cause of some fraction of the observed nighttime nitrogen dioxide concentrations. The overall reaction, as exhaust is diluted in air, is described by the reaction:

\[ 2 \text{NO} + \text{O}_2 \rightarrow 2 \text{NO}_2 \]  

(5)

The rate of formation of NO\(_2\) from reaction 5 is determined by:

\[ \frac{d(\text{NO}_2)}{dt} = k_2 (\text{NO})^2 (\text{O}_2). \]  

(6)

When dilution is complete, that is, when the NO concentration is in the ppm range, reaction 5 will result in a small contribution to the oxidation of nitric oxide. Since atmospheric exhaust gas dilution is very rapid, the amount of NO oxidized by this process is usually less than 10 percent of the total NO emissions from sunset to sunrise. Consequently, it is not unusual to have the maximum daily NO concentration recorded early in the morning.

E. METEOROLOGICAL EFFECTS

1. General

The diurnal urban emission pattern of oxidant-forming pollutants is fairly uniform from weekday to weekday. It is apparent, therefore, that variations in the pattern of oxidant concentrations must be due largely to meteorological factors. Dilution of oxidants is accomplished by the same process of atmospheric turbulence and transport that affects other gaseous contaminants. The large range of values over which these parameters extend often contributes significantly to the diurnal, daily, and seasonal variations of observed oxidant concentrations at a particular location. Atmospheric concentrations of oxidant are also dependent upon those meteorological variables affecting oxidant formation. Some of these factors are discussed in the following sections.

2. Sunlight

Light is measured in parameters of intensity and wavelength. Intensity is the measure in photons of the concentration of light, while wavelength is the measure of how much energy each photon contains. The longer the wavelength, the less energy is contained in each photon.

Interaction of light with an NO\(_2\) molecule to form NO and an O atom (reaction 1) is a reaction between a photon and the NO\(_2\) molecule. Thus, the rate of NO\(_2\) destruction, or of O atom formation, is directly proportional to the intensity of light and the concentration of NO\(_2\). Since ozone concentration is a function of O atom concentration (reaction 2), the ozone concentration by equation 4 is likewise directly proportional to the light intensity.

NO\(_2\) efficiently absorbs most wavelengths of light between 3,000 and 6,000 Å. Efficient absorption alone, however, does not guarantee that NO\(_2\) dissociation (reaction 1) will take place. It has been determined that
the dissociation of NO₂ is a function of wavelength.\textsuperscript{8, 29} Over 90 percent of NO₂ molecules are photolyzed when the light wavelength is between 3,000 and 3,700 Å.\textsuperscript{29} Above 3,700 Å, the percentage drops off rapidly; and above 4,200 Å, dissociation does not occur.

Disruption of the bond between O and NO in NO₂, as necessitated in reaction 1, requires approximately 72 kilocalories of energy per mole at 25\(^\circ\) C. This corresponds to the energy available in light at a wavelength of approximately 4,000 Å. At longer wavelengths, light cannot break the NO₂ bonds because insufficient energy is available.

The wavelength at which NO₂ dissociation fails to occur is not precisely defined because the individual molecules of NO₂ do not all possess the same ground state energy prior to absorption. Figure 2-4 shows that the transition from 100 percent to 0 percent dissociation occurs between 3,700 and 4,200 Å. This gradual transition indicates a variation in ground state energy of about 10 kilocalories per mole.

Figure 2-4. Nitrogen dioxide dissociation as a functional wavelength of light absorbed.

The relative distribution of wavelengths from sunlight reaching the earth's surface does not vary appreciably except in the presence of absorbing species or light scattering particles. Since polluted atmospheres contain variable amounts of NO₂, and since the NO₂ will absorb certain wavelengths of sunlight, there can be some changes in both the relative wavelength distribution and the intensity of sunlight at the earth's surface. Due to low ambient NO₂ concentrations, any such effects of NO₂ absorption on wavelength distribution and intensity are likely to be small.

A reduction as high as 80 percent of intensity near the region of 3,250 Å has been observed at the earth's surface during an intense photochemical air pollution episode.\textsuperscript{30} Since most of the NO₂ had disappeared from the atmosphere at this time, it cannot be responsible for the observed decrease in light intensity. Rather, the reduction has been generally attributed to the light-scattering effect of atmospheric aerosols formed as a by-product of the photochemical interactions of RH, NOₓ and SO₂.

Light intensity reductions of the magnitude observed would be expected to produce a substantial decrease in the rate of photochemical reactions. The actual effect, however, caused by aerosol diffusion is far more complex. As pointed out by Leighton,\textsuperscript{8} the available light energy is a function of the height within a given polluted air mass. Within the upper half of the air mass, the available light energy will tend to be the result of aerosol scattering, and this will be substantially greater than that available from just incident radiation. An opposite effect is observed in the lower half of the air mass. Thus, the formation of photochemical aerosols has the rather interesting effect of increasing the rate of photochemical reactions in the upper half of the polluted air mass and, at the same time, decreasing these rates in the lower half of the air mass. Thus, in areas where visibility reduction are severe, a less direct correlation between observed oxidant concentrations and light intensity measured at ground level would be expected. The total effect on oxidant levels of such intensity effects cannot be ascertained without quantitative data on vertical mixing within a polluted air mass.

2-12
Variations in sunlight intensity which most affect development of oxidants are those occurring as a function of time of day, time of year, and geographical location. Maximum intensities prevail around noon, with duration times of near maximum intensities varying according to season and latitude. Cloud cover is, of course, another important factor, as well as the atmospheric accumulation of light-scattering and light-absorbing pollutants.

The combination of the factors of light intensity and duration controls to some extent the amount of photooxidized materials which can be formed. In the United States, the maximum noonday intensity and the duration of nearly maximum light intensity do not vary appreciably with latitude during the summer months. In the region of 3,000 to 4,000 Å, the maximum total intensity is \( \gamma \times 10^{16} \) photons cm\(^{-2}\) sec\(^{-1}\), with the measurement remaining near this value for 4 to 6 hours.\(^9\) By contrast, the winter values vary from 0.7 \( \times 10^{16} \) to 1.5 \( \times 10^{16} \) photons cm\(^{-2}\) sec\(^{-1}\), depending on latitude, and time near maximum light intensity in the winter is reduced to 2 to 4 hours.\(^9\) These times and intensities are important controlling factors in determining the severity and duration of photochemical air pollution symptoms.

3. Temperature

The temperature of a polluted air mass determines the ground state energy of all chemical species in the system. High temperatures increase ground state energy. In Figure 2-4, for example, the transition curve can be slightly shifted to longer wavelengths by increasing the temperature and, thus, increasing the ground state energy of the system.\(^2\)\(^9\) Since most chemical reactions require addition or subtraction of energy, a temperature variation can also change the reaction rate.

Laboratory experiments have shown that a 40°F temperature rise increases the rate of NO and hydrocarbon oxidations by a factor of 2.\(^3\)\(^1\) There is also evidence in certain systems that a temperature increase of this magnitude results in a fourfold increase in rate of oxidant production.\(^3\)\(^2\) These are substantial changes which can affect the concentrations of photochemical air pollution products in the atmosphere.

Quantitative estimates of the effect of ambient temperature variations upon these manifestations cannot be made at this time. The restrictive nature of the laboratory experiments and the lack of knowledge concerning the variables and reactions involved are two of the factors which prohibit other than qualitative estimates.

F. REACTANT CONCENTRATION STUDIES

1. Environmental Chamber Studies

During the past 15 years, a number of laboratory studies have investigated the formation of photochemical air pollution. The results of such studies are in agreement with atmospheric measurements and present a consistent picture. Ultraviolet irradiation of air mixtures, containing concentrations in the ppm range of certain hydrocarbons and NO or NO\(_2\), leads to:

1. Oxidation of NO to NO\(_2\) and then to nitric acid.
2. Reactions of NO\(_2\) to form products such as peroxyacetyl nitrates and inorganic nitrates.
3. Oxidation of hydrocarbons to form aldehydes and ketones.
4. Formation of O\(_3\).
5. Increase in the oxidation rate of SO\(_2\) to SO\(_3\) and sulfate.

Rather extensive data are available on the consumption of reactant hydrocarbons and nitrogen oxides and on the formation of products from a number of irradiated hydrocarbon-nitrogen oxide systems.\(^1\)\(^3\) Acceptable carbon mass balances have been obtained for the photooxidation of hydrocarbons such as ethylene, propylene,\(^1\)\(^2\) 1-butene,\(^1\)\(^6\) trans-2-butene,\(^3\)\(^3\) n-butane,\(^1\)\(^9\) and pentanes.\(^1\)\(^9\) Major products vary from system to system and include formaldehyde, acetaldehyde, one or more other aldehydes or ketones, carbon monoxide, carbon dioxide, and peroxyacetyl nitrates;\(^1\)\(^3\) other important products include
ozone, nitrogen dioxide, and inorganic nitrates. Acceptable nitrogen balances have been obtained from irradiations of mixtures containing hydrocarbons such as ethylene and 1,3-butadiene, which produce little if any peroxyacyl nitrates. Acceptable carbon and nitrogen balances have not been reported from irradiation of nitrogen oxides with higher-molecular-weight hydrocarbons, such as the alkylbenzenes.

Several studies have been made of ultraviolet-irradiated automobile-exhaust systems. Ozone, nitrogen dioxide, formaldehyde and other aldehydes, and peroxyacyl nitrates have been identified and measured as products. As would be expected, the same types of products are found as in simpler mixtures. From the results of one study, the formation of aldehydes can be estimated to account for about 30 percent of the carbon atoms consumed from the hydrocarbons. Hydrocarbon consumption and formaldehyde and peroxyacetyl nitrate formation were determined from a small series of experiments on irradiated atmospheric samples collected between 7 and 8 a.m. in downtown Los Angeles. The formaldehyde accounted for 24 percent, and the peroxyacetyl nitrate 9 percent, of the carbon atoms consumed.

In addition to the product-yield-type studies discussed above, much attention has been directed to the quantification of reaction dynamics. It was realized at an early date, however, that rates of reactions and concentrations of products are not strictly proportional to concentration of reactants. Haagen-Smit first noted this effect during irradiation of flasks containing hydrocarbons and NOx in air. In view of the previous discussion concerning the complex hydrocarbon interaction with NO2 photolysis, such nonlinear effects are not unexpected. Since concentration ratios, as well as absolute concentrations, of the primary reactants affect oxidant concentrations and rates of formation, a summary of the available information on these topics is required in order to appreciate oxidant variations in polluted atmospheres. Nevertheless, in discussing these effects, it must be remembered that all of the data on this subject have been generated in experimental systems which only approximate the ambient atmospheres. Because of the limitations of atmospheric data, researchers have been unable to show as yet that concentration ratio effects are unequivocally operating in the real atmosphere. Nevertheless, the implications demand an examination of the experimental data.

Subsequent to the work of Haagen-Smit, several investigators have attempted to quantify the effect of changes in reactant concentrations and ratios. In one sense, all the studies could be deemed successful in that each laboratory was able to confirm and quantify the effects. There appears to be only a qualitative agreement, however, when comparing one study with another, and there is no general agreement concerning the meaning of the results when extrapolated to the atmosphere. As indicated, direct atmospheric data on this subject have not as yet been obtained. A major stumbling block is the lack of air monitoring data on atmospheric concentrations of reactive hydrocarbons. Such a lack means the applicable concentrations or ratios in the real atmosphere cannot at this time be clearly specified.

Examples of the relationships observed experimentally between reactant and oxidant concentrations are shown in Figures 2-5 through 2-8. It should be noted that the studies in Figures 2-5 and 2-7 refer to irradiation of mixtures containing propylene as the hydrocarbon, while the studies in Figures 2-6 and 2-8 refer to irradiated mixtures using auto exhaust as the hydrocarbon source. In spite of many differences in choice of initial reactants and experimental design, the qualitative agreement in these four studies is rather striking. All studies show a similar dependence of oxidant on hydrocarbon concentrations. All studies further reveal a noticeable dependence upon the NOx levels. From the previous discussion, it is not unrealistic to envision the complex NOx effect as the direct result of the competition between the reactions resulting in oxidant formation and destruction.
Figure 2-5. Maximum oxidant concentration as a function of propylene and nitric oxide reactant concentrations during 2-hour dynamic irradiation.12

Figure 2-6. Maximum oxidant concentration as a function of hydrocarbon and nitrogen oxide concentrations reacted during 1-hour dynamic irradiation of automobile exhaust mixtures.41

Figure 2-7. Maximum oxidant concentration as a function of propylene and nitric oxide reactant concentrations during static irradiations.37

Figure 2-8. Maximum oxidant concentration as a function of hydrocarbon and nitrogen oxide reactant concentrations during 2-hour dynamic irradiation of automobile exhaust mixtures.35

enabling the prediction of oxidant concentrations from a knowledge of emission and meteorological data. Unfortunately, such models are in an early experimental stage. Instead of such a model, however, it is still possible to extract qualitative information from monitoring data, but assumptions must be made to accomplish this purpose. If these

2. Atmospheric Studies

As previously indicated, a severe handicap exists when attempting to demonstrate a relationship between reactant concentrations and oxidant concentrations in polluted atmospheres. Theoretically, generation of an atmospheric simulation model should be feasible,
implications are kept in mind, there is little danger of overestimating the value of information generated in this manner.

One assumption is that observed concentrations of reactants and products are largely a function of meteorological factors. This assumption implies that day-to-day emissions are approximately constant, thus further implying that the day-to-day human activities leading to these emissions are relatively constant. Since this latter assumption has been shown to be approximately true for weekday conditions only, the following study will be confined to weekday data. Weekend emissions show substantial changes attributable to changes in human activities.

A necessary restriction in order to obtain acceptable correlation between stations and consequently between a single station and a large area air mass, is that air quality values are based on a large quantity of long-term average data. In lieu of detailed meteorological data, a further restriction is that data be examined only for those portions of the year which show the greatest atmospheric stability. Adherence to this latter restriction produces two desirable effects. First, it confines the study to those portions of the year with the greatest potential for oxidant formation. Second, it restricts the examination of the data to those days when there is the greatest likelihood of a direct relationship between morning and noontime meteorological factors.

Based on the discussed assumptions and restrictions, the CAMP data for a 3-year period during the months of June, July, and August from six cities were examined. Figure 2-9 shows the resulting plot of early morning hydrocarbon concentrations (6 to 9 a.m.) as a function of average maximum daily oxidant concentration (5-minute peak). A similar treatment of the data from three stations in Los Angeles is shown in Figure 2-10. Comparison of the experimental data in Figures 2-5 through 2-8 with the atmospheric data in Figures 2-9 and 2-10 shows that they are similar. No additional statement or discussion is justified in view of the gross techniques and assumptions used to analyze the atmospheric

![Figure 2-9. Effect of 6 a.m.-9 a.m. hydrocarbon concentrations on maximum daily oxidant concentrations during June, July, and August, 1965-1967.](image)

![Figure 2-10. Effect of 6 a.m.-9 a.m. hydrocarbon concentrations on maximum daily oxidant concentrations in Los Angeles, during April through September, 1962 and 1963.](image)
data. It should be apparent, however, that this method of data treatment can be made rigorous by application of statistical methods and consideration of measured day-to-day meteorological factors. Indeed, until such time as simulation models are perfected, it may be that this suggested approach is the only means of extracting ambient reactant-product information from air monitoring data.

The involvement of hydrocarbons in NO2 photolysis, with the resulting formation of excess ozone, can also be demonstrated by examination of the changes in pollutant concentrations occurring on any given day. For this purpose, 2 days in Philadelphia during July 1967, were chosen for examination. Philadelphia was chosen because, beginning in 1967, this city’s CAMP station measured nonmethane hydrocarbons and thus provided data for a better measure of the hydrocarbons involved in the formation of oxidants. Both days, July 18 and 22, were chosen because an appreciable oxidant formation occurred and thus they were characteristic of stabilized atmospheric conditions. Changes in pollutant concentrations are, therefore, more likely to be principally the result of emission variables and sunlight-induced reactions. July 18th (Figure 2-11) was a weekday; July 22nd (Figure 2-12) was a Saturday. These days were chosen to illustrate the effect of weekend changes in human activities on emissions.

In most respects the Philadelphia data shown in Figure 2-11 are typical of a weekday in Los Angeles. The only major difference is that, in Philadelphia, the early morning NO concentration is frequently less than the NO2 concentration, a contrast to Los Angeles observations. Experimentally, it has been shown that the only effect is a time delay when irradiating hydrocarbons in the presence of NO2 instead of NO. Hydrocarbon involvement and formation of ozone occur earlier in hydrocarbon-NO2 mixtures than in hydrocarbon-NO mixtures, since the light absorber (i.e., NO2) already is at an elevated concentration. It is evident, as shown in Figure 2-11, that the advent of sunrise leads to the rapid formation of oxidants. More complete analysis of this data would indicate that NO is being oxidized to NO2 and that hydrocarbons are being destroyed. It is interesting to note that secondary peaks near 11:30 a.m. in the hydrocarbon and NO2 curves are followed 1 hour later by a secondary oxidant peak. At the same time, and as expected, the NO remains at a near zero value, as required by the presence of elevated oxidant values. Saturday’s changes, shown in Figure 2-12, indicate that the major difference is one of time; the peak hydrocarbon and NO2 values occur 2 hours later than on weekdays. As expected, the oxidant peak is also 2 hours later.

A discussion of the relationship between ambient concentrations of hydrocarbons and photochemical oxidants may also be found in Air Quality Criteria for Hydrocarbons, NAPCA Publication No. AP-64.

G. FUTURE RESEARCH

In any evaluation of laboratory data on photochemical air pollution, it should be emphasized that environmental chamber experiments are relatively simple when compared to the highly complex and variable situations occurring in the atmosphere. The fact that various laboratory investigators have
obtained significantly different quantitative results illustrates this contrast. Such limited findings can only be applied to the ambient atmosphere in a suggestive and qualitative manner. There is an apparent need for more reliable and applicable quantitative information derived from direct atmospheric observations, as well as for refinement in the results obtained from irradiation chamber studies.

Relevant monitoring data in more detail are needed on reactant concentrations and meteorological factors. Because these elements control the size, ventilation rate, and mixing characteristics of polluted air masses, they determine to a large extent pollutant concentrations and photochemical reaction products.

The need for all types of basic irradiation chamber experimentation remains acute. A partial listing of such needed experiments is:

1. Kinetic measurements of the rates of reaction of hydroxyl radicals with various hydrocarbons. The observed overall rates of hydrocarbon consumption are not explained by reactions with atomic oxygen and ozone. The most likely intermediate species contributing to these reactions are hydroxyl radicals.

2. The relationship of oxidants, oxygenated hydrocarbons, organic nitrates, and other possible substances to such factors as reactant concentrations, reactant concentration ratio, temperature, light intensity, and water vapor. While the products accounting for most of the carbon atoms from lower molecular weight paraffinic hydrocarbons and olefinic hydrocarbons have been measured, the results of carbon balances from the reactions of alkylbenzenes and higher molecular weight paraffins are poor. Although the presence of dicarbonyl compounds from alkylbenzenes has been reported, quantitative measurements are lacking. Peroxybenzoyl nitrate, a new and minor product from the reaction of certain alkylbenzenes, is a potent eye irritant which has recently been identified in this type of research.

3. The relationship of aerosol formation to a variety of reaction parameters, including water vapor content. Studies of the role of sulfur dioxide, sulfuric acid, sulfates, nitric acid, nitrates, and organics in aerosol formation should also be extended. The composition of the organic fraction of aerosols is still unknown.

4. More measurements on the yields of organic and inorganic nitrates from a variety of hydrocarbon-nitrogen oxide systems. Just recently, the formation of inorganic nitrates has been shown to occur at an appreciable rate and as a direct result of photooxidation of hydrocarbons in the presence of nitrogen oxides.

5. Additional investigation of the importance of other primary absorbers or species made reactive by energy transfer. Such species include the products of aldehydes and singlet oxygen.

Of even greater urgency, detailed investigations of the atmospheric reactions in several representative urban atmospheres are needed. Such studies have been in progress in Los Angeles and have been initiated in the New York-New Jersey area. The types of information required includes:

1. Reactant and product composition obtained from irradiation of atmospheric samples with solar radiation at atmospheric temperatures. Such results can be used to verify kinetic and product composition results in the
laboratory. Such measurements have been made in a limited group of experiments on samples collected in Los Angeles.

2. Detailed results on the composition of ground-level samples and samples obtained aloft. Coordinated vertical distribution measurements along trajectories are needed to trace the reaction history of air masses. Oxidant concentrations should be related to hydrocarbon nitrogen oxide concentration levels. Such measurements should also emphasize clarification of the importance of various species identified in laboratory irradiations but not in the atmosphere, such as peroxybenzoyl nitrate, hydrogen peroxide, alkyl hydroperoxides, and dicarboxyls.

3. Measurement techniques to estimate the actual concentrations of radical intermediate species in urban atmospheres.

4. Measuring techniques to obtain hourly concentrations of total particulates, submicron particulates, sulfate, nitrate, lead, and other particulate species. Although a considerable body of measurements are now available on the diurnal variations of gaseous pollutants, comparable diurnal measurements of particulates are not available.

H. SUMMARY

The atmospheric reactions leading to formation of the photochemical oxidants, ozone and peroxyacyl nitrates, are reasonably well understood in a broad if not detailed sense. It is known that certain hydrocarbon pollutants interact with the photolytic cycle of nitrogen dioxide and, as a result, the hydrocarbons are oxidized to form various products. The product mixtures contain substantial concentrations of ozone, as well as such hydrocarbon products as aldehydes, ketones, and peroxyacyl nitrates.

Absorption of sunlight by nitrogen dioxide in the region of 3,000 to 4,000 Å results in dissociation of the nitrogen dioxide into nitric oxide and an oxygen atom. These oxygen atoms react principally with the oxygen in air to form ozone. A small portion of the oxygen atoms and ozone also react with certain hydrocarbons to form free-radical intermediates, as well as various products. In some unknown manner, these free radical intermediates compete with ozone for nitric oxide. One result is the very rapid oxidation of nitric oxide to nitrogen dioxide and an increased concentration of ozone.

Experimentally, this photochemical system can be reproduced in the laboratory, and data can be obtained relating oxidant concentrations to the concentrations and types of both hydrocarbons and oxides of nitrogen. For various reasons, however, these results cannot be extrapolated to the atmosphere in a quantitative manner. Because of the complexity of the atmospheric mixture and the necessary use of non-specific monitoring techniques, demonstration of the relationship of hydrocarbon and oxidant concentrations in the atmosphere is somewhat limited. A more precise examination of the parameters affecting atmospheric oxidant concentrations awaits the application of statistical and modeling techniques as well as improvements in measuring methods.

I. REFERENCES


10. Cvetanović, R.J. Addition of Atoms to Olefins in the Gas


38. Hamming, W.J. et al. Gasoline Composition and the Control of Smog. Joint Study by Western Oil and Gas Association and Los Angeles County Air Pollution Control District. Los Angeles, 1961.


Chapter 3.

ATMOSPHERIC PHOTOCHEMICAL OXIDANT CONCENTRATIONS

A. INTRODUCTION

This chapter is concerned with the measured levels of oxidants in urban atmospheres and some of the factors which affect these levels. It is intended to be a straightforward presentation of atmospheric measurements and their interpretation. The only point likely to cause confusion is the various terms used to describe oxidant concentrations. Two of these terms, "oxidant" and "total oxidant," are used to describe the "net" oxidizing ability of the air sampled. Use of these non-specific terms is required because the most common method of measurement does not differentiate between oxidizing or reducing agents in all cases. The terms "corrected oxidant" or "adjusted oxidant" are used to indicate that the measurements have been corrected for certain known responses caused by gases other than ozone or peroxyacetyl nitrate. The terms "ozone" or "peroxyacetyl nitrate" are used only when the method of measurement is specific.

B. CONCENTRATIONS OF OXIDANTS IN URBAN ATMOSPHERES

1. General Discussion

In the early 1950's, the Los Angeles County Air Pollution Control District (LACAPCD) established its first air monitoring network (12 stations), using automatic sampler-analyzers and thus enabling the District's staff to make detailed continuous observations of gaseous pollutants. Network equipment also included potassium iodide (KI) oxidant recorders; ozone photometers were added in 1958. In 1961, the State of California Department of Public Health organized a 16-station Statewide Cooperative Air Monitoring Network (SCAN). Six of the stations of SCAN were equipped and operated entirely by the Department; seven were equipped and operated jointly by the Department and by LACAPCD. The Public Health Service of the U.S. Department of Health, Education, and Welfare opened its Continuous Air Monitoring Project (CAMP) in Cincinnati in October 1961. By early 1962, five additional CAMP stations were operating in Chicago, Philadelphia, San Francisco, New Orleans, and Washington, in cooperation with local air pollution control agencies. The station in New Orleans was moved to St. Louis in 1964, and in 1965, the San Francisco Station equipment was moved to Denver.¹ Air monitoring in San Francisco was continued by the Bay Area Air Pollution Control District as a new SCAN station.

Today, data on oxidants are continuously obtained in many U.S. cities by local, State, and Federal agencies. In California alone, oxidants are continuously measured at about 40 air monitoring stations. The oxidant data presented in this report are from the CAMP stations operated by the Public Health Service,¹ from a few stations in California,² and from the local agency in Phoenix.³ Two of the California stations are in the Los Angeles area, where the highest concentrations of oxidants are found; two others are in large coastal cities; and the remaining two are in small cities, one on the coast and one in the Central Valley of California.
At the CAMP and LACAPCD stations, oxidant concentrations are measured with colorimetric analyzers using neutral-phosphate-buffered, 10 percent, KI reagent. At the SCAN stations, 20 percent KI is used. All stations of the networks, except those belonging to CAMP, report the peak instantaneous concentration of each day, the average concentration during each clock hour, and the maximum hourly average concentration of each day. CAMP reports the 5-minute peak concentration rather than the instantaneous peak, in addition to hourly averages and maximum hourly averages.

The peak concentration for each day, or the highest value for the day, may be of only a few minutes duration. The hourly average concentration is the average concentration during any one of the twenty-four 60-minute intervals beginning and ending on the hour, such as 6:00 to 6:59 p.m. The maximum hourly average is the highest hourly average each day. From these basic data, other data are obtainable, such as the monthly mean of the hourly averages or of the maximum hourly average concentrations.

In addition, as shown by Larsen, it is possible to convert the maximum concentration for one averaging time to that for another. He computes the expected maximum concentration \( C \) for a given averaging time by using the geometric mean \( (M_g) \), the standard geometric deviation \( (\sigma_g) \), and the "standard normal deviate" of no more than one occurrence in 1.67N trials \( (z) \). His formula is

\[
C = M_g \sigma_g z
\]

2. Oxidant Concentration Patterns

Table 3-1 shows the maximum hourly average concentrations and peak concentrations, as well as the number and percent of days when the maximum hourly average concentration exceeded 290, 200, and 100 \( \mu g/m^3 \) (0.15, 0.10, and 0.05 ppm) for 12 monitoring sites. These values vary greatly from city to city. The percent of days that oxidant concentrations exceed 290 \( \mu g/m^3 \) (0.15 ppm) in Los Angeles and Pasadena are of an order of magnitude greater than in other cities. It is also interesting to note that peak concentrations in St. Louis reached 1,670 \( \mu g/m^3 \) (0.85 ppm). The high peak concentration reported for the St. Louis air monitoring station, however, is one of a series of extraordinarily high readings which usually occur late at night and are of short duration. It is suspected that they result from emissions from a nearby large chemical complex rather than an atmospheric photochemical reaction. The cumulative frequency distribution of

<table>
<thead>
<tr>
<th>Station</th>
<th>Total days of available data</th>
<th>Number and percent of total days with maximum hourly average equal to or greater than concentration specified</th>
<th>Maximum hourly average, ppm</th>
<th>Peak concentration, ppm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Days</td>
<td>Percent of days</td>
<td>Days</td>
</tr>
<tr>
<td>Pasadena</td>
<td>728</td>
<td>299</td>
<td>41.1</td>
<td>401</td>
</tr>
<tr>
<td>Los Angeles</td>
<td>730</td>
<td>220</td>
<td>30.1</td>
<td>354</td>
</tr>
<tr>
<td>San Diego</td>
<td>623</td>
<td>35</td>
<td>5.6</td>
<td>130</td>
</tr>
<tr>
<td>Denver</td>
<td>623</td>
<td>14</td>
<td>4.9</td>
<td>51</td>
</tr>
<tr>
<td>St. Louis</td>
<td>582</td>
<td>14</td>
<td>2.4</td>
<td>59</td>
</tr>
<tr>
<td>Philadelphia</td>
<td>556</td>
<td>13</td>
<td>2.3</td>
<td>60</td>
</tr>
<tr>
<td>Sacramento</td>
<td>711</td>
<td>16</td>
<td>2.3</td>
<td>104</td>
</tr>
<tr>
<td>Cincinnati</td>
<td>613</td>
<td>10</td>
<td>1.6</td>
<td>55</td>
</tr>
<tr>
<td>Santa Barbara</td>
<td>723</td>
<td>11</td>
<td>1.5</td>
<td>76</td>
</tr>
<tr>
<td>Washington, D.C.</td>
<td>577</td>
<td>7</td>
<td>1.2</td>
<td>65</td>
</tr>
<tr>
<td>San Francisco</td>
<td>647</td>
<td>6</td>
<td>0.9</td>
<td>29</td>
</tr>
<tr>
<td>Chicago</td>
<td>530</td>
<td>0</td>
<td>0</td>
<td>24</td>
</tr>
</tbody>
</table>

\(^{a,b}\) months of data beginning February, 1965.

3-2
hourly average concentrations for these same 12 sites is presented in Table 3-2.

At most stations listed in Table 3-2, the concentrations are equal to or greater than 80 μg/m³ (0.04 ppm) 30 percent of the time. Conversely, the concentrations are less than 80 μg/m³ (0.04 ppm) 70 percent of the time. The differences among stations are due almost entirely to the high concentrations, which occur less than about 10 percent of the time.

There does not appear to be a relationship between the ranking of stations by yearly average concentrations and the ranking by peak or maximum concentrations. The yearly average from Table 3-2 ranges from 37 to 82 μg/m³ (0.019 to 0.042 ppm), which is approximately in the range of ozone concentrations found in remote, unpopulated areas.

Caution, however, must be used in the interpretation of this latter observation as well as other conclusions which may be drawn from the data presented in Table 3-2. For example, one might, on the basis of yearly average, conclude that Los Angeles, San Diego, Denver, and Santa Barbara had similar oxidant problems. Yet examination of the data in Table 3-1 shows that the peak concentration, maximum hourly average, and percent of days with elevated oxidant concent-

Table 3-2. CUMULATIVE FREQUENCY DISTRIBUTION OF HOURLY AVERAGE OXIDANT CONCENTRATIONS IN SELECTED CITIES, 1964-1965

<table>
<thead>
<tr>
<th>City</th>
<th>Percent of hours with concentrations equal to or greater than stated concentrations, ppm</th>
<th>1964-1965 yearly average, ppm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>90</td>
<td>70</td>
</tr>
<tr>
<td>Pasadena</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>Los Angeles</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>San Diego</td>
<td>0.01</td>
<td>0.02</td>
</tr>
<tr>
<td>Denver</td>
<td>0.01</td>
<td>0.02</td>
</tr>
<tr>
<td>St. Louis</td>
<td>0.01</td>
<td>0.02</td>
</tr>
<tr>
<td>Philadelphia</td>
<td>0.01</td>
<td>0.02</td>
</tr>
<tr>
<td>Sacramento</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>Cincinnati</td>
<td>0.01</td>
<td>0.02</td>
</tr>
<tr>
<td>Santa Barbara</td>
<td>0.02</td>
<td>0.02</td>
</tr>
<tr>
<td>Washington, D.C.</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>San Francisco</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>Chicago</td>
<td>0.01</td>
<td>0.01</td>
</tr>
</tbody>
</table>

*11 months of data beginning February 1965.*
Substantial concentrations for most of the hours in a day; the yearly average in most cities is in the order of 50 μg/m³ (0.04 ppm). Assuming a background of 40 to 80 μg/m³ (0.02 to 0.04 ppm) ozone permits calculation indicating an ozone destruction rate of 1,960 to 5,880 μg/m³ (1 to 3 ppm) per hour. Thus any nonurban-formed ozone would stand little chance of existing in an urban atmosphere. The yearly average oxidant values, therefore, represent urban photochemically formed oxidants. These yearly values are low because 75 percent of the averaged values are necessary near zero, as previously indicated.

Table 3-3 presents, on a yearly basis, the maximum hourly average concentration and the number of days when the maximum hourly average exceeded specified values at each of the CAMP sites, from 1964 through 1967.

### Table 3-3. SUMMARY OF TOTAL OXIDANT CONCENTRATIONS RECORDED AT CAMP SITES, 1964-1967

<table>
<thead>
<tr>
<th>City</th>
<th>Year</th>
<th>Days of valid data</th>
<th>Number of days with at least 1 hourly average equal to or exceeding</th>
<th>Maximum hourly average, ppm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.05 ppm</td>
<td>0.10 ppm</td>
</tr>
<tr>
<td>Chicago</td>
<td>1964</td>
<td>254</td>
<td>149</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>1965</td>
<td>275</td>
<td>120</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>1966</td>
<td>235</td>
<td>52</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>1967</td>
<td>255</td>
<td>113</td>
<td>16</td>
</tr>
<tr>
<td>Cincinnati</td>
<td>1964</td>
<td>303</td>
<td>137</td>
<td>36</td>
</tr>
<tr>
<td></td>
<td>1965</td>
<td>310</td>
<td>182</td>
<td>19</td>
</tr>
<tr>
<td></td>
<td>1966</td>
<td>208</td>
<td>54</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>1967</td>
<td>228</td>
<td>122</td>
<td>24</td>
</tr>
<tr>
<td>Denver</td>
<td>1965</td>
<td>285</td>
<td>226</td>
<td>51</td>
</tr>
<tr>
<td></td>
<td>1966</td>
<td>298</td>
<td>187</td>
<td>46</td>
</tr>
<tr>
<td></td>
<td>1967</td>
<td>166</td>
<td>76</td>
<td>12</td>
</tr>
<tr>
<td>Philadelphia</td>
<td>1964</td>
<td>269</td>
<td>124</td>
<td>37</td>
</tr>
<tr>
<td></td>
<td>1965</td>
<td>266</td>
<td>109</td>
<td>23</td>
</tr>
<tr>
<td></td>
<td>1966</td>
<td>315</td>
<td>145</td>
<td>52</td>
</tr>
<tr>
<td></td>
<td>1967</td>
<td>282</td>
<td>124</td>
<td>28</td>
</tr>
<tr>
<td>St. Louis</td>
<td>1964</td>
<td>253</td>
<td>156</td>
<td>26</td>
</tr>
<tr>
<td></td>
<td>1965</td>
<td>329</td>
<td>206</td>
<td>33</td>
</tr>
<tr>
<td></td>
<td>1966</td>
<td>292</td>
<td>174</td>
<td>33</td>
</tr>
<tr>
<td></td>
<td>1967</td>
<td>289</td>
<td>185</td>
<td>38</td>
</tr>
<tr>
<td>Washington, D.C.</td>
<td>1964</td>
<td>293</td>
<td>163</td>
<td>40</td>
</tr>
<tr>
<td></td>
<td>1965</td>
<td>284</td>
<td>150</td>
<td>25</td>
</tr>
<tr>
<td></td>
<td>1966</td>
<td>325</td>
<td>134</td>
<td>27</td>
</tr>
<tr>
<td></td>
<td>1967</td>
<td>322</td>
<td>137</td>
<td>27</td>
</tr>
</tbody>
</table>

3. Seasonal and Diurnal Variations

Seasonal and diurnal variations in oxidant concentrations result largely from (1) variations in emissions of oxidant-forming pollutants, (2) variations in the atmospheric transport and dilution, and (3) variations in other atmospheric variables involved in the photochemical formation of oxidant. Typically, each of these factors varies significantly over periods as short as a few hours; the latter two also vary significantly seasonally. It is not unexpected, therefore, to find considerable variations in observed ambient oxidant concentrations.

At most of the stations, the highest monthly mean concentrations occur during the period from late spring to early fall. Oxidant concentrations exhibit a daily as well as a seasonal variation, and the maximum
generally occurs around the noon-hour, the period when the shorter wavelength solar radiation, which is photochemically important, reaches the surface of the earth with greatest intensity. Table 3-4 gives oxidant concentrations recorded in selected cities during the month having the highest mean 1-hour average concentration averaged for the years 1964 and 1965. For these months, the means of all hourly concentrations and the means of the maximum daily 1-hour average concentrations are also listed.

The seasonal variation of oxidant concentrations by month is illustrated for three of the stations in Figures 3-1 and 3-2. Figure 3-1 illustrates the mean by month of all hourly average concentrations for Los Angeles, Denver, and Phoenix. Figure 3-2 shows the mean by month of daily maximum 1-hour average concentrations for the same cities. In these figures, the importance of solar radiation is readily apparent. Note that for Denver, the high values occur around midsummer. For Los Angeles, the high values shift toward late summer and autumn, apparently due in part to slower wind speeds and less cloudiness in these seasons. In addition, the characteristics of atmospheric transport in Los Angeles are more favorable to a day-to-day carryover of precursor pollutants in autumn than in midsummer, the result of the greater balance between the sea and land breezes.

In Figure 3-3, the diurnal variations of mean 1-hour average oxidant concentrations are shown for Los Angeles and St. Louis. Selected for the Los Angeles presentation is the calendar month which had the highest monthly mean average for the years 1964 and 1965, while the St. Louis presentation illustrates the calendar month which had the highest monthly mean average in 1966. Illustrated by Figure 3-4 is the diurnal variation of mean 1-hour average oxidant concentrations for a 3-day period, August 6-8, 1966, in Philadelphia when unusually high concentrations of oxidants were recorded. While there are some differences, all curves of Figures 3-3 and 3-4 show a distinct peak around noon. This peak results largely from the interaction of diurnal variations in emissions, solar radiation intensity, and atmospheric dilution.

Peak emissions occur with the morning rush-hour traffic, at a time when solar radiation and dilution are weak. As the emissions drop off toward midmorning and midday,

<table>
<thead>
<tr>
<th>Station</th>
<th>Month having highest mean 1-hour average oxidant concentration</th>
<th>Monthly mean of hourly average concentrations, ppm</th>
<th>Monthly mean of maximum daily 1-hour average concentrations, ppm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pasadena</td>
<td>July</td>
<td>0.075</td>
<td>0.24</td>
</tr>
<tr>
<td>Los Angeles</td>
<td>August</td>
<td>0.056</td>
<td>0.17</td>
</tr>
<tr>
<td>San Diego</td>
<td>October</td>
<td>0.050</td>
<td>0.11</td>
</tr>
<tr>
<td>Denver(^a)</td>
<td>July(^a)</td>
<td>0.050</td>
<td>0.11</td>
</tr>
<tr>
<td>St. Louis</td>
<td>May</td>
<td>0.042</td>
<td>0.072</td>
</tr>
<tr>
<td>Philadelphia</td>
<td>July</td>
<td>0.054</td>
<td>0.11</td>
</tr>
<tr>
<td>Sacramento</td>
<td>June</td>
<td>0.040</td>
<td>0.075</td>
</tr>
<tr>
<td>Cincinnati</td>
<td>July</td>
<td>0.048</td>
<td>0.098</td>
</tr>
<tr>
<td>Santa Barbara</td>
<td>May and September(^b)</td>
<td>0.042(^b)</td>
<td>0.064 and 0.072</td>
</tr>
<tr>
<td>Washington</td>
<td>May</td>
<td>0.041</td>
<td>0.072</td>
</tr>
<tr>
<td>San Francisco</td>
<td>May</td>
<td>0.031</td>
<td>0.046</td>
</tr>
<tr>
<td>Chicago</td>
<td>April</td>
<td>0.044</td>
<td>0.070</td>
</tr>
</tbody>
</table>

\(^a\)11 months of data beginning February 1965.  
\(^b\)1964-1965 average for the months of May and September.
radiation increases to a maximum around noon; dilution increases rapidly in the fore-
noon to reach a maximum around midafter-
noon. Consequently, the diurnal variations of 
oxidant concentration typically show a peak 
around noon.

4. Oxidant Measurement Parameters

a. Sulfur Dioxide

Sulfur dioxide is a prevalent air pollutant in 
many areas of the country. It causes a 
negative response equal to its concentration 
when oxidants are measured by the potassium 
iodide method. If the atmospheric concentra-
tion of ozone and of sulfur dioxide were 0.5 
ppm each, for example, the potassium iodide 
method would indicate an oxidant-concen-
tration of zero, even though 0.5 ppm \( \text{O}_3 \) was 
present in the atmosphere.

To eliminate such sulfur dioxide inter-
ference, chromium trioxide scrubbers were 
attached to CAMP oxidant-analyzers at the 
beginning of 1964.\(^5\) The scrubbers were 
provided for all CAMP stations except the one 
located in San Francisco. The post-1963 
CAMP oxidant data presented, therefore, 
were not affected by sulfur dioxide inter-
ference. The extent of the interference at San 
Francisco and at other stations in California is 
generally very small because of the typically 
low sulfur dioxide concentrations found 
there.

b. Nitrogen Dioxide

The 1-hour-average concentrations of 
oxidant adjusted for nitrogen dioxide during 
selected summer and winter months for each 
of four stations are shown in Tables 3-5 and 
3-6. In these tables, the concentrations are 
presented both adjusted and unadjusted for 
nitrogen dioxide.

Table 3-5 lists the monthly mean hourly 
average concentrations, while Table 3-6 lists 
monthly means of daily maximum hourly 
average concentrations. It was assumed in 
calculating the adjustment that the nitrogen

---

![Graph](https://via.placeholder.com/150)

**Figure 3-1.** Monthly variation of mean hourly oxidant concentrations for three selected cities.
Figure 3-2. Monthly variation of mean daily maximum 1-hour average oxidant concentrations for three selected cities.

dioxide response of the potassium iodide oxidant-analyzer was 20 percent of the concentration of NO₂. Because the response is variable, the adjusted concentrations and the percentage of the measured oxidant which is due to nitrogen dioxide may not be reliable. The tables do show, however, that, on the average, the adjustment is greater during the winter than during the summer months. They also show that the adjustment for the hourly average is greater than for the daily maximum hourly averages.

c. Nitric Oxide

Nitric oxide, unlike SO₂ and NO₂, does not have a direct effect on the KI method of measurement. It has been found, however, that the chromium trioxide scrubbers used to remove the previously discussed SO₂ interference convert a portion of the ambient NO to NO₂. Thus, since 1964, the oxidant data from the CAMP network have been corrected for this secondary interference related to NO by use of the following relationship:

\[ \text{CAMP reported oxidant} = O_X - 0.11 \text{ NO} \]

where:
- \( O_X \) = measured oxidant concentration
- NO = measured nitric oxide concentration

C. CONCENTRATIONS OF OZONE IN URBAN ATMOSPHERES

1. Adjusted Oxidant

At 2 of the 12 stations discussed previously, Los Angeles and Pasadena, continuous ozone measurement has been conducted with ultraviolet photometers. Ozone concentrations measured by photometers are generally
Figure 3-3. Diurnal variation of mean hourly average oxidant concentrations in Los Angeles and St. Louis.

Figure 3-4. Diurnal variation of mean hourly average oxidant concentrations in Philadelphia, August 6-8, 1966.

Table 3-5. MONTHLY MEAN HOURLY AVERAGE OXIDANT CONCENTRATIONS ADJUSTED FOR NITROGEN DIOXIDE FOR SELECTED SUMMER AND WINTER MONTHS, IN FOUR CITIES

<table>
<thead>
<tr>
<th>Station</th>
<th>Month</th>
<th>Mean concentration, ppm</th>
<th>Calculated NO₂ interference, %</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Unadjusted</td>
<td>Adjusted</td>
</tr>
<tr>
<td>Los Angeles</td>
<td>(Summer)</td>
<td>0.055</td>
<td>0.044</td>
</tr>
<tr>
<td>Sacramento</td>
<td>July 1964</td>
<td>0.042</td>
<td>0.036</td>
</tr>
<tr>
<td>Denver</td>
<td>July 1965</td>
<td>0.048</td>
<td>0.041</td>
</tr>
<tr>
<td>St. Louis</td>
<td>July 1964</td>
<td>0.035</td>
<td>0.029</td>
</tr>
<tr>
<td>Los Angeles</td>
<td>(Winter)</td>
<td>0.021</td>
<td>0.005</td>
</tr>
<tr>
<td>Sacramento</td>
<td>Jan 1965</td>
<td>0.020</td>
<td>0.015</td>
</tr>
<tr>
<td>Denver</td>
<td>Feb 1965*</td>
<td>0.029</td>
<td>0.019</td>
</tr>
<tr>
<td>St. Louis</td>
<td>Jan 1965</td>
<td>0.024</td>
<td>0.020</td>
</tr>
</tbody>
</table>

*12 days of data only.

similar to oxidant concentrations measured by oxidant recorders.

In Figure 3-5, the monthly variation in means of hourly average concentrations of ozone are compared to analogous concentrations of oxidants in Pasadena and in Los Angeles. In Figure 3-6, the monthly variation in means of the daily maximum hourly averages are compared.

In Figure 3-7, hour-by-hour comparisons of 1-hour average ozone and oxidant concentrations for Los Angeles and Pasadena are shown for the month of July 1964. The line termed "adjusted oxidant" shown in Figure 3-7 is calculated from the following:

\[
\text{Adjusted oxidant} = O_X - 0.2 \text{ NO}_2 + \text{SO}_2
\]
Table 3-6. MONTHLY MEAN DAILY MAXIMUM 1-HOUR AVERAGE OXIDANT CONCENTRATIONS ADJUSTED FOR NITROGEN DIOXIDE FOR SELECTED SUMMER AND WINTER MONTHS, IN FOUR CITIES

<table>
<thead>
<tr>
<th>Station</th>
<th>Month</th>
<th>Mean concentration, ppm</th>
<th>Calculated NO₂ interference, %</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Unadjusted</td>
<td>Adjusted</td>
</tr>
<tr>
<td>Los Angeles</td>
<td>(Summer)</td>
<td>July 1964</td>
<td>0.166</td>
</tr>
<tr>
<td>Sacramento</td>
<td>July 1965</td>
<td></td>
<td>0.085</td>
</tr>
<tr>
<td>Denver</td>
<td>July 1965</td>
<td></td>
<td>0.110</td>
</tr>
<tr>
<td>St. Louis</td>
<td>July 1964</td>
<td></td>
<td>0.078</td>
</tr>
<tr>
<td>Los Angeles</td>
<td>(Winter)</td>
<td>Jan 1965</td>
<td>0.037</td>
</tr>
<tr>
<td>Sacramento</td>
<td>Jan 1965</td>
<td></td>
<td>0.035</td>
</tr>
<tr>
<td>Denver</td>
<td>Feb 1965(^b)</td>
<td></td>
<td>0.073</td>
</tr>
<tr>
<td>St. Louis</td>
<td>Jan 1965</td>
<td></td>
<td>0.046</td>
</tr>
</tbody>
</table>

\(^a\) days of data when NO₂ measured at time of maximum hourly average oxidant concentration.
\(^b\)12 days of data only.

Figure 3-5. Comparison of the monthly variation in mean 1-hour average ozone and oxidant concentrations in Los Angeles and Pasadena, 1964-1965.

where:

- \(O_X\) = measured oxidant concentration
- \(NO_2\) = measured nitrogen dioxide concentration
- \(SO_2\) = measured sulfur dioxide concentration

The specific equation required to calculate this "adjusted oxidant" value will depend on whether the KI instrument is equipped with chromium trioxide scrubbers. In the case of the California data shown in Figure 3-7, no such scrubbers were used and both the positive effect of \(NO_2\) and the negative effect of \(SO_2\) were considered in the calculation of adjusted oxidant.
Figure 3-6. Comparison of the monthly variation of mean daily maximum 1-hour average ozone and oxidant concentrations in Los Angeles and Pasadena, 1964-1965.

Figure 3-7. Comparison of the hourly variation of mean 1-hour average concentrations of ozone, oxidant, and oxidant adjusted for NO₂ and SO₂ response, Los Angeles and Pasadena, July 1964.
As previously noted, chromium trioxide SO$_2$ scrubbers have been used at CAMP stations since 1964. Adjusted oxidant values for the CAMP network, therefore, can be calculated by subtracting solely the effect of NO$_2$. Thus, adjusted oxidant from the raw CAMP data is calculated:

$$\text{Adjusted oxidant} = O_X - 0.2 \text{NO}_2 - 0.11 \text{NO}$$

where:

- $O_X$ = measured oxidant concentration
- NO$_2$ = measured nitrogen dioxide concentration
- NO = measured nitric oxide concentration

Specific values for the constants in the above equation are dependent upon individual instrument response. Therefore, calibration with known quantities of interfering compounds is suggested.

The close agreement between adjusted oxidant and ozone concentrations indicates that the contribution to oxidant measurement by peroxycetyl nitrate (PAN) or other oxidants is very low. As will be discussed later, PAN concentrations are usually much lower than ozone concentrations.

The agreement between the unadjusted oxidant and ozone concentrations is also good. Adjusting for SO$_2$ and NO$_2$ did not cause much change in the agreement between the oxidant and ozone concentrations because the response of the oxidant-recorder to NO$_2$ was largely compensated by the opposite response to SO$_2$. If SO$_2$ is not present, or if scrubbers are used, the measured oxidant concentrations will be higher than the ozone concentrations by the amount of the response to NO$_2$.

2. Ozone Concentration Patterns

In a recent study made by the Research Triangle Institute under contract to the Public Health Service, the daily maximum 1-hour average ozone concentration was recorded for certain cities, and the number of days when this daily maximum exceeded 100, 200, and 290 $\mu$g/m$^3$ (0.05, 0.10, and 0.15 ppm) is reported in Table 3-7. Table 3-8 lists the mean 1-hour average ozone concentrations by hour for each city involved in the 1967 portion of the same study. Figure 3-8 shows the diurnal patterns of 1-hour average ozone concentrations experienced on 2 selected days at the Denver and Philadelphia CAMP sites. This graph illustrates the high levels of ozone concentrations that have been reached.

