

United States
Environmental Protection
Agency

Environmental Criteria and
Assessment Office
Research Triangle Park, NC 27711

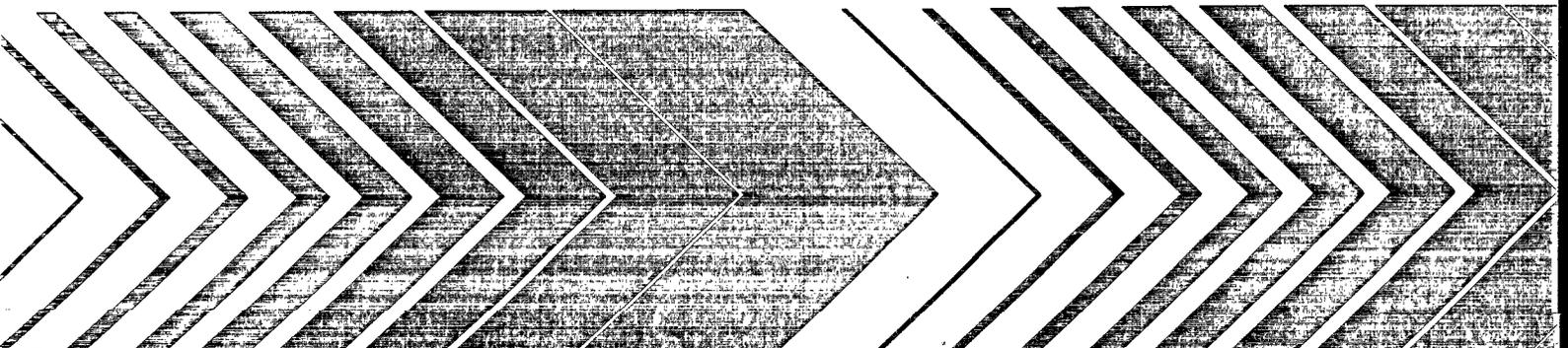
EPA-600/8-84-020aF
August 1986

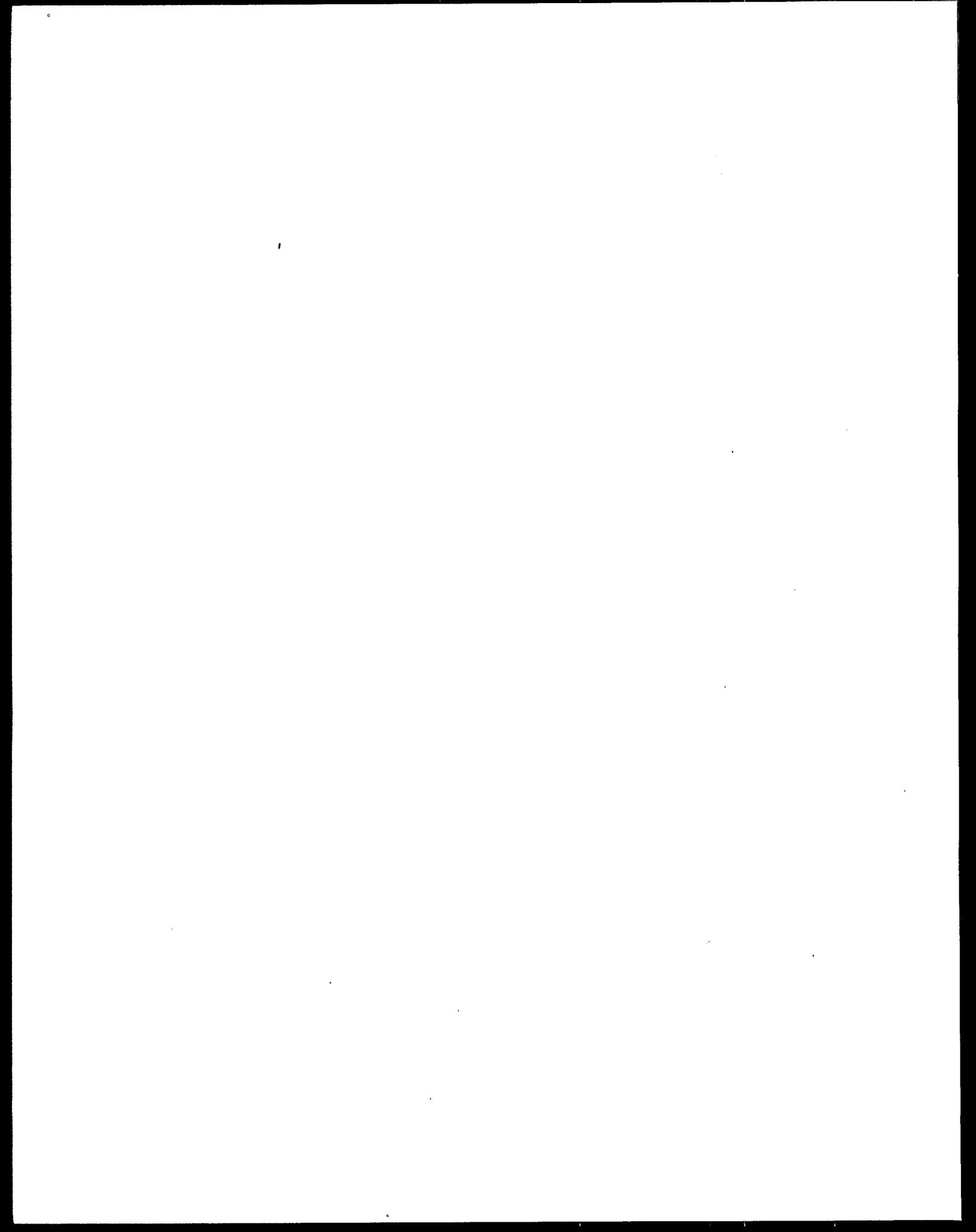
Research and Development



Air Quality Criteria for Ozone and Other Photochemical Oxidants

Volume I of V





EPA-600/8-84-020aF
August 1986

**Air Quality Criteria
for Ozone and Other
Photochemical Oxidants**

Volume I of V

Environmental Criteria and Assessment Office
Office of Health and Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Research Triangle Park, N.C. 27711

DISCLAIMER

This document has been reviewed in accordance with U.S. Environmental Protection Agency policy and approved for publication. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

ABSTRACT

Scientific information is presented and evaluated relative to the health and welfare effects associated with exposure to ozone and other photochemical oxidants. Although it is not intended as a complete and detailed literature review, the document covers pertinent literature through early 1986.

Data on health and welfare effects are emphasized, but additional information is provided for understanding the nature of the oxidant pollution problem and for evaluating the reliability of effects data as well as their relevance to potential exposures to ozone and other oxidants at concentrations occurring in ambient air. Information is presented on the following exposure-related topics: nature, source, measurement, and concentrations of precursors to ozone and other photochemical oxidants; the formation of ozone and other photochemical oxidants and their transport once formed; the properties, chemistry, and measurement of ozone and other photochemical oxidants; and the concentrations of ozone and other photochemical oxidants that are typically found in ambient air.

The specific areas addressed by chapters on health and welfare effects are the toxicological appraisal of effects of ozone and other oxidants; effects observed in controlled human exposures; effects observed in field and epidemiological studies; effects on vegetation seen in field and controlled exposures; effects on natural and agroecosystems; and effects on nonbiological materials observed in field and chamber studies.

AIR QUALITY CRITERIA FOR OZONE
AND OTHER PHOTOCHEMICAL OXIDANTS

	<u>Page</u>
VOLUME I	
Chapter 1. Summary and Conclusions	1-1
VOLUME II	
Chapter 2. Introduction	2-1
Chapter 3. Properties, Chemistry, and Transport of Ozone and Other Photochemical Oxidants and Their Precursors	3-1
Chapter 4. Sampling and Measurement of Ozone and Other Photochemical Oxidants and Their Precursors	4-1
Chapter 5. Concentrations of Ozone and Other Photochemical Oxidants in Ambient Air	5-1
VOLUME III	
Chapter 6. Effects of Ozone and Other Photochemical Oxidants on Vegetation	6-1
Chapter 7. Effects of Ozone on Natural Ecosystems and Their Components	7-1
Chapter 8. Effects of Ozone and Other Photochemical Oxidants on Nonbiological Materials	8-1
VOLUME IV	
Chapter 9. Toxicological Effects of Ozone and Other Photochemical Oxidants	9-1
VOLUME V	
Chapter 10. Controlled Human Studies of the Effects of Ozone and Other Photochemical Oxidants	10-1
Chapter 11. Field and Epidemiological Studies of the Effects of Ozone and Other Photochemical Oxidants	11-1
Chapter 12. Evaluation of Health Effects Data for Ozone and Other Photochemical Oxidants	12-1

TABLE OF CONTENTS

	<u>Page</u>
LIST OF TABLES	ix
LIST OF FIGURES	x
LIST OF ABBREVIATIONS	xi
AUTHORS, CONTRIBUTORS, AND REVIEWERS	xv
1. SUMMARY AND CONCLUSIONS	1-1
1.1 INTRODUCTION	1-1
1.2 PROPERTIES, CHEMISTRY, AND TRANSPORT OF OZONE AND OTHER PHOTOCHEMICAL OXIDANTS AND THEIR PRECURSORS	1-3
1.2.1 Descriptions and Properties of Ozone and Other Photochemical Oxidants	1-3
1.2.2 Nature of Precursors to Ozone and Other Photochemical Oxidants	1-4
1.2.3 Atmospheric Reactions of Ozone and Other Oxidants Including Their Role in Aerosol Formation	1-5
1.2.3.1 Formation and Transformation of Ozone and Other Photochemical Oxidants	1-6
1.2.3.2 Atmospheric Chemical Processes Involving Ozone	1-7
1.2.3.3 Atmospheric Reactions of PAN, H ₂ O ₂ , and HCOOH	1-8
1.2.4 Meteorological and Climatological Processes	1-9
1.2.4.1 Atmospheric Mixing	1-9
1.2.4.2 Wind Speed and Direction	1-11
1.2.4.3 Effects of Sunlight and Temperature	1-11
1.2.4.4 Transport of Ozone and Other Oxidants and Their Precursors	1-12
1.2.4.5 Stratospheric-Tropospheric Ozone Exchange	1-13
1.2.4.6 Stratospheric Ozone at Ground Level	1-14
1.2.4.7 Background Ozone from Photochemical Reactions	1-15
1.2.5 Sources, Emissions, and Concentrations of Precursors to Ozone and Other Photochemical Oxidants	1-16
1.2.5.1 Sources and Emissions of Precursors	1-16
1.2.5.2 Representative Concentrations in Ambient Air	1-17
1.2.6 Source-Receptor (Oxidant-Precursor) Models	1-19
1.2.6.1 Trajectory Models	1-19
1.2.6.2 Fixed-Grid Models	1-20
1.2.6.3 Box Models	1-20
1.2.6.4 Validation and Sensitivity Analyses for Dynamic Models	1-20
1.3 SAMPLING AND MEASUREMENT OF OZONE AND OTHER PHOTOCHEMICAL OXIDANTS AND THEIR PRECURSORS	1-21
1.3.1 Sampling and Measurement of Ozone and Other Photochemical Oxidants	1-21

TABLE OF CONTENTS
(continued)

		<u>Page</u>
	1.3.1.1 Quality Assurance and Sampling	1-21
	1.3.1.2 Measurement Methods for Total Oxidants and Ozone	1-22
	1.3.1.3 Calibration Methods	1-24
	1.3.1.4 Relationships of Total Oxidants and Ozone Measurements	1-26
	1.3.1.5 Methods for Sampling and Analysis of Peroxyacetyl Nitrate and Its Homologues	1-27
	1.3.1.6 Methods for Sampling and Analysis of Hydrogen Peroxide	1-30
	1.3.2 Measurement of Precursors to Ozone and Other Photochemical Oxidants	1-32
	1.3.2.1 Nonmethane Organic Compounds	1-32
	1.3.2.2 Nitrogen Oxides	1-34
1.4	CONCENTRATIONS OF OZONE AND OTHER PHOTOCHEMICAL OXIDANTS IN AMBIENT AIR	1-36
	1.4.1 Ozone Concentrations in Urban Areas	1-36
	1.4.2 Trends in Nationwide Ozone Concentrations	1-39
	1.4.3 Ozone Concentrations in Nonurban Areas	1-41
	1.4.4 Diurnal and Seasonal Patterns in Ozone Concentrations	1-44
	1.4.5 Spatial Patterns in Ozone Concentrations	1-46
	1.4.5.1 Urban-Nonurban Differences in Ozone Concentrations	1-46
	1.4.5.2 Geographic, Vertical, and Altitudinal Variations in Ozone Concentrations	1-47
	1.4.5.3 Other Spatial Variations in Ozone Concentrations	1-50
	1.4.6 Concentrations and Patterns of Other Photochemical Oxidants	1-51
	1.4.6.1 Concentrations	1-51
	1.4.6.2 Patterns	1-52
	1.4.7 Relationship Between Ozone and Other Photochemical Oxidants	1-53
1.5	EFFECTS OF OZONE AND OTHER PHOTOCHEMICAL OXIDANTS ON VEGETATION	1-55
	1.5.1 Limiting Values of Plant Response to Ozone	1-56
	1.5.2 Methods for Determining Ozone Yield Losses	1-58
	1.5.3 Estimates of Ozone-Induced Yield Loss	1-60
	1.5.3.1 Yield Loss: Determination by Regression Analysis	1-61
	1.5.3.2 Yield Loss: Determination from Discrete Treatment	1-67
	1.5.3.3 Yield Loss: Determination with Chemical Protectants	1-67
	1.5.3.4 Yield Loss: Determination from Ambient Exposures	1-69
	1.5.3.5 Yield Loss Summary	1-69

TABLE OF CONTENTS
(continued)

		<u>Page</u>
1.5.4	Effects on Crop Quality	1-72
1.5.5	Statistics Used to Characterize Ozone Exposures ..	1-72
1.5.6	Relationship Between Yield Loss and Foliar Injury	1-75
1.5.7	Physiological Basis of Yield Reductions	1-75
1.5.8	Factors Affecting Plant Response to Ozone	1-76
	1.5.8.1 Environmental Conditions	1-77
	1.5.8.2 Interaction with Plant Diseases	1-78
	1.5.8.3 Interaction of Ozone with Other Air Pollutants	1-78
1.5.9	Economic Assessment of Effects of Ozone on Agriculture	1-79
1.5.10	Effects of Peroxyacetyl Nitrate on Vegetation	1-90
	1.5.10.1 Factors Affecting Plant Response to PAN	1-90
	1.5.10.2 Limiting Values of Plant Response	1-90
	1.5.10.3 Effects of PAN on Plant Yield	1-91
1.6	EFFECTS OF OZONE ON NATURAL ECOSYSTEMS AND THEIR COMPONENTS	1-91
1.6.1	Responses of Ecosystems to Ozone Stress	1-91
1.6.2	Effects of Ozone on Producers	1-92
1.6.3	Effects of Ozone on Other Ecosystem Components and on Ecosystem Interactions	1-94
1.6.4	Effects of Ozone on Specific Ecosystems	1-95
1.6.5	Economic Valuation of Ecosystems	1-97
1.7	EFFECTS OF OZONE AND OTHER PHOTOCHEMICAL OXIDANTS ON NONBIOLOGICAL MATERIALS	1-98
1.8	TOXICOLOGICAL EFFECTS OF OZONE AND OTHER PHOTOCHEMICAL OXIDANTS	1-103
1.8.1	Introduction	1-103
1.8.2	Regional Dosimetry in the Respiratory Tract	1-104
1.8.3	Effects of Ozone on the Respiratory Tract	1-107
	1.8.3.1 Morphological Effects	1-107
	1.8.3.2 Pulmonary Function	1-110
	1.8.3.3 Biochemical Effects	1-116
	1.8.3.4 Host Defense Mechanisms	1-122
	1.8.3.5 Tolerance	1-127
1.8.4	Extrapulmonary Effects of Ozone	1-131
	1.8.4.1 Central Nervous System and Behavioral Effects	1-131
	1.8.4.2 Cardiovascular Effects	1-132
	1.8.4.3 Hematological and Serum Chemistry Effects	1-132
	1.8.4.4 Cytogenetic and Teratogenic Effects	1-134
	1.8.4.5 Other Extrapulmonary Effects	1-135
1.8.5	Interaction of Ozone with Other Pollutants	1-136
1.8.6	Effects of Other Photochemical Oxidants	1-136
1.9	CONTROLLED HUMAN STUDIES OF THE EFFECTS OF OZONE AND OTHER PHOTOCHEMICAL OXIDANTS	1-142

TABLE OF CONTENTS
(continued)

	<u>Page</u>
1.10 FIELD AND EPIDEMIOLOGICAL STUDIES OF THE EFFECTS OF OZONE AND OTHER PHOTOCHEMICAL OXIDANTS	1-151
1.11 EVALUATION OF HEALTH EFFECTS DATA FOR OZONE AND OTHER PHOTOCHEMICAL OXIDANTS	1-156
1.11.1 Health Effects in the General Human Population ...	1-156
1.11.2 Health Effects in Individuals with Preexisting Disease	1-162
1.11.3 Extrapolation of Effects Observed in Animals to Human Populations	1-163
1.11.4 Health Effects of Other Photochemical Oxidants and Pollutant Mixtures	1-164
1.11.5 Identification of Potentially At-Risk Groups	1-164
1.12 REFERENCES	1-166

LIST OF TABLES

<u>Table</u>	<u>Page</u>
1-1 Summary of ozone monitoring techniques	1-23
1-2 Ozone calibration techniques	1-25
1-3 Second-highest ozone concentrations among daily maximum 1-hr values in 1983 in Standard Metropolitan Statistical Areas with populations ≥ 1 million, given by census divisions and regions	1-37
1-4 Ozone concentrations for short-term exposures that produce 5 or 20 percent injury to vegetation grown under sensitive conditions	1-57
1-5 Summary of ozone concentrations predicted to cause 10 percent and 30 percent yield losses and summary of yield losses predicted to occur at 7-hr seasonal mean ozone concentrations of 0.04 and 0.06 ppm	1-64
1-6 Ozone concentrations at which significant yield losses have been noted for a variety of plant species exposed under various experimental conditions	1-68
1-7 Effects of ozone on crop yield as determined by the use of chemical protectants	1-70
1-8 Effects of ambient oxidants on yield of selected crops	1-71
1-9 Summary of estimates of regional economic consequences of ozone pollution	1-81
1-10 Summary of estimates of national economic consequences of ozone pollution	1-73
1-11 Summary table: Morphological effects of ozone in experimental animals	1-112
1-12 Summary table: Effects on pulmonary function of short-term exposures to ozone in experimental animals	1-116
1-13 Summary table: Effects on pulmonary function of long-term exposures to ozone in experimental animals	1-118
1-14 Summary table: Biochemical changes in experimental animals exposed to ozone	1-124
1-15 Summary table: Effects of ozone on host defense mechanisms in experimental animals	1-129
1-16 Summary table: Extrapulmonary effects of ozone in experimental animals	1-138
1-17 Summary table: Interaction of ozone with other pollutants in experimental animals	1-140
1-18 Summary table: Controlled human exposure to ozone	1-143
1-19 Summary table: Acute effects of ozone and other photochemical oxidants in field studies with a mobile laboratory	1-152

LIST OF FIGURES

<u>Figure</u>	<u>Page</u>
1-1 National trend in composite average of the second highest value among daily maximum 1-hour ozone concentrations at selected groups of sites, 1975 through 1983	1-40
1-2 Distributions of the three highest 1-hour ozone concentrations at valid sites (906 station-years) aggregated for 3 years (1979, 1980, and 1981) and the highest ozone concentrations at NAPBN sites aggregated for those years (24 station-years)	1-42
1-3 Relationship between ozone concentration, exposure duration, and a reduction in plant growth or yield	1-59
1-4 Examples of the effects of ozone on the yield of soybean and wheat cultivars	1-62
1-5 Examples of the effects of ozone on the yield of cotton, tomato, and turnip	1-63
1-6 Number and percentage of 37 crop species or cultivars predicted to show a 10 percent yield loss at various ranges of 7-hr seasonal mean ozone concentrations	1-66
1-7 Summary of morphological effects in experimental animals exposed to ozone	1-111
1-8 Summary of effects of short-term ozone exposures on pulmonary function in experimental animals	1-115
1-9 Summary of effects of long-term ozone exposures on pulmonary function in experimental animals	1-117
1-10 Summary of biochemical changes in experimental animals exposed to ozone	1-123
1-11 Summary of effects of ozone on host defense mechanisms in experimental animals	1-128
1-12 Summary of extrapulmonary effects of ozone in experimental animals	1-137
1-13 Summary of effects in experimental animals exposed to ozone combined with other pollutants	1-139
1-14 Group mean decrements in 1-sec forced expiratory volume during 2-hr ozone exposures with different levels of intermittent exercise	1-158

LIST OF ABBREVIATIONS

AChE	acetylcholinesterase
avg	average
BAKI	boric acid buffered potassium iodide
Be (⁷ Be)	beryllium (radioactive isotope of beryllium)
C	carbon, concentration
°C	degrees Celsius
CA	chromotropic acid
CC	closing capacity
CH ₃ C(O)O ₂	acetylperoxy radical
cm	centimeter
CNS	central nervous system
CO	carbon monoxide
CO ₂	carbon dioxide
COLD	chronic obstructive lung disease
conc., concn.	concentration
CV	closing volume
dbh	diameter at breast height
DNPH	2,4-dinitrophenylhydrazine
ECD	electron-capture detector
EDU	ethylenediurea
EKMA	Empirical Kinetic Modeling Approach
FEF	forced expiratory flow
Fe ₂ (SO ₄) ₃	ferric sulfate
FEV	forced expiratory volume
FEV ₁	forced expiratory volume in 1 sec
FID	flame ionization detector
f _R	respiratory frequency
FTIR	Fourier-transform infrared
FVC	forced vital capacity
G-6-PD	glucose-6-phosphate dehydrogenase
GC	gas chromatography
GPT	gas-phase titration

LIST OF ABBREVIATIONS
(continued)

GSH	glutathione
HC	hydrocarbon(s)
HCOOH	formic acid
HNO ₃	nitric acid
HNO ₄	peroxynitric acid
HO	hydroxy
HO ₂	hydroperoxy
HONO	nitrous acid
HPLC	high-pressure liquid chromatography; high-performance liquid chromatography
HPPA	3-(p-hydroxyphenyl)propionic acid
hr	hour(s)
hr/day	hours per day
HRP	horseradish peroxidase
H ₂ O ₂	hydrogen peroxide
H ₂ SO ₄	sulfuric acid
I	impact
I ⁻	iodide ion
IC	inspiratory capacity
I/O	ratio of indoor to outdoor ozone concentrations
IR	infrared
KIO ₃	potassium iodate
km	kilometer
LAAPCD	Los Angeles Air Pollution Control District
LCV	leuco crystal violet
LDH	lactate dehydrogenase
L/min	liters per minute
M	molar
m	meter(s)
MBTH	3-methyl-2-benzothiazolinone hydrazone
mi	mile(s)

LIST OF ABBREVIATIONS

(continued)

NADPH	nicotinamide adenine dinucleotide phosphate
NAPBN	National Air Pollution Background Network
NBKI	neutral buffered potassium iodide
$(\text{NH}_4)_2\text{SO}_4$	ammonium sulfate
NF	National Forest
nm	nanometer(s)
NMHC	nonmethane hydrocarbons
NMOC	nonmethane organic compounds
NO	nitric oxide
NO_2	nitrogen dioxide
NO_3	nitrogen trioxide
NO_x	nitrogen oxides
ΔN_2	nitrogen washout
NPSH	non-protein sulfhydryls
NR	natural rubber
N_2O	nitrous oxide
OH	hydroxyl group (or radical)
O_2	oxygen
O_3	ozone
OZIPP	Ozone Isopleth Plotting Package
PAN	peroxyacetyl nitrate
$P_A\text{O}_2$	alveolar partial pressure of oxygen
PBzN	peroxybenzoyl nitrate
PEFR	peak expiratory flow rate
pH	negative log of H ⁺ ion concentration
PPN	peroxypropionyl nitrate
ppb	parts per billion
ppm	parts per million
rad	radiation absorbed dose
RBC	red blood cell
RV	residual volume

LIST OF ABBREVIATIONS

(continued)

SaO ₂	arterial oxygen saturation
SAROAD	Storage and Retrieval of Aerometric Data
SBR	styrene-butadiene rubber
sec	second(s)
SG _{aw}	specific airway conductance
SNAAQs	Secondary National Ambient Air Quality Standards
SO ₂	sulfur dioxide
SO ₄	sulfate
SO _x	sulfur oxide(s)
SR _{aw}	specific airway resistance
SRM	Standard Reference Material
SURE	Sulfate Regional Experiment Sites
T	time, temperature
TF	tropopause-folding events
Tg/yr	teragrams per year
TGS-ANSA	triethanolamine, guaiacol(<i>o</i> -methoxyphenol), sodium metabisulfite; and 8-anilino-1-naphthalene sulfonic acid
TLC	total lung capacity
TSH	thyroid stimulating hormone
µg/m ³	microgram(s) per cubic meter
µm/hr	micrometer(s) per hour
UV	ultraviolet
V _T	tidal volume
\dot{V}_E	minute ventilation; expired volume per minute
VOC	volatile organic compounds
ZnSO ₄	zinc sulfate

AUTHORS, CONTRIBUTORS, AND REVIEWERS

Authors:

Dr. Richard M. Adams
Department of Agricultural and Resource Economics
Oregon State University
Corvallis, OR 97331

Dr. Donald E. Gardner
Northrop Services, Inc.
Environmental Sciences
P. O. Box 12313
Research Triangle Park, NC 27709

Dr. J. H. B. Garner
Environmental Criteria and Assessment Office
MD-52
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

Dr. Judith A. Graham
Health Effects Research Laboratory
MD-51
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

Dr. Milan J. Hazucha
School of Medicine
Center for Environmental Health and Medical Sciences
University of North Carolina
Chapel Hill, NC 27514

Dr. Jimmie A. Hodgeson
U.S. Environmental Protection Agency
Environmental Monitoring Systems Laboratory
26 West St. Clair
Cincinnati, OH 45268

Mr. Michael W. Holdren
Battelle, Columbus Laboratories
505 King Avenue
Columbus, OH 43201

Dr. Donald H. Horstman
Health Effects Research Laboratory
MD-58
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

Authors (continued):

Mr. James M. Kawecki
TRC Environmental Consultants, Inc.
2001 Wisconsin Avenue, N.W.
Suite 261
Washington, DC 20007

Dr. Jan G. Laarman
Department of Forestry
North Carolina State University
Raleigh, NC 27607

Mr. Thomas B. McMullen
Environmental Criteria and Assessment Office
MD-52
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

Dr. Daniel B. Menzel
Laboratory of Environmental Toxicology and Pharmacology
Duke University Medical Center
P. O. Box 3813
Durham, NC 27710

Mr. James A. Raub
Environmental Criteria and Assessment Office
MD-52
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

Ms. Beverly E. Tilton
Environmental Criteria and Assessment Office
MD-52
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

Dr. David T. Tingey
Environmental Research Laboratory
200 SW 35th Street
Corvallis, OR 97330

Dr. Halvor Westberg
Director, Laboratory for Atmospheric Research,
and Professor, Civil and Environmental Engineering
Washington State University
Pullman, WA 99164-2730

Dr. Arthur M. Winer
Assistant Director
Statewide Air Pollution Research Center
University of California
Riverside, CA 92521

Contributing Authors:

Dr. Robert Frank
Department of Environmental Health Sciences
Johns Hopkins School of Hygiene and Public Health
615 N. Wolfe Street
Baltimore, MD 21205

Dr. Michael D. Lebowitz
Department of Internal Medicine
College of Medicine
University of Arizona
Tucson, AZ 85724

Dr. Harold G. Richter
Office of Air Quality Planning and Standards
Monitoring and Data Analysis Division
MD-14
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

Mr. Elmer Robinson
Director, Mauna Loa Observatory
National Oceanic and Atmospheric Administration
(NOAA/CMCO)
P.O. Box 275
Hilo, HI 96720

SCIENCE ADVISORY BOARD
CLEAN AIR SCIENTIFIC ADVISORY COMMITTEE

The substance of this document was reviewed by the Clean Air Scientific Advisory Committee of the Science Advisory Board in public sessions.

SUBCOMMITTEE ON OZONE

Chairman

Dr. Morton Lippmann
Professor
Department of Environmental Medicine
New York University Medical Center
Tuxedo, New York 10987

Members

Dr. Mary O. Amdur
Senior Research Scientist
Energy Laboratory
Massachusetts Institute of Technology
Cambridge, Massachusetts 02139

Dr. Eileen G. Brennan
Professor
Department of Plant Pathology
Martin Hall, Room 213, Lipman Drive
Cook College-NJAES
Rutgers University
New Brunswick, New Jersey 08903

Dr. Edward D. Crandall
Professor of Medicine
School of Medicine
Cornell University
New York, New York 10021

Dr. James D. Crapo
Associate Professor of Medicine
Chief, Division of Allergy, Critical
Care and Respiratory Medicine
Duke University Medical Center
Durham, North Carolina 27710

Dr. Robert Frank
Professor of Environmental Health
Sciences
Johns Hopkins School of Hygiene
and Public Health
615 N. Wolfe Street
Baltimore, Maryland 21205

Professor A. Myrick Freeman II
Department of Economics
Bowdoin College
Brunswick, Maine 04011

Dr. Ronald J. Hall
Senior Research Scientist and Leader
Aquatic and Terrestrial Ecosystems
Section
Ontario Ministry of the Environment
Dorset Research Center
Dorset, Ontario
Canada POA1E0

Dr. Jay S. Jacobson
Plant Physiologist
Boyce Thompson Institute
Tower Road
Ithaca, New York 14853

Dr. Warren B. Johnson
Director, Atmospheric Science Center
SRI International
333 Ravenswood Avenue
Menlo Park, California 94025

Dr. Jane Q. Koenig
Research Associate Professor
Department of Environmental Health
University of Washington
Seattle, Washington 98195

Dr. Paul Kotin
Adjunct Professor of Pathology
University of Colorado Medical School
4505 S. Yosemite, #339
Denver, Colorado 80237

Dr. Timothy Larson
Associate Professor
Environmental Engineering and
Science Program
Department of Civil Engineering
University of Washington
Seattle, Washington 98195

Professor M. Granger Morgan
Head, Department of Engineering
and Public Policy
Carnegie-Mellon University
Pittsburgh, Pennsylvania 15253

Dr. D. Warner North
Principal
Decision Focus Inc., Los Altos
Office Center, Suite 200
4984 El Camino Real
Los Altos, California 94022

Dr. Robert D. Rowe
Vice President, Environmental and
Resource Economics
Energy and Resources Consultants, Inc.
207 Canyon Boulevard
Boulder, Colorado 80302

Dr. George Taylor
Environmental Sciences Division
P.O. Box X
Oak Ridge National Laboratory
Oak Ridge, Tennessee 37831

Dr. Michael Treshow
Professor
Department of Biology
University of Utah
Salt Lake City, Utah 84112

Dr. Mark J. Utell
Co-Director, Pulmonary Disease Unit
Associate Professor of Medicine and
Toxicology in Radiation Biology
and Biophysics
University of Rochester Medical
Center
Rochester, New York 14642

Dr. James H. Ware
Associate Professor
Harvard School of Public Health
Department of Biostatistics
677 Huntington Avenue
Boston, Massachusetts 02115

Dr. Jerry Wesolowski
Air and Industrial Hygiene Laboratory
California Department of Health
2151 Berkeley Way
Berkeley, California 94704

Dr. James L. Whittenberger
Director, University of California
Southern Occupational Health Center
Professor and Chair, Department of
Community and Environmental Medicine
California College of Medicine
University of California - Irvine
19772 MacArthur Boulevard
Irvine, California 92717

Dr. George T. Wolff
Senior Staff Research Scientist
General Motors Research Labs
Environmental Science Department
Warren, Michigan 48090

PROJECT TEAM FOR DEVELOPMENT
OF
Air Quality Criteria for Ozone and Other Photochemical Oxidants

Ms. Beverly E. Tilton, Project Manager
and Coordinator for Chapters 1 through 5, Volumes I and II
Environmental Criteria and Assessment Office (MD-52)
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

Mr. Norman E. Childs
Environmental Criteria and Assessment Office (MD-52)
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

Dr. J.H.B. Garner
Coordinator for Chapters 7 and 8, Volume III
Environmental Criteria and Assessment Office (MD-52)
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

Mr. Thomas B. McMullen
Environmental Criteria and Assessment Office (MD-52)
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

Mr. James A. Raub
Coordinator for Chapters 9 through 12, Volumes IV and V
Environmental Criteria and Assessment Office (MD-52)
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

Dr. David T. Tingey
Coordinator for Chapter 6, Volume III
Environmental Research Laboratory
U.S. Environmental Protection Agency
200 S.W. 35th Street
Corvallis, OR 97330

1. SUMMARY AND CONCLUSIONS

1.1 INTRODUCTION

This document is a revision of Air Quality Criteria for Ozone and Other Photochemical Oxidants, published in 1978 (U.S. Environmental Protection Agency, 1978). Its purpose is to provide the scientific basis for the derivation of National Ambient Air Quality Standards (NAAQS) by consolidating and assessing knowledge regarding the origin and distribution of ozone and other photochemical oxidants and the effects of these pollutants on humans, experimental animals, vegetation, terrestrial ecosystems, and nonbiological materials. Because the indirect contributions of the photochemical oxidants to visibility degradation, climatic changes, and acidic deposition cannot at present be quantified, these atmospheric effects and phenomena are not addressed in this document. They have been addressed, however, in other, recent air quality criteria documents (U.S. Environmental Protection Agency, 1982a,b).

Research has established that photochemical oxidants in ambient air consist mainly of ozone, peroxyacetyl nitrate, and nitrogen dioxide, and of considerably lesser amounts of other peroxyacyl nitrates, hydrogen peroxide, alkyl hydroperoxides, nitric and nitrous acids, and formic acid. Other oxidants suspected to occur in ambient air but only in trace amounts include peracids and ozonides. Only data on ozone, peroxyacyl nitrates, hydrogen peroxide, and formic acid are examined in this document. Coverage has been limited to these photochemical oxidants on the basis of available information on effects, ambient air concentrations, or both. Of these oxidants, only ozone and peroxyacetyl nitrate have been studied at concentrations having relevance for potential exposures of human populations or of vegetation, ecosystems, or nonbiological materials. Although by definition a photochemical oxidant, nitrogen dioxide is not included among the oxidants discussed in this document. Separate criteria documents are issued for oxides of nitrogen, and the second document in that series, completed in 1982, presented information on nitrosamines and inorganic nitrogen acids, as well as the oxides of nitrogen (U.S. Environmental Protection Agency, 1982a).

This document presents a review and evaluation of relevant literature on ozone and other photochemical oxidants published through early 1986. The document is not intended as a complete literature review, however; but is

intended, rather, to present current data of probable consequence for the derivation of national ambient air quality standards for protecting public health and welfare. The scientific information selected for review and comment in the text generally came from the more recent literature, with emphasis on studies conducted at or near pollutant concentrations found in ambient air. Generally, only published material that has undergone scientific peer review has been included. In the interest of admitting new and important information, however, some material not published in the open literature but meeting other standards of scientific reporting may have been included. In addition, the studies reviewed in the health- and welfare-related chapters met other selection criteria, including the appropriate use and satisfaction of statistical tests.

In the early chapters of this document, an overview is presented of the nature, origins, and distribution in ambient air of those organic and inorganic compounds that serve as precursors to ozone and other photochemical oxidants. The currently available measurement techniques for these precursors are briefly evaluated, inasmuch as the assessment of the occurrence of the precursors depends upon their accurate measurement. Similarly, an overview is presented of the chemical and physical processes in the atmosphere by which precursors give rise to the production of ozone and other photochemical oxidants. In addition, the properties of ozone and other photochemical oxidants are presented as background for understanding information presented in the chapters on health and welfare effects. Likewise, techniques for the measurement of ozone, total oxidants, and individual oxidant species other than ozone are evaluated, since the significance of aerometric and exposure data on these pollutants is dependent upon the accuracy and specificity of the analytical techniques used. Typical concentrations of the respective oxidants are presented to permit assessment of potential exposures of human populations and other receptors.

Remaining chapters of the document contain the actual air quality criteria; that is, quantitative and qualitative information that describes the nature of the health and welfare effects attributable to ozone and other photochemical oxidants and the concentrations at which these pollutants are thought to produce the observed effects.

Neither techniques nor strategies for the abatement of photochemical oxidants are reviewed in this document. Technology for controlling the emissions of nitrogen oxides and of volatile organic compounds is discussed in documents

issued by the Office of Air Quality Planning and Standards (OAQPS) of the U.S. Environmental Protection Agency (e.g., U.S. Environmental Protection Agency, 1978b, 1983). Likewise, research findings and issues germane to the scientific basis for control strategies are addressed in numerous documents issued by OAQPS and by the Office of Research and Development.

In addition, certain issues of direct relevance to standard-setting are not explicitly addressed in this document, but are addressed instead in documentation prepared by OAQPS as part of its regulatory analyses. Such analyses include: (1) discussion of what constitutes an "adverse effect," that is, the effect or effects the NAAQS are intended to protect against; (2) assessment of risk; and (3) discussion of factors to be considered in providing an adequate margin of safety. While scientific data contribute significantly to decisions regarding these three issues, their resolution cannot be achieved solely on the basis of experimentally acquired information. Final decisions on items (1) and (3) are made by the Administrator of the U.S. Environmental Protection Agency.

The legislative basis for the development and issuance of the air quality criteria and related information presented in this document is found in Sections 108 and 109 of the Clean Air Act (U.S.C., 1982).

1.2 PROPERTIES, CHEMISTRY, AND TRANSPORT OF OZONE AND OTHER PHOTOCHEMICAL OXIDANTS AND THEIR PRECURSORS

1.2.1 Descriptions and Properties of Ozone and Other Photochemical Oxidants

Ozone (O_3) and other photochemical oxidants occurring at low concentrations in ambient air, such as peroxyacetyl nitrate (PAN), hydrogen peroxide (H_2O_2), and formic acid (HCOOH), are characterized chiefly by their ability to remove electrons from, or to share electrons with, other molecules or ions (i.e., oxidation). The capability of a chemical species for oxidizing or reducing other chemical species is termed "redox potential" (positive or negative standard potential) and is expressed in volts. A reactive allotrope of oxygen that is only about one-tenth as soluble as oxygen in water, ozone has a standard potential of +2.07 volts in aqueous systems for the redox pair, O_3/H_2O (Weast, 1977). Hydrogen peroxide, which is highly soluble in water and other polar solvents, has a standard potential of +1.776 in the redox pair, H_2O_2/H_2O (Weast, 1977). No standard potential for peroxyacetyl nitrate in neutral or buffered aqueous systems, such as those that occur in biological

systems, appears in the literature. In acidic solution (pH 5 to 6), PAN hydrolyzes fairly rapidly (Lee et al., 1983; Holdren et al., 1984); in alkaline solution it decomposes with the production of nitrite ion and molecular oxygen (Stephens, 1967; Nicksic et al., 1967). An important property of PAN, especially in the laboratory, is its thermal instability. Its explosiveness dictates its synthesis for experimental and calibration purposes by experienced personnel only.

Formic acid is formed as a stable product in photochemical air pollution. It has the structure of both an acid and an aldehyde and in concentrated form is a pungent-smelling, highly corrosive liquid.

The toxic effects of oxidants are attributable to their oxidizing ability. Their oxidizing properties also form the basis of several measurement techniques for O_3 and PAN. The calibration of ozone and PAN measurements, however, is achieved via their spectra in the ultraviolet and infrared regions, respectively. All three pollutants of most concern in this document (O_3 , PAN, and H_2O_2) must be generated in situ for the calibration of measurement techniques. For ozone and H_2O_2 , generation of calibration gases is reasonably straightforward.

1.2.2 Nature of Precursors to Ozone and Other Photochemical Oxidants

Photochemical oxidants are products of atmospheric reactions involving volatile organic compounds (VOC) and oxides of nitrogen (NO_x), as well as hydroxyl (OH) and other radicals, oxygen, and sunlight (see, e.g., Demerjian et al., 1974; National Research Council, 1977; U.S. Environmental Protection Agency, 1978; Atkinson, 1985). The oxidants are largely secondary pollutants formed in the atmosphere from their precursors by processes that are a complex, non-linear function of precursor emissions and meteorological factors.

The properties of organic compounds that are most relevant to their role as precursors to ozone and other oxidants are their volatility, which governs their emissions into the atmosphere; and their chemical reactivity, which determines their lifetime in the atmosphere. Although vapor-phase hydrocarbons (compounds of carbon and hydrogen only) are the predominant organic compounds in ambient air that serve as precursors to photochemical oxidants, other volatile organic compounds are also photochemically reactive in those atmospheric processes that give rise to oxidants. In particular, halogenated organics (e.g., haloalkenes) that participate in photochemical reactions are

present in ambient air, although at lower concentrations than the hydrocarbons. They are oxidized through the same initial step involved in the oxidation of the hydrocarbons; that is, attack by hydroxyl radicals. Alkenes, haloalkenes, and aliphatic aldehydes are, as classes, among the most reactive organic compounds found in ambient air (e.g., Altshuller and Bufalini, 1971; Darnall et al., 1976; Pitts et al., 1977; U.S. Environmental Protection Agency, 1978, and references therein). Alkenes and haloalkenes are unique among VOC in ambient air in that they are susceptible both to attack by OH radicals (OH) and by ozone (Niki et al., 1983). Methane, halomethanes, and certain haloethenes are of negligible reactivity in ambient air and have been classed as unreactive by the U.S. Environmental Protection Agency (1980a,b). Since methane is considered only negligibly reactive in ambient air, the volatile organic compounds of importance as oxidant precursors are usually referred to as nonmethane hydrocarbons (NMHC) or, more properly, as nonmethane organic compounds (NMOC).

The oxides of nitrogen that are important as precursors to ozone and other photochemical oxidants are nitrogen dioxide (NO_2) and nitric oxide (NO). Nitrogen dioxide is itself an oxidant that produces deleterious effects, which are the subject of a separate criteria document (U.S. Environmental Protection Agency, 1982). Nitrogen dioxide is an important precursor (1) because its photolysis in ambient air leads to the formation of oxygen atoms that combine with molecular oxygen to form ozone; and (2) because it reacts with acetylperoxy radicals to form peroxyacetyl nitrate, a phytotoxicant and a lachrymator. Although ubiquitous, nitrous oxide (N_2O) is unimportant in the production of oxidants in ambient air because it is virtually inert in the troposphere.

1.2.3 Atmospheric Reactions of Ozone and Other Oxidants Including Their Role in Aerosol Formation

The chemistry of the polluted atmosphere is exceedingly complex, but an understanding of the basic phenomena is not difficult to acquire. Three processes occur: the emission of precursors to ozone from predominantly manmade sources; photochemical reactions that take place during the dispersion and transport of these precursors; and scavenging processes that reduce the concentrations of both O_3 and precursors along the trajectory.