### D. CONCENTRATIONS OF PEROXYACETYL NITRATE IN THE URBAN ATMOSPHERE

Utilizing gas chromatographic techniques with an electron-capture detector, PAN con-

<table>
<thead>
<tr>
<th>City</th>
<th>Time period</th>
<th>Number of days of valid data</th>
<th>Number of days with at least one hourly average equal to or exceeding</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.05 ppm</td>
</tr>
<tr>
<td>Cincinnati</td>
<td>9/15/67 to 9/29/67</td>
<td>15</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>10/3/67 to 10/16/67</td>
<td>15</td>
<td>3</td>
</tr>
<tr>
<td>Denver</td>
<td>10/17/67 to 11/1/67</td>
<td>15</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>8/10/68 to 10/11/68</td>
<td>58</td>
<td>51</td>
</tr>
<tr>
<td>Los Angeles</td>
<td>11/2/67 to 11/19/67</td>
<td>18</td>
<td>16</td>
</tr>
<tr>
<td>Philadelphia</td>
<td>6/1/68 to 7/31/68</td>
<td>59</td>
<td>47</td>
</tr>
</tbody>
</table>
Table 3-8. AVERAGE HOURLY OZONE CONCENTRATIONS IN SELECTED CITIES, 1967

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>0000</td>
<td>0.0032</td>
<td>0.0055</td>
<td>0.0119</td>
<td>0.0052</td>
</tr>
<tr>
<td>0100</td>
<td>0.0035</td>
<td>0.0065</td>
<td>0.0096</td>
<td>0.0078</td>
</tr>
<tr>
<td>0200</td>
<td>0.0036</td>
<td>0.0077</td>
<td>0.0103</td>
<td>0.0079</td>
</tr>
<tr>
<td>0300</td>
<td>0.0038</td>
<td>0.0061</td>
<td>0.0091</td>
<td>0.0074</td>
</tr>
<tr>
<td>0400</td>
<td>0.0031</td>
<td>0.0053</td>
<td>0.0108</td>
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</tr>
<tr>
<td>0500</td>
<td>0.0021</td>
<td>0.0040</td>
<td>0.0066</td>
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</tr>
<tr>
<td>0600</td>
<td>0.0010</td>
<td>0.0018</td>
<td>0.0033</td>
<td>0.0051</td>
</tr>
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<td>0700</td>
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<td>0.0041</td>
<td>0.0044</td>
</tr>
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<td>0.0108</td>
<td>0.0044</td>
<td>0.0067</td>
</tr>
<tr>
<td>0900</td>
<td>0.0075</td>
<td>0.0226</td>
<td>0.0078</td>
<td>0.0121</td>
</tr>
<tr>
<td>1000</td>
<td>0.0142</td>
<td>0.0292</td>
<td>0.0163</td>
<td>0.0383</td>
</tr>
<tr>
<td>1100</td>
<td>0.0178</td>
<td>0.0416</td>
<td>0.0270</td>
<td>0.0928</td>
</tr>
<tr>
<td>1200</td>
<td>0.0274</td>
<td>0.0444</td>
<td>0.0279</td>
<td>0.1204</td>
</tr>
<tr>
<td>1300</td>
<td>0.0282</td>
<td>0.0440</td>
<td>0.0338</td>
<td>0.1132</td>
</tr>
<tr>
<td>1400</td>
<td>0.0277</td>
<td>0.0369</td>
<td>0.0280</td>
<td>0.0872</td>
</tr>
<tr>
<td>1500</td>
<td>0.0197</td>
<td>0.0261</td>
<td>0.0271</td>
<td>0.0662</td>
</tr>
<tr>
<td>1600</td>
<td>0.0130</td>
<td>0.0178</td>
<td>0.0271</td>
<td>0.0238</td>
</tr>
<tr>
<td>1700</td>
<td>0.0055</td>
<td>0.0061</td>
<td>0.0232</td>
<td>0.0093</td>
</tr>
<tr>
<td>1800</td>
<td>0.0019</td>
<td>0.0043</td>
<td>0.0178</td>
<td>0.0048</td>
</tr>
<tr>
<td>1900</td>
<td>0.0019</td>
<td>0.0041</td>
<td>0.0162</td>
<td>0.0047</td>
</tr>
<tr>
<td>2000</td>
<td>0.0021</td>
<td>0.0039</td>
<td>0.0105</td>
<td>0.0046</td>
</tr>
<tr>
<td>2100</td>
<td>0.0023</td>
<td>0.0037</td>
<td>0.0110</td>
<td>0.0028</td>
</tr>
<tr>
<td>2200</td>
<td>0.0024</td>
<td>0.0037</td>
<td>0.0124</td>
<td>0.0021</td>
</tr>
<tr>
<td>2300</td>
<td>0.0028</td>
<td>0.0045</td>
<td>0.0122</td>
<td>0.0058</td>
</tr>
</tbody>
</table>

Concentrations were measured in Los Angeles during September and October of 1965, by the California Department of Public Health. Seven measurements per day were made for each of 16 weekdays in September and 19 weekdays in October. The mean 1-hour average concentrations of PAN and oxidant by hour of day for these periods are shown in Figure 3-9. The measurements were made near the downtown Los Angeles air-monitoring station.

![Graph showing diurnal variation of hourly ozone concentrations in Philadelphia and Denver.](image-url)

**Figure 3-8.** Diurnal variation of hourly ozone concentrations in Philadelphia and Denver.

3-12
Figure 3-9. Variation of mean 1-hour average oxidant and PAN concentrations, by hour of day, in downtown Los Angeles, 1965.
Beginning in June 1966, measurements of PAN have been made on the campus of the University of California at Riverside using the gas-chromatograph-with-electron-capture method mentioned. Samples are usually collected once each hour between 7:00 a.m. and 4:00 p.m., Pacific standard time. Other pollutants are also measured at this station which is operated by the Riverside Air Pollution Research Center and supported by the Public Health Service. In Figure 3-10, the mean 1-hour-average oxidant concentrations, as measured with a Mast analyzer, and the mean 1-hour-average PAN concentrations are shown by the hour of the day for the month of September 1966. The monthly mean hourly oxidant and PAN concentrations and the monthly mean of the daily maximum hourly average of 1 year's data are shown by month in Figure 3-11.

The comparison shown in Figure 3-11 is a good illustration of how specific averaging processes affect results. The considerable variations in daily maximum hourly concentrations as a function of time of year become much less obvious if the data include all hours. As stated previously, the latter damping effect is the result of including in the data those hours for which oxidant is necessarily at or near zero. Since these near-zero hours account for approximately 75 percent of the time of sampling, they have the effect of averaging out the elevated daytime values. Each method of averaging has its purpose, however, and subsequent interpretations of the results require careful consideration.

In Figure 3-10, there are two daily maxima for the oxidant and PAN concentrations. As will be discussed, the second maximum is probably due to the transport of pollutants from Los Angeles to Riverside. The PAN concentrations in Riverside are an order of magnitude lower than those in Los Angeles, while the concentrations of oxidants are of the same order of magnitude.

E. METEOROLOGICAL FACTORS

1. General Discussion

In any given area, the concentration of oxidant is dependent on many factors. Some of these, such as the concentration ratio of oxides of nitrogen to hydrocarbons, light intensity, and the reactivity of hydrocarbons, have been mentioned earlier. Other important factors are the size of the area, the meteorology, the topography, the number and distribution of sources, and the rates of emissions.

These latter factors are important because they affect the distribution of pollution over a city. Concentrations of oxidant on the upwind side of an area are substantially different from concentrations downwind. Oxidants may be transported many miles beyond the source area under certain meteorological and topographical conditions.

These spatial variations in concentrations make the location of air monitoring stations of great importance if representative air pollution data are to be collected for an area or city.
2. Transport

The transport of pollutants by wind in the Los Angeles Basin has been the subject of several studies.\textsuperscript{10,13,14} Most of the trajectories enter the basin from the west and, in general, surface winds are predominately from the ocean to the land during the spring, summer, and fall months.

To illustrate the eastward transport of oxidant, the diurnal variation of mean hourly average concentrations during October 1965, in West Los Angeles, Los Angeles, Azusa, and Riverside are shown in Figure 3-12. Data from the first three stations are from the Los Angeles County Air Pollution Control District, while data for the city of Riverside are from the Riverside County Air Pollution Control District.

The station at West Los Angeles is about 10 miles west, and Azusa is about 20 miles east, of downtown Los Angeles. Riverside is about 30 miles east of Azusa. As shown, the time of the peak oxidant concentrations follows those in West Los Angeles by about 1 hour in Los Angeles, 2 hours in Azusa, and 4 hours in Riverside.

Figure 3-12. Diurnal variation of mean 1-hour average oxidant concentrations at selected California sites, October 1965.
Oxidant concentrations at Riverside exhibited a double peak, as did the data on PAN and oxidant measured at the University of California at Riverside (Figure 3-10). The first peak at about 11:00 a.m. is attributed to pollutants generated at or near Riverside, while the peak at 4:00 p.m. is attributed to pollutants transported from the large and more densely populated Los Angeles metropolitan area.

As shown in Figure 3-13, which is a plot for carbon monoxide analogous to Figure 3-12, the afternoon peak concentration of carbon monoxide in Riverside is much smaller than the morning peak in Los Angeles. This suggests that the polluted air mass was diluted as it moved eastward to Riverside. On the other hand, the afternoon oxidant peak concentrations in Riverside were about as high as the peak concentrations in Los Angeles. It is possible, therefore, that, as the polluted air mass moved eastward, the oxidants continued to be formed at a rate about as great as the rate of dilution. It is also just as probable that the second peak at Riverside represents oxidant contributions from local as well as distant sources.

On the average, the direction of flow of surface wind and of pollutant transport appear to coincide, but they are not the only meteorological factors responsible for the transport of pollutants. Bell's appraisal of hour-by-hour development of oxidant concentrations indicates that other mechanisms, such as turbulence and downward motions created mechanically by airflow through mountain gaps, were also responsible for pollutant transport.\textsuperscript{11}

On some days, as observed by Stephens, the polluted air mass from the Los Angeles metropolitan area is defined by a sharp boundary which may not extend as far as Riverside.\textsuperscript{15} The reason for this sharp boundary, Stephens postulates, is that the temperature profile at the front of the air mass increased to the adiabatic lapse rate. At this point, rapid vertical ventilation had begun.

![Graph showing diurnal variation of mean 1-hour average carbon monoxide concentrations at selected California sites, October 1965.](image)

Figure 3-13. Diurnal variation of mean 1-hour average carbon monoxide concentrations at selected California sites, October 1965.

The prevailing winds are not always westerly. Under some meteorological conditions described by Bell,\textsuperscript{11} pollutants from Los Angeles have been transported out to sea and then southward to Oceanside\textsuperscript{14} and even to San Diego, a distance of over 100 miles. Under other conditions, pollutants have been transported from the sea northward to Ventura and Santa Barbara Counties.\textsuperscript{16, 17}

Preliminary data from a SCAN station recently established in Santa Cruz, California, indicate that a similar phenomenon may occur in the region of the San Francisco - Oakland metropolitan area. Under certain conditions, pollutants from the metropolitan area are transported out to sea and then brought back to shore by the local sea breeze to Santa Cruz, about 50 miles south. On these occasions, the hourly average oxidant concentrations have been as high as from 240 to
350 µg/m³ (0.12 to 0.18 ppm) in Santa Cruz. The very low concentrations of oxides of nitrogen and hydrocarbons measured during these occasions again suggest substantial oxidant formation in spite of high dilution.

3. Monitoring Station Location

Of the many air monitoring programs that measure oxidant values, the network operation of the Los Angeles County Air Pollution Control District has provided the most extensive experience. Determination of the number of sites and selection of the locations was regarded by its designers as one of the most important factors in planning a continuing air quality survey. From the early 1950's until July 1955, 14 stations were maintained. The locations of the 14 stations were selected on the basis of surface wind trajectories, population, and industrial concentrations. On the average, each station covered an area of about 70 square miles.

After evaluation of the data obtained during 1957 and 1965, it became apparent that fewer permanent stations strategically located in the most frequent pattern of the smog masses could achieve adequate warning of hazardous pollution levels. Subsequently, the number of stations for this purpose was reduced to six.

The Bay Area Pollution Control District also has a network of air monitoring stations. Examination of their data and data of the Los Angeles County Air Pollution Control District shows that average oxidant concentrations are not uniform throughout a metropolitan area.

In the Los Angeles Metropolitan Area during July 1965, for example, the mean of the daily maximum 1-hour average oxidant concentrations was 430 µg/m³ (0.22 ppm) at Burbank, 470 µg/m³ (0.24 ppm) at Pasadena, and 510 µg/m³ (0.26 ppm) at Azusa. On the other hand, in the city of Los Angeles, which is southerly and adjacent to both the Burbank and Pasadena stations, this mean was 270 µg/m³ (0.14 ppm). At North Long Beach, south of and adjacent to Los Angeles, it was 120 µg/m³ (0.06 ppm).

Thus, the data from a single CAMP or SCAN station may be indicative only of the concentrations in the immediate area where it is located. In order to determine the local variation in air quality throughout an urban area, a complete air sampling network is necessary.

F. SUMMARY

Examination of the aerometric data from several major U.S. cities indicates in all cases the presence of photochemically formed oxidants. This is not an unexpected finding since the precursor reactants, hydrocarbons and oxides of nitrogen, are inevitable by-products of current technology.

On a concentration basis, ozone has been identified as the major observed component of elevated oxidant levels. Demonstration of this fact, however, can be a most difficult task. One of the difficulties arises because the oxidant measuring method in common use is nonspecific and subject to several interferences, although it is true that the major atmospheric components which cause these interferences have been identified and thus can be given appropriate consideration. Nevertheless, the inherent danger of oxidant concentration artifacts must be borne in mind.

A complicating feature of oxidants is the fact that their appearance in the atmosphere is dependent on chemical reactions, and they occur, therefore, later in time than their precursor reactants. These chemical reactions are dependent on such variables as sunlight intensity, duration of sunlight, and temperature, in addition to variables affecting atmospheric dilution and dispersion. As a result, relationship between precursor emissions and atmospheric oxidant concentrations is much less direct and more difficult to quantify than is the case in the study of primary pollutants.

The second class of photochemical oxidants, peroxyacetyl nitrate, has not been routinely measured, although they have been identified in the atmosphere of several cities. These hydrocarbons peroxy derivatives have been shown to be intimately related to atmospheric photochemical ozone formation.
and therefore can be assumed to be present whenever oxidant levels are elevated.

By analysis of oxidant concentration data for 4 years at 12 stations, the daily maximum 1-hour-average concentration was shown to be equal to or exceeded 290 $\mu$g/m$^3$ (0.15 ppm) up to 41 percent of the time; the maximum 1-hour-average concentrations ranged from 250 to 1,140 $\mu$g/m$^3$ (0.13 to 0.58 ppm), with short-term peaks as high as 1,310 $\mu$g/m$^3$ (0.67 ppm); yearly averages for a 2-year period ranged from 370 to 820 $\mu$g/m$^3$ (0.19 to 0.42 ppm). Yearly averages are not representative of air quality with respect to oxidant pollution, however, because the 1-hour-average ozone concentration will necessarily be at or about zero for approximately 75 percent of the time, when photochemical reactions are minimal.

At four stations, maximum 1-hour-average ozone concentrations ranged from 140 to 510 $\mu$g/m$^3$ (0.07 to 0.26 ppm) in various studies. Adjusted oxidant and ozone concentration values are usually relatively the same.

G. REFERENCES

1. Air Quality Data from the National Air Sampling Network and Contributing State and Local Networks, 1966. Division of Air Pollution, Cincinnati, Ohio, 1967.
2. Data obtained from appropriate local agencies in California.
3. Data obtained from Maricopa County Health Dept. Phoenix, Arizona.
Chapter 4.

NATURAL SOURCES OF OZONE

A. INTRODUCTION

In order to make full use of aerometric data, it is necessary to know the relative magnitude of that portion of a pollutant's concentration which arises from nontechnological sources, often termed "background concentration." Such background concentration of an air pollutant represents an important factor in air pollution control, since it is a concentration which will persist even if all technological sources are eliminated. It is necessary to emphasize, however, that even in those cases where measurable natural emissions are considered relatively substantial, their contribution to current urban background concentrations is only a very small percentage of the total measure of ambient air quality. This is particularly the case with ozone, as indicated in this chapter. The largest natural source of ozone is the upper atmosphere, and a process of migration is therefore necessary in order to affect ground level air quality.

B. NATURAL SOURCES OF OZONE

Ozone can be formed naturally by electrical discharge. The electrical energy available from the atmosphere, however, is inadequate to form significant ozone concentrations over a large metropolitan area. Calculations reveal that in urban atmospheres the concentrations of ozone available from power-loss energy are several orders of magnitude less than 20 $\mu$g/m$^3$ (0.01 ppm).$^1$ Such a natural source of ozone, therefore, does not account for the high concentrations found in urban atmospheres.

Ozone is also formed naturally by the action of solar radiation in the stratosphere at altitudes of from 15,000 to 37,000 meters (50,000 to 120,000 feet). At these altitudes, ultraviolet radiation of wavelengths less than 2,450 Å causes oxygen molecules to photo lyze to atomic oxygen. The oxygen atoms then combine with oxygen molecules in the air to form ozone.

The total amount of ozone in the upper atmosphere, as measured spectrographically, has been found to vary between 2 and 4 millimeters.$^2$ The unit "millimeter of ozone" is the thickness the ozone layer would have if it were compressed at 1 atmosphere of pressure; 2 to 4 millimeters is equivalent, therefore, to between 490 and 980 $\mu$g/m$^3$ (0.25 to 0.50 ppm) for uniform distribution through a constant density atmosphere. Maximum ozone concentrations occur at altitudes of about 20 kilometers (12 miles).

The unit “centimeter of ozone per kilometer” (cm/km) also is used to express ozone concentrations determined spectrographically. Maximum ozone concentrations as high as 0.02 cm/km occur at altitudes of about 20 kilometers (12 miles).$^2$ At this altitude, the pressure is about 44 mm Hg. The concentration of 0.02 cm/km in terms of ppm is:

$$0.02 \text{ cm/km} \times 10^{-5} \text{ km/cm} \times 10^6 = 0.20 \text{ ppm}$$

If compressed with ozone-free air to sea level pressure, this concentration would be 390 $\mu$g/m$^3$ (0.20 ppm).

C. OZONE TRANSFER

Several explanations have been offered for the manner in which ozone is transferred from the upper atmosphere to the lower troposphere.$^2,^3$ The transfer apparently occurs in the vicinity of the jet stream and in weather-frontal zones. It is also postulated that such a transfer occurs directly across the tropopause region. The concentrations transferred to ground level, however, are not
believed to be high. Stable air systems, including temperature inversions, serve as barriers to the descent of stratospheric ozone. In the troposphere, ozone is destroyed by chemical, photochemical, and catalytic reactions; clouds and combustion gases rapidly dissociate ozone.

In the early 1950's, it was believed that stratospheric ozone was a significant source of surface ozone.\(^4\)\(^-\)\(^5\) Several investigators have reported concentrations of ozone or of oxidant up to a few-tenths of a ppm in nonurban areas.\(^5\)\(^-\)\(^7\)

It is now considered, however, that it is not possible to attribute high concentrations solely to natural sources with certainty, and that limitations in methodology may have contributed to the high readings.\(^7\) In agreement with those who postulate that high concentrations of stratospheric ozone do not reach ground level, certain researchers have found very little ozone at some remote sites. In general, it can be said that oxidant concentrations in remote areas range from less than 20 to about 100 $\mu g/m^3$ (0.01 to 0.05 ppm), with most of the measurements falling between 20 and 60 $\mu g/m^3$ (0.01 and 0.03 ppm). The proportion of these ozone concentrations which is due to transport from the stratosphere and the proportion which is due to transport from urban areas is uncertain. But, in any case, stratospheric ozone alone cannot account for the high concentrations found in urban atmospheres.

Ozone and oxidant concentrations have been measured in a variety of nonurban locations. The reported results of a number of these studies are discussed in this section.

In Greenland in July 1960, a maximum concentration of 25 $\mu g/m^3$ (0.013 ppm) was reported.\(^8\) In the Antarctic, the mean monthly surface ozone values from April 1957 to May 1958 ranged from 20 to 67 $\mu g/m^3$ (0.01 to 0.034 ppm).\(^9\) Measurements obtained at a French station in the Antarctic, from February 14, 1958 to January 15, 1959, indicated that daily mean concentrations during the months of September and October were almost tenfold higher than those of April and May.\(^10\) At the Amundsen-Scott station located at the geographical South Pole, ozone was measured using the chemiluminescent method of Regener from 1961 through 1964, and using the Mast method during 1963 and 1964. The monthly mean averages were from about 40 to 80 $\mu g/m^3$ (0.02 to 0.04 ppm) by the former method, and from 20 to 60 $\mu g/m^3$ (0.01 to 0.03 ppm) by the latter.\(^11\) Haagen-Smit, using the rubber-cracking method, reported concentrations of from 0 to 60 $\mu g/m^3$ (up to 0.03 ppm) in the beach and desert regions of California.\(^12\)

From December 1962 to August 1964, ozone concentrations were measured using balloonborne chemiluminescent ozone sondes at network stations of the Air Force Cambridge Research Laboratories.\(^13\) Of 867 soundings up to pressure levels of 500 millibars (18,280 feet or 5,570 meters at standard atmospheric conditions), 14 percent had maximum ozone concentrations greater than about 100 $\mu g/m^3$ (0.05 ppm) and 0.7 percent had maximum ozone concentrations greater than about 200 $\mu g/m^3$ (0.10 ppm). The concentration of 100 $\mu g/m^3$ (0.05 ppm) was exceeded most frequently at stations near large cities. The 200 $\mu g/m^3$ (0.10 ppm) concentration was exceeded at two stations only, near Bedford, Massachusetts, and Seattle, Washington.

When oxidants were measured at the Petawawa Forest, Chalk River, Ontario, the daily average concentration varied from slightly under 20 $\mu g/m^3$ (0.01 ppm) to slightly over 80 $\mu g/m^3$ (0.04 ppm).\(^14\) Measurements were made over a period of years using both the rubber-cracking method and a Mast meter, and the highest concentration encountered was 120 $\mu g/m^3$ (0.06 ppm) for 4 hours. Observed diurnal fluctuations revealed that oxidant values at night were usually less than 20 $\mu g/m^3$ (0.01 ppm), increasing in the morning hours to a peak around noon of 60 $\mu g/m^3$ (0.03 ppm), and then again dropping in the evening hours. From June 1, 1965, through August 13, 1965, measurements of
a 15-minute duration were made 24 hours a day. A total of 6,865 measurements were made. The mean concentration was 22 μg/m³ (0.011 ppm), and the maximum instantaneous reading was 120 μg/m³ (0.06 ppm). The cumulative frequency distribution of the concentrations measured in this study is shown in Figure 4-1.14

Using Mast analyzers, Berry determined the oxidant concentrations in valleys and on mountain tops in the Southern Appalachians.15 At one valley location, there was a strong diurnal variation, with a highest concentration of 80 μg/m³ (0.04 ppm) at about 2 p.m. Minimum concentrations, about 29 μg/m³ (0.015 ppm), occurred during the late evening and early morning hours. At other locations, the average oxidant concentrations generally ranged between 20 and 60 μg/m³ (0.01 and 0.03 ppm). At Green Knob, North Carolina, 2,394 instantaneous readings were made every 15 minutes from June 15, 1952, through July 11, 1962. The mean concentration was 33 μg/m³ (0.017 ppm), the maximum instantaneous reading was 140 μg/m³ (0.07 ppm), and the standard deviation was 16 μg/m³ (0.00790 ppm). At Pocahontas County in West Virginia, similar measurements were made from June 6 through July 6, 1961. Of 2,880 instantaneous measurements, the mean was 49 μg/m³ (0.025 ppm), the maximum was 125 μg/m³ (0.064 ppm), and the standard deviation was 28 μg/m³ (0.01475 ppm).16

In the summary of data by Junge, most of the ground-level concentrations reported were also in the range of about 20 to 60 μg/m³ (0.01 to 0.03 ppm).17 Spectrographic determinations indicate that, at surface level, the ozone concentrations range from 0 to 0.0004 cm/km (80 μg/m³, or 0.04 ppm).2

**D. SUMMARY**

Ozone can be formed naturally in the atmosphere by electrical discharge. Ozone is also formed in the stratosphere by the action of solar radiation, with maximum concentrations occurring at altitudes of about 20 kilometers (12 miles). These processes are not capable of forming significant concentrations at ground level over a large metropolitan area and thus do not account for the high concentrations of ozone found in some urban atmospheres. Maximum instantaneous ozone levels of from 20 to 100 μg/m³ (0.01 to 0.05 ppm) have been recorded in nonurban areas.

![Figure 4-1. Cumulative frequency distribution of average 15-minute ozone concentrations at Chalk River, Canada, June 1 to August 13, 1965.14](image-url)
E. REFERENCES

Chapter 5.

MEASUREMENT OF OXIDANTS, OZONE, AND PEROXYACETYL NITRATE IN AMBIENT AIR

A. INTRODUCTION

Total oxidants in the air may be defined as those compounds that will oxidize a reference material which is not capable of being oxidized by atmospheric oxygen. Methods for measuring oxidants, therefore, involve exposing an oxidizable substance to a sample of ambient air and determining the degree of oxidation which has occurred. There are many such methods, each one being dependent upon the reference substance chosen.

Two widely used methods of determining the degree of oxidation are colorimetric and coulometric analysis of the iodine released by atmospheric oxidants from a potassium iodide (KI) solution. Since these methods are the most widely used, they will be discussed in greater detail.

It should be recalled that:

1. Ambient air contains a mixture of oxidizing and reducing compounds—ozone, nitrogen dioxide, peroxyacetyl nitrate (PAN), sulfur dioxide, hydrogen sulfide, aldehydes, unsaturated hydrocarbons, and others.
2. The concentrations of these compounds may be constantly varying within relatively wide limits—from 0 to 1 ppm.

Since reducing compounds in air have an opposite effect on the reference material that is, cause a decrease in the degree of oxidation observed the result obtained by these KI methods is a “net” oxidant value rather than a total one unless empirical corrections are made. Thus the net value describes a condition of the air, rather than a specific compound concentration. For this reason, concentrated efforts are being made by researchers to obtain measurements for each specific oxidant present. Hopefully these efforts will enable air pollution scientists to better define this atmospheric “condition.”

Most oxidant data which have been collected are net determinations, and a high correlation between these values and other pollutant levels has been observed. Consequently until the more promising methods become routinely available net determination must serve as indicators of total oxidant levels. Oxidant measurement parameters are discussed in detail in Chapter 3.

It should be emphasized that in studies seeking a better evaluation of health effects, it is essential that data be obtained for individual oxidants, such as nitrogen dioxide, ozone, PAN, formaldehyde, acrolein, and organic peroxides, rather than data on total oxidants. Instrumentation currently available permits the accurate measurement of atmospheric ozone, nitrogen dioxide, and PAN. A further essential need exists to develop instrumentation capable of measuring the other individual gaseous pollutants which have the properties of oxidants.

Photochemical reactions and other problems derived from oxidants can be much better defined using specific methods for measurement of nitrogen dioxide and ozone, in preference to the traditional methods for determination of total oxidants.

B. REFERENCE METHOD FOR MEASUREMENT OF TOTAL OXIDANTS

Because of the complexity and variety of the methodology involved in oxidants determination, it has become necessary to adopt
one reference method for use in comparisons between techniques. The most widely used method for this purpose is the neutral-phosphate-buffered KI colorimetric technique described by Saltzman.\(^3\) In this method, oxidants are absorbed in neutral buffered 1 percent KI aqueous solution and the liberated tri-iodine ion is measured colorimetrically at 3,520 Å. Since one mole of ozone theoretically liberates one mole of iodine from this solution, the iodine measured is directly related to the concentration of ozone present in the air sample. Results may thus be expressed directly as equivalent volumes of ozone.

At present, there is no satisfactory means of testing this theoretical reaction. There is evidence that the amount of tri-iodine ions liberated by ozone is dependent on the design of the gas bubbler and on the phosphate concentration at neutral pH. Nevertheless, the error of measurement produced as a result of neglecting to account for the above variables is estimated to be no larger than ± 10 percent of the true value. Since ozone is believed to be the principal oxidant present in the atmosphere, this reference method has been adopted by NAPCA. A reference method in which the stoichiometry depends on fewer variables is, however, needed.

In addition to serving as a manual procedure for determining oxidants, the reference method may be used in conjunction with a “dynamic calibration” technique for instrumental methods, as follows: By passing scrubbed clean air over an ultraviolet lamp at a constant rate, ozone is generated at a constant rate. Analysis of the effluent with both the bubbler reference method and the particular system to be tested makes it possible to calibrate the various methods against this constant ozone source, and to express the results obtained as “ozone concentrations.” Since some instruments use different colorimetric techniques which require static liquid calibrations, comparisons of these liquid standards may be made using such a dynamic system. A diagram of the procedure is shown in Figure 5-1.

As a manual procedure for the determination of oxidants in the atmosphere, the neutral-buffered KI technique is useful if analysis is completed immediately after sampling. Rapid analysis is necessary because the iodine-color deteriorates with time. Concentrations of oxidant in a range from about 20 to 19,600 μg/m³ (0.01 to 10 ppm) can be determined using this method, although the accuracy for concentrations below 100 μg/m³ (0.05 ppm) is compromised because of the cumulative errors of sampling and analysis.

C. METHODS FOR MEASUREMENT OF TOTAL OXIDANTS

1. Continuous Methods Utilizing Potassium Iodide

a. Colorimetric

Continuous analysis of total oxidants is most commonly accomplished with instrumentation using a 10 to 20 percent solution of neutral–buffered potassium iodide as the absorbing medium. In devices using a colorimetric method sample air is passed at a known rate through a liquid absorbing reagent with a vacuum-pump arrangement. Oxidants in contact with the KI react to free iodine and the tri-iodine ion. These are determined colorimetrically in a continuous flow colorimeter at 3,520 Å. The continuous instrumental method is extremely sensitive to oxidizing substances and is suitable for determinations from 0 to 1,000 μg/m³ (0 to 0.5 ppm) ozone with an accuracy similar to the KI reference method. The negative interference caused by sulfur dioxide is often reduced by passing the air sample over chromium trioxide (CrO₃) scrubbers, but this technique has not been entirely successful, primarily because the effects of humidity on scrubber systems often render them ineffective.\(^3\) As discussed in Chapter 3, these scrubber systems also convert a portion of the ambient NO to NO₂, thus necessitating an additional correction factor. Despite these drawbacks, CrO₃ scrubbers remain the most generally used technique for removing SO₂ interferences; they are particularly useful for this purpose during periods of high SO₂ concentrations. Never-
theless, better methods for eliminating SO₂ interference are critically needed.

b. Coulometric

A coulometric method, commonly referred to as Mast-analysis, is also widely used for measurement of oxidants. In devices using this technique, sample air is passed in a manner similar to the colorimetric type of instrument into an electrolytic detector-cell containing KI. The free iodine liberated by the oxidants is reduced at the cathode of the cell, causing a current flow through an external circuit. The current flow is proportional to the amount of iodine liberated, and, in turn, to the oxidants entering the solutions. The current is measured with a microammeter which is usually calibrated directly in pphm of ozone.

A method utilizing this technique for the continuous measurement of total oxidants has been prepared for collaborative testing by the Intersociety Committee on Manual of Air Sampling and Analysis.5

c. Colorimetric versus Coulometric Methods

Since these are the types of instruments which are the most widely used for oxidant measurements, there have been studies conducted comparing the results obtained with the colorimetric and coulometric approaches. Field comparisons of measurements from colorimetric and coulometric oxidant analyzers, where readings did not exceed 390 μg/m³ (0.20 ppm) and corrections were made for NO₂ interferences, indicate that differences are no greater than would be expected if two colorimetric-type instruments were working side-by-side.6 Comparative data, including readings greater than 390 μg/m³ (0.20 ppm), show a correlation coefficient of 0.87 between readings of the two different instruments, both calibrated with respect to
known ozone-streams. In spite of the relatively good correlation obtained in this study, there were some instances when the agreement between the two methods was poor. To resolve some of these discrepancies, more comparative studies of this type are needed.

Perhaps the largest variations between the two methods are related to the differences in reagent formulation. Generally, 10 to 20 percent KI solutions are used in colorimetric instruments; at these concentrations, response to nitrogen dioxide is higher than at the 2 percent level used with coulometric instruments. Results of several studies are shown in Table 5-1. Analyzers utilizing other principles of detection vary in their sensitivity to nitrogen dioxide. As shown in Table 5-1, the coulometric cell analyzers register about 10 percent of the nitrogen dioxide concentration as ozone, and the U. V. photometer device shows the least interference from nitrogen dioxide.

Studies comparing the two methods indicate that both instruments must be checked and calibrated frequently and that variations are usually caused by changes occurring in the airflow or reagent flow and by interfering compounds. Table 5-2 gives some indication of the reliability of the continuous colorimetric oxidant analyzers used at seven California stations. These analyzers are dynamically and statically calibrated about twice a

<table>
<thead>
<tr>
<th>Range of NO₂ concentration in air, ppm</th>
<th>Method</th>
<th>NO₂ concentration (as ozone), %</th>
<th>Reference</th>
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</thead>
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<tr>
<td>&gt; 0 to 36</td>
<td>1% KI, pH 7 bubbler</td>
<td>8 to 11</td>
<td>8</td>
</tr>
<tr>
<td>1.5 to 5.5</td>
<td>2% KI, pH 7 bubbler</td>
<td>6.4</td>
<td>9</td>
</tr>
<tr>
<td>2.0</td>
<td>10% KI, pH 7 contact column</td>
<td>21</td>
<td>7</td>
</tr>
<tr>
<td>&gt; 0 to 1</td>
<td>20% KI, pH 7 contact column</td>
<td>30</td>
<td>8</td>
</tr>
<tr>
<td>0.24 to 0.43</td>
<td>20% KI, pH 7 contact column</td>
<td>12 to 47 (average: 25)</td>
<td>10</td>
</tr>
<tr>
<td>2.0</td>
<td>coulombic cell</td>
<td>10</td>
<td>7</td>
</tr>
<tr>
<td>2.0</td>
<td>U.V. photometer</td>
<td>2</td>
<td>7</td>
</tr>
</tbody>
</table>

*aBased on 4-liter sample.

<table>
<thead>
<tr>
<th>Change from previous calibration, %</th>
<th>Number of stations</th>
<th>Occurrences</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>% of total</td>
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<tr>
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<td>± 11 to 20</td>
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</tr>
<tr>
<td>± 31 to 40</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>
year. The percentage of change from the previous calibration indicates the uncertainty in measurements obtained during the time between calibrations. Table 5-2 shows that 62 percent of the time there was less than a 10 percent change, and 78 percent of the time there was less than a 20 percent change. Changes greater than 20 percent occurred at three of the seven stations, which indicates that some stations provide more reliable data than others.

2. Other Methods - Intermittent Sampling

a. Ferrous Ammonium Sulfate

The ferrous ammonium sulfate measurement technique is in use on an experimental basis by NAPCA because of its possible application to intermittent sampling by the gas sampling network. This method is based on the conversion of the ferrous ion by oxidation to the ferric ion. Samples of ambient air are drawn through an acidified ferrous ammonium sulfate solution. The ferric ion formed in the collection solution is complexed in the laboratory with ammonium thiocyanate, and the resultant color is measured on a spectrophotometer. The oxidized solution is stable after sampling and can be analyzed later. Depending on the sample flow rate and interval of collection, this method is useful in the range of 1 to 2,400 µg/m³ (0.0005 to 1.2 ppm). As with the KI method, this method suffers from the drawback of interferences from reducing compounds in the atmosphere. Another weakness is the lack of agreement with results obtained by other methods.

b. Alkaline Potassium Iodide

This method³ is intended for the manual determination of oxidants in a range from a few µg/m³ to about 39,000 µg/m³ (20 ppm). The advantage of this procedure over the neutral iodide, or reference, technique is that a delay between sampling and completion of analysis is allowed. Sampling is conducted in midget impingers containing 1 percent KI in a 1-normal sodium hydroxide solution. Because of the simplicity of the preferred reference neutral-buffered KI method, however, the alkaline procedure is not widely used.

c. Phenolphthalain

The phenolphthalain method is based on the oxidation of phenolphthalain to phenolphthalein by the oxidant present in the atmosphere. Phenolphthalein is pink in alkaline solution. Because the technique responds to pH and temperature changes, it also is not widely used at present, although it was used extensively to obtain oxidant data in the past. Measurement results obtained by this method are approximately twice those obtained by the KI method. Consequently, when data obtained by the two methods are compared, it is necessary to adjust for this difference.

D. METHODS SPECIFICALLY FOR MEASUREMENT OF OZONE

1. Chemiluminescence

The chemiluminescent method of measuring ozone was developed by Regener.¹¹,¹² If air containing ozone is passed in the dark across the surface of a chemiluminescent or fluorescent substance such as Rhodamine B adsorbed on silica gel, light is emitted. The intensity of emitted light can be measured with a photomultiplier tube. The chemiluminescent reaction between the ozone and Rhodamine B is highly specific to ozone and concentrations less than 2 µg/m³ (0.001 ppm) can be measured. Nitrogen dioxide, sulfur dioxide, and peroxyacetyl nitrate give negligible, if any, interference. The sensitivity for ozone detection of the chemiluminescent surface gradually decays with time, and daily calibration with a constant ozone source is required. Although the method has not been widely used for routine ambient air monitoring, it shows considerable promise and potential for this application and represents a needed attempt to obtain ozone specificity in measurement. Devices of this type are currently being thoroughly evaluated in the field and are available commercially.
2. Ultraviolet Photometry

Two instruments have been developed based on ozone's absorption of ultraviolet radiation. Ozone is unique in having a high optical opacity within the wavelength band centered at 2,500 to 2,600 \( \text{Å} \).\(^{13}\) The first instrument, based on the researches of Stairs,\(^{14}\) required an optical path-length of 250 feet between source and receiver units, which was a major difficulty.\(^{15}\)

The second instrument designed to monitor ozone is one developed and previously manufactured by the Harold Kruger Instrument Company. It was of a more conventional design, using 10-inch cells, a single source of radiation, and a double path of fixed wavelength at 3,537 \( \text{Å} \). One cell had ambient air passing through it, while the other cell sampled air with the ozone removed by catalytic decomposition.\(^{15}\)

The Kruger photometer was available commercially for several years but, because of electronic instability and temperature dependence, it required frequent attention. As a result, it was not used extensively. It is not available presently.

3. Trans-butene-2 Gas Phase Titration

This method, which has been only recently described,\(^{16}\) involves a periodic gas-phase titration of the air sample with trans-butene-2 prior to analysis for total oxidants. This reactive olefin selectively removes the ozone present in the air stream and provides a specific ozone concentration by difference. The technique is applicable to coulometric and colorimetric methods of iodine measurement but has been evaluated more fully with a colorimetric instrument.\(^{17}\)

4. Rubber Cracking

One of the earliest techniques used to identify ozone in ambient air was based on the rapid cracking of stressed rubber strips. A strip of a suitable rubber is bent double, stretched, and exposed to the air. Either the time until the rubber begins to crack or the depth of crack after a specified time can be related to the ozone concentration. Although it does not provide a direct quantitative measurement, this technique is useful for survey work, as it involves inexpensive equipment and is a simple operation. It cannot, however, be automated to obtain continuous data and, although presumed specific for ozone, other reactive species such as free radicals may contribute to rubber cracking over extended periods.\(^{18}\)

5. Other Chemical Methods

Hauser and Bradley\(^{19}\) have described a specific ozone technique using 1,2-Di-(4 pyridyl) ethylene as the absorbing medium and colorimetric analysis of the resulting aldehyde by the 3-methyl-2-benzothiazolone hydrazone method.\(^{3}\) This method can be applied in field analysis of atmospheric ozone because sampling and analysis need not be performed on the same day.

1-hexene, hydrogen peroxide, and PAN were found to interfere when a 24-hour sampling period was used. Glacial acetic acid, which is used in the collection solution, also presents a problem since it cannot be sent through the mails. Furthermore, the absorbing solution used freezes at 16° C and must be artificially warmed if used below this temperature. For a 30-minute sample collected in a bubbler at 0.5 liters/minute, the range of measurement for this technique is 100 to 2,300 \( \mu \text{g}/\text{m}^3 \) (0.05 to 1.2 ppm) ozone.

E. METHODS FOR DETERMINATION OF PEROXYACETYL NITRATE

1. Long-path Infrared Spectroscopy

Peroxyacetyl nitrates were discovered using long-path infrared spectroscopy. Although water vapor interferes with the principal absorption band used in this determination, an alternate band may be used to avoid this effect. Strong bands are located at 5.75 and 8.62 \( \mu \) and weaker bands at 7.3, 9.5, 10.0, 10.8, and 12.2 \( \mu \). A 500-meter-long-path infrared cell is used to detect PAN in polluted ambient air.\(^{20}\)
2. Gas Chromatography

Increased sensitivity for the detection of PAN is provided by gas chromatography using an electron-capture detector.\textsuperscript{21} The advantage of this detector is its relative insensitivity to other compounds which might interfere.

Short columns (9 to 18 inches in length by 1/8 inch in diameter) packed with carbowax coated on an inert substance are used for the separation. Retention time for PAN is 1 to 2 minutes. Two-milliliter samples containing 5 µg/m\(^3\) (0.001 ppm) PAN give detectable peaks. This method has been automated, but currently it is used primarily as part of a manual sampling program.

F. SUMMARY

The most widely used method for the analysis of atmospheric total oxidants is the neutral-buffered KI technique. This technique is currently recommended by NAPCA as the reference method. Oxidizing species in the atmosphere react with potassium iodide to release iodine. The iodine may then be measured either spectrophotometrically or coulometrically. Both of these principles have been used in instruments which are suitable for continuous determinations from 0 to 1,000 µg/m\(^3\) (0 to 0.5 ppm) ozone with a sensitivity of about 10 µg/m\(^3\) (0.005 ppm). The spectrophotometric technique also may be used on an intermittent basis provided that analysis is completed within 5 minutes after sampling. This limit is necessary because of the iodine color deterioration with time.

Although currently there is no widely used method for the specific measurement of ozone in the atmosphere, there are several promising instruments being evaluated for this purpose. These include a device employing the chemiluminescent reaction between ozone and Rhodamine B and measurement of the emitted light; concentrations less than 2 µg/m\(^3\) (0.001 ppm) can be measured by this technique. Another method uses a gas phase titration of the air sample with trans-butene-2 to selectively remove the ozone present. This technique is applicable to coulometric or colorimetric methods of iodine measurement, the concentration of ozone in the sample being obtained by difference. Chemical methods for the specific manual determination of ozone include the 1,2-Di-(4 pyridyl) ethylene method which may be used for 30-minute or 24-hour sampling periods. Though this technique suffers from several operational drawbacks, it is capable of measuring ozone concentrations from 100 to 2,300 µg/m\(^3\) (0.05 to 1.2 ppm) for a 30-minute sample.

The methods generally used for the measurement of PAN include long-path infrared spectroscopy and, more commonly, gas chromatography using an electron-capture detector. Sensitivity is in the µg/m\(^3\) (ppb) range and the latter procedure has been automated by some researchers.

There is a further need for the development of instruments capable of continuously measuring other individual oxidants such as formaldehyde, acrolein, and organic peroxides. Acquisition of data on ambient levels of individual oxidants will facilitate interpretation of the results of studies on the effects of photochemical oxidants.

G. REFERENCES


Chapter 6.

EFFECTS OF PHOTOCHEMICAL OXIDANTS ON VEGETATION AND CERTAIN MICROORGANISMS

A. INTRODUCTION

Injury to vegetation was one of the earliest manifestations of photochemical air pollution. A peculiar type of injury to leafy vegetables, ornamentals, and field crops, now characterized by banding, silvering, and stippling of the leaves, was first investigated by Middleton et al. in 1944 in a small area of Los Angeles County. By 1950, such injury had spread over a large segment of southern California and the San Francisco Bay Area. Plant injury of this kind has since spread to many widely separated areas of the United States, with increasing severity and with associated economic losses to both farmers and nurserymen.

Three specific phytotoxic materials have been isolated from the photochemical complex: ozone, nitrogen dioxide, and the peroxyacyl nitrates. The latter homologous series of compounds includes peroxyacetyl nitrate (PAN), peroxypropionyl nitrate (PPN), peroxybutyryl nitrate (PBN), and peroxyisobutyryl nitrate (PISOBN). Preliminary work has shown that PPN is several times as toxic to vegetation as PAN, while PBN and PISOBN are more toxic than PPN. Since PAN is the only member of the series which has received much study, and also since PPN and PBN are usually present but below detectable limits, discussion will be restricted to the effects of PAN. Discussion of the quantitative effects of ozone and PAN has to be limited to laboratory and controlled field exposures since, under ambient conditions, the effects of these compounds cannot be easily differentiated. The term “oxidant” will be used when discussing the toxic materials to which the plants are exposed under ambient conditions. Research in several laboratories using a variety of reaction systems suggests that additional phytotoxicants may be present in the photochemical complex. Synergistic effects between the toxicants discussed and other atmospheric contaminants may also produce injury to sensitive plant species. Available information suggests that ozone is the most important phytotoxicant of the photochemical complex.

Sensitive plants are useful biological indicators of photochemical air pollution. The most detailed study of this phenomenon was made in Los Angeles, with annual bluegrass and petunia as monitoring species. This study was designed to use plants to determine relative concentrations of photochemical pollution by attempting to establish a relationship between oxidant concentrations and plant injury. A similar survey used pinto beans, grown under greenhouse conditions. The injured leaf area in both studies was measured and showed poor correlation with oxidant levels, suggesting that unknown factors were affecting sensitivity, or that the oxidant index did not accurately measure the phytotoxic substances in the air. Tobacco has also been widely used as a monitor for photochemical pollutants. It was first suggested because its injury pattern seemed specific for the PAN-type of injury. The injury pattern and sensitivity of tobacco to ozone makes it a useful monitor in studying the extent, severity, and frequency of ambient oxidant.
B. SYMPTOMS OF THE EFFECTS OF PHOTOCHEMICAL AIR POLLUTANTS ON VEGETATION

The visible symptoms of injury to plants attributable to oxidant air pollution can be classified into three general categories: acute injury, severe injury occurring within several hours after exposure, identified by cell collapse with subsequent development of identifiable necrotic patterns; chronic injury, light to severe injury developing over an extended time period, identified by necrotic patterns with or without chlorotic or other pigmented patterns found in sensitive leaf tissues and not easily attributable to other than oxidant air pollutants; and physiological effects, including growth alterations, reduced yields, and changes in the quality of plant products. The acute symptoms are generally characteristic of a specific pollutant. Chronic injury patterns are often highly characteristic but by no means specific for the toxic agents. Disease, insects, nutrition, and other factors can produce leaf patterns similar to those induced by air pollution.

The most easily recognized effect of air pollutants is the necrotic patterns which develop after the occurrence of injury to cells and final collapse of the tissue. The initial plasmolysis is due to changes in cell-wall permeability and then to changes in structural integrity; a slightly water-soaked or bruised-looking area appears in the intact leaf. These areas generally dry out, leaving necrotic patterns characteristic of the toxicant.

Ozone-type injury to field crops was first observed as a "stippling" on grape leaves in California.49,50 Heggestad and Middleton51 found that the tobacco "weather fleck" symptoms found along the eastern seaboard could be reproduced by exposing tobacco to ozone fumigations. Daines et al.52,53 reported ozone-type injury to several crop plants in New Jersey, and indicated that continued production of these crops was seriously threatened. Since these early reports, ozone-type injury to sensitive crop plants has been reported from Florida to southern Ontario.54-62 The conifer "x-disease"63 and the citrus decline in California26 have also been related to ozone injury.

The initial effect of ozone is in the palisade cell layer and involves scattered groups of cells. In acute injury, the cells plasmolyze and the cell contents become disrupted and disintegrate with or without the production of a dark pigment, called "stipple" in the former case and "fleck" in the latter. With continuing exposure or with a high-level exposure, these upper-surface necrotic areas will enlarge, coalesce, and eventually injure the spongy cells and form necrotic lesions through the leaf. Ozone enters the leaf through the stomata, but the gas preferentially attacks the palisade cells. A detailed discussion of ozone symptoms with illustrations of several plants has recently been published.64

PAN-type injury, characterized by under-surface glazing or bronzing of the leaves of many plant species, has been observed primarily in California and in the states along the eastern seaboard.22,25,52,65 This type of injury has also been reported in other parts of the United States and in several metropolitan areas of foreign countries.21,66 Injury occurring in the field has been identified in spinach, beets, celery, tobacco, endive, romaine lettuce, Swiss chard, pepper, alfalfa, petunias, snapdragon, primrose, asters, and other plants.13,14,25,53

PAN has been accepted as the primary phytotoxicant which causes the oxidant-type injury initially described by Middleton et al.12 and more completely by Bobrov et al.3 and Glater et al.4,7 The initial collapse is in the spongy cells surrounding the air space into which a stomata opens. The effect in some cases is limited for the most part to cells nearest the lower epidermis. This results in a slight separation of the lower leaf epidermis, which produces a characteristic under-surface silvering, glazing, or bronzing. More acute injury causes the necrosis to extend through the entire leaf. Injury to the leaves of grasses, petunia, and tobacco causes a crossleaf banding associated with the sequential maturation
of cells from the tip to the base of the leaf. A detailed discussion of PAN symptoms with illustrations has recently been published.6,7

Chronic injury may be characterized by chlorosis or other color changes, in addition to several necrotic patterns. Chlorosis, the loss or reduction of chlorophyll, is a very common and nonspecific symptom in plants. Its appearance generally signifies either a deficiency of some nutrient required by the plant or a general metabolic disturbance. The loss of chlorophyll results in leaves with a pale-green or yellow color pattern. There are other color changes in leaves, often with general chlorosis, which are associated with maturity or senescence, e.g., the color change in the autumn. There are cases where the photochemical complex or its specific components will produce chlorosis in leaves which have been exposed to the pollutant at low levels over a long period of time. The pattern, however, is usually not distinctive, appearing only as an early senescence. The pigmentation of small areas of the palisade cells (stipple) seems to be characteristic of ozone injury in some plants, as a silvering or bronzing of the undersurface of some leaves is associated with PAN injury.

Physiological effects causing growth- and yield-reductions have been experienced in several areas of the United States.2,5,25 A serious decline in citrus, manifested by early loss of leaves, changes in water relationships, smaller fruit, and poor growth,24,26 is currently being experienced in the Los Angeles area. Similar effects are detected in vegetable and other horticultural crops of the area. Photochemical pollutants such as ozone and PAN have been suggested as responsible.26 Feder and Campbell6,8 exposed carnations continuously over a 2-month period to low levels of ozone [100 to 200 μg/m³ (0.05 to 0.10 ppm)] and reported a significant reduction in the number of flowers as well as a reduction in height and flower-stock length. Time to produce flowers was also lengthened. Experimentation has shown that some of these effects, such as early leaf drop, changes in water relationships, increased respiration, sub-normal growth of vegetables, and effects on flower development, can be alleviated by exposure to air filtered through activated carbon to remove photochemical oxidants.7,6,9 It should be noted that reduced growth and yields can occur without visible injury to the leaves.3,5,36

Taylor70 has reported on observed physiological upsets in trees due to oxidants. Others have reported on generally poor growth and early senescence due to oxidants.7 Hill and Littlefield71 have reported a reduction in photosynthesis at ozone concentrations which caused only slight visible injury. At the present time, there is no sound basis for accurately evaluating the significance of these observations and results. Additional work of this type, together with basic work such as that reviewed by Dugger et al.,72 will be required before an evaluation can be made of the physiological and biochemical effects of photochemical oxidants on the intact plant.

C. FACTORS AFFECTING RESPONSE OF VEGETATION TO PHOTOCHEMICAL AIR POLLUTANTS7,3

The response of a given species or variety of plant to a specific air pollutant cannot be predetermined on the basis of the known response of related plants to the same pollutant. Neither can the response be predetermined by the given known response of the plant to similar doses of a different pollutant. Genetic susceptibility and environmental influences must, therefore, be determined for each plant and pollutant.

1. Genetic Factors

Variability in response to pollutants is known to exist between species of a given genus and between varieties within a given species. Species variability has been well-documented in numerous genera, and major varietal or strain differences have been shown in such species as petunia, tobacco, white pine, soybean, tomato, and radish.5,9,74,78

Varietal variations have been studied most extensively in the species Nicotiana tabacum. Heggestad and Mense4,8 isolated and devel-
oped a sensitive tobacco strain (Bel-W3) and have compared its sensitivity to a number of other varieties. Macdowall et al.\textsuperscript{59} tested 32 varieties of tobacco and found the White Gold variety to be most susceptible. Menser\textsuperscript{76} and Reinert et al.\textsuperscript{77} have also studied and reported on varietal response differences in the field and laboratory associated with oxidant or ozone exposures. Injury to susceptible varieties of tobacco in the Connecticut River Valley fields and elsewhere has led to the development of more resistant varieties for commercial use. The more sensitive varieties are being used in monitoring programs and as biological material for studying the effects of air pollutants on plants.

A study was conducted in Wisconsin on varietal ozone susceptibility.\textsuperscript{79,80} Ozone-resistance in onions was found to be related to a dominant gene which controlled the response of the guard cells to ozone. This single-factor inheritance of sensitivity of onion to ozone is perhaps unique, but it deserves further investigation.

Feder\textsuperscript{81} reported a 40 percent reduction in pollen germination and a 50 percent reduction in pollen tube growth following a 5- to 24-hour exposure of tobacco to 200 $\mu$g/m$^3$ (0.10 ppm) of ozone. This could represent a major loss in reproductive capacity.

2. Environmental Factors

Field and laboratory observations and experiments have shown that the environmental conditions under which plants are grown and/or exposed have a major influence on their sensitivity to phytotoxic air pollutants. Macdowall et al.\textsuperscript{58} correlated the sensitivity of tobacco with oxidant levels and found that sensitivity is related to general soil conditions and to the interaction of several meteorological factors such as temperature, wind speed and direction, humidity, and light. By using these factors and an empirical factor (the coefficient of evaporation) and relating them to oxidant dose, the authors had some success in forecasting tobacco-fleck attacks.

During growth, the influence of light intensity on the sensitivity of plants is dependent on the phytotoxicant. Plants are more sensitive to PAN when grown under high light intensities,\textsuperscript{82} but are more sensitive to ozone when grown under low light intensities.\textsuperscript{82-84} Sensitivity of Poa annua during exposure to oxidant shows a positive correlation with increasing light intensity to at least 3,000 foot-candles.\textsuperscript{40} A similar response of plants to ozone has been reported.\textsuperscript{85} For some pollutants and with specific plants, sensitivity may increase with increasing light intensity up to full sunlight.

In a study on the standardization of Poa annua response as an index of smog concentrations, it was found that sensitivity increased as temperatures increased from 40° to 85° F.\textsuperscript{40} When plants are exposed under greenhouse conditions, it is almost impossible to separate the effect of temperature at the time of exposure from the effect of light intensity since a positive correlation exists between these two factors. Reported findings are in general agreement that sensitivity of plants to oxidants increases with temperature from 40° to 100° F. However, when plants were exposed to ozone under controlled lighting conditions,\textsuperscript{85} there was an inverse relationship between temperature and sensitivity as the temperature was raised from 65° to 85° F. This suggests that the positive correlation between sensitivity and temperature found under ambient conditions is due to the overriding influence of light intensity.

The effects of humidity during growth and exposure have not been well documented. Early work suggests that plants grown and/or exposed under high humidities are more sensitive than those grown at low humidities. The results of Menser\textsuperscript{86} on exposure of sensitive tobacco to ozone suggests the same trend; however, his results are not conclusive. Otto and Daines\textsuperscript{87} found a marked reduction in sensitivity of pinto bean and tobacco to ozone when they were exposed at 26 percent relative humidity as opposed to 51 percent relative humidity. However, they found varied plant responses to higher humidities. These
findings are in general agreement with earlier work. The overall importance of humidity is still poorly understood.\textsuperscript{73} Heggestad et al.\textsuperscript{88} noted that tobacco plants grown out-of-doors and in greenhouses in Utah and California were not as sensitive to either ambient oxidant or ozone as similar plants grown in eastern states where the relative humidity is higher.

The increased use of carbon dioxide as an additive for increased production in greenhouses may act to reduce the sensitivity to photochemical pollutants of plants grown under these conditions.\textsuperscript{83}

The relative importance of some meteorological factors, such as wind speed and barometric pressure, has seemed insignificant in relation to the primary factors already discussed. Plants appear to be more sensitive when high pollution levels occur from mid-morning to early afternoon than at other times during the day or night.

Research has largely neglected the influence of soil factors on the sensitivity of plants to phytotoxic air pollutants. There are numerous reports that plants grown under drought conditions are less susceptible to air pollutants than those grown under moist conditions. Field observations\textsuperscript{61} have shown that tobacco plants irrigated just prior to a natural fumigation are more sensitive than unirrigated plants, even when the unirrigated plants have a sufficient water supply to prevent wilting. Withholding of water from greenhouse and irrigated crops during times of high pollution potential has been recommended by several researchers as a preventive measure against pollution injury.

Various studies have shown that plant sensitivity to phytotoxic air pollutants increases when they are grown under low total-soil-fertility. This has been found under both natural and laboratory conditions. Other studies have shown that an increased nitrogen supply makes plants more sensitive, although differing results have been reported. General observation suggests that the healthier the plant, the greater its sensitivity toward air pollutants.\textsuperscript{73}

Plants are not as sensitive when grown in heavy soils. The effects of soil temperature, aeration (both oxygen and carbon dioxide), texture, compaction, and composition have not been studied. This is partly due to their seeming unimportance in relation to many other factors, but their effects need to be explored.

3. Other Factors

The growth stage of a plant is important in determining its sensitivity to air pollutants. The age of the leaf under exposure is of more specific importance than the age of the plant. Components of the photochemical complex cause different responses in sensitive plants. In annual bluegrass and tobacco, the youngest mature cells are the most sensitive.\textsuperscript{3,4,7} Since these cells develop progressively from the tip to the base of the leaf, the injury occurs as a band on the sensitive area of the leaf. This pattern is the generally accepted pattern for PAN injury. In other plants, where the cells mature rather uniformly within a given leaf, PAN injury affects the young leaves before they are fully expanded. Ozone injury generally does not develop until the leaves are mature, although Ting and Dugger\textsuperscript{84} in studying cotton, report greatest sensitivity in leaves about 75 percent expanded.

There is some evidence that oxidant or ozone injury may be reduced by pretreatment with the toxicant.\textsuperscript{7,79} In the sensitive onion variety, pretreatment was linked to stomatal action which prevented further entrance of ozone. Stomatal closure as related to pollutants has not been studied extensively; available information suggests, however, that the stomata of many species are affected by ozone.

Essentially no evidence is available on the possible synergistic or antagonistic effects of two or more pollutants in combination. Ethylene, propylene, acetylene, and nitrogen dioxide show no synergistic or antagonistic
action; neither do the photochemical phytotoxicants produced by irradiation of nitrogen dioxide propylene mixtures when ethylene and/or acetylene are mixed with the reaction systems. However, subthreshold levels of sulfur dioxide combined with subthreshold levels of ozone were reported to cause an ozone-like fleck on sensitive tobacco. This interaction has since been reported by several other investigators with positive results on most of the species studied. The response, in general, does not appear to be truly synergistic.

4. Discussion

The various factors affecting the response of vegetation to air pollution must be mediated through control of the stoma, other internal factors that affect cellular responses, or a combination of these. Although stomatal opening is an accepted prerequisite for the entrance of pollutants, there are certain plant tissues that are sensitive to a specific pollutant and not sensitive to another, even when stomatal function is not a factor. Work with irradiated auto exhaust best exemplifies this premise, since three toxicants were identified from the mixture by the differential responses of tobacco leaf tissue. Biochemical control of sensitivity to ozone and PAN is also suggested, since ozone first affects the mature tobacco leaves and PAN affects the younger leaves even though stomatal openings are present in leaves of both ages. Thus, although stomatal opening is a prerequisite for pollutant entrance, the injurious reaction of the pollutant within the leaf tissue is a biochemically mediated response, dependent upon unknown factors within the cells.

D. PROBLEMS IN DIAGNOSIS AND ASSESSMENT OF THE ECONOMIC IMPACT OF PHOTOCHEMICAL AIR POLLUTION ON VEGETATION

The plant is a product of its environment, responding in many ways to the stresses and support afforded by that environment. Air pollution must be considered as simply another quality of the environment, together with climate, soil, insects, disease, as well as genetic history and patterns of care or abuse by man. The injury created by an air pollutant may not only be modified or obscured by other environmental factors but, from these other factors, the plant may develop injuries which are difficult or impossible to distinguish from those caused by air pollution. Ornamental and agronomic crops grown under special management practices must be carefully examined before attributing poor growth to air pollution. Many bacterial, viral, and fungal diseases, as well as insects, can produce injury symptoms in plants which are quite similar to the symptoms produced by air pollutants. To properly diagnose air pollution effects on vegetation, therefore, the injuries must be observed in the field and supported by both measurements of the concentration in the ambient air and by laboratory fumigation studies, using different levels of the suspect pollutant. Furthermore, the observer must have a thorough knowledge of local cultural conditions.

While the markings on the leaves of a plant may be identified with an air pollutant, it is often quite difficult to evaluate these markings in relation to their effects on the intact plant. The question which must be answered is whether or not the plant has been so altered by the air pollutant as to significantly alter its growth, survival, yield, or use. In evaluating some of the leaf injuries the problem is relatively straightforward. This is especially true in cases where the appearance of the plant is paramount, or where there is a loss or a failure to develop a desired product. Other cases, however, require some assessment of the extent to which the leaf injury has affected the essential processes of the plant. Whether the air pollutants directly or indirectly upset the basic plant processes through injury to the leaf, however, is really immaterial to the question of an evaluation of economic loss.

A German report by Guderian et al. expresses a philosophy on the general problem of the effect of air pollutants on vegeta-
tion. The authors make a useful distinction between “injury” and “damage”. They define injury due to air pollution as any identifiable and measurable response of a plant to air pollution. They define damage which results from an air pollution injury as any identifiable and measurable adverse effect upon the desired or intended use or derived product of the plant. Thus, necrotic lesions on the leaves of lettuce when identifiable as due to an air pollutant are injury. Any assessment of damage requires a judgment that this injury affects the yield or use of the vegetation. The initial identification of injury also requires an informed judgment.

The appearance of romaine lettuce, as the appearance of many other truck crops, affects sales on the market. If the outer leaves have been marked by oxidant, the crop may not be marketable. The labor of stripping the outer leaves, with a resultant loss of shape, may not be justified. The yield in crates per acre may be unaffected, but the crop may still be a total loss. Thus both injury and damage have occurred.

In other crops, where the marketable product is not the leaves, the case is somewhat more complicated. Hill and Thomas showed that the yield of alfalfa was reduced in proportion to the area of the leaf destroyed. This offers a method of evaluating the damage sustained by such crops.

Leaf injury to a fruit tree becomes far more difficult to assess as “damage”. The effect of a given percentage of leaf destruction on total fruit yield or on the distribution of grades in the crop is not known. It is known that in severe cases, the growth of the tree will be affected, yet it is very difficult to evaluate the injury in terms of the value of subsequent crops. To properly evaluate such evidence in relation to a crop which is to be harvested 10 to 20 years from now requires more than the knowledge now available. In forest trees, the problem is similar; there is evidence of reduced growth-rings which can be attributed to prior leaf injury.

The problem of leaf injury to ornamentals is equally difficult to assess. If the injury is severe, the stock in a nursery may not be salable. If it is less severe, pruning may make the stock salable but at a reduced price. The damage in these cases usually can be estimated; however, the problem of injured stock held over for subsequent seasons is more difficult. Often such injured stock may represent a sizeable portion of the entire inventory. Then again, individual plants may appear fully recovered by the following season. In some cases, if growth of the plant is somewhat stunted, this can be a benefit to the grower.

After the stock has been sold and planted in a yard, park, or cemetery, the concept of damage becomes broader in scope. Damage may not be so much to the leaves of a plant as to the feelings of people concerned.

E. DOSE-INJURY RELATIONSHIPS OF PHOTOCHEMICAL AIR POLLUTION AND VEGETATION

The interrelations of time and concentration (dose) as they affect injury to plants are essential elements of air quality criteria. They have been inadequately studied and are therefore poorly understood. Discussion of time-concentration relationships is simplified by considering them in terms of three types of injury to plants: acute, chronic, and growth. There is little data in the literature relative to the effect of time-concentration relationships on the production of chronic injury, or in the reduction of growth, yield, or quality of plant material. There is also a dearth of information concerning the relationship of time and concentration on acute injury by PAN and mixtures of pollutants in the photochemical oxidant complex. There is more information in this area on the acute effects of ozone, but it is far from complete.

At the present time, it is impossible to cite more than cursory results on the effects of time-concentration relationships on plant growth. Table 6-1 summarizes the available information for PAN, ozone, and the photochemical complex as they affect the growth and biochemistry of plants.

No attempt has been made to tabulate the slight amount of information available on
chronic effects, since it cannot be separated easily from the results on growth or acute effects.

The acute effects of the photochemical toxicants have been the most widely studied. The fragmentary reports which are available for PAN and the photochemical complex are tabulated in Table 6-2. Relative phytotoxicity of four members of the peroxyacetyl nitrates are shown in Table 6-3. Threshold levels of injury for ozone are tabulated in Table 6-4.

The acute effects of ozone at various concentrations and times has received sufficient study to permit preliminary time-concentration curves to be constructed for several sensitivity groupings. One of the first time-concentration relations was reported by O’Gara. He was concerned only with an acute type of injury which develops over a relatively short period of time. He related concentration (c) and time of exposure (t) as:

\[ t(c-a) = b \]

The parameters “a” and “b” are dependent upon the species and variety of plant and the

<table>
<thead>
<tr>
<th>Species</th>
<th>Concentration, ( \text{ppm} )</th>
<th>Time</th>
<th>Effects</th>
<th>Reference</th>
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<tr>
<td><strong>PAN</strong>(^b)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bean ((\text{Phaseolus vulgaris, L.}))</td>
<td>1.0</td>
<td>0.5 hr</td>
<td>Reduction in photosynthesis</td>
<td>92</td>
</tr>
<tr>
<td></td>
<td>1.0</td>
<td>0.5 hr</td>
<td>Changes in chloroplasts</td>
<td>93</td>
</tr>
<tr>
<td><strong>Ozone</strong>(^b)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peanut ((\text{Arachis hypogaea}))</td>
<td>0.02</td>
<td>24-28 hr</td>
<td>Chlorosis-leaves may absicse</td>
<td>33</td>
</tr>
<tr>
<td>Radish ((\text{Raphanus sativus, L.}))</td>
<td>0.05</td>
<td>8.0 hr/day (20 days)</td>
<td>50 percent reduction in yield</td>
<td>36</td>
</tr>
<tr>
<td>Carnation ((\text{Dianthus caryophyllus, L.}))</td>
<td>0.07</td>
<td>60 days</td>
<td>50 percent reduction in floral development*</td>
<td>68</td>
</tr>
<tr>
<td>Tobacco ((\text{Nicotiana tabacum, L.})) Bel-W3</td>
<td>0.10</td>
<td>5.5 hr</td>
<td>50 percent reduction in pollen germination and pollen tube growth</td>
<td>81</td>
</tr>
<tr>
<td></td>
<td>Bel-W3</td>
<td>0.05</td>
<td>8.0 hr/day (20 days)</td>
<td></td>
</tr>
<tr>
<td><strong>Photochemical complex</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tobacco ((\text{Nicotiana tabacum, L.})) Bel-W3</td>
<td>0.03-0.22</td>
<td>700 hr</td>
<td>Severe leaf injury</td>
<td>69</td>
</tr>
<tr>
<td></td>
<td>Bel-B</td>
<td>0.03-0.22</td>
<td>700 hr</td>
<td>69</td>
</tr>
</tbody>
</table>

\(a\) Concentrations reported for the photochemical complex and ozone have been corrected to neutral KI values. Reported Mast oxidant values have been multiplied by a factor of 1.5.

\(b\) Results of laboratory exposures.
<table>
<thead>
<tr>
<th>Species</th>
<th>Concentration, ppm</th>
<th>Time</th>
<th>Effects</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PAN</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Petunia (Petunia hybrida, Vilm)</td>
<td>0.1</td>
<td>5.0 hr</td>
<td>3</td>
<td>16</td>
</tr>
<tr>
<td>Sensitive species</td>
<td>0.01</td>
<td>6.0 hr</td>
<td>2</td>
<td>29</td>
</tr>
<tr>
<td>Sensitive species (PPN)</td>
<td>0.002</td>
<td>6.0 hr</td>
<td>2</td>
<td>29</td>
</tr>
<tr>
<td>Bean (Phaseolus vulgaris, L.)</td>
<td>1.0</td>
<td>0.5 hr</td>
<td>3</td>
<td>82</td>
</tr>
<tr>
<td></td>
<td>0.1</td>
<td>5.0 hr</td>
<td>3</td>
<td>16</td>
</tr>
<tr>
<td><strong>Photochemical complex</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tobacco (Nicotiana tabacum, L.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bel-W3</td>
<td>0.06</td>
<td>1.0 hr</td>
<td>1</td>
<td>123</td>
</tr>
<tr>
<td></td>
<td>0.07</td>
<td>2.0 hr</td>
<td>2</td>
<td>95</td>
</tr>
<tr>
<td>White Gold</td>
<td>0.05</td>
<td>3.0 hr</td>
<td>1</td>
<td>58</td>
</tr>
<tr>
<td></td>
<td>0.10</td>
<td>3.0 hr</td>
<td>2</td>
<td>58</td>
</tr>
<tr>
<td>Bean (Phaseolus vulgaris, L.)</td>
<td>0.15 (max)</td>
<td>1.0 day</td>
<td>2</td>
<td>42</td>
</tr>
<tr>
<td>White Pine (Pinus strobus, L.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.10</td>
<td>4.0 hr</td>
<td>1</td>
<td>54</td>
</tr>
<tr>
<td><strong>Sulfur dioxide/ozone</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tobacco (Nicotiana tabacum, L.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bel-W3</td>
<td>0.25/0.03</td>
<td>2.0 hr</td>
<td>2</td>
<td>34</td>
</tr>
<tr>
<td>Bel-W3</td>
<td>0.10/0.03</td>
<td>4.0 hr</td>
<td>2</td>
<td>25</td>
</tr>
<tr>
<td>Bel-W3</td>
<td>0.05/0.05</td>
<td>8.0 hr/day (20 days)</td>
<td>1</td>
<td>35</td>
</tr>
<tr>
<td>Peanut (Arachis hypogaea)</td>
<td>0.03/0.02</td>
<td>6.0 hr</td>
<td>1</td>
<td>33</td>
</tr>
<tr>
<td>Tomato (Lycopersicon esculentum, Mill)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.25/0.05</td>
<td>4.0 hr</td>
<td>1</td>
<td>25</td>
</tr>
<tr>
<td>Radish (Raphanus sativus, L.)</td>
<td>0.25/0.05</td>
<td>4.0 hr</td>
<td>1</td>
<td>25</td>
</tr>
<tr>
<td></td>
<td>0.05/0.05</td>
<td>8.0 hr/day (20 days)</td>
<td>1</td>
<td>36</td>
</tr>
<tr>
<td>Pine (Pinus strobus, L.)</td>
<td>0.05/0.05</td>
<td>8.0 hr/day (20 days)</td>
<td>1-2</td>
<td>37</td>
</tr>
</tbody>
</table>

*aConcentrations reported for the photochemical complex and ozone have been corrected to neutral KI values. Reported Max oxidant values were multiplied by a factor of 1.5.*

*bSeverity of injury: 1 = slight, 2 = moderate, 3 = severe.
Table 6-3. RELATIVE PHYTOPHOTOXICITY OF FOUR MEMBERS OF THE PEROXYACYL NITRATES, INDICATED BY PRELIMINARY FUMIGATION ON TWO SPECIES OF PLANTS

<table>
<thead>
<tr>
<th>Species</th>
<th>Fumigation time, hr</th>
<th>Toxicant</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Conc., ppb</td>
<td>Injury, %</td>
<td>Conc., ppb</td>
<td>Injury, %</td>
<td>Conc., ppb</td>
<td>Injury, %</td>
</tr>
<tr>
<td>Bean, var. Pinto</td>
<td>0.5</td>
<td>–</td>
<td>100</td>
<td>90</td>
<td>100</td>
<td>80</td>
</tr>
<tr>
<td></td>
<td>1.0</td>
<td>140</td>
<td>55</td>
<td>24</td>
<td>7</td>
<td>30</td>
</tr>
<tr>
<td></td>
<td>4.0</td>
<td>40</td>
<td>90</td>
<td>10</td>
<td>86</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td>8.0</td>
<td>20</td>
<td>44</td>
<td>5</td>
<td>100</td>
<td>–</td>
</tr>
<tr>
<td>Petunia, var. Rosy morn</td>
<td>0.5</td>
<td>–</td>
<td>50</td>
<td>7</td>
<td>100</td>
<td>90</td>
</tr>
<tr>
<td></td>
<td>1.0</td>
<td>140</td>
<td>33</td>
<td>24</td>
<td>35</td>
<td>12</td>
</tr>
</tbody>
</table>

Table 6-4. THRESHOLD SUSCEPTIBILITY OF PLANTS TO ACUTE INJURY FROM OZONE

<table>
<thead>
<tr>
<th>Species</th>
<th>Concentration, ppm</th>
<th>Time, hr</th>
<th>Effects</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tobacco</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Nicotiana tabacum, L.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bel-W3</td>
<td>0.15</td>
<td>0.5</td>
<td>1</td>
<td>96</td>
</tr>
<tr>
<td></td>
<td>0.75</td>
<td>0.2</td>
<td>1</td>
<td>123</td>
</tr>
<tr>
<td></td>
<td>0.15</td>
<td>1.0</td>
<td>1</td>
<td>96</td>
</tr>
<tr>
<td></td>
<td>0.08</td>
<td>2.0</td>
<td>2</td>
<td>95</td>
</tr>
<tr>
<td></td>
<td>0.03</td>
<td>8.0</td>
<td>1</td>
<td>83</td>
</tr>
<tr>
<td></td>
<td>0.15</td>
<td>2.0</td>
<td>2</td>
<td>97</td>
</tr>
<tr>
<td>White Gold</td>
<td>0.05</td>
<td>3.0</td>
<td>1</td>
<td>58</td>
</tr>
<tr>
<td>Bean (Phaseolus vulgaris, L.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.15</td>
<td>0.5</td>
<td>1</td>
<td>96</td>
</tr>
<tr>
<td></td>
<td>0.40</td>
<td>0.33</td>
<td>1</td>
<td>41</td>
</tr>
<tr>
<td></td>
<td>0.08</td>
<td>4.0</td>
<td>1</td>
<td>25</td>
</tr>
<tr>
<td>Oat (Avena sativa)</td>
<td>0.12</td>
<td>2.0</td>
<td>1</td>
<td>60</td>
</tr>
<tr>
<td>White Pine (Pinus strobus, L.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.10</td>
<td>4.0</td>
<td>1</td>
<td>54</td>
</tr>
<tr>
<td>Alfalfa (Medicago sativa, L.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tomato (Lycopersicon esculentum, Mill.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.20</td>
<td>2.0</td>
<td>2</td>
<td>98</td>
</tr>
<tr>
<td>Radish (Raphanus sativus, L.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.08</td>
<td>1.0</td>
<td>1</td>
<td>124</td>
</tr>
<tr>
<td>Onion (Allium cepa, L.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peanut (Arachis hypogaea)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.02</td>
<td>24-48</td>
<td>1</td>
<td>33</td>
</tr>
</tbody>
</table>

\[a\] Concentrations have been corrected to neutral KI values. Reported
Mast oxidant values were multiplied by a factor of 1.5.

\[b\] Severity of Injury: 1 = slight, 2 = moderate, 3 = severe

6-10
degree of injury. Concentration is measured in parts per million and time in hours. O’Gara’s equation can be rearranged to:

\[ c = b/t + a \]

The plot of “c” versus “1/t” is a straight line. The parameter “a” is the intercept for 1/t = 0 or when “t” is infinitely large. This intercept could be considered the threshold concentration for injury. The O’Gara equation is a mathematical form which fits experimental data obtained from exposures limited in time. Guderian et al.\textsuperscript{90} do not believe that the O’Gara equation fits their observations and suggest an exponential relationship to best describe their data:

\[ t = Ke^{-a (c-r)} \]

where K, a, and r are parameters varying with species and degree of injury. In the middle time range, both forms of equations reasonably fit available data. The exponential form probably fits better over a wider range of time.

An expression of the degree of injury produced as time and concentration vary is needed in describing injury. Heck et al.\textsuperscript{96} presented this information graphically for pinto bean and Bel-W3 tobacco exposed to ozone. Mathematical surfaces of this type can make apparent the frequently steep slope in the injury-versus-concentration or injury-versus-time planes. The steepness in these slopes gives a relative measure of the degree of variability to be expected in data collected under practical control conditions. Similar reports, stressing the importance of considering both time and concentration, have recently appeared in the literature.\textsuperscript{84,99}

Such relationships give an insight into what may happen under a given set of circumstances. These relationships are probably universal and could be derived for any toxicant producing a definite acute-type of tissue collapse. Relationships of this type permit the assumption with reasonable assurance that as long as a certain concentration is not exceeded for a given period of time, no acute injury will occur. They do not indicate the severity of injury at higher concentrations for longer time periods. To extend any of these relationships beyond the available experimental data upon which they are based is questionable.

None of the experimental data presented provide any more than suggestions of what takes place in the field. They cover only single time-concentration relationships under standard conditions. They do not consider fluctuations in concentration in a given time interval or the effect of repeated fumigations over either several days or even several hours in one day. Further, most of the available data are from rather short-time exposures, that is, short as related to a growing season. To extrapolate any of the presentations to long time periods would give results of very questionable validity.

A number of investigators have reported the time-concentration effects of ozone on various species of plants. Table 6-4 summarizes some of the threshold-level (5 percent) time-concentration results with ozone. From these results, Tables 6-5 and 6-6 have been developed. These tables suggest the times and concentrations necessary to produce injury in sensitive, intermediate, and resistant plants at the threshold (5 percent) and 20 percent injury levels, respectively. The lower levels shown in Table 6-5 for the sensitive plants were taken essentially from Table 6-4. The remaining values in Tables 6-5 and 6-6 were developed from the threshold values by extensive study of ozone effects on a group of economically important plants. Table 6-7 gives a complete list of the plants which have been studied, places them in three sensitivity classes, and lists them alphabetically by family.

The sensitive range for 1/2, 1, 2, and 4 hours from Table 6-5 was analyzed using the O’Gara equation. This equation gives a good fit at the shorter time periods but gives a threshold concentration above experimental
values for infinite time. These results suggest that the O'Gara equation does not give an exact fit to experimental data with ozone, but that the approximate fit obtained can be used with a fair degree of confidence with the fragmentary data presently available for analysis.

F. EFFECTS OF PHOTOCHEMICAL OXIDANTS ON MICROORGANISMS

The bacteriocidal action of ambient photochemical oxidant has been demonstrated by Goetz and Tsuneishi. In fact, the BIA test, where bacteria of uniform density are exposed to a concentration of oxidant, was developed as a bacteriological analog for the measure of eye irritation from photochemical oxidant aerosols. Photochemical oxidant concentrations from irradiated auto exhaust containing as little as 0.125 ppm hydrocarbons proved bacteriostatic for common bacteria such as Eschericha coli.

G. EFFECTS OF OZONE ON MICROORGANISMS

The growth suppression of microorganisms by ozone is a well-known phenomenon. Ozone at higher concentrations readily oxidizes organic matter and is used in a variety of applications, such as in cold storage plants for the control and suppression of fungi and bacteria associated with food spoilage, drinking water purification, and the treatment of sewage. The value of ozone in preventing the spoilage of fruit and vegetables, however, is in question. The use of ozone with food and drinking water is permitted because safeguards can protect personnel from excessive exposures, but these same safeguards cannot be applied so as to permit the use of ozone for air sterilization in the general ventilation of homes and public buildings.

It is now recognized that ozone is not an

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Table 6-5. PROJECTED OZONE CONCENTRATIONS WHICH WILL PRODUCE, FOR SHORT-TERM EXPOSURES, 5 PERCENT INJURY TO ECONOMICALLY IMPORTANT VEGETATION GROWN UNDER SENSITIVE CONDITIONS

<table>
<thead>
<tr>
<th>Time, hr</th>
<th>Sensitive</th>
<th>Intermediate</th>
<th>Resistant</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.2</td>
<td>0.35-0.75</td>
<td>0.70-1.00</td>
<td>0.90 and up</td>
</tr>
<tr>
<td>0.5</td>
<td>0.15-0.30</td>
<td>0.25-0.60</td>
<td>0.50 and up</td>
</tr>
<tr>
<td>1.0</td>
<td>0.10-0.25</td>
<td>0.20-0.40</td>
<td>0.35 and up</td>
</tr>
<tr>
<td>2.0</td>
<td>0.07-0.20</td>
<td>0.15-0.30</td>
<td>0.25 and up</td>
</tr>
<tr>
<td>4.0</td>
<td>0.05-0.15</td>
<td>0.10-0.25</td>
<td>0.20 and up</td>
</tr>
<tr>
<td>8.0</td>
<td>0.03-0.10</td>
<td>0.08-0.20</td>
<td>0.15 and up</td>
</tr>
</tbody>
</table>

The lower levels obtained from data presented in Table 6-4.

Table 6-6. PROJECTED OZONE CONCENTRATIONS WHICH WILL PRODUCE, FOR SHORT-TERM EXPOSURES, 20 PERCENT INJURY TO ECONOMICALLY IMPORTANT VEGETATION GROWN UNDER SENSITIVE CONDITIONS

<table>
<thead>
<tr>
<th>Time, hr</th>
<th>Sensitive</th>
<th>Intermediate</th>
<th>Resistant</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.2</td>
<td>0.40-0.90</td>
<td>0.80-1.10</td>
<td>1.00 and up</td>
</tr>
<tr>
<td>0.5</td>
<td>0.20-0.40</td>
<td>0.35-0.70</td>
<td>0.60 and up</td>
</tr>
<tr>
<td>1.0</td>
<td>0.15-0.30</td>
<td>0.25-0.55</td>
<td>0.50 and up</td>
</tr>
<tr>
<td>2.0</td>
<td>0.10-0.25</td>
<td>0.20-0.45</td>
<td>0.40 and up</td>
</tr>
<tr>
<td>4.0</td>
<td>0.07-0.20</td>
<td>0.15-0.40</td>
<td>0.35 and up</td>
</tr>
<tr>
<td>8.0</td>
<td>0.05-0.15</td>
<td>0.10-0.35</td>
<td>0.30 and up</td>
</tr>
<tr>
<td>Species</td>
<td>Variety</td>
<td>Reference</td>
<td></td>
</tr>
<tr>
<td>-------------------------</td>
<td>-----------------------</td>
<td>-----------</td>
<td></td>
</tr>
<tr>
<td><strong>Sensitive</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chenopodiaceae</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spinach (<em>Spinacia oleracea, L.</em>)</td>
<td>Early Hybrid 7</td>
<td>100</td>
<td></td>
</tr>
<tr>
<td><strong>Cruciferae</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Radish (<em>Raphanus sativus, L.</em>)</td>
<td>Early Scarlet Red</td>
<td>124</td>
<td></td>
</tr>
<tr>
<td><strong>Curcurbitaceae</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Muskmelon (<em>Cucumis melo, L.</em>)</td>
<td></td>
<td>124</td>
<td></td>
</tr>
<tr>
<td><strong>Gramineae</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bromegrass (<em>Bromus inermis, L.</em>)</td>
<td>Sac Smooth</td>
<td>124</td>
<td></td>
</tr>
<tr>
<td>Oat (<em>Avena sativa</em>) Hexaploid</td>
<td></td>
<td>60</td>
<td></td>
</tr>
<tr>
<td>Oat (<em>Avena strigosa</em>) Diploid</td>
<td></td>
<td>60</td>
<td></td>
</tr>
<tr>
<td>Rye (<em>Secale cereale</em>)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Leguminosae</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bean (<em>Phaseolus vulgaris, L.</em>)</td>
<td>Black Valentine</td>
<td>124</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pinto</td>
<td>42,100,124</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sanilac</td>
<td>62</td>
<td></td>
</tr>
<tr>
<td>Peanut (<em>Arachis hypogaea</em>)</td>
<td></td>
<td>33</td>
<td></td>
</tr>
<tr>
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Table 6-7 (continued). LISTS OF PLANTS IN THREE SENSITIVITY GROUPS BY SENSITIVITY TO OZONE

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<td>Petunia (Petunia hybridra, Vilm.)</td>
<td>Unknown</td>
<td>124</td>
</tr>
<tr>
<td>Potato (Solanum tuberosum, L.)</td>
<td>Rose Charm</td>
<td>98</td>
</tr>
<tr>
<td></td>
<td>Irish</td>
<td>124</td>
</tr>
<tr>
<td>UMBELLIFERAE</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carrot (Daucus carota, L.)</td>
<td>Imperator</td>
<td>98</td>
</tr>
<tr>
<td>Parsnip (Pastinaca sativa, L.)</td>
<td>White Model</td>
<td>98</td>
</tr>
<tr>
<td>VERBENACEAE</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Verbena (Verbena sp.)</td>
<td>L.A.</td>
<td>100</td>
</tr>
<tr>
<td>VITACEAE</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grape (Vitis vinifera, L.)</td>
<td>Mission</td>
<td>98</td>
</tr>
<tr>
<td></td>
<td>Zing</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>Tokay</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>Concord</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>Unknown</td>
<td>50</td>
</tr>
<tr>
<td>SCROPHULARIACEAE</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Snapdragon (Antirrhinum majus)</td>
<td></td>
<td>124</td>
</tr>
</tbody>
</table>

Effective germicide at concentrations below the level of human sensitivity (80 μg/m³, or 0.04 ppm).\(^\text{119}\) The germicidal effectiveness of ozone varies with its concentration, the relative humidity, and the species of bacteria.\(^\text{105-107,120}\) Jordan and Carlson\(^\text{120}\) were unable to obtain any positive germicidal action for ozone against bacteria (Staphylococcus pyrogenes, Staphylococcus aureus and Bacillus pyocyaneus) grown on agar plates and exposed to 5,880 to 9,016 μg/m³ (3.0 to 4.6 ppm). Concentrations which killed dry typhus bacilli, staphylococci, or streptococci in the course of several hours, were found by Sawyer et al. to kill guinea pigs sooner.\(^\text{106}\)

Hibben found ozone toxic to the exposed moist fungus spores of some species, even at the 200 μg/m³ (0.1 ppm) level.\(^\text{108}\) Exposure to 980 and 1,960 μg/m³ (0.5 and 1.0 ppm) reduced or prevented germination of spores of all species tested. Ozone at 200 μg/m³ (0.1 ppm) for 4 hours or at 1,960 μg/m³ (1.0 ppm) for 2 hours stopped apical cell division of conidiophores of Alternaria solani and caused collapse of the apical cell wall.\(^\text{109}\)

Giese and Christensen found that protozoa
in hanging drop suspensions exposed to approximately 8 percent ozone in ozonized water were killed in from 4 minutes (colpidium) to a maximum of 64 minutes (Tillina).112

Scott and Lesher, in studies of the effects of ozone on Escherichia coli, found that ozone caused leakage of the cell contents into the medium, with lysis of some cells.111 They postulated that the primary effect of ozone was on the cell wall or membrane of the bacteria, probably by reaction with the double bonds of lipids, and that the leakage depended on the extent of the reaction. The bactericidal action of ozone on E. coli has been considered by Fetner and Ingols to be an all-or-none effect.112 Haines found that E. coli growth in a culture medium was retarded by 7,840 \( \mu g/m^3 \) (4 ppm) ozone in the atmosphere and that 19,600 \( \mu g/m^3 \) (10 ppm) ozone prevented bacterial growth.113

Elford and van den Ende found that 390 \( \mu g/m^3 \) (0.2 ppm) ozone in a moderately humid atmosphere exercises a very definite lethal effect against certain bacteria deposited from aerosol mists or suspensoids on various surfaces.122 The effectiveness depends on: (1) the type of surface on which the bacteria are deposited, (2) the medium in which the organisms are contained before being sprayed (a higher protein content favoring protection of the bacterium in the ultimate particle), and (3) the different resistances to ozone of the different types of organism. Relative humidity plays a dominant role in influencing the action of ozone, particularly when the gas is present in low concentrations. In relatively dry air, at relative humidities lower than 45 percent, there was no appreciable killing action on the bacterial aerosols studied, even when the ozone concentration was in excess of 1,960 \( \mu g/m^3 \) (1.0 ppm). In contrast, when Streptococcus salivarus, in a fine aerosol-broth suspension, was exposed at a relative humidity of 60 to 80 percent to 50 \( \mu g/m^3 \) (0.025 ppm) ozone for 30 minutes and deposited from the aerosol on glass plate surfaces, a kill of more than 90 percent resulted.

The effects exerted by humidity on fine suspensoid systems of bacteria depend on: (1) its purely physical effect on limiting particle size and settling rate, (2) its effect on the normal viability, and (3) the interaction between ozone and the organisms, as reflected in the increased death-rate of the latter when influenced by high humidity. When bacteria are covered with a protective coating of organic matter, as in coarse aerosols from suspensions containing appreciable amounts of serum protein such as in sneezes and coughs, ozone toxicity rapidly diminishes and much greater concentrations are necessary to achieve any appreciable killing effect on the organisms. Elford and van den Ende's evidence122 leads to the conclusion that ozone, at low concentrations which do not otherwise cause irritation of the human respiratory tract, cannot be expected to provide any effective protection against airborne bacterial infection through direct inactivation of the infectious carrier particulates. A summary of the effects of ozone on certain microorganisms is given in Table 6-8.

H. SUMMARY

Injury to vegetation was one of the earliest manifestations of photochemical air pollution. Due to this fact, sensitive plants have been useful biological indicators of this type of pollution. The visible symptoms of photochemical-oxidant-produced injury to plants may be classified as: 1) acute injury, identified by cell collapse with subsequent development of necrotic patterns; 2) chronic injury, identified by necrotic patterns with or without chlorotic or other pigmented patterns; and, 3) physiological effects, including growth alterations, reduced yields, and changes in the quality of plant products. The acute symptoms are generally characteristic of a specific pollutant while, though highly characteristic, chronic injury patterns are not. Injury to leaves by ozone is identified as a stippling or flecking. Such injury has occurred experimentally in the most sensitive species after exposure to 60 \( \mu g/m^3 \) (0.03 ppm) ozone for 8 hours. Injury will occur in shorter time
periods when low levels of sulfur dioxide are present. PAN-produced injury is characterized by an under-surface glazing or bronzing of the leaf. Such injury has occurred experimentally in the most sensitive species after exposure to 50 μg/m³ (0.01 ppm) PAN for 5 hours. Leaf injury has occurred in certain sensitive species after a 4-hour exposure to 100 μg/m³ (0.05 ppm) of total oxidant. Ozone appears to be the most important phytotoxicant in the photochemical complex.

There are a number of factors affecting the response of vegetation to photochemical air pollutants. Variability in response is known to exist between species of a given genus and between varieties within a given species. Varietal variations have been most extensively studied with tobacco. In fact, a sensitive tobacco strain, Bel-W3, has been isolated and developed for use as a biological indicator of ozone injury.

The influence of light intensity on the sensitivity of plants during growth appears to depend on the phytotoxicant. Plants are more sensitive to PAN when grown under high light intensities, but are more sensitive to ozone when grown under low light intensities. Reported findings are in general agreement that sensitivity of greenhouse-grown plants to oxidants increases with temperature from 40° to 100° F. However, there is some indication that this positive correlation may result from the overriding influence of light intensity on sensitivity.

The effects of humidity on the sensitivity of plants has not been well documented. General trends indicate that plants grown and/or exposed under high humidities are more sensitive than those grown at low humidities. Though there has been little research in this direction, there are indications that soil factors influence the sensitivity of plants to phytotoxic air pollutants. Plants grown under drought conditions are less susceptible than those grown under moist conditions. Studies indicate that plants appear to be more sensitive when they are grown in soil having low total fertility.

The age of the leaf under exposure is important in determining its sensitivity to air

<table>
<thead>
<tr>
<th>Organisms</th>
<th>Concentration, ppm</th>
<th>Experimental conditions</th>
<th>Effects</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bacteria</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Streptococcus salivarius</em></td>
<td>0.025</td>
<td>30 minutes Relative humidity = 60-80 percent, 20°C</td>
<td>90 percent mortality</td>
<td>122</td>
</tr>
<tr>
<td>Organisms sprayed in air in Singleton fine broth suspension and collected on agar plates</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>E. coli</em></td>
<td>4.0</td>
<td></td>
<td>Retarded growth</td>
<td>113</td>
</tr>
<tr>
<td>Ozone is admitted when bacteria are inoculated in Nelson’s medium</td>
<td>10.0</td>
<td></td>
<td>Prevented bacterial growth</td>
<td>113</td>
</tr>
<tr>
<td><em>Staphylococcus pyogenes, Bacillus pyocyaneus, Staphylococcus aureus.</em></td>
<td>3.0-4.6 (Acid KI method)</td>
<td>1.5-4.5 hours</td>
<td>No positive germicidal action</td>
<td>120</td>
</tr>
<tr>
<td><strong>Protozoa</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Colpidium</td>
<td>8.0</td>
<td>Between 4 minutes (Colpidium) and 64 minutes (Tillina), in descending order</td>
<td>100 percent mortality</td>
<td>121</td>
</tr>
<tr>
<td>Blepharisma</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paramecium</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amoeba</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Didinium</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tillina</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 6-8. SUMMARY OF EFFECTS OF OZONE ON BACTERIA AND PROTOZOA
pollutants. There is some evidence that oxidant or ozone injury may be reduced by pretreatment with the toxicant.

Identification of an injury to a plant as being caused by air pollution is an arduous undertaking. Even when the markings on the leaves of a plant may be identified with an air pollutant, it is often quite difficult to evaluate these markings in terms of their effect on the intact plant. Further difficulty arises in trying to evaluate the economic impact of air pollution damage to the plant.

The interrelations of time and concentration, or dose, as they affect injury to plants, are essential to air quality criteria. There are, however, only scant data relating concentrations and length of photochemical oxidant exposure to chronic injury and effects on reduction of plant growth, yield, or quality. There is also a dearth of information relating acute injury to concentrations and duration of exposure to PAN or mixtures of photochemical oxidants. A larger body of information exists on the acute effects of ozone but even in this instance, the information is far from complete. Sufficient data do exist, however, to present, in tabular form, ozone concentrations which will produce 5 percent injury to sensitive, intermediate, and resistant plants after given short-term exposure, as shown in Table 6-5. Information available lists 20 species and/or varieties as "sensitive," 55 as "intermediate in sensitivity," and 64 as "relatively resistant."

Bacteriostatic and bacteriocidal properties of photochemical oxidants in general have been demonstrated. The growth suppression of microorganisms by ozone is a well-known phenomenon, although ozone concentrations for this activity are undesirable from a human standpoint. The bacteriocidal activity of ozone varies with its concentration, the relative humidity, and the species of bacteria.

I. REFERENCES

2. Bobrov, R.A. The Effect of Smog on the Anatomy of

8. First and Second Technical and Administrative Reports on Air Pollution Control. Los Angeles County Air Pollution Control Board, Los Angeles, Calif. 1949-50; 1950-51.


41. Middleton, J.T. Response of Plants to Air Pollution. J. Air Pollution Control Assoc. 6(1):7-9, 50, May 1956.


46. Treschow, M. Evaluation of Vegetation Injury as an Air Pollution Criteria. J. Air Pollution Control Assoc. 15:266-269, June 1965.


60. Sechler, D. and D.R. Davis. Ozone Toxicity in Small

6-22


Chapter 7.

THE EFFECT OF OZONE ON MATERIALS

A. INTRODUCTION

The total extent of damage to materials caused by atmospheric levels of ozone is not known. Ozone may be a major contributor to the degradation of materials which is now attributed to "weathering." To date, the majority of the research in this area has been concerned with only two classes of materials, the elastomers and textiles. Within these two groups, certain specific organic compounds are much more sensitive to attack by atmospheric concentrations of ozone than animals or humans. There may be, therefore, economic incentives to control ozone levels well below the concentrations which may be established as hazardous to humans.

B. MECHANISMS OF OZONE ATTACK

Ozone is so active that, when concentrated, it becomes a materials-handling problem. In general, any organic material is incompatible with concentrated ozone. Bailey has reviewed the literature thoroughly from 1939 to 1957 for reactions of ozone with organic compounds, and he describes the reaction mechanisms in detail. Although it is incorrect to assume that all of these reactions will occur at atmospheric concentrations of ozone, it is possible that some of these mechanisms would be operant.

Many polymers are sensitive to atmospheric concentrations of ozone. Both chain-scissioning and crosslinking take place in polymers exposed to ozone at atmospheric levels. Chain-scissioning results in increased fluidity and a loss in tensile strength. Crosslinking increases the rigidity of the polymer, reducing elasticity, and brittleness may result.

An example of chain-scissioning is as follows:

\[ \text{RHC} \rightarrow \text{CHR'} + \text{RCHO_2} + \text{CHR'} \]

The sensitivity of polymers to chain-scissioning, therefore, is related to the prevalence of double bonds in their structure. Since almost all rubbers possess such a chemical structure, they are prone to this type of oxidative attack. A similar type of attack has been proposed for the degradation of dyes.

C. THE EFFECT OF OZONE ON RUBBER

Rubber is an economically important material that is highly sensitive to ozone attack. The most vulnerable generic formulations are natural, styrene-butadiene, polybutadiene, and synthetic polyisoprene. These types account for 85 percent by weight of the estimated 1969 national production of rubber. With the exception of natural rubber, the major use for each of these elastomers is in tires. Thus most of the rubber that is susceptible to ozone-cracking is exposed to the atmosphere in a state of tensile stress. Butyl, halogenated butyl, polychloroprene, vinyl-modified nitrile-butadiene, and carboxylated nitrile rubbers have some ozone resistance but require special formulation for optimum performance. Synthetic rubbers with saturated chemical structures, such as silicones, ethylene-propylene, chlorosulfonated polyethylene, polyacrylate, and fluorocarbon rubbers have inherent ozone resistance. These latter special-application materials, however, are relatively expensive and account for only a small fraction of the market on a weight basis.

A tensile stress is necessary to produce ozone-cracking of rubber. If strained as little
as 2 or 3 percent and exposed to an atmosphere containing 20 to 40 µg/m³ (0.01 to 0.02 ppm) ozone, cracks will develop perpendicular to the stress axis.\textsuperscript{11,12} Rubber in a relaxed state can be exposed for long periods of time to relatively high concentrations of ozone without visible cracking.\textsuperscript{17} The rubber is apparently protected by the formation of an ozonide film on the surface.\textsuperscript{4} The embrittled film cracks when strained and exposes fresh surface to the atmosphere. The strong dependence of crack-growth on nominal tensile stress has been explained in terms of fracture mechanics.\textsuperscript{18} However, the “Griffith theory” only partially explains observed relationships between number and size of cracks as a function of strain.\textsuperscript{5,18} Crack growth rate is also ozone concentration-dependent,\textsuperscript{18} which suggests that the rate of diffusion of ozone to the root of a crack is the rate-controlling factor.