The specific atmospheric reactions of ozone and of other photochemical oxidants such as peroxyacetyl nitrate and hydrogen peroxide are becoming

increasingly well-characterized. The reactions of these species result in products and processes that may have significant environmental and health- and welfare-related implications, including effects on biological systems, nonbiological materials, and such phenomena as visibility degradation and acidification of cloud and rain water.

1.2.3.1 Formation and Transformation of Ozone and Other Photochemical Oxidants. In the troposphere, ozone is formed through the dissociation of NO_2 by sunlight to yield an oxygen atom, which then reacts with molecular oxygen (O_2) to produce an O_3 molecule. If it is present, NO can react rapidly with O_3 to form NO_2 and an O_2 molecule. In the absence of competing reactions, a steady-state or equilibrium concentration of O_3 is soon established between O_3 , NO_2 , and NO (National Research Council, 1977). The injection of organic compounds into the atmosphere upsets the equilibrium and allows the ozone to accumulate at higher than steady-state concentrations. The length of the induction period before the accumulation of O_3 begins depends heavily on the initial NO/NO_2 and NMOC/NO_x ratios (National Research Council, 1977).

The major role played by organic compounds in smog reactions is attributable to the hydroxyl radical (OH), since it reacts with essentially all organic compounds (e.g., Atkinson, 1985; Herron and Huie, 1977, 1978; Dodge and Arnts, 1979; Niki et al., 1981). Aldehydes, which are constituents of automobile exhaust as well as decomposition products of most atmospheric photochemical reactions involving hydrocarbons, and nitrous acid (HONO), are important sources of OH radicals, as is O_3 itself. Other free radicals, such as hydro- and alkylperoxy radicals and the nitrate (NO_3) radical play important roles in photochemical air pollution.

The presence of organic compounds, oxides of nitrogen, and sunlight does not mean that the photochemical reactions will continue indefinitely. Termination reactions gradually remove NO_2 from the reaction mixtures, such that the photochemical cycles slowly come to an end unless fresh NO and NO_2 emissions are injected into the atmosphere. Compounds containing nitrogen, such as PAN, nitric acid (HNO_3), and peroxyntic acid (HNO_4), as well as organic and inorganic nitrates, are formed in these termination reactions.

Recent studies on the photooxidation of organic compounds under simulated atmospheric conditions have been reasonably successful. The rate constants for the reaction of OH radicals with a large number of organic compounds have been measured (e.g., Atkinson et al., 1979; Atkinson et al., 1985). The

mechanisms of the reactions of paraffinic compounds are fairly well understood, as are those of olefinic compounds, at least for the smaller compounds. Photooxidation reactions of the aromatic compounds, however, are poorly understood.

In the presence of NO_x , natural hydrocarbons (i.e., those organic compounds emitted from vegetation) can also undergo photooxidation reactions to yield O_3 , although most naturally emitted hydrocarbons are olefins and are scavengers as well as producers of O_3 (e.g., Lloyd et al., 1983; Atkinson et al., 1979; Kamens et al., 1982; Killus and Whitten, 1984; Atkinson and Carter, 1984).

1.2.3.2 Atmospheric Chemical Processes Involving Ozone. Ozone can react with organic compounds in the boundary layer of the troposphere (Atkinson and Carter, 1984). It is important to recognize, however, that organics undergo competing reactions with OH radicals in the daytime (Atkinson et al., 1979; Atkinson, 1985) and, in certain cases, with NO_3 radicals during the night (Japar and Niki, 1975; Carter et al., 1981a; Atkinson et al., 1984a,b,c,d,e; Winer et al., 1984), as well as photolysis, in the case of aldehydes and other oxygenated organics. Only for organics whose ozone reaction rate constants are greater than $\sim 10^{-21} \text{ cm}^3 \text{ molecule}^{-1} \text{ sec}^{-1}$ can consumption by ozone be considered to be atmospherically important (Atkinson and Carter, 1984).

Ozone reacts rapidly with the acyclic mono-, di-, and tri-alkenes and with cyclic alkenes. The rate constants for these reactions range from $\sim 10^{-18}$ to $\sim 10^{-14} \text{ cm}^3 \text{ molecule}^{-1} \text{ sec}^{-1}$ (Atkinson and Carter, 1984), corresponding to atmospheric lifetimes ranging from a few minutes for the more reactive cyclic alkenes, such as the monoterpenes, to several days. In polluted atmospheres, a significant portion of the consumption of the more reactive alkenes will occur via reaction with ozone rather than with OH radicals, especially in the afternoons during photochemical oxidant episodes. Reactions between ozone and alkenes can result in aerosol formation (National Research Council, 1977; Schuetzle and Rasmussen, 1978), with alkenes of higher carbon numbers the chief contributors.

Because of their respective rate constants, neither alkanes (Atkinson and Carter, 1984) nor alkynes (Atkinson and Aschmann, 1984) are expected to react with ozone in the atmosphere, since competing reactions with OH radicals have higher rate constants (Atkinson et al., 1979; Atkinson, 1985).

The aromatics react with ozone, but quite slowly (Atkinson and Carter, 1984), such that their reactions with ozone are expected to be unimportant in the atmosphere. Cresols are more reactive toward ozone than the aromatic hydrocarbons (Atkinson and Carter, 1984), but their reactions with OH radicals (Atkinson, 1985) or NO_3 radicals (Carter et al., 1981a; Atkinson et al., 1984d) predominate.

For oxygen-containing organic compounds, especially those without carbon-carbon double bonds, reactions with ozone are slow. For carbonyls and ethers (other than ketene) that contain unsaturated carbon-carbon bonds, however, much faster reactions are observed (Atkinson and Carter, 1984).

Certain reactions of ozone other than its reactions with organic compounds are important in the atmosphere. Ozone reacts rapidly with NO to form NO_2 , and subsequently with NO_2 to produce the nitrate (NO_3) radical and an oxygen molecule. Photolysis of ozone can be a significant pathway for the formation of OH radicals, particularly in polluted atmospheres when ozone concentrations are at their peak.

Ozone may play a role in the oxidation of SO_2 to H_2SO_4 , both indirectly in the gas phase (via formation of OH radicals and Criegee biradicals) and directly in aqueous droplets.

1.2.3.3 Atmospheric Reactions of PAN, H_2O_2 , and HCOOH . Because PAN is in equilibrium with acetyl peroxy radicals and NO_2 , any process that leads to the removal of either of these species will lead to the decomposition of PAN. One such process is the reaction of NO with acetyl peroxy radicals. This can lead, however, to the formation of OH radicals. Thus, PAN remaining overnight from an episode on the previous day can react with NO emitted from morning traffic to produce OH radicals (Cox and Roffey, 1977; Carter et al., 1981b) that will enhance smog formation on that day (e.g., Tuazon et al., 1981). In the absence of significant NO concentrations, and in regions of moderate to lower temperatures, PAN will persist in the atmosphere (Wallington et al., 1984; Aikin et al., 1983) and contribute to the long-range transport of NO_x .

Although hydrogen peroxide formed in the gas phase from the reactions of hydroperoxyl radicals plays a role in HO_x chemistry in the troposphere, and especially in the stratosphere (Crutzen and Fishman, 1977; Cox and Burrows, 1979), its major importance arises from its high solubility in water. The latter ensures that a large fraction of gaseous H_2O_2 will be taken up in aqueous droplets. Over the past decade, evidence has accumulated that H_2O_2

dissolved in cloud, fog, and rainwater may play an important, and, in acidic droplets (i.e., $\text{pH} \lesssim 5$), even a dominant role in the oxidation of SO_2 to H_2SO_4 (e.g., Hoffman and Edwards, 1975; Martin and Damschen, 1981; Chameides and Davis, 1982; Calvert and Stockwell, 1983, 1984; Schwartz, 1984). Hydrogen peroxide may also play a role in the oxidation of NO_2 dissolved in aqueous droplets, although relevant data are limited and additional research is required (see, e.g., Gertler et al., 1984). Substantial uncertainties remain concerning the quantitative role of H_2O_2 in acidification of aqueous particles and droplets (Richards et al., 1983).

Because it can be scavenged rapidly into water droplets, formic acid can potentially function as an oxidant in cloud water and rain water. Thus, HCOOH is an example of a compound that is a non-oxidant or weak oxidant in the gas phase but that is transformed upon incorporation in aqueous solutions into an effective oxidizer of S(IV) . Although much uncertainty remains concerning the quantitative role of HCOOH and the higher organic acids, they potentially play a minor but still significant role in the acidification of rain.

1.2.4 Meteorological and Climatological Processes

Meteorological and climatological processes are important in determining the extent to which precursors to ozone and other photochemical oxidants can accumulate, and thereby the concentrations of ozone and other oxidants that can result. The meteorological factors most important in the formation and transport of ozone and other photochemical oxidants in the lower troposphere are: (1) degree of atmospheric stability; (2) wind speed and direction; (3) intensity and wavelength of sunlight; and (4) synoptic weather conditions. These factors are in turn dependent upon or interrelated with geographic, seasonal, and other climatological factors.

Incursions of ozone from the stratosphere are an additional source of the ozone found in the lower troposphere. The physical and meteorological mechanisms by which ozone is brought into the troposphere from the stratosphere are important in determining the resulting ground-level concentrations, ground-level locations impacted, and the seasonality of incursions of stratospheric ozone.

1.2.4.1 Atmospheric Mixing. The concentration of a pollutant in ambient air depends significantly on the degree of atmospheric mixing that occurs from the time the pollutant or its precursors are emitted and the arrival of the pollutant at the receptor. The rate at which atmospheric mixing proceeds and the

extent of the final dilution depends on the amount of turbulent mixing that occurs and on wind speed and direction. Atmospheric stability is one of the chief determinants of turbulent mixing since pollutants do not spread rapidly within stable layers nor do they mix upward through stable layers to higher altitudes.

Temperature inversions, in which the temperature increases with increasing altitude, represent the most stable atmospheric conditions. Surface inversions (base at ground level) and elevated inversions (the entire layer is above the surface) are both common (Hosler, 1961; Holzworth, 1964) and both can occur simultaneously at the same location. Surface inversions show a diurnal pattern, forming at night in the absence of solar radiation but breaking up by about mid-morning as the result of surface heating by the sun (Hosler, 1961; Slade, 1968). Elevated inversions can persist throughout the day and pollutants can be trapped between the ground surface and the base of the inversion. The persistence of elevated inversions is a major meteorological factor contributing to high pollutant concentrations and photochemical smog conditions along the California coast (Hosler, 1961; Holzworth, 1964; Robinson, 1952). In coastal areas generally, such as the New England coast (Hosler, 1961) and along the Great Lakes (Lyons and Olsson, 1972), increased atmospheric stability (and diminished mixing) occurs in summer and fall as the result of the temperature differential between the water and the land mass.

The depth of the layer in which turbulent mixing can occur (i.e., the "mixing height") shows geographical dependence. Summer morning mixing heights are usually >300 m in the United States except for the Great Basin (part of Oregon, Idaho, Utah, Arizona, and most of Nevada), where the mixing height is ~200 m (Holzworth, 1972). By mid-morning, mixing heights increase markedly such that only a few coastal areas have mixing heights <1000 m.

Summer afternoon mixing heights are generally an indication of the potential for recurring photochemical oxidant problems. Photochemical smog problems in the United States are somewhat unexpected since the lowest afternoon mixing height is ~600 m (Holzworth, 1972). Elevated inversions having bases <500 m (i.e., low-level inversions) occur in the United States, however, with the following frequencies: 90 percent on the California coast; \geq 20 percent on the Atlantic coast (New Jersey to Maine); \geq 5 percent along the Great Lakes; and 5 to 10 percent from Louisiana to Arkansas and eastward to about Atlanta, Georgia. For most areas of the United States, though, the persistence through the

afternoon of low-level stable layers is a rare event, occurring on <1 day in 20 (Holzworth and Fisher, 1979).

1.2.4.2 Wind Speed and Direction. For areas in which mixing heights are not restrictive, wind speed and, in some cases, wind direction are major determinants of pollution potential. Since strong winds dilute precursors to ozone and other photochemical oxidants, a location may have good ventilation despite the occurrence of persistent inversions (e.g., San Francisco). Conversely, light winds can result in high oxidant levels even if the mixing layer is deep.

The frequency of weak winds, then, is important in oxidant formation. In industrialized, inland areas east of the Mississippi River, surface inversions in the morning coupled with wind speeds ≤ 2.5 m/sec (≤ 6 mi/hr) occur with a frequency ≥ 50 percent (Holzworth and Fisher, 1979). These surface inversions break up by afternoon, however, permitting dispersion.

The effects of wind speed and direction include the amount of dilution occurring in the source areas, as well as along the trajectory followed by an urban or source-area plume. Regions having steady prevailing winds, such that a given air parcel can pass over a number of significant source areas, can develop significant levels of pollutants even in the absence of weather patterns that lead to the stagnation type of air pollution episodes. The Northeast states are highly susceptible to pollutant plume transport effects, although some notable stagnation episodes have also affected this area (e.g., Lynn et al., 1964). Along the Pacific Coast, especially along the coast of California, coastal winds and a persistent low inversion layer contribute to major pollutant buildups in urban source areas and downwind along the urban plume trajectory (Robinson, 1952; Neiburger et al., 1961).

1.2.4.3 Effects of Sunlight and Temperature. The effects of sunlight on photochemical oxidant formation, aside from the role of solar radiation in meteorological processes, are related to its intensity and its spectral distribution. Intensity varies diurnally, seasonally, and with latitude, but the effect of latitude is strong only in the winter. Experimental studies have verified the effects on oxidant formation of light intensity (Peterson, 1976; Demerjian et al., 1980) and its diurnal variations (Jeffries et al., 1975; 1976), as well as on the overall photooxidation process (Jaffee et al., 1974; Winer et al., 1979).

A correlation between high oxidant concentrations and warm, above-normal temperatures has been demonstrated generally (Bach, 1975; Wolff and Lioy, 1978) and for specific locations, e.g., St. Louis (Shreffler and Evans, 1982). Coincident meteorology appears to be the cause of the observed correlation. Certain synoptic weather conditions are favorable both for the occurrence of higher temperatures and for the formation of ozone and other oxidants, so that temperature is often used to forecast the potential for high oxidant concentrations (e.g., Wolff and Lioy, 1978; Shreffler and Evans, 1982). Data from smog chamber studies show an effect of temperature on ozone formation (e.g., Carter et al., 1979; Countess et al., 1981), but the effect is thought to result from the volatilization and reaction of chamber wall contaminants as the temperature is increased.

1.2.4.4 Transport of Ozone and Other Oxidants and Their Precursors. The levels of ozone and other oxidants that will occur at a given receptor site downwind of a precursor source area depend upon many interrelated factors, which include but are not restricted to: (1) the concentrations of respective precursors leaving the source area; (2) induction time; (3) turbulent mixing; (4) wind speed and wind direction; (5) scavenging during transport; (6) injection of new emissions from source areas in the trajectory of the air mass; and (7) local and synoptic weather conditions.

Ozone and other photochemical oxidants can be transported hundreds of miles from the place of origin of their precursors, as documented by the numerous studies on transport phenomena that were described in the 1978 criteria document for ozone and other photochemical oxidants (U.S. Environmental Protection Agency, 1978). In that document, transport phenomena were classified into three categories, depending upon transport distance: urban-scale, mesoscale, and synoptic-scale. In urban-scale transport, maximum concentrations of O_3 are produced about 20 miles or so (and about 2 to 3 hours) downwind from the major pollutant source areas. In mesoscale transport, O_3 has been observed up to 200 miles downwind from the sources of its precursors. Synoptic-scale transport is associated with large-scale, high-pressure air masses that may extend over and persist for many hundreds of miles.

Urban-scale transport has been identified as a significant, characteristic feature of the oxidant problem in the Los Angeles Basin (Tiao et al., 1975), as well as in San Francisco, New York, Houston, Phoenix, and St. Louis (e.g., Altshuller, 1975; Coffey and Stasiuk, 1975; Shreffler and Evans, 1982;

Wolff et al., 1977a). Simple advection of a photochemically reactive air mass, local wind patterns, and diurnal wind cycles appear to be the main factors involved in urban-scale transport.

Mesoscale transport is in many respects an extension of urban-scale transport and is characterized by the development of urban plumes. Bell documented cases in 1959 in which precursors from the Los Angeles Basin and the resultant oxidant plume were transported over the coastal Pacific Ocean, producing elevated oxidant concentrations in San Diego County the next day (Bell, 1960). Similar scales of transport have been reported by Cleveland et al. (1976a,b) for the New York-Connecticut area; by Wolff and coworkers and others (Wolff et al., 1977a,b; Wolff and Lioy, 1978; Clark and Clarke, 1982; Clarke et al., 1982; Vaughan et al., 1982) for the Washington, DC-Boston corridor; and by Westberg and coworkers for the Chicago-Great Lakes area (Sexton and Westberg, 1980; Westberg et al., 1981). These and other studies have demonstrated that ozone-oxidant plumes from major urban areas can extend downwind about 100 to 200 miles and can have widths of tens of miles (Sexton, 1982), frequently up to half the length of the plume.

Synoptic-scale transport is characterized by the general and widespread occurrence of elevated oxidants and precursors on a regional or air-mass scale as the result of certain favorable weather conditions, notably, slow-moving, well-developed high-pressure, or anti-cyclonic, systems characterized by weak winds and limited vertical mixing (Korshover, 1967; 1975). The size of the region that can be affected has been described by Wolff and coworkers, who reported the occurrence of haze and elevated ozone levels in an area extending from the Midwest to the Gulf Coast (Wolff et al., 1982) and the occurrence of elevated ozone concentrations extending in a virtual "ozone river" from the Gulf Coast to New England that affected anywhere from a few hundred square miles to a thousand square miles during a 1-week period in July 1977 (Wolff and Lioy, 1980).

1.2.4.5 Stratospheric-Tropospheric Ozone Exchange. The fact that ozone is formed in the stratosphere, mixed downward, and incorporated into the troposphere, where it forms a more or less uniformly mixed background concentration, has been known in various degrees of detail for many years (Junge, 1963). It is widely accepted that the long-term average tropospheric background concentration of about 30 ppb to 50 ppb results primarily, though not

exclusively, from the transfer of stratospheric ozone into the upper troposphere, followed by subsequent dispersion throughout the troposphere (e.g., Kelly et al., 1982).

The exchange of ozone between the stratosphere and the troposphere in the middle latitudes occurs to a major extent in events called "tropopause folds" (TF) (Reiter, 1963; Reiter and Mahlman, 1965; Danielsen, 1968; Reiter, 1975; Danielsen and Mohnen, 1977; Danielsen, 1980), in which the polar jet stream plays a major role. From recent studies, Johnson and Viezee (1981) proposed four types or mechanisms of TF injection and concluded that two of these, both of which are consistent with theory, could cause substantial effects in terms of high ozone concentrations at ground level. They concluded, in addition, that all low-pressure trough systems may induce TF events and cause the trans-tropopause movement of ozone-rich air into the troposphere (Johnson and Viezee, 1981).

1.2.4.6 Stratospheric Ozone at Ground Level. From a detailed review of studies on background tropospheric ozone, Viezee and Singh (1982) concluded that the stratosphere is a major but not the sole source of background ozone in the unpolluted troposphere, a conclusion reached by other investigators as well (e.g., Kelly et al., 1982). The stratospheric ozone reservoir shows a strong seasonal cycle that is reflected at ground-level. At some stations that monitor background ozone levels, average spring background levels may be as high as 80 ppb, with average fall levels ranging from 20 to 40 ppb (e.g., Singh et al., 1977; Mohnen, 1977; U.S. Environmental Protection Agency, 1978). Viezee and Singh (1982) and Viezee et al. (1983) concluded that relatively high ozone concentrations can occur for short periods of time (minutes to a few hours) over local areas as a result of stratospheric intrusions.

A number of investigators have attempted to quantify the amount of the surface ozone that can be attributed to stratospheric sources. The method most commonly used is based on the assumption that beryllium-7 (^7Be) is a unique tracer for air parcels of stratospheric origin. Calculated correlations between surface ozone and ^7Be show, however, that their relationship is highly variable (e.g., Kelly et al., 1982; Ferman and Monson, 1978; Johnson and Viezee, 1981; Husain et al., 1977). Singh et al. (1980) and Viezee and Singh (1982) have pointed out problems with using this technique to quantify the contribution of stratospheric ozone to surface ozone. Singh et al. (1980)

concluded that "the experimental technique involving a $^7\text{Be}/\text{O}_3$ ratio to estimate the daily stratospheric component of ground-level O_3 is unverified and considered to be inadequate for air quality applications" (p. 1009). This group of investigators have suggested, however, that ^7Be may be used, under appropriate meteorological conditions, as a qualitative tracer for air masses of stratospheric origin (Johnson and Viezee, 1981; Viezee et al., 1979).

Other methods used to attempt to quantify the stratospheric component of surface ozone include aircraft observations of TF events coupled with calculations of downward ozone flux, and examination of surface ozone data records. From such data, Viezee et al. (1983) concluded that direct ground-level contributions from stratospheric ozone are infrequent (<1 percent of the time), short-lived, and associated with ozone concentrations ≤ 0.1 ppm.

Notwithstanding difficulties with quantifying its contribution to surface ozone, however, stratospheric ozone is clearly present in atmospheric surface layers, and the meteorological mechanisms responsible have been described by a number of investigators (e.g., Danielsen, 1968; Wolff et al., 1979; Johnson and Viezee, 1981).

1.2.4.7 Background Ozone from Photochemical Reactions. Whereas stratospheric ozone is thought by many investigators to be the dominant contributor to background levels of ozone, as discussed above, other investigators have concluded that as much as two-thirds of the annual average background concentrations may result from photochemical reactions. For example, Altshuller (1986), in a recent review article, has concluded that photochemically generated ozone should equal or exceed the stratospheric contribution at lower-elevation remote locations; and that photochemically generated ozone from manmade emissions probably constitutes most of the ozone measured at more polluted rural locations during the warmest months of the year. His conclusions were based, in part, on an analysis of global circulation (e.g., Levy et al., 1985) and photochemical modeling studies (e.g., Fishman and Seiler, 1983; Fishman and Carney, 1984; Fishman et al., 1985; Dignon and Hameed, 1985). In these modeling studies, the photochemical contribution to background ozone levels was estimated to range from ~15 ppb (long-term) to ~80 ppb (summertime), depending on the level of NO_x emissions assumed.

Studies on the role of NO_x in nonurban ozone photochemistry have shown that ozone formation at many of the locations is not NO_x -limited, but depends on VOC reactions, as well (e.g., Martinez and Singh, 1979; Kelly et al., 1984;

Liu et al., 1984). Background NO_x concentrations at most remote, clean locations range from $\lesssim 0.05$ ppb upward. Mean concentrations of NO_x at nonurban locations in the United States east of the Rocky Mountains range from ~ 1 ppb to 10 ppb (Altshuller, 1986; see also Section 1.2.5.2.4 and Chapter 3). These background concentrations of NO_x are higher than previously thought (see, e.g., Singh et al., 1980; Kelly et al., 1984, regarding global models and assumed reservoirs of NO_x).

The contributions of biogenic VOC to background ozone, although a matter of controversy in recent years, appear not to be significant under most atmospheric conditions, since ambient air concentrations of biogenic VOC are quite low, even at rural sites (Altshuller, 1983).

Thus, photochemistry and stratospheric intrusions are both regarded as contributing to background ozone concentrations, but the apportionment of background to respective sources remains a matter of investigation.

1.2.5 Sources, Emissions, and Concentrations of Precursors to Ozone and Other Photochemical Oxidants

As noted earlier, photochemical production of ozone depends both on the presence of precursors, volatile organic compounds (VOCs) and nitrogen oxides (NO_x), emitted by manmade and by natural sources; and on suitable conditions of sunlight, temperature, and other meteorological factors. Because of the intervening requirement for meteorological conditions conducive to the photochemical generation of ozone, emission inventories are not as direct predictors of ambient concentrations of secondary pollutants such as ozone and other oxidants as they are for primary pollutants.

1.2.5.1 Sources and Emissions of Precursors. Emissions of manmade VOCs (excluding several relatively unreactive compounds such as methane) in the United States have been estimated at 19.9 Tg/yr for 1983 (U.S. Environmental Protection Agency, 1984). Retrospective estimates show that manmade VOC emissions rose from about 18.5 Tg/yr in 1940 to about 27.1 Tg/yr in 1970 (U.S. Environmental Protection Agency, 1986). An examination of trends in manmade VOC emissions for 1970 through 1983 shows that the annual emission rate for manmade VOCs decreased some 26 percent during this period. The main sources nationwide are industrial processes, which emit a wide variety of VOCs, such as chemical solvents; and transportation, which includes the emission of VOCs in gasoline vapor as well as in gasoline combustion products. Estimates of

biogenic emissions of organic compounds in the United States are highly inferential but data suggest that the yearly rate is the same order of magnitude as manmade emissions. Most of the biogenic emissions actually occur during the growing season, however, and the kinds of compounds emitted are different from those arising from manmade sources.

Emissions of manmade NO_x in the United States were estimated at 19.4 Tg/yr for 1983. Retrospective estimates show that manmade NO_x emissions rose from about 6.8 Tg/yr in 1940 to about 18.1 Tg/yr in 1970 (U.S. Environmental Protection Agency, 1986). Annual emissions of manmade NO_x were some 12 percent higher in 1983 than in 1970, but the rate leveled off in the late 1970s and exhibited a small decline from about 1980 through 1982 (U.S. Environmental Protection Agency, 1984). The increase over the period 1970 through 1983 had two main causes: (1) increased fuel combustion in stationary sources such as power plants; and (2) increased fuel combustion in highway motor vehicles, as the result of the increase in vehicle miles driven. Total vehicle miles driven increased by 42 percent over the 14 years in question.

Estimated biogenic NO_x emissions are based on uncertain extrapolations from very limited studies, but appear to be about an order of magnitude less than manmade NO_x emissions.

1.2.5.2 Representative Concentrations in Ambient Air.

1.2.5.2.1 Hydrocarbons in urban areas. Most of the available ambient air data on the concentrations of nonmethane hydrocarbons (NMHC) in urban areas have been obtained during the 6:00 to 9:00 a.m. period. Since hydrocarbon emissions are at their peak during that period of the day, and since atmospheric dispersion is limited that early in the morning, NMHC concentrations measured then generally reflect maximum diurnal levels. Representative data for urban areas show mean NMHC concentrations between 0.4 and 0.9 ppm.

The hydrocarbon composition of urban atmospheres is dominated by species in the C_2 to C_{10} molecular-weight range. The paraffinic hydrocarbons (alkanes) are most prominent, followed by aromatics and alkenes. Based on speciation data obtained in a number of urban areas, alkanes generally constitute 50 to 60 percent of the hydrocarbon burden in ambient air, aromatics 20 to 30 percent, with alkenes and acetylene making up the remaining 5 to 15 percent (Sexton and Westberg, 1984).

1.2.5.2.2 Hydrocarbons in nonurban areas. Rural nonmethane hydrocarbon concentrations are usually one to two orders of magnitude lower than those

measured in urban areas (Ferman, 1981; Sexton and Westberg, 1984). In samples from sites carefully selected to guarantee their rural character, total NMHC concentrations ranged from 0.006 to 0.150 ppm C (e.g., Cronn, 1982; Seila, 1981; Holdren et al., 1979). Concentrations of individual species seldom exceeded 0.010 ppm C. The bulk of species present in rural areas are alkanes; ethane, propane, n-butane, iso-pentane, and n-pentane are most abundant. Ethylene and propene are sometimes present at ≤ 0.001 ppm C, and toluene is usually present at ~ 0.001 ppm C. Monoterpene concentrations are usually ≤ 0.020 ppm C. During the summer months, isoprene concentrations as high as 0.150 ppm C have been measured (Ferman, 1981). The maximum concentrations of isoprene usually encountered, however, are in the range of 0.030 to 0.040 ppm C.

1.2.5.2.3 Nitrogen oxides in urban areas. Concentrations of NO_x , like hydrocarbon concentrations, tend to peak in urban areas during the early morning, when atmospheric dispersion is limited and automobile traffic is dense. Most NO_x is emitted as nitric oxide (NO), but the NO is rapidly converted to NO_2 , initially by thermal oxidation and subsequently by ozone and peroxy radicals produced in atmospheric photochemical reactions. The relative concentrations of NO versus NO_2 fluctuate day-to-day, depending on diurnal and day-to-day fluctuations in ozone levels and photochemical activity.

Urban NO_x concentrations during the 6:00 to 9:00 a.m. period in 10 cities ranged from 0.05 to 0.15 ppm in studies done in the last 5 to 7 years (e.g., Westberg and Lamb, 1983; Richter, 1983; Eaton et al., 1979), although concentrations two to three times higher occur in cities such as Los Angeles. Concurrent NMHC measurements for these 10 cities showed that NMHC/ NO_x ratios ranged from 5 to 16.

1.2.5.2.4 Nitrogen oxides in nonurban areas. Concentrations of NO_x in clean remote environments are usually < 0.5 ppb (Logan, 1983). In exceptionally clean air, NO_x concentrations as low as 0.015 ppb have been recorded (Bollinger et al., 1982). Concentrations of NO_x at nonurban sites in the northeastern United States appear to be higher than NO_x concentrations in the west by a factor of ten (Mueller and Hidy, 1983). From the limited amount of data available, NO_x concentrations in unpopulated nonurban areas in the west average ≤ 1 ppb; but in nonurban northeastern areas average NO_x can exceed 10 ppb.

1.2.6 Source-Receptor (Oxidant-Precursor) Models

In order to apply knowledge of the atmospheric chemistry of precursors, and of ozone and other photochemical oxidants, during their dispersion and transport, models describing these phenomena have been developed in a variety of forms over the past 15 years. Most of these models relate the rates of precursor emissions from mobile and stationary sources, or precursor atmospheric concentrations, to the resulting ambient concentrations of secondary pollutants that impact receptors at downwind sites. For this reason they have been described as source-receptor models.

Current air quality, or source-receptor, models can be classified as either statistical or computational-dynamic. Statistical models are generally based on a statistical analysis of historical air quality data, and are not explicitly concerned with atmospheric chemistry or meteorology. An example of statistical models is the linear rollback concept.

Computational, or dynamic, models attempt to describe mathematically the atmospheric chemical and physical processes influencing air pollution formation and impacts. Examples of computational models include trajectory and fixed-grid airshed models. Two phenomenologically different approaches have been employed in dynamic models with respect to the coordinate systems chosen. A coordinate system fixed with respect to the earth is termed Eulerian, while in Lagrangian models the reference frame moves with the air parcel whose behavior is being simulated.

1.2.6.1 Trajectory Models. In trajectory models, a moving-coordinate system describes pollutant transport as influenced by local meteorological conditions. Trajectory models provide dynamic descriptions of atmospheric source-receptor relationships that are simpler and less expensive to derive than those obtained from fixed-cell models.

The simplest form of trajectory model is the empirical kinetic modeling approach (EKMA). This approach was developed from earlier efforts (Dimitriades, 1972) to use smog chamber data to develop graphical relationships between morning NMOC and NO_x levels and afternoon maximum concentrations of ozone. In applying EKMA, the Ozone Isopleth Plotting Package (OZIP) (Whitten and Hogo, 1978) is used to generate ozone isopleths at various levels of sophistication corresponding to "standard" EKMA, "city-specific" EKMA, or the simplified trajectory model (F.R., 1979). The EKMA isopleths generated are used to determine the relative degree of control of precursor emissions needed to achieve a given percentage reduction in ozone.

The use of EKMA in ozone abatement programs is relatively widespread. It is therefore worth noting the general control implications of EKMA isopleths. For areas with high levels of morning precursor emissions and meteorology conducive to oxidant formation, such as Los Angeles, for example, EKMA isopleths predict that (1) at high NMOC/NO_x concentration ratios, reductions in NO_x will decrease ozone formation; (2) at moderate NMOC/NO_x ratios, reductions in NMOC and NO_x will decrease ozone formation; and (3) at very low NMOC/NO_x ratios, increases in NO_x will inhibit ozone formation. These predictions cannot be assumed to apply to all urban areas, or even to all high-oxidant urban areas, since the shape of the EKMA isopleths is a function of numerous factors, many of which are location-specific. For discussions of the specific assumptions employed in EKMA and the underlying chemistry and meteorology, the primary literature should be consulted (e.g., Dimitriades, 1970, 1972, 1977a,b; Dodge, 1977a,b; Whitten and Hogo, 1977; U.S. Environmental Protection Agency, 1977, 1978; Whitten, 1983). Likewise, the primary literature should be consulted for additional data and discussions on the respective effects on ozone formation of controlling NMHC and NO_x (e.g., Liu and Grisinger, 1981; Chock et al., 1981; Kelly, 1985; Kelly et al., 1986; Glasson and Tuesday, 1970; Dimitriades, 1970, 1972, 1977a,b).

1.2.6.2 Fixed-Grid Models. Fixed-grid models, also called regional airshed models, are based on two- or three-dimensional arrays of grid cells and are the most sophisticated source-receptor models presently available. Such models are computationally complex and require the most extensive set of input data; but they also provide the most realistic treatment of the various processes involved in photochemical air pollution formation.

1.2.6.3 Box Models. Box models (Hanna, 1973; Demerjian and Schere, 1979; Derwent and Hov, 1980) are the simplest of dynamic models. They treat the atmosphere as a single cell, bounded by the mixing layer, having an area on the order of 100 square miles.

1.2.6.4 Validation and Sensitivity Analyses for Dynamic Models. All presently available source-receptor models require a degree of simplifying assumptions to deal with practical limitations imposed by existing computer capabilities, time and cost constraints, or lack of knowledge concerning inputs such as boundary conditions, emissions, or detailed reaction mechanisms. The reliability and applicability of any particular model therefore depends upon its specific limitations, data requirements, and degree of validation against

experimental data from ambient air measurements or environmental chamber runs. Reliability and applicability also depend on the quality of the chemical kinetics mechanisms used to define the O_3 -HC- NO_x relationship.

Attempts are made to validate model predictions by comparing them with real observations; and operating parameters are often varied to determine the sensitivity of the model to respective parameter changes (Gelinas and Vajk, 1979). In addition, the extent of agreement between the results from two simulations can be tested. In this way, completely different models may be compared, or an internal component, such as the chemical kinetics mechanisms, may be substituted and the model run again to ascertain the effect of such substitutions.

1.3 SAMPLING AND MEASUREMENT OF OZONE AND OTHER PHOTOCHEMICAL OXIDANTS AND THEIR PRECURSORS

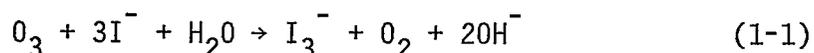
1.3.1 Sampling and Measurement of Ozone and Other Photochemical Oxidants

The analysis of ozone and other, related atmospheric oxidants includes requirements for representative sampling, specific and sensitive measurement methodologies, methods for the generation of standard samples, absolute methods for the calibration of these standards, and procedures by which to provide quality assurance for the whole measurement process. In this summary, recommended procedures are given for all of these analytical steps. Because of the large existing data base that employed measurements for "total oxidants," non-specific iodometric techniques are discussed and compared to current specific O_3 measurements.

1.3.1.1 Quality Assurance and Sampling. A quality assurance program is employed by the U.S. Environmental Protection Agency for assessing the accuracy and precision of monitoring data and for maintaining and improving the quality of ambient air data. Procedures and operational details have been prescribed in each of the following areas: selection of analytical methods and instrumentation (i.e., reference and equivalent methods); method specifications for gaseous standards; methods for primary and secondary (transfer standards) calibration; instrumental zero and span check requirements, including frequency of checks, multiple-point calibration procedures, and preventive and remedial maintenance requirements; procedures for air pollution episode monitoring; methods for recording and validating data; and information on documenting

quality control (U.S. Environmental Protection Agency, 1977b). Design criteria for O₃ monitoring stations, to help ensure the quality of aerometric data, have been established (U.S. Environmental Protection Agency, 1977a; National Research Council, 1977).

1.3.1.2 Measurement Methods for Total Oxidants and Ozone. Techniques for the continuous monitoring of total oxidants and O₃ in ambient air are summarized in Table 1-1. The earliest methods used for routinely monitoring oxidants in the atmosphere were based on iodometry. When atmospheric oxidants are absorbed in potassium iodide (KI) reagent, the iodine produced,



is measured by ultraviolet absorption in colorimetric instruments and by amperometric means in electrochemical instruments. The term "total oxidants" is of historical significance only and should not be construed to mean that such measurements yield a sum of the concentrations of the oxidants in the atmosphere. The various oxidants in the atmosphere react to yield iodine at different rates and with different stoichiometries. Only ozone reacts immediately to give a quantitative yield of iodine. As discussed below, the total oxidants measurement correlates fairly well with the specific measurement of ozone, except during periods when significant nitrogen dioxide (NO₂) and sulfur dioxide (SO₂) interferences are present. The major problem with the total oxidants measurement was the effect of these interferences. Total oxidants instruments have now been replaced by specific ozone monitors in all aerometric networks and in most research laboratories. Biases among and between total oxidants and ozone methods are still important, however, for evaluating existing data on health and welfare effects levels where concentrations were measured by total oxidants methods.

The reference method promulgated by EPA for compliance monitoring for ozone is the chemiluminescence technique based on the gas-phase ozone-ethylene reaction (F.R., 1971). The technique is specific for ozone, the response is a linear function of concentration, detection limits of 0.001 to 0.005 ppm are readily obtained, and response times are 30 seconds or less. Prescribed methods of testing and prescribed performance specifications that a commercial analyzer must meet in order to be designated as a reference method or an equivalent method have been published by EPA (F.R., 1975). An analyzer may be

TABLE 1-1. SUMMARY OF OZONE MONITORING TECHNIQUES

Principle	Reagent	Response	Minimum detection limit	Response time, 90% FS ^a	Major interferences	References
Continuous colorimetric	10(20)% KI buffered at pH = 6.8	Total oxidants	0.010 ppm	3 to 5 minutes	NO ₂ (+20%, 10%KI) SO ₂ (-100%)	Littman and Benolite (1953) Tokiwa et al. (1972)
Continuous electrochemical	2% KI buffered at pH = 6.8	Total oxidants	0.010 ppm	1 minute	NO ₂ (+6%) SO ₂ (-100%)	Brewer and Milford (1960) Mast and Saunders (1962) Tokiwa et al. (1972)
Chemiluminescence	Ethylene, gas-phase	O ₃ -specific	0.005 ppm	< 30 seconds	None ^b	Nederbragt et al. (1965) Stevens and Hodgeson (1973)
Chemiluminescence	Rhodamine-B	O ₃ -specific	0.001 ppm	< 1 minute	None	Regener (1960, 1964) Hodgeson et al. (1970)
Ultraviolet photometry	None	O ₃ -specific	0.005 ppm	30 seconds	Species that absorb at 254 nm ^c	Bowman and Horak (1972)

^aFS = full response.

^bA signal enhancement of 3 to 12% has been reported for measurement of O₃ in humid versus dry air (California Air Resources Board, 1976).

^cNo significant interferences have been reported in routine ambient air monitoring. If abnormally high concentrations of species that absorb at 254 nm (e.g., aromatic hydrocarbons and mercury vapor) are present, some positive response may be expected. If high aerosol concentrations are sampled, inlet filters must be used to avoid a positive response.

designated as a reference method if it is based on the same principle as the reference method and meets performance specifications. An acceptable equivalent method must meet the prescribed performance specifications and also show a consistent relation with the reference method.

The designated equivalent methods are based on either the gas-solid chemiluminescence procedure or the ultraviolet absorption procedure (Table 1-1). The first designated equivalent method was based on ultraviolet absorption by ozone of the mercury 254 nm emission line. The measurement is in principle an absolute one, in that the ozone concentration can be computed from the measured transmission signal since the absorption coefficient and pathlength are accurately known. In the gas-solid chemiluminescence analyzer, the reaction between ozone and Rhodamine-B adsorbed on activated silica produces chemiluminescence, the intensity of which is directly proportional to ozone concentration.

1.3.1.3 Calibration Methods. All the analyzers discussed above must be calibrated periodically with ozonized air streams, in which the ozone concentration has been determined by some absolute technique. This includes the ultraviolet (UV) absorption analyzer, which, when used for continuous ambient monitoring, may experience ozone losses in the inlet system because of contamination.

A primary ozone calibration system consists of a clean air source, ozone generator, sampling manifold, and means for measuring absolute ozone concentration. The ozone generator most often used is a photolytic source employing a mercury lamp that irradiates a quartz tube through which clean air flows at a controlled rate (Hodgeson et al., 1972). Once the output of the generator has been calibrated by a primary reference method, it may be used to calibrate O₃ transfer standards, which are portable generators, instruments, or other devices used to calibrate analyzers in the field. Reference calibration procedures that have been used for total oxidants and ozone-specific analyzers in the United States are summarized in Table 1-2.

The original reference calibration procedure promulgated by EPA was the 1 percent neutral buffered potassium iodide (NBKI) method (F.R., 1971). This technique was employed in most of the United States, with the exception of California. The California Air Resources Board (CARB) (1976) and the Los Angeles Air Pollution Control District (LAAPCD) employed different versions of iodometric techniques. Procedural details of the calibration methods are

TABLE 1-2. OZONE CALIBRATION TECHNIQUES

Method	Reagent	Primary standard ^a	Organization and dates	Purpose	Bias, $[O_3]_1/[O_3]_{UV}$
1% NBKI	1% KI, phosphate buffer pH = 6.8	Reagent grade arsenious oxide	EPA 1971-1976	Primary reference procedure	1.12 ± 0.05^b
2% NBKI ^c	2% KI phosphate buffer pH = 6.8	Reagent grade potassium biiodate	CARB until 1975	Primary reference procedure	1.20 ± 0.05^b
1% Unbuffered KI	1% KI pH = 7		LAAPCD until 1975	Primary reference procedure	0.96^d
UV photometry	None	O_3 absorptivity at Hg 254 nm emission line	All 1979-present	Primary reference procedure ^e	
Gas-phase titration (GPT)	Nitric oxide standard reference gas	Nitric oxide SRM (50 ppm in N_2) from NBS	EPA, States 1973-present	Alternative reference procedure (1973-1979) Transfer standard (1979-present)	1.030 ± 0.015^f
1% BAKI	1% KI, boric acid buffer pH = 5	Standard KIO_3 solutions	EPA 1975-1979	Alternative reference procedure	1.00 ± 0.05

^aIn the case of the iodometric methods, the primary standard is the reagent used to prepare or standardize iodine solutions.

^bThe uncertainty limits represent the range of values obtained in several independent studies.

^cPre-humidified air used for the ozone source.

^dOnly one study available (DeMore et al., 1976).

^eUV photometry used as reference method by CARB since 1975. This technique used as an interim, alternative reference procedure by EPA from 1976 to 1979.

^fThis is the value reported in the latest definitive study (Fried and Hodgeson, 1982). Previous studies reported biases ranging from 0 to 10 percent (Burton et al., 1976; Paur and McElroy, 1979).