Andrews has taken advantage of the fracture mechanics behavior of various rubbers to compound for ozone resistance and other desired properties.\textsuperscript{19} Blends of natural rubber with an ethylene-propylene copolymer are more resistant to ozone-cracking than natural rubber alone. Specimens of blends from 0 to 50 percent ethylene-propylene, strained from 5 to 100 percent, were exposed to 980 mg/m³ (500 ppm) ozone in air. Micro-cracks were observed in all of the blends, the crack density increasing with a decrease of ethylene-propylene rubber. The addition of the ethylene-propylene copolymer raises the critical stored energy necessary to propagate the observed micro-cracks into the macro-cracks characteristic of ozone damage.

Antiozonant additives, such as p-phenylenediamine derivatives, have been developed and protect elastomers from ozone degradation. These chemicals are expensive, however, and add to the cost of rubber products. One company spent $2 million for research and development prior to constructing a multimillion-dollar plant to produce one type of antiozonant.\textsuperscript{20} The addition of 1.5 percent antiozonants to automobile tires costs up to $0.50 per tire. With the 1969 production of automobile tires reported to be over 203 million,\textsuperscript{21} the cost to the public of antiozonants in tires alone could approach $100 million. Antiozonants are also used in conveyor belts, automotive rubber parts, wire and cable, and other products as well as tires.

The use of antiozonants has its limitations. The amount required to be effective increases with the anticipated amount of ozone to be encountered.\textsuperscript{22} In some cases these inhibitors may provide only temporary protection against ozone damage, because some of them migrate to the surface of the rubber product with usage.\textsuperscript{4} Oils, gasoline, and other chemicals tend to extract antiozonants from the rubber, leaving it again susceptible to ozone attack. Thus, in spite of preventive measures, ozone-cracking of rubber products is still a major problem.

A number of factors affect the rate of attack of ozone on rubber:\textsuperscript{4} (1) the amount of tensile stress, (2) the type of rubber compound, (3) concentration of ozone, (4) period of exposure, (5) rate of diffusion of ozone to the rubber surface, and (6) temperature. Dose-response data, therefore, are necessarily dependent on these factors. Also, the method for reporting damage varies from one researcher to another. For example, damage may be expressed as time to initiate either micro-cracks or visible cracks, or it may be expressed in crack-depth or crack-growth rate. The following examples of dose-response data should be considered with these factors in mind.

Bradley and Haagen-Smit evaluated the rubber formulation indicated in Table 7-1 for susceptibility to ozone-cracking.\textsuperscript{13}

This formulation was selected for its sensitivity to ozone. Strips were strained approximately 100 percent by bending. These specimens, if exposed to 39,000 mg/m³ (2 percent or 20,000 ppm) ozone in air would crack instantaneously and break completely within 1 second.

Results of Bradley and Haagen-Smit's standard tests, using a gas flow rate of 1.5
liters per minute through a 13-mm tube at room temperature, are given in Table 7-2.

Edwards and Story have determined the effects of ozone on "hot" (Polysar-S) and "cold" (Polysar-krylene) styrene-butadiene rubbers (SBR) containing various amounts of antiozonant.\textsuperscript{22} The ingredients added to the base polymer are listed in Table 7-3. The results are given in Table 7-4.

Thin polybutadiene specimens were exposed, under constant load, to room air for which the average concentrations of ozone had been determined.\textsuperscript{23} The specimens exposed in the summer months, to average ozone concentrations of about 94 \( \mu g/m^3 \) (0.048 ppm), failed by breaking into two separate parts after 150 to 250 hours. In the fall, at average ozone concentrations of 82 \( \mu g/m^3 \) (0.042 ppm), specimens failed between 400 and 500 hours. In the winter, at average ozone concentrations of 47 \( \mu g/m^3 \) (0.024 ppm), failures occurred between 500 and 700 hours. These data show the strong dependence of cracking rate on the average concentration of ozone.

The behavior of rubber exposed to ozone under laboratory conditions correlates well with the service behavior of tires in localities where atmospheric ozone concentrations are high.\textsuperscript{24} The relative susceptibility of white sidewalls made from different formulations remains the same, whether in a laboratory test, in which they are exposed to as much as 980 \( \mu g/m^3 \) (0.5 ppm) ozone, or in the ambient air of the Los Angeles area. The rate of cracking is thus a function of ozone concentration.

As the tread wear on passenger car tires improves, more or better antiozonants will

---

**Table 7-1. FORMULATION OF HIGHLY OZONE-SENSITIVE RUBBER\textsuperscript{13}**

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Parts by weight</th>
<th>Percent by weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rubber</td>
<td>100</td>
<td>35.91</td>
</tr>
<tr>
<td>Tire reclaim</td>
<td>125</td>
<td>44.88</td>
</tr>
<tr>
<td>SRF black</td>
<td>33</td>
<td>11.85</td>
</tr>
<tr>
<td>Stearic acid</td>
<td>1.5</td>
<td>0.54</td>
</tr>
<tr>
<td>Pina tar</td>
<td>8.4</td>
<td>3.02</td>
</tr>
<tr>
<td>Zinc oxide</td>
<td>4.7</td>
<td>1.69</td>
</tr>
<tr>
<td>Mercaptopbenzoiazole</td>
<td>0.8</td>
<td>0.29</td>
</tr>
<tr>
<td>Diphenylguanidine</td>
<td>0.1</td>
<td>0.03</td>
</tr>
<tr>
<td>Sulfur</td>
<td>5</td>
<td>1.79</td>
</tr>
</tbody>
</table>

\textsuperscript{a}Cured 40 minutes at 45 psi steam.

**Table 7-2. EFFECT OF OZONE ON RUBBER\textsuperscript{a,13}**

<table>
<thead>
<tr>
<th>Ozone concentration ( \mu g/m^3 )</th>
<th>Time to first sign of crack at 4X magnification, minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>( 40 )</td>
<td>0.02</td>
</tr>
<tr>
<td>( 510 )</td>
<td>0.26</td>
</tr>
<tr>
<td>( 880 )</td>
<td>0.45</td>
</tr>
</tbody>
</table>

\textsuperscript{a}Special formulation of Bradley and Haagen-Smit, strained 100 percent.

\textsuperscript{b}Determined by neutral KI.

**Table 7-3. TIRE SIDEWALL FORMULATION\textsuperscript{22}**

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Parts by Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polymer (hot or cold SBR)</td>
<td>100</td>
</tr>
<tr>
<td>Ciroxol 2 X H</td>
<td>10</td>
</tr>
<tr>
<td>FEF Black</td>
<td>30</td>
</tr>
<tr>
<td>SRF Black</td>
<td>10</td>
</tr>
<tr>
<td>Zinc Oxide</td>
<td>3</td>
</tr>
<tr>
<td>Stearic Acid</td>
<td>2</td>
</tr>
<tr>
<td>Antiozonant (Santoflex AW)</td>
<td>Variable</td>
</tr>
<tr>
<td>Crystex</td>
<td>2</td>
</tr>
</tbody>
</table>

**Table 7-4. EFFECTS OF OZONE ON SIDEWALL FORMULATIONS CONTAINING VARIOUS ANTIOZONANT CONCENTRATIONS\textsuperscript{a,22}**

<table>
<thead>
<tr>
<th>Polymer</th>
<th>Antiozonant concentration, (Santoflex AW), percent</th>
<th>Rate of cracking, ( 10^{-4} ) in./hr</th>
<th>Time to first sign of crack, ( \mu g/m^3 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>&quot;Hot&quot; SBR</td>
<td>0.32</td>
<td>0.69</td>
<td>87</td>
</tr>
<tr>
<td>(Polysar-S)</td>
<td>0.63</td>
<td>0.35</td>
<td>170</td>
</tr>
<tr>
<td></td>
<td>1.25</td>
<td>0.13</td>
<td>460</td>
</tr>
<tr>
<td>&quot;Cold&quot; SBR</td>
<td>0</td>
<td>1.58</td>
<td>38</td>
</tr>
<tr>
<td>(Polysar-drylene)</td>
<td>0.32</td>
<td>0.85</td>
<td>77</td>
</tr>
<tr>
<td></td>
<td>0.63</td>
<td>0.57</td>
<td>105</td>
</tr>
<tr>
<td></td>
<td>1.25</td>
<td>0.24</td>
<td>250</td>
</tr>
</tbody>
</table>

\textsuperscript{a}Specimens were strained initially at 100 percent and exposed at 120°F to an ozone concentration of 490 ±100 \( \mu g/m^3 \) (0.25 ± 0.05 ppm).

\textsuperscript{b}Added to enable comparison with data in Table 7-2. First sign of crack was assumed to be 10\textsuperscript{-4} inch crack depth, visible at 4X magnification. Data are not found in reference 22.
have to be added to sidewall formulations to prevent sidewall cracking from becoming the limiting factor in tire life. Thus, part of the cost of premium tires will be due to atmospheric ozone.

D. THE EFFECT OF OZONE ON FABRICS AND DYES

1. Damage to Textile Fabrics

Ozone attacks cellulose by two mechanisms.\(^2\)\(^5\) One is a free-radical chain mechanism involving oxygen in the propagating step, and the other appears to be an electrophilic attack on double bonds. Bogaty et al.\(^2\)\(^6\) have shown that ozone attack at atmospheric levels results in the deterioration of wet cotton textiles. Two types of cotton fabric, duck and printed cloth, were exposed, both wet and dry, for 50 days to an atmosphere containing between 40 and 120 µg/m\(^3\) (0.02 and 0.06 ppm) ozone. The deterioration of the two fabrics was shown by comparing dissolved fabric fluidity values before and after exposure. The value for the wet duck cloth increased from 2.6 to 9.5 rhe (a calibrated measure of fluidity which is inversely proportional to viscosity), while the value for the wet printed cloth increased from 8 to 16 rhe. Both fabrics, when wet, also showed a 20 percent loss in tensile strength due to the exposure. When exposed dry, neither fluidity values nor tensile strengths were changed appreciably. The fabrics demonstrated that increasing the ozone concentration increased the amount of damage to the cellulose.

Morris and Young\(^2\)\(^7\) found in their laboratory experiments that light as well as humidity is necessary to change appreciably breaking strength and cellulose fluidity. In the absence of light, 980 µg/m\(^3\) (0.5 ppm) ozone at 21° C (70° F) and 72 percent relative humidity for 1,200 hours had little degrading effect on Acala 4-42 and Pima S-1 cotton fabrics.\(^2\)\(^8\) In a later study, Kerr et al.\(^2\)\(^9\) showed that light was not necessary to degrade cotton in air containing ozone. Cotton print cloth dyed with C. I. Vat Blue 29 was exposed at 25° C in an airtight chamber containing a shallow pan filled with water so as to increase the relative humidity in the chamber. Purified air to which oxygen had been added was passed through another chamber containing three 4-watt ozone bulbs and then directly into the exposure chamber. The concentration of ozone was adjusted by varying the number of ozone lamps in operation, the amperage used, and the amount of pure oxygen added to the air intake. The concentration of the ozone fed into the chamber was 1960 ± 200 µg/m\(^3\) (1 ± 0.1 ppm), and the exit concentration, recorded with a Mast Ozone Meter, was 980 µg/m\(^3\) (0.5 ppm). Specimens were removed at 3-day intervals and either washed or soaked. Control specimens were kept in light-tight chambers at 21° C (70° F) and 65 percent relative humidity and were given the same cycle of washing or soaking. After 60 days of exposure, the washed fabrics had an 18.2 percent greater strength loss than did the controls. Cellulose fluidity values also indicated degradation caused by exposure to ozone. The washed fabrics exposed to ozone had a fluidity value of 9.27 rhe as compared to a value for the control samples of 5.37 rhe.

Peters and Saville\(^3\)\(^0\) have reported that in their experiments, the effects of high ozone concentrations on breaking strengths are significant for white nylon and polyester fabrics but are not significant for cotton, acetate, or fiberglass. Based on these results and the results of other researchers, the relative susceptibility in increasing order of different fibers to ozone attack is cotton, acetate, nylon, and polyester.

2. Fading of Dyes

The first evidence of ozone causing the fading of dyes was obtained when acetate fiber samples dyed with Disperse Blue-27 (developed for nitrogen dioxide resistance) were field-evaluated.\(^3\)\(^1\) These samples were exposed to the atmosphere in light-tight containers in Pittsburgh, Pa., which has a high nitrogen dioxide concentration, and Ames, Iowa, where a low nitrogen dioxide concen-
tration is recorded. After 6 months, samples in Ames had faded but had not in Pittsburgh. Laboratory exposure of similar samples to 200 \( \mu g/m^3 \) (0.1 ppm) ozone duplicated the fading observed in Ames. It was further found that all blue anthraquinone dyes were sensitive to change as were certain anthraquinone red dyes. Azo red and yellow dyestuffs and diphenylamine yellow dyes were not sensitive to ozone. Thus fading in Ames was attributed to naturally occurring high levels of ozone.

In previously described research, Kerr et al.\(^{29}\) measured the fading of vat-dyed cotton fabrics caused by ozone. The results are presented in Table 7-5 and indicate that the rate of fading and the maximum amount of the fading which will occur are both dependent upon the environment. The soaked fabrics faded in ozone more rapidly and to a greater extent than did the laundered fabrics. It was suggested that laundering produced a change in the dye and improved its ozone resistance.

Salvin exposed wool, cotton, nylon, acetate, orlon, and polyester fabrics, all dyed with ozone sensitive dyes, to the atmosphere of the cities of Chicago and Los Angeles and the rural, nonindustrial areas of Sarasota, Florida, and Phoenix, Arizona.\(^{32,33}\) In Los Angeles, where ozone concentration is high, the dyes were most affected, whereas those exposed in Chicago were the least affected. In humid Florida, dyes faded more than they did in dry Arizona. The extent of ozone fading was related to different concentrations and durations of exposure in the different localities.

Customer complaints of fading have been attributed to attack by ozone.\(^ {34,35}\) The fading of polyester materials was not a problem until cotton/polyester fabrics were finished for permanent press. The type of permanent press treatment which resulted in fading used a magnesium chloride catalyst which formed a soluble complex with the blue dyes used on polyester. The dyes migrated to the finish, where they are susceptible to ozone fading.\(^ {34}\)

A combination of high humidity and ozone has caused fading of nylon carpets.\(^ {35}\) The fading rates of any one dye in ozone-containing atmospheres is a function of the nylon structure as well as the environment. This observation is attributed to the differences in adsorption onto and diffusion into differentnylons. The greater the surface area exposed and the more open the microstructure, the more sensitive the nylon is expected to be to ozone fading. Swelling of nylon due to high humidity should increase both the surface area and the rate of diffusion into the fiber.\(^ {35}\)

By experimentations, combinations of fabrics, dyes, and treatments can be selected to eliminate ozone fading.\(^ {35}\) The additional expense both in research and the use of more costly materials, however, is passed on to the

<table>
<thead>
<tr>
<th>Number of days treated</th>
<th>Washed fabrics</th>
<th>Soaked fabrics</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Gray scale</td>
<td>NBS units(^a)</td>
</tr>
<tr>
<td>12</td>
<td>4.0</td>
<td>1.5</td>
</tr>
<tr>
<td>24</td>
<td>2.5</td>
<td>4.5</td>
</tr>
<tr>
<td>36</td>
<td>2.5</td>
<td>4.5</td>
</tr>
<tr>
<td>48</td>
<td>2.0</td>
<td>6.0</td>
</tr>
<tr>
<td>60</td>
<td>2.0</td>
<td>6.0</td>
</tr>
</tbody>
</table>

\(^a\) NBS units = \( \Delta L^2 + \Delta a^2 + \Delta b^2 \) where:

- \( L = 10 \sqrt{R_d} \) (\(R_d = \text{reflectance}\))
- \( a = \text{measure of green to red color change}\).
- \( b = \text{measure of blue to yellow color change}\).
- \( \Delta = \text{difference between exposed and unexposed}\).
customer and is an indirect cost of ozone in the atmosphere. Since dyes have been found to fade also at ozone levels produced by natural processes, it is extremely difficult to determine what fraction of additional cost may be due to man-made ozone levels.

E. THE NEED FOR FUTURE RESEARCH

It is highly probable that present knowledge of ozone damage to materials is only a small fraction of the total. The interaction effects between ozone and other pollutants on materials has not been investigated. From thermodynamics, it would be expected that ozone would oxidize both nitrogen tetroxide \( \text{N}_2\text{O}_4 \) and sulfur dioxide \( \text{SO}_2 \) to nitrogen pentoxide \( \text{N}_2\text{O}_5 \) and sulfur trioxide \( \text{SO}_3 \) respectively. At the low concentrations found in the atmosphere, the kinetics of the reactions in the gas phase may be too slow to be considered. When adsorbed on solid surfaces of materials, however, the reactions may be catalyzed to appreciable rates. \( \text{N}_2\text{O}_5 \) and \( \text{SO}_3 \) form nitric acid and sulfuric acid respectively when dissolved in water. Many materials are attacked by these acids, and the diurnal condensation-evaporation cycle of atmospheric moisture on material surfaces would be expected to concentrate these dissolved acids if they did not react immediately with the material. In this manner, damaging concentration levels could be reached.

An example of this type of behavior was reported by Morris.\(^{2,8}\) A good statistical correlation was obtained between breaking strengths of cotton fabrics and the pH values of water extracted from them. The breaking strength decreased with a decrease in pH. Correlations were also obtained with relative humidity and the amount of sunlight at the exposure sites. Breaking strength decreased with increases of both humidity and amount of sunlight. The amount of nonfibrous material taken from fabric samples exposed in California in May and June was 1.32 percent and the pH of the water extract was 4.93. In contrast, the value for the unexposed sample was 0.29 percent, with a pH of 6.65.

Much more dose-response data are needed before the economic impact of ozone can be calculated. Although there may be sufficient data for rubber, data for other organic materials such as fabrics, dyes, paints, and plastics are sadly lacking. There have been no investigations to determine the dose-response relationships of combinations of ozone with other pollutants on either organic or inorganic materials.

F. SUMMARY

Although the total extent of ozone-associated damage to materials is not known, ozone may very well be a major contributor to the "weathering" of materials. Ozone is an extremely active compound, and generally any organic material is incompatible with concentrated ozone. Many organic polymers are subject to chemical alteration from exposure to very small concentrations of ozone, including some ambient concentrations. This sensitivity usually increases with the number of double bonds in the chemical structure of the polymer.

Rubber is an economically important material that is highly sensitive to ozone attack. The most vulnerable generic groups of rubber are natural, styrene-butadiene, polybutadiene, and synthetic polyisoprene. Although a tensile stress is necessary for ozone to produce cracking of rubber, rubber products are usually used in this state. Other factors which determine the rate of ozone-attack on rubber are the type of rubber compound, the concentration of ozone, the period of exposure, the rate of diffusion of ozone to the rubber surface, and temperature. Although rubber in a relaxed state can be exposed for long periods of time without visible cracks forming, cracks can develop from exposure to an atmosphere containing 20 to 40 \( \mu \)g/m\(^3\) (0.01 to 0.02 ppm) ozone if the rubber is under a strain of as little as 2 or 3 percent. Antiozonant additives have been developed and are capable of protecting elastomers from ozone degradation. These additives are expensive, and they sometimes migrate with usage to the surface of the rubber product and thus afford only limited protection. Oils, gasoline, and
other chemicals may extract antiozonants from rubber and thus also decrease the resistance of the rubber product to ozone attack.

Ozone attacks the cellulose in textile fabrics through both a free-radical chain-mechanism and an electrophilic attack on double bonds. Light and humidity appear to be factors which must be present before an appreciable alteration occurs in the breaking strength and fluidity of fibers. The relative susceptibility of different fibers to ozone attack, in increasing order, appears to be: cotton, acetate, nylon, and polyester.

Certain dyes are susceptible to fading during exposure to ozone. The rate and extent of fading appear to be dependent on ozone concentration, length of exposure, type of material used, and environmental factors, such as relative humidity and the presence or absence of other atmospheric pollutants. Technology is capable of selecting combinations of fabrics, dyes, and processing which can eliminate ozone fading, but the cost of this will be passed on to the consumer in the form of increased costs.

To calculate the economic costs of ozone air pollution, dose-response relationships must be formulated. At present, this type of data exists only for rubber. Little, if any information is available on dose-response relationships of the effect of combinations of ozone with other air pollutants on materials.

G. REFERENCES


Chapter 8.

TOXICOLOGICAL APPRAISAL OF PHOTOCHEMICAL OXIDANTS

A. INTRODUCTION

Photochemical oxidants such as ozone, oxides of nitrogen, and peroxycyloc nitrites are gases which tend to exert their toxic effect by entering the body through inhalation. If present in sufficient concentrations, these gases are capable of causing death to various organisms. At sublethal concentrations, they may alter, impair, or otherwise interfere with normal respiratory processes.

Alterations in pulmonary function and in the mechanical properties of the lungs are among the effects found as a result of inhalation of these compounds. Other effects which have been investigated include pathological changes in the lungs, chemical and biochemical changes both in the lungs and in other organs, and eye irritation.

The toxicological effects of three major pollutant groups, ozone, "oxidants" (mixtures of substances produced by photochemical reactions), and peroxycyloc nitrites, are discussed in this chapter. Each pollutant group is treated separately, and human and animal data are treated separately within each section.

B. OZONE

1. Animal Data

a. Acute Toxicity

The earliest experiments to determine the acute toxicity of ozone on a variety of animal species were carried out by Bohr and Maar in 1904 and by Hill and Flack in 1912. Gross examination of the respiratory organs showed acute inflammation of the respiratory tract, often with hemorrhage and edema (abnormal accumulation of fluid). Death due to this reaction sometimes occurred within several hours after the exposure was terminated, but usually within 24 hours.

Mittler et al. have determined the LD₅₀ for ozone in a variety of laboratory animals. The LD₅₀ of a toxic substance is defined as that dose expected to kill 50 percent of a population of experimental animals. These results are shown in Table 8-1. In most of the studies carried out, animals were exposed to a constant level of ozone for a given period of time. There were no pathological changes in the lungs of rats exposed to concentrations up to 6,290 μg/m³ (3.2 ppm) for 18 to 22 hours. Data from other sources, although less comprehensive, indicate that the observations of Mittler et al. might occur at much lower concentrations. With exposures from 7,800 to 11,800 μg/m³ (4 to 6 ppm) there were hemorrhagic changes in the lung; above 6 ppm, both hemorrhage and edema occurred (Table 8-2). There are no data at present on the extent of penetration of ozone, but the occurrence of edema at the alveolar level presumably reflects some local damage. Very little is known of the uptake of ozone by pulmonary tissues or of its half-life following uptake.

Table 8-1. LD₅₀ OF OZONE FOR VARIOUS SPECIES AFTER 3-HOUR EXPOSURE

<table>
<thead>
<tr>
<th>Species</th>
<th>Ozone, ppm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Guinea pigs</td>
<td>51.7</td>
</tr>
<tr>
<td>Rabbits</td>
<td>36.0</td>
</tr>
<tr>
<td>Mice</td>
<td>21.0</td>
</tr>
<tr>
<td>Rats</td>
<td>21.8</td>
</tr>
<tr>
<td>Cats</td>
<td>34.5</td>
</tr>
<tr>
<td>Ozone, ppm (C)</td>
<td>Duration of exposure (t), hours</td>
</tr>
<tr>
<td>---------------</td>
<td>-------------------------------</td>
</tr>
<tr>
<td>3.6-4.0</td>
<td>18</td>
</tr>
<tr>
<td>3.0-3.5</td>
<td>20</td>
</tr>
<tr>
<td>2.8-3.2</td>
<td>22</td>
</tr>
<tr>
<td>4.4</td>
<td>18</td>
</tr>
<tr>
<td>3.8-4.4</td>
<td>20</td>
</tr>
<tr>
<td>3.5-4.1</td>
<td>22</td>
</tr>
<tr>
<td>5.0-5.8</td>
<td>18</td>
</tr>
<tr>
<td>6.0-7.7</td>
<td>18</td>
</tr>
</tbody>
</table>

are not the only factors which determine toxicity. In a recent review, Stokinger has listed other factors which influence the response to a specific level of ozone. These include:

1. **Age**: When exposed to 7,800 µg/m³ (4 ppm) for 4 hours, young mice were found to be 2 or 3 times more susceptible to the acute toxic effects of ozone than older animals.

2. **Temperature**: Susceptibility of rats and mice to the toxic effects of ozone is doubled when the temperature is raised from 75°F to 90°F.

3. **Exercise**: Exercise enhances the toxicity of ozone for rats. 1,960 µg/m³ (1 ppm), a level without obvious acute effects, becomes lethal if animals are made to undergo forced activity intermittently for a few minutes each hour during exposure to the gas.

4. **Dosage Rate**: The LD₅₀ dose of ozone decreases with increase in the length of exposure up to 3 hours; it then remains constant. This appears to be valid over a concentration range of from 1,960 to 98,000 µg/m³ (1 to 50 ppm).

5. **Respiratory Infection**: Mice infected with *Klebsiella pneumoniae* (a disease-producing bacterium) and then exposed to 1,960 µg/m³ (1 ppm) ozone have shown shortened survival time and increased mortality. Mice infected with streptococcus (Group C) and then exposed to 160 µg/m³ (0.08 ppm) ozone for 3 hours exhibited increased mortality. This decreased resistance to respiratory infection is noted when the ozone exposures occur either prior to or following infection.

6. **Reducing Agents and Drugs**: Prophylactic administration of reducing agents (de-oxidizing compounds) such as ascorbic acid (vitamin C), both alone and in combination with cysteine (an amino-acid) and glucuronate, substantially reduced the edemagenic response of animals to O₃.

7. **Intermittent Exposure**: The edemagenic response, and the associated alterations in pulmonary function, are reduced markedly by interruption of ozone administration and exposure to air. Intervals as brief as 15 or 20 minutes in repeated 30-minute exposures to 7,800 µg/m³ (4 ppm) ozone reduced edema and mortality among laboratory animals.

In addition to these factors, Skillen confirmed Fairchild's observation of a variation in the effect of ozone exposure on rats with varying thyroid status. Three groups of rats were used. The first received meal containing 0.15 percent propylthiouracil (PTU), a drug which decreases thyroid activity; the second received meal containing 0.01 percent strong desiccated thyroid; the third received untreated meal. After 4 weeks of feeding, these animals were continuously exposed to 11,800
μg/m³ (6 ppm) ozone. The average survival times of the three groups were: greater than 10 hours for those rats with reduced thyroid function; 6.7 hours for rats with unaltered thyroid activity; and 2.2 hours for those rats with stimulated thyroid function.

(1) Effects on the pulmonary organs.

Changes in pulmonary function.

Several workers have determined the extent of pulmonary edema and change in pulmonary function in animals following exposure to ozone. Schell et al. exposed 75 rats to 3,900 μg/m³ (2 ppm) ozone for 3 hours. After their removal from the exposure chamber the oxygen uptake, tidal volume, and frequency of breathing of the rats was measured on a closed circuit respirometer. The rats were killed in groups of five, without further exposure, at intervals over a period of 960 hours. The lungs were excised and weighed before and after drying. The results are shown in Figures 8-1 and 8-2. It was found that the water-content of the lungs increased during the post-exposure period, the increase reaching a maximum after 12 hours. These authors expressed pulmonary edema in terms of water in the lung per kilogram body-weight instead of per unit dry-lung-weight (Figure 8-1). If this exposure to ozone is considered a stress, then changes in body-weight might be expected to occur, especially over the long post-exposure period of 1,000 hours. No data are given on changes in body weights and therefore the changes in lung-water-content given in this paper must be interpreted with caution.

The authors also reported an increase in the dry-lung weights during the first 250 hours of the post-exposure period (Figure 8-1). Again the results are expressed with reference to

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![Figure 8-1](image-url). Changes in lung water and tissue of rats exposed to 3,900 μg/m³ (2 ppm) ozone for 3 hours.\(^5\)
In a second series of experiments, Scheel et al. exposed rabbits to ozone concentrations increasing from 15,700 to 88,200 μg/m³ (8 to 45 ppm) for 1 hour each week. As in the rat experiments, there was a reduction in tidal volume and oxygen uptake, reaching a minimum within 1 hour after exposure (Figure 8-3). These parameters increased during the

Figure 8-2. Average respiratory response of 75 rats exposed to 3,900 μg/m³ (2 ppm) ozone for 3 hours.5

body-weight. Inspection of the data shows an apparent twofold increase in the dry-lung-weight, which can presumably be ascribed to residual cells, protein, etc., resulting from pulmonary edema. The increase could also be partly due, however, to an acute loss of body-weight.

A decrease in minute ventilation (the volume of air breathed per minute), tidal volume (volume per breath), and oxygen uptake occurred immediately after exposure and reached minimum recorded values 8 hours after exposure (Figure 8-2). At 20 hours after exposure, all measurements had returned to initial values. The initial decrease in the minute ventilation and oxygen uptake of the animals could have reflected a decrease in activity (metabolic rate) as a secondary result of the irritating effects of the exposure. One factor which might cause a decrease in activity is the occurrence of pulmonary edema. The simultaneous increase in minute ventilation and decrease in oxygen consumption after 40 hours suggests that delayed impairment occurred. All the data given in this study on changes in pulmonary function were expressed with reference to hours after exposure.

Figure 8-3. Average respiratory response of eight rabbits following first exposure to 29,000 μg/m³ (15 ppm) ozone for 30 minutes.5

following 4 hours of the post-exposure period, but remained below the pre-exposure values for more than 2 days. After repeated exposures to ozone, the rabbits showed not only a marked decrease in tidal volume as a result of the exposure, but also changes in oxygen consumption. For example, the normal control animals consumed 104 to 136 cc of oxygen per minute, whereas the repeatedly exposed animals were able to consume only 68 to 98 cc of oxygen per minute before the tenth exposure. This may have been the consequence, however, of their subjection to considerable stress for 2-1/2 months. Decreased oxygen uptake could be the result of decreased activity or of loss of weight, both of which could be secondary to the effects of ozone.

Murphy et al. exposed guinea pigs to from 590 to 2,650 μg/m³ (0.3 to 1.35 ppm)
ozone for a period of 2 hours. Measurements of respiratory rates, tidal volumes, and total respiratory flow resistances were made before, during, and after exposure. It was observed that respiratory rates increased and tidal volumes decreased during exposure to all concentrations (Figure 8-4). The maximum changes were significantly different \( p < 0.05 \) from the pre-exposure control values obtained for each concentration shown. After a maximum 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 levels when the animals were returned to clean air. Total respiratory flow resistances were not significantly altered during inhalation of ozone concentrations of 670 to 1,330 \( \mu \text{g/m}^3 \) (5 to 7 ppm) for 2 hours.\(^1\) Measurement of flow resistance, frequency of breathing, tidal volume, and lung compliance were made before, during, and after exposure. The results are shown in Figure 8-5. A threefold increase in flow resistance occurred during exposure and reached a maximum after 1 hour. There was a 50 percent increase in frequency of breathing and a small decrease in tidal volume. Lung compliance (a parameter which had not been measured previously) decreased 50 percent. After exposure, all measurements returned to pre-exposure values within 180 minutes.

Pathological changes.

Gross autopsy findings of pulmonary edema and hemorrhage following acute ozone exposure have been known for some time.\(^1\)\(^1\)\(^1\)

Scheel\(^5\) et al. have provided histopathologic evidence of injury caused by a single acute exposure of 1,960 or 6,270 \( \mu \text{g/m}^3 \) (1.0 or 3.2 ppm) ozone for 4 hours in mice and by repeated intermittent exposures of 15,700 to 88,200 \( \mu \text{g/m}^3 \) (8 to 45 ppm) ozone for 1 hour in rabbits. No gross pulmonary edema was observed in mice killed immediately after exposure to 1,960 \( \mu \text{g/m}^3 \) (1 ppm), but moderately engorged blood vessels and capillaries containing an excess of leukocytes (white blood cells) were found. Mice killed 20 hours after exposure showed mild edema and migration of the leukocytes into the alveolar spaces. Superficial desquamation of the epithelium (peeling away of surface cells) in the bronchi and bronchioles was also observed. Inhalation of 6,270 \( \mu \text{g/m}^3 \) (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 varying degrees of extravasation of red cells (migration of cells from the capillaries into the tissues) accompanied the edema. Damage to the respiratory tract consisted of loss of epithelium from the bronchioles, and sheets of desquamated epithelial cells were seen in the lumen. Repeated exposures of rabbits to ozone have resulted in damage to the epithelium of the respiratory tract, increasing in severity from the lower trachea to the bronchioles. Growth of fibrous tissue was found in the walls of the bronchioles and in the alveolar ducts. The external surface had a puckered and granular appearance in one rabbit after the 49th exposure, suggestive of shrinkage of fibrous tissue. The time of onset of the fibrous changes was not determined, but the respiratory tract of one rabbit killed after the 25th exposure contained no such lesions.

Chemical and biochemical changes.

The elastic behavior of the lungs is determined by both fixed tissue elements and other substances, such as surfactant (a natural surface-active agent lining the alveoli), or by edema. There is evidence that these factors might be affected by the chemical changes that have been observed in the lung tissue following exposure to ozone.

Lung tissue "ground substance" may include hyaluronic acid, chondroitin sulfuric acids A, B and C, heparin, keratosulfate, and the proteins collagen and elastin. Theoretically, the latter compounds would be expected to be oxidized by ozone to yield aldehydes. In one experiment, Buell\(^1\)\(^2\) et al. used
Figure 8-4. Effect of ozone exposure on respiration of guinea pigs.\textsuperscript{9}
four litter-mate rabbits; 2 were exposed to 1,960 μg/m³ (1 ppm) ozone for 1 hour, 1 of which was sacrificed immediately after exposure and the other 24 hours later. Another was killed immediately after exposure to 9,800 μg/m³ (5 ppm) for 1 hour. The fourth was used as a control. After hydrolysis of the protein fraction of the lung tissue by the enzymes hyaluronidase, pepsin, elastase, and collagenase, the present of carbonyl compounds (e.g., aldehydes and ketones) was observed. The carbonyl compounds were identified spectrophotometrically as the corresponding dinitrophényl-hydrazone. It was concluded that the carbonyl compounds were most likely derived from lung proteins and that structural changes in lung tissue might have occurred. Ozone might also affect the ground substance, since the data suggest that oxidative degradation of hyaluronic acid could have occurred. The similarity of data obtained from animals sacrificed immediately after exposure and 24 hours after exposure suggests that no reversal of the process occurred within that period of time. The authors suggested that decreased slippage and decreased flexibility of elastic protein molecules might occur if the ground substance is destroyed by ozone. The effect could be enhanced by intra- and inter-molecular cross-
linking of aldehydes with the fibers of elastic protein molecules.

This experiment confirmed the results of preliminary experiments in which three sets of rabbits were used, one set to develop the methodology and two sets to determine if the observation was real. In all cases, no carbonyl compounds were found in the lungs of control rabbits.

Mendenhall and Stoking have exposed saline washings obtained from the lungs of mice to 9,800 to 15,700 μg/m³ (5 to 8 ppm) ozone and noted rapid increases in the film pressure (the force opposing surface tension). The authors suggested that if analogous changes were to occur in vivo, the consequence would be an increase in the distensibility of the lungs, considered by the authors to be conducive to the development of emphysema. This effect was not confirmed in another study in which saline washings from the lungs of dogs were exposed in vitro to similar concentrations of ozone. If pulmonary edema were to supervene because of ozone exposure, one would expect to observe an effect different from that seen by Mendenhall and Stoking; that is, there would be a reduction of surfactant and an associated increase in surface tension especially at low lung volumes, a resultant decrease in alveolar stability, and a reduction in distensibility of the lungs.

Frank et al. have exposed the right lung of rabbits to 4,300 to 23,500 μg/m³ (2.2 to 12.1 ppm) ozone for 3 hours, the left lung having been collapsed prior to exposure. When edema occurred in the right lung, changes in surfactant behavior were observed in the left lung of some animals; no such changes were observed in the absence of edema in the right lung. These results suggest that ozone is not only capable of inducing chemical changes in exposed lungs but also that the products of such changes are capable of producing deleterious effects or compensatory responses in non-exposed lungs.

Skilley et al. have induced pulmonary edema in rats by exposing them to 11,800 μg/m³ (6 ppm) ozone for 4 hours. The animals were sacrificed immediately after exposure and their lungs removed. Significant increases (p < 0.001) in lung weight, lung protein content and lung serotonin (5-hydroxytryptamine) were observed. The source of the increase in lung serotonin could have been due in part to the increase in the number of pulmonary mast-cells and their subsequent disintegration. Whether this increase in serotonin is related to pulmonary edema remains to be demonstrated.

(2) Systemic effects.

Biochemical changes.

Several experiments described in greater detail later in this chapter have provided evidence that ozone may exert an effect at the cellular level which is similar to that of radiation. In these studies, free radicals have been suggested as the basic biochemical mechanism of ozone-associated cellular damage; this same chemical species is also believed to mediate radiation-induced cell damage. Based on cellular changes of human red blood cells in vitro after exposure to 78,000 μg/m³ (40 ppm) ozone for 2 hours, and mouse red blood cells in vivo after exposure to 15,700 μg/m³ (8 ppm) for 4 hours, Goldstein et al. have suggested that ozone-induced cellular damage may be mediated by its ability to react with the double-bonds of unsaturated fatty acids, resulting in the formation of free radicals and other intermediate species which might be responsible for the cellular damage. These free radicals (from ozonide and peroxide intermediates) may also enter into similar reactions with other unsaturated fatty acids, initiating a chain reaction of cellular damage. Thus, Goldstein et al. imply that cellular damage may result both directly from ozonolysis and secondarily from lipid peroxidation initiated by the products of ozonolysis.

Immunology.

Scheel et al. reported the presence of circulating antibodies (precipitins) in the serum of rabbits exposed to ozone. Ordinarily,
such antibody responses are elicited after introduction of a foreign protein into an animal. After exposing eight rabbits to 19,600 µg/m³ (10 ppm) ozone for 1 hour each week for a 6-week period, these investigators reported a positive precipitin test when serum from the exposed rabbits was overlaid with an antigen prepared by exposing a 0.1 percent solution of egg albumin to dry gas containing 4,500 to 4,900 µg/m³ (2,300 to 2,500 ppm) ozone. The data indicate that the interaction of ozone and albumin resulted in denaturation or change in the conformation of the albumin.

Buell undertook to reproduce Scheel’s results but was unable to confirm the precipitin reaction using the exposure of rabbits to ozone. Buell also employed a variety of sensitive techniques in an attempt to demonstrate immunologic responses in other ways. It is likely that cross-reactions between native and heat-denatured ovalbumins of the type described by Scheel are largely due to the impurities conalbumin or ovomucoid.

A report by Erban indicates that various noxious stimuli may evoke or elicit nonspecific responses in rabbits. The serum globulin fraction, for example, in particular the alpha-globulin fraction, increased in rabbits exposed daily for 9-1/2 hours to 36 mg/m³ sulfur dioxide (13.8 ppm) for 80 days. Thus, it should be concluded that an immunological response attributable to ozone exposure is not yet proven, although the possibility exists that exposures to ozone might aggravate pre-existing sensitivity or immunologic processes.

Brinkman et al. have shown that inhalation of 390 to 490 µg/m³ (0.20 to 0.25 ppm) ozone for periods of 30 to 60 minutes by mice, rats, rabbits, and man increased the rate of sphering of red cells in vitro, the cells losing their characteristic biconcave shape more rapidly following exposure of diluted blood to radiation. This would indicate an acceleration in the aging of the cells. These changes were largest after 1 hour and diminished toward the control after 6 hours exposure.

Mountain has reported that the glutathione content of lung extracts of mice was reduced following acute ozone exposure. Glutathione is known to be essential to the integrity of the red blood cell. It exists in both the oxidized (GSSG) and reduced (GSH) form and may be enzymatically converted from one form to the other. Only GSH is physiologically active, and it is this form which has been measured by various investigators. It is possible that exposure to ozone either oxidizes the sulphydryl group of glutathione or prevents the formation of the reduced form. Either possibility would result in an abnormal excess of the oxidized form. GSSG is known to combine with hemoglobin in vitro, resulting in a decreased affinity of the blood for oxygen; this in turn causes a decrease in the amount of oxygen released to the tissues. The presence of GSH inhibits the formation of the hemoglobin-GSSG complex due to the presence of its free sulphydryl group, and thus oxidation of glutathione must be almost complete if the mechanism is to be effective in vivo.

**Effects of ozone on other organs.**

Chemical and biochemical changes have been observed in the heart, liver, and brain of animals following inhalation of ozone. Brinkman et al. have exposed adult mice to 390 µg/m³ (0.2 ppm) ozone for 5 hours daily for 3 weeks. Structural changes in the cell membranes and in the nuclei of myocardial muscle fibers were produced which were reversible after about 1 month following exposure. The physiological implications of these changes were not discussed. Skillen et al. have demonstrated a significant decrease (p < 0.001) in brain serotonin following exposure of rats to 11,800 µg/m³ (6 ppm) for 4 hours. The mechanism of this response is unknown.

Acute exposure of laboratory animals to high concentrations of ozone causes stimulation of the activity of the liver. Scheel et al.
exposed rats to 17,990 µg/m³ (9.2 ppm) ozone for 45 minutes. A large increase in the liver RNA/DNA ratio was observed during the first 4 hours after exposure, but the ratio rapidly returned to normal. Murphy et al. found significant increases (p < 0.01) in liver-weight per unit body-weight and liver alkaline phosphatase activity following a single exposure of rats to 6,100 µg/m³ (3.1 ppm) for 20 hours. No significant increases were observed after an exposure of 7,470 µg/m³ (3.8 ppm) for 4 hours. In both of these studies, no attempt was made to determine the minimal exposure conditions which would produce these effects. The role of alkaline phosphatase within the animal body is only partially known, although it has frequently been used as an index of the response to stress.

Since inhaled ozone itself can directly affect only the lining of the respiratory tract, changes in extra-pulmonary organs may be the result of the breakdown of material within the lung with a secondary systemic effect. It should be emphasized that the biochemical changes which take place in the liver and brain are the result of abnormally high exposures to ozone, and, subject to the animal's survival, are potentially reversible.

b. Effects of Prolonged Exposure to Ozone

According to Stokinger's review, at least three long-term effects of repeated exposures to ozone have been recognized. These are: (1) long-term pulmonary effect, (2) lung tumor acceleration, and (3) aging. An additional effect, (4) the development of tolerance following low level exposure to ozone, is also related to chronic toxicity.

(1) Long-term pulmonary effects.

Stokinger et al. reported that chronic bronchitis, bronchiolitis, and emphysematous and fibrotic changes in the lung tissues occur in mice, rats, hamsters, and guinea pigs exposed 6 hours daily, 5 days a week for 14-1/2 months to a concentration slightly above 1,960 µg/m³ (1 ppm) ozone. These irreversible changes also develop in animals tolerant to acute inflammatory effects.

(2) Lung tumor acceleration.

Acceleration of lung tumorigenesis (adenoma) in a strain of mice susceptible to such tumors occurred from daily ozone exposures of about 1,960 µg/m³ (1 ppm). At 15 months, a tumor incidence of 85 percent was seen in the ozone-exposed, as against 38 percent in the control mice; the average number of tumors per exposed mouse was 1.9 compared with 1.5 in the controls.

(3) Aging.

There are some suggestive data that exposure to ozone may accelerate the aging process. Bjorksten has presented evidence that aging may be due to irreversible cross-linking between macromolecules, principally proteins and nucleic acids. He included aldehydes in his list of active cross-linking agents. Aldehydes are potential cross-linking agents, and may be produced in the lung by ozone exposure.

Hutker et al. exposed mice and guinea pigs for 23 months to irradiated automobile exhaust containing 390 to 1,960 µg/m³ (0.2 to 1.0 ppm) ozone; also present in the mixture were 23 to 115 mg/m³ (20 to 100 ppm) carbon monoxide, 6 to 36 ppm hydrocarbons, and 0.7 to 3.9 ppm nitrogen oxides. It was noted that irradiated auto exhaust increased susceptibility to pulmonary infection and chronic disease (bronchitis), particularly in the latter half of the animal's life, although no change in immunological response was demonstrated.

Stokinger reports an accelerated or premature aging in rabbits after 1 year of weekly 1-hour exposures to ozone. His evidence included: premature calcification of the sternocostal cartilage, unthrifty appearance and coarseness of the pelage (hairy system of the body), severe depletion of body fat, and general signs of senescence, such as dull cornea and sagging conjunctivae.
It has been suggested that the radiomimetic properties of ozone are implicated in its effects on aging. Fetner,\textsuperscript{19} in demonstrating chromosome breakages in human cell cultures from exposure to 15,700 \( \mu \text{g/m}^3 \) (8 ppm) ozone for 5 or 10 minutes, suggested that the effects of ozone and irradiation are additive. This is supported by the data of Brinkman\textsuperscript{20} et al.

Buell\textsuperscript{13} suggested that the interaction of ozone and moisture (water) results in the formation of atomic- or radical-oxygen and molecular oxygen, while Alder\textsuperscript{38} postulated the formation of a variety of radicals. Although high-energy sources such as X-radiation undoubtedly decompose water into a variety of free radicals, it has not been shown that ozone can form more than one.

(4) Development of tolerance to low-level exposures.

One feature of the response to oxidants, and in particular, ozone, which has stimulated considerable interest is the apparent development of tolerance to the short-term effects of these agents in laboratory animals. Tolerance is the acquired capacity of a pre-treated host to exhibit a lesser response to a challenge than would be observed in a comparable but naïve (non-pretreated) host. Tolerance is demonstrable directly at a single brief 1 hour or less exposure to very low levels, from 590 to 5,900 \( \mu \text{g/m}^3 \) (0.3 to 3.0 ppm) ozone. Tolerance occurs after a nonedemagenic as well as an edemagenic dose. In rats, a tolerance is sometimes developed that lasts for a month or longer, while in mice, a tolerance up to 14 weeks has been observed.

It must be emphasized that ozone is not the only compound which is capable of producing tolerance. Nitrogen dioxide, phosgene, and phenylthiourea are among many others. It is interesting that tolerance evoked by one agent can provide cross-protection against one or more irritants. For example, a single exposure of rats or mice to 980 to 9,800 \( \mu \text{g/m}^3 \) (0.5 to 5 ppm) ozone for 1 to 5 hours will induce protection against the acute pulmonary effects of nitrogen dioxide, hydrogen peroxide, ketene, phosgene, hydrogen sulfide, and nitrosyl chloride.\textsuperscript{39} The development of pulmonary edema is the toxic effect shown by all of these compounds. Although pretreatment by injection of phenylthiourea will provide tolerance against inhalation of a lethal dose of ozone, the reverse is not true; inhalation of ozone will not provide a cross-tolerance against challenge by injection of thiourate. The development of tolerance to ozone has also been noted by Coffin\textsuperscript{40} et al., employing altered response to bacterial infection as the parameter (Table 8-3).\textsuperscript{38} This suggests that the cellular mechanism responsible for resistance to infection, presumably the macrophage system, is capable of exhibiting ozone tolerance. In the reported experiments, however, tolerance on the basis of edema development cannot be precluded, since the ozone concentration of 1,960 \( \mu \text{g/m}^3 \) (1 ppm) is probably edemagenic for mice. Experiments reported by Pace\textsuperscript{41} et al., in which in vitro exposure of standard tissue cultures of live cells were employed, indicate, however, that protection can be conferred by previous exposure to ozone. This argues strongly that an epicellular or membrane effect or an intra-cellular mechanism is responsible for tolerance, since isolated cells in synthetic medium should be uninfluenced by hormonal or other mechanisms mediated through humoral agents.

Henschler\textsuperscript{42} has demonstrated tolerance to nitrogen dioxide in mice. It was found that those mice which had been pregassed only once were almost totally protected, whereas those which were repeatedly pregassed were only partially protected. It was concluded that the first exposure protected against the following exposures, thus inhibiting increased tolerance build-up.

In another experiment, Henschler\textsuperscript{43} induced tolerance and eventually managed to challenge the animals with a lethal dose. It is significant that none died with pulmonary edema; the predominant gross findings were
Table 8-3. DEVELOPMENT OF TOLERANCE TO THE MORTALITY-ENHANCING EFFECT OF OZONE ON STEPTOCOCCAL INFECTION

<table>
<thead>
<tr>
<th>Number of mice per group</th>
<th>Ambient air</th>
<th>1 ppm ozone for 3 hours</th>
<th>1 ppm ozone for 3 hours, repeated 24 hours later</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>35</td>
<td>75</td>
<td>40</td>
</tr>
<tr>
<td>20</td>
<td>40</td>
<td>90</td>
<td>70</td>
</tr>
<tr>
<td>20</td>
<td>20</td>
<td>65</td>
<td>35</td>
</tr>
<tr>
<td>20</td>
<td>20</td>
<td>80</td>
<td>55</td>
</tr>
<tr>
<td>20</td>
<td>10</td>
<td>70</td>
<td>10</td>
</tr>
<tr>
<td>100</td>
<td>25%</td>
<td>76%</td>
<td>46%</td>
</tr>
</tbody>
</table>

*The differences in mortality between the group exposed to ambient air and the group exposed to 1 ppm for 3 hours, and between the latter group and the group exposed twice to ozone, were statistically significant (p < 0.001).

massive hemorrhage. The mechanisms of tolerance are uncertain. The possibility that a gaseous irritant will produce tolerance is apparently governed partially by its solubility. Gases and vapors with a relatively low solubility can contact all parts of the respiratory tract from the nasal passages down to the bronchioli and alveoli. Irritants which reach these inner recesses of the lung are referred to as deep lung irritants, and it is these which are capable of producing tolerance. Irritants which ordinarily do not reach the alveoli in sufficient concentration, however, appear to be devoid of tolerance development. Thus materials such as sulfur dioxide, chlorine, ammonia, and isocyanates, when inhaled in low concentrations, do not have the ability to promote protective tolerance against subsequent challenge.