^gThis procedure also recommended a standard I_3^- solution absorptivity to be used instead of the preparation of standard iodine solutions.

summarized in Table 1-2. A number of studies conducted between 1974 and 1978 revealed several deficiencies with KI methods, the most notable of which were poor precision or interlaboratory comparability and a positive bias of NBKI measurements relative to simultaneous absolute UV absorption measurements. The positive bias observed is peculiar to the use of phosphate buffer in the NBKI techniques. The bias was not observed in the unbuffered LAAPCD method (which nevertheless suffered from poor precision), nor in the 1 percent EPA KI method without phosphate buffer (Hodgeson et al., 1977), nor in a KI procedure that used boric acid as buffer (Flamm, 1977). A summary of results of these prior studies was presented in the previous criteria document (U.S. Environmental Protection Agency, 1978) and in a review by Burton et al. (1976). Correction factors for converting NBKI calibration data to a UV photometry basis are given in Table 1-2 and discussed in Chapter 4 (Section 4.2.4.2.1).

Subsequently, EPA evaluated four alternative reference calibration procedures and selected UV photometry on the basis of superior accuracy and precision and simplicity of use (Rehme et al., 1981). In 1979 regulations were amended to specify UV photometry as the reference calibration procedure (F.R., 1979). Laboratory photometers used as reference systems for absolute O_3 measurements have been described by DeMore and Patapoff (1976) and Bass et al. (1977).

These laboratory photometers contain long path cells (1 to 5 m) and employ sophisticated digital techniques for making effective double beam measurements of small absorbancies at low ozone concentrations. A primary standard UV photometer is one that meets the requirements and specifications given in the revised ozone calibration procedures (F.R., 1979e). Since these are currently available in only a few laboratories, EPA has allowed the use of transfer standards, which are devices or methods that can be calibrated against a primary standard and transferred to another location for calibration of O_3 analyzers. Examples of transfer standards that have been used are commercial O_3 photometers, calibrated generators, and gas-phase titration (GPT) apparatus. Guidelines on transfer standards have been published by EPA (McElroy, 1979).

1.3.1.4. Relationships of Total Oxidants and Ozone Measurements. The temporal and quantitative relationship between simultaneous total oxidants and ozone measurements has been examined in this chapter because of the existence of a data base obtained by total oxidants measurements. Such a comparison is complicated by the relative scarcity of simultaneous data, the presence of both positive (NO_2) and negative (SO_2) interferences in total oxidants measurements

of ambient air, and the change in the basis of calibration. In particular, the presence of NO₂ and SO₂ interferences prevent the establishment of a definite quantitative relationship between ozone and oxidants data. Nevertheless, some interesting conclusions can be drawn and are summarized below.

Studies concluded in the early to mid-1970s were reviewed in the previous criteria document (U.S. Environmental Protection Agency, 1978). Averaged data showed fairly good qualitative and quantitative agreement between diurnal variations of total oxidants and ozone. For example, uncorrected monthly averaged data from Los Angeles and St. Louis showed distinct morning and evening peaks resulting from NO₂ interference (Stevens et al., 1972a,b). The most recent comparison in the literature involved simultaneous ozone and total oxidant measurements in the Los Angeles basin by the California Air Resources Board (1978) in 1974, 1976, and 1978. The maximum hourly data pairs were correlated (Chock et al., 1982) and yielded the following regression equation for 1978, in which a large number (927) of data pairs were available:

$$\begin{aligned} \text{Oxidant (ppm)} &= 0.870 \text{ O}_3 + 0.005 \\ (\text{Correlation coefficient} &= 0.92) \end{aligned} \quad (1-2)$$

The oxidant data were uncorrected for NO₂ and SO₂ interferences, and on individual days maximum oxidant averages were both higher than and lower than ozone averages.

In summary, specific ozone measurements agree fairly well with total oxidants corrected for NO₂ and SO₂ interferences, and in such corrected total oxidants measurements ozone is the dominant contributor. Indeed, it is difficult to discern the presence of other oxidants in corrected total oxidant data. Without corrections there can be major temporal discrepancies between ozone and oxidants data, which are primarily a result of oxidizing and reducing interferences with KI measurements. As a result of these interferences, on any given day the total oxidant values may be higher than or lower than simultaneous ozone data. The measurement of ozone is a more reliable indicator than total oxidant measurements of oxidant air quality.

1.3.1.5 Methods for Sampling and Analysis of Peroxyacetyl Nitrate and Its Homologues. Only two analytical techniques have been used to obtain significant data on ambient peroxyacetyl nitrate (PAN) concentrations. These are gas chromatography with electron capture detection (GC-ECD) and long-path Fourier

transform infrared (FTIR) spectrometry. Atmospheric data on PAN concentrations have been obtained predominantly by GC-ECD because of its relative simplicity and superior sensitivity. These techniques have been described in this chapter along with attendant methods of sampling, PAN generation, absolute analysis, and calibration.

By far the most widely used technique for the quantitative determination of ppb concentrations of PAN and its homologues is GC-ECD (Darley et al., 1963; Stephens, 1969). With carbowax or SE30 as a stationary phase, PAN, peroxypropionyl nitrate (PPN), peroxybenzoyl nitrate (PBzN), and other homologues (e.g., peroxybutyryl nitrate) are readily separated from components such as air, water, and other atmospheric compounds, as well as ethyl nitrate, methyl nitrate, and other contaminants that are present in synthetic mixtures. Electron-capture detection provides sensitivities in the ppb and sub-ppb ranges. Typically, manual air samples are collected in 50- to 200-ml ungreased glass syringes and purged through the gas-sampling valve. Continuous analyses are performed by pumping ambient air through a gas sampling loop of an automatic valve, which periodically injects the sample onto the column. Samples collected from the atmosphere should be analyzed as soon as possible because PAN and its homologues undergo thermal decomposition in the gas phase and at the surface of containers. The recent work of Singh and Salas (1983a,b) on the measurement of PAN in the free (unpolluted) troposphere (see Chapter 5) is illustrative of current capabilities for measuring low concentrations. A minimum detection limit of 0.010 ppb was obtained.

The literature contains conflicting reports on the effects of variable relative humidity on PAN measurements by GC-ECD. If a moisture effect is suspected in a PAN analysis, the bulk of this evidence suggests that humidification of PAN calibration samples (to a range approximating the humidity of the samples being analyzed) would be advisable.

Conventional long-path infrared spectroscopy and Fourier-transform infrared spectroscopy (FTIR) have been used to detect and measure atmospheric PAN. Sensitivity is enhanced by the use of FTIR. The most frequently used IR bands have been assigned and the absorptivities reported in the literature (Stephens, 1964; Bruckmann and Willner, 1983; Holdren and Spicer, 1984) permit the quantitative analysis of PAN without calibration standards. The absorptivity of the 990 cm^{-1} band of PBzN, a higher homologue of PAN, has been reported by Stephens (1969). Tuazon et al. (1978) describes an FTIR system operable at

pathlengths up to 2 km for ambient measurements of PAN and other trace constituents. This system employed an eight-mirror multiple reflection cell with a 22.5-m base path. Tuazon et al. (1981b) reported maximum PAN concentrations ranging from 6 to 37 ppb over a 5-day smog episode in Claremont, CA. Hanst et al. (1982) made measurements with a 1260-m folded optical path system during a 2-day smog episode in Los Angeles in 1980. An upper limit of 1 ppb of PBzN was placed, and the maximum PAN concentration observed was 15 ppb. The large internal surface area of the White cells may serve to promote the decomposition or irreversible adsorption of reactive trace species such as PAN. High volume sampling rates and inert internal surface materials are used to minimize these effects.

Because of the thermal instability of dilute PAN samples and the explosive nature of liquefied PAN, calibration samples are not commercially available and must be prepared. Earlier methods used to synthesize PAN have been summarized by Stephens (1969). The photolysis of alkyl nitrites in oxygen was the most commonly used procedure and may still be used by some investigators. As described by Stephens et al. (1965), the liquefied crude mixture obtained at the outlet of the photolysis chamber is purified by preparative-scale GC. [CAUTION: Both the liquid crude mixture and the purified PAN samples are violently explosive and should be handled behind explosion shields using plastic full-face protection, gloves, and a leather coat at all times.] The pure PAN is usually diluted to about 1000 ppm in cylinders pressurized with nitrogen to 100 psig and stored at reduced temperatures, <15°C.

Gay et al. (1976) have used the photolysis of chlorine: aldehyde: nitrogen dioxide mixtures in air or oxygen for the preparation of PAN and a number of its homologues at high yields. This procedure has been utilized in a portable PAN generator that can be used for the calibration of GC-ECD instruments in the field (Grosjean, 1983; Grosjean et al., 1984).

Several investigators have recently reported on a condensed-phase synthesis of PAN by nitration of peracetic acid in a hydrocarbon solvent. High yields are produced of a pure product free of other alkyl nitrates (Hendry and Kenley, 1977; Kravetz et al., 1980; Nielsen et al., 1982; Holdren and Spicer, 1984). After the nitration is complete, the hydrocarbon fraction containing PAN concentrations of 2 to 4 mg/ml can be stored at -20°C for periods longer than a year (Holdren and Spicer, 1984).

The most direct method for absolute analysis of these PAN samples is by infrared absorption, using the IR absorptivities mentioned earlier. Conventional IR instruments and 10-cm gas cells can analyze gas standards of concentrations >35 ppm. Liquid microcells can be used for the analysis of PAN in isooctane solutions. The alkaline hydrolysis of PAN to acetate ion and nitrite ion in quantitative yield (Nicksic et al., 1967) provides a means independent of infrared for the quantitative analysis of PAN. Following hydrolysis, nitrite ion may be quantitatively analyzed by the Saltzman colorimetric procedure (Stephens, 1969). The favored procedures now use ion chromatography to analyze for nitrite (Nielsen et al., 1982) or acetate (Grosjean, 1983; Grosjean et al., 1984) ions. Another calibration procedure has been proposed that is based on the thermal decomposition of PAN in the presence of excess and measured NO concentrations (Lonneman et al., 1982). The acetylperoxy radical, $\text{CH}_3\text{C}(\text{O})\text{O}_2$, and its decomposition products rapidly oxidize nitric oxide (NO) to NO_2 with a stoichiometry that has been experimentally determined.

1.3.1.6 Methods for Sampling and Analysis of Hydrogen Peroxide. Hydrogen peroxide (H_2O_2) is significant in photochemical smog as a chain terminator; as an index of the hydroperoxyl radical (HO_2) concentration (Bufalini and Brubaker, 1969; Demerjian et al., 1974); and as a reactant in the aqueous-phase oxidation of SO_2 to SO_4^{-2} and in the acidification of rain (Penkett et al., 1979; Dasgupta, 1980a,b; Martin and Damschen, 1981; Overton and Durham, 1982).

One of the major problems, however, in assessing the role of atmospheric H_2O_2 has been a lack of adequate measurement methodology. Earlier measurements (Gay and Bufalini, 1972a,b; Bufalini et al., 1972; Kok et al., 1978a,b) reporting H_2O_2 concentrations of 0.01 to 0.18 ppm are now believed to be far too high, and to be the result of artifact H_2O_2 formation from reactions of absorbed O_3 (Zika and Saltzman, 1982; Heikes et al., 1982; Heikes, 1984). Maximum tropospheric H_2O_2 concentrations predicted by modeling calculations (Chameides and Tan, 1981; Logan et al., 1981) and observed in recent field studies (Das et al., 1983) are on the order of 1 ppb.

Almost all of the methods used for the measurement of atmospheric H_2O_2 have used aqueous traps for sampling. Atmospheric O_3 , however, which is also absorbed at concentrations much higher than H_2O_2 , reacts in the bulk aqueous phase and at surfaces to produce H_2O_2 and thus is a serious interference (Zika and Saltzman, 1982; Heikes et al., 1982; Heikes, 1984). The removal of absorbed O_3 by purging immediately after sample collection may remove or substantially

reduce this interference (Zika and Saltzman, 1982; Das et al., 1983). Another problem identified with aqueous sampling is that other atmospheric species (in particular, SO_2) may interfere with the generation of H_2O_2 in aqueous traps and also react with collected H_2O_2 to reduce the apparent concentration measured (Heikes et al., 1982).

Of the techniques that have been used for the measurement of aqueous and gas-phase H_2O_2 , only the chemiluminescence and enzyme-catalyzed methods are summarized below. The other techniques are now believed to have inadequate sensitivity and specificity for the atmospheric concentrations actually present. In addition, the use of a tunable diode infrared laser source should eliminate the problem associated with nearby water bands, and this method is currently under investigation for atmospheric measurements (unpublished work in progress, Schiff, 1985).

Hydrogen peroxide in the atmosphere may be detected at low concentrations by the chemiluminescence obtained from copper(II)-catalyzed oxidation of luminol (5-amino-2,3-dihydro-1,4-phthalazinedione) by H_2O_2 (Armstrong and Humphreys, 1965; Kok et al., 1978a,b). This technique as initially employed suffered the interferences from O_3 and SO_2 discussed above for aqueous traps. Das et al. (1982) employed a static version of the method of Kok et al. (1978a) to measure H_2O_2 concentrations in the 0.01 to 1 ppb range. In addition, samples were purged with argon immediately after collection to eliminate, reportedly, the O_3 interference. Recently, a modified chemiluminescence method has been reported which used hemin, a blood component, as a catalyst for the luminol-based H_2O_2 oxidation (Yoshizumi et al., 1984).

The most promising chemical approach employs the catalytic activity of the enzyme horseradish peroxidase (HRP) on the oxidation of organic substrates by H_2O_2 . The production or decay of the fluorescence intensity of the substrate or reaction product is measured as it is oxidized by H_2O_2 , catalyzed by HRP. Some of the more widely used substrates have been scopoletin (6-methoxy-7-hydroxy-1,2-benzopyrone) (Andreae, 1955; Perschke and Broda, 1961); 3-(p-hydroxyphenyl)propionic acid (HPPA) (Zaitsev and Okhura, 1980); and leuco crystal violet (LCV) (Mottola et al., 1970).

The decrease in the fluorescence intensity of scopoletin is measured as a function of H_2O_2 concentration. Detection limits have been reported to be quite low (10^{-11} M). The chief disadvantage of this approach is that the concentration of H_2O_2 must be within a narrow range to obtain an accurately

measureable decrease in fluorescence. Oxidation of LCV produces intensely colored crystal violet, which has a molar absorption coefficient of $10^5 \text{ M}^{-1} \text{ cm}^{-1}$ at the analytical wavelength, 596 nm. The detection limit reported was 10^{-8} M in 5 cm cells. Two quite similar hydrogen donor substrates have been used. Zaitso and Okhura (1980) employed 3-(*p*-hydroxyphenyl) propionic acid and more recently the *p*-hydroxyphenyl acetic acid homologue is being used (Kunen et al., 1983; Dasgupta and Hwang, 1985). The measurement of the fluorescence intensity of the product dimer provides a quite sensitive means for the assay of H_2O_2 .

As with O_3 , H_2O_2 calibration standards are not commercially available and are usually prepared at the time of use. The most convenient method for preparing aqueous samples containing micromolar concentrations of H_2O_2 is simply the serial dilution of commercial grade 30 percent H_2O_2 (Fisher Analytical Reagent). Techniques for the convenient generation of gas-phase standards are not available. A technique often used for generating ppm concentrations of H_2O_2 in air involves the injection of microliter quantities of 30 percent H_2O_2 solution into a metered stream of air that flows into a Teflon bag. Aqueous and gas-phase samples are then standardized by conventional iodometric procedures (Allen et al., 1952; Cohen et al., 1967).

1.3.2 Measurement of Precursors to Ozone and Other Photochemical Oxidants

1.3.2.1 Nonmethane Organic Compounds. Numerous analytical methods have been employed to determine nonmethane organic compounds (NMOC) in ambient air. Measurement methods for the organic species may be grouped according to three major classifications: nonmethane hydrocarbons, aldehydes, and other oxygenated compounds.

Nonmethane hydrocarbons have been determined primarily by methods that employ a flame ionization detector (FID) as the sensing element. Early methods for the measurement of total nonmethane hydrocarbons did not provide for speciation of the complex mixture of organics in ambient air. These methods, still in use for analysis of total nonmethane organic compounds, are essentially organic carbon analyzers, since the response of the FID detector is essentially proportional to the number of carbon atoms present in the organic molecule (Sevcik, 1975). Carbon atoms bound, however, to oxygen, nitrogen, or halogens give reduced relative responses (Dietz, 1967). The FID detector has been utilized both as a stand-alone continuous detection system (non-speciation

analyzers have indicated an overall poor performance of the commercial instruments when calibration or ambient mixtures containing nonmethane organic compounds (NMOC) concentrations less than 1 ppm C were analyzed (e.g., Reckner, 1974; McElroy and Thompson, 1975; Sexton et al., 1982). The major problems associated with the non-speciation analyzers have been summarized in a recent technical assistance document published by the U.S. Environmental Protection Agency (1981). The document also presents ways to reduce some of the existing problems.

Because of the above deficiencies, other approaches to the measurement of nonmethane hydrocarbons are currently under development. The use of gas chromatography coupled to an FID system circumvents many of the problems associated with continuous non-speciation analyzers. This method, however, requires sample preconcentration because the organic components are present at part-per-billion (ppb) levels or lower in ambient air. The two main preconcentration techniques in present use are cryogenic collection and the use of solid adsorbents (McClenny et al., 1984; Jayanty et al., 1982; Westberg et al., 1980; Ogle et al., 1982). The preferred preconcentration method for obtaining speciated data is cryogenic collection. Speciation methods involving cryogenic preconcentration have also been compared with continuous nonspeciation analyzers (e.g., Richter, 1983). Results indicate poor correlation between methods at ambient concentrations below 1 part-per-million carbon (ppm C).

Aldehydes, which are both primary and secondary pollutants in ambient air, are detected by total NMOC and NMHC speciation methods but can not be quantitatively determined by those methods. Primary measurement techniques for aldehydes include the chromotropic acid (CA) method for formaldehyde (Altshuller and McPherson, 1963; Johnson et al., 1981), the 3-methyl-2-benzothiazolene (MBTH) technique for total aldehydes (e.g., Sawicki et al., 1961; Hauser and Cummins, 1964), Fourier-transform infrared (FTIR) spectroscopy (e.g., Hanst et al., 1982; Tuazon et al., 1978, 1980, 1981a), and high-performance liquid chromatography employing 2,4-dinitrophenyl-hydrazine derivatization (HPLC-DNPH) for aldehyde speciation (e.g., Lipari and Swarin, 1982; Kuntz et al., 1980). The CA and MBTH methods utilize wet chemical procedures and spectrophotometric detection. Interferences from other compounds have been reported for both techniques. The FTIR method offers good specificity and direct in situ analysis of ambient air. These advantages are offset, however, by the relatively high cost and lack of portability of the instrumentation.

On the other hand, the HPLC-DNPH method not only offers good specificity but can also be easily transported to field sites. A few intercomparison studies of the above methods have been conducted and considerable differences in measured concentrations were found. The data base is still quite limited at present, however, and further intercomparisons are needed.

Literature reports describing the vapor-phase organic composition of ambient air indicate that the major fraction of material consists of unsubstituted hydrocarbons and aldehydes. With the exception of formic acid, other oxygenated species are seldom reported. The lack of oxygenated hydrocarbon data is somewhat surprising since significant quantities of these species are emitted into the atmosphere by solvent-related industries and since at least some oxygenated species appear to be emitted by vegetation. In addition to direct emissions, it is also expected that photochemical reactions of hydrocarbons with oxides of nitrogen, O_3 , and hydroxyl radicals will produce significant quantities of oxygenated species. Difficulties during sample collection and analysis may account for the apparent lack of data. Attempts have been made to decrease adsorption by deactivating the reactive surface or by modifying the compound of interest (Osman et al., 1979; Westberg et al., 1980). Additional research efforts should focus on this area.

1.3.2.2 Nitrogen Oxides. Aside from the essentially unreactive nitrous oxide (N_2O), only two oxides of nitrogen occur in ambient air at appreciable concentrations: nitric oxide (NO) and nitrogen dioxide (NO_2). Both compounds, together designated as NO_x , participate in the cyclic reactions in the atmosphere that lead to the formation of ozone. Other minor reactive oxides of nitrogen in ambient air include peroxyacyl nitrates, nitrogen trioxide, dinitrogen pentoxide, and peroxyntic acid.

The preferred means (Federal Reference Method) of measuring NO and NO_2 is the chemiluminescence method (F.R., 1976). The measurement principle is the gas-phase chemiluminescent reaction of O_3 and NO (Fontijn et al., 1970). While NO is determined directly in this fashion, NO_2 is detected indirectly by first reducing or thermally decomposing the gas quantitatively to NO with a converter. The reaction of NO and O_3 forms excited NO_2 molecules that release light energy that is proportional to the NO concentration. Although the NO chemiluminescence is interference-free, other nitrogen compounds do interfere when directed through the NO_2 converter. The magnitude of these interferences is dependent upon the type of converter used (Winer et al., 1974; Joshi and

Bufalini, 1978). The detection limit of commercial chemiluminescence instruments for NO_2 measurement is $2.5 \mu\text{g}/\text{m}^3$ (0.002 ppm) (Katz, 1976).

Development of an instrument based on the chemiluminescent reaction of NO_2 with luminol (5-amino-2, 3-dihydro-1, 4-phthalazine dione) has been reported by Maeda et al. (1980). Wendel et al. (1983) have reported modifications of this luminol-based method in which better response time and less interference from O_3 have been achieved.

Other acceptable methods for measuring ambient NO_2 levels, including two methods designated as equivalent methods, are the Lyshkow-modified Griess-Saltzman method, the instrumental colorimetric Griess-Saltzman method, the triethanolamine method, the sodium arsenite method, and the TGS-ANSA method [TGS-ANSA = triethanolamine, guaiacol (*o*-methoxyphenol), sodium metabisulfite, and 8-anilino-1-naphthalene sulfonic acid]. The sodium arsenite method and the TGS-ANSA method were designated as equivalent methods in 1977. For descriptions of these methods, the reader is referred to the EPA criteria document for nitrogen oxides (U.S. Environmental Protection Agency, 1982). While some of these methods measure the species of interest directly, others require oxidation, reduction, or thermal decomposition of the sample, or separation from interferences, before measurement. None of these other techniques, however, is widely used to monitor air quality.

Careful adherence to specified calibration procedures is essential for obtaining accurate NO_x measurements. The U.S. Environmental Protection Agency (1975) has issued a technical assistance document that describes in detail the two acceptable calibration methods for NO_x : (1) the use of standard reference materials (SRMs) and (2) gas-phase titration (GPT) of NO with O_3 . The SRM for NO is a cylinder of compressed NO in N_2 ; the mixture is both accurate and stable (Hughes, 1975). The SRM for NO_2 is the NO_2 permeation tube (O'Keefe and Ortman, 1966; Scaringelli et al., 1970). The gas-phase titration, described by Rehme et al. (1974), is based upon the bimolecular reaction of NO with O_3 to form NO_2 plus O_2 . The U.S. Environmental Protection Agency (1975) recommends the combined use of GPT plus SRM procedures, using one technique to check the other.

1.4 CONCENTRATIONS OF OZONE AND OTHER PHOTOCHEMICAL OXIDANTS IN AMBIENT AIR

In the context of this document, the concentrations of ozone and other photochemical oxidants found in ambient air are important for: (1) assessing potential exposures of human and other receptors; (2) determining the range of ambient air concentrations of ozone and other photochemical oxidants relative to demonstrated "effects levels" (Chapters 6-12); (3) determining indoor-outdoor gradients for exposure analyses; (4) assessing whether the concentrations of oxidants other than ozone, singly, collectively, or in combination with ozone, are cause for concern; and (5) evaluating the adequacy of ozone as a control surrogate for other photochemical oxidants, if concentrations of the other oxidants are cause for concern given the effects and the "effects levels" of those oxidants.

1.4.1 Ozone Concentrations in Urban Areas

In Table 1-3, 1983 ozone concentrations for Standard Metropolitan Statistical Areas (SMSAs) having populations \geq 1 million are given by geographic area, demarcated according to United States Census divisions and regions (U.S. Department of Commerce, 1982). The second-highest concentrations among daily maximum 1-hour values measured in 1983 in the 38 SMSAs having populations of at least 1 million ranged from 0.10 ppm in the Ft. Lauderdale, Florida; Philadelphia, Pennsylvania; and Seattle, Washington, areas to 0.37 ppm in the Los Angeles-Long Beach, California, area. The second-highest value among daily maximum 1-hour ozone concentrations for 35 of the 38 SMSAs in Table 1-3 equaled or exceeded 0.12 ppm. The data clearly show, as well, that the highest 1-hour ozone concentrations in the United States occurred in the northeast (New England and Middle Atlantic states), in the Gulf Coast area (West South Central states), and on the west coast (Pacific states). Second-highest daily maximum 1-hour concentrations in 1983 in the SMSAs within each of these three areas averaged 0.16, 0.17, and 0.21 ppm, respectively. It should be emphasized that these three areas of the United States are subject to those meteorological and climatological factors that are conducive to local oxidant formation, or transport, or both. It should also be emphasized that 9 of the 16 SMSAs in the country with populations \geq 2 million are located in these areas.

TABLE 1-3. SECOND-HIGHEST OZONE CONCENTRATIONS AMONG DAILY MAXIMUM 1-hr VALUES IN 1983 IN STANDARD METROPOLITAN STATISTICAL AREAS WITH POPULATIONS \geq 1 MILLION, GIVEN BY CENSUS DIVISIONS AND REGIONS^a

Division and region	SMSA	SMSA population, millions	Second-highest 1983 O ₃ concn., ppm
<u>Northeast</u>			
New England	Boston, MA	>2	0.18
Middle Atlantic	Buffalo, NY	1 to <2	0.12
	Nassau-Suffolk, NY	>2	0.17
	Newark, NJ	1 to <2	0.25
	New York, NY/NJ	>2	0.19
	Philadelphia, PA/NJ	>2	0.10
	Pittsburgh, PA	>2	0.14
<u>South</u>			
South Atlantic	Atlanta, GA	>2	0.17
	Baltimore, MD	>2	0.19
	Ft. Lauderdale-Hollywood, FL	1 to <2	0.10
	Miami, FL	1 to <2	0.12
	Tampa-St. Petersburg, FL	1 to <2	0.14
	Washington, DC/MD/VA	>2	0.17
<u>South</u>			
West South Central	Dallas-Ft. Worth, TX	>2	0.16
	Houston, TX	>2	0.28
	New Orleans, LA	1 to <2	0.12
	San Antonio, TX	1 to <2	0.12
<u>North Central</u>			
East North Central	Chicago, IL	>2	0.17
	Detroit, MI	>2	0.17
	Cleveland, OH	1 to <2	0.15
	Cincinnati, OH/KY/IN	1 to <2	0.15
	Milwaukee, WI	1 to <2	0.18
	Indianapolis, IN	1 to <2	0.14
	Columbus, OH	1 to <2	0.12
West North Central	St. Louis, MO/IL	>2	0.18
	Minneapolis-St. Paul, MN/WI	>2	0.13
	Kansas City, MO/KS	1 to <2	0.13

TABLE 1-3 (cont'd). SECOND-HIGHEST OZONE CONCENTRATIONS AMONG DAILY MAXIMUM 1-hr VALUES IN 1983 IN STANDARD METROPOLITAN STATISTICAL AREAS WITH ≥ 1 MILLION, GIVEN BY CENSUS DIVISIONS AND REGIONS^a

Division and region	SMSA	SMSA population, millions	Second-highest 1983 O ₃ concn., ppm
<u>West</u>			
Mountain	Denver-Boulder, CO	1 to <2	0.14
	Phoenix, AZ	1 to <2	0.16
Pacific	Los Angeles-Long Beach, CA	>2	0.37
	San Francisco-Oakland, CA	>2	0.17
	Anaheim-Santa Ana-Garden Grove, CA	1 to <2	0.28
	San Diego, CA	1 to <2	0.20
	Seattle-Everett, WA	1 to <2	0.10
	Riverside-San Bernardino-Ontario, CA	1 to <2	0.34
	San Jose, CA	1 to <2	0.16
	Portland, OR/WA	1 to <2	0.12
	Sacramento, CA	1 to <2	0.15

^aStandard Metropolitan Statistical Areas and geographic divisions and regions as defined by Statistical Abstract of the United States (U.S. Department of Commerce, 1982).

Source: U.S. Environmental Protection Agency (1984).

Emissions of manmade oxidant precursors are usually correlated with population, especially emissions from area source categories such as transportation and residential heating (Chapter 3). Accordingly, when grouped by population, the 80 largest SMSAs had the following median values for their collective second-highest daily maximum 1-hour ozone concentrations in 1983: populations ≥ 2 million, 0.17 ppm O₃; populations of 1 to 2 million, 0.14 ppm O₃; and populations of 0.5 to 1 million, 0.13 ppm O₃. As noted above, however, coincident meteorology favorable for oxidant formation undoubtedly contributes to the apparent correlation between population and ozone levels.

Among all stations reporting valid ozone data (≥ 75 percent of possible hourly values per year) in 1979, 1980, and 1981 (collectively, 906 station-years) in the United States, the median second-highest 1-hour ozone value was 0.12 ppm, and 5 percent of the stations reported second-highest 1-hour values ≥ 0.28 ppm.

A pattern of concern in assessing responses to ozone in human populations and in vegetation is the occurrence of repeated or prolonged multiday periods when the ozone concentrations in ambient air are in the range of those known to elicit responses (see Chapters 10 and 12). In addition, the number of days of respite between such multiple-day periods of high ozone is of possible consequence. Data show that repeated, consecutive-day exposures to or respites from specified concentrations are location-specific. At a site in Dallas, Texas, for example, daily maximum 1-hour concentrations were ≥ 0.06 ppm for 2 to 7 days in a row 37 times in a 3-year period (1979 through 1981). A concentration of ≥ 0.18 ppm was recorded at that site on only 2 single days, however, and no multiple-day recurrences of that concentration or greater were recorded over the 3-year period. At a site in Pasadena, California, daily maximum 1-hour concentrations ≥ 0.18 ppm recurred on 2 to 7 consecutive days 33 times in that same 3-year period (1979 through 1981) and occurred, as well, on 21 separate days. These and other data demonstrate the occurrence in some urban areas of multiple-day potential exposures to relatively high concentrations of ozone.

1.4.2. Trends in Nationwide Ozone Concentrations

Trends in ozone concentrations nationwide are important for estimating potential exposures in the future of human populations and other receptors, as well as for examining the effectiveness of abatement programs. The determination of nationwide trends requires the application of statistical tests to comparable, representative, multiyear aerometric data. The derivation of recent trends in ozone concentrations and the interpretation of those trends is complicated by two potentially significant factors that have affected aerometric data since 1979: (1) the promulgation by EPA in 1979 of a new calibration procedure for ozone monitoring (see Chapter 4); and (2) the introduction by EPA of a quality-assurance program that has resulted in improved data-quality audits. The effects of these factors on ozone concentration measurements are superimposed on the effects on concentrations of any changes in meteorology or in precursor emission rates that may have occurred over the same time span.

The nationwide trends in ozone concentrations for a 9-year period, 1975 through 1983, are shown in Figure 1-1 (U. S. Environmental Protection Agency, 1984). The data given are trends as gauged by the composite average of the

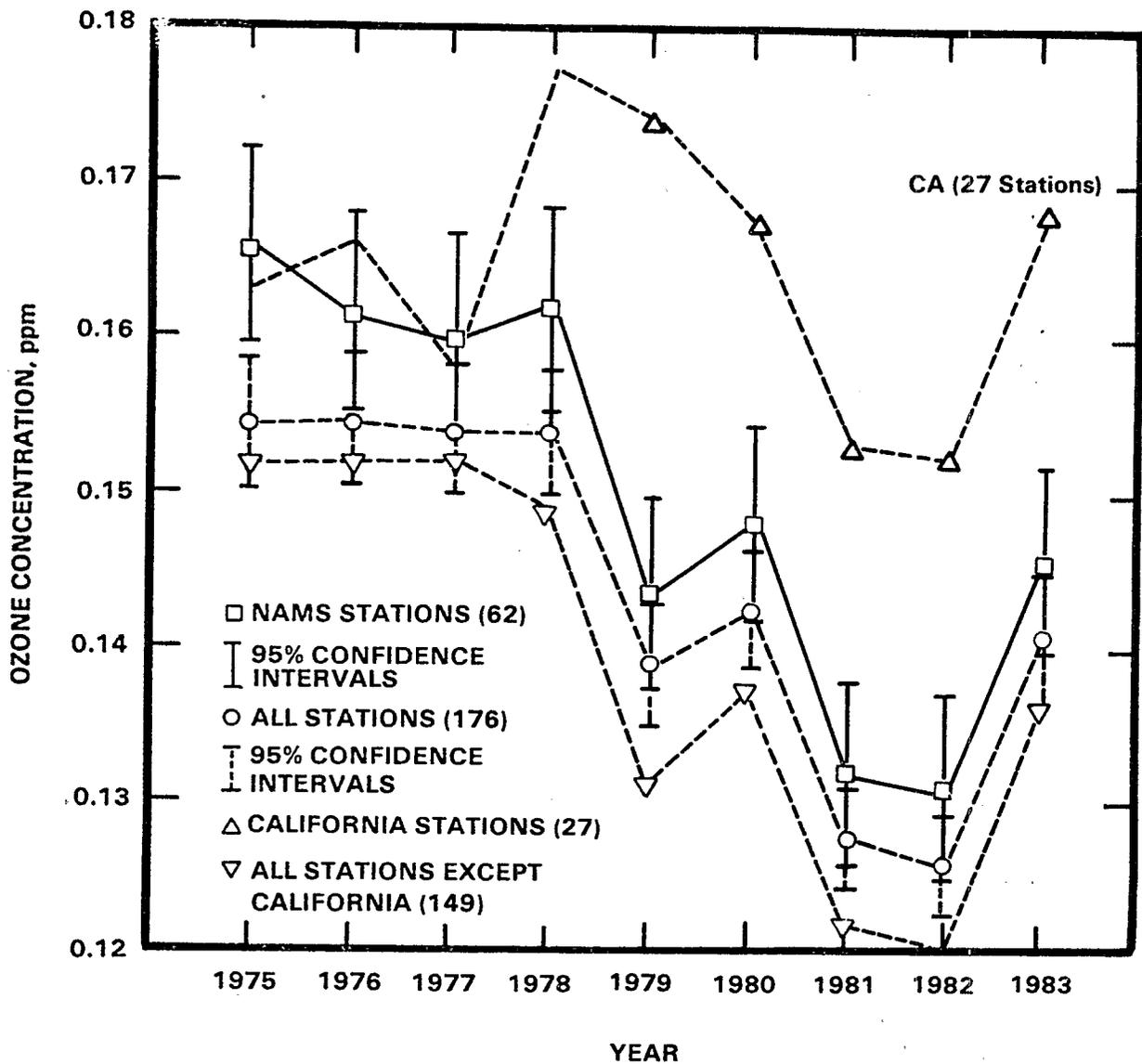


Figure 1-1. National trend in composite average of the second highest value among daily maximum 1-hour ozone concentrations at selected groups of sites, 1975 through 1983.

Source: U.S. Environmental Protection Agency (1984).

second-highest value among daily maximum 1-hour ozone concentrations. Data from four subsets of monitoring stations, most of them urban stations, are given: (1) California stations only; (2) all stations except those in California; (3) all stations including those in California; and (4) all National Air Monitoring Stations (NAMS), which report data directly to EPA. Only stations reporting ≥ 75 percent of possible hourly values in the respective years are represented in the data.

For the entire 9-year period, 1975 through 1983, all subsets of monitoring stations show a decline in the composite second-highest daily maximum 1-hour ozone concentration. Between 1979, when the new, more accurate calibration procedure was promulgated, and 1982, a small decline of 9 to 10 percent in nationwide ozone concentrations occurred. From 1982 to 1983, however, concentrations increased by about 10 percent in California, by about 14 percent outside California, and by about 12 percent nationwide (all states). Recently published data for 1984 from a somewhat smaller number of sites (163) (U.S. Environmental Protection Agency, 1986) show a decrease in nationwide ozone concentrations from 1983 to 1984, with 1984 levels approximating those recorded in 1981. The portion of the apparent decline in ozone nationwide from 1975 through 1984 that is attributable to the calibration change of 1979 cannot be determined by simply applying a correction factor to all data, since not all monitoring stations began using the UV calibration procedure in the same year.

Figure 1-2 shows the nationwide frequency distributions of the first-, second-, and third-highest 1-hour O_3 concentrations at predominantly urban stations aggregated for 1979, 1980, and 1981, as well as the highest 1-hour O_3 concentration at site of the National Air Pollution Background Network (NAPBN) aggregated for the same 3 years. As shown by Figure 1-2, 50 percent of the second-highest 1-hour values from non-NAPBN sites in this 3-year period were 0.12 ppm or less and about 10 percent were equal to or greater than 0.20 ppm. At the NAPBN sites, the collective 3-year distribution (1979 through 1981) is such that about 6 percent of the values are less than 0.10 ppm and fewer than 20 percent are higher than 0.12 ppm.

1.4.3. Ozone Concentrations in Nonurban Areas

Few nonurban areas have been routinely monitored for ozone concentrations. Consequently, the aerometric data base for nonurban areas is considerably less substantial than for urban areas. Data are available, however, from two

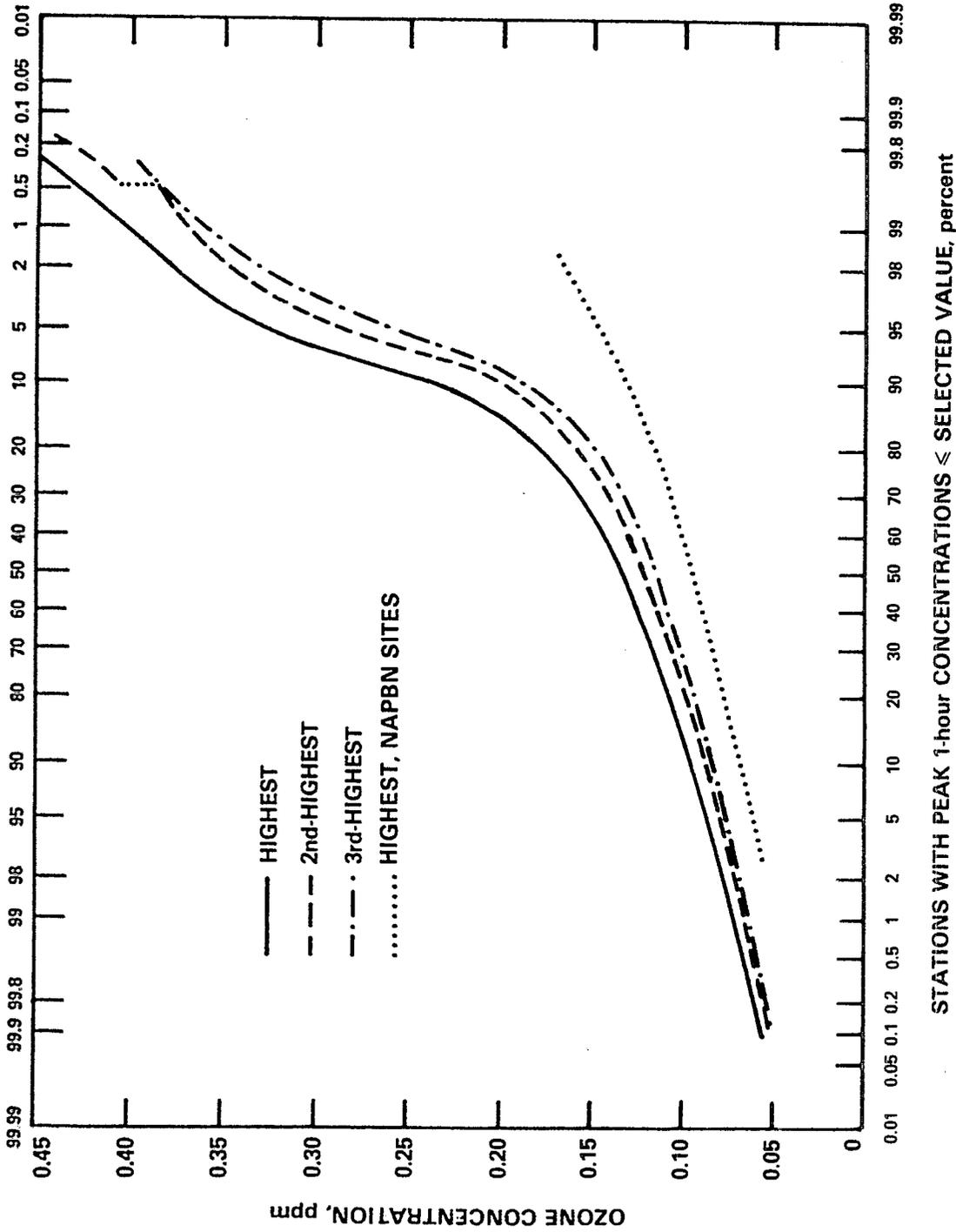


Figure 1-2. Distributions of the three highest 1-hour ozone concentrations at valid sites (906 station-years) aggregated for 3 years (1979, 1980, and 1981) and the highest ozone concentrations at NAPBN sites aggregated for those years (24 station-years).

Source: U.S. Environmental Protection Agency (1980, 1981, 1982).

special-purpose networks, the National Air Pollution Background Network (NAPBN) and the Sulfate Regional Experiment network (SURE). Data on maximum 1-hour concentrations and arithmetic mean 1-hour concentrations reveal that maximum 1-hour concentrations at nonurban sites classified as rural (SURE study, Martinez and Singh, 1979; NAPBN studies, Evans et al., 1983) can sometimes exceed the concentrations observed at sites classified as suburban (SURE study, Martinez and Singh, 1979). For example, maximum 1-hour ozone concentrations measured in 1980 at Kisatchie National Forest (NF), Louisiana; Custer NF, Montana; and Green Mt. NF, Vermont, were 0.105, 0.070, and 0.115 ppm, respectively. Arithmetic mean 1-hour concentrations for 1980 were 0.028, 0.037, and 0.032 ppm at the respective sites. For four nonurban (rural) sites in the SURE study, maximum 1-hour ozone concentrations were 0.106, 0.107, 0.117, and 0.153; and mean 1-hour concentrations ranged from 0.021 to 0.035 ppm. At the five nonurban (suburban) sites of the SURE study, maximum concentrations were 0.077, 0.099, 0.099, 0.080, and 0.118 ppm, respectively. The mean 1-hour concentrations at those sites were 0.023, 0.030, 0.025, 0.020, and 0.025 ppm, respectively.

Ranges of concentrations and the maximum 1-hour concentrations at some of the NAPBN and SURE sites show the probable influence of ozone transported from urban areas. In one documented case, for example, a 1-hour peak ozone concentration of 0.125 ppm at an NAPBN site in Mark Twain National Forest, Missouri, was measured during passage of an air mass whose trajectory was calculated to have included Detroit, Cincinnati, and Louisville in the preceding hours (Evans et al., 1983).

The second-highest concentration among all the daily maximum 1-hour concentrations measured at the NAPBN sites appear to be about one-half the corresponding concentrations measured at urban sites in the same years. No trend in concentrations at these NAPBN sites is discernible in the data record for 1979 through 1983.

These data corroborate the conclusion given in the 1978 criteria document (U.S. Environmental Protection Agency, 1978) regarding urban-nonurban and urban-suburban gradients; i.e., nonurban areas may sometimes sustain higher peak ozone concentrations than those found in urban areas.

1.4.4. Diurnal and Seasonal Patterns in Ozone Concentrations

Since the photochemical reactions of precursors that result in ozone formation are driven by sunlight, as well as by emissions, the patterns of ozone occurrence in ambient air depend on daily and seasonal variations in sunlight intensity. The typical diurnal pattern of ozone in ambient air has a minimum ozone level around sunrise (near zero in most urban areas), increasing through the morning to a peak concentration in early afternoon, and decreasing toward minimal levels again in the evening. The 1978 criteria document ascribed the daily ozone pattern to three simultaneous processes: (1) downward transport of ozone from layers aloft; (2) destruction of ozone through contact with surfaces and through reaction with nitric oxide (NO) at ground level; and (3) in situ photochemical production of ozone (U.S. Environmental Protection Agency, 1978; Coffey et al., 1977; Mohnen, 1977; Reiter, 1977). Obviously, meteorology is a controlling factor; if strong winds disperse the precursors or heavy clouds intercept the sunlight, high ozone levels will not develop. Another important variation on the basic diurnal pattern appears in some localities as a secondary peak in addition to the early afternoon peak. This secondary peak may occur any time from midafternoon to the middle of the night and is attributed to ozone transported from upwind areas where high ozone levels have occurred earlier in the day. Secondary peak concentrations can be higher than concentrations resulting from the photochemical reactions of locally emitted precursors (Martinez and Singh, 1979). At one nonurban site in Massachusetts (August 1977), for example, primary peak concentrations of about 0.11, 0.14, and 0.14 occurred at noon, from noon to about 4:00 p.m., and at noon, respectively, on 3 successive days of high ozone levels (Martinez and Singh, 1979). Secondary peaks at the same site for those same 3 days were about 0.150, 0.157, and 0.130 ppm at about 6:00 p.m., 8:00 p.m., and 8:00 p.m., respectively (Martinez and Singh, 1979).

Because weather patterns, ambient temperatures, and the intensity and wavelengths of sunlight all play important roles in oxidant formation, strong seasonal as well as diurnal patterns exist. The highest ozone levels generally occur in the spring and summer (second and third quarters), when sunlight reaching the lower troposphere is most intense and stagnant meteorological conditions augment the potential for ozone formation and accumulation. Average summer afternoon levels can be from 150 to 250 percent of the average winter afternoon levels. Minor variations in the smog season occur with location,

however. In addition, it is possible for the maximum and second-highest 1-hour ozone concentration to occur outside the two quarters of highest average ozone concentrations. Exceptions to seasonal patterns are potentially important considerations with regard to the protection of crops from ozone damage, especially since respective crops have different growing seasons in terms of length, time of year, and areas of the country in which they are grown.

In addition to the seasonal meteorological conditions that obtain in the lower troposphere, stratospheric-tropospheric exchange mechanisms exist that produce relatively frequent but sporadic, short-term incursions into the troposphere of stratospheric ozone (see Chapter 3). Such incursions show a seasonal pattern, usually occurring in late winter or early spring.

Percentile distributions, by season of the year, of concentration data from all eight NAPBN sites show that the arithmetic mean 1-hour concentration (averaged over a minimum of 3 years of data at each site, for 1977 through 1983) was higher in the second quarter of the year (April, May, June) at seven of the eight stations; and was only negligibly lower than the third-quarter value at the eighth station. The maximum 1-hour concentrations at respective sites, aggregated over 3 to 6 years, depending on the data record for each site, ranged from 0.050 ppm at Custer NF, MT (in the fourth quarter) to 0.155 ppm at Mark Twain NF, MO (in the third quarter). The second-highest 1-hour concentration among maximum daily 1-hour values ranged from 0.050 ppm at Custer NF, MT (in the fourth quarter), to 0.150 ppm at Mark Twain NF, MO (in the third quarter). The data also show that 99 percent of the 1-hour concentrations measured were well below 0.12 ppm, even in the second quarter of the year, when incursions of stratospheric ozone are expected to affect, at least to some degree, the concentrations measured at these stations. Excursions above 0.12 ppm were recorded in 1979 and 1980 at NAPBN sites; but none were recorded in 1981 (Evans et al., 1983; Lefohn, 1984).

Because of the diurnal patterns of ozone, averaging across longer-term periods such as a month, a season, or longer masks the occurrence of peak concentrations (see, e.g., Lefohn and Benedict, 1985). This is an obvious and familiar statistical phenomenon. It is pointed out, however, because it has direct relevance to the protection of public health and welfare. Averaging times must correspond to, or be related in a consistent manner to, the pattern of exposure that elicits untoward responses.

1.4.5 Spatial Patterns in Ozone Concentrations

In addition to temporal variations, both macro- and microscale spatial variations in ozone concentrations occur that have relevance ranging from important to inconsequential for exposure assessments. Differences in concentrations or patterns of occurrence, or both, are known to exist, for example, between urban and nonurban areas, between indoor and outdoor air, within large metropolises, and between lower and higher elevations. The more important variations are summarized below.

1.4.5.1 Urban-Nonurban Differences in Ozone Concentrations. Ozone concentrations differ between urban and rural, between urban and remote, and even between rural and remote sites, as discussed in part in the preceding section on temporal variations. The variations with area and type of site are variations both in the timing and the magnitude of the peak concentrations, and, in the case of transported ozone, are related to the temporal variations between urban and nonurban areas discussed above. Data from urban, suburban, rural, and remote sites (see, e.g., SAROAD, 1985a-f; Martinez and Singh, 1979; Lefohn, 1984; Evans, 1985; respectively) corroborate the conclusion drawn in the 1978 criteria document (U.S. Environmental Protection Agency, 1978) that ozone concentrations can sometimes be higher in some suburban or even rural areas downwind of urban plumes than in the urban areas themselves; and, furthermore, that higher concentrations can persist longer in rural and remote areas, largely because of the absence of nitric oxide (NO) for chemical scavenging. In nonurban areas downwind of urban plumes, peak concentrations can occur, as the result of transport, at virtually any hour of the day or night, depending upon many factors, such as the strength of the emission source, induction time, scavenging, and wind speed (travel time) and other meteorological factors. The dependence of the timing of peak exposures upon these transport-related factors is well-known and is illustrated here by two studies. Evans et al. (1983) calculated multiday trajectories for air parcels reaching a nonurban sites in the Mark Twain National Forest, Missouri, during an episode. Four separate trajectories, all of which passed over the Ohio River Valley and the Great Lakes region, impacted the forest site at different times in a 24-hour period (in which the maximum 1-hour concentration measured was 0.125 ppm). Subsequently, regional cloud cover and rains produced shifts in air flow and also reduced the potential for ozone formation, alleviating the impact at the site. Kelly et al. (1986) showed in the Detroit area that peak

ozone concentrations occurred at distances of 10 to 70 km (ca. 6 to 43 mi) north-northeast of the urban center. Consequently, it would be possible for peak ozone concentrations to occur in the late afternoon or early evening in nonurban areas downwind of Detroit. Kelly et al. (1986) also found that concentrations diminished again beyond 70 km (ca. 43 mi) downwind of the urban center. Thus, as illustrated by these and similar data, beyond the distance traversed in the time required for maximum ozone formation in an urban plume, ozone concentrations will decrease (unless fresh emissions are injected into the plume) as the rate of ozone formation decreases, the plume disperses, surface deposition or other scavenging occurs, and meteorological conditions intervene.

It is not surprising, therefore, that in rural areas lying beyond the point of maximum ozone formation, for a given set of conditions, peak concentrations are lower and average diurnal profiles are flatter than in urban and near-urban areas (see, e.g., SAROAD, 1985b-f, for rural and remote sites). In remote areas beyond the influence of urban plumes, average peak concentrations will be still lower and average diurnal profiles still flatter (see e.g., Evans, 1985). Exceptions to these generalizations occur, of course, because of the complex interactions of topography, meteorology, and photochemistry.

Such temporal and spatial differences between ozone concentrations in urban versus nonurban areas are important considerations for accurately assessing actual and potential exposures for human populations and for vegetation in nonurban areas, especially since the aerometric data for nonurban areas are far from abundant.

1.4.5.2 Geographic, Vertical, and Altitudinal Variations in Ozone Concentrations.

Although of interest and concern when estimating global ozone budgets, demonstrated variations in ozone concentrations with latitude and the lesser variations with longitude have little practical significance for assessing exposure within the contiguous United States. The effects on ozone concentrations of latitude and longitude within the contiguous states are minor, and are reflected in the aerometric data bases. Of more importance, ozone concentrations are known to increase with increasing height above the surface of the earth. Conversely, they may be viewed as decreasing with proximity to the surface of the earth, since the earth's surface acts as a sink for ozone (see, e.g., Seiler and Fishman, 1981; Galbally and Roy, 1980; Oltmans, 1981, cited in Logan et al., 1981). The most pertinent vertical and altitudinal gradients in

ozone concentrations are: (1) increases in concentration with height above the surface of the ground (regardless of altitude); (2) increases in concentration with altitude; and (3) variations in concentrations with elevation in mountainous areas, attributable to transport and overnight conservation of ozone aloft, nocturnal inversions, trapping inversions, upslope flows, and other, often location-specific interactions between topography, meteorology, and photochemistry.

The importance of monitoring concentrations at the proper height above the surface of the ground has been known for a long time, and EPA guidance on the placement of monitoring instruments (see Chapter 4) is predicated on the existence of a vertical gradient as ozone is depleted by reaction with ground-level emissions of NO or by deposition on reactive surfaces such as vegetation. Data illustrative of the near-surface gradient were reported by Pratt et al. (1983), who measured ozone concentrations at two separate heights (3 and 9 or 6 and 9 meters) above the ground at three rural, vegetated sites. Although the maximum mean difference between 3 and 9 meters was 3 ppb, this difference was similar to the mean difference between sites at the same height. Given the height of some vegetation canopies, especially forests, even such small differences over a spread of 6 meters should probably be taken into consideration when interpreting reported dose-response functions.

The gradual increase in ozone concentrations with altitude has been documented by a number of workers (see e.g., Viezee et al., 1979; Seiler and Fishman, 1981; Oltmans, 1981, as cited in Logan et al., 1981). There is a general increase in concentration with altitude, but as described by Seiler and Fishman (1981) and Oltmans (1981; cited in Logan et al., 1981), for example, two relatively pronounced gradients exist, one between the surface of the earth and 2 km (ca. 1 mi) and one even more pronounced between 8 and 12 km (ca. 5 and 7.5 mi).

Increases in concentrations with altitude could potentially be of some consequence for passengers and airline personnel on high-altitude flights in the absence of adequate ventilation-filtration systems (see Chapter 11). Variations with height above the surface and with elevation, in mountainous areas, however, should be taken into account to ensure the accurate assessment of exposures and the accurate derivation of dose-response functions, especially for forests and other vegetation.

Variations in ozone concentrations with elevation, not always consistent or predictable, have been reported by researchers investigating the effects of ozone on the mixed-conifer forest ecosystem of the San Bernardino Mountains of California. Measurements taken at four monitoring stations at four different elevations showed that peak ozone concentrations occurred progressively later in the day at progressively higher elevations (Miller et al., 1972). Ozone concentrations >0.10 ppm occurred for average durations of 9, 13, 9, and 8 hr/day at the four respective stations, going from lower to higher elevations. The occurrence for 13 hr/day of concentrations >0.10 ppm at the station at 817 m (2860 ft) was probably the result of contact of that zone of the mountainside with the inversion layer (U.S. Environmental Protection Agency, 1978). Nighttime concentrations rarely decreased below 0.05 ppm at the mountain crest; whereas at the lowest elevation, the basin station at 442 m (1459 ft), the nighttime concentration usually decayed to near zero. Trapping inversions were major contributors to the elevational gradients observed in this study, which was conducted in the 1970s. Oxidant concentrations within the inversion were found not to be uniform but to occur in multiple layers and strong vertical gradients. The important result of the trapping of oxidants in the inversion layers was the prolonged contact of high terrain with oxidants at night (U.S. Environmental Protection Agency, 1978).

In a more recent report, Wolff et al. (1986) described measurements made in July 1975 at three separate elevations at High Point Mountain in northeastern New Jersey. The daily ozone maxima were similar at different elevations. At night, however, ozone concentrations were nearly zero in the valley but increased with elevation on the mountainside. Greater cumulative doses (number of hours at >0.08 ppm) were sustained at the higher elevations, 300 and 500 m, respectively (ca. 990 and 1650 ft, respectively). Wolff et al. (1986) related this phenomenon to the depth of the nocturnal inversion layer and the contact with the mountainside of ozone conserved aloft at night.

These concentration gradients with increased elevation are important for accurately describing concentrations at which injury or damage to vegetation, especially forests, may occur. Researchers investigating the effects of ozone on forest ecosystems have seldom measured nighttime ozone concentrations because the stomates of most species are thought to be closed at night, thus preventing the internal flux of ozone that produces injury or damage (see Chapter 6). If stomates remain even partially open at night, however, the

possible occurrence of nighttime peak concentrations of ozone, the occurrence of multiple peaks in a 24-hour period, or the persistence of elevated concentrations that do not decay to near zero overnight should not be overlooked. Furthermore, the lack of NO for nighttime scavenging in nonurban areas and the persistence of ozone overnight at higher elevations will result in the presence of relatively higher concentrations in such areas at sunrise when the stomates open and photosynthesis begins. This possibility requires that exposure assessments, in the absence of sufficient aerometric data for forests and other vegetated areas, take such factors into consideration.

1.4.5.3 Other Spatial Variations in Ozone Concentrations. Other spatial variations are important for exposure assessments for human populations. Indoor-outdoor gradients in ozone concentrations are known to occur even in buildings or vehicles ventilated by fresh air rather than air conditioning (e.g., Sabersky et al., 1973; Thompson et al., 1973; Peterson and Sabersky, 1975). Ozone reacts with surfaces inside buildings, so that decay may occur fairly rapidly, depending upon the nature of interior surfaces and furnishings (e.g., Davies et al., 1984; Contant et al., 1985). Ratios of indoor-to-outdoor (I/O) ozone concentrations are quite variable, however, since cooling and ventilation systems, air infiltration or exchange rates, interior air circulation rates, and the composition of interior surfaces all affect indoor ozone concentrations. Ratios (I/O, expressed as percentages) in the literature thus vary from 100 percent in a non-air-conditioned residence (Contant et al., 1985); to 80 ± 10 percent (Sabersky et al., 1973) in an air-conditioned office building (but with 100 percent outside air intake); to 10 to 25 percent in air-conditioned residences (Berk et al., 1981); and to as low as near zero in air-conditioned residences (Stock et al., 1983; Contant et al., 1985).

On a larger scale, within-city variations in ozone concentrations can occur, even though ozone is a "regional" pollutant. Data show, for example, relatively homogeneous ozone concentrations in New Haven, Connecticut (SAROAD, 1985a), a moderately large city that is downwind of a reasonably well-mixed urban plume (Wolff et al., 1975; Cleveland et al., 1976a,b). In a large metropolis, however, appreciable gradients in ozone concentrations can exist from one side of the city to the other, as demonstrated for New York City (Smith, 1981), and for Detroit (Kelly et al., 1986). Such gradients should be taken into consideration, where possible, in exposure assessments.

1.4.6 Concentrations and Patterns of Other Photochemical Oxidants

1.4.6.1 Concentrations. No aerometric data are routinely obtained by Federal, state, or local air pollution agencies for any photochemical oxidants other than nitrogen dioxide and ozone. The concentrations presented in this document for non-ozone oxidants were all obtained in special field investigations. The limitations in the number of locations and areas of the country represented in the information presented simply reflect the relative paucity of data in the published literature.

The four non-ozone photochemical oxidants for which at least minimal concentration data are available are formic acid, peroxyacetyl nitrate (PAN), peroxypropionyl nitrate (PPN), and hydrogen peroxide (H_2O_2). Peroxybenzoyl nitrate has not been clearly identified in ambient air in the United States.

The highest concentrations of PAN reported in the older literature, 1960 through the present, were those found in the Los Angeles area: 70 ppb (1960), 214 ppb (1965); and 68 ppb (1968) (Renzetti and Bryan, 1961; Mayrsohn and Brooks, 1965; Lonneman et al., 1976; respectively).

The highest concentrations of PAN measured and reported in urban areas in the past 5 years were 42 ppb at Riverside, California, in 1980 (Temple and Taylor, 1983) and 47 ppb at Claremont, California, also in 1980 (Grosjean 1981). These are clearly the maximum concentrations of PAN reported for California and for the entire country in this period. Other maximum PAN concentrations measured in the last decade in the Los Angeles Basin have been in the range of 11 to 37 ppb. Average concentrations of PAN in the Los Angeles Basin in the past 5 years have ranged from 4 to 13 ppb (Tuazon et al., 1981a; Grosjean, 1983; respectively). The only published study covering urban PAN concentrations outside California in the past 5 years is that of Lewis et al. (1983) for New Brunswick, New Jersey, in which the average PAN concentration was 0.5 ppb and the maximum was 11 ppb during September 1978 through May 1980. Studies outside California from the early 1970s through 1978 showed average PAN concentrations ranging from 0.4 ppb in Houston, Texas, in 1976 (Westberg et al., 1978) to 6.3 ppb in St. Louis, Missouri, in 1973 (Lonneman et al., 1976). Maximum PAN concentrations outside California for the same period ranged from 10 ppb in Dayton, Ohio, in 1974 (Spicer et al., 1976) to 25 ppb in St. Louis (Lonneman et al., 1976).

The highest PPN concentration reported in studies over the period 1963 through the present was 6 ppb in Riverside, California (Darley et al., 1963).

The next highest reported PPN concentration was 5 ppb at St. Louis, Missouri, in 1973 (Lonneman et al., 1976). Among more recent data, maximum PPN concentrations at respective sites ranged from 0.07 ppb in Pittsburgh, Pennsylvania, in 1981 (Singh et al., 1982) to 3.1 ppb at Staten Island, New York (Singh et al., 1982). California concentrations fell within this range. Average PPN concentrations at the respective sites for the more recent data ranged from 0.05 ppb at Denver and Pittsburgh to 0.7 ppb at Los Angeles in 1979 (Singh et al., 1981).

Altshuller (1983) has succinctly summarized the nonurban concentrations of PAN and PPN by pointing out that they overlap the lower end of the range of urban concentrations at sites outside California. At remote locations, PAN and PPN concentrations are lower than even the lowest of the urban concentrations by a factor of 3 to 4.

The concentrations of H_2O_2 reported in the literature to date must be regarded as inaccurate since ozone is now thought to be an interference in all methods used to date except FTIR (Chapter 4). Measurements by FTIR, the most specific and accurate method now available, have not demonstrated unambiguously the presence of H_2O_2 in ambient air, even in the high-oxidant atmosphere of the Los Angeles area. (The limit of detection for a 1-km-pathlength FTIR system is around 0.04 ppm.)

Recent data indicate the presence in urban atmospheres of only trace amounts of formic acid: ≤ 15 ppb, measured by FTIR (Tuazon et al., 1981b). Estimates in the earlier literature (1950s) of 600 to 700 ppb of formic acid in smoggy atmospheres were erroneous because of faulty measurement methodology (Hanst et al., 1982).

1.4.6.2 Patterns. The patterns of formic acid ($HCOOH$), PAN, PPN, and H_2O_2 may be summarized fairly succinctly. Qualitatively, diurnal patterns are similar to those of ozone, with peak concentrations of each of these occurring in close proximity to the time of the ozone peak. The correspondence in time of day is not exact, but is close. As demonstrated by the work of Tuazon et al. (1981b), ozone concentrations return to baseline levels somewhat faster than the concentrations of PAN, $HCOOH$, or H_2O_2 (PPN was not measured).

Seasonally, winter concentrations (third and fourth quarters) of PAN are lower than summer concentrations (second and third quarters). The percentage of PAN concentrations ($PAN/O_3 \times 100$) relative to ozone, however, is higher in winter than in summer. Data are not readily available on the seasonal patterns of the other non-ozone oxidants.

Indoor-outdoor data on PAN are limited to one report (Thompson et al., 1973), which confirms the pattern to be expected from the known chemistry of PAN; that is, it persists longer indoors than ozone. Data are lacking on indoor-outdoor ratios for the other non-ozone oxidants.

1.4.7 Relationship Between Ozone and Other Photochemical Oxidants

The relationship between ozone concentrations and the concentrations of PAN, PPN, H_2O_2 , and $HCOOH$ is important only if these non-ozone oxidants are shown to produce potentially adverse health or welfare effects, singly, in combination with each other, or in various combinations with ozone at concentrations corresponding to those found in ambient air. If only ozone is shown to produce adverse health or welfare effects in the concentration ranges of concern, then only ozone must be controlled. If any or all of these other four oxidants are shown to produce potentially adverse health or welfare effects, at or near levels found in ambient air, then such oxidants will also have to be controlled. Since ozone and all four of the other oxidants arise from reactions among the same organic and inorganic precursors, an obvious question is whether the control of ozone will also result in the control of the other four oxidants.

Controlled-exposure studies on these non-ozone oxidants have employed concentrations much higher than those found in ambient air (see Chapters 9 and 10). Because PAN may have contributed, however, to the eye irritation symptoms reported in earlier epidemiological studies, and because PAN is the most abundant of these non-ozone oxidants, the relationship between ozone and PAN concentrations in ambient air remains of interest.

The patterns of PAN and ozone concentrations are not quantitatively similar but do show qualitative similarities for most locations at which both pollutants have been measured in the same study. That a quantitative, monotonic relationship between ozone and PAN is lacking, however, is shown by the range of PAN-to-ozone ratios, expressed as percentages, between locations and at the same location, as reported in the review of Altshuller (1983).

Certain other information bears out the lack of a monotonic relationship between PAN and ozone. Not only are PAN-ozone relationships not consistent between different urban areas, but they are not consistent in urban versus nonurban areas, in summer versus winter, in indoor versus outdoor environments, or even, as the data show, in location, timing, or magnitude of diurnal peak

concentrations within the same city. Data obtained in Houston by Jorgen et al. (1978), for example, show variations in peak concentrations of PAN and in relationships to ozone concentrations of those peaks among three separate monitoring sites. Temple and Taylor (1983) have shown that PAN concentrations are a greater percentage of ozone concentrations in winter than in the remainder of the year in California. Lonneman et al. (1976) demonstrated that PAN, absolutely and as a percentage of ozone, is considerably lower in nonurban than in urban areas. Thompson et al. (1973), in what is apparently the only published report on indoor concentrations of PAN, showed that PAN persists longer than ozone indoors. (This is to be expected from its enhanced stability at cooler-than-ambient temperatures such as found in air-conditioned buildings.) Tuazon et al. (1981b) demonstrated that PAN persists in ambient air longer than ozone, its persistence paralleling that of nitric acid, at least in the locality studied (Claremont, CA). Reactivity data presented in the 1978 criteria document for ozone and other photochemical oxidants indicated that all precursors that give rise to PAN also give rise to ozone. The data also showed, however, that not all precursors giving rise to ozone also give rise to PAN, and that not all that give rise to both are equally reactive toward both, with some precursors preferentially giving rise, on the basis of units of product per unit of reactant, to more of one product than the other (U.S. Environmental Protection Agency, 1978).

In the review cited earlier, Altshuller (1983) examined the relationships between ozone and a variety of other smog components, including PAN, PPN, H_2O_2 , $HCOOH$, aldehydes, aerosols, and nitric acid. He concluded that "the ambient air measurements indicate that ozone may serve directionally, but cannot be expected to serve quantitatively, as a surrogate for the other products" (Altshuller, 1983). It must be emphasized that the issue Altshuller examined was whether ozone could serve as an abatement surrogate for all photochemical products, not just the subset of non-ozone oxidants of concern in this document. Nevertheless, a review of the data presented indicates that his conclusion is applicable to the non-ozone oxidants examined in this document.

1.5 EFFECTS OF OZONE AND OTHER PHOTOCHEMICAL OXIDANTS ON VEGETATION

Foliar injury on vegetation is one of the earliest and most obvious manifestations of O_3 injury. The effects of O_3 are not limited to visible injury, however. Impacts can range from reduced plant growth and decreased yield, to changes in crop quality and alterations in susceptibility to abiotic and biotic stresses. The plant foliage is the primary site of O_3 effects, although significant secondary effects, including reduced growth (both roots and foliage) and yield, can occur.

Ozone exerts a phytotoxic effect only if a sufficient amount reaches the sensitive cellular sites within the leaf. The O_3 diffuses from the ambient air into the leaf through the stomata, which can exert some control on O_3 uptake, to the active sites within the leaf. Ozone injury will not occur if (1) the rate of O_3 uptake is low enough that the plant can detoxify or metabolize O_3 or its metabolites; or (2) the plant is able to repair or compensate for the effects (Tingey and Taylor, 1982). This is analogous to the plant response to SO_2 (Thomas et al., 1950). Cellular disturbances that are not repaired or compensated are ultimately expressed as visible injury to the leaf or as secondary effects that can be expressed as reduced root growth, or reduced yield of fruits or seeds, or both.

Plant growth and yield are the end products of a series of biochemical and physiological processes related to uptake, assimilation, biosynthesis, and translocation. Sunlight drives the processes that convert carbon dioxide into the organic compounds (assimilation) necessary for plant growth and development. In addition to nutrients supplied through photosynthesis, the plant must extract from the soil the essential mineral nutrients and water for plant growth. Plant organs convert these raw materials into a wide array of compounds required for plant growth and yield. A disruption or reduction in the rates of uptake, assimilation, or subsequent biochemical reactions will be reflected in reduced plant growth and yield. Ozone would be expected to reduce plant growth or yield if (1) it directly impacted the plant process that was limiting plant growth; or (2) it impacted another step sufficiently so that it becomes the step limiting plant growth (Tingey, 1977). Conversely, O_3 will not limit plant growth if the process impacted by O_3 is not or does not become rate-limiting. This implies that not all effects of O_3 on plants are reflected in growth or yield reductions. These conditions also suggest that there are combinations of O_3 concentration and exposure duration that the plant can

experience that will not result in visible injury or reduced plant growth and yield. Indeed, numerous studies have demonstrated combinations of concentration and time that did not cause a significant effect on the plant growth or yield.

Ozone induces a diverse range of effects on plants and plant communities. These effects are usually classified as either injury or damage. Injury encompasses all plant reactions such as reversible changes in plant metabolism (e.g., altered photosynthesis), leaf necrosis, altered plant quality, or reduced growth that does not impair yield or the intended use of the plant (Guderian, 1977). In contrast, damage or yield loss includes all effects that reduce or impair the intended use or the value of the plant. Thus, for example, visible foliar injury to ornamental plants, detrimental responses in native species, and reductions in fruit and grain production are all considered damage or yield loss. Although foliar injury is not always classified as damage, its occurrence is an indication that phytotoxic concentrations of O_3 are present. The occurrence of injury indicates that additional studies should be conducted in areas where vegetation shows foliar injury to assess the risk of O_3 to vegetation and to determine if the intended use or value of the plants is being impaired.

1.5.1 Limiting Values of Plant Response to Ozone

Several approaches have been used to estimate the O_3 concentrations and exposure durations that induce foliar injury. Most of these studies used short-term exposures (less than 1 day) and measured visible injury as the response variable. One method for estimating the O_3 concentrations and exposure durations that would induce specific amounts of visible injury involves exposing plants to a range of O_3 concentrations and exposure durations, and then evaluating the data by regression analysis (Heck and Tingey, 1971). The data obtained by this method for several species are summarized in Table 1-4 to illustrate the range of concentrations required to induce foliar injury (5% and 20%) on sensitive, intermediate, and less sensitive species.

An alternative method for estimating the O_3 concentrations and exposure durations that induce foliar injury is the use of the limiting-value approach (Jacobson, 1977). The limiting-value method, which was developed from a review of the literature, identified the lowest concentration and exposure duration reported to cause visible injury on various plant species. The analysis was based on more than 100 studies of agricultural crops and 18

TABLE 1-4. OZONE CONCENTRATIONS FOR SHORT-TERM EXPOSURES THAT PRODUCE 5 OR 20 PERCENT INJURY TO VEGETATION GROWN UNDER SENSITIVE CONDITIONS^a
(ppm)

Exposure time, hr	Ozone concentrations that may produce 5% (20%) injury:		
	Sensitive plants	Intermediate plants	Less sensitive plants
0.5	0.35 - 0.50 (0.45 - 0.60)	0.55 - 0.70 (0.65 - 0.85)	≥0.70 (0.85)
1.0	0.15 - 0.25 (0.20 - 0.35)	0.25 - 0.40 (0.35 - 0.55)	≥0.40 (0.55)
2.0	0.09 - 0.15 (0.12 - 0.25)	0.15 - 0.25 (0.25 - 0.35)	≥0.30 (0.40)
4.0	0.04 - 0.09 (0.10 - 0.15)	0.10 - 0.15 (0.15 - 0.30)	≥0.25 (0.35)
8.0	0.02 - 0.04	0.07 - 0.12	≥0.20 (0.30)

^aThe concentrations in parenthesis are for the 20% injury level.

Source: U.S. Environmental Protection Agency (1978).

studies of tree species. The analysis yielded the following range of concentrations and exposure durations that were likely to induce foliar injury (U.S. Environmental Protection Agency, 1978):

1. Agricultural crops:
 - a. 0.20 to 0.41 ppm for 0.5 hr.
 - b. 0.10 to 0.25 ppm for 1.0 hr.
 - c. 0.04 to 0.09 ppm for 4.0 hr.
2. Trees and shrubs:
 - a. 0.20 to 0.51 ppm for 1.0 hr.
 - b. 0.10 to 0.25 ppm for 2.0 hr.
 - c. 0.06 to 0.17 ppm for 4.0 hr.

It should be emphasized that both methods described above can estimate concentrations and exposure durations that might induce visible injury, but that neither method can predict impacts of O₃ on crop yield or intended use.

The concept of limiting values also was used to estimate the O₃ concentrations and exposure durations that could potentially reduce plant growth and yield (U.S. Environmental Protection Agency, 1978). The data were analyzed

and plotted in a manner similar to the approach used by Jacobson (1977) (Figure 1-3). In Figure 1-3 the line bounds mean O_3 concentrations and exposure durations below which effects on plant growth and yield were not detected. This graphical analysis used data from both greenhouse and field studies and indicated that the lower limit for reduced plant performance was a mean O_3 concentration of 0.05 ppm for several hours per day for exposure periods greater than 16 days. At 10 days the O_3 response threshold increased to about 0.10 ppm, and to about 0.30 ppm at 6 days.

1.5.2 Methods for Determining Ozone Yield Losses

Diverse experimental procedures have been used to study the effects of O_3 on plants, ranging from studies done under highly controlled conditions, to exposures in open-top chambers, and to field exposures without chambers. In general, the more controlled conditions are most appropriate for investigating specific responses and for providing the scientific basis for interpreting and extrapolating results. These systems are powerful tools for adding to an understanding of the biological effects of air pollutants. To assess, however, the impact of O_3 on plant yield and to provide data for economic assessments, deviations from the typical environment in which the plant is grown should be minimized. For field crops, this implies that the studies should be conducted in the field, but for crops that are typically grown in glass houses, the studies should be conducted under glass-house conditions.

To improve estimates of yield loss in the field, the National Crop Loss Assessment Network (NCLAN) was initiated by EPA in 1980 to estimate the magnitude of crop losses caused by O_3 (Heck et al., 1982). The primary objectives of NCLAN were:

1. To define the relationships between yields of major agricultural crops and O_3 exposure as required to provide data necessary for economic assessments and the development of National Ambient Air Quality Standards;
2. To assess the national economic consequences resulting from the exposure of major agricultural crops to O_3 ;
3. To advance understanding of the cause and effect relationships that determine crop responses to pollutant exposures.

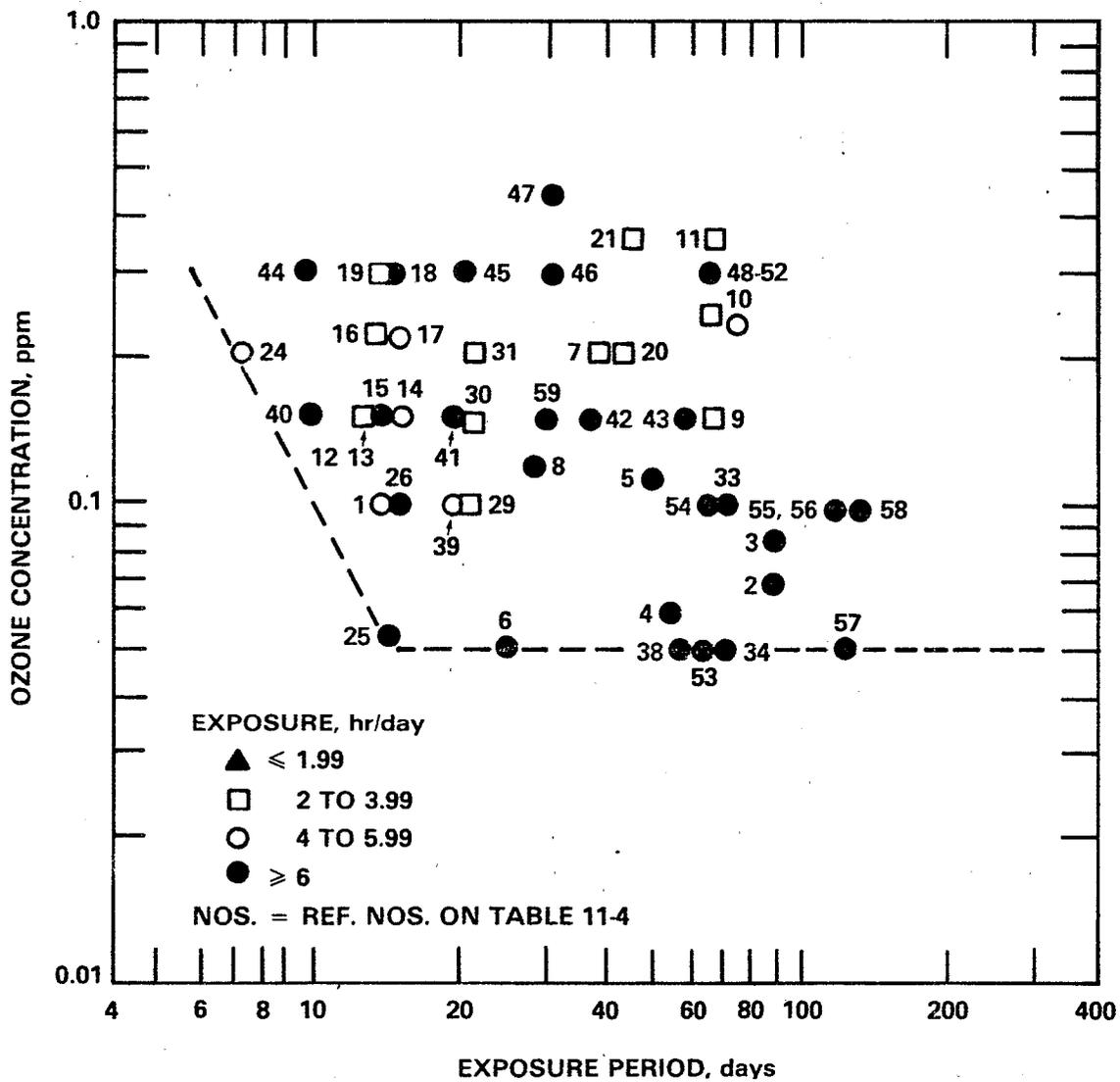


Figure 1-3. Relationship between ozone concentration, exposure duration, and reduction in plant growth or yield (see Table 6-18; also U.S. EPA, 1978).

Source: U.S. Environmental Protection Agency (1978).

In the NCLAN studies, the cultural conditions used approximated typical agronomic practices, and open-top field exposure chambers were used to minimize perturbations to the plant environment during the exposure. The studies have attempted to use a range of realistic O_3 concentrations and sufficient replication to permit the development of exposure-response models. In the NCLAN studies, plants were exposed to a range of O_3 concentrations. Chambers were supplied with either charcoal-filtered air (control), ambient air, or ambient air supplemented with O_3 to provide concentrations three or four levels greater than ambient. Consequently, the O_3 exposures were coupled to the ambient O_3 level; days with the highest ambient O_3 were also the same days when the highest concentrations occurred in a specific treatment in a chamber. As the ambient O_3 varied from day-to-day, the base to which additional O_3 was added also varied. This coupling of the O_3 exposures to the ambient environment means that high O_3 concentrations occurred in the chambers when the environmental and air chemistry conditions, in the ambient air, were conducive for producing elevated ambient O_3 levels. The plant response data have been analyzed using regression approaches. The exposures were typically characterized by a 7-hr (9:00 a.m. to 4:00 p.m.) seasonal mean O_3 concentration. This is the time period when O_3 was added to the exposure chambers.

1.5.3 Estimates of Ozone-Induced Yield Loss

Yield loss is defined as an impairment or decrease in the intended use of the plant. Included in the concept of yield loss are reductions in aesthetic values, the occurrence of foliar injury (changes in plant appearance), and losses in terms of weight, number, or size of the plant part that is harvested. Yield loss may also include changes in physical appearance, chemical composition, or ability to withstand storage; which collectively are traits called crop quality. Losses in aesthetic values are difficult to quantify. For example, because of its aesthetic value, the loss of or adverse effect on a specimen plant in a landscape planting may result in a greater economic loss than that incurred by the same impact on a plant of the same species growing as a part of natural plant community. Foliar injury symptoms may decrease the value of ornamental plants with or without concomitant growth reductions. Similarly, foliar injury on crops in which the foliage is the marketable plant part (e.g., spinach, lettuce, cabbage) can substantially reduce marketability and thus can constitute yield loss. Attainment of the limiting values for

ozone previously discussed in this section should be sufficient to prevent foliar injury and thereby reduce this type of yield loss. Most studies of the relationship between yield loss and ozone concentration have focused on yields as measured by weight of the marketable plant organ, and that kind of yield loss will be the primary focus of this section.

Studies have been conducted, frequently using open-top field exposure chambers, to estimate the impact of O_3 on the yield of various crop species. These studies can be grouped into two types, depending on the experimental design and statistical methods used to analyze the data: (1) studies that developed predictive equations relating O_3 exposure to plant response, and (2) studies that compared discrete treatment levels to a control. The advantage of the regression approach is that exposure-response models can be used to interpolate results between treatment levels.

When the regression approach was used to estimate yield loss, O_3 was added to either charcoal-filtered or ambient air to create a range of O_3 concentrations. In summarizing the data, O_3 -induced yield loss was derived from a comparison of the performance of the plants in charcoal-filtered air, although other reference concentrations have been used. Various regression techniques have been used to derive exposure-response functions. The use of regression approaches permits the estimation of the O_3 impact on plant yield over the range of concentrations, not just at the treatment means as is the case with analysis of variance methods.

1.5.3.1 Yield Loss: Determination by Regression Analysis. Examples of the relationship between O_3 concentration and plant yield are shown in Figures 1-4 and 1-5. These cultivars and species were selected because they also illustrated the type of year-to-year variation in plant response to ozone that may occur. The derived regression equations can be used to determine the concentrations that would be predicted to cause a specific yield loss or to estimate the predicted yield loss that would result from a specific O_3 concentration. Both approaches have been used to summarize the data on crop responses to O_3 using the Weibull function (Rawlings and Cure, 1985). As an example of response, the O_3 concentrations that would be predicted to cause a 10 or 30 percent yield loss have been estimated (Table 1-5). A brief review of the data in this table indicates that for some species mean yield reductions of 10 percent were predicted when the 7-hr seasonal mean O_3 concentration exceeded 0.04 to 0.05 ppm. Concentrations of 0.028 to 0.033 ppm were predicted to

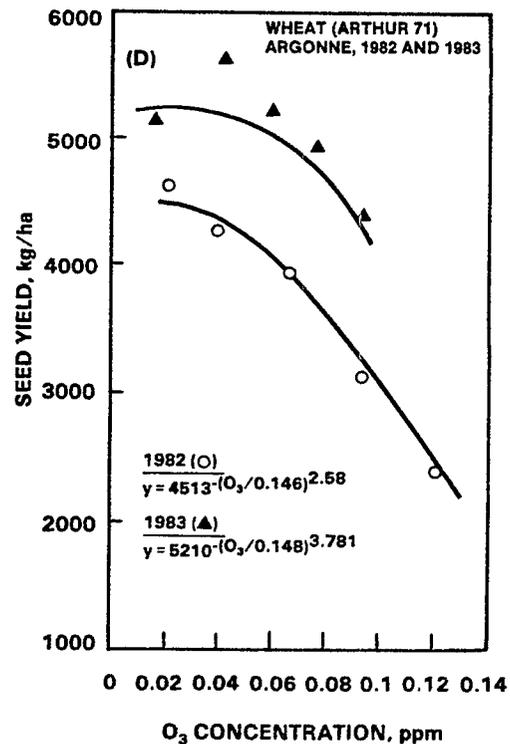
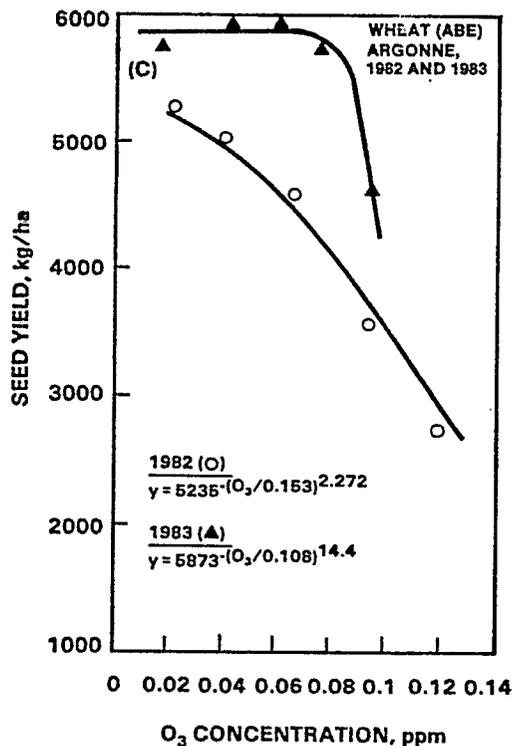
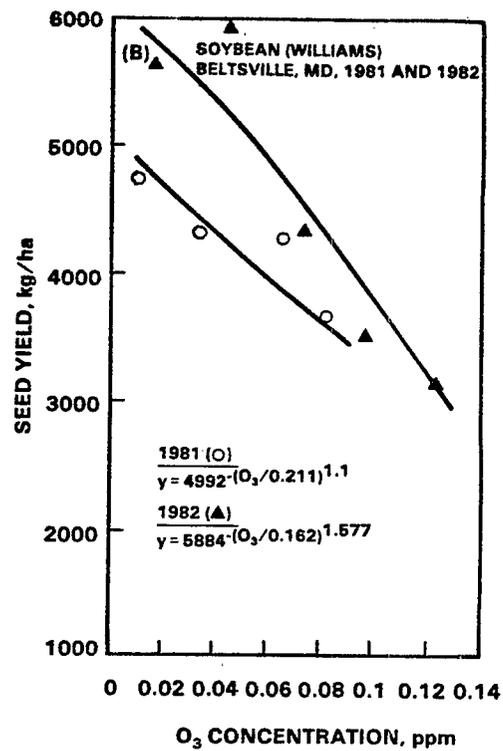
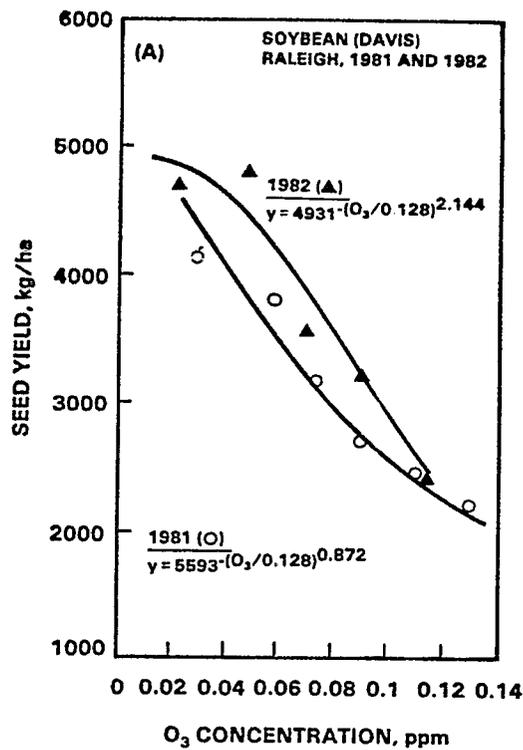


Figure 1-4. Examples of the effects of ozone on the yield of soybean and wheat cultivars. The O₃ concentrations are expressed as 7-hr seasonal mean concentrations. The cultivars were selected as examples of O₃ effects and of year-to-year variations in plant response to O₃.