The development of tolerance produces certain characteristics after such a challenge. These are:

1. Pathology: The usual changes associated with pulmonary edema are either reduced or absent.
2. Water content: The usual increase in lung water content is either reduced or does not occur.
3. Biochemistry: The activity of serum alkaline phosphatase and of adrenal succinic dehydrogenase remains either normal or only slightly altered in tolerant animals. In addition, there is significantly less oxidation of lung GSH in the animal pretreated with ozone.

It must be pointed out that the existing data suggest that rodents are particularly likely to develop tolerance. Quilligan et al., using several different conditions of exposure and challenge, were unable to demonstrate tolerance to ozone in baby chicks. It is not known whether humans can develop tolerance to ozone.

It would appear that the development of tolerance is a useful tool to determine the mechanism for the production of ozone-induced pulmonary edema. In the light of Henschler's observation that repeated intermittent exposures of mice to nitrogen dioxide produced less protection against challenge with a lethal dose than a single pre-exposure, tolerance may not be significant in populations continuously exposed to low levels of oxidants. Also, indices other than mortality need to be developed in animals before tolerance development can be appraised in humans.

c. Interaction with Other Agents

(1) Increased Susceptibility to bacterial infection

It has been observed that animals challenged with aerosols of infectious organisms suffer a higher incidence of infection when previously exposed to ozone, irradiated auto exhaust, or other common air pollutants. The suggested explanation is that the various pollutants inhibit, inactivate, or otherwise impair two distinct functions: the mucociliary streaming, that is, the action of cilia (tiny, hair-like, mobile, projections of cells) in the nasal and upper respiratory passages to clear particulates and prevent them from entering the lungs; and phagocytosis by the alveolar macrophages which surround and digest foreign particles in the lungs.

Coffin et al. studied the effect of ozone exposure on rabbits by means of
pulmonary lavage to obtain data on alteration of the cells. They found that exposure for 3 hours to ozone at concentrations from about 1,960 μg/m³ (1 ppm) and above elicited an influx of heterophilic (polymorphonuclear) leukocytes into the pulmonary airway (p < 0.001); this is illustrated in Figure 8-6. The elevation of heterophiles was noted immediately after cessation of the 3-hour exposure, peaked at 6 hours, and was still evident in reduced amounts 24 hours later (Figure 8-7). The authors interpreted this finding as indicating an acute reversible inflammatory response to irritation. At the higher exposure levels used, there was correlation with inflammation noted in tissue sections, while at lower levels, cells obtained by lavage indicated an irritation response not detected by histologic examination.

These same investigators noted by similar lavage techniques that administration of ozone also reduced the number and in vitro phagocytic abilities of the pulmonary alveolar macrophages. The reduction in phagocytosis was noted following 3-hour exposures to concentrations of 1,310 to 7,800 μg/m³ (0.67 to 4 ppm) ozone (p < 0.005). The authors suggest that this reduced phagocytosis conferred by ozone may play a role in the increased survival in the lungs of bacteria, with consequent enhancement of infection for ozone-exposed animals. (Table 8-4).

![Diagram](image1)

**Figure 8-6.** Cellular response to exposure to ozone for 3 hours.45

![Diagram](image2)

**Figure 8-7.** Increase in percent heterophiles following exposure to 9,800 μg/m³ (5 ppm) ozone for 3 hours.45

<table>
<thead>
<tr>
<th>Ozone dose, ppm</th>
<th>Phagocytic index x 100</th>
<th>Number of rabbits</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>498</td>
<td>20</td>
</tr>
<tr>
<td>0.67</td>
<td>442</td>
<td>6</td>
</tr>
<tr>
<td>1.25</td>
<td>341</td>
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<td>2.50</td>
<td>303</td>
<td>6</td>
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<td>3.75</td>
<td>281</td>
<td>6</td>
</tr>
<tr>
<td>5.00</td>
<td>286</td>
<td>6</td>
</tr>
<tr>
<td>9.50</td>
<td>291</td>
<td>6</td>
</tr>
</tbody>
</table>

Table 8-4. DEPRESSION OF PHAGOCYTOSIS AFTER 3-HOUR EXPOSURE TO OZONE 45

Holzman et al., on the basis of determinations from pulmonary wash-out fluid, have demonstrated that a 3-hour exposure to 9,800 μg/m³ (5 ppm) ozone reduced the activity of the bactericidal enzyme, lysozyme, presumably due to in vivo oxidation of the enzyme.

In experiments reported by Coffin et al. and Stokinger and Coffin, mice were exposed to ozone at various levels for 3 hours and subsequently to a bacterial aerosol of streptococcus (Group C). Enhancement of mortality occurred from ozone concentrations of 160 μg/m³ (0.08 ppm) and above (p < 0.05); these data are shown in Table 8-5. The same investigators also showed that prolongation of pulmonary bacterial
It was concluded that exposure to ozone significantly reduces the resistance of mice and hamsters to subsequent respiratory infection due to *K. pneumoniae*. Statistical evaluation of the data indicated higher mortality, shorter survival time, and a lower LD₅₀ for *K. pneumoniae* in animals exposed to ozone as compared with controls.

In a similar series of experiments conducted by Purvis⁴⁹ mice were exposed to 7,470 to 8,000 µg/m³ (3.8 to 4.1 ppm) ozone for 3 hours. The exposures took place 1 to 27 hours before and 3 to 27 hours after challenge with *K. pneumoniae* aerosol. It was found that, within 19 hours after exposure to ozone, the resistance of mice suffering from respiratory infection initiated by challenge with an aerosol of *K. pneumoniae* was significantly reduced. The same effect was observed in infected animals exposed to ozone up to 27 hours after the challenge with the infectious aerosol.

(2) Increased susceptibility to histamine

In an attempt to define a mechanism for the edemagenic effect of ozone, Easton¹⁰ et al. noted that ozone-exposed guinea pigs appeared to be more susceptible to the toxic action of histamine, a chemical substance released by the tissues in allergic reactions. It was observed that pre-exposure to 9,800 µg/m³ (5 ppm) ozone for 2 hours followed by a histamine challenge of from 0.9 to 1.4 mg/kg (injected 1.5 to 2.0 hours after the end of exposure) resulted in increased mortality compared with an air-exposed control group; these results are illustrated in Figure 8-8.

Autopsy of the dead animals showed that the members of both groups died of acute bronchoconstriction. The increased susceptibility to histamine was detectable for 12 hours after the termination of exposure to ozone. The minimum concentration of ozone necessary to produce such an increase in susceptibility was found to be 980 to 1,960 µg/m³ (0.5 to 1.0 ppm). This concentration is only one-twentieth of that required to produce death from pulmonary edema due to ozone alone. Since there was no significant

---

<table>
<thead>
<tr>
<th>Ozone ppm</th>
<th>Number of mice per group</th>
<th>Percent Mortality</th>
<th>Step only</th>
<th>Step + ozone</th>
<th>Difference percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>.52</td>
<td>40</td>
<td>13</td>
<td>80</td>
<td>67⁺</td>
<td></td>
</tr>
<tr>
<td>.35</td>
<td>40</td>
<td>0</td>
<td>60</td>
<td>60⁺</td>
<td></td>
</tr>
<tr>
<td>.30</td>
<td>40</td>
<td>8</td>
<td>63</td>
<td>55⁺</td>
<td></td>
</tr>
<tr>
<td>.28</td>
<td>40</td>
<td>3</td>
<td>40</td>
<td>37⁺</td>
<td></td>
</tr>
<tr>
<td>.20</td>
<td>40</td>
<td>8</td>
<td>50</td>
<td>42⁺</td>
<td></td>
</tr>
<tr>
<td>.18</td>
<td>40</td>
<td>0</td>
<td>63</td>
<td>63⁺</td>
<td></td>
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<td>.17</td>
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<td>8</td>
<td>35</td>
<td>27⁺</td>
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</tr>
<tr>
<td>.08</td>
<td>40</td>
<td>15</td>
<td>38</td>
<td>23⁺</td>
<td></td>
</tr>
<tr>
<td>.07</td>
<td>40</td>
<td>15</td>
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<td>20</td>
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<tr>
<td>.07</td>
<td>40</td>
<td>8</td>
<td>5</td>
<td>-3</td>
<td></td>
</tr>
</tbody>
</table>

⁺p < 0.05.
difference between lung water content in the two groups, it was concluded that pulmonary edema did not contribute to the increased susceptibility to histamine. It was found that the increased susceptibility only occurred when exposure to ozone took place before challenge with histamine. There was no increase in susceptibility in guinea pigs injected with histamine prior to exposure. This is in contrast to the increased susceptibility to *K. pneumoniae* before and after exposure to ozone.

**d. Mechanisms of Ozone Toxicity in Animals**

The existing data suggest that ozone exerts its toxic effect primarily on the respiratory system and secondarily on the other extrapulmonary systems. In the latter case, some of the responses are essentially those of stress and are not specific for ozone. The exact mechanisms by which ozone causes damage to the respiratory system, however, are still unknown. Research to elucidate such mechanisms is currently being conducted in three major areas—the acute toxic edema reaction, the effects on oxygen transport by the red cells, and the development of tolerance. The latter is used frequently as a model system to determine the mechanism of the edema reaction.

In animals, it is suggested that some responses to ozone are due to irritation at the local levels. An example is the immediate increase in pulmonary flow resistance observed during exposure to ozone, and its rapid return to normal after exposure. Because of the delayed edemagenic response observed frequently after short-term exposure to high concentrations of ozone, it is likely that the formation of pulmonary edema operates by some other mechanism. Data are available from various sources which suggest that ozone exerts a direct effect on the cell membrane and not by way of a neural or hormonal pathway.

Fairchild et al. provided evidence that ozone interacts with sulphhydryl groups along the respiratory tract and in the lung. Their conclusions were based on the protection against the effects of ozone provided by administration of organic sulfur-containing compounds. How ozone reaches the active sulphhydril groups is open to some question. It is possible that the intact molecule reacts with the receptor site, but it is more likely that, since ozone is so reactive, it reacts with water to form molecular oxygen or other active forms of oxygen.  

Glutathione is one compound which contains a free sulphhydryl group and which has been implicated in the mechanism for the formation of pulmonary edema. It has been observed that lung GSH levels were maintained in ozone-tolerant animals but not in non-tolerant animals. This situation could be brought about in two ways—either through interaction of ozone with the sulphhydryl group or through interference with the enzymatic pathways (pentose phosphate shunt) which are responsible for maintaining adequate levels of reduced glutathione. It should be pointed out that glutathione is not itself diminished, but is converted to the oxidized form and is therefore inactive. The processes of inflammation and repair are accompanied by an increased utilization, by glucose, of the pentosephosphate pathway in the injured area. The decrease in the levels of GSH
would inhibit this pathway and hence inhibit the process of repair. This would explain the delayed edemagenic response frequently observed after exposure to acute concentrations of ozone.

Buell et al. have demonstrated changes in the formation of lung proteins and ground substances in rabbits after exposure to ozone; these changes were not reversible within 24 hours after exposure. The potential intra- and inter-molecular cross-linking of aldehydes with fibers of elastic protein molecules would result in decreased flexibility of the latter and hence imply a simultaneous reduction in the elasticity of the lung. It would be desirable for such a study to be put on a quantitative basis so that the extent of and subsequent renaturation or replacement of lung tissue could be determined.

The radiomimetic properties of ozone have been suggested as a basis for its edemagenic response, although the data obtained so far provide conflicting conclusions which have already been noted in this report. The evidence in support of this theory, however, is as follows:

1. The reducing agents which protect against ozone also protect against irradiation effects;
2. Both ozone and X-radiation produce chromosomal aberrations; and

Dixon and Mountain have suggested that the liberation of histamine, thought to result from ozone exposure, has a role in the formation of pulmonary edema. However, Easton et al. were unable to demonstrate a significant depletion of lung histamine in guinea pigs exposed to ozone. Additional evidence that ozone or one of its reaction products exerts its edemagenic effect by a direct interaction with the cell membrane is given by Stokinger in a recent review. He cites a personal communication from Pace who has shown that ozone affects cultures of liver and HeLa cells (cultured human cancer cells) in much the same way as it affects the cells of an intact animal.

It must be concluded that, although there are a number of facts which are suggestive of certain mechanisms, the data obtained so far are inadequate to provide a mechanism for ozone-induced pulmonary edema. The responses of the extra-pulmonary organs to exposures of high concentrations of ozone (possibly excluding effects on oxygen transport by red blood cells) are essentially those of stress. These are not specific for ozone and are frequently encountered in animals assaulted by a variety of toxic chemicals. A neuro-humoral mechanism has been suggested, but its exact nature is still unclear.

e. Summary

A detailed summary of the effects of ozone in animals is shown in Table 8-6. The concentrations listed are the lowest for which the observed effect has been recorded. Based on experimental data, it has been found that:

1. Pathological changes, including bronchitis, bronchiolitis, emphysema, and fibrosis, are produced by prolonged exposure to 390 to 1,960 µg/m³ (0.2 to 1.0 ppm) ozone.
2. Changes in pulmonary function are produced by exposure to 590 µg/m³ (0.3 ppm) ozone for periods of up to 2 hours. These changes, which revert to normal following exposure, are decreased tidal volume, increased frequency of breathing, and increased flow resistance.
3. Chemical changes in the structural proteins of the lung are found following exposure to 1,960 µg/m³ (1 ppm) for 1 hour.
4. Mortality rates resulting from histamine, bacterial infections, age, and exercise are increased as a result of exposure to from 1,960 to 9,800 µg/m³ (1 to 5 ppm) ozone for 2 to 6 hours.
5. Biochemical changes in the lung and other organs are found following exposure to 5,900 to 11,800 µg/m³
<table>
<thead>
<tr>
<th>Ozone,$^a$ $\mu g/m^3$</th>
<th>Ozone,$^a$ ppm</th>
<th>Length of exposure</th>
<th>Observed effect(s)</th>
<th>Species</th>
<th>Reference</th>
</tr>
</thead>
</table>
| 160                    | 0.08          | 3 hours          | Local effects  
Short-term exposures  
Increased susceptibility to streptococcus (Group C)  
30% increase in frequency of breathing;  
20% decrease in tidal volume                                                                                                                                 | Mice    | Coffin$^40$ et al. |
| 670                    | 0.34          | 2 hours          | Increased flow resistance                                                                                                                                                                                                                                           | Guinea pigs | Murphy$^9$ et al. |
| 1,330                  | 0.68          | 2 hours          | No significant increase in flow resistance                                                                                                                                                                                                                       | Guinea pigs | Murphy$^9$ et al. |
| 1,960                  | 1.00          | 1 hour           | Chemical changes in ground substance and lung protein                                                                                                                                                                                                                | Rabbits | Buell$^{12}$ et al. |
| 1,960                  | 1.00          | 4 hours          | Engorged blood vessels and excess leukocytes in lung capillaries  
Increased susceptibility to *Klebsiella pneumoniae*                                                                                                                                                   | Mice, hamsters | Müller and Ehrlich$^{48}$ |
| 2,120                  | 1.08          | 2 hours          | Increased flow resistance                                                                                                                                                                                                                                           | Guinea pigs | Murphy$^9$ et al. |
| 2,550                  | 1.30          | 3 hours          | Increased susceptibility to *Klebsiella pneumoniae*                                                                                                                                                      | Mice, hamsters | Müller and Ehrlich$^{48}$ |
| 3,900                  | 2.00          | 3 hours          | Increased lung weight, decreased tidal volume, decreased minute ventilation                                                                                                                                  | Rats    | Schel$^5$ et al. |
| 6,290                  | 3.20          | 4 hours          | Gross pulmonary edema                                                                                                                                                                                                                                               | Mice    | Schel$^5$ et al. |
| 9,800                  | 5.00          | 2 hours          | Increased lung compliance, increased susceptibility to histamine                                                                                                                                           | Guinea pigs | Easton and Murphy$^{10}$ |
| 9,800                  | 5.00          | 3 hours          | Decreased activity of bacteriocidal lysozyme                                                                                                                                                              | Mice, rabbits | Holzman$^{46}$ et al. |
| 11,800                 | 6.00          | 4 hours          | Gross pulmonary edema, increased lung serotonin                                                                                                                                                           | Rats    | Skillen$^{17}$ et al. |
| 20,000                 | 15.00         | 30 min.          | Decreased tidal volume, decreased $O_2$ consumption                                                                                                                                                      | Rabbits | Schel$^5$ et al. |
| 41,000                 | 21.00         | 3 hours          | 50% mortality                                                                                                                                                                                              | Mice    | Mittler$^9$ et al. |
| 41,000                 | 21.80         | 3 hours          | 50% mortality                                                                                                                                                                                              | Mice    | Mittler$^9$ et al. |
| 67,980                 | 34.50         | 3 hours          | 50% mortality                                                                                                                                                                                              | Cats    | Mittler$^9$ et al. |
| 71,000                 | 36.00         | 3 hours          | 50% mortality                                                                                                                                                                                              | Rabbits | Mittler$^9$ et al. |
| 101,370                | 51.70         | 3 hours          | 50% mortality                                                                                                                                                                                              | Guinea pigs | Mittler$^9$ et al. |
| 1,650                  | 0.84          | 4 hours/5 days/2 wk | Increased susceptibility to *Klebsiella pneumoniae*                                                                                                                                                      | Mice, hamsters | Müller and Ehrlich$^{48}$ |

$^a$The concentrations of ozone listed are the lowest for which the observed effect has been recorded.
### Table 8-6. (continued).—SUMMARY OF THE EFFECTS OF OZONE IN ANIMALS

<table>
<thead>
<tr>
<th>Ozone, $^a$ $\mu g/m^3$</th>
<th>Ozone, $^a$ ppm</th>
<th>Length of exposure</th>
<th>Observed effect(s)</th>
<th>Species</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1,960</td>
<td>1.00</td>
<td>continuous</td>
<td>Bronchitis, bronchiolitis, emphysematous and fibrotic changes; acceleration of lung tumor development</td>
<td>Mice</td>
<td>Stokinger$^3$ et al.</td>
</tr>
<tr>
<td>15,700 to 88,000</td>
<td>8 to 45</td>
<td>1 hr/wk up to 49 wk</td>
<td>Damage to epithelium of the lower trachea and bronchioles; fibrosis</td>
<td>Rabbits</td>
<td>Scheel$^5$ et al.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Systemic effects</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Short-term exposures</td>
<td></td>
<td></td>
</tr>
<tr>
<td>390</td>
<td>0.20</td>
<td>30 min.</td>
<td>Increased sphering of red blood cells when irradiated</td>
<td>Rabbits, rats, mice</td>
<td>Brinkman$^{18}$ et al.</td>
</tr>
<tr>
<td>390</td>
<td>0.20</td>
<td>6 hours</td>
<td>Decreased voluntary running activity</td>
<td>Mice</td>
<td>Stokinger$^6$</td>
</tr>
<tr>
<td>1960</td>
<td>1.00</td>
<td>6 hours</td>
<td>60% increase in mortality as a result of exercise for 15 min/hr</td>
<td>Rats</td>
<td>Stokinger$^6$</td>
</tr>
<tr>
<td>6100</td>
<td>3.10</td>
<td>20 hours</td>
<td>Increased liver weight; increased liver alkaline phosphatase</td>
<td>Rats</td>
<td>Murphy$^{31}$ et al.</td>
</tr>
<tr>
<td>7800</td>
<td>4.00</td>
<td>4 hours</td>
<td>Decreased mortality with age: young 50% mortality, old 10% mortality</td>
<td>Mice</td>
<td>Stokinger$^6$</td>
</tr>
<tr>
<td>11800</td>
<td>6.00</td>
<td>4 hours</td>
<td>Decreased brain serotonin</td>
<td>Rats</td>
<td>Skillen$^{30}$ et al.</td>
</tr>
<tr>
<td>390</td>
<td>0.20</td>
<td>5 hr/day/3 wk</td>
<td>Structural changes in heart myocardial fibers</td>
<td>Mice</td>
<td>Stokinger$^6$</td>
</tr>
</tbody>
</table>

$^a$The concentrations of ozone listed are the lowest for which the observed effect has been recorded.

(3 to 6 ppm) ozone for periods of more than 4 hours.

6. Increased susceptibility to bacterial infection is produced by a 3-hour exposure to 160 $\mu g/m^3$ (0.08 ppm) ozone.

### 2. Human Data

Much of the data on effects of exposure to ozone in humans depend on measurement of pulmonary function. Definitions of the various measures of pulmonary function may be found in any one of a number of textbooks on pulmonary physiology, or in the *Air Quality Criteria for Particulate Matter*, NAPCA Publication No. AP-49.$^{5,4}$

#### a. Occupational Exposures to Ozone

References to human occupational exposures to ozone are numerous. Dadlez$^{5,5}$ in 1928, found that 2,940 $\mu g/m^3$ (1.5 ppm) ozone rendered the atmosphere intolerable. He cites an investigation by D'Arsonval who found that 780 $\mu g/m^3$ (0.4 ppm) ozone in the atmosphere produced symptoms of discomfort and irritation which were apparent in about 30 minutes. In 1931, Flury$^{5,6}$ gave the following symptoms characteristic of ozone exposure: 920 $\mu g/m^3$ (0.47 ppm) causes distinct irritation of mucous membranes, 1,840 $\mu g/m^3$ (0.94 ppm) causes coughing, irritation, and exhaustion within 1.5 hours; and 5,900 $\mu g/m^3$ (3 ppm) causes sleepiness in 1 hour. At higher concentrations, ozone causes increased pulse, sleepiness, and continued headache. It may also result in dyspnea (difficult breathing), pulmonary edema, and peribronchial complications.

More recently (1957), Kleinfeld$^{5,7}$ et al. have reported several cases of severe ozone intoxication in welders using a consumable electrode technique which was new at that
time. Three plants were investigated, and in all cases the ozone concentration was monitored at the breathing zone of the consumable electrode machine. In the first case, an ozone concentration of 490 \( \mu g/m^3 \) (0.25 ppm) was found. The workers had no complaints, and clinical findings were noncontributory. In the second case, the ozone concentration ranged from 590 to 1,570 \( \mu g/m^3 \) (0.3 to 0.8 ppm). Two out of four welders complained of chest constriction and throat irritation. Clinical examination disclosed no abnormalities. In the third case, the ozone concentrations were 17,990 \( \mu g/m^3 \) (9.2 ppm). A trichloroethylene degreaser was located about 50 feet feet from the welding area. Concentrations of nickel carbonyl and nitrogen oxides were negligible. Air tests for phosgene in the welding area were negative. The authors gave case histories of three workers, although others also complained of severe headaches, throat irritation, and lassitude. One of the cited patients developed severe dyspnea and substernal oppression and, on admission to a hospital, he was found to be in pulmonary edema. Chest x-rays showed diffuse peribronchial infiltration consistent with peribronchial pneumonia. The patient made a slow recovery over a 2-week period but still complained of fatigue and exertional dyspnea after 9 months. The two other workers had similar but less critical symptoms.

Challen et al. have performed a similar clinical and environmental survey. Ozone concentrations of 1,570 to 3,330 \( \mu g/m^3 \) (0.8 to 1.7 ppm) were found. Eleven out of 14 workers who were directly involved in welding complained of respiratory symptoms. No further symptoms were reported when ozone concentrations were reduced to 390 \( \mu g/m^3 \) (0.2 ppm). The results of this study, however, were complicated by the fact that concentrations of trichlorethylene up to 1,275 mg/m\(^3\) (238 ppm) were also present.

Young et al. have made the first study of pulmonary functional changes in workers exposed to ozone. Seven men engaged in argon-shielded electric arc welding, all smokers, were studied. The concentration of ozone in the welding shop was estimated by the rubber cracking technique and found to be 390 to 590 \( \mu g/m^3 \) (0.2 to 0.3 ppm). The following measurements of pulmonary function were made: vital capacity (VC), functional residual capacity (FRC), maximal mid inspiratory flow rate (MMFR), 0.75-second forced expiratory volume (FEV\(_{0.75}\)), and carbon monoxide diffusing capacity (DL\(_{CO}\)) at rest and at exercise. There was no convincing evidence that functional impairment develops in association with long-term exposure to 390 to 590 \( \mu g/m^3 \) (0.2 to 0.3 ppm) ozone in these seven smokers. Similar exposures and studies in non-smokers should be conducted.

The available data on occupational exposures of humans to ozone are summarized in Table 8-7.

b. Human Experimentation

Experiments on human subjects were first carried out by Jordan and Carlson in 1913. Three men were exposed to 9,800 to 19,600 \( \mu g/m^3 \) (5 to 10 ppm) ozone for a brief period. The subjects developed headache and drowsiness, one of them to the point of falling asleep. The authors suggested that the headache was due to irritation and hyperemia (abnormal congestion of blood) in the frontal sinuses. Similar findings were observed in dogs exposed to ozone.

Griswold et al. have reported changes in pulmonary function in a single subject exposed to 2,940 to 3,920 \( \mu g/m^3 \) (1.5 to 2 ppm) ozone for 2 hours. High temperatures complicated the exposure and may have influenced the results. At this concentration, ozone was associated with a lack of coordination and inability to express thoughts, plus respiratory symptoms such as chest pain and cough. Measurements of vital capacity were made before and immediately after exposure. No measurements of diffusing capacity were made. There was a slight (13 percent) reduction in vital capacity, which returned to normal after 22 hours. The forced expiratory
<table>
<thead>
<tr>
<th>Ozone, mg/m³</th>
<th>Ozone, ppm</th>
<th>Subjective complaints</th>
<th>Clinical findings attributed to ozone</th>
<th>Measurements of pulmonary function</th>
<th>Other comments</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>490</td>
<td>0.25</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td></td>
<td>Kleinfeld⁷⁷ et al.</td>
</tr>
<tr>
<td>590 to 1,570</td>
<td>0.3 to 0.8</td>
<td>Chest constriction and throat irritation in 2 to 4 subjects</td>
<td>None</td>
<td>None</td>
<td></td>
<td>Kleinfeld⁷⁷ et al.</td>
</tr>
<tr>
<td>17,990 (peak concentration)</td>
<td>9.2 (peak concentration)</td>
<td>Severe headaches, throat irritation, and lassitude in 7 or 8 subjects</td>
<td>None</td>
<td>None</td>
<td>Negligible nickel carbonyl and oxides of nitrogen. Trichloroethylene degreaser located 50 ft from welding area. Tests for phosgene negative.</td>
<td>Kleinfeld⁷⁷ et al.</td>
</tr>
<tr>
<td>390</td>
<td>0.2</td>
<td>–</td>
<td>None</td>
<td>None</td>
<td></td>
<td>Challen⁵⁸ et al.</td>
</tr>
<tr>
<td>1,570 to 3,330</td>
<td>0.8 to 1.7</td>
<td>Dry mouth and throat, irritation of nose and eyes, disagreeable smell in 11 of 14 subjects</td>
<td>None</td>
<td>None</td>
<td>Concentration of trichloroethylene up to 238 ppm found</td>
<td>Challen⁵⁸ et al.</td>
</tr>
<tr>
<td>390 to 590</td>
<td>0.2 to 0.3</td>
<td>Irritating odor, soreness of eyes, and dryness of mouth, throat, and trachea in 1 of 7 subjects</td>
<td>None</td>
<td>VC decreased in 3 of 7 subjects. FRC decreased in 2 of 7 subjects. DLCO decreased in 1 of 7 subjects.</td>
<td>All decreases in pulmonary function measurements were small. All subjects were smokers.</td>
<td>Young⁵⁹ et al.</td>
</tr>
<tr>
<td>780</td>
<td>0.4</td>
<td>Discomfort and irritation in about 30 minutes</td>
<td>None</td>
<td>None</td>
<td></td>
<td>Dadlez⁵⁵ (D’Arsonval)</td>
</tr>
<tr>
<td>920</td>
<td>0.47</td>
<td>Distinct irritation of mucous membranes</td>
<td>None</td>
<td>None</td>
<td></td>
<td>Flury⁵⁶</td>
</tr>
<tr>
<td>1,840</td>
<td>0.94</td>
<td>Coughing, irritation, and exhaustion, within 1-1/2 hours</td>
<td>None</td>
<td>None</td>
<td></td>
<td>Flury⁵⁶</td>
</tr>
<tr>
<td>5,900</td>
<td>3.0</td>
<td>Sleepiness within 1 hour</td>
<td>None</td>
<td>None</td>
<td></td>
<td>Flury⁵⁶</td>
</tr>
</tbody>
</table>
volume (FEV \textsubscript{3.0}) was reduced by 16.8 percent and was still slightly below normal after 22 hours. There was a slight reduction in the maximum breathing capacity. It is possible that the very slight reduction in vital capacity was due, in part, to the symptoms noted.

Bennett\textsuperscript{62} has exposed two groups of six volunteers to ozone for 3 hours a day, 6 days a week, for 12 weeks; one group at a concentration of 390 \(\mu g/m^3\) (0.2 ppm), the other at 980 \(\mu g/m^3\) (0.5 ppm). Ozone was produced by an ultraviolet ozone generator in which pure oxygen was used to prevent the simultaneous formation of nitrogen oxides. Measurements of vital capacity and forced expiratory volume (FEV \textsubscript{1.0}) were made. The group exposed to 390 \(\mu g/m^3\) (0.2 ppm) ozone experienced no symptoms, and their vital capacity and forced expiratory volume showed no significant change. The subjects experienced an average of 0.66 upper respiratory infections per person during the period

November through January, compared with an average of 0.95 per person suffered by a control group in a nearby laboratory. The group exposed to 980 \(\mu g/m^3\) (0.5 ppm) had no symptoms, although they could detect the ozone by smell. Their vital capacities showed a light downward trend, but the decreases were not significant. Their forced expiratory volume (FEV \textsubscript{1.0}) showed a significant decrease during the last few weeks of exposure (Figure 8-9). The results given in this paper represent only the average value for the entire group, and no information was given concerning the range of response among individuals. The conclusions of the author that airway-narrowing had taken place are quite plausible, since vital capacity was not altered.

Young\textsuperscript{63} et al. have made a detailed study of the effect of low concentrations of ozone on pulmonary function in man. These authors exposed 11 subjects, 10 men and 1 woman, aged 20 to 45 years, to 1,180 to 1,570 \(\mu g/m^3\)

![Graph](attachment:image_url)

**Figure 8-9.** Effect of exposure to 390 and 980 \(\mu g/m^3\) (0.2 and 0.5 ppm) ozone on six subjects.\textsuperscript{62}
(0.6 to 0.8 ppm) ozone for 2 hours. The ozone was generated by the action of two small ultraviolet bulbs on a stream of dry, filtered, air which was fed into the inspired air just before it reached the mixing box. Satisfactory evidence was obtained that the generator was not producing nitrogen oxides simultaneously. In this experiment, the subjects served as their own controls by breathing air instead of ozone for a period of 2 hours prior to exposure; in addition to the 16 ozone experiments, 11 experiments using air as a control were performed on the 11 subjects. Pulmonary function measurements were determined immediately before the experiments and at 24 and 48 hours after the completion of the breathing periods. In two subjects, mixing efficiency was determined by the helium dilution method, and the mechanical properties of the lungs were studied before and after inhalation of ozone. Ten of the 11 subjects experienced substernal soreness and tracheal irritation for 6 to 12 hours. There was a 25 percent reduction in the carbon monoxide diffusing capacity (DL\textsubscript{CO}) with inhalation of ozone for 2 hours (Table 8-8). There was also a 7 percent drop in the DL\textsubscript{CO} after breathing air for 2 hours. The drop was attributed to the progressive shift in perfusion distribution in the lung, which had occurred as a result of rest in the upright position. The results of individual experiments, however, showed that change in the DL\textsubscript{CO} after breathing air was scattered in a random manner, whereas a decrease in the DL\textsubscript{CO} following ozone exposure was noted in every one of the 16 experiments. The results of ventilatory tests carried out in 10 of the 11 subjects are shown in Table 8-9. Vital capacity and the forced expiratory volume (FEV\textsubscript{0.75}) were unaffected by 2 hours of air breathing but fell about 10 percent after breathing ozone. This difference was not statistically significant. The maximal mid-expiratory flow rate (MMFR) fell about 15 percent, but this change was not statistically significant. The authors point out that one subject had such large decreases in all of these measurements

<table>
<thead>
<tr>
<th></th>
<th>(\text{DL}_{\text{CO}})</th>
<th>Standard deviation</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control exposure</td>
<td>20.7</td>
<td>5.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exposure to air</td>
<td>19.3</td>
<td>4.40</td>
<td></td>
<td></td>
</tr>
<tr>
<td>for 2 hours</td>
<td>-1.4</td>
<td>0.822</td>
<td>1.64</td>
<td>0.2 to 0.1</td>
</tr>
<tr>
<td>Change ((\Delta) air)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control exposure</td>
<td>22.5</td>
<td>3.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exposure to ozone for 2 hours</td>
<td>17.1</td>
<td>2.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change ((\Delta) ozone)</td>
<td>-5.4</td>
<td>0.84</td>
<td>6.40</td>
<td>0.001</td>
</tr>
<tr>
<td>Difference in effect of exposures ((\Delta) ozone - (\Delta) air)</td>
<td>-4.0</td>
<td>3.84</td>
<td>3.31</td>
<td>0.01 to 0.001</td>
</tr>
</tbody>
</table>
that his changes were responsible for almost half of the effect shown by the whole group. The mixing efficiency and dynamic compliance of the lung remained normal after ozone exposure. The airway resistance was slightly greater after ozone than before, however, in each of the two subjects measured, but remained within normal limits. It was concluded that the lowering of the diffusing capacity was due to a thickening of the alveolar wall. It was calculated that an increase of 50 percent in the thickness of the alveolar capillary membrane was necessary to produce the change observed; this could be achieved by only 13 ml of edema fluid. The authors suggested that injury might be confined to the lower regions of the lungs, since they are known to receive a higher portion of the ventilation than the upper regions. Although it was not proved, the speed of recovery indicated that edema rather than inflammation was responsible for such changes. The small reduction in vital capacity could be due to a physical limitation.

Hallett exposed ten subjects to 1,960 to 7,800 μg/m³ (1 to 4 ppm) ozone for periods of up to 30 minutes. Measurements of vital capacity, 1- and 3-second forced expiratory volumes, and maximal breathing capacities were made with a Collins spirometer. Maximum expiratory flow rate was measured using a Wright peak flowmeter, and functional residual capacity was determined using the closed circuit helium technique. Carbon monoxide diffusing capacity was determined by the steady-state method. Ozone was generated from pure oxygen and was monitored by a Mast ozone meter but, due to a calibration problem, there was some uncertainty about the actual exposure. Results are given for 11 subjects, 6 of whom could not tolerate the dose for a full 30 minutes. All had symptoms after 10 minutes. The symptoms usually lasted for more than an hour and, in some, for several hours, especially headache and shortness of breath. Measurements of pulmonary function are confusing, since some showed increases and some showed decreases. Measurements of vital capacity showed four out of eight subjects exhibiting decreases of greater than 10 percent; five out of eight subjects showing a decrease in the forced expiratory volume (FEV₁₋₀) of greater than 10 percent; five out of six subjects showing a decrease in maximum expiratory flow rate; five out of eight subjects showing a decrease in maximal breathing capacity; and seven out of eleven subjects showing a decrease in the carbon monoxide diffusing capacity, the smallest decrease being 20 percent. Since no measurements were made of the mechanical properties of the lungs, it is difficult to relate these changes to any one factor. It is possible that both mechanical changes and irritative symptoms contributed to the changes.

Goldsmith et al. have attempted to demonstrate changes in airway resistance following exposure to ozone. Four subjects were exposed to 200, 780, 1,180, and 1,960 μg/m³ (0.1, 0.4, 0.6, and 1.0 ppm) ozone for periods of 1 hour. During exposure, simple pulmonary function tests such as Wright peak flowmeter and pufmeter (used to estimate MMFR) were used to detect any rapid untoward reaction, but none was observed. Airway

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**Table 8.9. EFFECT OF INHALING EITHER AIR OR 1,180 to 1,570 μg/m³ (0.6 to 0.8 ppm) OZONE FOR 2 HOURS ON VITAL CAPACITY (VC), INDIRECT MAXIMAL BREATHING CAPACITY (FEV₀.₇₅ x 40), AND MAXIMUM MID-EXPIRATORY FLOW RATE (MMFR), ON 10 MALE SUBJECTS**

<table>
<thead>
<tr>
<th>INHALANT</th>
<th>VC, liters</th>
<th>FEV₀.₇₅ x 40, liters/min.</th>
<th>MMFR, liters/sec</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before</td>
<td>4.40</td>
<td>135</td>
<td>3.80</td>
</tr>
<tr>
<td>After</td>
<td>4.44</td>
<td>136</td>
<td>3.94</td>
</tr>
<tr>
<td>Change (Δ air)</td>
<td>+ 0.04</td>
<td>+1</td>
<td>+0.14</td>
</tr>
<tr>
<td>P</td>
<td>0.3-0.2</td>
<td>0.7-0.6</td>
<td>0.6-0.5</td>
</tr>
<tr>
<td>Ozone</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before</td>
<td>4.33</td>
<td>131</td>
<td>3.60</td>
</tr>
<tr>
<td>After</td>
<td>4.06</td>
<td>118</td>
<td>3.08</td>
</tr>
<tr>
<td>Change (Δ ozone)</td>
<td>-0.37</td>
<td>-13</td>
<td>-0.52</td>
</tr>
<tr>
<td>P</td>
<td>0.10-0.05</td>
<td>0.05-0.02</td>
<td>0.05-0.02</td>
</tr>
<tr>
<td>p (Δ ozone - Δ air)</td>
<td>0.05-0.02</td>
<td>0.05-0.02</td>
<td>0.10-0.05</td>
</tr>
</tbody>
</table>
resistance was measured in triplicate, using the body plethysmograph technique before, immediately, 1 hour, and 24 hours after exposure; these data are shown in Figure 8-10. The subject who had the highest pre-exposure airway resistance showed a 45 percent increase after exposure to 200 μg/m³ (0.1 ppm), and had slight hemoptysis (expectoration of blood). The pooled data show increased airway resistance with increasing concentrations which became significant at 1,960 μg/m³ (1.0 ppm). To pool the data, all of each subject’s tests were averaged and the relative value, a percent of this average, was calculated.

c. Summary of Human Exposure to Ozone

Prolonged exposure of humans to ozone, under occupational and experimental conditions, has produced the following effects:

1. No apparent effects were observed at

![Graph showing the effect of ozone on airway resistance.](image-url)

Figure 8-10. Effect of ozone on airway resistance.⁶⁵
concentrations up to 390 mg/m³ (0.2 ppm).
2. The threshold level at which nasal and throat irritation will result appears to be about 590 mg/m³ (0.3 ppm).
3. Concentrations of 980 mg/m³ (0.5 ppm) have caused a 20 percent decrease in forced expiratory volume, observed after 8 weeks of intermittent exposure (3 hours a day, 6 days a week); this change has returned to normal during a post-exposure period of 6 weeks.

Experimental appraisal of short-term exposures to ozone show the following results (Table 8-10):
1. Up to 200 mg/m³ (0.1 ppm) for 1 hour is apparently without significant effect.
2. Concentrations of 200 to 780 mg/m³ (0.1 to 0.4 ppm) for 1 hour have not been shown to produce effects, but there is a lack of information for this concentration range.
3. Exposure to concentrations of 980 to 1,960 mg/m³ (0.5 to 1.0 ppm) for periods of 1 to 2 hours produces changes in pulmonary function. These are: increased airway resistance, decreased vital capacity, decreased carbon monoxide diffusing capacity, and decreased forced expiratory volume.
4. Some people are unable to tolerate concentrations of 1,960 to 5,900 mg/m³ (1.0 to 3.0 ppm) over a period of about 2 hours. Extreme fatigue and lack of coordination are experienced.
5. Concentrations of about 17,600 mg/m³ (9.0 ppm) produce severe pulmonary edema and possible acute bronchiolitis.

C. OXIDANTS

In addition to the experimental exposure of both animals and humans to ozone, considerable work has been performed to evaluate the effects of exposure to “oxidants.” These exposures have usually involved photochemical smog obtained from either ambient air or the synthetic irradiation of automobile exhaust, and thus ozone has generally been one of the substances present in these “oxidant mixtures.”

1. Animal Data

a. Direct Effects of Photochemical Oxidants

(1) Effects on pulmonary tissue.

Changes in pulmonary tissue.

Studies carried out to show the effect of both ambient air and high concentrations of auto exhaust on pulmonary function in laboratory animals have indicated that some increase in lung flow resistance occurs as a result of exposure to oxidants.

Swann⁶⁶,⁶⁷ et al. have studied the effect of ambient air on the flow resistance of guinea pigs. The first study took place over a period of 2 years. Guinea pigs were located at three stations within the Los Angeles area. At each station, one group was exposed to filtered air and a second group exposed to ambient air. Measurement of expiratory flow resistance (mostly airway resistance) at monthly intervals, using the forced pressure oscillation technique, showed no essential difference between the two groups in each station.

These data provided a basis for further study of guinea pigs exposed to ambient air when the oxidant level was above 40 percent of the alert, or greater than 390 mg/m³ (0.2 ppm). It was observed that some ambient air breathers could show a significant increase in resistance on the days during which oxidant levels reached 980 mg/m³ (0.5 ppm) or higher. The animals exposed to charcoal filtered air, however, also experienced this increase in resistance, although to a lesser extent. The ambient-air-breathing animals also showed an increase in resistance on days when the oxidant levels were considerably lower, 590 mg/m³ (0.30 ppm). It is possible that other components of ambient air, such as hydrocarbons and oxides of nitrogen, may also have been elevated on the days when resistance was elevated. The data provided in this paper are inadequate to permit analysis of the relative importance of these various pollutants on respiratory resistance, although cold, wet days
<table>
<thead>
<tr>
<th>Concentration</th>
<th>Duration</th>
<th>Subjects</th>
<th>Symptoms</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>up to 7,800</td>
<td>10-30 min</td>
<td>11</td>
<td>Headache, shortness of breath, lasting more than 1 hour</td>
<td>VC: mean decrease 16.5% (4/8 subjects showed decrease &gt;10%); FEV1: mean decrease 20% (5/8 subjects showed decrease &gt;10%); MMEF: mean decrease 10.5% (5/6 subjects showed a decrease); MBC: mean decrease 12% (5/8 subjects showed a decrease); DLCO decreased 20-50% in 7/11 subjects, increased 10-50% in 4/11 subjects. Only 5/11 tolerated dose for full 30 mins. Wide variation in DLCO.</td>
</tr>
<tr>
<td>200</td>
<td>1 hour</td>
<td>4 male</td>
<td></td>
<td>Airway resistance: mean increase 3.3% at 0 hours after exposure (1/4 subjects showed an increase of 45%).</td>
</tr>
<tr>
<td>780</td>
<td>1 hour</td>
<td>4 male</td>
<td>Odor</td>
<td>Airway resistance: mean increase 3.5% at 0 hours after exposure (1/4 subjects showed an increase of 60%), mean increase 12.6% 1 hour after exposure;</td>
</tr>
<tr>
<td>1,180</td>
<td>1 hour</td>
<td>4 male</td>
<td>Odor</td>
<td>Airway resistance: mean increase 5.8% at 0 hours after exposure (1/4 subjects showed an increase of 75%), mean increase 5% 1 hour after exposure;</td>
</tr>
<tr>
<td>1,960</td>
<td>1 hour</td>
<td>4 male</td>
<td>Throat irritation and cough</td>
<td>Airway resistance: mean increase 19.3% at 0 hours after exposure (3/4 subjects showed an increase of &gt;20%); mean increase 5% 1 hour after exposure.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>One subject had history of asthma, and experienced hemoptysis 2 days after 1 ppm.</td>
</tr>
</tbody>
</table>

Goldsmith and Nadel
### Table 8-10. SUMMARY OF DATA ON HUMAN EXPERIMENTAL EXPOSURE TO OZONE

<table>
<thead>
<tr>
<th>Ozone, $\mu g/m^3$</th>
<th>Ozone, ppm</th>
<th>Length of exposure</th>
<th>No. of subjects</th>
<th>Subjective complaints</th>
<th>Measurements of pulmonary function</th>
<th>Other comments</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>9,800 to 19,600</td>
<td>5 to 10</td>
<td>Not available</td>
<td>3 male</td>
<td>Drowsiness, headache</td>
<td>None</td>
<td>Measurement of $O_3$ probably inaccurate</td>
<td>Jordan et al.</td>
</tr>
<tr>
<td>2.940 to 3.920</td>
<td>1.5 to 2</td>
<td>2 hours</td>
<td>1 male</td>
<td>C.N.S. depression, lack of coordination, chest pain, cough for 2 days, tiredness for 2 weeks</td>
<td>VC: decreased 13%, returned to normal in 22 hours, FEV$_{1.0}$ decreased 16.8%, slightly below normal after 22 hours, MBC: decreased very slightly.</td>
<td>—</td>
<td>Gnswoot et al.</td>
</tr>
<tr>
<td>390</td>
<td>0.2</td>
<td>3 hr/day, 6 days/wk for 12 wk</td>
<td>6 male</td>
<td>None</td>
<td>VC: no change FEV$_{1.0}$ no change</td>
<td>0.66 upper respiratory infections/person in 12 weeks - CF control group had 0.95 in the same period</td>
<td>Bennett et al.</td>
</tr>
<tr>
<td>980</td>
<td>0.5</td>
<td>3 hr/day, 6 days/wk for 12 wk</td>
<td>6 male</td>
<td>No irritating symptoms but could detect ozone by smell</td>
<td>VC: slight decrease but not significant; FEV$_{1.0}$ significant decrease toward end of 12 weeks. Returned to normal during 6 weeks after exposure.</td>
<td>0.80 upper respiratory infections/person in 12 weeks</td>
<td>—</td>
</tr>
<tr>
<td>1,180 to 1,570</td>
<td>0.6-0.8</td>
<td>2 hours</td>
<td>11 male, 1 female</td>
<td>Subternal soreness and tracheal irritation 6-12 hours after exposure, disappearing within 12-24 hours in 10/11 subjects</td>
<td>DLCO: mean decrease of 25% (11/11 subjects), VC: mean decrease of 10% which was significant (10/10 subjects); FEV$_{0.5}$ x 40: mean decrease of 10% which was significant (10/10 subjects); MMFR: mean decrease of 15% which was not significant; Mixing efficiency: no change (2/2 subjects); Airway resistance: slight increase but within normal limits; Dynamic compliance: no change (2/2 subjects).</td>
<td>—</td>
<td>Young et al.</td>
</tr>
</tbody>
</table>

827
would appear to be associated with an increase in resistance. Murphy et al. exposed guinea pigs to irradiated and nonirradiated auto exhaust for periods of 4 hours. Intact unanesthetized guinea pigs were exposed through face masks attached to an exposure manifold, and were placed in body plethysmographs. Before, during, and after exposure, measurements were made of tidal volume, respiratory rate, and total respiratory (lung and chest wall) flow resistance, as described by Mead. The results of these measurements are shown in Figures 8-11 and 8-12. Chemical differences were noted between the two forms of auto exhaust. Comparison of concentrations in irradiated and nonirradiated atmospheres of approximately equal dilution ratios show the photochemical formation of aldehydes, nitrogen dioxide, and total oxidant at the expense of nitric oxide and olefin (Table 8-11). Marked increases in total expiratory flow resistance occurred rapidly during exposure to irradiated exhaust. This was accompanied by a decrease in respiratory rate and a small increase in tidal volume. These functions rapidly returned to baseline levels following removal of the animals to a clean-air atmosphere. The response to nonirradiated exhaust was relatively small, and when it occurred could possibly have been due to carbon monoxide. In this study, the increase in flow resistance was associated with a decrease in frequency of breathing and an increase in tidal volume. In this respect, the response was similar to that of guinea pigs exposed to sulfur dioxide and different from guinea pigs exposed to ozone or to nitrogen dioxide. The increase in flow resistance noted in this experiment occurred at low oxidant levels compared to the concentration of ozone (1,960 μg/m³ or 1 ppm) required to produce changes in flow resistance. The data suggest that components of the exhaust other than oxidants play a role. It must be pointed out that changes in resistance were of only borderline significance when concentrations of auto exhaust comparable to those found in ambient air were used (I-1142 in Figure 8-12; experiments H and I in Table 8-11).

Pathological changes.

Bils and Bils and Romanovsky have reported ultrastructural alterations of the alveolar tissue of mice exposed to heavy Los Angeles smog containing 780 μg/m³ (0.4 ppm) total oxidants and to synthetic photochemical smog prepared by irradiating 14,000 μg/m³ (8 ppm) propylene, and 3,440 μg/m³ (2.8 ppm) NO to yield 1,470 to 980 μg/m³

Table 8-11. CHEMICAL AGENTS IN EXHAUST-CONTAMINATED ATMOSPHERES

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Dil. ratio-air/ex.</th>
<th>CO²</th>
<th>Total oxidant</th>
<th>NO₂</th>
<th>NO</th>
<th>Formald.</th>
<th>Acrolein</th>
<th>Olefin</th>
<th>HC/NOₓ ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Irr.</td>
<td>150</td>
<td>290</td>
<td>0.78</td>
<td>5.50</td>
<td>1.00</td>
<td>1.93</td>
<td>0.17b</td>
<td>8.90b</td>
<td>4.0</td>
</tr>
<tr>
<td>B. Irr.</td>
<td>155</td>
<td>310</td>
<td>0.80</td>
<td>2.66</td>
<td>0.21</td>
<td>2.42</td>
<td>0.20b</td>
<td>12.90</td>
<td>3.5</td>
</tr>
<tr>
<td>C. Nonirr.</td>
<td>160</td>
<td>300</td>
<td>0.02</td>
<td>1.58</td>
<td>4.27</td>
<td>0.12</td>
<td>0.07</td>
<td>17.80</td>
<td>2.9</td>
</tr>
<tr>
<td>D. Irr.</td>
<td>360</td>
<td>86</td>
<td>0.82</td>
<td>1.64</td>
<td>0.16</td>
<td>1.11</td>
<td>0.11</td>
<td>3.20</td>
<td>2.5</td>
</tr>
<tr>
<td>E. Irr.</td>
<td>330</td>
<td>95</td>
<td>0.57</td>
<td>2.23</td>
<td>0.20</td>
<td>1.02</td>
<td>0.09</td>
<td>1.60</td>
<td>2.5</td>
</tr>
<tr>
<td>F. Irr.</td>
<td>407</td>
<td>85</td>
<td>0.45</td>
<td>2.95</td>
<td>0.59</td>
<td>1.39</td>
<td>0.10</td>
<td>1.53</td>
<td>2.1</td>
</tr>
<tr>
<td>G. Nonirr.</td>
<td>375</td>
<td>85</td>
<td>0.00</td>
<td>0.38</td>
<td>2.58</td>
<td>0.38</td>
<td>0.02</td>
<td>5.37</td>
<td>2.6</td>
</tr>
<tr>
<td>H. Irr.</td>
<td>1,350</td>
<td>34</td>
<td>0.35</td>
<td>0.49b</td>
<td>0.25b</td>
<td>0.54b</td>
<td>0.57b</td>
<td>1.22b</td>
<td>2.4</td>
</tr>
<tr>
<td>I. Irr.</td>
<td>935</td>
<td>47</td>
<td>0.33</td>
<td>0.43</td>
<td>0.17b</td>
<td>0.39b</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

aAll concentrations in ppm except olefins in μg per liter.
bIndicates analysis at manifold was not obtained and figure refers to a representative analysis at the irradiation chamber.