Source: Soybean data from Heck et al. (1984); wheat data from Kress et al. (1985).

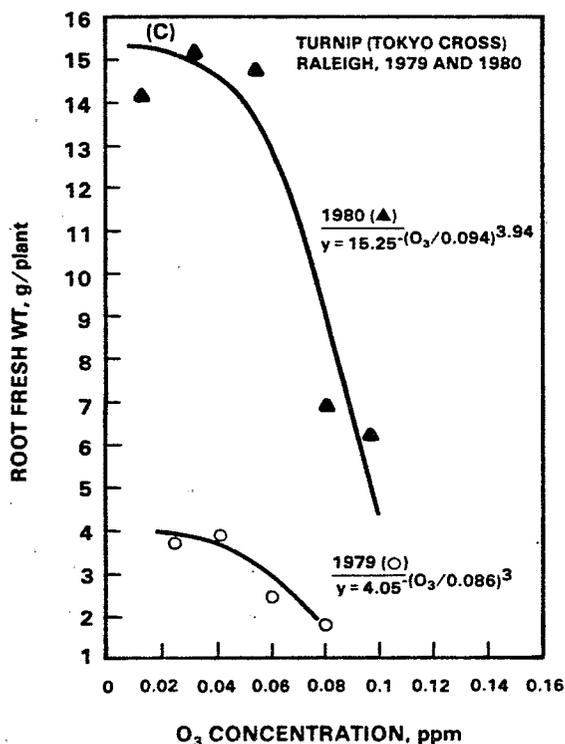
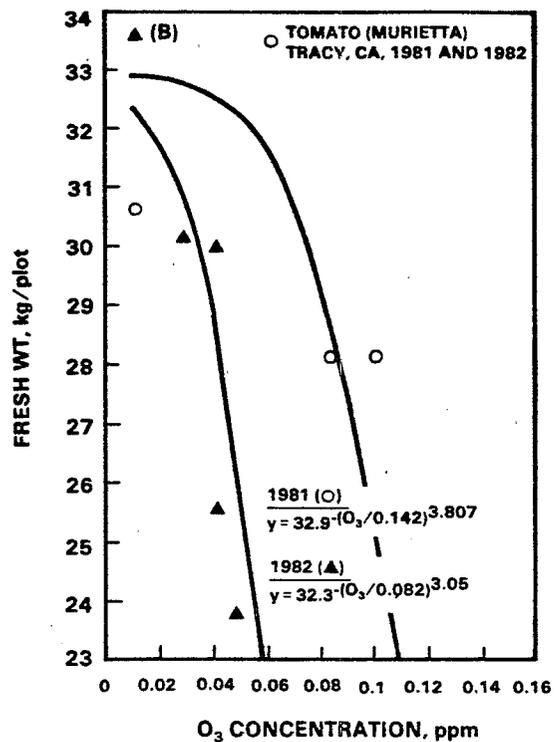
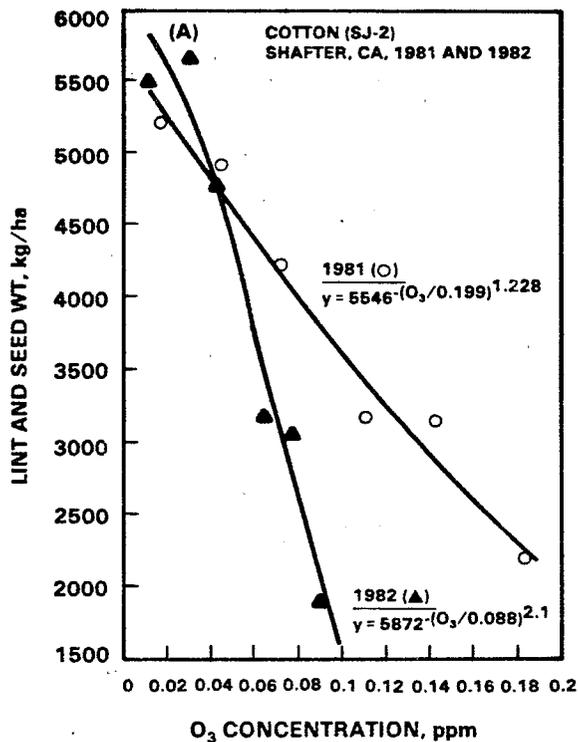


Figure 1-5. Examples of the effects of ozone on the yield of cotton, tomato, and turnip. The O₃ concentrations are expressed as 7-hr seasonal mean concentrations. The species were selected as examples of O₃ effects and of year-to-year variations in plant response to O₃.

Source: Cotton and tomato data from Heck et al. (1984); turnip data from Heagle et al. (1985).

TABLE 1-5. SUMMARY OF OZONE CONCENTRATIONS PREDICTED TO CAUSE 10 PERCENT AND 30 PERCENT YIELD LOSSES AND SUMMARY OF YIELD LOSSES PREDICTED TO OCCUR AT 7-hr SEASONAL MEAN OZONE CONCENTRATIONS OF 0.40 and 0.06 ppm^a

Species	O ₃ concentrations, ppm, predicted to cause yield losses of:		Percent yield losses predicted to occur at 7-hr seasonal mean O ₃ concentration of:	
	10%	30%	0.04 ppm	0.06 ppm
<u>Legume crops</u>				
Soybean, Corsoy	0.048	0.082	6.4	16.6
Soybean, Davis (81)	0.038	0.071	11.5	24.1
Soybean, Davis (CA-82)	0.048	0.081	6.4	16.5
Soybean, Davis (PA-82)	0.059	0.081	2.0	10.4
Soybean, Essex	0.048	0.099	7.2	14.3
Soybean, Forrest	0.076	0.118	1.7	5.3
Soybean, Williams	0.039	0.093	10.4	18.1
Soybean, Hodgson	0.032	0.066	15.4	18.4
Bean, Kidney	0.033	0.063	14.9	28
Peanut, NC-6	0.046	0.073	6.4	19.4
<u>Grain crops</u>				
Wheat, Abe	0.059	0.095	3.3	10.4
Wheat, Arthur 71	0.056	0.094	4.1	11.7
Wheat, Roland	0.039	0.067	10.3	24.5
Wheat, Vona	0.028	0.041	28.8	51.2
Wheat, Blueboy II	0.088	0.127	0.5	2.8
Wheat, Coker 47-27	0.064	0.107	2.2	8.4
Wheat, Holly	0.099	0.127	0.0	0.9
Wheat, Oasis	0.093	0.135	0.4	2.4
Corn, PAG 397	0.095	0.126	0.3	1.5
Corn, Pioneer 3780	0.075	0.111	1.4	5.1
Corn, Coker 16	0.133	0.175	0.0	0.3
Sorghum, DeKalb-28	0.108	0.186	0.0	2.7
Barley, Poco	0.121	0.161	0.0	0.5
<u>Fiber crops</u>				
Cotton, Acala SJ-2 (81)	0.044	0.096	8.3	16.2
Cotton, Acala SJ-2 (82)	0.032	0.055	16.1	35.1
Cotton, Stoneville	0.047	0.075	4.6	16.2
<u>Horticultural crops</u>				
Tomato, Murrieta (81)	0.079	0.108	0.8	3.7
Tomato, Murrieta (82)	0.040	0.059	10.3	31.2
Lettuce, Empire	0.053	0.075	0.0	16.8
Spinach, America	0.046	0.082	6.8	17.2
Spinach, Hybrid	0.043	0.082	2.6	9.2
Spinach, Viroflay	0.048	0.080	6.0	16.7
Spinach, Winter Bloom	0.049	0.080	5.8	16.5
Turnip, Just Right	0.043	0.064	7.7	24.9
Turnip, Pur Top W. G.	0.040	0.064	10.1	26.5
Turnip, Shogoin	0.036	0.060	13.0	29.7
Turnip, Tokyo Cross	0.053	0.072	3.3	15.6

^aThe yield losses are derived from Weibull equations and are based on the control yields in charcoal-filtered air.

Source: Derived from Heck et al. (1984).

cause a 10 percent yield loss in Vona wheat, kidney bean, and Hodgson soybean. At a 7-hr seasonal mean O_3 concentration of 0.04 ppm, mean yield reductions ranged from zero percent in sorghum, barley, and a corn cultivar to a high of 28.8 percent in Vona wheat.

A histogram of the 7-hr seasonal mean O_3 concentrations predicted to cause a 10 percent yield loss (Table 1-5) is given in Figure 1-6 to help illustrate the range of concentrations and their relative frequency of occurrence. The data in Figure 1-6 are based on 37 species or cultivar yield-response functions developed from studies in open-top field exposure chambers. Approximately 57 percent of the species or cultivars were predicted to exhibit 10 percent yield reductions at 7-hr seasonal mean concentrations below 0.05 ppm. Thirty-five percent of plant types were predicted to display a 10 percent yield loss at 7-hr mean concentrations between 0.04 and 0.05 ppm. Seven-hr seasonal mean concentrations in excess of 0.08 ppm were required to cause a 10 percent yield loss in almost 19 percent of the species or cultivars. The data indicate that approximately 11 percent of the species or cultivars would display a 10 percent loss at 7-hr seasonal mean concentrations below 0.035 ppm, suggesting that these plant types are very sensitive to O_3 -induced yield losses.

A review of the data in Table 1-5 indicates that the grain crops were apparently generally less sensitive than the other crops to O_3 . Mean yield reductions at 0.04 ppm were predicted to be less than 5 percent for all the species and cultivars tested except for the Roland and Vona wheat cultivars. The data also demonstrate that sensitivity differences within a species may be as large as differences between species. For example, at 0.04 ppm O_3 , estimated yield losses ranged from 2 to 15 percent in soybean and from 0 to 28 percent in wheat. In addition to differences in sensitivity among species and cultivars, the data in Figures 1-4 and 1-5 illustrate year-to-year variations in plant response to O_3 .

Several exposure-response models, ranging from simple linear to complex nonlinear models, have been used to describe the relationship between plant yield and O_3 exposure. When exposure-response models are used, it is important for the fitted equations not to show systematic deviation from the data points and for the coefficient of determination (R^2) to be high. Although linear regression equations have been used to estimate yield loss, there appear to be systematic deviations from the data for some species and cultivars even though

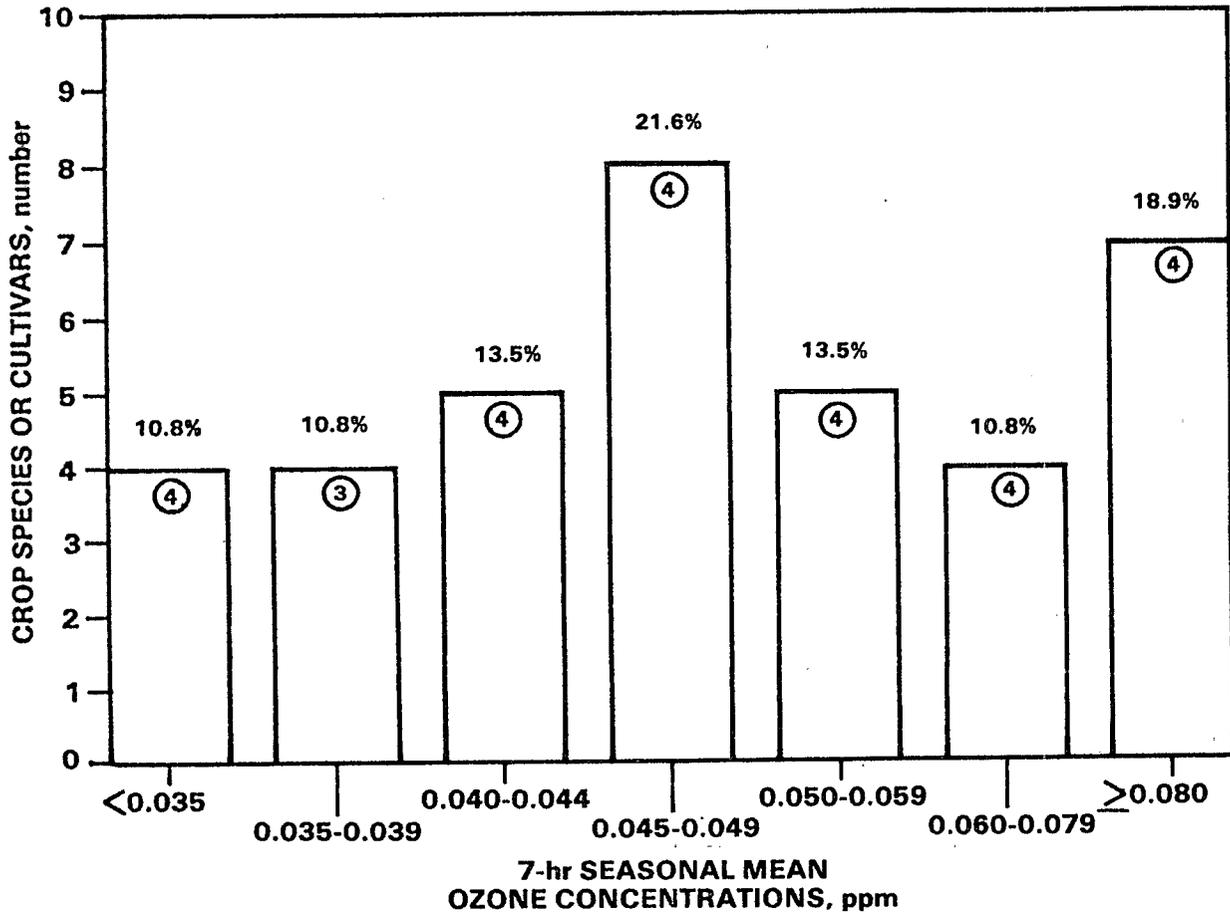


Figure 1-6. Number and percentage of 37 crop species or cultivars predicted to show a 10 percent yield loss at various ranges of 7-hr seasonal mean ozone concentrations. Concentration ranges and 10% yield loss data are derived from Table 1-5. Data represent 12 separate crop species; circled numbers represent separate species for each concentration range.

the equations have moderate-to-high coefficients of determination (R^2). Plateau-linear or polynomial equations appear to fit the data better. More recently, a Weibull model has been used to estimate percentage yield loss (Heck et al., 1983). The Weibull model yields a curvilinear response line that seems to provide a reasonable fit to the data. Based on available data, it is recommended that curvilinear exposure-response functions be used to describe and analyze plant response to O_3 .

1.5.3.2 Yield Loss: Determination from Discrete Treatments. In addition to the use of regression approaches in some studies, various other approaches have been used to investigate the effects of O_3 on crop yield. These studies were designed to test whether specific O_3 treatments were different from the control rather than to develop exposure-response equations. In general, these data were analyzed using analysis of variance. To summarize the data from studies that used discrete treatments, the lowest O_3 concentration that significantly reduced yield was determined from analyses done by the authors (Table 1-6). The lowest concentration reported to reduce yield was frequently the lowest concentration used in the study; hence it was not always possible to estimate a no-effect exposure concentration. In general, the data indicate that O_3 concentrations of 0.10 ppm (frequently the lowest concentration used in the studies) for a few hours per day for several days to several weeks generally caused significant yield reductions. Although it appears from this analysis that a higher O_3 concentration was required to cause an effect than was estimated from the regression studies, it should be noted that the concentrations derived from the regression studies were based on a 10 percent yield loss, while in studies using analysis of variance (Table 1-6) the 0.10 ppm concentration frequently induced mean yield losses of 10 to 50 percent.

1.5.3.3 Yield Loss: Determination with Chemical Protectants. Chemical protectants (antioxidants) have been used to estimate the impact of ambient O_3 on crop yield. In these studies, some plots were treated with the chemical and others were not. Yield loss was determined by comparing the yield in the plots treated with the chemical to the yield in untreated plots. When chemical protectants are used, care must be used in interpreting the data because the chemical itself may alter plant growth. The chemical may not be effective against all concentrations of all pollutants in the study area, which would result in an underestimation of yield loss. With an understanding of these limitations, however, researchers have concluded that chemical protectants are

TABLE 1-6. OZONE CONCENTRATIONS AT WHICH SIGNIFICANT YIELD LOSSES HAVE BEEN NOTED FOR A VARIETY OF PLANT SPECIES EXPOSED UNDER VARIOUS EXPERIMENTAL CONDITIONS

Plant species	Exposure duration	Yield reduction, % of control	O ₃ concentration, ppm	Reference
Alfalfa	7 hr/day, 70 days	51, top dry wt	0.10	Neely et al. (1977)
Alfalfa	2 hr/day, 21 day	16, top dry wt	0.10	Hoffman et al. (1975)
Pasture grass	4 hr/day, 5 days/wk, 5 wk	20, top dry wt	0.09	Horsman et al. (1980)
Ladino clover	6 hr/day, 5 days	20, shoot dry wt	0.10	Blum et al. (1982)
Soybean	6 hr/day, 133 days	55, seed wt/plant	0.10	Heagle et al. (1974)
Sweet corn	6 hr/day, 64 days	45, seed wt/plant	0.10	Heagle et al. (1972)
Sweet corn	3 hr/day, 3 days/wk, 8 wk	13, ear fresh wt	0.20	Oshima (1973)
Wheat	4 hr/day, 7 day	30, seed yield	0.20	Shannon and Mulchi (1974)
Radish	3 hr	33, root dry wt	0.25	Adedipe and Ormrod (1974)
Beet	2 hr/day, 38 days	40, storage root dry wt	0.20	Ogata and Maas (1973)
Potato	3 hr/day, every 2 wk, 120 days	25, tuber wt	0.20	Pell et al. (1980)
Pepper	3 hr/day, 3 days/wk, 11 wk	19, fruit dry wt	0.12	Bennett et al. (1979)
Cotton	6 hr/day, 2 days/wk, 13 wk	62, fiber dry wt	0.25	Oshima et al. (1979)
Carnation	24 hr/day, 12 days	74, no. of flower buds	0.05-0.09	Feder and Campbell (1968)
Coleus	2 hr	20, flower no.	0.20	Adedipe et al. (1972)
Begonia	4 hr/day, once every 6 days for a total of 4 times	55, flower wt	0.25	Reinert and Nelson (1979)
Ponderosa pine	6 hr/day, 126 days	21, stem dry wt	0.10	Wilhour and Neely (1977)
Western white pine	6 hr/days, 126 days	9, stem dry wt	0.10	Wilhour and Neely (1977)
Loblolly pine	6 hr/day, 28 days	18, height growth	0.05	Wilhour and Neely (1977)
Pitch pine	6 hr/day, 28 days	13, height growth	0.10	Wilhour and Neely (1977)
Poplar	12 hr/day, 5 mo	+1333, leaf abscission	0.041	Wilhour and Neely (1977)
Hybrid poplar	12 hr/day, 102 days	58, height growth	0.15	Patton (1981)
Hybrid poplar	8 hr/day, 5 day/wk, 6 wk	50, shoot dry wt	0.15	Patton (1981)
Red maple	8 hr/day, 6 wk	37, height growth	0.25	Dochinger and Townsend (1979)
American sycamore	6 hr/day, 28 days	9, height growth	0.05	Kress and Skelly (1982)
Sweetgum	6 hr/day, 28 days	29, height growth	0.10	Kress and Skelly (1982)
White ash	6 hr/day, 28 days	17, total dry wt	0.15	Kress and Skelly (1982)
Green ash	6 hr/day, 28 days	24, height growth	0.10	Kress and Skelly (1982)
Willow oak	6 hr/day, 28 days	19, height growth	0.15	Kress and Skelly (1982)
Sugar maple	6 hr/day, 28 days	12, height growth	0.15	Kress and Skelly (1982)

an objective method of assessing the effects of O_3 on crop yield, especially in conjunction with other methods. Results of several studies with chemical protectants showed decreased crop yield from exposure to ambient oxidants (Table 1-7). Crop yields were reduced 18 to 41 percent when the ambient oxidant concentration exceeded 0.08 ppm for 5 to 18 days over the growing season of the crop.

1.5.3.4 Yield Loss: Determination from Ambient Exposures. A number of research studies have demonstrated that ambient O_3 concentrations in a number of locations in the United States are sufficiently high to impair plant yield. Of studies to determine the impact of ambient oxidants (primarily O_3) on plant yield, most have compared the yield differences between plants grown in ambient air and those grown in charcoal-filtered air. Early research documented that ambient oxidants reduced the yield and quality of citrus, grape, tobacco, cotton, and potato (U.S. Environmental Protection Agency, 1978). Subsequent studies substantiated the impacts of ambient oxidants on plant yield (Table 1-8). Over several years, bean yields varied from a 5 percent increase to a 22 percent decrease in response to O_3 concentrations in excess of 0.06 ppm (Heggstad and Bennett, 1981).

Studies conducted on eastern white pine in the southern Appalachian mountains showed that ambient O_3 may have reduced the radial growth of sensitive individuals as much as 30 to 50 percent annually over the last 15 to 20 years (Mann et al., 1980). Field studies in the San Bernardino National Forest showed that during the last 30 years ambient O_3 may have reduced height growth of ponderosa pine by as much as 25 percent, radial growth by 37 percent, and the total wood volume produced by 84 percent (Miller et al., 1982). Calculations of biomass in these studies were based, however, on apparent reductions in radial growth without standardization of radial growth data with respect to tree age.

1.5.3.5 Yield Loss Summary. Several general conclusions can be drawn from the various approaches used to estimate crop yield loss. The data from the comparisons of crop yield in charcoal-filtered and unfiltered air (ambient exposures) clearly show that ambient levels of O_3 are sufficiently elevated in several parts of the country to impair the growth and yield of plants. The data from the chemical protectant studies support and extend this conclusion to other plant species. Both approaches indicate that the effects occur at low mean concentrations, with only a few O_3 occurrences greater than 0.08 ppm.

TABLE 1-7. EFFECTS OF OZONE ON CROP YIELD AS DETERMINED BY THE USE OF CHEMICAL PROTECTANTS^a

Species	Yield reductign, % of control	O ₃ exposure, ppm	Reference
Beans (green)	41	>0.08 for total of 27 hr over 3.5 months	Manning et al. (1974)
Onion	38	>0.08 on 5 days out of 48	Wukasch and Hofstra (1977b)
Tomato	30	>0.08 on 15 days over 3 months	Legassicke and Ormrod (1981)
Bean (dry)	24	>0.08 on 11 days (total of 34 hr) over 3 months	Temple and Bisessar (1979)
Tobacco	18	>0.08 on 14 days during the summer	Bisessar and Palmer (1984)
Potato	36	>0.08 ppm on 18 days (total of 68 hr) over 3 months	Bisessar (1982)
Potato	25	- ^c	Clarke et al. (1983)

^aAll the species were treated with the antioxidant, EDU, except the bean study by Manning et al. (1974) which used the systemic fungicide, benomyl.

^bYield reduction was determined by comparing the yields of plants treated with chemical protectants (control) to those that were not treated.

^cThis study was run over 2 years when the O₃ doses were 65 and 110 ppm-hr, respectively, but the yield loss was similar both years.

TABLE 1-8. EFFECTS OF AMBIENT OXIDANTS ON YIELD OF SELECTED CROPS

Plant species	O ₃ concentration, ppm	Exposure duration	Yield, % reduction from control	Location of study	Reference
Tomato (Fireball 861 VR)	0.035 (0.017-0.072)	99 day average (6:00 a.m. - 9:00 p.m.)	33, fruit fresh wt	New York	MacLean and Schneider (1976)
Bean (Tendergreen)	0.041 (0.017-0.090)	43 day average (6:00 a.m. - 9:00 p.m.)	26, pod fresh wt		
Snap bean (3 cultivars: Astro, BBL 274, BBL 290)	0.042	3 mo average (9:00 a.m. - 8:00 p.m.)	1, pod wt	Maryland	Heggestad and Bennett (1981)
Soybean (4 cultivars: Cutler, York, Clark, Dare)	>0.05	31% of hr between 8:00 a.m. - 10:00 p.m. from late June to mid-September over three summers; 5% of the time the concentration was >0.08 ppm	20, seed wt	Maryland	Howell et al. (1979); Howell and Rose (1980)
Forbs, grasses, sedges	0.052	1979, 8 hr/day average (10:00 a.m. - 6:00 p.m.), April-September	32, total above-ground biomass	Virginia	Duchelle et al. (1983)
	0.051	1980, 8 hr/day average (10:00 a.m. - 6:00 p.m.), April-September	20, total above-ground biomass	Virginia	
	0.035	1981, 8 hr/day average (10:00 a.m. - 6:00 p.m.), April-September	21, total above-ground biomass		
Sweet corn (Bonanza)	>0.08	58% of hr (6:00 a.m. - 9:00 p.m.), 1 July-6 September	9, ear fresh wt	California	Thompson et al. (1976a)
(Monarch Advance)	>0.08		28, ear fresh wt		

Growth and yield data from the previous criteria document (U.S. Environmental Protection Agency, 1978), shown in Figure 1-3, indicate that effects on growth and yield of several plant species occurred when the mean O_3 concentration (for 4 to 6 hr/day) exceeded 0.05 ppm for at least 2 wk. The data from the regression studies, conducted to develop exposure-response functions for estimating yield loss, indicated that at least 50 percent of the species/cultivars tested were predicted to display a 10 percent yield loss at 7-hr seasonal mean O_3 concentrations of 0.05 ppm or less. Most of the data from the discrete treatment studies did not use levels low enough to support these values directly. The magnitude of yield losses reported at 0.10 ppm, however, indicate that maintenance of a substantially lower concentration than 0.10 ppm is needed to prevent O_3 effects, although a specific value cannot be derived from the discrete treatment studies.

1.5.4 Effects on Crop Quality

Based on results of the few studies that have been conducted, O_3 can reduce crop quality in addition to reducing the total yield of the crop. Quality is a general term that includes many features of the crop, such as nutritional composition, appearance, taste, and ability to withstand storage and shipment. Examples of O_3 -induced alterations in quality are decreased oil in soybean seeds (Howell and Rose, 1980; Kress and Miller, 1983); decreased β -carotene, vitamin C, and carbohydrates in alfalfa (Thompson et al., 1976b; Neely et al., 1977); and increased reducing sugars that are associated with undesirable darkening when potatoes are used to make potato chips (Pell et al., 1980).

1.5.5 Statistics Used to Characterize Ozone Exposures

The characterization and representation of plant exposures to O_3 has been, and continues to be a major problem. Research has not yet clearly identified which components of the pollutant exposure cause the plant response. Most studies have characterized the exposure by the use of mean O_3 concentrations, although various averaging times have been used. Some studies have also used cumulative O_3 dose. The difficulty of selecting an appropriate statistic to characterize plant exposure has been summarized by Heagle and Heck (1980). Ambient and experimental O_3 exposures have been presented as

seasonal, monthly, weekly, or daily means; peak hourly means; number of hours above a selected concentration; or the number of hours above selected concentration intervals. None of these statistics adequately characterize the relationships among O_3 concentration, exposure duration, interval between exposures, and plant response. The use of a mean concentration (with long averaging times) (1) implies that all concentrations of O_3 are equally effective in causing plant responses and (2) minimizes the contributions of the peak concentrations to the response. The mean treats low-level, long-term exposures the same as high-concentration, short-term ones. Thus, the use of a long-term mean concentration ignores the importance of peak concentrations; to ignore the peaks is inconsistent with the literature.

The total ozone dose (concentration multiplied by time) has been used to describe plant exposure; however, it suffers from the same problem as the mean. The total dose is simply the summation of the ppm-hr over the study period, which also treats all concentrations as being equally effective. Several investigators have attempted to give greater importance to peak O_3 concentrations. For example, Oshima et al. (1977a,b) and Lefohn and Benedict (1982) have summed only the ppm-hr of exposure greater than some preselected value. Larsen et al. (1983) have introduced the concept of "impact" to describe the effects of O_3 and SO_2 on soybeans. The "impact (I)" is calculated similarly to total dose, except the concentration is raised to an exponent greater than one ($I = C^W \times T$); this method of calculation effectively gives greater weight to the higher concentrations. More recently, Larsen and Heck (1984) have suggested the term "effective mean" to describe an approach in which greater importance is given to higher concentrations. The "effective mean" is defined as the average hourly impact raised to an exponent and divided by the duration.

Several lines of evidence suggest that higher concentrations should be regarded as having the greater influence in determining the impact of O_3 on vegetation. Studies have shown that plants can tolerate some combinations of exposure duration and concentration without exhibiting foliar injury or effects on growth or yield, illustrating that not all concentrations are equally effective in causing a response. From the toxicological perspective, it is the peaks or concentrations above some level that are most likely to have an impact. Effects occur on vegetation when the amount of pollutant that the plant has absorbed exceeds the ability of the organism to repair or compensate for the impact.

Studies with beans and tobacco (Heck et al., 1966) showed that a dose (concentration times time) distributed over a short period induced more injury than did the same dose distributed over a longer period. Tobacco studies showed that the O_3 concentration was substantially more important than exposure duration in causing foliar injury (Tonneijck, 1984). In beans, foliar injury occurred when the internal O_3 flux exceeded $115 \mu\text{moles}/\text{m}^2$ in 1 hr (Bennett, 1979). A single 3-hr exposure, however, at approximately half the concentration (0.27 compared with 0.49 ppm) required a 64 percent greater internal flux of O_3 to produce the same amount of foliar injury as the 1-hr exposure required. More recently, Amiro et al. (1984) showed that higher concentrations were more important than low concentrations in causing injury. Their study also suggested the existence of a biochemical injury threshold (i.e., the O_3 uptake rates that plants can experience without incurring visible foliar injury). The greater importance of concentration compared to exposure duration has also been reported by other authors (e.g., Heck and Tingey, 1971; Henderson and Reinert, 1979; Reinert and Nelson, 1979).

Studies with soybean (Johnston and Heagle, 1982), tobacco (Heagle and Heck, 1974), and bean (Runeckles and Rosen, 1977) showed that plants exposed to a low level of O_3 for a few days became more sensitive to subsequent O_3 exposures. In studies with tobacco, Mukammal (1965) showed that a high O_3 concentration on one day caused substantial injury, whereas an equal or higher concentration on the second day caused only slight injury. Using stress ethylene as an indicator of O_3 effects, Stan and Schicker (1982) showed that a series of successive short exposures was more injurious to plants than a continuous exposure at the same O_3 concentration for the same total exposure period. Walmsley et al. (1980) continuously exposed radishes to O_3 for several weeks and found that the plants acquired some O_3 tolerance. The acquired tolerance displayed two components: (1) the exposed plants developed new leaves faster than the controls, and (2) there was a progressive decrease in sensitivity of the new leaves to O_3 . The newer leaves also displayed a slower rate of senescence. The observations by Elkley and Ormrod (1981) that the O_3 uptake decreased during a 3-day study period may provide an explanation for the results with radish.

Not only are concentration and time important but the dynamic nature of the O_3 exposure is also important; i.e. whether the exposure is at a constant or variable concentration. Musselman et al. (1983) recently showed that constant concentrations of O_3 caused the same types of plant responses as

variable concentrations at equivalent doses. Constant concentrations, however, had less effect on plant growth responses than variable concentrations at similar doses. Exposures of radishes to ambient O_3 in open-top exposure chambers showed that significant yield reductions occurred when the maximum O_3 concentration exceeded 0.06 ppm at least 10 percent of the days when the crop was growing (Ashmore, 1984). Initial studies have compared the response of alfalfa to daily peak and episodic O_3 exposure profiles that gave the equivalent total O_3 dose over the growing season (Hogsett et al., 1985). Alfalfa yield was reduced to a greater extent in the episodic than in the daily peak exposure. This study also illustrates the problem with the 7-hr seasonal mean concentration; i.e., it does not properly account for the peak concentrations. The plants that displayed the greater growth reduction (in the episodic exposure) were exposed to a significantly lower 7-hr seasonal mean concentration. Studies with SO_2 also showed that plants exposed to variable concentrations exhibited a greater plant response than those exposed to a constant concentration (McLaughlin et al., 1979; Male et al., 1983).

1.5.6 Relationship Between Yield Loss and Foliar Injury

Because plant growth and production depend on photosynthetically functional leaves, various studies have been conducted to determine the association between foliar injury and yield for species in which the foliage is not part of the yield. Some research has demonstrated significant yield loss with little or no foliar injury (e.g., Tingey et al., 1971; Tingey and Reinert, 1975; Kress and Skelly, 1982; Feder and Campbell, 1968; Adedipe et al., 1972). Other studies showed that significant foliar injury was not always associated with yield loss (Heagle et al., 1974; Oshima et al., 1975). The relative sensitivities of two potato cultivars were reversed when judged by foliar injury versus yield reductions (Pell et al., 1980). In field corn, foliar injury occurred at a lower O_3 concentration than yield reductions; but as the O_3 concentration increased, yield was reduced to a greater extent than foliar injury was increased (Heagle et al., 1979a). In wheat, foliar injury was not a good predictor of O_3 -induced yield reductions (Heagle et al., 1979b).

1.5.7 Physiological Basis of Yield Reductions

As discussed earlier in this summary, plant growth is the summation of a series of biochemical and physiological processes related to uptake, assimilation, biosynthesis, and translocation. An impairment in these processes may lead to reduced plant yield if the process is limiting.

For plant growth to occur, plants must assimilate CO_2 and convert it into organic substances; an inhibition in carbon assimilation may be reflected in plant growth or yield. In several species O_3 (at 0.05 ppm and higher) inhibited photosynthesis, as measured by gas-exchange (e.g., U.S. Environmental Protection Agency, 1978; Coyne and Bingham, 1978; Black et al., 1982; Bennett and Hill, 1974; Yang et al., 1983). Biochemical studies showed that O_3 (0.12 ppm for 2 hr) inhibited an enzyme that catalyzes the assimilation of CO_2 (Pell and Pearson, 1983).

Ozone, in addition to decreasing the total amount of CO_2 that is assimilated, alters that pattern by which the reduced amount of assimilate is partitioned throughout the plant. There is generally less photosynthate translocated to the roots and to the reproductive organs (e.g., Tingey et al., 1971; Jacobson, 1982; Oshima et al., 1978, 1979; Bennett et al., 1979). This reduces root size and marketable yield as well as rendering the plant more sensitive to injury from environmental stresses. Another consequence of reduced root growth and altered carbon allocation is an impairment of symbiotic nitrogen fixation (U.S. Environmental Protection Agency, 1978; Ensing and Hofstra, 1982).

The reproductive capacity (flowering and seed set) is reduced by O_3 in ornamental plants, soybean, corn, wheat, and other plants (Adedipe et al., 1972; Feder and Campbell, 1968; Heagle et al., 1972, 1974; Shannon and Mulchi, 1974). These data suggest that O_3 impairs the fertilization process in plants. This suggestion has been confirmed in tobacco and corn studies using low concentrations of O_3 (0.05 to 0.10 ppm) for a few hours (Feder, 1968; Mumford et al., 1972).

Ozone both in the field and in chamber studies stimulates premature senescence and leaf drop (Menser and Street, 1962; Heagle et al., 1974; Heggstad, 1973; Pell et al., 1980; Hofstra et al., 1978). In part, the O_3 -induced yield reduction has been attributed to premature senescence. The premature leaf drop decreases the amount of photosynthate that a leaf can contribute to plant growth.

1.5.8 Factors Affecting Plant Response to Ozone

Numerous factors influence the type and magnitude of plant response to O_3 . Most studies of the factors influencing plant response have been limited to effects on foliar injury; however, some studies have measured yield and

some have researched the physiological basis for the influences. The parameters studied include environmental factors, biological factors, and interactions with other air pollutants.

1.5.8.1 Environmental Conditions. Environmental conditions before and during plant exposure are more influential than post-exposure conditions in determining the magnitude of the plant response. The influence of environmental factors has been studied primarily under controlled conditions, but field observations have substantiated the results. Most studies have evaluated the influence of only a single environmental factor and have relied primarily upon foliar injury as the plant response measure. Some generalizations of the influence of environmental factors can be made:

1. Light conditions that are conducive to stomatal opening appear to enhance O_3 injury (U.S. Environmental Protection Agency, 1978). Light is required to induce stomatal opening, which permits the plant to absorb pollutants.
2. No consistent pattern relating plant response to temperature has been observed (U.S. Environmental Protection Agency, 1978). Plants do not appear to be as sensitive at extremely high or low temperatures, however, as they are under more moderate conditions.
3. Plant injury tends to increase with increasing relative humidity (U.S. Environmental Protection Agency, 1978). The relative humidity effect appears to be related to stomatal aperture, which tends to increase with increasing relative humidity. McLaughlin and Taylor (1981) demonstrated that plants absorb significantly more O_3 at high humidity than at low humidity. It is generally accepted that plants in the eastern United States are injured by lower concentrations of O_3 than their counterparts in California; this phenomenon has been attributed to differences in humidity (U.S. Environmental Protection Agency, 1978).
4. As soil moisture decreases, plant water stress increases and there is a reduction in plant sensitivity to O_3 (U.S. Environmental Protection Agency, 1978). The reduced O_3 sensitivity is apparently related to stomatal closure, which reduces O_3 uptake (U.S. Environmental Protection Agency, 1978; Olszyk and Tibbitts, 1981; Tingey et al., 1982). Water stress does not confer a permanent tolerance to O_3 ;

once the water stress has been alleviated, the plants regain their sensitivity to O_3 (Tingey et al., 1982).

1.5.8.2 Interaction with Plant Diseases. Ozone can affect the development of disease in plant populations. Laboratory evidence suggests that O_3 (at ambient concentrations or greater for 4 hr or more) inhibits infection by pathogens and subsequent disease development (Laurence, 1981; Heagle, 1982; U.S. Environmental Protection Agency, 1978). Increases, however, in diseases from "stress pathogens" have been noted. For example, plants exposed to O_3 were more readily injured by Botrytis than plants not exposed to O_3 (Manning et al., 1970a,b; Wukasz and Hofstra, 1977a,b; Bisessar, 1982). Both field and laboratory studies have confirmed that the roots and cut stumps of O_3 -injured ponderosa and Jeffrey pines are more readily colonized by a root rot (Heterobasidion annosus). The degree of infection was correlated with the foliar injury (James et al., 1980; Miller et al., 1982). Studies in the San Bernardino National Forest showed that O_3 -injured trees were predisposed to attack by bark beetles and that fewer bark beetles were required to kill an O_3 -injured tree (Miller et al., 1982).

1.5.8.3 Interaction of Ozone with Other Air Pollutants. The report of Menser and Heggstad (1966) provided the initial impetus for studying the interaction of O_3 with SO_2 . They showed that Bel W-3 tobacco plants exposed to O_3 (0.03 ppm) or SO_2 (0.24 to 0.28 ppm) were uninjured but that substantial foliar injury resulted when the plants were exposed to both gases simultaneously. Subsequent studies have confirmed and extended the observation that combinations of O_3 and SO_2 may cause more visible injury than expected based on the injury from the individual gases. This injury enhancement (synergism) is most common at low concentrations of each gas and also when the amount of foliar injury induced by each gas, individually, is small. At higher concentrations or when extensive injury occurs, the effects of the individual gases tend to be less than additive (antagonistic). In addition to foliar injury, the effects of pollutant combinations have also been investigated in relation to other plant effects, and these have been discussed in several reviews and numerous individual reports (e.g., Reinert et al., 1975; Ormrod, 1982; Jacobson and Colavito, 1976; Heagle and Johnston, 1979; Olszyk and Tibbitts, 1981; Flagler and Youngner, 1982; Foster et al., 1983; Heggstad and Bennett, 1981; Heagle et al., 1983a).

Field studies have investigated the influence of SO_2 on plant response to O_3 at ambient and higher concentrations in several plant species: soybean (Heagle et al., 1983b; Reich and Amundson, 1984), beans (Oshima, 1978; Heggstad and Bennett, 1981), and potatoes (Foster et al., 1983). In these studies, O_3 altered plant yield but SO_2 had no significant effect and did not interact with O_3 to reduce plant yield unless the SO_2 exposure concentrations and frequency of occurrence were much greater than the concentrations and frequencies of occurrence typically found in the ambient air in the United States.

The applicability of the yield results from pollutant combination studies to ambient conditions is not known. An analysis of ambient air monitoring data for instances of co-occurrence of O_3 and SO_2 indicated that at sites where the two pollutants were monitored, they both were present for ten or fewer periods during the growing season (Lefohn and Tingey, 1984). Co-occurrence was defined as the simultaneous occurrence of hourly averaged concentrations of 0.05 ppm or greater for both pollutants. At this time, it appears that most of the studies of the effects on pollutant combinations (O_3 and SO_2) on plant yield have used a longer exposure duration and a higher frequency of pollutant co-occurrence than are found in the ambient air.

Only a few studies have investigated the effects of O_3 when combined with pollutants other than SO_2 , and no clear trend is available. Preliminary studies using three-pollutant mixtures (O_3 , SO_2 , NO_2) showed that the additions of SO_2 and NO_2 (at low concentrations) caused a greater growth reduction than O_3 alone.