Carbon monoxide concentration calculated from raw exhaust/dilution ratio.
Figure 8-11. Respiratory response of guinea pigs breathing auto exhaust.
(0.75 to 0.5 ppm) total oxidants. Exposure to natural smog was for 2 to 3 hours, whereas exposure to synthetic smog was for 3 hours. The data from these experiments are summarized in Tables 8-12 and 8-13. The pattern of the ultrastructural changes appeared to be the same for both natural and synthetic smog. The severity of the changes appeared to increase with increase in age, with irreversible changes (loss of wall cells) appearing at about age 15 months. The lining epithelium and basement membrane seemed to withstand smog well even though the endothelium was seriously affected. The edema-like condition was only apparent in the lining epithelium when the mice reached 20 to 21 months.

In addition to these studies, Rounds and Bils\textsuperscript{72} treated cultures of alveolar wall cells from rats and rabbits in vitro with NaNO\textsubscript{2}; it was assumed that NaNO\textsubscript{2} simulated the introduction of nitrogen dioxide to the cell. A reversible inhibition of respiratory activity of the cells was observed. Electron microscopy of the living cells and of fixed material during NaNO\textsubscript{2} treatment showed that there were changes in the shape of the nucleus and ultrastructure of the mitochondria of the alveolar wall cell. These changes occurred as a

Figure 8-12. Effect of auto exhaust on expiratory flow resistance of guinea pigs.\textsuperscript{68}
Table 8-12. ULTRASTRUCTURAL ALTERATIONS IN ALVEOLAR TISSUE OF MICE AFTER EXPOSURE TO MORE THAN 780 µg/m³ (0.4 ppm) OXIDANT FOR 2 TO 3 HOURS⁷⁰

<table>
<thead>
<tr>
<th>Age</th>
<th>Time of investigation</th>
<th>Cellular effects and pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 months</td>
<td>During and after exposure.</td>
<td>No apparent change.</td>
</tr>
<tr>
<td>9 months</td>
<td>During exposure.</td>
<td>Few normal mitochondria in epithelial cell walls. Disrupted cytoplasm containing lamellar inclusions or fragments. Some epithelial and endothelial swelling.</td>
</tr>
<tr>
<td>9 months</td>
<td>14 hours after exposure</td>
<td>Marked recovery. Difficult to distinguish between experimental and control animals.</td>
</tr>
<tr>
<td>21 months</td>
<td>During exposure.</td>
<td>Similar to 9-month-old mice, but cytoplasmic fragments and proteinaceous material were present in alveoli. Practically no epithelial wall cells present.</td>
</tr>
</tbody>
</table>

Table 8-13. ULTRASTRUCTURAL ALTERATIONS IN ALVEOLAR TISSUE OF MICE AFTER 3–HOUR EXPOSURE TO FROM 980 TO 1,470 µg/m³ (0.50 TO 0.75 ppm) OXIDANT IN SYNTHETIC PHOTOCHEMICAL SMOG

<table>
<thead>
<tr>
<th>Age</th>
<th>Time of investigation</th>
<th>Cellular effects and pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>6 months</td>
<td>Immediately after exposure</td>
<td>Difficult to distinguish treated from control tissues. Possibly an increase in breakage of the endothelial membrane in treated animals.</td>
</tr>
<tr>
<td>8 months</td>
<td>Immediately after exposure</td>
<td>Wall cells contained fewer normal mitochondria. These cells were vacuolated with many lamellar inclusions, possibly indicating increased mitochondrial transformation. Slight epithelial and endothelial swelling.</td>
</tr>
<tr>
<td></td>
<td>18 hours after exposure</td>
<td>Alveolar tissue recovered so that it was difficult to distinguish treated from controls.</td>
</tr>
<tr>
<td>15 months</td>
<td>Immediately after exposure</td>
<td>Swelling and disruption of both epithelial and endothelial lining of alveolar cell. Basement membrane still intact. Increase in number of alveolar phagocytes. Observed decrease in wall cells, seen also in controls and therefore attributed to aging.</td>
</tr>
<tr>
<td></td>
<td>6 to 8 hours after exposure</td>
<td>Disrupted wall cell cytoplasm but some normal wall cells remained. Large alveolar phagocytes, some in almost complete contact with epithelial lining. Serious disorganization of the connective tissue in some localized interstitial areas.</td>
</tr>
<tr>
<td></td>
<td>24 hours after exposure</td>
<td>Most wall cells seemed to be lost. Some epithelial and endothelial lining cells remained intact. Integrity of basement membrane maintained. Rupture of the lining membranes was apparent.</td>
</tr>
<tr>
<td>20 months</td>
<td>Immediately after exposure</td>
<td>Cytoplasm of large alveolar cell walls quite disorganized. Most of the surrounding basement membranes, connective tissue, and lining membranes also severely altered. Disruption of both epithelial and endothelial lining membranes. Most alveolar phagocytes appeared normal.</td>
</tr>
<tr>
<td></td>
<td>18 hours after exposure</td>
<td>Exaggerated swelling of cell lining. Both epithelial and endothelial blebs contained substances that could have produced an edematous condition. Remains of degenerating wall cells present. Remaining wall cells contained same type of dense membranous material as that seen in alveoli.</td>
</tr>
</tbody>
</table>
result of both a single exposure to 37,600 µg/m³ (20 ppm) nitrogen dioxide for 1 hour and chronic exposure to 28,200 µg/m³ (15 ppm) nitrogen dioxide for 4 hours a day for 10 days. Increasing the dose of nitrogen dioxide to 150 µg/m³ (80 ppm) for 55 minutes produced more drastic and irreversible lamellar transformation of the wall cell mitochondria.

**Development of lung tumors.**

Certain studies have reported that aging experimental mice breathing air with relatively high levels of photochemical oxidant pollution show increased incidence of benign pulmonary adenomas. Gardner⁷³ has exposed several strains of mice at four exposure stations in the Los Angeles area. Monthly average “oxidant” concentration varied from 40 to 140 µg/m³ (0.02 to 0.07 ppm), and daily 24-hour maxima averaged from 140 to 490 µg/m³ (0.07 to 0.25 ppm). The results indicated a trend toward an increased incidence of pulmonary adenomas in aging experimental mice breathing ambient air when compared to the incidence in control animals who breathed filtered air.

In an attempt to confirm this observed trend, Gardner⁷⁴ et al. conducted an additional study utilizing rats exposed to either ambient or filtered Los Angeles air. The results of this second study indicated that there was an increased incidence of chronic nephritis in male rats exposed to ambient air, although no statistically significant differences concerning the incidence of lung tumors were noted between the mice exposed to the ambient atmosphere and their respective controls.

Catcott and Kotin⁷⁵ did not find an increase in lung tumors in dogs from polluted areas of Los Angeles, although this species seems to be resistant to pulmonary lesions for anatomical reasons.

Kotin⁷⁶,⁷⁷ et al. exposed A-strain and C₅⁷ black mice to 1,960 to 7,470 µg/m³ (1.0 to 3.8 ppm) ozonized gasoline. The results are shown in Table 8-14. These data show an association between exposure to ozonized gasoline and an increase in lung tumor incidence.

Kotin and Falk⁷⁸ have pointed out that the physical state of natural carcinogenic hydrocarbons is one of adsorption on soot particles in a size range compatible with deposition and retention of particles. They have also noted that atmospheric irritants, including photochemical oxidants, cause respiratory epithelial changes which facilitate deposition and retention of particles.

It is often implied that an increase in the frequency of pulmonary adenoma is equivalent to a demonstration that the exposure in question had produced or enhanced a carcinogenic effect. True carcinomas in C₅⁷ black mice have been produced by the combined exposure to ozonized gasoline and to influenza virus,⁷⁹,⁸⁰ and it has recently been

<table>
<thead>
<tr>
<th>Group</th>
<th>Strain</th>
<th>Week of first tumor appearance</th>
<th>Number of mice surveyed</th>
<th>Number of tumor-bearing mice</th>
<th>Percent tumor-bearing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Washed air</td>
<td>C₅⁷</td>
<td>56</td>
<td>376</td>
<td>6</td>
<td>1.6</td>
</tr>
<tr>
<td>Ozonized gasoline</td>
<td>C₅⁷</td>
<td>71</td>
<td>155</td>
<td>15</td>
<td>9.6</td>
</tr>
<tr>
<td>Washed air</td>
<td>A-strain</td>
<td>28</td>
<td>45</td>
<td>11</td>
<td>24.0</td>
</tr>
<tr>
<td>Ozonized gasoline</td>
<td>A-strain</td>
<td>24</td>
<td>15</td>
<td>6</td>
<td>40.0</td>
</tr>
</tbody>
</table>
reported that experimental animals preexposed to sulfur dioxide and then exposed to inhaled polynuclear hydrocarbons developed a true metastasizing bronchial carcinoma.\textsuperscript{81} Other combinations, such as polynuclear hydrocarbons with soot and with hematite, have been reported to produce experimental lung cancer in animals. No true lung cancers have been reported, however, from experimental exposures to either ozone alone or any other combination or ingredient of photochemical oxidants.

(2) Systemic effects.

Changes in fertility and neonatal mortality

Huetter et al.,\textsuperscript{37} in studying the effect of irradiated auto exhaust on mice, observed decreased fertility and survival rate of offspring in those mice exposed to irradiated auto exhaust containing ozone in concentrations of 1,180 to 1,960 μg/m\textsuperscript{3} (0.6 to 1.0 ppm); other pollutants present in the exhaust were carbon monoxide (70 to 115 mg/m\textsuperscript{3}, or 60 to 100 ppm), hydrocarbons (20 to 36 ppm), and nitrogen oxides (2.9 to 3.9 ppm).\textsuperscript{37} Similar results have been obtained by Lewis\textsuperscript{82} et al., who studied the effect of irradiated auto exhaust on the reproduction of mice. One hundred-fifty virgin females were preconditioned to either filtered air of irradiated auto exhaust for a period of 16 hours daily for 46 days. Estimates of total oxidant levels ranged from 200 to 980 μg/m\textsuperscript{3} (0.1 to 0.5 ppm) in the first experiment and 590 to 1,960 μg/m\textsuperscript{3} (0.3 to 1.0 ppm) in the second experiment; carbon monoxide, hydrocarbons, and nitrogen oxides were also present. At 12 to 13 weeks, the mice were permitted to mate with randomly paired, similarly preconditioned males for an 11-day mating period. Results of the first experiment indicated that preconditioning of males with irradiated auto exhaust doubled the non-pregnancy average of their mates. It was determined, moreover, that the reproductive tracts and gonads of the nonpregnant females and their respective mates were normal.

In the second experiment, it was found that preconditioning of males to irradiated auto exhaust resulted in an increased mortality in neonatal mice. The authors suggest that air pollutants in irradiated auto exhaust may alter the genetic composition and possibly other cellular components of sperm.

Stress response.

Exposure to photochemical oxidants has produced stress responses in animals. It is well known that an increased urinary 17-ketogenic steroid output is found in response to a wide variety of stresses. Harvey\textsuperscript{83} et al. subjected guinea pigs to psychological and physiological stresses and measured their elevated 17-ketogenic steroid output by Selye's general alarm reaction. The guinea pigs were then exposed to a Los Angeles photochemical smog. At the end of the stress period, the levels rapidly returned to normal. The subsequent exposure to smog increased the 17-ketogenic steroid output, but the differences were small compared to the control group. It was suggested by the authors that smog stress induces the general alarm reaction.

Huetter\textsuperscript{37} et al. reported a stress adaption response in mice exposed over a period of 2 years to irradiated auto exhaust containing a concentration of ozone of 80 to 390 μg/m\textsuperscript{3} (0.04 to 0.2 ppm). Their indices to stress adaptation included a reduction in the spontaneous activity of the mice, followed by a return to pre-exposure activity level.

b. Indirect Effects of Photochemical Oxidants

Altered response to other agents.

Exposure to photochemical oxidants has been shown to lower the resistance of animals to certain other deleterious agents. Levels of laboratory-synthesized smog, with oxidant levels well below the peak concentrations found in heavily polluted areas, have enhanced mortality from streptococcal pneumonia.

Murphy\textsuperscript{68} et al. have studied the combined effects of the edemagenic agent α-naphthylthiourea (ANTU) with both irradiated and nonirradiated auto exhaust. These data are shown in Table 8-15. Except for one experi-
ment (G), there was a consistently higher mortality rate due to ANTU in the exhaust-exposed group. Although the mortality differences between the two groups were small, they were statistically significant (p < 0.02). The authors concluded that increased mortality was due to increased stress and not due to the lethality of the exhaust itself.

Coffin and Blommer⁸⁴ have studied the susceptibility of laboratory animals to bacterial infection following exposure to auto exhaust. Mice were exposed to auto exhaust containing 13,800 to 115,000 μg/m³ (12 to 100 ppm) carbon monoxide and 160 to 1,310 μg/m³ (0.08 to 0.67 ppm) oxidant for four hours. Clean air was used in control experiments. Immediately after exposure, the animals were subjected to a bacterial aerosol of streptococcus (Group C) introduced at a rate of 100,000 organisms per mouse. It was observed that exposure to exhaust containing 115,000 μg/m³ (100 ppm) carbon monoxide and an oxidant range of 690 to 1,310 μg/m³ (0.35 to 0.67 ppm) caused enhanced mortality from streptococcal pneumonia. Mortality was 53 percent for those pretreated with exhaust and 11 percent for the controls. A “no effect” level was reached at 28,750 μg/m³ (25 ppm) carbon monoxide and 240 μg/m³ (0.12 ppm) oxidant. It was concluded that laboratory-synthesized photochemical smog can enhance mortality to streptococcal pneumonia at levels of carbon monoxide and oxidant which are well below the peak concentration in heavily polluted air. In view of the fact that ozone at 160 μg/m³ (0.08 ppm) and above has been shown to enhance mortality from respiratory infection,⁴⁰ it would appear reasonable to ascribe the enhancement reported in this experiment to the oxidant content of the auto smog.

c. Summary

Several general statements can be made regarding the experimental exposure of laboratory animals to mixtures of photochemical oxidants.

1. Long-term exposure to ambient air in Los Angeles produces an increase in flow resistance in guinea pigs during peak oxidant periods when the oxidant level exceeds 980 μg/m³ (0.5 ppm).

2. Long-term exposure to irradiated auto exhaust with oxidant levels from 390 to 1,960 μg/m³ (0.2 to 1.0 ppm) produces a decrease in fertility, an increase in neonatal mortality, and a stress adaptation response in mice.

3. During short-term exposure to irradiated auto exhaust containing up to 1,570 μg/m³ (0.8 ppm) oxidant for periods of up to 6 hours, the following changes in pulmonary function are observed in guinea pigs: increased tidal volume, increased minute volume, and increased flow resistance. These changes return to normal

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Dilution ratio, air/exhaust</th>
<th>Number treated per group</th>
<th>0 hr post exposure</th>
<th>3 hr post exposure</th>
<th>24 hr post exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Air</td>
<td>Exhaust</td>
<td>Air</td>
<td>Exhaust</td>
<td>Air</td>
</tr>
<tr>
<td>A. Irr.</td>
<td>150</td>
<td>10</td>
<td>0</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td>B. Irr.</td>
<td>155</td>
<td>15</td>
<td>27</td>
<td>47</td>
<td>27</td>
</tr>
<tr>
<td>C. Nonirr.</td>
<td>160</td>
<td>15</td>
<td>13</td>
<td>40</td>
<td>13</td>
</tr>
<tr>
<td>D. Irr.</td>
<td>360</td>
<td>20</td>
<td>0</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>E. Irr.</td>
<td>330</td>
<td>15</td>
<td>0</td>
<td>6.7</td>
<td>0</td>
</tr>
<tr>
<td>F. Irr.</td>
<td>407</td>
<td>15</td>
<td>27</td>
<td>53</td>
<td>40</td>
</tr>
<tr>
<td>G. Nonirr.</td>
<td>375</td>
<td>15</td>
<td>13</td>
<td>0</td>
<td>20</td>
</tr>
</tbody>
</table>

⁸⁴ANTU (α-naphthylthiourea) injected intraperitoneally, 30 minutes before exposure, at dosage of 15 mg/kg in all experiments except F; experiment F, 20 mg/kg.
immediately following exposure. In addition, irreversible alveolar tissue changes in aged mice, decreased spontaneous running activity, and increased susceptibility to streptococcal pneumonia in young adult mice are observed.

It should be noted that experimental exposure to irradiated auto exhaust usually involves variable concentrations of carbon monoxide, hydrocarbons, and nitrogen oxides, as well as “oxidants.” The studies of the effects of oxidants on animals are summarized in tabular form in Table 8-16. Apart from the production of eye irritation, no studies have been carried out on the effects of photochemical oxidant mixtures on humans.

D. PEROXYACYL NITRATES

1. Animal Data

Lethality

Data on the lethality of peroxycetyl nitrate (PAN) are sparse, but that which is available suggests that it is less lethal to mice than ozone, about the same as nitrogen dioxide, and more lethal than sulfur dioxide.

Campbell et al. exposed mice to high concentrations of PAN, 480 to 700 µg/m³ (97 to 145 ppm) as measured at the chamber outlet, for 2 hours at 80°F. The studies demonstrated that the majority of mice exposed to 540 µg/m³ or 110 ppm or more, PAN died within a month (Figure 8-13). It was observed that mice exposed to higher concentrations died earlier than those exposed to the lower concentrations. Median lethal exposures characteristically produced a delayed mortality pattern, with most deaths occurring in the second and third week after exposure. Mortality was greater among older mice than younger mice, and it was greater at higher temperatures. It was not influenced appreciably by changes in relative humidity.

2. Human Data

Effects on Pulmonary Function

Experiments carried out on humans have suggested that exposure to PAN results in increased oxygen uptake during exercise. Smith has carried out a group of studies on male college students averaging 21 years of age. The subjects were exposed to 1,485 µg/m³ (0.3 ppm) PAN by breathing through the mouth (nose clamps were used) for 5 minutes while at rest, and then the subjects were engaged immediately in 5 minutes of exercise on a bicycle ergometer. Both air containing PAN and air free of PAN were used without the knowledge of the subjects. Since the pollutant has no characteristic smell or taste, it was considered that the experiment was carried out in a “blind” fashion. Some of the data are presented in Table 8-17, and it can be noted that there was a statistically significant increase in oxygen uptake during exercise, without any change at rest. Expiration velocity was reduced after exercise. The changes could possibly be a reflection of an increase in the work of breathing or due to an increase in airway resistance. Because the report of this work does not adequately describe the experimental design or the statistical analysis, these results merit replication before conclusive statements can be made.

3. Discussion

The data obtained so far on the effects of PAN in animals and man are too incomplete to enable conclusions to be drawn regarding any effects of this pollutant at ambient concentrations.

E. SENSORY IRRITATION

1. Animal Data

Effects of Air Pollutants on the Eye

Experiments with various types of air pollutants have thus far failed to show significant physical or chemical effects on the eyes of exposed rabbits. Hine et al. exposed healthy albino rabbits to pure sulfur dioxide at 26,200 µg/m³ (10 ppm); nitrogen dioxide at 37,600 µg/m³ (20 ppm); and ozone, 3,720 to 5,490 µg/m³ (1.9 to 2.8
<table>
<thead>
<tr>
<th>Oxidant, ( \mu g/m^3 )</th>
<th>Oxidant, ppm</th>
<th>Source</th>
<th>Length of exposure</th>
<th>Observed effect(s)</th>
<th>Species</th>
<th>Reference</th>
</tr>
</thead>
</table>
| > 240           | > 0.12      | Irr. auto exhaust | 4 hours          | Short-term exposures  
Increased mortality from streptococcal pneumonia. | Mice    | Coffin and Blommer \(^{34}\)  
Murphy \(^{68}\) et al. |
| 650 to 1,610    | 0.33-0.82   | Irr. auto exhaust | 4 hours          | Increased expiratory flow resistance - 20 to 120%, increased inspiratory flow resistance - 40%. Decreased respiratory frequency - 15-35%.  
Alveolar tissue changes in animals aged 9 months or over. Increased severity with age. Damage at 9 months reversible, at 21 months irreversible. Disruption of epithelial walls; cytoplasmic fragments and proteinaceous material in alveoli. | Guinea pigs | Bils \(^{70}\)  
Bils and Romanovsky \(^{71}\) |
| > 780           | > 0.4       | Smog   | 2-3 hours         | Long-term exposures  
Increase in flow resistance (increase also occurred at lower oxidant levels). | Mice    | Swann \(^{66}\) et al.  
Swann and Balchum \(^{57}\) |
| 1,960 to 7,470  | 1.0-3.8     | Ozonized gasoline | Continuous       | Increased frequency of lung tumors seen after 24 weeks | Mice    | Kotin and Falk \(^{76}\)  
Kotin \(^{77}\) et al.  
Kotin \(^{77}\) et al. |
| 650 to 1,610    | 0.33-0.82   | Irr. auto exhaust | Continuous       | Systemic effects  
Short-term exposures  
8 to 80% decrease in spontaneous running activity. | Mice    | Murphy \(^{68}\) et al.  
Lewis \(^{82}\) et al. |
| 200 to 980      | 0.1 - 0.5   | Irr. auto exhaust | 16 hour day/46 days | Decrease in fertility. Doubling of non-pregnancy average. | Mice    | Lewis \(^{82}\) et al.  
Hueter \(^{37}\) et al. |
| 390 to 1,960    | 0.2-1.0     | Auto exhaust | Continuous       | Stress adaptation response, i.e. reduction in spontaneous running activity returning to pre-exposure levels. | Mice    |  
|
| 590 to 1,960    | 0.3 - 1.0   | Irr. auto exhaust | 16 hour day/46 days | Increased neonatal mortality due to preconditioning of males. | Mice    |  
|
Table 8-17. COMPARISONS OF MEANS AND PERCENTAGE CHANGE IN OXYGEN UPTAKE IN HEALTHY MALE COLLEGE STUDENTS AFTER INHALATION OF 1,485 µg/m³ (0.3 ppm) PEROXYACETYL NITRATE AND FILTERED AIR \(^{86}\)

<table>
<thead>
<tr>
<th>Activity</th>
<th>Oxygen uptake, liters/min</th>
<th>Change, percent</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Air</td>
<td>PAN</td>
</tr>
<tr>
<td>Rest for 5 minutes</td>
<td>1.65</td>
<td>1.66</td>
</tr>
<tr>
<td>Exercise for 5 minutes</td>
<td>10.32</td>
<td>10.55</td>
</tr>
<tr>
<td>Recovery for 5 minutes</td>
<td>3.30</td>
<td>3.34</td>
</tr>
</tbody>
</table>

\(^{a}\)Statistically significant (\(p < 0.05\)).

ppm); and to the vapors of acrolein, 4,580 to 18,320 µg/m\(^3\) (2 to 8 ppm); and a di-epoxide (3 to 12 ppm). These compounds were also used in various mixtures, with and without the addition of a saline aerosol. Two types of synthetic smog were also used: UV-irradiated ozonized gasoline, with or without nitrogen dioxide, and irradiated auto exhaust. The parameters measured were the regeneration rate of excoriated corneas, changes in the degree of chemosis (excessive edema of the conjunctiva), and iritis (inflammation of the iris). A single 4-hour exposure or 25 to 34 intermittent 1-hour exposures to the various gases produced no significant effects. In a

Figure 8-13. Cumulative mortality of mice from exposure to PAN.\(^{86}\)
similar study, Mettier et al. were unable to demonstrate ophthalmologic or biochemical effects on intact and de-epithelialized corneas of exposed rabbits.

2. Human Data

a. Olfactory Effects

Witheridge and Yaglou have reported that the odor of ozone could be detected by people with a keen sense of smell at 20 μg/m³ (0.01 ppm), and that normal people could detect the odor at 30 μg/m³ (0.015 ppm); the measuring techniques used in this study were less accurate at these low concentrations than methods of later studies. The authors conducted a series of experiments in which a number of subjects were placed in a poorly ventilated room and body odor intensity was allowed to reach a point of equilibrium. An ozonator was turned on and adjusted to produce the minimum ozone intensity consistent with minimal body odors. The subjects then evaluated the ozone and body odors. The authors claimed that the ozone odor intensity was not affected by the presence or absence of body odor. They found that the perceived body odor was due to the effect of the ozone on the mucous membranes of the nose, rather than on the odor agents themselves.

Henschler et al. have performed careful studies of the olfactory threshold and symptoms in 10 to 14 male volunteer test subjects exposed for 30 minutes to a series of different concentrations of ozone. Changes in sensation were recorded by the subjects at 5-minute intervals while in the chamber. The lowest concentration of ozone used, 40 μg/m³ (0.02 ppm), was recognized immediately by 9 of 10 exposed subjects. Thus Henschler et al. indicate that the odor threshold is below 40 μg/m³ (0.02 ppm). Following exposure to 40 μg/m³ (0.02 ppm), the subjects reported that the odor diminished rapidly. Within a period of 30 seconds to 12 minutes (average of 5 minutes), the odor was no longer perceptible. At 100 μg/m³ (0.05 ppm) ozone, 13 of 14 subjects indicated that the odor was considerably stronger and the odor perception lasted longer (2 to 30 minutes, with an average of 13 minutes).

b. Experimental Studies of Eye Irritation

The most obvious reaction of humans exposed to photochemical air pollution is the development of eye irritation with, in some instances, lacrimation. A number of complicated problems arise, however, when attempts are made to provide precise experimental measurements of eye irritation and to determine exactly what factors are responsible for reported irritation.

Ozone, the principal contributor to ambient oxidant levels, is not an eye irritant. The major photochemical products identified as eye irritants are acrolein, peroxycetyl nitrate (PAN), and peroxybenzoyl nitrate (PBzN). The recently discovered PBzN compound has been demonstrated to exhibit an eye irritation potency 200 times that of formaldehyde. Problems associated with studies of eye irritation generally fall into one of two categories: (1) Human experimental studies have been directed toward a definition of the substances responsible for irritation, and attempts have been made to correlate experimental data with those obtained from epidemiological studies, and (2) Difficulties have been encountered in experimental studies resulting from variables which include the measurement of a subjective response, individual differences in sensitivity, synthetic atmospheres, and the multiphasic nature of photochemical reactions.

First, there is the problem of obtaining clean air samples for irradiation, that is, samples which are uncontaminated with hydrocarbons and nitrogen oxides. It is difficult to cleanse the residual traces of reacted products from previous experiments from exposure chambers. Thus, additional impurities often build up.

Second, problems stem from the fact that measurement of eye irritation in humans is necessarily the measurement of a subjective response. Physiologic and psychologic factors contribute to the sensitivity of a particular
individual. Wide variations in response to a given irritant may depend on differences in sensitivity between individuals and between irritants; age, the older persons being more sensitive and sustaining irritant effects for longer periods; inflation due to other causes; and choice of experimental subjects.

A third complication is the nature of photochemical reactions themselves, which are fundamentally multiphasic with a complex time course (Figure 8-14). Thus it takes several hours for reactions to take place at realistic concentrations of hydrocarbons and nitrogen oxides. Both in chamber irradiations of hydrocarbons and nitrogen oxides and in polluted atmospheres, the earliest reaction is the conversion of nitric oxide to nitrogen dioxide. This is followed, about an hour later, by a peak in eye irritation response, and then by the maximal oxidant or ozone value, which occurs about 2 hours after peak eye irritation. The measured oxidant level, for practical purposes, may be considered as a weighted sum of the nitrogen dioxide and ozone.

3. Discussion

The characteristic pungent odor of ozone can be detected instantaneously at very low concentrations (less than 40 μg/m³, or 0.02 ppm, depending on individual acuity); at 100 μg/m³ (0.05 ppm), the odor is considerably stronger and persists longer. The odor persisted for an average of 5 minutes after the lower exposure, and an average of 13 minutes after the higher exposure.

The following conclusions can be reached on the basis of the existing data on eye irritation:

1. The effective eye irritants are the products of photochemical reactions.
2. Although oxidant concentrations may correlate with the severity of eye irritation, a direct cause-effect relationship has not been demonstrated. Ozone, the principal contrib-

![Figure 8-14. Analytical data for auto exhaust chamber experiments.](image)

8-39
that aldehydes and peroxynitrite contribute to a major extent, but it is probable that unidentified compounds are also responsible.

A thorough discussion relating to the eye irritation effects of air pollution will be found in the companion document, AP-64, *Air Quality Criteria for Hydrocarbons*.

**F. SUMMARY**

The data have been presented separately for both human exposures and animal studies; effects of ozone, oxidants, and PAN have been discussed and summarized separately. Table 8-18 summarizes the toxicologic studies of ozone exposure, and Table 8-19 summarizes the toxicologic studies of oxidant exposure. The data existing on the effects of PAN are extremely limited at the present time.

### Table 8-18. SUMMARY OF TOXICOLOGIC STUDIES OF OZONE EXPOSURE

<table>
<thead>
<tr>
<th>Effect</th>
<th>Ozone concentrations, ppm</th>
<th>µg/m³</th>
<th>Length of exposure</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morbidity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Local effects</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Perception of pungent odor (man)</td>
<td>0.02</td>
<td>40</td>
<td>&lt; 5 minutes</td>
<td>Henschler(^9^) et al.</td>
</tr>
<tr>
<td>Increased frequency of breathing. Decreased tidal volume (guinea pigs)</td>
<td>0.34</td>
<td>670</td>
<td>2 hours</td>
<td>Murphy(^9^) et al.</td>
</tr>
<tr>
<td>Chemical changes in lungs (rabbits)</td>
<td>1.00</td>
<td>1,960</td>
<td>1 hour</td>
<td>Buell(^12^) et al.</td>
</tr>
<tr>
<td>Engorged blood vessels and excess leukocytes in lung capillaries (mice)</td>
<td>1.00</td>
<td>1,960</td>
<td>4 hours</td>
<td>Scheel(^5^) et al.</td>
</tr>
<tr>
<td>Increased flow resistance (guinea pigs)</td>
<td>1.08</td>
<td>2,120</td>
<td>2 hours</td>
<td>Murphy(^9^) et al.</td>
</tr>
<tr>
<td>Increased lung weight. Decreased tidal volume, decreased minute ventilation (rats)</td>
<td>2.00</td>
<td>3,920</td>
<td>3 hours</td>
<td>Scheel(^5^) et al.</td>
</tr>
<tr>
<td>Gross pulmonary edema (rats)</td>
<td>3.20</td>
<td>6,290</td>
<td>4 hours</td>
<td>Scheel(^5^) et al.</td>
</tr>
<tr>
<td>Decreased lung compliance (guinea pigs)</td>
<td>5.00</td>
<td>9,800</td>
<td>2 hours</td>
<td>Easton(^10^) et al.</td>
</tr>
<tr>
<td>Gross pulmonary edema. Increased lung serotonin (rats)</td>
<td>6.00</td>
<td>11,800</td>
<td>4 hours</td>
<td>Skillen(^17^) et al.</td>
</tr>
<tr>
<td>Decreased tidal volume, decreased oxygen consumption (rabbits)</td>
<td>15.00</td>
<td>29,000</td>
<td>30 minutes</td>
<td>Scheel(^5^) et al.</td>
</tr>
<tr>
<td>Bronchitis, bronchiolitis, emphysematous and fibrotic changes; acceleration of lung tumor development (mice)</td>
<td>1.00</td>
<td>1,960</td>
<td>Continuous (at least 1 year)</td>
<td>Stokinger(^33^) et al.</td>
</tr>
<tr>
<td>Systemic effects</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Decreased voluntary running activity (mice)</td>
<td>0.20</td>
<td>390</td>
<td>6 hours</td>
<td>Stokinger(^6^)</td>
</tr>
<tr>
<td>Increased liver weight. Increased liver alkaline phosphatease (rats)</td>
<td>3.10</td>
<td>6,100</td>
<td>20 hours</td>
<td>Murphy(^31^) et al.</td>
</tr>
<tr>
<td>Decreased brain serotonin (rats)</td>
<td>6.00</td>
<td>11,800</td>
<td>4 hours</td>
<td>Skillen(^30^) et al.</td>
</tr>
<tr>
<td>Structural changes in heart myocardial fibers (mice)</td>
<td>0.20</td>
<td>390</td>
<td>5 hours/day/3 weeks (total 105 hours)</td>
<td>Brinkman(^18^) et al.</td>
</tr>
</tbody>
</table>

8-40
Table 8-18. (continued) SUMMARY OF TOXICOLOGIC STUDIES OF OZONE EXPOSURE

<table>
<thead>
<tr>
<th>Effect</th>
<th>Ozone concentrations, ppm</th>
<th>Ozone concentrations, µg/m³</th>
<th>Length of exposure</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Increased susceptibility to <em>Streptococcus</em> (Group C)</td>
<td>0.08</td>
<td>160</td>
<td>3 hours</td>
<td>Coffin⁴⁰ et al.</td>
</tr>
<tr>
<td>Increased susceptibility to <em>Klebsiella pneumoniae</em> (mice, hamsters)</td>
<td>0.84</td>
<td>1,650</td>
<td>4 hours/5 days/2 weeks</td>
<td>Miller⁴⁸ et al.</td>
</tr>
<tr>
<td>Increased susceptibility to <em>Klebsiella pneumoniae</em> (mice, hamsters)</td>
<td>1.30</td>
<td>2,550</td>
<td>3 hours</td>
<td>Miller⁴⁸ et al.</td>
</tr>
<tr>
<td>Increased susceptibility to histamine (guinea pigs)</td>
<td>5.0</td>
<td>9,800</td>
<td>2 hours</td>
<td>Easton¹⁰ et al</td>
</tr>
<tr>
<td>Increased mortality with exercise (15 minutes/hour) (rats)</td>
<td>1.0</td>
<td>1,960</td>
<td>6 hours</td>
<td>Stokinger⁶</td>
</tr>
<tr>
<td>Decreased mortality with age. Young—50% mortality; old—10% mortality</td>
<td>4.0</td>
<td>7,800</td>
<td>4 hours</td>
<td>Stokinger⁶</td>
</tr>
<tr>
<td>Decreased survival time due to stimulated thyroid activity (rats)</td>
<td>6.0</td>
<td>11,800</td>
<td>4 hours</td>
<td>Skillen⁷</td>
</tr>
<tr>
<td>LD₅₀ mice</td>
<td>21.0</td>
<td>41,000</td>
<td>3 hours</td>
<td>Mittler³ et al.</td>
</tr>
<tr>
<td>LD₅₀ rats</td>
<td>21.8</td>
<td>42,000</td>
<td>3 hours</td>
<td>Mittler³ et al.</td>
</tr>
<tr>
<td>LD₅₀ cats</td>
<td>34.5</td>
<td>67,980</td>
<td>3 hours</td>
<td>Mittler³ et al.</td>
</tr>
<tr>
<td>LD₅₀ rabbits</td>
<td>36.0</td>
<td>71,000</td>
<td>3 hours</td>
<td>Mittler³ et al.</td>
</tr>
<tr>
<td>LD₅₀ guinea pigs</td>
<td>51.7</td>
<td>101,400</td>
<td>3 hours</td>
<td>Mittler³ et al.</td>
</tr>
</tbody>
</table>

Table 8-19. SUMMARY OF TOXICOLOGIC STUDIES OF OXIDANT EXPOSURE ON ANIMALS

<table>
<thead>
<tr>
<th>Effect</th>
<th>Oxidant concentration, ppm</th>
<th>Oxidant concentration, µg/m³</th>
<th>Length of exposure</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morbidity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Local Effects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Increased flow resistance; decreased frequency of breathing (guinea pigs)</td>
<td>0.33–0.82</td>
<td>650–1,610</td>
<td>4 hours</td>
<td>Murphy⁶⁸ et al.</td>
</tr>
<tr>
<td>Changes in alveolar tissue in mice</td>
<td>&gt;0.4</td>
<td>780</td>
<td>2–3 hours</td>
<td>Büls⁷⁰, ⁷¹ et al.</td>
</tr>
<tr>
<td>Increase in flow resistance (guinea pigs)</td>
<td>&gt;0.5</td>
<td>980</td>
<td>continuous</td>
<td>Swann⁶⁶, ⁶⁷ et al.</td>
</tr>
<tr>
<td>Increase in lung tumors (mice)</td>
<td>1.0–3.8 (from ozonized gasoline)</td>
<td>1,960–7,470</td>
<td>24 weeks</td>
<td>Kotin⁷⁶, ⁷⁷ et al.</td>
</tr>
<tr>
<td>Systemic Effects</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Decrease in spontaneous running activity (mice)</td>
<td>0.33–0.82</td>
<td>650–1,610</td>
<td>6 hours</td>
<td>Murphy⁶⁸ et al.</td>
</tr>
<tr>
<td>Decrease in fertility. Doubling of non-pregnancy average (mice)</td>
<td>0.1–0.5</td>
<td>200–980</td>
<td>16 hours day/46 days (total, 736 hours)</td>
<td>Lewis⁸² et al.</td>
</tr>
<tr>
<td>Mortality</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Increased neonatal mortality due to pre-conditioning of males (mice)</td>
<td>0.3–1.0</td>
<td>590–1,960</td>
<td>16 hours day/46 days (total, 736 hours)</td>
<td>Lewis⁸² et al.</td>
</tr>
<tr>
<td>Increased mortality from streptococcal pneumonia</td>
<td>&gt;0.12</td>
<td>240</td>
<td>4 hours</td>
<td>Coffin and Blommer⁸⁴</td>
</tr>
</tbody>
</table>
G. REFERENCES


Chapter 9.

EPIDEMIOLOGICAL APPRAISAL OF PHOTOCHEMICAL OXIDANTS

A. INTRODUCTION

The possibility that photochemical air pollution could be a major health hazard has been of growing concern. A number of systematic studies have been conducted in an attempt to obtain an association between episodes of high oxidant pollution and general mortality, acute illness, aggravation of chronic respiratory disease, impairment of performance, or untoward symptoms such as eye irritation. The purpose of this chapter is to review the data and conclusions of these studies. In some cases, the data have been reanalyzed. Areas of insufficient knowledge and the relevance of the existing data to air quality criteria are emphasized.

B. ACUTE EFFECTS OF PHOTOCHEMICAL OXIDANTS

To identify the acute health effects of photochemical oxidant pollution, observations of the same populations or communities are made during periods of high-level pollution and during periods of low-level pollution. These health effects are associated with short-term pollutant concentrations measured as 24-hour averages, hourly maxima, or instantaneous peak concentrations.

1. Daily Mortality in Relation to Variations in Oxidant Levels

a. Mortality Among Residents Age 65 Years and Older

A study of the relationship between daily concentrations of photochemical oxidants and daily mortality among residents of Los Angeles County age 65 years and older was reported in March 1955, March 1956, and February 1957. The number of deaths per day was related to 2 indices: daily temperature at the downtown weather bureau, and oxidant concentrations from August through November of 1954 and July through November of 1955. (Table 9-1 and Figure 9-1) Daily mortality clearly exceeded the average during a heat-wave occurring in late August and early September of 1955. High concentrations of photochemical oxidants occurred immediately before, during, and after the heat-wave. Daily mortality decreased, however, when the temperatures dropped while elevated oxidant levels persisted.

Thus the oxidant level did not independently affect daily mortality counts. An inconsistent pattern was observed during other months when daily mortality was compared with both temperature and oxidant gradients (Table 9-1). For example, on days when the maximum temperature fell to between 70°-79° F, variations in oxidant levels from low to medium to high showed no consistent relationship to the average number of deaths. For the months covered by this study, therefore, daily mortality among persons age 65 years and older in Los Angeles County was strongly influenced by a heat wave, but was not altered consistently by variations in oxidant concentrations.

b. Mortality and Heat Waves

Since there is a meteorological association between high temperatures and high oxidant concentrations in the Los Angeles basin, the conclusions of the previous paragraph do not preclude a relationship between increased mortality and the simultaneous occurrence of high temperatures and high oxidant concentrations. That is, high oxidant concentrations may augment the mortality effect of high temperature. The results of a study by Oechsli
Table 9-1. AVERAGE NUMBER OF DEATHS PER DAY DUE TO CARDIAC AND RESPIRATORY CAUSES AMONG RESIDENTS OF LOS ANGELES COUNTY, AGE 65 AND OLDER, AS RELATED TO TEMPERATURE AND OXIDANT CONCENTRATIONS BY MONTH, 1954–1955

<table>
<thead>
<tr>
<th>Concentration, ppm</th>
<th>Number of days</th>
<th>Average number of deaths</th>
<th>Number of days</th>
<th>Average number of deaths</th>
<th>Number of days</th>
<th>Average number of deaths</th>
<th>Number of days</th>
<th>Average number of deaths</th>
<th>Number of days</th>
<th>Average number of deaths</th>
<th>Number of days</th>
<th>Average number of deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>50°-59°F</td>
<td>60°-69°F</td>
<td>70°-79°F</td>
<td>80°-89°F</td>
<td>90°-99°F</td>
<td>100°F and above</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>August, 1954</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low (.00 - .24)</td>
<td>8</td>
<td>29.9</td>
<td>4</td>
<td>28.2</td>
<td>4</td>
<td>31.5</td>
<td>3</td>
<td>39.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medium (.25 - .49)</td>
<td>22</td>
<td>32.2</td>
<td>7</td>
<td>30.1</td>
<td>12</td>
<td>31.8</td>
<td>3</td>
<td>39.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High (.50 +)</td>
<td>1</td>
<td>32.0</td>
<td>1</td>
<td>32.0</td>
<td>1</td>
<td>32.0</td>
<td>1</td>
<td>32.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>September, 1954</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low (.00 - .24)</td>
<td>1</td>
<td>26.0</td>
<td>1</td>
<td>26.0</td>
<td>1</td>
<td>26.0</td>
<td>1</td>
<td>26.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medium (.25 - .49)</td>
<td>25</td>
<td>32.1</td>
<td>5</td>
<td>29.6</td>
<td>16</td>
<td>35.1</td>
<td>4</td>
<td>36.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High (.50 +)</td>
<td>4</td>
<td>33.3</td>
<td>4</td>
<td>33.3</td>
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and Buechley\textsuperscript{2} of mortality associated with three Los Angeles heat-waves—in 1939, 1955, and 1963—provide information on this multiple-relationship. Daily mortality during these heat-waves was compared with daily mortality occurring immediately before and after each heat-wave and with mortality during the same season in 1947, when no heat-wave occurred.

Significant increases in the mortality ratio were observed with increases in temperature during each of the three heat waves. Above average deviations were greatest among the more elderly persons. Of the three incidents, the one occurring in 1963 is notable for a considerable diminution of excess mortality, even though temperatures reached the same peak levels as in 1939 and 1955. It has been surmised that the use of air conditioning was one of several possible reasons for the reduction in mortality in 1963. There was no discernible difference in the magnitude of the mortality response to heat in 1939 and 1955. It can be assumed that considerably less photochemical oxidant pollution accompanied the 1939 heat wave than was present in 1955, although oxidant was not being measured in 1939. The comparison of the 1939 and 1955 heat waves suggests, under the above assumption, that high photochemical oxidant concentrations do not augment the mortality effect of high temperatures.

Figure 9-1. Comparison of deaths of persons, 65 years of age and over, and maximum daily temperatures, Los Angeles County, July 1 to November 30, 1955.\textsuperscript{1}
c. Mortality of Nursing Home Residents

An attempt was made to establish whether nursing home patients, many of whom were chronically ill and thought to be unusually susceptible to atmospheric pollution, experienced greater mortality on or immediately after days of high oxidant concentrations.\(^1\) Deaths and transfers to hospitals among residents of 16 Los Angeles nursing homes having a total of 358 beds were recorded for 1954. An unusually large number of patients appeared to have been transferred to hospitals following a particularly heavy episode of smog during 1 week of the study period. A larger study of the nursing home population was conducted from July through December of 1955, during which all such homes in Los Angeles County containing 25 or more beds were surveyed. The number of institutions ranged from 90 to 92, with a total of 3,734 to 3,826 beds. Daily mortality, the corresponding maximum daily temperature, and the occurrence of smog-alert days with ozone concentrations of 590 \(\mu g/m^3\) (0.30 ppm) or higher are shown in Figure 9-2. The heat-wave in late August and early September again showed a striking effect on mortality; at all other times, variations in daily mortality did not appear to be related to the occurrence of smog-alert days.

d. Two-Community Study

Massey\(^3\) et al. compared daily mortality in two areas of Los Angeles County. These areas were selected for both similarities in temperature and differences in air pollution levels. Two synthetic communities containing a combined population of 944,391 persons were thus formed. The pollutant variables used in this analysis were the daily maximum and mean oxidant levels as established using the KI method, and sulfur dioxide and carbon monoxide concentrations. The synthetic communities were subdivided into smaller units,

![Figure 9-2. Comparison of nursing home deaths, maximum daily temperature, and '"smog alert" days in Los Angeles County, July through December 1955.](image-url)
each of which was represented by a single temperature or air pollution station. The mean number of daily deaths in the low pollution area was subtracted from the mean number of deaths in the high pollution area, and the differences were examined by correlation and regression analysis with respect to differences in pollution. No significant correlations between mortality differences and differences in pollutant levels were observed.

e. Mortality from Cardiac and Respiratory Diseases

Hechter and Goldsmith[4] analyzed the effect of pollutant concentrations on average daily mortality from cardiac and respiratory diseases in Los Angeles County for the years 1956 through 1958. Daily mortality, averaged within each month of the study, fluctuated between 1.0 and 1.3 per 100,000 population. These fluctuations were approximately 180 degrees out of phase with fluctuations in oxidant and temperature values, and approximately in phase with maximum carbon monoxide concentrations (Figure 9-3). To remove the major effect of season of year, the authors fitted Fourier curves to the data and found a single cycle of Fourier functions to fit oxidant and temperature fluctuations in carbon monoxide and cardiorespiratory mortality (Figure 9-4). The residual variations from

Figure 9-3. Comparison of maximum concentrations of oxidant and carbon monoxide, maximum temperature, and daily death rate for cardiac and respiratory causes, Los Angeles County, 1956-1958.4
these fitted curves for each of the variables were presumed to be independent of season. The relationship between pollution or temperature on 1 day with the value of the same variable on the successive or following days was also accounted for in the analysis. When residuals from the fitted curves were thus analyzed, no significant correlations between pollutants and mortality for cardiorespiratory diseases were found. Neither were there significant correlations when a lag of 1 to 4 days was applied.

Mills⁵ has attempted to relate cardiorespiratory deaths to the occurrence of smog in Los Angeles. Although he observed an association between oxidant levels and excess deaths, he failed to take into account seasonal fluctuations of each, thus precluding the comparison of this observation with other studies in this section.

The analysis of mortality data by fitting them to Fourier curves derived from the same data may mask a real effect of environmental factors on mortality. In the absence of a simultaneous study of a less-polluted comparison community, no conclusive statement can be made regarding the effect of oxidant levels on community mortality.

f. Discussion

A variety of methods have been used to examine whether short-term variations in photochemical oxidant concentrations have been associated with excess mortality in Los Angeles. The results have not demonstrated such an effect. Studies comparing the mortality

![Figure 9-4. Fourier curves fitted to data in Figure 9-3](image-url)
experience of Los Angeles with that of a less-polluted community are needed before firm conclusions can be drawn about the observed lack of a pollutant-related mortality.

2. Hospital Admissions in Relation to Oxidant Levels

a. Los Angeles County Hospital
Admissions, 1954

A study of hospital admissions for the period September through December of 1954 has examined records on admissions to Los Angeles County Hospital in several categories,¹ namely, admissions for asthma in children under 10 years of age, for tuberculosis, for other respiratory conditions, and for other causes. Total admissions, the number of persons with acute conditions in all units, and the number who died, were tabulated. No significant associations with oxidant levels were observed. Weekly admissions to an additional group of hospitals with a total of 2,224 beds were similarly examined. The data appeared to show some seasonal trend, particularly for certain diseases of the lung, but no association between oxidant air pollution and hospital admissions due to diseases of the cardiovascular or respiratory systems was observed.