1.5.9 Economic Assessment of Effects of Ozone on Agriculture

Evidence from the plant science literature clearly demonstrates that O_3 at ambient levels will reduce yields of some crops (see Chapter 6, Section 6.4.3.2.2). In view of the importance of U.S. agriculture to both domestic and world consumption of food and fiber, such reductions in crop yields could adversely affect human welfare. The plausibility of this premise has resulted in numerous attempts to assess, in monetary terms, the losses from ambient O_3 or the benefits of O_3 control to agriculture. Many of these assessments have been performed since publication of the 1978 O_3 criteria document (U.S. Environmental Protection Agency, 1978). The utility of these post-1978 studies in regulatory decision-making can be evaluated in terms of how well the requisite biological, aerometric, and economic inputs conform to specific criteria, as discussed in Section 6.5 of Chapter 6.

While a complete discussion of the criteria for evaluating economic assessments is not appropriate here, it is instructive to highlight certain key issues. First, the evidence on crop response to O_3 should reflect how crop yields will respond under actual field conditions. Second, the air quality data used to frame current or hypothetical effects of O_3 on crops should represent the actual exposures sustained by crops in each production area. Finally, the assessment methodology into which such data are entered should (1) capture the economic behavior of producers and consumers as they adjust to changes in crop yields and prices that may accompany changes in O_3 air quality; and (2) ideally, should accurately reflect institutional considerations, such as regulatory programs, that may result in market distortions.

The assessments of O_3 damages to agriculture found in the literature display a range of procedures for calculating economic losses, from simple monetary calculation procedures to more complex economic assessment methodologies. The simple procedures calculate monetary effects by multiplying predicted yield or production changes resulting from exposure to O_3 by an assumed constant crop price, thus failing to recognize possible crop price changes arising from yield changes as well as not accounting for the processes underlying economic response. Conversely, a rigorous economic assessment will provide estimates of the benefits of air pollution control that account for producer-consumer decision-making processes, associated market adjustments, and perhaps some measure of distributional consequences between affected parties. It is important to distinguish between those studies based on naive or simple models and those based on correct procedures, since the naive procedure may be badly biased, leading to potentially incorrect policy decisions.

Most of the post-1978 economic assessments focus on O_3 effects in specific regions, primarily California and the Corn Belt (Illinois, Indiana, Iowa, Ohio, and Missouri). This regional emphasis may be attributed to the relative abundance of data on crop response and air quality for selected regions, as well as the national importance of these agricultural regions. Economic estimates for selected regions are presented in Table 1-9. In addition to reporting the monetary loss or benefit estimates derived from each assessment, this table provides some evaluation of the adequacy of the plant science, aerometric, and economic data, and assumptions used in each assessment. Adequacy as defined here does not mean that the estimates are free of error; rather, it implies that the estimates are based on the most defensible biologic,

TABLE 1-9. SUMMARY OF ESTIMATES OF REGIONAL ECONOMIC CONSEQUENCES OF OZONE POLLUTION

Reference and study region	Crops	Annual benefits of control, \$ million		Evaluation of critical data and assumptions ^a		Additional comments
		Plant response data	Aerometric data	Economic model data	Economic model data	
Adams et al. (1982); Southern California	12 annual crops: beans, broccoli, cantaloupes, carrots, cauliflower, celery, lettuce, onions, potatoes, tomatoes, cotton, and sugar beets.	Inadequate; uses Larsen-Heck (1976) foliar injury models converted to yield losses.	Adequate; exposure measured as cumulative seasonal exposure in excess of California standard (0.08 ppm), from hourly data collected for sites closest to production regions.	Adequate; a price endogenous mathematical (quadratic) programming model reflecting agronomic, environmental, and economic conditions in 1976.	Economic effect measured as a change in economic surplus (sum of consumers and producers' surpluses) between base case (actual O ₃ levels in 1976) and economic surplus that would be realized if all regions were in compliance with 1971 photochemical oxidant standard of 0.08 ppm.	
Lueng et al. (1982); Southern California	9 crops: lemons, oranges (Valencia and Navel), strawberry, tomato, alfalfa, avocado, lettuce, and celery.	Inadequate; O ₃ -yield response functions estimated from secondary data on crop yields.	Adequate for some regions; exposure measured in average monthly concentration in ppm for 12 hr period (7:00 a.m. to 7:00 p.m.). Data from 61 California Air Resources Board monitoring sites.	Adequate on demand side; economic model is composed of linear supply and demand curves for each crop estimated with data from 1958-1977, but ignores producer-level adjustments.	Economic effect is measured as a change in economic surplus between base case (1975) and a clean air environment reflecting zero O ₃ .	
Howitt et al. (1984a,b); California	13 crops: alfalfa, barley, beans, celery, corn, cotton, grain sorghum, lettuce, onions, potatoes, rice, tomatoes, and wheat.	Adequate for some crops; most response functions derived from NCLAN data through 1982. Surrogate responses used for celery, onions, rice and potatoes are questionable.	Adequate; California Air Resources Board data for monitoring sites closest to rural production areas. Exposure measured as the seasonal 7-hr average in each production area for compatibility with NCLAN exposure.	Adequate; economic model similar to Adams et al. (1982) but includes some perennial crops and reflects 1978 economic and technical environment.	Economic effects measured as changes in economic surplus across three O ₃ changes from 1978 actual levels. These include changes in ambient O ₃ to 0.04, 0.05, and 0.08 ppm across all regions.	

TABLE 1-9 (cont'd). SUMMARY OF ESTIMATES OF REGIONAL ECONOMIC CONSEQUENCES OF OZONE POLLUTION

Reference and study region	Crops	Evaluation of critical data and assumptions ^a			Additional comments
		Annual benefits of control, \$ million	Plant response data	Aerometric data	
Rowe et al. (1984); San Joaquin Valley in California	14 annual and perennial crops: alfalfa, barley, beans, carrots, corn, cotton, grain sorghum, grass hay, grapes, pasture, potatoes, safflower, tomatoes and wheat.	\$43 to \$117 depending on degree of control, measured in 1978 dollars.	Adequate for some crops; response functions based on both experimental and secondary data. Most crops from NCLAN data. Responses for the remaining crops were based on surrogate responses of similar crops in the data set.	Adequate; 4 exposure levels were tested. The average hourly concentration was used in most functions to predict changes. All data were from California Air Resources Board monitoring sites in predominantly rural areas.	Economic effects measured as the change in economic surplus between the 1978 base case and three increasingly stringent control scenarios: (1) a 50% reduction in no. of hr > 0.10 ppm; (2) meeting the current standard of 0.10 ppm; and (3) meeting an O ₃ standard of 0.08 ppm.
Adams and McCarl (1985); Corn Belt	3 crops: corn, soybeans, and wheat.	\$668 (in 1980 dollars)	Adequate; O ₃ yield response information from NCLAN for 3 yr (1980-1982). Yield adjustments estimated from Weibull response models.	Adequate except for linkage of 7-hr seasonal mean to hourly standards. Data are interpolated from SAROAD monitoring sites by Kriging procedure, measured as 1980 seasonal 7-hr average. Regulatory analysis assumes that O ₃ is log-normally distributed.	Economic estimates represent changes in economic surplus (sum of consumers' and producers' surpluses) between current (1980) O ₃ levels and increases and decreases in ambient O ₃ levels. Reduction to a uniform ambient level of 0.04 ppm across all regions results in benefits of \$668 million.

TABLE 1-9 (cont'd). SUMMARY OF ESTIMATES OF REGIONAL ECONOMIC CONSEQUENCES OF OZONE POLLUTION

Reference and study region	Crops	Annual benefits of control, \$ million			Evaluation of critical data and assumptions ^a		Additional comments
		Plant response data	Aerometric data	Economic model data			
Mjelde et al. (1984); Illinois	3 crops: corn, soybeans, and wheat.	Ranges from \$55 to \$220 annually for period 1976 to 1980.	Adequate when cross-checked against NCLAN data; responses are estimated from secondary (non-experimental) data on actual farmer yield, input, and O ₃ concentrations. Results are translated into yield effects and compared to NCLAN data from Illinois.	Adequate; same Krige data set as used in Adams and McCarl (1985), except only for Illinois and cover 5 yr (1976-1980). Exposure is measured as seasonal 7-hr average to facilitate comparison with NCLAN response estimates.	Adequate at producers level; economic model consists of a series of annual relationships on farmers' profits (These functions are adjusted to represent changes in O ₃ ($\pm 25\%$) for each year. Model does not include consumer (demand) effects).	The estimates represent increases in farmers' profits that could arise for a 25% reduction in O ₃ for each year (1976-1980). Years with higher ambient levels have highest potential increase in profits for changes.	
Page et al. (1982); Ohio River Basin	3 crops: corn, soybeans and wheat.	\$7.022 measured as present value of producer losses for period 1976 to 2000. Annualized losses are approx. \$270 in 1976 dollars.	Inadequate; crop losses provided by Loucks and Armentano (1982); responses derived by synthesis of existing experimental data.	Inadequate; dose measured as cumulative seasonal exposure for a 7-hr period (9:30 a.m. to 4:30 p.m.) Monitoring sites at only 4 locations were used to characterize the regional exposure.	Inadequate; the economic model consists of regional supply curves for each crop. The predicted changes in production between "clean air" case and each scenario are used to shift crop supply curves. The analysis ignores price changes from shifts in supply.	Losses are measured as differences in producer surplus across the various scenarios. Since prices are assumed fixed (in real terms) over the period, no consumer effects are measured.	

TABLE 1-9 (cont'd). SUMMARY OF ESTIMATES OF REGIONAL ECONOMIC CONSEQUENCES OF OZONE POLLUTION

Reference and study region	Crops	Annual benefits of control, \$ million	Evaluation of critical data and assumptions ^a		Additional comments
			Plant response data	Aerometric data	
Benson et al. (1982); Minnesota	4 crops: alfalfa, wheat, corn, and potatoes. Cultivar believed to be limited to one per crop.	\$30.5 (measured in 1980 dollars)	Inadequate; but innovative crop loss models estimated using experimental yield-O ₃ data from other researchers. Crop loss modeling includes both chronic and episodic response and crop development stage as factors in yield response, by regressing yield on O ₃ exposures for various time windows, during the growing season.	Adequate; air quality data are for state of Minnesota for 1979 and 1980. Exposure measured several ways but generally as a daily exposure statistic reflecting either sum of hourly averages or the mean hourly average.	The economic effect measured in terms of short-run profit changes for Minnesota producers. If yields are assumed to change only in Minnesota then losses to Minnesota producers are \$30.5 million. If yields change in Minnesota and the rest of U.S., then producers gain \$67 million as a result of increases in crop prices.

^aAdequacy as defined here does not mean that the estimates are free of error; rather, it implies that the estimates are based on the most defensible biological, aerometric, or economic information and models currently available.

^bKriging is a spatial interpolation procedure that has been used to generate O₃ concentration data for rural areas in which no monitoring sites have been established. See Heck et al. (1983b).

aerometric, or economic information and models currently available in the literature. The estimates can then be ranked relative to the strength of these data and assumptions. Of the eight regional studies reviewed, most have adequate economic models, but only four are judged adequate across all input categories. Further, most regional studies abstract from the interdependencies that exist between regions, which limits their utility in evaluating secondary national ambient air quality standards (SNAAQs).

National-level studies can overcome this limitation of regional analyses by accounting for economic linkages between groups and regions. A proper accounting for these linkages, however, requires additional data and more complex models, and frequently poses more difficult analytical problems. Thus, detailed national assessments tend to be more costly to perform. As a result, there are fewer assessments of pollution effects at the national than at the regional level. Six national-level assessments performed since the last criteria document was published in 1978 are reported in Table 1-10. Of these, two used the simple "price times quantity" approach to quantify dollar effects. Four used more defensible economic approaches. As with Table 1-9, an evaluation of the adequacy of critical plant science, aerometric, and economic data is presented, along with the estimates of benefits or damages.

As is evident from the evaluation, most of the national studies reviewed here suffer from either plant science and aerometric data problems, incomplete economic models, or both. As a result of these limitations, decision-makers should be cautious in using these estimates to evaluate the efficiency of alternative SNAAQs. Two of the studies, however, are judged to be much more adequate in terms of the three critical areas of data inputs. Together, they provide reasonably comprehensive estimates of the economic consequences of changes in ambient air O_3 levels on agriculture.

In the first of these studies, Kopp et al. (1984) measured the national economic effects of changes in ambient air O_3 levels on the production of corn, soybeans, cotton, wheat, and peanuts. In addition to accounting for price effects on producers and consumers, the assessment methodology used is notable in that it placed emphasis on developing producer-level responses to O_3 -induced yield changes (from NCLAN data) in 200 production regions. The results of the Kopp et al. (1984) study indicated that a reduction in O_3 from 1978 regional ambient levels to a seasonal 7-hr average of approximately 0.04 ppm would result in a \$1.2 billion net benefit in 1978 dollars. Conversely,

TABLE 1-10. SUMMARY OF ESTIMATES OF NATIONAL ECONOMIC CONSEQUENCES OF OZONE POLLUTION

Study	Crops	Annual benefits of control, \$ billion	Evaluation of critical data and assumptions ^a		Additional comments
			Plant response data	Economic model data	
Ryan et al. (1981)	16 crops: alfalfa, beets, broccoli, corn cabbage, corn (sweet and field), hay, lima beans, oats, potatoes, sorghum, soybeans, spinach, tobacco, tomatoes, and wheat.	\$1.747 (in 1980 dollars).	Inadequate; yield-response information derived from a synthesis of 5 yield studies in the literature prior to 1980. Synthesized response functions estimated for both chronic and acute exposures for six crops. For the remaining 10 crops surrogates are used. Yield changes are based on reductions in O ₃ to meet 1980 Federal standard of 0.12 ppm in non-compliance counties.	Inadequate; naive economic model. Monetary impact calculated by multiplying changes in county production by crop price in 1980. Measures impact on producers only.	Dollar estimate is for the 531 counties exceeding the Federal standard of 0.12 ppm. This study is essentially an updated version of Benedict et al. (1971) reported in 1978 criteria document.
Shriner et al. (1982)	4 crops: corn, soybeans, wheat, and peanuts. Multiple cultivars of all crops but peanuts.	\$3.0 (in 1978 dollars).	Adequate; analysis uses NCLAN response data for 1980. Functions estimated in linear form. Yield changes reflect difference between 1978 ambient O ₃ levels of each county and assumed background of 0.025 ppm concentration.	Unknown; exposure may be measured as highest 7-hr average, rather than 7-hr NCLAN average. Rural ambient concentrations for 1978 estimated by Kriging ^b procedure applied to SAROAD data.	Dollar estimates are for all counties producing the four crops. As with Ryan et al. (1981), estimates are for producer level effects only.
Adams and Crocker (1984)	3 crops: corn, soybeans, and cotton. Two corn cultivars, three soybean, two cotton.	\$2.2 (in 1980 dollars).	Adequate; analysis uses NCLAN O ₃ -yield data for 1980 and 1981. Functions estimated in linear form. Yield changes measured between 1980 ambient levels and an assumed O ₃ concentration of 0.04 ppm across all production regions.	Adequate; 1980 ambient O ₃ levels estimated by Kriging of SAROAD monitoring sites, translated into a seasonal 7-hr average.	Economic estimate measured in terms of changes in consumer and producer surpluses associated with the change in O ₃ .

^a ^b

TABLE 1-10 (cont'd). SUMMARY OF ESTIMATES OF NATIONAL ECONOMIC CONSEQUENCES OF OZONE POLLUTION

Study	Crops	Annual benefits of control, \$ billion			Evaluation of critical data and assumptions ^a		Additional comments
		Plant response data	Aerometric data	Economic model data			
Adams et al. (1984a)	4 crops: corn, soybeans, wheat, and cotton. Two cultivars for corn and cotton, three for soybeans and wheat.	Adequate; analysis uses NCLAN O ₃ -yield data for 1980 through 1982. Yield changes measured between 1980 ambient levels and 25% reduction in O ₃ across all regions. Functions estimated in both linear and quadratic form.	Adequate; same as Adams and Crocker (1984).	Inadequate producer model; same as Adams and Crocker (1984), except that analysis examines range of economic estimates reflecting variability in yield predictions resulting from sample size and functional form.	Same as Adams and Crocker (1984). Linear functions result in higher yield losses and hence higher economic loss estimates. Reported estimate (\$2.4 billion) is for quadratic response function.		
Kopp et al. (1984)	5 crops: corn, soybeans, wheat, cotton, and peanuts. Multiple cultivars of each crop except peanuts.	Adequate; analysis uses NCLAN O ₃ yield response data for 1980 through 1982. Yield losses (for estimates reported here) measured as the difference between ambient 1978 O ₃ and a level assumed to represent compliance with an 0.08 ppm standard.	Adequate; same as Adams and Crocker (1984) and Adams et al. (1984b) but for 1978 growing season.	Adequate; economic model consists of producer-level models, by crop, for numerous production regions. Predicted yield changes are used to generate supply shifts for each region/crop combined with crop demand relationships to estimate producer and consumer surpluses.	In addition to measuring the change in economic surplus for various assumed O ₃ levels, the analysis also includes an examination of the sensitivity of the estimates to the nature of the demand relationships used in the model.		
Adams et al. (1984b)	6 crops: barley, corn, soybeans, cotton, wheat, and sorghum. Multiple cultivars used for each crop except barley and grain sorghum; two for cotton, three for wheat, two for corn, and nine for soybean.	Adequate; analysis uses NCLAN O ₃ yield response data for 1980 through 1983. Yield changes reflect changes from 1980 ambient O ₃ of 10 and 40% reduction and a 25% increase for each response.	Adequate; same as above but for 1980 and 1976 through 1980 periods.	Adequate; economic model consists of two components: a series of farm-level models for each of 55 production regions and a national model of crop use and demand. Yield changes are used to generate regional supply shifts used in national model.	Consumer surplus estimated for both domestic and foreign markets; producer surplus nationally and by region. The analysis includes a range of economic estimates reflecting changes in response and O ₃ data and assumptions.		

^a Adequacy as defined here does not mean that the estimates are free of error; rather, it implies that the estimates are based on the most defensible biologic, aerometric, or economic information and models currently available.

^b Kriging is a spatial interpolation procedure that has been used to generate O₃ concentration data for rural areas in which no monitoring sites have been established. See Heck et al. (1983b).

an increase in O_3 to an assumed ambient concentration of 0.08 ppm (seasonal 7-hr average) across all regions produced a net loss of approximately \$3.0 billion.

The second study, by Adams et al. (1984b), is a component of the NCLAN program. The results were derived from an economic model of the U.S. agricultural sector that includes individual farm models for 55 production regions integrated with national supply-and-demand relationships for a range of crop and livestock activities. Using NCLAN data, the analysis examined yield changes for six major crops (corn, soybeans, wheat, cotton, grain, sorghum, and barley) that together account for over 75 percent of U.S. crop acreage. The estimated annual benefits (in 1980 dollars) from O_3 adjustments are substantial, but make up a relatively small percentage of total agricultural output (about 4 percent). Specifically, in this analysis, a 25 percent reduction in ozone from 1980 ambient levels resulted in benefits of \$1.7 billion. A 25 percent increase in ozone resulted in an annual loss (negative benefit) of \$2.363 billion. When adjusted for differences in years and crop coverages, these estimates are quite close to the Kopp et al. (1984) benefit estimates.

While the estimates from both Kopp et al. (1984) and Adams et al. (1984b) were derived from conceptually sound economic models and from the most defensible plant science and aerometric data currently available, there are several sources of uncertainty. These include the issue of exposure dynamics (7-hr per day exposures from the NCLAN experiments versus longer exposure periods, such as 12-hr exposures), and the lack of environmental interactions, particularly O_3 -moisture stress interactions, in many of the response experiments. Also, the O_3 data in both studies are based on a limited set of the monitoring sites in the SAROAD system of EPA, mainly sites in urban and suburban areas. While the spatial interpolation process used for obtaining O_3 concentration data (Kriging) results in a fairly close correspondence between predicted and actual O_3 levels at selected validation points, validation requires more monitoring sites in rural areas. The economic models, with their large number of variables, and parameters, and the underlying data used to derive these values, contain potential sources of uncertainty, including the effects on benefits estimates of market-distorting factors such as the Federal farm programs.

The inclusion of these possible improvements in future assessments is not likely, however, with the possible exception of market-distorting factors, to alter greatly the range of agricultural benefits provided in the Kopp et al. (1984) and Adams et al. (1984b) studies, for several reasons. First, the

current studies cover about 75 to 80 percent of U.S. agricultural crops (by value). For inclusion of the other 20 percent to change the estimates significantly would require that their sensitivities to O_3 be much greater than for the crops included to date. Second, model sensitivity analyses from existing studies indicate that changes in key plant science parameters must be substantial to translate into major changes in economic estimates. From experience to date it seems unlikely that use of different dose measures or interaction effects would result in changes of the magnitude already addressed in some of the sensitivity analyses. Third, even if there are such changes, there are likely to be countervailing responses; e.g., longer exposure periods may predict greater yield losses but O_3 -water stress tends to dampen or reduce the yield estimates. Finally, it should be noted that potential improvements in economic estimates are policy-relevant only to the extent that they alter the relationship between total benefits and total costs of that policy. Uncertainties in other effects categories are probably greater.

In conclusion, the recent economic estimates of benefits to agriculture of O_3 control, particularly those estimates by Kopp et al. (1984) and Adams et al. (1984b), meet the general criteria discussed in Section 6.5 and hence provide the most defensible evidence given in the literature to date of the general magnitude of such effects. Relative to estimates given in the 1978 criteria document (U.S. Environmental Protection Agency, 1978) and economic information on most other O_3 effects categories (non-agricultural), these two studies, in combination with the underlying NCLAN data on yield effects, provide the most comprehensive economic information to date on which to base judgments regarding the economic efficiency of alternative SNAAQs. As noted above, there are still gaps in plant science and aerometric data and a strong need for meteorological modeling of O_3 formation and transport processes for use in formulating rural O_3 scenarios. With regard to the economic data and models used, the impact of factors that upset free-market equilibria needs further analysis. Additionally, it must be emphasized that none of the studies has accounted for the compliance costs of effecting changes in O_3 concentrations in ambient air. For a cost-benefit analysis to be complete, the annualized estimated benefits to agriculture that would result from O_3 control would have to be combined with benefits accruing to other sectors and then compared with the overall annualized compliance costs.

1.5.10 Effects of Peroxyacetyl Nitrate on Vegetation

Peroxyacetyl nitrate (PAN) is a highly phytotoxic air pollutant that is produced by photochemical reactions similar to those that produce O_3 . Both O_3 and PAN can coexist in the photochemical oxidant complex in ambient air. The effects of PAN were a concern in southern California for almost 20 years before the phytotoxicity of O_3 under ambient conditions was identified. The symptoms of photochemical oxidant injury that were originally described (prior to 1960) were subsequently shown to be identical with the symptoms produced by PAN. Following the identification of PAN as a phytotoxic air pollutant, PAN injury (foliar symptoms) has been observed throughout California and in several other states and foreign countries.

1.5.10.1 Factors Affecting Plant Response to PAN. Herbaceous plants are sensitive to PAN and cultivar differences in sensitivity have been observed in field and controlled studies. Trees and other woody species, however, are apparently resistant to visible foliar injury from PAN (Taylor, 1969; Davis, 1975, 1977).

Taylor et al. (1961) demonstrated that there is an absolute requirement for light before, during, and after exposure or visible injury from PAN will not develop. Field observations showed that crops growing under moisture stress developed little or no injury during photochemical oxidant episodes while, adjacent to them, recently irrigated crops were severely injured (Taylor, 1974).

Only a few studies have investigated the effects of PAN and O_3 mixtures on plants. When plants were exposed to both gases at their respective injury thresholds, no interaction between the gases was found (Tonneijck, 1984). At higher concentrations, the effects were less than additive. Studies with petunia confirmed that O_3 tended to reduce PAN injury (Nouchi et al., 1984).

1.5.10.2 Limiting Values of Plant Response. The limiting-value method has been used to estimate the lowest PAN concentration and exposure duration reported to cause visible injury on various plant species (Jacobson, 1977). The analysis yielded the following range of concentrations and exposure durations likely to induce foliar injury: (1) 200 ppb for 0.5 hr; (2) 100 ppb for 1.0 hr; and (3) 35 ppb for 4.0 hr.

Other studies, however, suggest that these values need to be lowered by 30 to 40 percent to reduce the likelihood of foliar injury (Tonneijck, 1984). For example, foliar injury developed on petunia plants exposed at 5 ppb PAN

for 7 hr (Fukuda and Terakado, 1974). Under field conditions, injury symptoms may develop on sensitive species when PAN concentrations reach approximately 15 ppb for 4 hr (Taylor, 1969).

1.5.10.3 Effects of PAN on Plant Yield. Only a few limited studies have been conducted to determine the effects of PAN on plant growth and yield. In greenhouse studies, radish, oat, tomato, pinto bean, beet, and barley were exposed to PAN concentrations of up to 40 ppb for 4 hr/day, twice/wk, from germination to crop maturity (Taylor et al., 1983). No significant effects on yield were detected. This is supportive of field observations, in which foliar injury from ambient PAN exposures was found but no evidence was seen of reduced yield in these crops. In contrast, lettuce and Swiss chard exposed to PAN concentrations of up to 40 ppb for 4 hr/day, twice/wk, from germination to crop maturity showed yield losses up to 13 percent (lettuce) and 23 percent (Swiss chard) without visible foliar injury symptoms (Taylor et al., 1983). The results indicate that PAN at concentrations below the foliar-injury threshold can cause significant yield losses in sensitive cultivars of leafy vegetable crops. In addition, photochemical oxidant events have caused foliar injury on leafy vegetables (Middleton et al., 1950) for which the foliage is the marketable portion. After severe PAN damage, entire crops may be unmarketable or else extensive hand work may be required to remove the injured leaves before the crop may be marketed.

A comparison of PAN concentrations likely to cause either visible injury or reduced yield with measured ambient concentrations (see Chapter 5) indicates that it is unlikely that ambient PAN will impair the intended use of plants in the United States except in some areas of California and possibly in a few other localized areas.

1.6 EFFECTS OF OZONE ON NATURAL ECOSYSTEMS AND THEIR COMPONENTS

1.6.1 Responses of Ecosystems to Ozone Stress

The responses to ozone of individual species and subspecies of herbaceous and woody vegetation are well documented. They include (1) injury to foliage, (2) reductions in growth, (3) losses in yield, (4) alterations in reproductive capacity, and (5) alterations in susceptibility to pests and pathogens, especially "stress pathogens" (National Research Council, 1977; U.S. Environmental Protection Agency, 1978; this document, Chapter 6). The responses elicited by

ozone in individual species and subspecies of primary producers (green plants) have potential consequences for natural ecosystems because effects that alter the interdependence and interrelationships among individual components of populations can, if the changes are severe enough, perturb ecosystems. Because, however, of the numerous biotic and abiotic factors known to influence the response of ecosystem components such as trees (see, e.g., Cowling, 1985; Manion, 1985), it is difficult to relate natural ecosystem changes to ozone specifically, and especially to ozone alone. Ozone can only be considered a contributing factor.

Evidence indicates that any impact of ozone on ecosystems will depend on the responses to ozone of the producer community. Producer species (trees and other green plants) are of particular importance in maintaining the integrity of an ecosystem, since producers are the source, via photosynthesis, of all new organic matter (energy/food) added to an ecosystem. Any significant alterations in producers, whether induced by ozone or other stresses, can potentially affect the consumer and decomposer populations of the ecosystem, and can set the stage for changes in community structure by influencing the nature and direction of successional changes (Woodwell, 1970; Bormann, 1985), with possibly irreversible consequences (see, e.g., Odum, 1985; Bormann, 1985).

1.6.2 Effects of Ozone on Producers

In forest ecosystems, tree populations are the producers. As such, they determine the species composition, trophic relationships, and energy flow and nutrient cycling of forest ecosystems (Ehrlich and Mooney, 1983). Ozone-induced effects on the growth of trees has been clearly demonstrated in controlled studies (see Chapter 6). For example, Kress and Skelly (1982) showed the following reductions in growth in height in seedlings exposed to ozone for 6 hr/day for 28 days: American sycamore, 9 percent (0.05 ppm O_3); sweetgum, 29 percent (0.10 ppm O_3); green ash, 24 percent (0.10 ppm); willow oak, 19 percent (0.15 ppm O_3); and sugar maple, 25 percent (0.15 ppm). Similar results have been obtained for other tree species by other investigators (e.g., Dochinger and Townsend, 1979; Mooi, 1980; Patton, 1981; Kress et al., 1982). Some species, however, have been shown to exhibit increased growth in short-term ozone exposures (e.g., yellow poplar and white ash; Kress and Skelly, 1982). Hogsett et al. (1985) found reductions in growth in height, in

radial growth, and in root growth in slash pine seedlings exposed for up to 112 days to 7-hr seasonal mean concentrations of 0.104 ppm O_3 (with a 1-hr daily maximum of 0.126 ppm O_3) and 0.076 ppm O_3 (with a 1-hr daily maximum of 0.094 ppm O_3).

Field studies on the Cumberland Plateau (near Oak Ridge, TN) have shown reductions in growth in eastern white pine exposed to ambient air O_3 concentrations ≥ 0.08 ppm (1-hr) (Mann et al., 1980), with 1-hr concentrations ranging over the multi-year study from 0.12 ppm to 0.2 ppm (McLaughlin et al., 1982). It should be noted, however, that in the McLaughlin et al. (1982) study trees classified as ozone-tolerant sustained greater percentage reductions in radial growth in the last 4 years (1976 to 1979) of the 1962 to 1979 period for which growth was examined than the reductions observed in trees classified as ozone-sensitive. In the Blue Ridge Mountains of Virginia, Benoit et al. (1982) found reductions in radial growth of sensitive eastern white pine in a multi-year study in which 1-hr O_3 concentrations were generally 0.05 to 0.07 ppm but peaked at ≥ 0.12 ppm on as many as 5 consecutive days at a time.

The concentrations of ozone reported for sites on the Cumberland Plateau and in the Blue Ridge Mountains may not fully represent the actual exposures at those sites, however, since measurements were made in the daytime only. For species in which stomates remain open at night, such as eastern white pine, the possible occurrence of peak ozone concentrations at night, from transported urban plumes, is an important consideration for accurately assessing concentration-response relationships.

Exposures of trees and other producers to ozone have been shown to reduce photosynthesis (e.g., Miller et al., 1969; Botkin et al., 1972; Barnes, 1972; Carlson, 1979; Coyne and Bingham, 1981; Yang et al., 1983; Reich and Amundson, 1985) and to alter carbohydrate allocation, especially the partitioning of photosynthate between roots and tops (e.g., Price and Treshow, 1972; Tingey et al., 1976; McLaughlin et al., 1982). Krause et al. (1984) have associated growth reductions in ozone-exposed seedlings with foliar leaching. All three of these effects have been postulated as mechanisms of the reduced growth seen in ozone-exposed vegetation.

Responses to ozone are not uniform among plants of the same species and the same approximate age. Differential responses have been attributed in part to differences in genetic potential (e.g., Mann et al., 1980; Coyne and Bingham,

1981; Benoit et al., 1982). In addition, the age of the plant and its developmental stage at time of exposure influence its response to ozone (see Chapter 6). Other factors, as well, influence the types and magnitude of plant responses to ozone, including such macro- and microenvironmental factors as temperature, relative humidity, soil moisture, light intensity, and soil fertility (see Chapter 6).

Trees may respond rapidly to O_3 stress. Needles of sensitive eastern white pine usually exhibit injury symptoms within a few days after exposure to high O_3 concentrations. In other instances, responses are more subtle and may not be observable for years because trees are perennials and must therefore cope over time with the cumulative effects of multiple short- and long-term stresses. Reductions in the growth of annual rings observed in ponderosa, Jeffrey, and eastern white pine have been attributed to the exposure of the trees to O_3 over a period of 10 to 20 years (Miller and Elderman, 1977; Miller et al., 1982; McLaughlin et al., 1982; Benoit et al., 1982). Decline and dieback of red spruce in the northeastern United States and reduced growth rates of red spruce, balsam fir, and Fraser fir in central West Virginia and western Virginia also have been attributed to stresses, to which air pollution is a possible contributor, that began at least 20 years ago (Johnson and Siccama, 1983; Adams et al., 1985).

1.6.3 Effects of Ozone on Other Ecosystem Components and on Ecosystem Interactions

Evidence for the effects of ozone on other ecosystem components indicates that most are indirect, occurring chiefly as a result of the direct effects of ozone on trees and other producers. Significant alterations in producer species can change the ability of a species to compete and thus can influence the nature and direction of successional changes in the ecosystem. Likewise, significant alterations in producers can result in changes in the consumer and decomposer populations that depend on producers as their food source. Studies in the San Bernardino Mountain ecosystems in the 1970s have provided some evidence of successional shifts and of predisposition to infestation by pests and pathogens as the result of oxidant-induced changes in ponderosa and Jeffrey pines (see Section 1.6.4 below).

Marked morphological deterioration of the common lichen species, Hypogymnia enteromorpha, was documented in areas of the San Bernardino Mountains having high oxidant concentrations. A comparison of the species of lichens found

growing on ponderosa and Jeffrey pine with collections from the early 1900's indicated the presence of 50 fewer species (Sigal and Nash, 1983).

McCool et al. (1979) and Parmeter et al. (1962) reported decreases in mycorrhizal infections and rootlets in ozone-stressed citrange (a citrus hybrid) and ponderosa pine, respectively. Mahoney (1982), on the other hand, found no evidence of impairment in the development of mycorrhizal associations in loblolly pine seedlings exposed to ozone plus sulfur dioxide; however, shoot dry weight was decreased by 12 percent.

The effects of ozone on mycorrhizae are of particular note here, since mycorrhizae are essential for the optimal development of most plants because of the functions they perform. Mycorrhizal fungi increase the solubility of minerals, improve the uptake of nutrients for host plants, protect roots against pathogens, produce plant growth hormones, and move carbohydrates from one plant to another (Hacskeylo, 1972). Ozone may disrupt the association between mycorrhizal fungi and plants, possibly by inhibiting photosynthesis and reducing the amounts of sugars and carbohydrates available for transfer from leaves of producers to the roots. Mycorrhizae are known to be sensitive to alterations in carbon allocation to the roots in host plants (Hacskeylo, 1973).

Because of the complex interactions among plants, pests, pathogens, and other biotic and abiotic factors, Laurence and Weinstein (1981) have emphasized the critical importance of examining pollutant-pathogen and pollutant-insect interactions in determining the growth impact of a pollutant. Manion (1985) has emphasized the necessity of taking non-pollutant stresses, both biotic and abiotic, into account when attempting to attribute changes in forest ecosystems to air pollutants.

1.6.4 Effects of Ozone on Specific Ecosystems

One of the most thoroughly studied ecosystems in the United States is the mixed-conifer forest ecosystem in the San Bernardino Mountains of southern California. Sensitive plant species there began showing injury in the early 1950's (Miller and Elderman, 1977) and the source of the injury was identified as oxidants (ozone) in 1962 (Miller et al., 1963). In an inventory begun in 1968, Miller found that sensitive ponderosa and Jeffrey pines were being selectively removed by oxidant air pollution. Mortality of 8 and 10 percent was found in two respective populations of ponderosa pine studied between 1968

and 1972. Monitoring in that period showed ozone concentrations ≥ 0.08 ppm for ≥ 1300 hours, with concentrations rarely decreasing below 0.05 ppm at night near the crest of the mountain slope (Miller, 1973).

In a subsequent interdisciplinary study (1973 through 1978), biotic and abiotic components and ecosystem processes were examined. The ecosystem components most directly affected were various tree species, the fungal microflora of needles, and the foliose lichens on the bark of trees. In May through September, 1973 through 1978, 24-hr-average ozone concentrations ranged from about 0.03 to 0.04 ppm to about 0.10 to 0.12 ppm. (Monitoring was done by the Mast meter through 1974 and by the UV method from 1975 through 1978). Foliar injury on sensitive ponderosa and Jeffrey pine was observed when the 24-hr-average ozone concentrations were 0.05 to 0.06 ppm (Miller et al., 1982). Injury, decline, and death of these species were associated with the major ecosystem changes observed (Miller et al., 1982).

Growth reductions attributable to oxidant air pollution were calculated by McBride et al. (1975) for ponderosa pine saplings. Assuming 1910 to 1940 to be a period of low oxidant pollution and 1944 to 1974 a period of high oxidant pollution, they used radial growth increments (dbh) to calculate an oxidant-induced decrease in diameter of 40 percent. On the basis of the 3-year growth of saplings in filtered and nonfiltered air in portable greenhouses, they calculated oxidant-induced reductions of 26 percent in height growth (McBride et al., 1975). No standardized methods for determining tree ring widths were available at the time of this study.

Carbon flow and mineral nutrient cycling were influenced by the accumulation of litter under stands with the most severe needle injury and by defoliation, as well as by a reduction in the number of species and the population density of the fungi that normally colonize living needles and later participate in decomposition. The most likely result of heavy litter accumulation is a reduction in pine seedling establishment and greater establishment and growth of oxidant-tolerant understory species on some sites and oxidant-tolerant trees on other sites (Miller et al., 1982).

Changes in the energy available to trees influenced the biotic interactions, so that weakened ponderosa pines were more susceptible to attack by predators such as bark beetles and to pathogens such as root rot fungi (Stark and Cobb, 1969). Fewer western pine beetles were required to kill weakened trees (Dahlsten and Rowney, 1980); and stressed pines became more susceptible

to root rot fungi (James et al., 1980) and showed a decrease in mycorrhizal rootlets and their replacement by saprophytic fungi (Parmeter et al., 1962).

Accelerated rates of mortality of ponderosa and Jeffrey pine in the forest overstory, resulting from O_3 injury, root rot, and pine beetle attack, and in some cases, removal by fire, changed the basic structure of the forest ecosystem (Phase IV; Bormann, 1985) by causing replacement of the dominant conifers with self-perpetuating, fire-adapted, O_3 -tolerant shrub and oak species, which are considered less beneficial than the former pine forest and which inhibit reestablishment of conifers (Miller et al., 1982).

Injury to vegetation in other ecosystems has also been reported. Duchelle et al. (1983) found reductions in the growth and productivity of graminoid and forb vegetation in the Shenandoah National Park, where 1-hr ozone concentrations ranged from 0.08 to 0.10 ppm in the 3-year study period, with 1-hr concentrations >0.06 ppm occurring for 1218, 790, and 390 hours in 1979, 1980, and 1981, respectively. Treshow and Stewart (1973) fumigated species that grow in the Salt Lake Valley and the Wasatch Mountains in Utah and found key, dominant species to be ozone-sensitive. The National Park Service (1985) has recently reported ozone-induced injury to vegetation in the Santa Monica Mountains National Recreational Area, the Sequoia and Kings Canyon National Parks, Indiana Dunes National Lakeshore, Great Smoky Mountains National Park, and the Congaree Swamp National Monument. The impact of injury to vegetation in these ecosystems has not been appraised.

It should be emphasized that the relative importance of a given species in a given ecosystem must be considered in any assessment of the impact of ozone (or other stresses) on an ecosystem. Ozone has not had the impact on other ecosystems that it has had on the San Bernardino mixed-conifer forest because the plant species injured do not have a role equal in importance to the role of ponderosa and Jeffrey pines in the San Bernardino ecosystem.

1.6.5 Economic Valuation of Ecosystems

At the present time, economists and ecologists remain unable to devise a mutually acceptable framework for estimating the economic value of ecosystems. In addition, the credibility of any attempt to estimate at present the economic value of ecosystems would be diminished by a lack of scientific data (1) on the time-course of the manifestation of stress-induced effects on ecosystems, (2) on the point at which ecosystems lose the capacity for self-repair, and

(3) on the points at which they begin to lose their ability to provide, respectively, priced and unpriced benefits to society. In addition, estimation of the economic losses that might be associated with the specific effects of ozone on ecosystems requires other data that are presently in short supply, i.e., better and more aerometric data and better and more data on additional variables, so that significant contributions from abiotic factors other than ozone, as well as from biotic factors, can be credibly estimated.

1.7 EFFECTS OF OZONE AND OTHER PHOTOCHEMICAL OXIDANTS ON NONBIOLOGICAL MATERIALS

Over two decades of research show that ozone damages certain nonbiological materials; the amount of damage to actual in-use materials, however, is poorly characterized. Knowledge of indoor/outdoor ozone gradients, for example, has expanded considerably in recent years, and this type of exposure information has not been incorporated in materials damage studies. Moreover, virtually all materials research on photochemical oxidants has focused on ozone. Theoretically, a number of the less abundant oxidants may equal or surpass ozone in reactivity with certain materials, but this possibility has not been tested empirically. In the absence of photochemical pollution, oxidative damage to certain materials still occurs from atmospheric oxygen, but at a much reduced rate and through different chemical mechanisms. Generally, ozone damages elastomers by cracking along the line of physical stress, whereas oxygen causes internal damage to the material.

The materials most studied in ozone research are elastomers and textile fibers and dyes. Natural rubber and synthetic polymers of butadiene, isoprene, and styrene, used in products like automobile tires and protective outdoor electrical coverings, account for most of the elastomer production in the United States. The action of ozone on these compounds is well known, and dose-response relationships have been established and corroborated by several studies. These relationships, however, must be correlated with adequate exposure information based on product use. For these and other economically important materials, protective measures have been formulated to reduce the rate of oxidative damage. When antioxidants and other protective measures are incorporated in elastomer production, the dose-cracking rate is reduced considerably, although the extent of reduction differs widely according to the material and the type and amount of protective measures used.

The formation of cracks and the depth of cracking in elastomers are related to ozone dose and are influenced greatly by humidity and mechanical stress. Dose is defined as the product of concentration and time of exposure. The importance of ozone dose was demonstrated by Bradley and Haagen-Smit (1951), who used a specially formulated ozone-sensitive natural rubber. Samples exposed to ozone at a concentration of 20,000 ppm cracked almost instantaneously, and those exposed to lower concentrations took a proportionately longer time to crack. At concentrations of 0.02 to 0.46 ppm, and under 100-percent strain, the cracking rate was directly proportional to the time of exposure, from 3 to 65 min.

Similar findings were reported by Edwards and Storey (1959), who exposed two SBR elastomers to ozone at a concentration of 0.25 ppm for 19 to 51 hr under 100-percent strain. With ozone doses of 4.75 ppm-hr to 12.75 ppm-hr, a proportional rate in cracking depth was observed, averaging 2.34 $\mu\text{m/hr}$ for cold SBR and 4.01 $\mu\text{m/hr}$ for hot SBR. When antiozonants were added to the compounds, the reduction in cracking depth rate was proportional to the amount added. Haynie et al. (1976) exposed samples of a tire sidewall to ozone at concentrations of 0.08 and 0.5 ppm for 250 to 1000 hr under 10 and 20 percent-strain. Under 20-percent strain, the mean cracking rate for 0.08 ppm was 1.94 $\mu\text{m/hr}$. From these and other data, they estimated that at the ozone standard of the time (0.08 ppm, 1-hr average), and at the annual NO_x standard of 0.05 ppm, it would take 2.5 years for a crack to penetrate cord depth.

In addition to stress, factors affecting the cracking rate include atmospheric pressure, humidity, sunlight, and other atmospheric pollutants. Veith and Evans (1980) found a 16-percent difference in cracking rates reported from laboratories located at various geographic elevations.