Brant⁶,⁷ studied patients with respiratory or cardiovascular diagnoses who were admitted to or discharged from Los Angeles County Hospital between August 8 and December 25, 1954. Patients were excluded who were younger than 9 or older than 90 years of age, as were those who had not resided for at least 3 years in an area within 8 miles of downtown Los Angeles. Meteorological data including temperature and relative humidity were included in the analysis. Although the method of selection of patients for the study was not described, the process of selection yielded 246 cardiovascular admissions and 122 respiratory admissions. Total oxidant as measured about a mile from the hospital was used as an index of air pollution. Multiple regression analyses were used to relate hospital admissions to atmospheric and meteoro-

logical variables. Calculations included regressions of hospital admissions as late as 4 weeks after the occurrence of a given set of environmental measurements, and a significant correlation between periods of high oxidant, low relative humidity, and low temperature, and hospital admissions weeks later for cardiovascular conditions was observed. Because there is no biologically plausible explanation for this correlation, the association must be considered fortuitous. In contrast, there was a negative correlation between admissions for cardiovascular disease and oxidant levels either on the day of admission or for 2 weeks prior to admission.

b. Hospital Admissions in the Los Angeles Metropolitan Area

Based on Blue Cross records, Sterling,⁸,⁹ et al. assembled data from Los Angeles hospitals for the period of March 17 to October 26, 1961. Diagnoses were grouped according to "highly relevant," "relevant," and "irrelevant" disorders. Classified as "highly relevant" were allergic disorders, inflammatory diseases of the eye, acute upper respiratory infections, influenza, and bronchitis. "Relevant" disorders were considered to include diseases of the heart, rheumatic fever, vascular diseases, and other diseases of the respiratory system. All other illnesses were classified as "irrelevant."

The authors found that the mean number of admissions varied by day of week. They were higher in the first-half than in the second-half of the week. They also observed (1) oxidant precursors were exceptionally low on Sundays, (2) both oxidant and ozone levels were low on Saturdays and Sundays, (3) nitric oxide was exceptionally low on Wednesdays. After correcting for the day of the week with respect to pollutant and admission frequencies, "highly relevant" and "relevant" illnesses were found to show significant correlations with oxidant levels, carbon monoxide, and ozone. "Highly relevant" conditions were also correlated with oxides of nitrogen and particulate matter. No significant correlations between hospital admissions and ten-
perature were found, in contrast to the findings discussed in section B.1 and in Figure 9-3, although only data for March and April were used. Using the same data, Sterling et al. studied the association of duration of hospital stay and air pollution. The correlations were not statistically significant.

Because of the limited period of the year utilized for study, and extremely low correlation coefficients, it is difficult to conclude that a meaningful relationship has been demonstrated by these studies.

c. Discussion

Studies reviewed in this section have failed to adequately demonstrate any relationship between daily hospital admissions and variations in concentrations of photochemical oxidants.

3. Aggravation of Respiratory Diseases by Oxidant Pollution

a. Aggravation of Asthma

As a result of reports by physicians that asthmatic attacks are frequently associated with smog periods, Schoettlin and Landau undertook a study to determine whether such a phenomenon does take place. Five physicians selected 157 patients, 137 of whom participated in the study. All resided and worked in the Pasadena area. Fifty-four of the patients were younger than 15 years of age. Most had had asthma for at least 2 years, and all but 17 had been residents of the Los Angeles area for 5 or more years. Daily records of the time of onset and severity of asthmatic attacks were maintained by the patients for the period September 3 through December 9, 1956. Relatively high air pollution levels occur during these months of the year. The data were collected weekly. In the event a subject neglected to submit a report, the investigators telephoned and requested one.

The study showed that the peak period for asthma attacks was between midnight and 6:00 a.m., while the maximum oxidant levels were recorded between 10:00 a.m. and 4:00 p.m. A delay in response to the inciting agent might explain this late period for asthma attacks. Of the 3,435 attacks reported, less than 5 percent were spontaneously associated with smog by the patients, and none of these were severe. One-third of the attacks spontaneously associated with smog were reported by a single patient. The correlation-coefficient between the number of attacks per day and the maximum oxidant reading was 0.37. Addition of other variables to the analysis did not significantly alter these results. There was no significant difference in the average number of attacks per day for days above the median maximum oxidant level (0.13 ppm by the phenolphthalein method) compared with days below the median. However, there was a significant increase in the mean number of attacks on days when daily maximum oxidant levels exceeded 0.25 ppm by the phenolphthalein method, the equivalent of 250 μg/m³ or 0.13 ppm by the KI method, contrasted with days when the daily maximum oxidant level fell below this level. The authors suggested that this may indicate a threshold-level for oxidants above which there could be a physiologic response. There was also a significant association between attack-rates on days in which plant damage occurred, with oxidant concentrations of about 200 μg/m³ (0.10 ppm) by the KI method, in contrast to days without plant damage. This effect was most pronounced for persons who had lived in the area 10 or more years. The data were examined further to see whether a small number of the subjects might be responsible for the observed correlation. Eight individuals, or 6 percent of the total panel, were identified whose attacks corresponded most often to days on which plant damage occurred, but there was no attribute common to these individuals. Thus a small portion of people suffering from asthma appeared to be responsive to levels of photochemical oxidants sufficient to cause vegetation damage.

b. Aggravation of Emphysema and Chronic Bronchitis

Several studies have been conducted to determine if air pollution aggravates the con-
dition of subjects suffering from chronic bronchitis and emphysema.

Motley\textsuperscript{11} et al. reported on the results of lung function tests on 66 volunteers, 46 of whom had pulmonary emphysema. Lung function tests were performed on subjects in rooms from which the oxidants were removed by activated charcoal filters. Twenty-one subjects stayed in the filtered rooms for periods between 2 and 4 hours, 20 subjects between 18 and 20 hours, and 25 subjects between 40 and 90 hours. No accurate measurements of oxidants were obtained but, during the periods of the study, oxidant concentrations ranged from 390 to 1,370 \(\mu g/m^3\) (0.2 to 0.7 ppm) and ozone from 390 to 1,040 \(\mu g/m^3\) (0.2 to 0.53 ppm) at monitoring stations several miles from the chamber. Air was classified as smoggy when there was a definite odor of ozone, reduced visibility, eye irritation, and the prediction of smog by the Los Angeles Air Pollution Control District. Lung function measurements were made of vital capacity, \(FEV_{3.0}\), and maximal breathing capacity. Residual volume and air distribution measurements were also recorded. An improvement in lung function was observed, particularly a decrease in the residual lung volume in emphysematous subjects who remained in the chamber for 40 or more hours and who entered it on smoggy days. No significant changes in lung volume measurements were obtained when normal subjects breathed filtered air. No significant changes were observed when emphysematous subjects entered the chamber on non-smoggy days. Variations in the smoking habits of test subjects could have significantly influenced the results, but no information on such variations was reported.

At Los Angeles County Hospital, Remmers and Balchum\textsuperscript{12} utilized a room with an air conditioning system and a filter which could be used at the discretion of the investigator to remove photochemical oxidants, ozone, and nitrogen oxides from ambient air and partially remove particulate matter. Studies were conducted in September and October of 1964 and from March to November of 1965. Subjects performed lung function tests one or more times daily while they lived in the room. In general, they spent 1 week in the air in the room without air filtration, and 1 week with the air filtered. During both weeks, air-conditioning was adjusted to maintain a room temperature of \(72^\circ \pm 3^\circ F\) and a relative humidity of \(50 \pm 5\) percent. A third week was spent in a room arranged for exposure to ambient atmosphere. Respiratory function tests of airway resistance, diffusing capacity, other pulmonary function tests, blood oxygen tension, and oxygen consumption during exercise were performed. Several tests were repeated while the patients were exercising. Because of the elaborate nature of the experimental system, only a small number of subjects could be studied. Eleven subjects were cigarette smokers, and one stopped smoking when he entered the study. Most subjects had moderate to severe respiratory impairment from bronchitis or emphysema. Hence, any impairment of respiratory function could be interpreted as a fairly serious deterioration of pulmonary status.

Preliminary examination of the data indicated that airway resistance was affected by elevated oxidant concentrations when observations were made over a range of 100 to 450 \(\mu g/m^3\) (0.05 to 0.23 ppm). Determination of a threshold level for this effect cannot be made from these data because of the infrequent observations over the entire range of exposure. The subjects were also exposed to ambient air containing not only oxidants, but other substances as well. It is possible that the observed effects were caused partially by removal of other pollutants such as aerosols, particulates, PAN, or aldehydes. Cigarette smoking also varied and apparently was more frequent when the ambient air was not being filtered, thus possibly confounding the results. Another test which seemed to show significant changes with air pollution was the consumption of oxygen during exercise. Alterations in rate and depth of breathing could also have accounted for some of these effects,
but these possibilities were not investigated. Further analyses of these data were carried out for purposes of this report, and the results are shown in Table 9-2.

In view of the possible influence of cigarette smoking on these test results, data for cigarette smokers must be interpreted cautiously. The majority of the nonsmokers in this study did show decreases in airway resistance corresponding to decreases in oxidant exposure, but observations were far too infrequent for inferences to be drawn about a possible threshold level for this effect.

Rokaw and Massey\textsuperscript{13} conducted a study of the effects of environmental variables on pulmonary function in a group of 25 patients in a chronic disease hospital in Los Angeles, over a period of 18 months. All of the patients had chronic, nontuberculous, respiratory diseases, predominantly pulmonary em-

Table 9-2. CORRELATION OF MORNING AND EARLY AFTERNOON OXIDANT LEVELS WITH OXYGEN CONSUMPTION AND AIRWAY RESISTANCE OF 15 PATIENTS WITH CHRONIC RESPIRATORY DISEASE\textsuperscript{12}

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\textsuperscript{a} The first two correlations are with "resting" oxygen consumption, which was measured around 11 a.m.; the third correlation is with "exercise" oxygen consumption, which was measured around 3 p.m.

\textsuperscript{b} Airway resistance is "resting"; values given are averages of four measurements made throughout the day

\textsuperscript{c} a.m. Oxidant was measured around 9:30 a.m.

\textsuperscript{d} p.m. Oxidant was measured around 1:30 p.m.

\textsuperscript{e} Smoking History: S = smoker, NS = nonsmoker, SS = stopped smoking when study began. [ - Indicates patients were tested during the same period.

\textsuperscript{f} "z" values were found by converting the individual correlations to t values, using the relationship

\[ t_i = \frac{(r_i - 1)}{(1 - r_i)(n - 1)} \]

then summing the t values over all patients; the sums have variance

\[ \Sigma (t_i) = \Sigma [(n_i - 2)(n - 4)] \]

\[ \Sigma (t_i) / \sqrt{\Sigma \Sigma (t_i)} \]

which is approximately N(0,1).

\textsuperscript{g} Significant at the 0.01 level.

\textsuperscript{h} Significant at the 0.001 level.
physema. Each subject underwent a series of pulmonary function tests 4 times weekly; in addition, functional residual capacity by the helium dilution method was determined monthly. Air pollution data were obtained from a station about a quarter of a mile upwind from the hospital. Statistical methods of analysis were employed to detect association between changes in pulmonary function and air pollution levels. The results showed a marked variability in the performance of pulmonary function tests; the variability was greater in the group of patients than in normal subjects. A correlation between performance and ambient carbon monoxide was observed for some but not all of the subjects; no such response was observed during major smog episodes, and no seasonal pattern of performance for the group was observed. [This is in contrast to the findings of Mc Kerrow, who found yearly cycles in ventilatory function tests in a group of ex-miners with pneumoconiosis working in a car-assembly plant in South Wales.] The general level of oxidant in the hospital area was not as high as in some parts of the Los Angeles basin; the mean oxidant was 120 µg/m³ (0.06 ppm), with a maximum of 820 µg/m³ (0.42 ppm). It is possible that the experiment did not detect any effect of oxidant on simple respiratory function tests because; (1) the group was too ill to respond, (2) fluctuations in the subjects' disease states or cigarette smoking habits were the major determinants of test performance, or (3) the subjects were exposed to a relatively low level of oxidant.

Shoetlin studied the long-term effects of community air pollution, occupational exposure to air pollution, and smoking among Armed Forces veterans living in the Domiciliary Unit and Chronic Disease Annex of the Los Angeles Veterans Administration Center. Day to day variations in the physical status of men with chronic respiratory disease were studied in relation to changes in environmental conditions in the coastal area of the Los Angeles basin. Two groups of men were selected. The first group, consisting of 528 veterans who had no positive signs or symptoms of respiratory disease, was used as a control. The second group of 326 men was selected on the basis of the presence of at least 2 symptoms of respiratory disease (including cough, sputum, shortness of breath, and wheezing) for 2 or more years, and the presence of abnormal breath sounds. Men from these two groups were matched by age and smoking history, and the resulting pairs were studied weekly by means of repeated pulmonary function tests and responses to a respiratory symptom questionnaire. About 65 percent of the men selected cooperated in the study. An air pollution monitoring station was set up at the site, and a companion program of observing vegetation damage was established. Analysis of variance showed no statistically significant effects of air pollution on respiratory symptoms or functions. The maximal oxidant values and oxidant precursor values, however, consistently accounted for more of the variation in frequency of symptoms and clinical signs for the diseased group than for the control group (Table 9-3). For example, the maximal oxidant precursor accounted for 30 percent of the variation in symptoms of the diseased group and 17

<table>
<thead>
<tr>
<th>Environmental factors</th>
<th>Diseased group</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Symptoms</td>
<td>Signs</td>
</tr>
<tr>
<td>Mean oxidant</td>
<td>0.11</td>
<td>0.13</td>
</tr>
<tr>
<td>Maximal oxidant</td>
<td>0.17</td>
<td>0.16</td>
</tr>
<tr>
<td>Maximal oxidant precursor</td>
<td>0.30</td>
<td>0.17</td>
</tr>
<tr>
<td>Pollen count</td>
<td>0.02</td>
<td>0.05</td>
</tr>
<tr>
<td>Maximal temperature</td>
<td>0.05</td>
<td>0.03</td>
</tr>
<tr>
<td>Maximal relative humidity</td>
<td>0.05</td>
<td>0.05</td>
</tr>
</tbody>
</table>
percent of variations in the Puffmeter test, whereas the proportion of the variation accounted for in the control group was insignificant.

c. Discussion

A study of 137 patients with asthma demonstrated a significant increase in the number of asthma attacks on days when photochemical oxidant levels exceeded 250 µg/m³ (0.13 ppm) by the KI method. Using the information presented in Chapter 3, section B.1, the peak value of 250 µg/m³ oxidant might be expected to be associated with a maximum hourly oxidant concentration of 100 to 120 µg/m³ (0.05 to 0.06 ppm), depending on localized conditions.

studied under usual conditions of ambient oxidant exposure and then compared in a clean, filtered room have shown improvement in ventilatory function. The data are not adequate for determining the threshold level at which improved ventilatory function first occurred. In a study performed in a location with a mean oxidant concentration of 120 µg/m³ (0.06 ppm), however, no association between variations in oxidant concentration and the performance of ventilatory function tests by patients with chronic respiratory disease was demonstrable. In another study, no statistically significant association was found between oxidant concentrations and respiratory symptoms or functions in a selected group of subjects with or without chronic respiratory disease.

4. Impairment of Performance Associated with Oxidant Pollution

a. Athletic Performance

Wayne et al. have studied the athletic performance in 21 competitive meets of student cross-country track runners at San Marino High School, Los Angeles County, from 1959 to 1964. Oxidant measurements for the hour of the race, and 1, 2, and 3 hours before the race were related to the running time for each athlete. The effects of oxides of nitrogen, temperature, relative humidity, wind velocity, and wind direction were all considered but did not reveal any relationship to the running times. A significant relationship was observed between oxidant levels and the percent of team members whose performance decreased compared to their performance in the immediately previous home meet, as is shown in Figure 9-5. The correlations between performance and oxidant levels were quite high but diminished as the interval between the oxidant measurement and the time of the meet increased. The authors speculated as to possible mechanisms for this association and pointed out that a direct effect on oxygen utilization could occur or that there might be detrimental effects of discomfort from eye and respiratory irritation. The data provide convincing evidence that some component of the air which was measured as oxidant had a causal effect on team performance. With increasing levels of oxidant, there was a manifest impairment of team performance in this study, conducted

![Figure 9-5. Relationship between oxidant level in the hour before an athletic event and percent of team members with decreased performance.](image-url)
over a range of oxidant concentrations from 60 to 590 $\mu$g/m³ (0.03 to 0.30 ppm). No threshold level for this effect can be determined since the possibility always exists that a team would always have a certain number of individuals who would fail to improve their previous performance.

b. Automobile Accidents

A study of the association of automobile accidents with days of elevated oxidant levels was performed because of the possibility that oxidant pollution may impair performance either directly, by interfering with oxygen transport or utilization, or indirectly, by eye discomfort and respiratory irritation. Ury applied a sign-test and a non-parametric correlation analysis to data for each daylight hour of each weekday in the 3-month period from August through October, for both 1963 and 1965. There was a total of 90 sets of data for testing, covering 9 hours daily and 5 days weekly. The sign-test results of each set were obtained by taking successive pairs of weeks (first week compared with second; third week compared with fourth, etc.) and scoring a plus (+) if the week with the higher oxidant had more accidents for that set and a minus (-) if it had fewer, and awarding a tie if the accident frequency or the oxidant levels were equal (Table 9-4). No particular pattern was shown by hours of the day, but a pattern did appear based on the day of the week.

Table 9-4. SIGN-TEST DATA FOR TESTING THE ASSOCIATION OF OXIDANT LEVELS WITH ACCIDENTS, LOS ANGELES, AUGUST THROUGH OCTOBER, 1963 AND 196517

<table>
<thead>
<tr>
<th>Weekday</th>
<th>Year</th>
<th>Plus</th>
<th>Minus</th>
<th>Tie</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monday</td>
<td>1963</td>
<td>17</td>
<td>23</td>
<td>5</td>
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<tr>
<td></td>
<td>1965</td>
<td>21</td>
<td>21</td>
<td>8</td>
</tr>
<tr>
<td>Tuesday</td>
<td>1963</td>
<td>25</td>
<td>14</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>1965</td>
<td>25</td>
<td>21</td>
<td>6</td>
</tr>
<tr>
<td>Wednesday</td>
<td>1963</td>
<td>23</td>
<td>18</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>1965</td>
<td>24</td>
<td>21</td>
<td>9</td>
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<tr>
<td>Thursday</td>
<td>1963</td>
<td>29</td>
<td>13</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>1965</td>
<td>27</td>
<td>19</td>
<td>6</td>
</tr>
<tr>
<td>Friday</td>
<td>1963</td>
<td>24</td>
<td>25</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>1965</td>
<td>25</td>
<td>21</td>
<td>6</td>
</tr>
<tr>
<td>Total</td>
<td>1963</td>
<td>118</td>
<td>93</td>
<td>26</td>
</tr>
<tr>
<td></td>
<td>1965</td>
<td>122</td>
<td>103</td>
<td>35</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>240</td>
<td>196</td>
<td>61</td>
</tr>
</tbody>
</table>

The data from this study indicate a statistically significant relationship between oxidant levels and automobile accidents. The method of analysis employed in the study does not lend itself to the determination of a threshold level for this effect. Similar results were obtained with the non-parametric correlation test. Carbon monoxide, oxides of nitrogen, or particulates, which would also have been relatively increased during the periods when oxidant was elevated, could also have contributed to the observed results. Furthermore, increased traffic density at certain times may have caused increases in both oxidant levels and accident rates. Thus, additional statistical analyses are indicated.

c. Ventilatory Performance

The ventilatory performance, measured by the Wright Peak Flow Meter, of two groups of elementary schoolchildren living in the Los Angeles basin was assessed twice monthly for 11 months by McMillan et al. One group of 50 children resided in an area exposed to seasonally high photochemical oxidant concentrations. The other group of 28 children lived in a less polluted area. During the 11 months of the study, no correlations were found between acute changes in photochemical oxidant pollution and ventilatory performance. Contrary to expectation, persistently higher ventilatory performance results were obtained from the children residing in the more polluted of the two communities. Several important differences existed, however, between these two groups of children. The incidence of upper respiratory tract illness was reported to be three times greater in the children of the less polluted community, in which lower ventilatory performance was measured. A significant ethnic difference was also present; the majority of children in the less polluted community were from a single ethnic group, whereas the other group of children were ethnically heterogeneous. Thus the expected impairment of photochemical oxidant pollution on ventilatory performance was not found either because the group differences other than air...
pollution exposure had a ventilatory performance effect opposite to that of oxidant pollution, or because differences in oxidant exposure of the magnitude found in this study produced no functional impairment. In either case, variations in photochemical oxidant pollution, from daily averages of 100 to 550 μg/m³ (0.05 to 0.28 ppm), had no demonstrable effect on the ventilatory performance of the 50 schoolchildren living in the more polluted of the two communities.

d. Discussion

In a study of school athletic meets, it was shown that team performance decreased as hourly oxidant concentrations increased over the range of 60 to 590 μg/m³ (0.03 to 0.30 ppm). It has also been shown that there were significantly more automobile accidents on days of high oxidant concentration. A minimum threshold level for the oxidant effect on athletic performance or accident frequency cannot be determined from the data available.

No acute or chronic effects on ventilatory performance of elementary schoolchildren were found in a study conducted during an 11 month period in Los Angeles basin when daily average photochemical oxidant concentrations varied from 100 to 550 μg/m³ (0.05 to 0.28 ppm).

5. Eye Irritation in Relation to Variations in Oxidant Levels

a. Panel Studies

A variety of individuals in various parts of Los Angeles have been studied to determine the occasions and the types of pollutants responsible for eye irritation, one of the most frequently reported symptoms associated with exposure to oxidant pollution. The first set of studies was conducted by the Air Pollution Foundation in 1954.19

During the first period observers were asked to report eye irritation on Tuesdays and Fridays. This was later changed to just those days for which eye-irritating levels of pollution were predicted. In general, the observers were office and factory workers; one of the panels consisted of a group of staff members of the California Institute of Technology. The eye irritation data were compared with instantaneous values of oxidant concentrations as measured by potassium iodide recorder. The data are shown in Figures 9-6 and 9-7 and in Tables 9-5 and 9-6. The “expert” panel (experienced scientists) and the other panel did not significantly differ in the correlations of eye irritation with oxidants, carbon monoxide, particulates, and aldehydes. During the second period, August through November of 1955, similar panels observed eye irritation effects during each day of the work week. In this study maximum oxidant concentrations were compared with reported eye irritation effects.

From the data provided by the Air Pollution Foundation studies, linear mathematical relationships between maximum oxidant values and mean maximum eye irritation values were derived. The data from these studies demonstrated increasing eye irritation with increasing concentrations of oxidant pollution over the range of instantaneous values from 100 to 880 μg/m³ (0.05 to 0.45 ppm), although no clearly demarcated threshold level for this effect is apparent (See Figure 9-6).

Other studies on eye irritation have been performed, including one in which a panel of

<table>
<thead>
<tr>
<th>Station</th>
<th>Number of daily observations</th>
<th>Variance, D (or r²)</th>
<th>Average eye irritation score</th>
<th>Average oxidant concentration, ppm</th>
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</thead>
<tbody>
<tr>
<td>5</td>
<td>25</td>
<td>0.88</td>
<td>26.2</td>
<td>0.13</td>
</tr>
<tr>
<td>8</td>
<td>29</td>
<td>0.68</td>
<td>22.0</td>
<td>0.10</td>
</tr>
<tr>
<td>4L</td>
<td>24</td>
<td>0.76</td>
<td>21.9</td>
<td>0.21</td>
</tr>
<tr>
<td>2</td>
<td>30</td>
<td>0.06</td>
<td>21.3</td>
<td>0.11</td>
</tr>
<tr>
<td>3</td>
<td>67</td>
<td>0.56</td>
<td>18.2</td>
<td>0.15</td>
</tr>
<tr>
<td>4E</td>
<td>66</td>
<td>0.65</td>
<td>13.0</td>
<td>0.17</td>
</tr>
<tr>
<td>11</td>
<td>344</td>
<td>0.18</td>
<td>18.8</td>
<td>0.14</td>
</tr>
</tbody>
</table>
Figure 9-6. Regression curves relating eye irritation and simultaneous oxidant concentrations from a number of stations in the Los Angeles area.19

Figure 9-7. Variation of mean maximum eye irritation, as judged by a panel of "experts" with maximum oxidant concentrations, Pasadena, August-November, 1955.
employees of the Los Angeles Air Pollution Control District was queried during the period 1955-58. A group of environmental sanitation workers in the San Francisco Bay Area was also studied during the same period. Neither of these panels reported anything other than a tendency to experience increasing occurrence of eye irritation with increasing oxidant levels. As in all such studies, there were some individuals who reported eye irritation even when there was no oxidant present.

b. Student Nurse Study

Hammer et al. reported on respiratory and eye symptoms among two groups of student nurses studied during a 24-day period from October 29 through November 25, 1962, in Santa Barbara and Los Angeles. In general, the symptoms were more frequent among the Los Angeles students than among those in Santa Barbara. The choice of period for study was intended to obviate the effect of season or of major respiratory and infectious disease. The relationships between the mean frequency of symptom by day as measured by eye irritation, coughing, and sneezing, and the daily maximum oxidant level is shown for the Los Angeles students in Figure 9-8. Unfortunately, a complete set of oxidant measurements was not available. This accounts for the gap in the distribution shown. It is of interest that on 1 day in which an oxidant level of 450 µg/m³ (0.23 ppm) was observed in Santa Barbara, no unusual symptom frequency was reported by the students. Data plotted in Figure 9-8 again show a relationship between increasing eye irritation and maximum daily photochemical oxidant concentrations over the range of 200 to 880 µg/m³ (0.10 to 0.55 ppm).

c. Evaluation of Filters for Removing Eye Irritants from Polluted Air

A study was conducted to evaluate the sensory effectiveness of air-filter media for removing eye irritants from polluted air in downtown Los Angeles. Eye irritation in two groups of 20 female telephone company employees, similar with respect to age and job characteristics and employed in identical adjacent rooms, was evaluated each work-day (123 study days) from May to November of 1956. Active and dummy filter units were switched periodically between the two rooms so that the groups were alternately exposed to test and control conditions. The sensory response of the subjects was measured daily at 11:00 a.m. by means of a questionnaire; simultaneous measurements of oxidants, particulate matter, and nitrogen dioxide were obtained within each of the two rooms and immediately outside the building.
Figure 9-8. Relationship between oxidant concentrations and selected symptoms in Los Angeles, October 29 through November 25, 1962.21
The differences in eye irritation between the activated-carbon-filtered and non-filtered test situations were in all cases highly significant (Table 9-7). A statistically significant correlation between eye irritation and oxidant concentrations occurred in the non-filtered room (Table 9-8). The scatter diagram of results (see Figure 9-9) suggests an eye irritation threshold as the concentration of oxidants exceeded 200 \( \mu g/m^3 \) (0.10 ppm). The index of eye irritation for the study groups increased progressively as oxidant concentrations exceeded the 200 \( \mu g/m^3 \) (0.10 ppm) level.

Nitrogen dioxide concentrations were reduced by the activated-carbon filters during their early use but, after a period of time, nitrogen dioxide concentrations in the filtered atmosphere increased. No significant correlations between eye irritation and nitrogen dioxide levels were observed, nor were significant correlations found between eye irritation and concentrations of particulate matter.

d. Photochemical Oxidant and Eye Irritation in Locations Other Than California

Oxidant measurements at levels likely to be associated with eye irritation have been re-

<table>
<thead>
<tr>
<th>Test condition</th>
<th>Eye irritation index</th>
<th>Oxidants, pphm&lt;sup&gt;b&lt;/sup&gt;</th>
<th>NO&lt;sub&gt;2&lt;/sub&gt;, pphm</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.032&lt;sup&gt;a&lt;/sup&gt; Activated carbon filter</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean, non-filtered room</td>
<td>1.99</td>
<td>9.8</td>
<td>1.5</td>
</tr>
<tr>
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<td>1.01</td>
<td>0.49</td>
<td>0.41</td>
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<td>0.98</td>
<td>9.4</td>
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<td>&lt;&lt;0.01</td>
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</tr>
<tr>
<td>0.016 Activated carbon filter</td>
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<tr>
<td>Mean, non-filtered room</td>
<td>2.95</td>
<td>8.4</td>
<td>3.4</td>
</tr>
<tr>
<td>Mean, filtered room</td>
<td>1.41</td>
<td>1.8</td>
<td>1.6</td>
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<tr>
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<td>1.54</td>
<td>6.7</td>
<td>1.8</td>
</tr>
<tr>
<td>Probability that the difference could have occurred by chance</td>
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<td>&lt;&lt;0.01</td>
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<tr>
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<td></td>
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<tr>
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<td>13.9</td>
<td>2.7</td>
</tr>
<tr>
<td>Mean, filtered room</td>
<td>2.35</td>
<td>4.9</td>
<td>5.7</td>
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<tr>
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<td>9.0</td>
<td>3.0</td>
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<td>&lt;0.01</td>
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<td>7.3</td>
<td>4.7</td>
</tr>
<tr>
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<td>4.9</td>
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<td>Particulate filter</td>
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<td>Mean, non-filtered room</td>
<td>2.13</td>
<td>5.7</td>
<td>6.3</td>
</tr>
<tr>
<td>Mean, filtered room</td>
<td>1.91</td>
<td>3.4</td>
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<tr>
<td>Difference between means</td>
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<td>0.8</td>
</tr>
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<td>Probability that the difference could have occurred by chance</td>
<td>c</td>
<td>&lt;0.01</td>
<td>&lt;0.02</td>
</tr>
</tbody>
</table>

<sup>a</sup> Refers to air detention time in seconds
<sup>b</sup> Measured by the KI method.
<sup>c</sup> Difference not significant.
ported from a number of other cities (Chapter 3). Circumstantial evidence of increased eye irritation has been reported in Washington, D.C., Denver, New York City, and St. Louis. An epidemiologic study of eye irritation was carried out by McCarron et al. on a population living in midtown Manhattan. The investigators established a system of weekly health reports by families, based on the presence or absence of certain symptoms. In October 1963, there were substantial increases in the frequency of new reports of eye irritation (increasing from about 2 to nearly 5 percent of the population). Oxidant measurements, made at some distance away, had increased during the period under study. Unfortunately, clear conclusions from these data cannot be drawn; there were high levels of sulfur oxide pollution, of particulate pollution, and of carbon monoxide. It is quite possible that eye irritation symptoms in New York City result from mixed pollution of both the oxidizing and reducing type.

e. Discussion

From these data, it can be only concluded that eye irritation in relation to increased

![Graph showing mean index of eye irritation versus oxidant concentration.](image)

Figure 9-9. Mean index of eye irritation versus oxidant concentration.22
levels of air pollution can occur elsewhere than in California. It is of great importance that this impression is documented and quantified by additional panel studies.

Several studies in California have shown that the incidence of eye irritation increases progressively when the ambient oxidant levels exceed 200 μg/m³ (0.10 ppm). Using the information presented in Chapter 3, section B.1, the instantaneous value of 200 μg/m³ (0.10 ppm) oxidant, which is related to eye irritation, might be expected to be associated with a maximum hourly average oxidant concentration of 50 to 100 μg/m³ (0.025 to 0.50 ppm), depending on localized conditions. Eye irritation in a group of subjects in downtown Los Angeles was diminished significantly in a room with an activated-carbon air-filter when compared simultaneously with results obtained in a non-filtered room.

When interpreting implied relationships associating eye irritation and ambient oxidant levels, care must be exercised in conclusions regarding cause and effect. Experimental studies have shown that ozone, the principal contributer to ambient oxidant levels, is not an eye irritant, as discussed in Chapter 8, Section E. Peroxyacyl nitrates have been shown to be powerful eye irritants; even more irritating is peroxybenzoyl nitrate. Formaldehyde and acrolein, also products of the photochemical system, have been shown to produce eye irritation. A postulated explanation for the relationship between ambient oxidant levels and eye irritation is that “oxidant” is a measure of the photochemical activity which produces the aforementioned eye irritants.

C. CHRONIC EFFECTS OF PHOTOCHEMICAL OXIDANTS

To define the chronic effects of prolonged exposure to photochemical oxidant pollution, investigators have contrasted health characteristics of populations living in clean and polluted communities. Results from these studies must be interpreted with caution, for the populations of two communities are often different in many respects other than air pollution exposure. In carefully designed epidemiologic studies, the principal determinants of the disease condition being investigated are identified, and population samples from each study area are matched on these determinants. Thus, information on socio-economic status, climate, cigarette

Table 9-8. PEARSON PRODUCT-MOMENT CORRELATION COEFFICIENTS BETWEEN EYE IRRITATION AND ENVIRONMENTAL FACTORS IN A NONFILTERED ROOM

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<td>Oxidants concentration, by</td>
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<td></td>
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<td>Irritation</td>
<td>Particulate</td>
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<td>Temperature</td>
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<td>vs Oxidants concentration,</td>
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<td>by Phenolphtalein</td>
<td>by KI method</td>
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<td>method</td>
<td>NO₂ concentrations</td>
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<tr>
<td></td>
<td>vs Oxidants concentration,</td>
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<tr>
<td></td>
<td>by KI method</td>
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</tbody>
</table>
smoking habits, and occupation must usually be obtained in two-community air pollution studies, and the similarity in these characteristics of the study population must be documented. When differences other than air pollution exposure are present in the study areas, demonstration of the same air pollution—health effect association in studies of other communities is usually required before the association can be firmly accepted.

In general, epidemiologic identification of the chronic effects of air pollution requires replicated studies of large, well-defined populations over relatively long periods of times by competent investigators producing consistent results. These requirements are not adequately met in this section. Although the results of the reported studies are therefore only suggestive, they are an essential part of air quality criteria because they can caution the public about hazardous substances in the atmosphere. To prove or disprove the existence of a long-term effect may require an excessively long or hazardous period of exposure and measurement. Studies which strongly suggest that a pollutant may be a health hazard are sufficient to warrant preventive action until such time as the pollutant—health effect association is convincingly proved or disproved.

1. Mortality in Areas of High and Low Oxidant Pollution

a. Lung Cancer Mortality

It is known that active chemical carcinogens are found in polluted atmospheres and that ozone, a principal component of photochemical oxidant pollution, has radiomimetic properties. Beginning in 1957, a prospective study of lung cancer among 69,160 members of the California Department of the American Legion was undertaken. This represented between 50 and 60 percent of the membership mailing list of eligible individuals. This population under-
b. Chronic Respiratory Disease Mortality

Mortality rates for other chronic respiratory diseases as reported in the above American Legion Study\textsuperscript{2,6} were somewhat higher in Los Angeles than in San Francisco and San Diego Counties, particularly among persons resident for 10 or more years in their respective counties (Table 9-11). Studies by Winklestein\textsuperscript{7,8} et al., however, have demonstrated the strong effect of socioeconomic level on chronic respiratory disease mortality. For this reason, the reported relationship between chronic respiratory disease mortality and area of residence in California should be considered as tenuous until the possible effect of differences in socioeconomic level among

| Table 9-9. TOTAL LUNG CANCER MORTALITY IN AN AMERICAN LEGION STUDY |
| POPULATION, CALIFORNIA, 1958–1962\textsuperscript{26} |
| |
| | Los Angeles County | San Francisco Bay Area and San Diego Counties | All other Calif. counties |
| | Mortality rate\textsuperscript{a} | Total deaths | Mortality rate\textsuperscript{a} | Total deaths | Mortality rate\textsuperscript{a} | Total deaths |
| Age-adjusted\textsuperscript{b} | 95.9 | n/a | 104.5 | n/a | 75.3 | n/a |
| Age-smoking adjusted | 95.4 | n/a | 102.0 | n/a | 75.5 | n/a |
| Resident\textsuperscript{c} | | | | | | |
| At least 10 yr | 96.6 | 79 | 106.3 | 58 | 79.9 | 69 |
| Less than 10 yr | 76.7 | 27 | 69.1 | 13 | 68.5 | 30 |
| Unknown | 123.4 | 12 | 215.3 | 10 | 65.2 | 6 |

\textsuperscript{a} Deaths per 100,000 man-years.
\textsuperscript{b} Age-adjusted by the direct method to the total study population.
\textsuperscript{c} Age and smoking adjusted.

| Table 9-10. LUNG CANCER DEATHS AND RELATIVE RISKS PER 100,000 MAN-YEARS |
| OF AN AMERICAN LEGION STUDY POPULATION, BY EXTENT OF CIGARETTE SMOKING |
| AND RESIDENCE, CALIFORNIA, 1958–1962\textsuperscript{26} |
| |
| | Los Angeles County | S.F. Bay Area and San Diego Counties | All other counties\textsuperscript{b} |
| | Cigarette smoking lifetime history\textsuperscript{a} | Rate | Relative risk | Rate | Relative risk | Rate | Relative risk |
| None | 28.1 | 2.5 | 43.9 | 3.9 | 11.2 | 1.0 |
| Less than one pack | 63.6 | 5.7 | 77.1 | 6.9 | 61.0 | 5.4 |
| About one pack | 126.0 | 11.3 | 134.5 | 12.0 | 124.9 | 11.2 |
| More than one pack | 241.3 | 21.5 | 226.0 | 20.2 | 137.5 | 12.3 |

\textsuperscript{a} Age-adjusted by the direct method to the total study population.
\textsuperscript{b} Nonsmokers in all other counties taken as unit risk.
Table 9-11. TOTAL CHRONIC RESPIRATORY DISEASE MORTALITY IN AN AMERICAN LEGION STUDY POPULATION, CALIFORNIA, 1958-1962a

<table>
<thead>
<tr>
<th>Residency</th>
<th>Los Angeles County</th>
<th>San Francisco Bay Area and San Diego Counties</th>
<th>All other Counties</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mortality rateb</td>
<td>Total deaths</td>
<td>Mortality rateb</td>
</tr>
<tr>
<td>Resident 10 yrs.</td>
<td>38.4</td>
<td>31</td>
<td>28.3</td>
</tr>
<tr>
<td>Resident less than 10 yrs.</td>
<td>41.2</td>
<td>14</td>
<td>45.6</td>
</tr>
<tr>
<td>Unknown</td>
<td>139.1</td>
<td>12</td>
<td>59.8</td>
</tr>
<tr>
<td>TOTAL</td>
<td>46.7</td>
<td>57</td>
<td>34.0</td>
</tr>
</tbody>
</table>

a Age and smoking adjusted by the direct method to the total study population.
b Per 100,000 man-years.

discussion

No obvious effect of prolonged exposure to photochemical oxidants on lung cancer mortality rates was evident. A relationship between chronic respiratory disease mortality and photochemical oxidant pollution was suggested in an isolated study in which data on other important variables were not obtained. A considerable amount of further epidemiologic study is needed to test this association.

2. General Morbidity in Areas of High and Low Oxidant Pollution

a. State of California Health Survey

In 1954, weekly studies were initiated to estimate the total morbidity among the general population of California throughout the year.1 In addition, the population aged 65 years and over was studied separately. As shown in Figures 9-10 and 9-11, a significant association of morbidity with periods of high air pollution within Los Angeles County was lacking, even when selected conditions (colds, asthma, hay fever, and respiratory conditions) were considered. The data in Figure 9-10, however, show that the incidence of illness and injury is consistently greater for the population age 65 years and older in Los Angeles than in the remainder of California. The average weekly incidence was about 7 percent for the non-Los Angeles population and about 10.4 percent for the Los Angeles population, approximately 50 percent greater. These data are only suggestive of an air pollution effect, since differences in population density, race, socioeconomic level and other important factors are known to exist between Los Angeles and the remainder of California.

A general health survey was again undertaken in 1956, with one goal being the evaluation of photochemical air pollution in Los Angeles, in the San Francisco Bay Area, and in the rest of the state.29 Because of the widespread public interest in air pollution, special precautions were taken to emphasize that this study was undertaken as a general health survey. Special efforts were made to prevent the household interviewers from suggesting the air pollution objective of the study to the subject. The study was designed to explore the following four questions:

1. How extensive is the air pollution problem in Los Angeles in relation to the rest of the state?
2. Does air pollution affect a few people much of the time and many people occasionally?
3. Is air pollution a serious source of discomfort?
4. Does air pollution cause dissatisfaction with living conditions in residential communities or in places of employment?

A probability sample of 3,545 households was selected as representative of all the people in the state, in the ratio of 1 to 1,055. The only populations not included were those in institutions and service camps. Interviewing took place during May and June of 1956 on the basis of a sample designed by the United States Census Bureau. In each household sampled, one adult was randomly chosen and predesignated for a personal interview. These sub-samples were appropriately weighted.

From the survey data presented in Table 9-12, it can be seen that asthma, cough, and nose and throat complaints were somewhat more frequent in Los Angeles, Orange, and San Diego Counties than in the San Francisco Bay area counties or the rest of the state. Differences, though statistically significant, were small. Bronchitis was reported by an equal proportion of sampled persons in the Los Angeles area and in the rest of the state. Sinus conditions and hay fever were most common in the rest of the state. Those persons who admitted to suffering from chronic conditions during the previous year were asked whether they felt worse on some days and, if so, why. The interviewers made no mention of smog or air pollution; the answers which referred to air pollution were considered spontaneous. In Figure 9-12, the responses obtained from residents of Los Angeles County and the San Francisco Bay area are compared. The proportion of the population who attributed a worsening of

![Graph showing rate of illness and injury per 100 persons by week ending August 2 to November 28, 1954.](image)

**Figure 9-10.** Relationship of high smog periods to incidence of illness and injury, persons 65 years and over, August 2 - November 28, 1954.
Figure 9-11. Relationship of high smog periods to incidence of selected conditions for persons of all ages, August 2 - November 28, 1954.¹

Table 9-12. SELECTED RESPIRATORY CONDITIONS REPORTED BY GENERAL POPULATION SAMPLE, CALIFORNIA, MAY 1956²³

<table>
<thead>
<tr>
<th>Conditions reported</th>
<th>California</th>
<th>Los Angeles, Orange, and San Diego Counties</th>
<th>San Francisco Bay Area Counties³</th>
<th>Rest of State</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Frequency</td>
<td>Frequency</td>
<td>Percent</td>
<td>Frequency</td>
</tr>
<tr>
<td>Bronchitis</td>
<td>309</td>
<td>156</td>
<td>5</td>
<td>71</td>
</tr>
<tr>
<td>Asthma</td>
<td>188</td>
<td>104</td>
<td>3</td>
<td>45</td>
</tr>
<tr>
<td>Cough</td>
<td>1,341</td>
<td>746</td>
<td>22</td>
<td>323</td>
</tr>
<tr>
<td>Sinus</td>
<td>1,202</td>
<td>576</td>
<td>17</td>
<td>302</td>
</tr>
<tr>
<td>Hayfever</td>
<td>695</td>
<td>265</td>
<td>8</td>
<td>221</td>
</tr>
<tr>
<td>Nose complaints</td>
<td>751</td>
<td>445</td>
<td>13</td>
<td>186</td>
</tr>
<tr>
<td>Throat complaints</td>
<td>848</td>
<td>505</td>
<td>15</td>
<td>192</td>
</tr>
<tr>
<td>Number of persons interviewed²</td>
<td>6,939</td>
<td>3,450</td>
<td>100</td>
<td>1,846</td>
</tr>
</tbody>
</table>


³Percentages will not add to 100 due to reports of multiple conditions or of no conditions.

³The general sample of 6,939 persons was drawn from the State's civilian, non-institutional population, age 18 years or older.
panel was limited to those age 30 years and older. They were reinterviewed on four occasions, twice in 1957, once in 1958, and once in 1959. Data were obtained as to the relative severity and the consequences of illness from respiratory conditions. No patterns of mortality of changes in morbidity were discovered which would indicate significant differences in diverse areas of the state and hence might be attributed to exposure to air pollution.

b. Chronic Respiratory Disease Survey of Telephone Workers

In comparative studies on outdoor telephone workers, Deane et al., used standardized respiratory survey techniques to study a group of West Coast workmen whose general occupation, medical status, and social status were similar to workmen on the East Coast and in the United Kingdom. In the older group (age 50-59 years), respiratory symptoms were more frequent in the Los Angeles than in the San Francisco population. Persistent cough and phlegm in the 50-59 year age-group were reported by 31.4 percent of the group in Los Angeles, compared with 16.3 percent in San Francisco. The differences between these two groups could not be accounted for by differences in smoking habits. There were not important differences in the results of pulmonary function tests. In the younger group (age 40-49 years), the frequencies of various respiratory symptoms were not consistently different between Los Angeles and San Francisco residents.

Questions regarding eye irritation were also included. The 40-49 age group in San Francisco reported eye irritation about 10 percent of the time; the 50-59 age group, about 4 percent. The comparable figures for Los Angeles were 30 percent and 29 percent respectively. Perhaps more important is the fact that over 50 percent of those in both age groups in San Francisco had never experienced eye irritation, while the corresponding figure for Los Angeles was less than 10 percent.
c. Discussion

Several surveys suggest a higher incidence of both chronic respiratory disease symptoms and of other symptoms, including asthma and nose and throat complaints, among residents of Los Angeles than in other areas of California, including the San Francisco Bay area.

3. Effects of Photochemical Oxidant Pollution on Community Satisfaction

a. State of California General Health Survey

At the end of the general health survey undertaken in California in 1956 and previously described,\(^2\)\(^9\) direct questions concerning the effects of air pollution were asked. Seventy-five percent of the surveyed population from Los Angeles County was "bothered" by air pollution, in contrast to 24 percent in the San Francisco Bay area, and 22 percent in the rest of the state. These contrasts were also reported for the working populations from these areas (Table 9-13). Of the total number who were bothered by air pollution, 17 percent in Los Angeles considered moving because of it, in contrast to 4 percent in San Francisco. Of the same total number, 9 percent in Los Angeles considered changing jobs because of air pollution, in contrast to 3 percent in San Francisco (Table 9-14). About 20 percent of the state's residents who had moved out of an air-polluted area said the pollution had some influence on their decision to move; 4 percent gave air pollution as their sole reason for moving. Among those who had moved from California communities because of air pollution, 75 percent had moved out of Los Angeles County, 8 percent had moved out of the San Francisco Bay area, and 17 percent had moved out of other areas of the state. The reasons given for all of these moves are listed in Table 9-15. Air pollution was given as the

<table>
<thead>
<tr>
<th>Table 9-13. PERCENT OF SURVEY RESPONSES OF GENERAL AND WORKING POPULATIONS &quot;BOtherED&quot; BY AIR POLLUTION, BY MAJOR GEOGRAPHIC AREAS IN CALIFORNIA, MAY 1956(^2)(^9)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Responses</strong></td>
</tr>
<tr>
<td>--------------------------------</td>
</tr>
<tr>
<td>General population sample</td>
</tr>
<tr>
<td><strong>Percent</strong></td>
</tr>
<tr>
<td>Not bothered by air pollution</td>
</tr>
<tr>
<td>Bothered by air pollution</td>
</tr>
<tr>
<td>Either at home or work</td>
</tr>
<tr>
<td>Both at home and at work</td>
</tr>
<tr>
<td>At home only</td>
</tr>
<tr>
<td>At work only</td>
</tr>
<tr>
<td>Total at home</td>
</tr>
<tr>
<td>Total at work</td>
</tr>
<tr>
<td>Working Population Sample</td>
</tr>
<tr>
<td><strong>Percent</strong></td>
</tr>
<tr>
<td>Not bothered by air pollution</td>
</tr>
<tr>
<td>Bothered by air pollution</td>
</tr>
<tr>
<td>Either at home or work</td>
</tr>
<tr>
<td>Both at home and at work</td>
</tr>
<tr>
<td>At home only</td>
</tr>
<tr>
<td>At work only</td>
</tr>
<tr>
<td>Total at home</td>
</tr>
<tr>
<td>Total at work</td>
</tr>
</tbody>
</table>

\(^{a}\)Percent are rounded independently.
Table 9-14. PERCENT OF SURVEY RESPONSES OF GENERAL AND WORKING POPULATIONS, “BOtherED” BY AIR POLLUTION, WHO HAVE CONSIDERED MOVING OR CHANGING THEIR JOBS FOR THIS REASON, BY MAJOR GEOGRAPHIC AREAS IN CALIFORNIA, MAY 1956<sup>29</sup>

<table>
<thead>
<tr>
<th>Responses</th>
<th>California</th>
<th>Los Angeles County</th>
<th>San Francisco Bay Area</th>
<th>Rest of State</th>
</tr>
</thead>
<tbody>
<tr>
<td>General population sample, bothered by air pollution</td>
<td>2,616</td>
<td>1,904</td>
<td>326</td>
<td>386</td>
</tr>
<tr>
<td>Persons bothered, percent</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Have given serious consideration to moving</td>
<td>15</td>
<td>17</td>
<td>4</td>
<td>12</td>
</tr>
<tr>
<td>Have not given serious consideration to moving</td>
<td>85</td>
<td>83</td>
<td>96</td>
<td>88</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Working population, sample workers bothered by air pollution at work</td>
<td>1,410</td>
<td>1,012</td>
<td>190</td>
<td>208</td>
</tr>
<tr>
<td>Persons bothered, percent</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Have given serious consideration to changing jobs</td>
<td>8</td>
<td>9</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Have not given serious consideration to changing jobs</td>
<td>92</td>
<td>91</td>
<td>97</td>
<td>97</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>

Table 9-15. REASONS FOR MOVING FROM THREE AREAS OF CALIFORNIA, IN RESPONSE TO GENERAL POPULATION SURVEY, MAY 1956<sup>29</sup>

<table>
<thead>
<tr>
<th>Reason for moving from area</th>
<th>Los Angeles County, percent</th>
<th>San Francisco Bay Area, percent</th>
<th>Rest of State, percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Financial</td>
<td>62</td>
<td>51</td>
<td>52</td>
</tr>
<tr>
<td>Personal</td>
<td>32</td>
<td>27</td>
<td>32</td>
</tr>
<tr>
<td>Health</td>
<td>4</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Air Pollution</td>
<td>2</td>
<td>13</td>
<td>10</td>
</tr>
<tr>
<td>Weather</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total percent</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>
reason for 13 percent of the moves from Los Angeles County since 1947, compared with 2 percent prior to 1947. For other areas of the state, the proportion of moves attributed to air pollution was negligible.