Ozone has been found to affect the adhesion of plies (rubber-layered strips) in tire manufacturing. Exposure to ozone concentrations of 0.05 to 0.15 ppm for a few hours significantly decreased adhesion in an NR/SBR blend, causing a 30-percent decrease at the highest ozone level. This adhesion problem worsened at higher relative humidities. When fast-blooming waxes and antiozonants or other antioxidants were added, only the combination of protective measures allowed good adhesion and afforded protection from ozone and sunlight attack. Wenghoefer (1974) showed that ozone (up to 0.15 ppm), especially in combination with high relative humidity (up to 90 percent), caused greater adhesion losses than did heat and NO_2 with or without high relative humidity.

The effects of ozone on dyes have been known for nearly three decades. In 1955, Salvin and Walker exposed certain red and blue anthraquinone dyes to a 0.1 ppm concentration of ozone and noted fading, which until that time was thought to be caused by NO_2 . Subsequent work by Schmitt (1960, 1962) confirmed the fading action of ozone and the importance of relative humidity in the absorption and reaction of ozone in vulnerable dyes. The acceleration in fading of certain dyes by high relative humidity was noted later by Beloin (1972, 1973) at an ozone concentration of 0.05 ppm and relative humidity of 90 percent. Kamath et al. (1982) also found that a slight rise in relative humidity (85 to 90 percent) caused a 20-percent dye loss in nylon fibers.

Both the type of dye and the material in which it is incorporated are important factors in a fabric's resistance to ozone. Haynie et al. (1976) and Upham et al. (1976) found no effects from ozone concentrations of 0.1 to 0.5 ppm for 250 to 1000 hr under high and low relative humidity (90 vs. 50 percent) on royal blue rayon-acetate, red rayon-acetate, or plum cotton. On the other hand, Haylock and Rush (1976, 1978) showed that anthraquinone dyes on nylon fibers were sensitive to fading from ozone at a concentration of 0.2 ppm at 70 percent relative humidity and 40°C for 16 hr. Moreover, the same degree of fading occurred in only 4 hr at 90 percent relative humidity. At higher concentrations, there was a parallel increase in fading. Along with Heuvel et al. (1978) and Salvin (1969), Haylock and Rush (1976, 1978) noted the importance of surface area in relation to the degree of fading. In explaining this relationship, Kamath et al. (1982) found that ozone penetrated into the fiber itself and caused most of the fading through subsequent diffusion to the surface.

Field studies by Nipe (1981) and laboratory work by Kamath et al. (1982) showed a positive association between ozone levels and dye fading of nylon materials at an ozone concentration of 0.2 ppm and various relative humidities. In summary, dye fading is a complex function of ozone concentration, relative humidity, and the presence of other gaseous pollutants. At present, the available research is insufficient to quantify the amount of damaged material attributable to ozone alone. Anthraquinone dyes incorporated into cotton and nylon fibers appear to be the most sensitive to ozone damage.

The degradation of fibers from exposure to ozone is poorly characterized. In general, most synthetic fibers like modacrylic and polyester are relatively resistant, whereas cotton, nylon, and acrylic fibers have greater but varying

sensitivities to the gas. Ozone reduces the breaking strength of these fibers, and the degree of reduction depends on the amount of moisture present. Under laboratory conditions, Bogaty et al. (1952) found a 20 percent loss in breaking strength in cotton textiles under high-moisture conditions after exposure to a 0.06 ppm concentration of ozone for 50 days; they equated these conditions to a 500- to 600-day exposure under natural conditions. Kerr et al. (1969) found a net loss of 9 percent in breaking strength of moist cotton fibers exposed to ozone at a concentration of 1.0 ppm for 60 days. The limited research in this area indicates that ozone in ambient air may have a minimal effect on textile fibers, but additional research is needed to verify this conclusion.

The effects of ozone on paint are small in comparison with those of other factors. Past studies have shown that, of various paints, only vinyl and acrylic coil coatings are affected, and that this impact has a negligible effect on the useful life of the material coated. Preliminary results of current studies have indicated a statistically significant effect of ozone and relative humidity on latex house paint, but the final results of those studies are needed before conclusions can be drawn.

For a number of important reasons, the estimates of economic damage to materials are far from reliable. Most of the available studies are now outdated in terms of the ozone concentrations, technologies, and supply-demand relationships that prevailed when the studies were conducted. Additionally, little was (and is) known about the physical damage functions, and cost estimates were simplified to the point of not properly recognizing many of the scientific complexities of the impact of ozone. Assumptions about exposure to ozone generally ignored the difference between outdoor and indoor concentrations. Also, analysts have had difficulty separating ozone damage from other factors affecting materials maintenance and replacement schedules. For the most part, the studies of economic cost have not marshalled factual observations on how materials manufacturers have altered their technologies, materials, and methods in response to ozone. Rather, the analysts have merely made bold assumptions in this regard, most of which remain unverified through the present time.

Even more seriously, the studies followed engineering approaches that do not conform with acceptable methodologies for measuring economic welfare. Almost without exception, the studies reported one or more types of estimated or assumed cost increases borne by materials producers, consumers, or both. The recognition of cost increase is only a preliminary step, however, towards

evaluating economic gains and losses. The analysis should then use these cost data to proceed with supply and demand estimation that will show how materials prices and production levels are shifted. Because the available studies fail to do this, there is a serious question as to what they indeed measure.

Increased ozone levels increase sales for some industries even as they decrease welfare for others. For example, manufacturers of antiozonants for automobile tires conceivably stand to increase sales as ozone increases, while purchasers of tires stand to pay higher prices. This is only one illustration of a fundamental analytical deficiency in the various studies of materials damage: the absence of a framework for identifying gainers and losers, and the respective amounts they gain and lose.

Among the various materials studies, research has narrowed the type of materials most likely to affect the economy from increased ozone exposure. These include elastomers and textile fibers and dyes. Among these, natural rubber used for tires is probably the most important economically for the following reasons: (1) significant ambient air exposure and long use life; (2) significant unit cost; and (3) large quantities and widespread distribution.

The study by McCarthy et al. (1983) calculated the cost of antiozonants in tires for protection against ozone along with the economic loss to the retread industry. While limitations in this study preclude the reliable estimation of damage costs, the figures indicate the magnitude of potential damage from exposure to ozone in ambient air.

Research has shown that certain textile fibers and dyes and house paint are also damaged by ozone, but the absence of reliable damage functions make accurate economic assessments impossible. Thus, while damage to these materials is undoubtedly occurring, the actual damage costs cannot be estimated confidently.

It is apparent from the review presented in this chapter that a great deal of work remains to be done in developing quantitative estimates of materials damage from photochemical oxidant exposures. This is not meant to deprecate the years of research reported in this document, for much has been gained in refining the initial methodologies used for assessing damage. Yocom et al. (1985) have summarized the current state of knowledge:

We have learned that some costs may be difficult to quantify either because they are minimal or because they are overshadowed by other factors, such as wear or obsolescence. We have learned that damage functions are complex and are influenced by the presence of other pollutants and by weather. We have learned that more accurate estimates of materials in place may be obtained using selective sampling and extrapolation. And we have learned that a mere cost-accounting of damage does not present a true estimate of economic cost if it does not account for the welfare effects induced by shifts in the supply-demand relationship.

1.8 TOXICOLOGICAL EFFECTS OF OZONE AND OTHER PHOTOCHEMICAL OXIDANTS

1.8.1 Introduction

The biological effects of O_3 have been studied extensively in animals and a wide array of toxic effects have been ascribed to O_3 inhalation. Although much has been accomplished to improve the existing data base, refine the concentration-response relationships and interpret better the mechanisms of O_3 effects, many of the present data were not accumulated with the idea that quantitative comparisons to man would be drawn. In many cases, only qualitative comparisons can be made. To maximize the extent that animal toxicological data can be used to estimate the human health risk of exposure to O_3 , the qualitative as well as quantitative similarities between the toxicity of O_3 to animals and man must be considered more carefully in the future. Significant advances have been made in understanding the toxicity of O_3 through appropriate animal models. This summary highlights the significant results of selected studies that will provide useful data for better predicting and assessing, in a scientifically sound manner, the possible human responses to O_3 .

Summary figures and tables are presented in the following sections. The practical purpose of this presentation of the data is to help the reader focus on what types of effects or responses have been reported, what concentrations have been tested (1.0 ppm and lower), and as a convenient list of references with each of the biological parameters measured. Studies were selected for inclusion in these figures and tables on the basis of specific criteria presented below:

1. Studies have been cited when the reported effects are clearly due to O_3 exposure. Effects due to mixtures of O_3 with other pollutants have been summarized in a separate figure and table. Studies involving exercise, diet deficiencies, or other possible modifiers of response to O_3 have not been included.

2. Cited studies report the effects of O_3 exposure over a broad range of animal species and strains and for varying lengths of time. Specific details on animal species, exposure duration, and observed biological effects can be obtained from the tables in Chapter 9.
3. Each closed symbol on the figures represents one or more studies conducted at that particular concentration that caused effects. Specific references can be found in the accompanying tables.
4. Each open circle represents one or more studies that used the given concentration, but reported no significant effects. No-effect levels are also indicated by brackets in the accompanying tables.
5. Only pulmonary function effects were divided by short-term (<14 days) and long-term exposures to follow the discussion in the text.

In order to keep this section brief and concise, it was necessary to be somewhat selective in determining what and how this information would be presented. A number of important factors, such as the specific length of exposure, were not included. Also, the parameter selected to illustrate a specific response was usually broad and very general. For example, the category "decreases in macrophage function" includes such diverse endpoints as measurements of lysosomal and phagocytic activity, macrophage mobility, or chemotactic response. These responses may or may not be related to one another. Thus, care must be taken in how these data are used and interpreted. The only appropriate use is to gain an overview of the broad array of the effects of ozone and the concentrations which did and did not cause these effects.

1.8.2 Regional Dosimetry in the Respiratory Tract

The amount of O_3 acting at a given site in the lung is related to the airway luminal concentration at that level. As a result, O_3 does not immediately interact with cellular components of the respiratory tract. Instead, it first comes into contact with the mucous or surfactant layer lining the airway. It should be noted that O_3 is quite reactive chemically. Reactions with components of this layer cause an increase in total absorption of O_3 in the upper airways and in a reduction of the amount of O_3 reaching sensitive tissues. The site

at which uptake and subsequent interaction occur and the local dose (quantity of O_3 absorbed per unit area per time), along with cellular sensitivity, will determine the type and extent of the injury. Also, the capacity for responding to a specific dose may vary between animals and humans because of dissimilarities in detoxification systems, pharmacokinetics, metabolic rates, genetic makeup, or other factors. Thus, along with the above, a knowledge of the complex process of gas transport and absorption is crucial to understanding the effects of O_3 and other oxidants in humans.

The animal studies that have been conducted on ozone absorption are beginning to indicate the quantity and site of O_3 uptake in the respiratory tract. Experiments on the nasopharyngeal removal of O_3 in animals suggest that the fraction of O_3 uptake depends inversely on flow rate, that uptake is greater for nose than for mouth breathing, and that tracheal and chamber concentrations are positively correlated. Only one experiment measured O_3 uptake in the lower respiratory tract, finding 80 to 87 percent uptake by the lower respiratory tract of dogs (Yokoyama and Frank, 1972). At present, however, there are no reported results for human nasopharyngeal or lower respiratory tract absorption. Caution must be used in estimating nasopharyngeal uptake for normal respiration based upon experiments employing unidirectional flows.

To further an understanding of O_3 absorption, mathematical models have been developed to simulate the processes involved and to predict O_3 uptake by various regions and sites within the respiratory tract. The model of Aharonson et al. (1974) has been used to analyze nasopharyngeal uptake data. Applied to O_3 data, the model indicates that the average mass transfer coefficient in the nasopharyngeal region increases with increasing air flow, but the actual percent uptake decreases.

Three models have been developed to simulate lower respiratory uptake (McJilton et al., 1972; Miller et al., 1978b, 1985). These models are very similar in their treatment of O_3 in the airways (taking into account convection, diffusion, wall losses, and ventilatory patterns) and in their use of morphological data to define the dimensions of the airways and liquid lining. The models differ in their treatment of the mechanism of absorption. Both of the models of Miller and co-workers take into account chemical reactions of O_3 with constituents of the liquid lining, whereas the model of McJilton et al. does not. The models of Miller et al. differ in their treatment of chemical

reactions, as well as in the fact that the newer model includes chemical reactions of O_3 in additional compartments, such as tissue and blood.

Tissue dose is predicted by the models of Miller et al. to be relatively low in the trachea, to increase to a maximum between the junction of the conducting airways and the gas-exchange region, and then to decrease distally. This is not only true for animal simulations (guinea pig and rabbit) but it is also characteristic of the human simulations (Miller et al., 1978b; 1985).

A comparison of the results of Miller and co-workers with morphological data (that shows the centriacinar region to be most affected by O_3) indicates qualitative agreement between predicted tissue doses and observed effects in the pulmonary region. However, comparisons in the tracheobronchial region indicate that dose-effect correlations may be improved by considering other expressions of dose such as total absorption by an airway and by further partitioning of the mucous layer compartment in mathematical models. Further research is needed to define toxic mechanisms, as well as to refine our knowledge of important chemical, physical, and morphological parameters.

At present, there are few experimental results that are useful in judging the validity of the modeling efforts. Such results are needed, not only to understand better the absorption of O_3 and its role in toxicity, but also to support and to lend confidence to the modeling efforts. With experimental confirmation, models which further our understanding of the role of O_3 in the respiratory tract will become practical tools.

The consistency and similarity of the human and animal lower respiratory tract dose curves obtained thus far lend strong support to the feasibility of extrapolating to man the results obtained on animals exposed to O_3 . In the past, extrapolations have usually been qualitative in nature. With additional research in areas which are basic to the formulation of dosimetry models, quantitative dosimetric differences among species can be determined. If in addition, more information is obtained on species sensitivity to a given dose, significant advances can be made in quantitative extrapolations and in making inferences about the likelihood of effects of O_3 in man. Since animal studies are the only available approach for investigating the full array of potential disease states induced by exposure to O_3 , quantitative use of animal data is in the interest of better establishing O_3 levels to which man can safely be exposed.

1.8.3 Effects of Ozone on the Respiratory Tract

1.8.3.1 Morphological Effects. The morphological changes which follow exposure to less than $1960 \mu\text{g}/\text{m}^3$ (1.0 ppm) O_3 are very similar in all species of laboratory mammals studied. Of the many specific cell types found in the respiratory system, two types, ciliated cells and type 1 alveolar epithelial cells, are the cells most damaged morphologically following O_3 inhalation. Ciliated cells are found in the conducting airways, e.g., trachea, bronchi, and nonrespiratory bronchioles. Ciliated cells function in the normal clearance of the airways and the removal of inhaled foreign material. Following O_3 exposure of experimental animals, damaged ciliated cells have been reported in all of these conducting airways (Schwartz et al., 1976; Castleman et al., 1977). In rats, damage to ciliated cells appears most severe at the junction of the conducting airways with the gas exchange area (Stephens et al., 1974a; Schwartz et al., 1976). Damage to type 1 alveolar epithelial cells is limited to those cells located near this junction, i.e., the centriacinar or proximal alveolar region of the pulmonary acinus (Stephens et al., 1974b; Schwartz et al., 1976; Castleman et al., 1980; Barry et al., 1983; Crapo et al., 1984). Type 1 alveolar cells form most of the blood-air barrier where gas exchange occurs. Severely damaged ciliated and type 1 alveolar epithelial cells are shed (sloughed) from the tissue surface and are replaced by multiplication of other cell types less damaged by O_3 (Evans et al., 1985). This process has been most extensively studied in the centriacinar region where nonciliated bronchiolar cells and type 2 alveolar epithelial cells become more numerous (Evans et al., 1976a,b,c; Lum et al., 1978). Some of these nonciliated bronchiolar and type 2 cells differentiate into ciliated and type 1 cells, respectively. Cell multiplication in bronchioles may be more than that required for replacement of damaged ciliated cells, and nonciliated bronchiolar cells may become hyperplastic (Castleman et al., 1977; Ibrahim et al., 1980; Eustis et al., 1981) and sometimes appear as nodules (Zitnik et al., 1978; Moore and Schwartz, 1981; Fujinaka et al., 1985). Inflammatory changes characterized by a variety of leukocytes with alveolar macrophages predominating, intramural edema, and fibrin are also seen in the centriacinar region (Stephens et al., 1974a; Schwartz et al., 1976; Castleman et al., 1977; Fujinaka et al., 1985).

The damage to ciliated and centriacinar type 1 alveolar epithelial cells and the inflammatory changes tend to occur soon after exposure to concentrations of O_3 as low as $392 \mu\text{g}/\text{m}^3$ (0.2 ppm). Damage to centriacinar type 1 alveolar

epithelium in rats has been well documented as early as 2 hours after exposure to O_3 concentrations of $980 \mu\text{g}/\text{m}^3$ (0.5 ppm) (Stephens et al., 1974a). In the same publication the authors report damage to centriacinar type 1 alveolar epithelial cells after 2 hours exposure to $392 \mu\text{g}/\text{m}^3$ (0.2 ppm) O_3 , but this portion of their report is not documented by published micrographs (Stephens et al., 1974a). Loss of cilia from cells in the rat terminal bronchiole occurs following exposure to $980 \mu\text{g}/\text{m}^3$ (0.5 ppm) O_3 for 2 hours (Stephens et al., 1974a). Damage to ciliated cells has been seen following exposure of both rats and monkeys to $392 \mu\text{g}/\text{m}^3$ (0.2 ppm) O_3 , 8 hr/day for 7 days (Schwartz et al., 1976; Castleman et al., 1977). Centriacinar inflammation has been reported as early as 6 hours after exposure to $980 \mu\text{g}/\text{m}^3$ (0.5 ppm) O_3 (Stephens et al., 1974b) and 4 hours after exposure to $1568 \mu\text{g}/\text{m}^3$ (0.8 ppm) O_3 (Castleman et al., 1980).

During long-term exposures, the damage to ciliated cells and to centriacinar type 1 cells and centriacinar inflammation continue, though at a reduced rate. Damage to cilia has been reported in monkeys following 90-day exposure to $980 \mu\text{g}/\text{m}^3$ (0.5 ppm) O_3 , 8 hr/day (Eustis et al., 1981) and in rats exposed to $980 \mu\text{g}/\text{m}^3$ (0.5 ppm) O_3 , 24 hr/day for 180 days (Moore and Schwartz, 1981). Damage to centriacinar type 1 cells was reported following exposure of young rats to $490 \mu\text{g}/\text{m}^3$ (0.25 ppm) O_3 , 12 hrs/day for 42 days (Barry et al., 1983; Crapo et al., 1984). Changes in type 1 cells were not detectable after $392 \mu\text{g}/\text{m}^3$ (0.2 ppm) O_3 , 8 hr/day for 90 days but were seen in rats exposed to $980 \mu\text{g}/\text{m}^3$ (0.5 ppm) for the same period (Boorman et al., 1980). Centriacinar inflammatory changes persist during 180-day exposures of rats to $980 \mu\text{g}/\text{m}^3$ (0.5 ppm) O_3 , 24 hr/day (Moore and Schwartz, 1981) and one-year exposures of monkeys to $1254 \mu\text{g}/\text{m}^3$ (0.64 ppm) O_3 , 8 hr/day (Fujinaka et al., 1985).

Remodeling of distal airways and centriacinar regions occurs following long-term exposures to O_3 . Rats develop respiratory bronchioles between the terminal bronchiole to alveolar duct junction seen in control rats (Boorman et al., 1980; Moore and Schwartz, 1981). In monkeys, distal airway remodeling results in increased volumes of respiratory bronchioles which have thicker walls and a smaller internal diameter (Fujinaka et al., 1985). The walls of centriacinar alveoli are also thickened (Schwartz et al., 1976; Boorman et al., 1980; Barry et al., 1983; Crapo et al., 1984; Last et al., 1984a). Studies of the nature of these thickened interalveolar septa and bronchiolar walls revealed increases in inflammatory cells, fibroblasts, and amorphous

extracellular matrix (Last et al., 1984a; Fujinaka et al., 1985). Three studies provide morphological evidence of mild fibrosis (i.e., local increase of collagen) in centriacinar interalveolar septa following exposure to $< 1960 \mu\text{g}/\text{m}^3$ ($< 1 \text{ ppm}$) of O_3 (Last et al., 1979; Boorman et al., 1980; Moore and Schwartz, 1981). Changes in collagen location or amounts, or both, which occur with the remodeling of the distal airways, were reported in two of those studies (Boorman et al., 1980; Moore and Schwartz, 1981).

While morphometry of small pulmonary arteries is not commonly studied in O_3 -exposed animals, pulmonary artery walls thickened by muscular hyperplasia and edema were reported in rabbits exposed to $784 \mu\text{g}/\text{m}^3$ (0.4 ppm) O_3 , 6 hr/day, 5 days/week for 10 months (P'an et al., 1972). Thickened intima and media in pulmonary arterioles were reported in monkeys exposed to $1254 \mu\text{g}/\text{m}^3$ (0.64 ppm) O_3 , 8 hr/day for 1 year (Fujinaka et al., 1985).

Several of the effects of O_3 inhalation persisted after the O_3 inhalation ended and the animals breathed only filtered air several days or weeks. Lungs from rats exposed to $1568 \mu\text{g}/\text{m}^3$ (0.8 ppm) O_3 for 72 hours appeared normal 6 days after the end of the exposure (Plopper et al., 1978). However, incomplete resolution of the nonciliated bronchiolar epithelial hyperplasia was reported in monkeys 7 days after 50 hours exposure to $1568 \mu\text{g}/\text{m}^3$ (0.8 ppm) O_3 (Castleman et al., 1980) and in mice 10 days after a 20-day exposure to $1568 \mu\text{g}/\text{m}^3$ (0.8 ppm) O_3 , 24 hr/day (Ibrahim et al., 1980). Centriacinar inflammation and distal airway remodeling were still apparent 62 days after a 180-day exposure to $980 \mu\text{g}/\text{m}^3$ (0.5 ppm) O_3 , 24 hr/day (Moore and Schwartz, 1981).

While not all species of laboratory mammals have been studied following a single O_3 exposure regimen or using the same morphological techniques because investigators have asked different biological questions, there is a striking similarity of morphological effects in the respiratory system of all species studied. The cell types most damaged are the same. One of these cells, the type 1 alveolar epithelial cell, has a wide distribution in the pulmonary acinus and yet is damaged only in one specific location in all species studied. The other, the ciliated cell, appears damaged wherever it is located in the conducting airways. Damage to these cells is seen within hours after exposure to concentrations of O_3 much lower than 1 ppm and continues during exposures of weeks or months. Hyperplasia of other cell types is reported to start early in the exposure period, to continue throughout a long-term exposure, and when studied, to persist following postexposure periods of days or weeks.

Centriacinar inflammation is also seen early and is reported throughout long exposure periods. Duration of centriacinar inflammation during postexposure periods has been studied less often and appears dependent upon length of the exposure period.

Other effects which have been reported in fewer studies or in a more limited number of species include distal airway remodeling and thickened pulmonary arteriolar walls. Remodeling of distal airways has only been reported in rats and monkeys after long-term exposures. In rats, remodeling of distal airways has been reported to persist for several weeks after the O₃ exposure has ended. Thickened pulmonary arteriolar walls have been reported only twice, once after long-term exposure of rabbits and once after long-term exposure of monkeys.

Studies on the morphologic effects of O₃ exposures of experimental animals are summarized in Figure 1-7 and Table 1-11 (see Section 1.8.1 for criteria used to summarize the studies).

1.8.3.2 Pulmonary Function. One of the limitations of animal studies is that many pulmonary function tests comparable to those conducted after acute exposure of human subjects are difficult to interpret. Methods exist, however, for obtaining similar measurements of many variables pertinent to understanding the effects of ozone on the respiratory tract, particularly after longer exposure periods. A number of newer studies reported here reflect recent advances in studying the effects of O₃ on pulmonary function in small animals.

Changes in lung function following ozone exposure have been studied in mice, rats, guinea pigs, rabbits, cats, dogs, sheep, and monkeys. Short-term exposure for 2 hr to concentrations of 431 to 980 µg/m³ (0.22 to 0.5 ppm) produces rapid, shallow breathing and increased pulmonary resistance during exposure (Murphy et al., 1964; Yokoyama, 1969; Watanabe et al., 1973; Amdur et al., 1978). The onset of these effects is rapid and the abnormal breathing pattern usually disappears within 30 min after cessation of exposure. Other changes in lung function measured following short-term ozone exposures lasting 3 hr to 14 days are usually greatest 1 day following exposure and disappear by 7 to 14 days following exposure. These effects are associated with premature closure of the small, peripheral airways and include increased residual volume, closing volume, and closing capacity (Inoue et al., 1979).

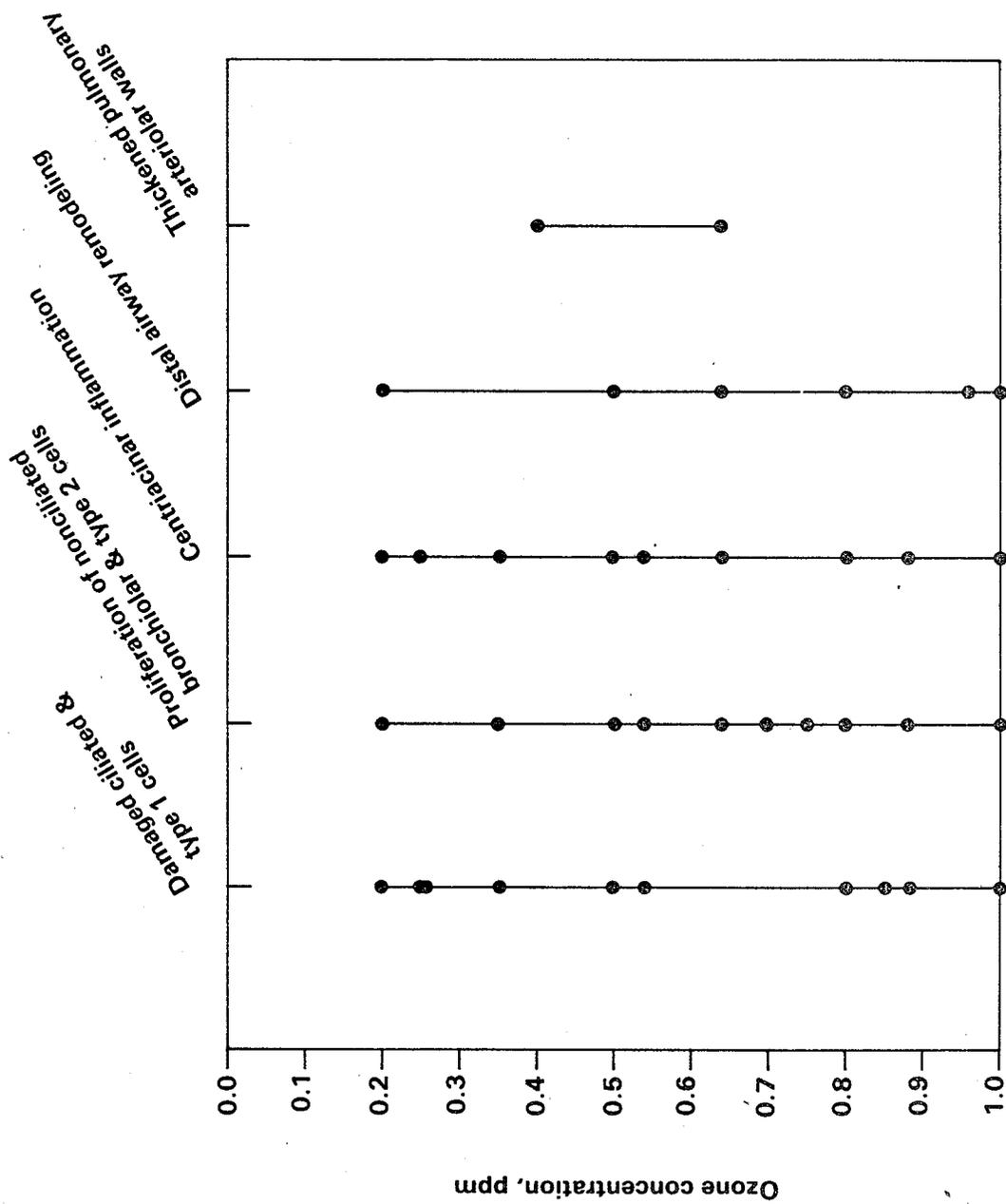


Figure 1-7. Summary of morphological effects in experimental animals exposed to ozone. See Table 1-11 for reference citations of studies summarized here.

TABLE 1-11. SUMMARY TABLE: MORPHOLOGICAL EFFECTS OF OZONE
IN EXPERIMENTAL ANIMALS

Effect/response	O ₃ concentration, ppm	References
Damaged ciliated and type 1 cells	[0.2], 0.5, 0.8	Boorman et al. (1980)
	0.2, 0.5, 0.8	Schwartz et al. (1976)
	0.2, 0.35	Castleman et al. (1977)
	0.25	Barry et al. (1983)
	0.25	Crapo et al. (1984)
	0.26, 0.50, 1.0	Boatman et al. (1974)
	0.5	Stephens et al. (1974b)
	0.5	Moore and Schwartz (1981)
	0.5	Evans et al. (1985)
	0.5, 0.8	Eustis et al. (1981)
	0.5, 0.8	Mellick et al. (1975, 1977)
	0.54, 0.88	Stephens et al. (1974a)
	0.8	Castleman et al. (1980)
	0.8	Plopper et al. (1978)
	0.85	Stephens et al. (1978)
Proliferation of non- ciliated bronchiolar and type 2 cells	0.2, 0.35	Castleman et al. (1977)
	0.35, 0.50, 0.70, 0.75, 1.0	Evans et al. (1976b)
	0.5	Evans et al. (1985)
	0.5	Zitnik et al. (1978)
	0.5	Moore and Schwartz (1981)
	0.5, 0.8	Eustis et al. (1981)
	0.54, 0.88	Freeman et al. (1974)
	0.64	Fujinaka et al. (1985)
	0.7	Evans et al. (1976a)
	0.8	Castleman et al. (1980)
	0.8	Lum et al. (1978)
	0.8	Ibrahim et al. (1980)
	1.0	Cavender et al. (1977)
Centriacinar inflammation	[0.2], 0.5, 0.8	Boorman et al. (1980)
	0.2	Plopper et al. (1979)
	0.2, 0.5, 0.8	Schwartz et al. (1976)
	0.25	Barry et al. (1983)
	0.25	Crapo et al. (1984)
	0.35	Castleman et al. (1977)
	0.5	Stephens et al. (1974b)
	0.5	Moore and Schwartz (1981)
	0.5, 0.8	Mellick et al. (1975, 1977)
	0.5, 0.8	Brummer et al. (1977)
	0.5, 0.8	Last et al. (1979)
	0.54, 0.88	Stephens et al. (1974a)
	0.54, 0.88	Freeman et al. (1974)
	0.64	Fujinaka et al. (1985)
	0.8	Castleman et al. (1980)
1.0	Freeman et al. (1973)	

TABLE 1-11 (continued). SUMMARY TABLE: MORPHOLOGICAL EFFECTS OF OZONE IN EXPERIMENTAL ANIMALS

Effect/response	O ₃ concentration, ppm	References
Distal airway remodeling	[0.2], 0.5, 0.8	Boorman et al. (1980)
	0.2, 0.5, 0.8	Schwartz et al. (1976)
	0.5	Moore and Schwartz (1981)
	0.64, 0.96	Last et al. (1984a)
	0.64	Fujinaka et al. (1985)
	1.0	Freeman et al. (1973)
Thickened pulmonary arteriolar walls	0.4	P'an et al. (1972)
	0.64	Fujinaka et al. (1985)

Studies of airway reactivity following short-term ozone exposure of 1 to 2 hr duration in experimental animals show that O₃ increases the reactivity of the lungs to a number of stimuli. Mild exercise, histamine aerosol inhalation, and breathing air with reduced oxygen or elevated carbon dioxide concentrations caused rapid, shallow breathing in conscious dogs immediately following 2-hr exposures to 1100 to 1666 µg/m³ (0.56 to 0.85 ppm) of O₃ (Lee et al., 1979, 1980). Aerosolized ovalbumin caused an increased incidence of anaphylaxis in mice preexposed to 980 or 1568 µg/m³ (0.5 or 0.8 ppm) of O₃ continuously for 3 to 5 days (Osebold et al., 1980). In addition, increased airway sensitivity to histamine or cholinomimetic drugs administered by aerosol or injection has been noted in several species after exposure to 980 to 5880 µg/m³ (0.5 to 3.0 ppm) of O₃ (Easton and Murphy, 1967; Lee et al., 1977; Abraham et al., 1980, 1984a,b; Gordon and Amdur, 1980; Gordon et al., 1981, 1984; Roum and Murlas, 1984). The mechanism responsible for O₃-induced bronchial reactivity is still uncertain but may involve more than one specific factor. Ozone has been shown to cause increased sensitivity of vagal sensory endings in the dog airway (Lee et al., 1977, 1979, 1980). Ozone exposure may also enhance the airway responsiveness to bronchoconstrictors by altering sensitivity of the airway smooth muscle directly or through released cellular mediators (Gordon et al., 1981, 1984; Abraham et al., 1984a,b). In some species, increased airway hyperreactivity may be explained by increased transepithelial permeability or decreased thickness of the airway mucosa (Osebold et al., 1980; Abraham et al., 1984b). Ozone exposure may also decrease airway hyperreactivity by causing mucous

hypersecretion, thereby limiting the airway penetration of inhaled bronchoconstrictors (Abraham et al., 1984a).

The time course of airway hyperreactivity after exposure to 980 to 5880 $\mu\text{g}/\text{m}^3$ (0.5 to 3.0 ppm) of O_3 suggests a possible association with inflammatory cells and pulmonary inflammation (Holtzman et al., 1983a,b; Sielczak et al., 1983; Fabbri et al., 1984; O'Byrne et al., 1984a,b; Murlas and Roum, 1985). However, the time course of responsiveness is variable in different species and the relationships between airway inflammation and reactivity at different concentrations of O_3 are not well understood. Additional studies that demonstrate increased collateral resistance following 30 min local exposure of O_3 or histamine in sublobar bronchi of dogs (Gertner et al., 1983a,b,c,1984) suggest that other mechanisms, along with amplification of reflex pathways, may contribute to changes in airway reactivity depending not only on the concentration of O_3 in the airways but also on the extent of penetration of ozone into the lung periphery.

The effects of short-term exposures to O_3 on pulmonary function and airway reactivity in experimental animals are summarized in Figure 1-8 and Table 1-12 (see Section 1.8.1 for criteria used in developing this summary).

Exposures of 4 to 6 weeks to ozone concentrations of 392 to 490 $\mu\text{g}/\text{m}^3$ (0.2 to 0.25 ppm) increased lung distensibility at high lung volumes in young rats (Bartlett et al., 1974; Raub et al., 1983). Similar increases in lung distensibility were found in older rats exposed to 784 to 1568 $\mu\text{g}/\text{m}^3$ (0.4 to 0.8 ppm) for up to 180 days (Moore and Schwartz, 1981; Costa et al., 1983; Martin et al., 1983). Exposure to O_3 concentrations of 980 to 1568 $\mu\text{g}/\text{m}^3$ (0.5 to 0.8 ppm) increased pulmonary resistance and caused impaired stability of the small peripheral airways in both rats and monkeys (Wegner, 1982; Costa et al., 1983; Yokoyama et al., 1984; Kotlikoff et al., 1984). The effects in monkeys were not completely reversed by 3 months following exposure; lung distensibility had also decreased in the postexposure period, suggesting the development of lung fibrosis which has also been suggested morphologically and biochemically.

The effects of long-term exposures to ozone on pulmonary function and airway reactivity in experimental animals are summarized in Figure 1-9 and Table 1-13 (see Section 1.8.1 for criteria used in developing this summary).

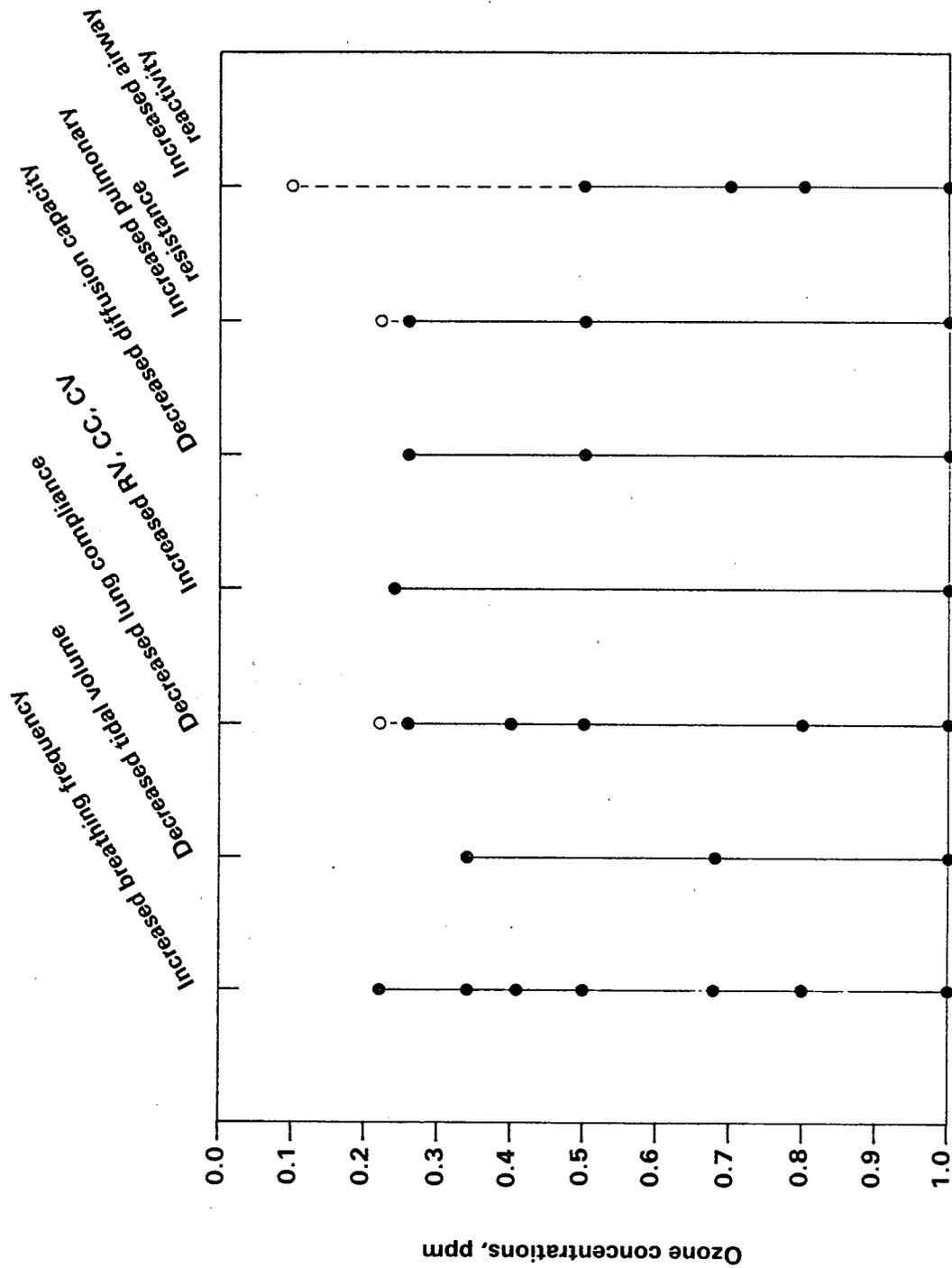


Figure 1-8. Summary of effects of short-term ozone exposures on pulmonary function in experimental animals. See Table 1-12 for reference citations of studies summarized here.

TABLE 1-12. SUMMARY TABLE: EFFECTS ON PULMONARY FUNCTION OF SHORT-TERM EXPOSURES TO OZONE IN EXPERIMENTAL ANIMALS

Effect/response	O ₃ concentration, ppm	References
Increased breathing frequency	0.22, 0.41, 0.8 0.34, 0.68, 1.0 0.5	Amdur et al. (1978) Murphy et al. (1964) Yokoyama (1969)
Decreased tidal volume	0.34, 0.68, 1.0	Murphy et al. (1964)
Decreased lung compliance	[0.22], 0.41, 0.8 0.26, 0.5, 1.0 1.0	Amdur et al. (1978) Watanabe et al. (1973) Yokoyama (1974)
Increased residual volume (RV), closing capacity (CC), and closing volume (CV)	0.24 - 1.0	Inoue et al. (1979)
Decreased diffusion capacity	0.26, 0.5, 1.0	Watanabe et al. (1973)
Increased pulmonary resistance	[0.22] 0.26, 0.5, 1.0 0.5 1.0	Amdur et al. (1978) Watanabe et al. (1973) Yokoyama (1969) Yokoyama (1974)
Increased airway reactivity	[0.1]-0.8 [0.1]-0.8, 1.0 0.5, 1.0 0.7 1.0	Gordon and Amdur (1980) Gordon et al. (1981, 1984) Abraham et al. (1980, 1984a,b) Lee et al. (1977) Holtzman et al. (1983a,b)

1.8.3.3 Biochemical Effects. The lung is metabolically active, and several key steps in metabolism have been studied after O₃ exposure. Since the procedures for such studies are invasive, this research has been conducted only in animals. Effects, to be summarized below, have been observed on antioxidant metabolism, oxygen consumption, proteins, lipids, and xenobiotic metabolism.