Eye irritation appeared to be the most frequently reported effect of air pollution; in some instances, this symptom was accompanied by nasal irritation (Table 9-16). About 20 percent of the respondents expressed a dissatisfaction with the communities in which they lived. Air pollution had not been mentioned at this point in the interview. The reasons for community dissatisfaction are shown in Table 9-17. A far greater proportion of dissatisfied Los Angeles residents (32 percent) attributed their dissatisfaction to air pollution than residents of the San Francisco Bay area (1 percent) or for the rest of the state (6 percent).

b. Survey of Los Angeles Physicians

A joint committee of the Los Angeles County Medical Association and the Tuber-
culosis and Health Association of Los Angeles County carried out a survey of Los Angeles physicians in December, 1960. A 1-in-16 sample of the physicians registered in practice in the county during 1958 was drawn, resulting in a sample of 526 from a total of 9,228 physicians. A pretested questionnaire was mailed with a letter signed by the chairman of the air pollution subcommittee. A followup was also mailed, and telephone calls were made to the offices of those physicians who had not responded.

Three-hundred fifty of the questionnaires were returned. Of those, 307 (58 percent) were completed and tabulated. The words "air pollution" did not appear in the questionnaire, although bias could have been introduced by the fact that the chairman of the air pollution subcommittee attached a letter to the questionnaire. Seventy-seven percent of the physicians believed that air pollution adversely affected the health of their patients. Two-thirds of the responding physicians felt that air pollution was a factor adversely

| Table 9-16. AIR POLLUTION EFFECTS REPORTED IN GENERAL POPULATION SURVEY, BY TYPE OF COMMUNITY AND BY MAJOR GEOGRAPHIC AREAS IN CALIFORNIA, MAY 1956 |
|-------------------------------------------------|----------------|----------------|----------------|----------------|----------------|
| Air pollution effects                          | California     | Los Angeles    | San Francisco  | Rest of State  |               |
|                                                | At home | At work | At home | At work | At home | At work | At home | At work |               |
| General population sample                      | 6,939  | 3,732  | 2,892  | 1,577  | 1,846  | 1,028  | 2,201  | 1,127  |               |
| Respondents bothered by air pollution          | 2,616  | 1,410  | 1,904  | 1,012  | 326    | 190    | 386    | 208    |               |
| Percent bothered by air pollution             | 38     | 37     | 66     | 64     | 18     | 18     | 17     | 19     |               |
| Air pollution effects cited:                   |          |         |         |         |         |         |         |         |               |
| Eyes, effects                                  | 75     | 76     | 89     | 88     | 38     | 39     | 41     | 51     |               |
| Eye irritation                                 | 44     | 46     | 54     | 53     | 17     | 18     | 23     | 30     |               |
| Eye and nasal irritation                       | 23     | 24     | 26     | 27     | 15     | 14     | 14     | 19     |               |
| Eye irritation and annoying                    | 5      | 3      | 6      | 4      | 4      | 3      | 3      | 2      |               |
| Eye, nasal irritation, and annoying           | 3      | 3      | 3      | 4      | 2      | 4      | 2      | 1      |               |
| Nasal irritation, eye not mentioned            | 10     | 9      | 5      | 4      | 22     | 23     | 22     | 21     |               |
| Nasal irritation                              | 8      | 8      | 4      | 3      | 19     | 19     | 18     | 21     |               |
| Nasal irritation and annoying                  | 2      | 1      | 1      | 1      | 3      | 4      | 4      | 4      |               |
| Annoying only                                  | 5      | 7      | 2      | 3      | 17     | 26     | 10     | 8      |               |
| Other effects, only                           | 5      | 2      | 2      | 1      | 5      | 5      | 17     | 4      |               |
| No effects reported                           | 5      | 6      | 2      | 4      | 18     | 7      | 10     | 16     |               |
| Total percent                                 | 100    | 100    | 100    | 100    | 100    | 100    | 100    | 100    |               |

9-29
affecting chronic respiratory disease. One-third of the physicians had advised one or more of their patients to leave the Los Angeles area for health reasons; air pollution was a factor mentioned in two-thirds of these instances. By extrapolation from the sample, assuming it to be representative, it was estimated that physicians had advised over 10,000 patients to move; it was reported that approximately 25 percent of the patients had done so. Nearly one-third of the physicians had themselves considered moving from the Los Angeles area because of air pollution. Among other environmental factors mentioned were overcrowding and traffic congestion, but these were of very small magnitude in relation to the reported difficulties with air pollution.

c. Discussion

A significantly large proportion of Los Angeles residents were subjectively bothered by air pollution when compared with residents of the San Francisco Bay area and the rest of the state. A larger proportion of residents who were so bothered had considered moving or had moved from Los Angeles than residents of other areas of California.

Eye irritation, at times accompanied by nasal irritation, was the most frequently reported nuisance effect of air pollution, according to the California Health Survey.

One-third of sampled physicians in the Los Angeles area advised one or more of their patients to leave the area for health reasons, and nearly one-third of the physicians had themselves considered moving from the Los Angeles area because of air pollution.

D. SUMMARY

Epidemiologic studies have been conducted relating photochemical air pollution with mortality, hospital admissions, aggravation of respiratory diseases, impairment of human performance, and eye irritation. The effects of prolonged oxidant exposure on mortality, morbidity, ventilatory function, and community satisfaction have also been studied.

1. Review of Results from Cited Studies

No convincing relationship was observed between short-term variations in photochemical oxidants and (1) daily mortality or (2) hospital admissions.

A study of 137 patients with asthma demonstrated significantly more asthma attacks on days when photochemical oxidant concentrations exceeded 250 μg/m³ (0.13 ppm). Such a peak oxidant value might be expected to be associated with a maximum hourly average oxidant concentration of 100 to 120 μg/m³ (0.05 to 0.06 ppm), depending on localized conditions.

Chronic respiratory disease patients removed from an ambient atmosphere of elevated oxidant concentrations to a room from which pollutants were filtered have shown improvement in ventilatory function. In two other studies, no significant association was found between variations in ambient oxidant levels and changes in respiratory symptoms or function in patients with chronic respiratory disease.

The team performance of high school cross-country track runners was impaired on days of elevated oxidant concentrations mea-
sured 1 hour before the commencement of each race; hourly oxidant concentrations ranged from 60 to 590 μg/m³ (0.3 to 0.30 ppm), although no threshold for this effect can be determined from the available data. Significantly more automobile accidents have also occurred on days of high oxidant concentrations.

Eye irritation appears to increase progressively when oxidant concentrations exceed 200 μg/m³ (0.10 ppm). This oxidant value, related to eye irritation might be expected to be associated with a maximum hourly average oxidant concentration of 50 to 100 μg/m³ (0.025 to 0.50 ppm), depending on localized conditions. Eye irritation, at times accompanied by nasal irritation, was the most frequently reported nuisance effect of air pollution in California. A postulated explanation for the relationship between ambient oxidant levels and eye irritation is that the level of oxidants is a measure of the photochemical activity which produced the eye irritants.

Lung cancer mortality rates were similar among California residents studied in both high-and low-oxidant pollution areas. A relationship between noncancerous chronic respiratory disease mortality and long-term photochemical oxidant exposure has been suggested in an isolated study in which other important variables were not analyzed. Factors other than oxidant exposure could well have accounted for these observations, and considerable documentation from other epidemiologic studies is required to substantiate these findings. Several surveys have also reported a higher incidence of both chronic respiratory disease symptoms and of other respiratory symptoms, including asthma and nose and throat complaints, among residents of Los Angeles than in other areas of California.

A significantly larger proportion of Los Angeles residents have been subjectively bothered by air pollution than residents of the San Francisco Bay area and the rest of the state. A larger proportion of residents who were bothered by air pollution have considered moving or have moved from Los Angeles than residents of other areas of California. One-third of the physicians sampled in the Los Angeles area had advised one or more of their patients to leave the area for health reasons, and nearly one-third of the physicians had themselves considered moving from Los Angeles because of air pollution.

2. Future Research Needs

Relatively scanty information has been gathered on community health effects of photochemical oxidants. The relatively localized nature of the oxidant problem accounts for this gap in knowledge. Data reported in this chapter need to be augmented, particularly with regard to lung cancer mortality and the prevalence of chronic respiratory disease. The known irritant potential of ozone, a major component of photochemical pollution, should be explored to establish more exactly how exacerbations of illness in subjects with asthma and chronic bronchitis on days of elevated oxidant concentrations may be related to the presence of ozone. Studies to date suggest that this portion of the population may be more sensitive to oxidant variations. Studies of the acute effects of oxidant pollution could be further refined by careful selection of comparison communities in which differences in exposure to photochemical oxidant levels are maximized. Both chronic and acute effects associated with air pollution exposure can be identified more readily through such studies.

Systemic effects of oxidant exposure were suggested in the athletic performance study and in the study on accident frequency. Replication and extension of these studies are clearly indicated. Physiologic and psychomotor tests to elucidate pathological mechanisms for these effects would add important confirmatory evidence and point the way to the development of dose-response relationships.

Well-designed prevalence studies in clean and polluted communities would provide useful quantitative data on the prevalence of chronic respiratory disease, heart disease, and
possibly chronic eye pathology in relation to prolonged oxidant exposure. The effect of oxidant exposure on growth and development in the first years of life is a clearly feasible prospective study. Such a study may provide important information on the action of this pollutant during a period of life when man is highly sensitive to environmental influences.

The impairment by oxidant pollution of immune mechanisms of response to infectious agents, suggested in animal studies, has not been explored in community studies. Classical epidemiologic and laboratory methods to study acute respiratory illness rates in populations could be applied profitably to the photochemical oxidant problem.

3. Discussion

Accumulated studies reviewed in this chapter reveal an inadequacy of epidemiologic information on the health effects of photochemical oxidant pollution. Consistent results for some effects, obtained by various investigators under varying conditions of exposure, are lacking. The few demonstrated associations between oxidant exposure and health effects, such as asthma, pulmonary function, or athletic performance, are inadequate to establish minimum threshold levels for each effect.

Reported studies do suggest, however, that photochemical oxidants are potentially hazardous environmental contaminants. Subjects with chronic respiratory disease seem to be the most threatened by such exposure, but virtually all segments of the population may experience eye irritation at levels of oxidants frequently present in the ambient air. Hazards to normal respiratory function, optimum athletic performance, and safe automobile driving have also been suggested. An association, although not necessarily a cause-effect relationship, has been shown between ambient levels of photochemical oxidants and eye irritation. Since one of the objectives of air pollution control is to promote good health and minimize exposures to potentially hazardous pollutants, the information provided by reported studies can not be discounted.

E. REFERENCES


Chapter 10.

SUMMARY AND CONCLUSIONS

A. INTRODUCTION

This document is a consolidation and assessment of the current state of knowledge on the origin and effects of the group of air pollutants known as photochemical oxidants on health, vegetation, and materials. The purpose of this chapter is to provide a concise picture of the information contained in this document, including conclusions which are believed reasonable to consider in evaluating concentrations of photochemical oxidants which are known to have an effect on either health or welfare. Although nitrogen dioxide is considered one of the photochemical oxidants, it is to be subject of a separate report. Consequently, nitrogen dioxide is discussed in this document only to the extent that it participates in the formation and reactions of other photochemical oxidants. The information and data contained in this document comprise the best available bases, and provide the rationale for development of specific levels of standards of photochemical oxidants in the ambient air for protection of public health and man’s environment.

B. NATURE OF PHOTOCHEMICAL OXIDANTS

Photochemical oxidants result from a complex series of atmospheric reactions initiated by sunlight. When reactive organic substances and nitrogen oxides accumulate in the atmosphere and are exposed to the ultraviolet component of sunlight, the formation of new compounds, including ozone and peroxyacyl nitrates, takes place.

Absorption of ultraviolet light energy by nitrogen dioxide results in its dissociation into nitric oxide and an oxygen atom. These oxygen atoms for the most part react with air oxygen to form ozone. A small portion of the oxygen atoms and ozone react also with certain hydrocarbons to form free radical intermediates and various products. In some complex manner, the free radical intermediates and ozone react with the nitric oxide produced initially. One result of these reactions is the very rapid oxidation of the nitric oxide to nitrogen dioxide and an increased concentration of ozone.

The photochemical system generally is capable of duplication in the laboratory. For various reasons, however, laboratory results cannot be quantitatively extrapolated to the atmosphere. Theoretically generation of an atmospheric simulation model should be feasible, enabling the prediction of ambient oxidant concentrations from a knowledge of emission and meteorological data. The development of such a model, however, is dependent on the acquisition of more reliable and applicable quantitative information derived from direct atmospheric observations, as well as on the refinement of results obtained from irradiation chamber studies.

C. ATMOSPHERIC PHOTOCHEMICAL OXIDANT CONCENTRATIONS

The presence of photochemically formed oxidants has been indicated in all of the major U.S. cities for which aerometric data have been examined. On a concentration basis, ozone has been identified as the major component of the oxidant levels observed. Difficulties arise, however, in interpreting data obtained by the most commonly used oxidant measuring method; this method is nonspecific and subject to several interferences. Adjusted oxidant concentrations, obtained by correcting potassium iodide oxidant measurements
for known interferences, have been found to be relatively close to concurrent measurements of ozone alone.

Since photochemical oxidants are the products of atmospheric chemical reactions, the relationship between precursor emissions and atmospheric oxidant concentrations is much less direct than is the case for primary pollutants. A further complicating situation is the dependence of these photochemical reactions on intensity and duration of sunlight, and on temperature.

In an analysis of oxidant concentration data for 4 years and 12 stations, the daily maximum 1-hour average concentration was equal to or exceeded 290 µg/m³ (0.15 ppm) up to 41 percent of the time; maximum 1-hour average concentrations ranged from 250 to 1,140 µg/m³ (0.13 to 0.58 ppm); short-term peaks were as high as 1,310 µg/m³ (0.67 ppm). Yearly averages, commonly applied to other pollutants, are not representative of air quality with respect to oxidant pollution, because 1-hour average ozone concentrations will necessarily be at or about zero for approximately 75 percent of the time when photochemical reactions are minimal.

Peroxyacyl nitrates, through not routinely measured, have been identified in the atmosphere of several cities. These compounds may be assumed to be present whenever oxidant levels are elevated.

D. NATURAL SOURCES OF OZONE

Ozone can be formed naturally in the atmosphere by electrical discharge, and in the stratosphere by solar radiation, by processes which are not capable of producing significant urban concentrations of this pollutant. Maximum instantaneous ozone levels of from 20 to 100 µg/m³ (0.01 to 0.05 ppm) have been recorded in nonurban areas.

E. MEASUREMENT OF PHOTOCHEMICAL OXIDANTS

The most widely used technique for the analysis of atmospheric total oxidants is based on the reaction of these compounds with potassium iodide to release iodine. The iodine may then be measured by either colorimetric or coulometric methods. Calibrating the oxidant measurement method used against a known quantity of ozone provides a measurement of the net oxidizing properties of the atmosphere in terms of an equivalent concentration of ozone. Most oxidant measurements are currently being made by the colorimetric method, although coulometric analyzers are used in a number of laboratory and field studies.

In order to generate comparable data, it is essential that all measurements be made by techniques which have been calibrated against the same standard or reference method. Since at the present time there is no standard method for the determination of total oxidants, the National Air Pollution Control Administration recommends use of the neutral-buffered 1 percent potassium iodide colorimetric technique as the method against which all instruments and other methods should be compared. In addition to serving as a manual procedure for determining oxidants, the reference method may be used in conjunction with a “dynamic calibration” technique for instrumental methods.

Reducing agents such as sulfur dioxide produce a negative interference in oxidant determination. Such interference can be reduced, however, by passing the air stream through a chromium trioxide scrubber prior to measurement. Unfortunately, a portion of the nitric oxide which may be present in the air stream is oxidized to nitrogen dioxide by the scrubber. This results in an apparent increase in the oxidant measurement of about 11 percent of the concentration of nitric oxide. Moreover, a portion of the atmospheric nitrogen dioxide concentration will also contribute to the oxidant measurement. Peroxyacyl nitrate concentrations are usually small and contribute only a very slight amount to the oxidant reading.

There are several means for the specific measurement of atmospheric ozone. Instrumental methods include chemiluminescent analysis based on the reaction of ozone with Rhodamine B, gas phase olefin titration, and
ultraviolet and infrared spectroscopy. A semi-quantitative method for ozone measurement is based on its ability to produce cracks in stretched rubber. Peroxyacyl nitrates can be measured in the atmosphere by gas chromatography with the use of an electron-capture detector.

For a better evaluation of the results of research on the effects of photochemical oxidants, it is essential that data be obtained for individual oxidants such as nitrogen dioxide, ozone, PAN, formaldehyde, acrolein, and organic peroxides. These data would either replace or complement data on total oxidants. Instrumentation currently available permits the accurate measurement of atmospheric ozone, nitrogen dioxide, and PAN. There exists, however, a further need to develop instruments capable of measuring other individual gaseous pollutants which have the properties of oxidants. Photochemical reactions and problems derived from oxidants can be much better defined using specific methods for measurement in preference to the traditional total oxidants determination.

F. EFFECTS OF PHOTOCHEMICAL OXIDANTS ON VEGETATION AND MICROORGANISMS

Injury to vegetation is one of the earliest manifestations of photochemical air pollution, and sensitive plants are useful biological indicators of this type of pollution. The visible symptoms of photochemical oxidant produced injury to plants may be classified as: (1) acute injury, identified by cell collapse with subsequent development of necrotic patterns; (2) chronic injury, identified by necrotic patterns with or without chlorotic or other pigmented patterns; and, (3) physiological effects, identified by growth alterations, reduced yields, and changes in the quality of plant products. The acute symptoms are generally characteristic of a specific pollutant; though highly characteristic, chronic injury patterns are not. Ozone injury to leaves is identified as a stippling or flecking. Such injury has occurred experimentally in the most sensitive species after exposure to 60 \( \mu g/m^3 \) (0.03 ppm) ozone for 8 hours. Injury will occur in shorter time periods when low levels of sulfur dioxide are present. PAN-produced injury is characterized by an under-surface glazing or bronzing of the leaf. Such injury has occurred experimentally in the most sensitive species after exposure to 50 \( \mu g/m^3 \) (0.01 ppm) PAN for 5 hours. Leaf injury has occurred in certain sensitive species after a 4-hour exposure to 100 \( \mu g/m^3 \) (0.05 ppm) total oxidant. Ozone appears to be the most important phytotoxicant in the photochemical complex.

There are a number of factors affecting the response of vegetation to photochemical air pollutants. Variability in response is known to exist between species of a given genus and between varieties within a given species; varietal variations have been most extensively studied with tobacco. The influence of light intensity on the sensitivity of plants to damage during growth appears to depend on the phytotoxicant. Plants are more sensitive to PAN when grown under high light intensities, but are more sensitive to ozone when grown under low light intensities. Reported findings are in general agreement that sensitivity of greenhouse-grown plants to oxidants increases with temperature, from 10° to 38° C (40° to 100° F), but this positive correlation may result from the overriding influence of light intensity on sensitivity. The effects of humidity on the sensitivity of plants has not been well documented. General trends indicate that plants grown and/or exposed under high humidities are more sensitive than those grown at low humidities. There has been little research in this direction, but there are indications that soil factors such as drought and total fertility influence the sensitivity of plants to phytotoxic air pollutants. The age of the leaf under exposure is important in determining its sensitivity to air pollutants.

There is some evidence that oxidant or ozone injury may be reduced by pretreatment with the toxicant.
Identification of injury to a plant as being caused by air pollution is a difficult undertaking. Even when the markings on the leaves of a plant may be identified with an air pollutant, there is the further difficulty of evaluating the injury in terms of its effect on the intact plant. Additional problems arise in trying to evaluate the economic impact of air pollution damage to a plant.

The interrelations of time and concentration (dose) as they affect injury to plants are essential to air quality criteria. There are, however, only scant data relating concentrations and length of photochemical oxidant exposure to chronic injury and effects on reduction of plant growth, yield, or quality. There is also a dearth of information relating concentrations to acute injury. A larger body of information exists on the acute effects of ozone, but even in this instance, the information is far from complete. Sufficient data do exist, however, to tabularly present ozone concentrations which will produce 5 percent injury to sensitive, intermediate, and resistant plants after a given short-term exposure, as shown in Table 10-1. Information available lists 20 species and/or varieties as sensitive, 55 as intermediate in sensitivity, and 64 as relatively resistant.

Bacteriostatic and bactericidal properties of photochemical oxidants in general have been demonstrated. The growth suppression of microorganisms by ozone is a well-known phenomenon, although the ozone concentrations for this activity are undesirable from a human standpoint. The bactericidal activity of ozone varies with its concentration, the relative humidity, and the species of bacteria.

### G. EFFECT OF OZONE ON MATERIALS

The detailed, quantitative extent of damage to materials caused by atmospheric levels of ozone is unknown, but generally any organic material is adversely affected by concentrated ozone. Many polymers are extremely sensitive to even very small concentrations of ozone, this sensitivity increasing with the number of double bonds in the structure of the polymer.

Economically, rubber is probably the most important material sensitive to ozone attack, particularly styrene-butadiene, natural, polybutadiene, and synthetic polyisoprene. Anti-ozonant additives have been developed and are capable of protecting elastomers from ozone degradation; synthetic rubbers with inherent resistance to ozone are also available. These additives are expensive, however, and add to the cost of the end product; in addition, increasing amounts of antiozonants are required as the amount of ozone which is to be encountered increases, and sometimes only temporary protection is provided.

Ozone attacks the cellulose in fabrics through both a free radical chain mechanism and an electrophilic attack on double bonds; light and humidity appear necessary for appreciable alterations to occur. The relative susceptibility of different fibers to ozone attack appears to be, in increasing order, cotton, acetate, nylon, and polyester.

<table>
<thead>
<tr>
<th>Time, hr</th>
<th>Ozone concentrations producing injury in three types of plants, ppm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Sensitive</td>
</tr>
<tr>
<td>0.2</td>
<td>0.35–0.75</td>
</tr>
<tr>
<td>0.5</td>
<td>0.15–0.30</td>
</tr>
<tr>
<td>1.0</td>
<td>0.10–0.25</td>
</tr>
<tr>
<td>2.0</td>
<td>0.07–0.20</td>
</tr>
<tr>
<td>4.0</td>
<td>0.05–0.15</td>
</tr>
<tr>
<td>8.0</td>
<td>0.03–0.10</td>
</tr>
</tbody>
</table>

10-4
Certain dyes are susceptible to fading during exposure to ozone. The rate and extent of fading is also dependent upon other environmental factors such as relative humidity and the presence of air pollutants other than ozone, as well as the length and concentration of ozone exposure and the type of material exposed.

H. TOXICOLOGICAL STUDIES OF PHOTOCHEMICAL OXIDANTS

1. Effects of Ozone in Animals

The major physiological effects of ozone are on the respiratory system. Inhalation of ozone at concentrations greater than about 5,900 μg/m³ (3 ppm) for several hours produces hemorrhage and edema in the lungs. This reaction can be fatal to animals. Rats and mice appear to be more sensitive than rabbits, cats, and guinea pigs. The toxicity is greater for young animals and for exercising animals. It is abated by intermittency of exposure, by prophylactic administration of chemical reducing agents, or by introducing agents into the diet which reduce the activity of the thyroid gland. At exposures less than those which produce edema in the lungs, changes in the mechanical properties of the lung occur. These are accompanied by increased breathing rates and increased oxygen consumption. Repeated non-fatal exposures to concentrations greater than 15,700 μg/m³ (8 ppm) for 30 minutes have produced fibrosis in the respiratory tract of rabbits, with the damage increasing in severity over the length of the respiratory tract from the trachea to the bronchioles.

Short-term exposures to ozone also produce chemical changes in the lung tissue elements of animals. A study conducted on a small number of rabbits showed that inhalation of 1,960 to 9,800 μg/m³ (1 to 5 ppm) ozone for 1 hour can produce denaturation of the structural lung proteins. Ozone also appears to oxidize the sulphydryl groups of amino acids in the lung.

Short-term exposures to ozone also produce changes in organs other than the lung. Concentrations of 5,900 μg/m³ (3 ppm) for 20 hours can stimulate some adaptive liver enzymes. Inhalation of 390 to 490 μg/m³ (0.2 to 0.25 ppm) ozone for 30 to 60 minutes makes the red blood cells of mice, rabbits, rats, and man more sensitive to the shape-altering effects of irradiation. Exposure of blood to ozone in vitro produces interference with the release of oxygen from red blood cells; this suggests that ozone exposure could impair the delivery of oxygen to the tissues. Ozone exposures at concentrations from 1,310 to 7,800 μg/m³ (0.67 to 4.0 ppm) have been shown to reduce the in vitro phagocytic abilities of the pulmonary alveolar macrophages. A 3-hour exposure to 9,800 μg/m³ (5 ppm) ozone has been shown to reduce the activity of bactericidal enzyme, presumably due to in vivo oxidation of the enzyme.

Ozone inhalation increases the vulnerability of animals to other agents. A single exposure to ozone at a concentration of 160 μg/m³ (0.08 ppm) for 3 hours has increased the mortality among mice from inhalation of pathogenic bacteria. This occurred when the bacteria were administered both before and after exposure to ozone. Ozone also increases the toxicity of histamine in guinea pigs.

Long-term effects of ozone exposure include, in some species, the development of tolerance to biological effects of ozone, production of fibrotic changes in the lungs, and a possible increase in the rate of aging. While tolerance has been shown in rodents, it has not been shown in chickens, and it is not certain whether or not it occurs in man. In species where tolerance to ozone exposure has been demonstrated, information is not available concerning the duration and mechanism of tolerance following repeated exposure. The aging effect may be similar to the changes produced by exposure to free radicals or by irradiation.

2. Effects of Ozone in Humans

Some studies of human exposures to ozone have focused on the determination of the threshold level at which odor can be detected, and on the occurrence of changes in pulmonary function. Nine out of 10 subjects exposed to 40 μg/m³ (0.02 ppm) ozone were able to detect the odor immediately, and it
persisted for an average of 5 minutes. Thirteen of 14 subjects exposed to 100 μg/m³ (0.05 ppm) ozone indicated the odor is considerably stronger at this concentration, and the odor persisted for an average of 13 minutes.

Occupational exposure of humans to ozone concentrations of up to 490 μg/m³ (0.25 ppm) has not produced detectable changes in pulmonary function. Respiratory symptoms and a decrease in vital capacity in three out of seven smokers who had been occupationally exposed to ozone have occurred at concentrations greater than 590 μg/m³ (0.3 ppm).

Experimental exposures of humans have been carried out at concentrations ranging from 200 to 7,800 μg/m³ (0.1 to about 4 ppm) for periods of up to 2 hours. Exposure to 390 μg/m³ (0.2 ppm) for 3 hours daily, 6 days a week, for 12 weeks has not produced any change in ventilatory function tests. Similar exposure to 980 μg/m³ (0.5 ppm) produced a decrease in the forced expiratory volume during the last 4 weeks of exposure, with recovery taking place in a subsequent 6-week period. In each of 11 subjects, exposure to 1,180 to 1,570 μg/m³ (0.6 to 0.8 ppm) for 2 hours resulted in an impairment of the diffusing capacity of the lung. Small decreases in vital capacity and forced expiratory volume were observed in some of these subjects. Resistance to flow of air in the respiratory tract increased slightly in some subjects after exposure to 200 to 1,180 μg/m³ (0.1 to 0.6 ppm) for 1 hour, and increased consistently in each of four subjects after exposure to 1,960 μg/m³ (1 ppm) for 1 hour.

Data obtained from animal experimentation cannot be used directly to define the ozone concentrations above which human health will be affected. Animal mortality studies, however, can be useful in determining the factors involved in toxicity. While the concentrations of ozone used in the determination of short-term non-fatal effects in animals are rarely found in ambient air, the changes in pulmonary function observed during and after exposure to these concentrations call attention to the possibility that similar effects may be observed in humans.

When interpreting the research conducted thus far using human subjects, it must be noted that occupational exposures differ from experimental exposures, because it is difficult in an occupational environment to define the exact nature and dose of the pollutants present.

3. Effects of Peroxyacetyl Nitrate

Experimental studies with peroxycetyl nitrate (PAN) in animals indicate that mortality may be delayed for 7 to 14 days after exposure; however, the exposure levels required to produce this mortality never occur in ambient atmospheres.

A single experimental study of healthy human subjects exposed to 1,485 μg/m³ (0.3 ppm) peroxycetyl nitrate indicated only that there may be a small increase in oxygen uptake with exercise. Sensitive pulmonary function tests were not obtained.

The data from animal and human studies are sparse and inadequate for determining the toxicological potential of peroxycetyl nitrate. It would appear, however, that at the concentrations of this compound known to occur in ambient atmospheres, PAN does not present any recognized health hazard.

4. Effects of Mixtures Containing Photochemical Oxidants on Animals

Studies have been conducted on animals exposed to both synthetic and natural photochemical smog. Synthetic smog has been produced by the irradiation of diluted motor vehicle exhaust or by irradiation of air mixtures containing nitrogen oxides and certain hydrocarbons. Exposures to irradiated motor vehicle exhaust are complicated by the simultaneous presence of carbon monoxide and other non-oxidant substances which include high concentrations of formaldehyde. Guinea pigs show increased respiratory volume during a four-hour exposure to irradiated exhaust containing 1,570 μg/m³ (0.8 ppm) total oxidant.
Exposure of mice to both natural and synthetic smog for 3 hours, at concentrations greater than 780 \( \mu g/m^3 \) (0.4 ppm) oxidants have produced changes in the fine structure of the lung. The nature and extent of the damage was the same after exposure to either type of smog with the same oxidant levels. The severity of the damage increased with age and became irreversible at age 21 months.

Chronic exposure of guinea pigs to ambient air with an average oxidant concentration of from 40 to 140 \( \mu g/m^3 \) (0.02 to 0.07 ppm) leads to a significant increase in flow resistance when the peak oxidant concentrations exceed 980 \( \mu g/m^3 \) (0.5 ppm).

When male mice, prior to mating, were given long-term exposures to irradiated auto exhaust containing from 200 to 1960 \( \mu g/m^3 \) (0.1 to 1.0 ppm) oxidant, a decrease in fertility and an increase in neonatal mortality of their offspring resulted; the irradiated mixture also contained varying concentrations of carbon monoxide, nitrogen oxides, and hydrocarbons. Similar exposures also cause a reduction in spontaneous running activity, which results in an adaptation response.

Thus a number of experimental studies have demonstrated that changes in lung tissue or lung function occur when animals are exposed for several hours to photo-oxidized mixtures containing 980 \( \mu g/m^3 \) (0.5 ppm) or more of oxidants.

5. Effects of Mixtures Containing Photochemical Oxidants on Humans

Laboratory studies of human exposure to photochemical smog have involved primarily the measurement of eye irritation. Based on the existing data, it appears that: (1) the effective eye irritants are the products of photochemical reactions; (2) although oxidant concentrations may correlate with the severity of eye irritation, a direct cause-effect relationship has not been demonstrated since ozone, the principal contributor to ambient oxidant levels is not an eye irritant; (3) the precursors of the eye irritants are organic compounds in combination with oxides of nitrogen, the most potent being aromatic hydrocarbons; (4) the chemical identities of the effective irritants in synthetic systems are known as being formaldehyde, peroxybenzoyl nitrate (PBzN), peroxyacetyl nitrate (PAN), and acrolein, although the latter two contribute to only a minor extent; and (5) the substances causing eye irritation in the atmosphere have not been completely defined.

I. EPIDEMIOLOGICAL STUDIES OF PHOTOCHEMICAL OXIDANTS

Several studies have examined daily mortality rates in localities where photochemical air pollution occurs, to determine if a relationship exists with increased levels of oxidant. Such an association has not been shown. These studies, however, pose a number of unresolved questions. One of these is, what is the effect of temperature, either alone or in combination with oxidants? In some of the most severe episodes, there has been an associated increase in environmental temperature, sufficient to cause excess mortality by itself. Several studies of mortality among residents in nursing homes in Los Angeles showed such excess mortality. In recent heat wave and air pollution episodes, however, large proportions of the elderly and ill persons in nursing homes have been protected by air conditioning.

Evidence of increased morbidity has been sought through study of general hospital admissions, but no unequivocal association between photochemical air pollution and increased morbidity has been shown. Additional studies are indicated for improved definition. Peak oxidant values of 250 \( \mu g/m^3 \) (0.13 ppm), which might be expected in relation to maximum hourly average levels of 100 to 120 \( \mu g/m^3 \) (0.05 to 0.06 ppm), have been associated with aggravation of asthma. No association between ambient oxidant concentrations and changes in respiratory symptoms or function was shown, however, in two separate studies of subjects with preexisting chronic respiratory disease. Non-smoking subjects with chronic respiratory disease did, however, demonstrate less airway resistance when they were studied in a room where the ambient air
of Los Angeles was filtered before entry. No acute or chronic effects of oxidant pollution on ventilatory performance of elementary schoolchildren were demonstrated in a study conducted in two communities within the Los Angeles basin.

Impairment of performance by high school athletes has been observed when photochemical oxidants ranged from 60 to 590 μg/m³ (0.03 to 0.3 ppm) for 1 hour immediately prior to the start of activities. Significantly, more automobile accidents have also occurred on days of high oxidant concentrations, but no threshold level for this effect could be determined from the analysis.

Among the general community, eye irritation is a major effect of photochemical air pollution. In Southern California, it has affected more than three-fourths of the population. Eye irritation under conditions prevalent in Los Angeles is likely to occur in a large fraction of the population when oxidant concentrations in ambient air increases to about 200 μg/m³ (0.10 ppm). This oxidant value might be expected to be associated with a maximum hourly average oxidant concentration of 50 to 100 μg/m³ (0.025 to 0.50 ppm), depending on localized conditions. According to survey data gathered in 1956, asthma, cough, and nose and throat complaints were more frequent in Los Angeles, Orange, and San Diego counties than in the San Francisco Bay area or in the rest of the State.

Casual reports of the presence of the symptoms of eye irritation have been recorded in many cities in the United States. Epidemiologic studies have been inadequate, however, to relate these symptoms clearly to measured exposures to photochemical oxidants. In fact, one of the major photochemical oxidants, ozone, is not an eye irritant. That eye irritation is experienced whenever the oxidant level exceeds a certain value is an indication that oxidant concentrations correlate well with other aspects of the photochemical complex; oxidant levels are probably a measure of the photochemical activity which produces the eye irritants. On the other hand, it must be recognized that reactions of ozone with hydrocarbons do lead to hydrocarbon fragments which are eye irritants. Nor can the possibility be discounted that ozone in the photochemical complex may exert a synergistic effect on eye irritation. Because the oxidant reading measured only the net oxidizing property of the atmosphere, however, the same amount of eye irritation experienced in two different geographical locations from identical irritants could be associated with different levels of oxidant, if other pollutants differed in their concentration.

J. AREAS FOR FUTURE RESEARCH

1. Environmental Aspects of Photochemical Oxidants

   1. Research should be conducted to further identify the substance(s) which cause eye irritation.

   2. The nature of the photochemical aerosol, its behavior at different pressures of water vapor, and the nature of the surface layer of the particulates remains to be determined.

   3. The role of sulfur dioxide in the formation of photochemical aerosols and in the impairment of visibility should be investigated.

   4. Mechanisms of photochemical oxidant formation should be explained.

2. Toxicity of Ozone, Photochemical Oxidants, and Peroxyacetyl Nitrates

   1. The effect of ozone and PAN in combination with other pollutants found in ambient air should be investigated. Considerable information is available on the separate effects of ozone, nitrogen dioxide, and sulfur dioxide, but data on the combined effects of defined concentrations of these gases are sparse. The effect of particulates (dust, saline droplets, oil, soots, etc.) should be determined alone and in combination with the gases. Additional variables such as
humidity and temperature should be controlled and recorded. These experiments should be carried out with materials, vegetation, animals, and, under appropriate conditions, in man.

2. Experiments with human exposures to gas mixtures should include a comparison between the respiratory effects shown in healthy subjects and those shown in patients with chronic respiratory disease, care being taken to respect the rights of experimental subjects.

3. Existing data demonstrate that tolerance occurs only in rodents. Indices other than mortality are required to demonstrate tolerance in animals. If such indices can be developed, then a study is needed to see if a similar phenomenon occurs in man.

4. The mechanisms of systemic effects of ozone (headache, fatigue, impaired oxygen transport by hemoglobin, inability to concentrate, etc.) have yet to be explained.

5. The rate and site of uptake of ozone and its fate following uptake should be determined in vegetation and animals.

6. The mechanism for the production of ozone-induced pulmonary edema remains unexplained.

7. Additional research is needed to define the role of peroxycacetyl nitrates in the production of eye irritation.

3. Epidemiology of Photochemical Oxidants

1. Of high priority is the need to study eye and respiratory irritation in metropolitan areas outside of California. Studies should be supplemented by pulmonary function tests.

2. Although the effects of episodes of high pollution levels have been studied with respect to mortality, morbidity, impairment of performance, etc., additional studies are needed at different sites and for different effects. These should include congenital malformations, stillbirths, hospitals admissions for miscarriage, and alterations in the sex ratio of newborns.

3. The examination of children has received insufficient attention in epidemiologic studies of the health effects of air pollution. This should be undertaken with respect to the effects of photochemical oxidants using simple pulmonary function tests. Emphasis should be placed on further studies of the incidence of asthma attacks during episodes of high pollution.

K. CONCLUSIONS

Derived from a careful evaluation of the studies cited in this document, the conclusions given below represent the best judgment of the scientific staff of the National Air Pollution Control Administration of the effects that may occur when various levels of photochemical oxidants are reached in the ambient air. The more detailed information from which the conclusions were derived, and the qualifications that entered into the consideration of these data, can be found in the appropriate chapter of this document.

1. Human Exposure
   a. Ozone

(1) Long-term exposure of human subjects.
   (a) Exposure to a concentration of up to 390 \( \mu g/m^3 \) (0.2 ppm) for 3 hours a day, 6 days a week, for 12 weeks, has not produced any apparent effects (Chapter 8, section B.2.)

   (b) Exposure to a concentration of 980 \( \mu g/m^3 \) (0.5 ppm) for 3 hours a day, 6 days a week, has caused a decrease in the 1-second forced expiratory volume (FEV\(_{1.0}\)) after 8 weeks (Chapter 8, section B.2)

(2) Short-term exposure of human subjects.
   (a) Exposure to a concentration of 40 \( \mu g/m^3 \) (0.02 ppm) was detected immediately by 9 of 10 subjects.
After an average of 5 minutes exposure, subjects could no longer detect ozone (Chapter 8, section E.2).

(b) Exposure to a concentration of 590 \( \mu g/m^3 \) (0.3 ppm) for 8 hours appears to be the threshold for nasal and throat irritation (Chapter 8, section E.2.)

(c) Exposure to concentrations of from 1,180 to 1,960 \( \mu g/m^3 \) (0.6 to 1.0 ppm) for 1 to 2 hours may impair pulmonary function by causing increased airway resistance, decreased carbon monoxide diffusing capacity, decreased total capacity, and decreased forced expiratory volume (Chapter 8, section B.2.2).

(d) Exposure to concentrations of from 1,960 to 5,900 \( \mu g/m^3 \) (1.0 to 3.0 ppm) for 10 to 30 minutes is intolerable to some people (Chapter 5, section B.2.)

(e) Exposure to a concentration of 17,600 \( \mu g/m^3 \) (9.0 ppm) produces severe illness (Chapter 5, section B.2.)

b. Oxidants

(1) Long-term exposure of human subjects.

Exposure to ambient air containing an oxidant concentration of about 250 \( \mu g/m^3 \) (0.13 ppm) (maximum daily value) has caused an increase in the number of asthmatic attacks in about 5 percent of a group of asthmatic patients. Such a peak value would be expected to be associated with a maximum hourly average concentration of 100 to 120 \( \mu g/m^3 \) (0.05 to 0.06 ppm) (Chapter 9, section B.3.)

(2) Short-term exposure of human subjects.

(a) Exposure to an atmosphere with peak oxidant concentrations of 200 \( \mu g/m^3 \) (0.1 ppm) and above has been associated with eye irritation. Such a peak concentration would be expected to be associated with a maximum hourly average concentration of 50 to 100

\( \mu g/m^3 \) (0.025 to 0.05 ppm) (Chapter 9, section B.3.)

(b) Exposure to an atmosphere with average hourly oxidant concentrations ranging from 60 to 590 \( \mu g/m^3 \) (0.03 to 0.30 ppm) has been associated with impairment of performance of student athletes (Chapter 9, section B.4.)

2. Other Exposures

a. Photochemical Oxidants

(1) Effects on vegetation and laboratory animals.

(a) Exposure to concentrations of about 60 \( \mu g/m^3 \) (0.03 ppm) ozone for 8 hours or to 0.01 ppm peroxyacetyl nitrate for 5 hours has been associated with the occurrence of leaf lesions in the most sensitive species, under laboratory conditions (Chapter 6, section E.)

(b) Exposure to ambient air containing oxidant concentrations of about 100 \( \mu g/m^3 \) (0.05 ppm) for 4 hours has been associated with leaf injury to the most sensitive species (Chapter 6, section E.)

(c) Experimental exposures of laboratory animals to ozone concentrations of from 160 to 2,550 \( \mu g/m^3 \) (0.08 to 1.30 ppm) for 3 hours has resulted in increased susceptibility to bacterial infection (Chapter 8, section B.1.)

b. Ozone Effects on Susceptible Materials

(1) Polymers.

(a) Many polymers, especially rubber, are extremely sensitive to very small concentrations. To provide protection, antiozonant additives are used, but are expensive and add to the cost of the end product (Chapter 7).

(2) Cellulose and dyes.

(a) The cellulose in fabrics is attacked by ozone, with subsequent weakening of the fabric. Similarly, certain dyes are susceptible to fading during exposure to ozone (Chapter 7). Tables 10-2
<table>
<thead>
<tr>
<th>Effect</th>
<th>Exposure</th>
<th>Duration</th>
<th>Comment</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vegetation damage&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.03 ppm, 60 µg/m³</td>
<td>8 hours</td>
<td>Sensitive species; laboratory conditions</td>
<td>Heck and Dunning</td>
</tr>
<tr>
<td>Cracking of stretched rubber</td>
<td>0.02 ppm, 40 µg/m³</td>
<td>1 hour</td>
<td>Vulcanized natural rubber</td>
<td>Bradley and Haagen-Smit</td>
</tr>
<tr>
<td>Odor detection</td>
<td>0.02 ppm, 40 µg/m³</td>
<td>&lt;5 minutes</td>
<td>Odor detected in 9 of 10 subjects</td>
<td>Henschler et al.</td>
</tr>
<tr>
<td>Increased susceptibility of laboratory animals to bacterial infection</td>
<td>0.08 ppm, 160 µg/m³</td>
<td>3 hours</td>
<td>Demonstrated in mice at 160 µg/m³ and in mice at 2550 µg/m³</td>
<td>Coffin et al.</td>
</tr>
<tr>
<td>Respiratory irritation (nose and throat), chest constriction</td>
<td>1.30 ppm, 2,550 µg/m³</td>
<td>Continuous during working hours</td>
<td>Occupational exposure of welders, other pollutants probably also present</td>
<td>Miller et al.</td>
</tr>
<tr>
<td>Changes in pulmonary function: Diminished FEV&lt;sub&gt;1.0&lt;/sub&gt; after 8 weeks</td>
<td>0.30 ppm, 590 µg/m³</td>
<td>3 hours/day, 6 days/week, for 12 weeks</td>
<td>Change returns to normal 6 weeks after exposure. No changes observed at 390 µg/m³ (0.2 ppm)</td>
<td>Kleinfield et al.</td>
</tr>
<tr>
<td>Small decrements in VC, FRC, and DL&lt;sub&gt;CO&lt;/sub&gt; in, respectively, 3, 2, and 1 out of 7 subjects</td>
<td>0.20 ppm, 390 µg/m³</td>
<td>Continuous during working hours</td>
<td>Occupational exposure. All 7 subjects smoked. Normal values for VC, FRC, and DL&lt;sub&gt;CO&lt;/sub&gt; based on predicted value.</td>
<td>Bennett</td>
</tr>
<tr>
<td>Impaired diffusion capacity (DL&lt;sub&gt;CO&lt;/sub&gt;)</td>
<td>0.60 ppm, 1,180 µg/m³</td>
<td>2 hours</td>
<td>Experimental exposure of 11 subjects</td>
<td>Young et al.</td>
</tr>
<tr>
<td>Increased airway resistance</td>
<td>0.80 ppm, 1,570 µg/m³</td>
<td>1 hour</td>
<td>Significant increase in 2 of 4 subjects at 200 µg/m³ (0.1 ppm) and 4 of 4 subjects at 1960 µg/m³ (1.0 ppm)</td>
<td>Young et al.</td>
</tr>
<tr>
<td>Reduced VC, severe cough, inability to concentrate</td>
<td>1.00 ppm, 1,960 µg/m³</td>
<td>2 hours</td>
<td>High temperatures. One subject.</td>
<td>Griswold et al.</td>
</tr>
<tr>
<td>Acute pulmonary edema</td>
<td>2.00 ppm, 3,900 µg/m³</td>
<td>2 hours</td>
<td>Refers to peak concentration of occupational exposure. Most of exposure was to lower level</td>
<td>Kleinfeld et al.</td>
</tr>
</tbody>
</table>

<sup>a</sup>Similar vegetation damage also occurs upon exposure to 0.01 ppm peroxyacetyl nitrate for 5 hours.
<table>
<thead>
<tr>
<th>Effect</th>
<th>Exposure</th>
<th>Duration</th>
<th>Comment</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vegetation damage</td>
<td>0.05</td>
<td>100</td>
<td>4 hours Leaf injury to sensitive species</td>
<td>MacDowall et al</td>
</tr>
<tr>
<td>Eye irritation</td>
<td>Exceeding</td>
<td>Peak</td>
<td>Result of panel response.</td>
<td>Renzetti and Gobran</td>
</tr>
<tr>
<td></td>
<td>0.1</td>
<td>values</td>
<td>Such a peak value would be expected to be associated with a maximum</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.1</td>
<td></td>
<td>hourly average concentration of 50 to 100 µg/m³ (0.025 to 0.05 ppm)</td>
<td></td>
</tr>
<tr>
<td>Aggravation of respiratory diseases—asthma</td>
<td>0.13&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Maximum daily value</td>
<td>Patients exposed to ambient air. Value refers to oxidant level at which number of attacks increased</td>
<td>Schoettlin and Landau</td>
</tr>
<tr>
<td>Impaired performance of student athletes</td>
<td>0.03 to 0.30</td>
<td>1 hour</td>
<td>Exposure for 1 hour immediately prior to race</td>
<td>Wayne et al.</td>
</tr>
<tr>
<td></td>
<td>60 to 590</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup>Calculated from a measured value of 0.25 ppm (phenolphthalein method) which is equivalent to 0.13 ppm by the KI method.
and 10-3 present these conclusions in tabular form.

L. RESUMÉ

Under the conditions prevailing in the areas where studies were conducted, adverse health effects, as shown by impairment of performance of student athletes, occurred over a range of hourly average oxidant concentrations from 60 to 590 μg/m³ (0.03 to 0.3 ppm). An increased frequency of asthma attacks in a small proportion of subjects with this disease was shown on days when oxidant concentrations exceeded peak values of 250 μg/m³ (0.13 ppm), a level that would be associated with an hourly average concentration ranging from 100 to 120 μg/m³ (0.05 to 0.06 ppm). Adverse health effects, as manifested by eye irritation, were reported by subjects in several studies when photochemical oxidant concentrations reached instantaneous levels of about 200 μg/m³ (0.10 ppm), a level that would be associated with an hourly average concentration ranging from 60 to 100 μg/m³ (0.03 to 0.05 ppm).

Adverse effects on sensitive vegetation were observed from exposure to photochemical oxidant concentrations of about 100 μg/m³ (0.05 ppm) for 4 hours. Adverse effects on materials from exposure to photochemical oxidants have not been precisely quantified, but have been observed at the levels presently occurring in many urban atmospheres.

It is reasonable and prudent to conclude that, when promulgating ambient air quality standards, consideration should be given to requirements for margins of safety that would take into account possible effects on health, vegetation, and materials that might occur below the lowest of the above levels.
APPENDIX

CONVERSION BETWEEN VOLUME
AND MASS UNITS OF CONCENTRATION

The physical state of gaseous air pollutants at atmospheric concentrations generally may be described by the ideal gas law:

\[ pv = nRT \]  \hspace{1cm} (1)

Where:  
\( p \) = absolute pressure of gas  
\( v \) = volume of gas  
\( n \) = number of moles of gas  
\( R \) = unival gas constant  
\( T \) = absolute temperature

The number of moles (\( n \)) may be calculated from the weight of pollutant (\( w \)) and its molecular weight (\( m \)) by:

\[ n = \frac{w}{m} \]  \hspace{1cm} (2)

Substituting equation 2 into equation 1 and rearranging yields:

\[ v = \frac{wrt}{pm} \]  \hspace{1cm} (3)

Parts per million refers to the volume of pollutant (\( v \)) per million volumes of air (\( V \)).

\[ 1 \text{ ppm} = \frac{v}{10^6 V} \]  \hspace{1cm} (4)

Substituting equation (3) into equation (4) yields:

\[ \text{ppm} = \frac{w}{V} \cdot \frac{RT}{pm10^6} \]  \hspace{1cm} (5)

Using the appropriate values for variables in equation 5 a conversion from volume to mass units of concentration for \( \text{O}_3 \) may be derived as shown below.

\[ T = 298^\circ \text{ K (25^\circ \text{ C})} \]  
\( p = 1 \text{ atm} \)  
\( m = 48 \text{ g/mole} \)  
\( R = 8.21 \times 10^{-2} \text{ l-atm/mole}^\circ \text{ K} \)

\[ \text{ppm} = \frac{w(g) \times 10^6 (\mu g/g)}{V(\ell) \times 10^{-3} \text{ (m}^3/\ell)} \]

\[ \frac{8.21 \times 10^{-2} (\ell\text{-atm/mole}^\circ \text{K}) \times 298(\circ \text{K})}{1(\text{atm}) \times 48 \text{ (g/mole)} \times 10^6} \]

\[ 1 \text{ ppm} = 1,960 \mu g/m^3 \]

\[ 1 \mu g/m^3 = 0.51 \times 10^{-3} \text{ ppm} \]
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