The lung contains several compounds (e.g., vitamin E, sulfhydryls, glutathione) and enzymes (e.g., glutathione peroxidase, glutathione reductase, glucose-6-phosphate dehydrogenase, and superoxide dismutase) that function as antioxidants, thereby defending the lung against oxidant toxicity from the

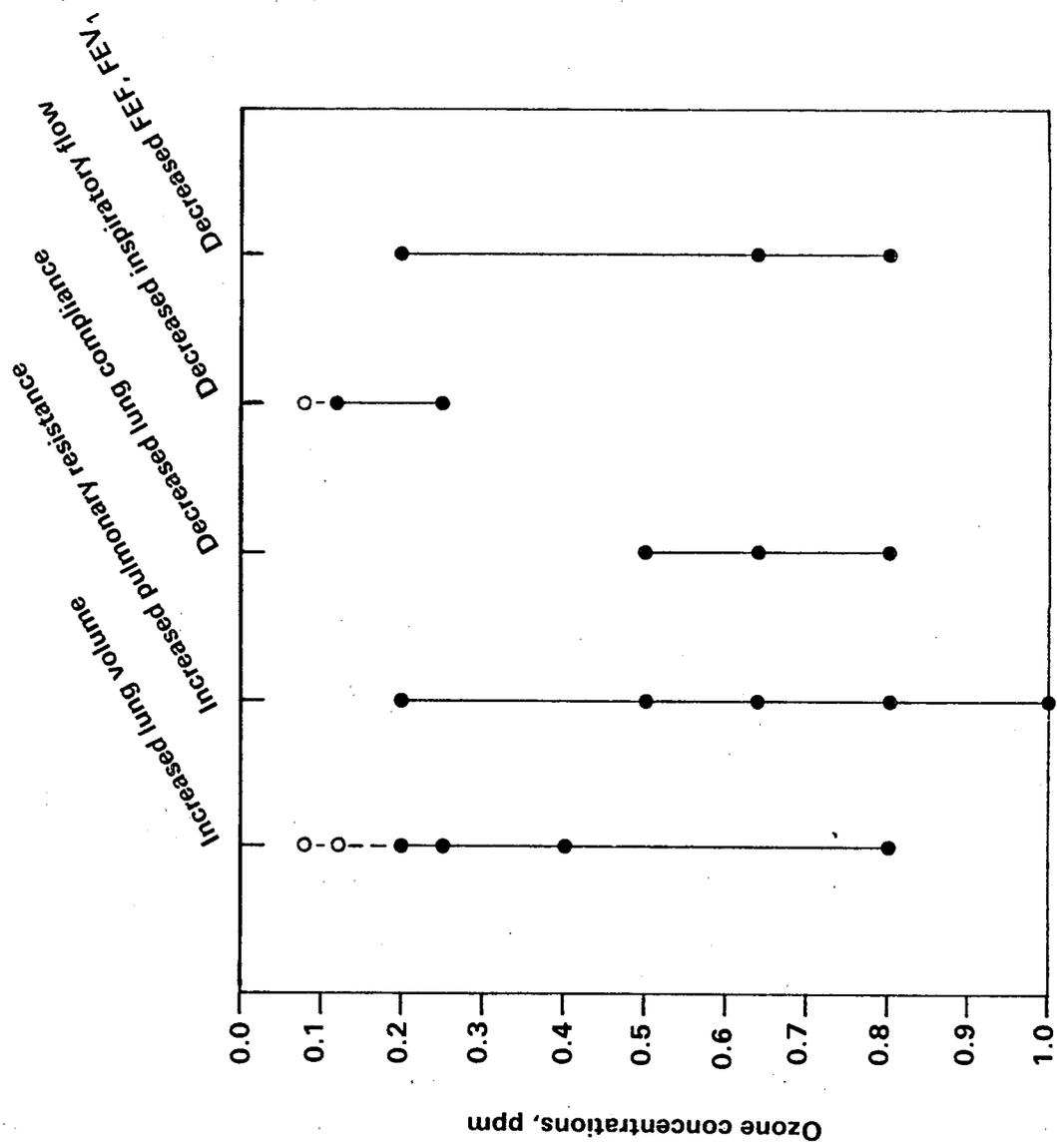


Figure 1-9. Summary of effects of long-term ozone exposures on pulmonary function in experimental animals. See Table 1-13 for reference citations of studies summarized here.

TABLE 1-13. SUMMARY TABLE: EFFECTS ON PULMONARY FUNCTION OF LONG-TERM EXPOSURES TO OZONE IN EXPERIMENTAL ANIMALS

Effect/response	O ₃ concentration, ppm	References
Increased lung volume	[0.08], [0.12], 0.25 0.2 [0.2], 0.8 0.4	Raub et al. (1983) Bartlett et al. (1974) Costa et al. (1983) Martin et al. (1983)
Increased pulmonary resistance	0.2, 0.8 0.5, 1.0 0.64 0.64	Costa et al. (1983) Yokoyama et al., 1984 Wegner (1982) Kotlikoff et al., 1984
Decreased lung compliance	0.5, 0.8 0.64	Eustis et al. (1981) Wegner (1982)
Decreased inspiratory flow	[0.08], 0.12, 0.25	Raub et al. (1983)
Decreased forced expiratory volume (FEV ₁) and flow (FEF ₁)	0.2, 0.8 0.64	Costa et al. (1983) Wegner (1982)

oxygen in air, from oxidants produced during metabolic processes, and from oxidizing air pollutants such as ozone. Obviously, this protection is only partial for O₃ since exposure to ozone causes numerous effects on lung structure, function, and biochemistry. Acute exposure to high ozone levels (2920 µg/m³, 2 ppm) typically decreases antioxidant metabolism, whereas repeated exposures to lower levels (between 272 and 1568 µg/m³, 0.2 and 0.8 ppm) increases this metabolism (DeLucia et al., 1975b). In rats maintained on normal diets, this response has been observed after a week of continuous or intermittent exposure to 392 µg/m³ (0.2 ppm) O₃ (Mustafa, 1975; Mustafa and Lee, 1976; Plopper et al., 1979). Similar responses are seen in monkeys and mice, but at higher concentrations (980 µg/m³, 0.5 ppm) (Fukase et al., 1978; Mustafa and Lee, 1976).

The effects of O₃ on oxygen consumption have been studied since oxygen consumption is a fundamental parameter of cellular metabolism, reflecting energy production by cells. As with antioxidant metabolism, acute exposure to

high ozone levels ($\geq 3920 \mu\text{g}/\text{m}^3$; ≥ 2 ppm) decreases metabolism (and thus, oxygen consumption); repeated exposure to lower levels ($> 1568 \mu\text{g}/\text{m}^3$, 0.8 ppm) increases oxygen consumption (Mustafa et al., 1973; Schwartz et al., 1976; Mustafa and Lee, 1976). Effects in rats on normal diets have been observed after a short-term exposure to ozone levels as low as $392 \mu\text{g}/\text{m}^3$ (0.2 ppm) (Schwartz et al., 1976; Mustafa et al., 1973; Mustafa and Lee, 1976). Monkeys are affected at a higher level of ozone ($980 \mu\text{g}/\text{m}^3$, 0.5 ppm).

Similar patterns of response for both antioxidant metabolism and oxygen consumption are observed after exposure to ozone. A 7-day exposure to ozone produces linear concentration-related increases in activities of glutathione peroxidase, glutathione reductase, glucose-6-phosphate dehydrogenase, and succinate oxidase (Mustafa and Lee, 1976; Chow et al., 1974; Schwartz et al., 1976; Mustafa et al., 1973). Rats on a vitamin E-deficient diet experience an increase in enzyme activities at $196 \mu\text{g}/\text{m}^3$ (0.1 ppm) ozone as compared to $392 \mu\text{g}/\text{m}^3$ (0.2 ppm) in animals on normal diets (Chow et al., 1981; Mustafa and Lee, 1976; Mustafa, 1975). Research on these enzymes has shown that there is no significant difference in effects from continuous versus intermittent exposure; this, along with concentration-response data, suggests that the concentration of ozone is more important than duration of exposure in causing these effects (Chow et al., 1974; Schwartz et al., 1976; Mustafa and Lee, 1976).

Duration of exposure still plays a role, however. During exposures up to 1 or 4 weeks, antioxidant metabolism and O_2 consumption generally do not change on the first day of exposure; by about day 2, increases are observed and by about day 4 a plateau is reached (Mustafa and Lee, 1976; DeLucia et al., 1975a). Recovery from these effects occurs by 6 days post-exposure (Chow et al., 1976). This plateauing of effects in the presence of exposure does not result in long-term tolerance. If rats are re-exposed after recovery is observed, the increase in enzyme activities is equivalent to that observed in animals exposed for the first time (Chow et al., 1976).

The influence of age on responsiveness is also similar for antioxidant metabolism and oxygen consumption (Elsayed et al., 1982a; Tyson et al., 1982; Lunan et al., 1977). Suckling neonates (5 to 20 days old) generally exhibited a decrease in enzyme activities; as the animals grew older (up to about 180 days old), enzyme activities generally increased with age. Species differences may exist in this response (Mustafa and Lee, 1976; Mustafa et al., 1982; Chow

et al., 1975; DeLucia et al., 1975a). Studies in which monkeys have been compared to rats did not include a description of appropriate statistical considerations applied (if any); thus, no definitive conclusions about responsiveness of monkeys versus rats can be made.

The mechanism responsible for the increase in antioxidant metabolism and oxygen consumption is not known. The response is typically attributed, however, to concurrent morphological changes, principally the loss of type 1 cells and an increase in type 2 cells that are richer in the enzymes measured.

Monoxygenases constitute another class of enzymes investigated after ozone exposure. These enzymes function in the metabolism of both endogenous (e.g., biogenic amines, hormones) and exogenous (xenobiotic) substances. The substrates acted upon are either activated or detoxified, depending on the substrate and the enzyme. Acute exposure to 1470 to 1960 $\mu\text{g}/\text{m}^3$ (0.75 to 1 ppm) ozone decreased cytochrome P-450 levels and enzyme activities related to both cytochrome P-450 and P-448. The health impact of these changes is uncertain since only a few elements of a complex metabolic system were measured.

The activity of lactate dehydrogenase is increased in lungs of vitamin E-deficient rats receiving a short-term exposure to 196 $\mu\text{g}/\text{m}^3$ (0.1 ppm) ozone (Chow et al., 1981). Higher levels caused a similar response in rats, but not in monkeys, on normal diets (Chow et al., 1974, 1977). This enzyme is frequently used as a marker of cellular damage because it is released upon cytotoxicity. It is not known, however, whether the increase in this enzyme is a direct reflection of cytotoxicity or whether it is an indicator of an increased number of type 2 cells and macrophages in the lungs.

An increase in a few of the measured activities of lysosomal enzymes has been shown in the lungs of rats exposed to $\geq 1372 \mu\text{g}/\text{m}^3$ (0.7 ppm) ozone (Dillard et al., 1972; Castleman et al., 1973a; Chow et al., 1974). This response is most likely the result of an increase in inflammatory cells in the lungs rather than an induction of enzymes, since lysosomal enzymes in alveolar macrophages decrease after in vivo or in vitro exposure to ozone (Hurst et al., 1970; Hurst and Coffin, 1971).

As discussed previously, long-term exposure to high O_3 concentrations causes mild lung fibrosis (i.e., local increase of collagen in centriacinar interalveolar septa). This morphological change has been correlated with biochemical changes in the activity of prolyl hydroxylase (an enzyme that catalyzes the production of hydroxyproline) and in hydroxyproline content (a

component of collagen that is present in excess in fibrosis) (Last et al., 1979; Bhatnagar et al., 1983). An increase in collagen synthesis has been observed, with $980 \mu\text{g}/\text{m}^3$ (0.5 ppm) O_3 being the minimally effective concentration tested (Hussain et al., 1976a,b; Last et al., 1979). During a prolonged exposure, prolyl hydroxylase activity increases by day 7 and returns to control levels by 60 days of exposure. When a short-term exposure ceases, prolyl hydroxylase activity returns to normal by about 10 days post-exposure, but hydroxyproline levels remain elevated 28 days post-exposure. Thus, the product of the increased synthesis, collagen, remains relatively stable. One study (Costa et al., 1983) observed a small decrease in collagen levels of rats at 392 and $1568 \mu\text{g}/\text{m}^3$ (0.2 and 0.8 ppm) O_3 after an intermittent exposure for 62 days.

The effects of O_3 on increasing collagen content may be progressive; i.e., after a 6-week intermittent exposure of rats to 0.64 or 0.96 ppm O_3 ceased, collagen levels 6 week post-exposure were elevated over the levels immediately after exposure (Last et al., 1984b). Also, there appears to be little difference between continuous and intermittent exposure in increasing collagen levels in rat lungs (Last et al., 1984b). Thus, the intermittent clean air periods were not sufficient to permit recovery.

Although the ability of O_3 to initiate peroxidation of unsaturated fatty acids in vitro is well established, few in vivo studies of lung lipids have been conducted. Generally, ozone decreases unsaturated fatty acid content of the lungs (Roehm et al., 1972) and decreases incorporation of fatty acids into lecithin (a saturated fatty acid) (Kyei-Aboagye et al., 1973). These alterations, however, apparently do not alter the surface-tension-lowering properties of lung lipids that are important to breathing (Gardner et al., 1971; Huber et al., 1971).

One of the earliest demonstrated effects of ozone was that very high concentrations caused mortality as a result of pulmonary edema. As more sensitive techniques were developed, lower levels ($510 \mu\text{g}/\text{m}^3$, 0.26 ppm) were observed to increase the protein content of the lung (Hu et al., 1982). Since some of the excess protein could be attributed to serum proteins, the interpretation was that edema had occurred. This effect was more pronounced several hours after exposure ceased. At higher concentrations, a loss of carrier-mediated transport from the air side of the lung to the blood side was observed (Williams et al., 1980). These changes imply an effect on the barrier function

of the lung, which regulates fluxes of various substances with potential physiological activities across the alveolar walls.

The biochemical effects observed in experimental animals exposed to O_3 are summarized in Figure 1-10 and Table 1-14 (see Section 1.8.1 for criteria used in developing this summary).

1.8.3.4 Host Defense Mechanisms. Reports over the years have presented substantial evidence that exposure to ozone impairs the antibacterial activity of the lung, resulting in an impairment of the lung's ability to kill inhaled microorganisms. Suppression of this biocidal defense of the lung can lead to microbial proliferation within the lung, resulting in mortality. The mortality response is concentration-related and is significant at concentrations as low as 157 to 196 $\mu\text{g}/\text{m}^3$ (0.08 to 0.1 ppm) (Coffin et al., 1967; Ehrlich et al., 1977; Miller et al., 1978a; Aranyi et al., 1983). The biological basis for this response appears to be that ozone or one of its reactive products can impair or suppress the normal bactericidal functions of the pulmonary defenses, which results in prolonging the life of the infectious agent, permitting its multiplication and ultimately, in this animal model, resulting in death. Such infections can occur because of O_3 effects on a complex host defense system involving alveolar macrophage functioning, lung fluids, and other immune factors.

The data obtained in various experimental animal studies indicate that short-term ozone exposure can reduce the effectiveness of several vital defense systems including (1) the ability of the lung to inactivate bacteria and viruses (Coffin et al., 1968; Coffin and Gardner, 1972b; Goldstein et al., 1974, 1977; Warshauer et al., 1974; Bergers et al.; 1983. Schwartz and Christman, 1979; Ehrlich et al., 1979); (2) the mucociliary transport system (Phalen et al., 1980; Frager et al., 1979; Kenoyer et al., 1981); (3) the immunological system (Campbell and Hilsenroth, 1976; Fujimaki et al., 1984; Thomas et al., 1981b; Aranyi et al., 1983; and (4) the pulmonary macrophage (Dowell et al., 1970; Goldstein et al., 1971a,b, and 1977; Hadley et al., 1977; McAllen et al., 1981; Witz et al., 1983; Hurst et al., 1970; Hurst and Coffin, 1971; Amoruso et al., 1981). Studies have also indicated that the activity level of the test subject and the presence of other airborne chemicals are important variables that can influence the determination of the lowest effective concentration of the pollutant (Gardner et al., 1977; Aranyi et al., 1983; Ehrlich, 1980, 1983; Grose et al., 1980, 1982; Phalen et al., 1980; Goldstein et al., 1974; Illing et al., 1980).

Increased O₂ consumption
 Increased lysosomal activities
 and prolyl hydroxylase activity
 Increased lung hydroxylase activity
 Altered glycoprotein secretions
 and permeability changes
 Increased alveolar protein
 secretions
 Increased LDH activity
 Increased NADPH-cytochrome c
 reductase activity
 Increased GSH metabolism
 HPSH
 Decreased unsaturated
 fatty acids

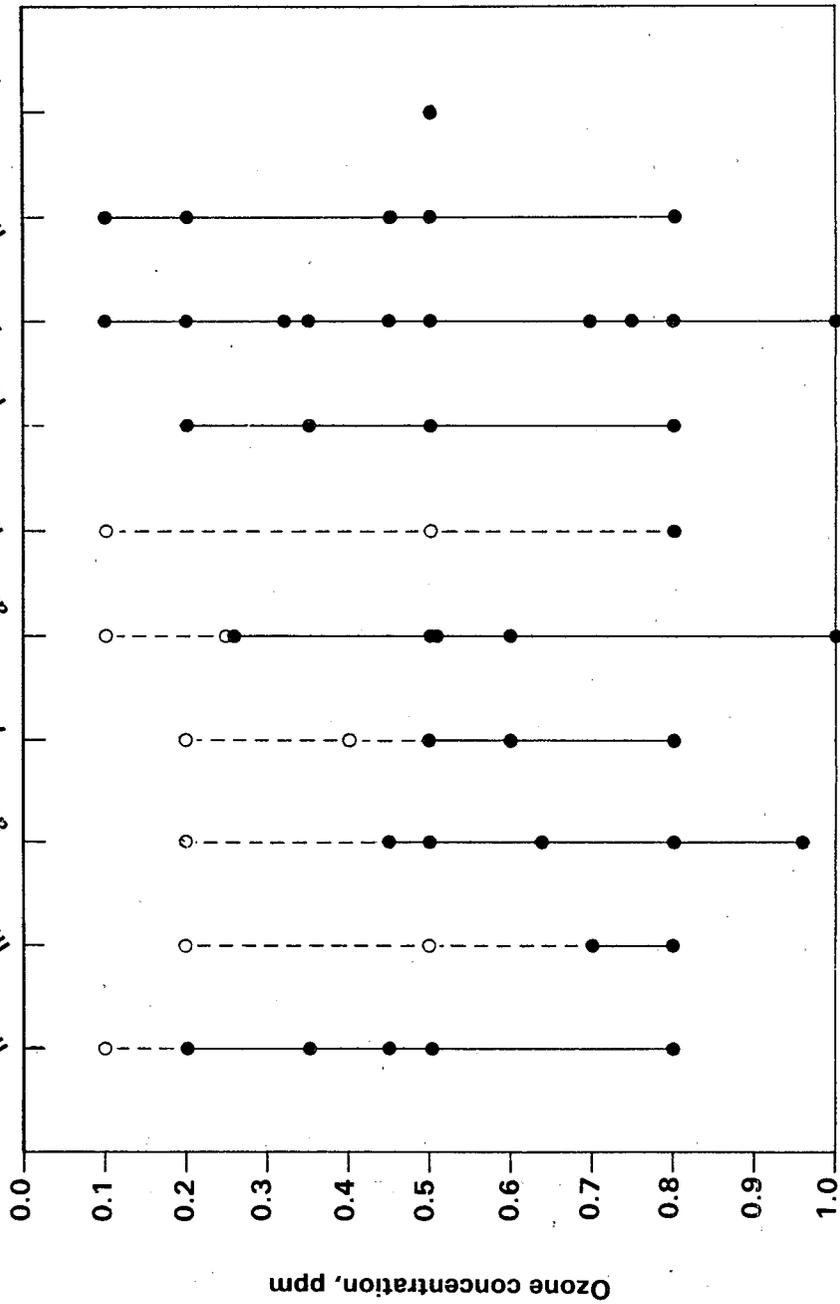


Figure 1-10. Summary of biochemical changes in experimental animals exposed to ozone. See Table 1-14 for reference citations of studies summarized here.

TABLE 1-14. SUMMARY TABLE: BIOCHEMICAL CHANGES
IN EXPERIMENTAL ANIMALS EXPOSED TO OZONE

Effect/response	O ₃ concentration, ppm	References
Increased O ₂ consumption	[0.1], 0.2	Mustafa (1975)
	[0.1], 0.2, 0.35, 0.5, 0.8	Mustafa and Lee (1976)
	0.2, 0.5, 0.8	Mustafa et al. (1973)
	0.2, 0.5, 0.8	Schwartz et al. (1976)
	0.45	Mustafa et al. (1982)
	0.8	Chow et al. (1976)
	0.8	Elsayed et al. (1982a)
Increased lysosomal enzyme activities	[0.2], [0.5], 0.8	Chow et al. (1974)
	0.7, 0.8	Dillard et al. (1972)
	0.7, 0.8	Castleman et al. (1973a,b)
Increased lung hydroxyproline and prolyl hydroxylase activity	[0.2], 0.5, 0.8	Hussain et al. (1976a,b)
	0.2, 0.8	Costa et al. (1983)
	0.45, 0.8	Bhatnagar et al. (1983)
	0.5, 0.64, 0.96	Last et al. (1979, 1984b)
	0.5	Last and Greenberg (1980)
0.8	Hesterberg and Last (1981)	
Altered mucus glycoprotein secretions	[0.2], [0.4], 0.5, 0.6, 0.8	Last and Kaizu (1980)
	0.5, 0.6, 0.8	Last and Cross (1978)
	0.6, 0.8	Last et al. (1977)
Increased alveolar protein and permeability changes	[0.1], 0.26, 0.51, 1.0	Hu et al. (1982)
	[0.25], 0.5, 1.0	Alpert et al. (1971a)
	0.6, 1.0	Williams et al. (1980)
	1.0	Reasor et al. (1979)
Increased LDH activity	[0.1]	Chow et al. (1981)
	[0.5], 0.8	Chow et al. (1977)
	0.8	Chow and Tappel (1973)
Increased NADPH - cytochrome c reductase activity	0.2, 0.35, 0.8	Mustafa and Lee (1976)
	0.2, 0.5, 0.8	Schwartz et al. (1976)
	0.2, 0.5, 0.8	DeLucia et al. (1972, 1975a,b)
Increased GSH metabolism	[0.1]	Chow et al. (1981)
	0.1, 0.2	Plopper et al. (1979)
	0.2, 0.35, 0.5, 0.8	Mustafa and Lee (1976)
	0.2, 0.5, 0.8	Chow et al. (1974)
	0.2, 0.5, 0.8	DeLucia et al. (1972, 1975a,b)
	0.2, 0.5, 0.8	Schwartz et al. (1976)
	0.2, 0.5, 1.0	Fukase et al. (1975)
	0.32	Moore et al. (1980)
	0.45	Mustafa et al. (1982)
0.5	Chow et al. (1975)	

TABLE 1-14 (continued). SUMMARY TABLE: BIOCHEMICAL CHANGES
IN EXPERIMENTAL ANIMALS EXPOSED TO OZONE

Effect/response	O ₃ concentration, ppm	References
	0.5, 1.0	Fukase et al. (1978)
	0.7, 0.75, 0.8	Chow and Tappel (1972, 1973)
	0.8	Elsayed et al. (1982a,b; 1983)
	0.8	Chow et al. (1976)
	0.9	Tyson et al. (1982)
	0.9	Lunan et al. (1977)
Increased NPSH	0.1, 0.2	Plopper et al. (1979)
	0.2, 0.5, 0.8	DeLucia et al. (1975b)
	0.45	Mustafa et al. (1982)
	0.8	Chow et al. (1976)
Decreased unsaturated fatty acids	0.5	Roehm et al., 1972

Ciliated cells are damaged by O₃ inhalation, as demonstrated by major morphological changes in these cells including necrosis and sloughing or by the shortening of the cilia in cells attached to the bronchi. Sufficient ciliated cell damage should result in decreased transport of viable and non-viable particles from the lung. Rats exposed to 784, 1568, 1960, or 2352 µg/m³ (0.4, 0.8, 1.0, or 1.2 ppm) for times as short as 4 hr have decreased short-term clearance of particles from the lung (Phalen et al., 1980; Frager et al., 1979; Kenoyer et al., 1981). Short-term clearance is mostly due to mucus transport of particles, and the decreased short-term clearance is an anticipated functional result predicted from morphological observations. The mucous glycoprotein production of the trachea is also altered by O₃ exposure. Mucous glycoprotein biosynthesis, as measured *ex vivo* in cultured tracheal explants from exposed rats, was inhibited by short-term continuous exposure to 1568 µg/m³ (0.8 ppm) of O₃ for 3 to 5 days (Last and Cross, 1978; Last and Kaizu, 1980; Last et al., 1977). Glycoprotein synthesis and secretion recovered to control values after 5 to 10 days of exposure and increased to greater than control values after 10 days of exposure. With this increase in production of mucus, investigators have found that the velocity of the tracheal mucus was

significantly reduced following a 2 hr exposure to $1960 \mu\text{g}/\text{m}^3$ (1.0 ppm) (Abraham et al., 1980).

A problem remains in assessing the relevance of these animal data to humans. Green (1984) reviewed the literature and compared the host antibacterial defense systems of the rodent and man and found that these two species had defenses that are very similar and thus provide a good basis for a qualitative extrapolation. Both defenses consist of an aerodynamic filtration system, a fluid layer lining the respiratory membranes, a transport mechanism for removing foreign particles, microorganisms, and pulmonary cells, and immune secretions of lymphocytes and plasma cells. In both rodents and humans, these components act in concert to maintain the lung free of bacteria.

If the animal models are to be used to reflect the toxicological response occurring in humans, then the endpoint for comparison of such studies should be morbidity rather than mortality. A better index of O_3 effect in humans might be the increased prevalence of infectious respiratory illness in the community. Such a comparison may be proper since both mortality from respiratory infections (animals) and morbidity from respiratory infections (humans) can result from a loss in pulmonary defenses (Gardner, 1984). Whether the microorganisms used in the various animal studies are comparable to the organisms responsible for the respiratory infections in a community still requires further investigation.

Ideally, studies of pulmonary host defenses should be performed in man, using epidemiological or volunteer methods of study. Unfortunately, such studies have not been reported yet. Attention must therefore be paid to the results of host-defense experiments conducted with animals.

In the area of host defense of the lung against infection, present knowledge of the physiology, metabolism, and function have come primarily from the study of various animal systems, but it is generally accepted that the basic mechanisms of action of these defense cells and systems function similarly in both animals and man. There are also human data to support this statement, especially in such areas as immunosuppression, ciliostasis, and alveolar macrophages. The effects seen in animals represent alterations in basic biological systems. One can assume that similar alterations in basic defense mechanisms could occur in humans since they possess equivalent pulmonary defense systems. It is understood, however, that different exposure levels may be required to produce similar responses in humans. The concentration of

O₃ at which effects become evident can be influenced by a number of factors, such as preexisting disease, virulence of the infectious agent, dietary factors, concurrent exposure to other pollutants, exercise, or the presence of other environmental stresses, or a combination of these. Thus, one could hypothesize that humans exposed to O₃ could experience effects on host defense mechanisms. At the present time, however, one cannot predict the exact concentration at which effects may occur in man nor the severity of the effects.

The effects of O₃ on host defense mechanisms in experimental animals are summarized in Figure 1-11 and Table 1-15 (see Section 1.8.1 for criteria used in developing this summary).

1.8.3.5 Tolerance. Examination of responses to short-term, repeated exposures to O₃ clearly indicates that with some of the parameters measured, animals have an increased capacity to resist the effects of subsequent exposure. This tolerance persists for varying times, depending on the degree of development of the tolerance. Previous exposure to low concentrations of O₃ will protect against the effects of subsequent exposure to lethal doses and the development of lung edema (Stokinger et al., 1956; Fairchild, 1967; Coffin and Gardner, 1972a; Chow, 1984). The prolongation of mucociliary clearance reported for O₃ can also be eliminated by pre-exposure to a lower concentration (Frager et al., 1979). This effect is demonstrated for a short period of time and is lost as soon as the mucus secretion rate returns to normal. However, not all of the toxic effects of O₃, such as reduced functioning activity of the pulmonary defense system (Gardner et al., 1972); hyperplasia of the type 2 cells (Evans et al., 1971, 1976a,b); increased susceptibility to respiratory disease (Gardner and Graham, 1977); loss of pulmonary enzymatic activity (Chow, 1976, Chow et al., 1976); and inflammatory response (Gardner et al., 1972) can be totally prevented by prior treatment with low levels of O₃. Dungworth et al. (1975) and Castleman et al. (1980) have attempted to explain tolerance by careful examination of the morphological changes that occur with repeated O₃ exposures. These investigators suggest that during continuous exposure to O₃ the injured cells attempt to initiate early repair of the specific lesion. The repair phase results in a reduction of the effect first observed but lasts only for a short time since the recovered cells are as sensitive to re-exposure to O₃ as the pre-exposed counterpart (Plopper et al., 1978). This information is an important observation because it implies that the decrease in susceptibility to O₃ persists only as long as the exposure to O₃ continues. The biochemical studies of Chow et al. (1976) support this conclusion.

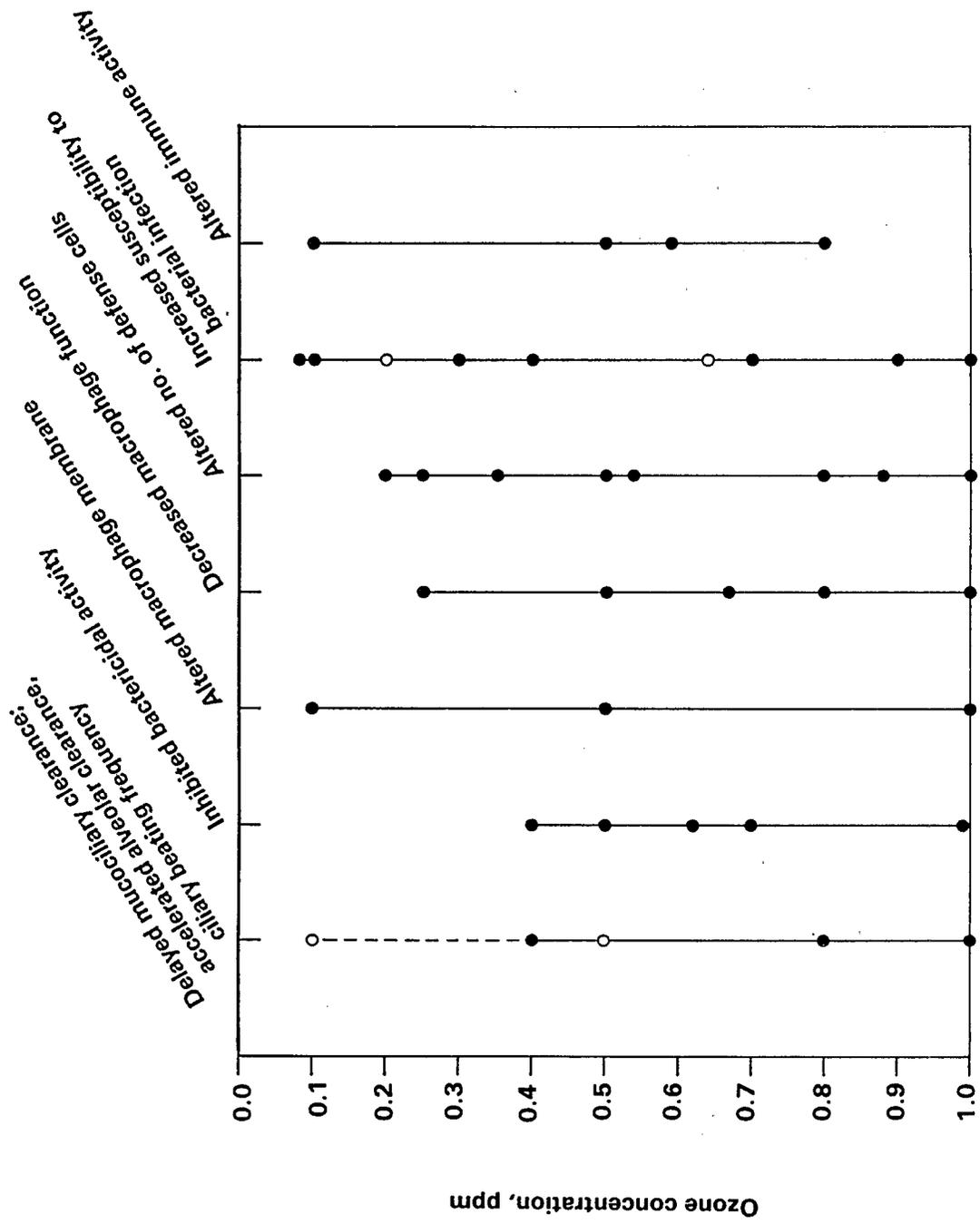


Figure 1-11. Summary of effects of ozone on host defense mechanisms in experimental animals. See Table 1-15 for reference citations of studies summarized here.

TABLE 1-15. SUMMARY TABLE: EFFECTS OF OZONE ON HOST DEFENSE MECHANISMS IN EXPERIMENTAL ANIMALS

Effect/response	O ₃ concentration, ppm	References
Delayed mucociliary clearance; accelerated alveolar clearance, ciliary beating frequency	[0.1]	Grose et al. (1980)
	0.4, 0.8, 1.0	Kenoyer et al. (1981)
	[0.5]	Friberg et al. (1972)
	[0.5], 1.0	Abraham et al. (1980)
	0.8	Phalen et al. (1980)
	1.2	Frager et al. (1979)
Inhibited bactericidal activity	0.4	Coffin and Gardner (1972b)
	0.4	Goldstein et al. (1972)
	0.5	Friberg et al. (1972)
	0.62	Goldstein et al. (1971b)
	0.7	Bergers et al. (1983)
	0.7	Warshauer et al. (1974)
0.99	Goldstein et al. (1971a)	
Altered macrophage membrane	0.1, 1.0	Gardner et al. (1971)
	0.5	Dowell et al. (1970)
	0.5	Hadley et al. (1977)
	0.5, 1.0	Goldstein et al. (1977)
Decreased macrophage function	0.25, 0.5	Hurst et al. (1970)
		Hurst and Coffin (1971)
	0.5	Alpert et al. (1971b)
	0.5, 0.67	Coffin et al. (1968)
	0.5, 0.67	Coffin and Gardner (1972b)
	0.8	Schwartz and Christman (1979)
	1.0	Shingu et al. (1980)
	1.0	McAllen et al. (1981)
Altered no. of defense cells	0.2	Plopper et al. (1979)
	0.2, 0.35, 0.5, 0.8	Dungworth et al. (1975)
	0.2, 0.35	Castleman et al. (1977)
	0.2, 0.5, 0.8	Boorman et al. (1977, 1980)
	0.25	Barry et al. (1983)
	0.5	Zitnik et al. (1978)
	0.5, 0.88	Stephens et al. (1974a)
	0.5	Last et al. (1979)
	0.5, 0.88	Brummer et al. (1977)
	0.5, 0.8	Eustis et al. (1981)
	0.54, 0.88	Freeman et al. (1974)
	0.8	Castleman et al. (1980)
	1.0	Freeman et al. (1973)
1.0	Cavender et al. (1977)	

TABLE 1-15 (continued). SUMMARY TABLE: EFFECTS OF OZONE ON HOST DEFENSE MECHANISMS IN EXPERIMENTAL ANIMALS

Effect/response	O ₃ concentration, ppm	References
Increased susceptibility to infection	0.08	Coffin et al. (1967)
	0.08, 0.1	Miller et al. (1978a)
	0.1	Ehrlich et al. (1977)
	0.1	Aranyi et al. (1983)
	0.1, 0.3	Illing et al. (1980)
	[0.2], 0.4, 0.7	Bergers et al. (1983)
	0.3	Abraham et al. (1982)
	0.5	Wolcott et al. (1982)
	[0.64]	[Sherwood et al. (1984)]
	0.7, 0.9	Coffin and Blommer (1970)
1.0	Thomas et al. (1981b)	
Altered immune activity	0.1	Aranyi et al. (1983)
	0.5, 0.8	Osebold et al. (1979, 1980)
	0.5, 0.8	Gershwin et al. (1981)
	0.59	Campbell and Hilsenroth (1976)
	0.8	Fujimaki et al. (1984)

At this time, there are a number of hypotheses proposed to explain the mechanism of this phenomenon (Mustafa and Tierney, 1978; Schwartz et al., 1976; Mustafa et al., 1977; Berliner et al., 1978; Gertner et al., 1983b; Bhatnagar et al., 1983). Evidence by Nambu and Yokoyama (1983) indicates that although the pulmonary antioxidant system (glutathione peroxidase, glutathione reductase, and glucose-6-phosphate dehydrogenase) may play an active role in defending the lung against ozone, it does not explain the mechanism of tolerance in that the development of tolerance does not coincide with the described biochemical enhancement of the antioxidant system in the lungs of rats.

From this literature, it would appear that tolerance, as seen in animals, may not be the result of any one single biological process, but instead may result from a number of different events, depending on the specific response measured. Tolerance does not imply complete or absolute protection, because continuing injury does still occur, which could potentially lead to nonreversible pulmonary changes.

Tolerance may not be long-lasting. During O₃ exposure, the increase in antioxidant metabolism reaches a plateau and recovery occurs a few days after

exposure ceases. Upon re-exposure, effects observed are similar to those that occurred during the primary exposure (Chow et al., 1976).

1.8.4 Extrapulmonary Effects of Ozone

It is still believed that O_3 , on contact with respiratory system tissue, immediately reacts and thus is not absorbed or transported to extrapulmonary sites to any significant degree. However, several studies suggest that possibly products formed by the interaction of O_3 and respiratory system fluids or tissue can produce effects in lymphocytes, erythrocytes, and serum, as well as in the parathyroid gland, the heart, the liver, and the CNS. Ozone exposure also produces effects on animal behavior that may be caused by pulmonary consequences of O_3 , or by nonpulmonary (CNS) mechanisms. The mechanism by which O_3 causes extrapulmonary changes is unknown. Mathematical models of O_3 dosimetry predict that very little O_3 penetrates to the blood of the alveolar capillaries. Whether these effects result from O_3 or a reaction product of O_3 which penetrates to the blood and is transported is the subject of speculation.

1.8.4.1 Central Nervous System and Behavioral Effects. Ozone significantly affects the behavior of rats during exposure to concentrations as low as $235 \mu\text{g}/\text{m}^3$ (0.12 ppm) for 6 hr. With increasing concentrations of O_3 , further decreases in unspecified motor activity and in operant learned behaviors have been observed (Konigsberg and Bachman, 1970; Tepper et al., 1982; Murphy et al., 1964; and Weiss et al., 1981). Tolerance to the observed decrease in motor activity may occur on repeated exposure. At low O_3 exposure concentrations ($490 \mu\text{g}/\text{m}^3$, 0.25 ppm), an increase in activity is observed after exposure ends. Higher O_3 concentrations ($980 \mu\text{g}/\text{m}^3$, 0.5 ppm) produce a decrease in rodent activity that persists for several hours after the end of exposure (Tepper et al., 1982, 1983).

The mechanism by which behavioral performance is reduced is unknown. Physically active responses appear to enhance the effects of O_3 , although this may be the result of an enhanced minute volume that increases the effective concentration delivered to the lung. Several reports indicate that it is unlikely that animals have reduced physiological capacity to respond, prompting Weiss et al. (1981) to suggest that O_3 impairs the inclination to respond. Two studies indicate that mice will respond to remove themselves from an atmosphere containing greater than $980 \mu\text{g}/\text{m}^3$ (0.5 ppm) (Peterson and Andrews, 1963, Tepper et al., 1983). These studies suggest that the aversive effects

of O_3 may be due to lung irritation. It is unknown whether lung irritation, odor, or a direct effect on the CNS causes change in rodent behavior at lower O_3 concentrations.

1.8.4.2 Cardiovascular Effects. Studies on the effects of O_3 on the cardiovascular system are few, and to date there are no reports of attempts to confirm these studies. The exposure of rats to O_3 alone or in combination with cadmium ($1176 \mu\text{g}/\text{m}^3$, $0.6 \text{ ppm } O_3$) resulted in measurable increases in systolic pressure and heart rate (Revis et al., 1981). No additive or antagonistic response was observed with the combined exposure. Pulmonary capillary blood flow and PaO_2 decreased 30 min following exposure of dogs to $588 \mu\text{g}/\text{m}^3$ (0.3 ppm) of O_3 (Friedman et al., 1983). The decrease in pulmonary capillary blood flow persisted for as long as 24 hr following exposure.

1.8.4.3 Hematological and Serum Chemistry Effects. The data base for the effects of O_3 on the hematological system is extensive and indicates that O_3 or one of its reactive products can cross the blood-gas barrier, causing changes in the circulating erythrocytes (RBC) as well as significant differences in various components of the serum.

Effects of O_3 on the circulating RBCs can be readily identified by examining either morphological and/or biochemical endpoints. These cells are structurally and metabolically well understood and are available through relatively non-invasive methods, which makes them ideal candidates for both human and animal studies. A wide range of structural effects have been reported in a variety of species of animals, including an increase in the fragility of RBCs isolated from monkeys exposed to $1470 \mu\text{g}/\text{m}^3$ (0.75 ppm) of O_3 4 hr/day for 4 days (Clark et al., 1978). A single 4-hr exposure to $392 \mu\text{g}/\text{m}^3$ (0.2 ppm) also caused increased fragility as well as sphering of RBCs of rabbits (Brinkman et al., 1964). An increase in the number of RBCs with Heinz bodies was detected following a 4-hr exposure to $1666 \mu\text{g}/\text{m}^3$ (0.85 ppm). The presence of such inclusion bodies in RBCs is an indication of oxidant stress (Menzel et al., 1975a).

These morphological changes are frequently accompanied by a wide range of biochemical effects. RBCs of monkeys exposed to $1470 \mu\text{g}/\text{m}^3$ (0.75 ppm) of O_3 for 4 days also had a decreased level of glutathione (GSH) and decreased acetylcholinesterase (AChE) activity, an enzyme bound to the RBC membranes. The RBC GSH activity remained significantly lower 4 days postexposure (Clark et al., 1978).

Animals deficient in vitamin E are more sensitive to O_3 . The RBCs from these animals, after being exposed to O_3 , had a significant increase in the activity of GSH peroxidase, pyruvate kinase, and lactic dehydrogenase, but had a decrease in RBC GSH after exposure to $1568 \mu\text{g}/\text{m}^3$ (0.8 ppm) for 7 days (Chow and Kaneko, 1979). Animals with a vitamin E-supplemented diet did not have any changes in glucose-6-phosphate dehydrogenase (G-6-PD), superoxide dismutase, or catalase activities. At a lower level ($980 \mu\text{g}/\text{m}^3$, 0.5 ppm), there were no changes in GSH level or in the activities of GSH peroxidase or GSH reductase (Chow et al., 1975). Menzel et al. (1972) also reported a significant increase in lysis of RBCs from vitamin E-deficient animals after 23 days of exposure to $980 \mu\text{g}/\text{m}^3$ (0.5 ppm). These effects were not observed in vitamin E-supplemented rats. Mice on a vitamin E-supplemented diet and those on a deficient diet showed an increase in G-6-PD activity after an exposure of $627 \mu\text{g}/\text{m}^3$ (0.32 ppm) of O_3 for 6 hr. Decreases observed in AChE activity occurred in both groups (Moore et al., 1980).

Other blood changes are attributed to O_3 . Rabbits exposed for 1 hr to $392 \mu\text{g}/\text{m}^3$ (0.2 ppm) of O_3 showed a significant drop in total blood serotonin (Veninga, 1967). Six- and 10-month exposures of rabbits to $784 \mu\text{g}/\text{m}^3$ (0.4 ppm) of O_3 produced an increase in serum protein esterase and in serum trypsin inhibitor. This latter effect may be a result of thickening of the small pulmonary arteries. The same exposure caused a significant decrease in albumin levels and an increase in alpha and gamma globulins (P'an and Jegier, 1971, 1976; P'an et al., 1972; Jegier, 1973). Chow et al. (1974) reported that the serum lysozyme level of rats increased significantly after 3 days of continuous exposure to O_3 but was not affected when the exposure was intermittent (8 hr/day, 7 days). The O_3 concentration in both studies was $1568 \mu\text{g}/\text{m}^3$ (0.8 ppm) of O_3 .

Short-term exposure to low concentrations of O_3 induced an immediate change in the serum creatine phosphokinase level in mice. In this study, the O_3 doses were expressed as the product of concentration and time. The $C \times T$ value for this effect ranged from 0.4 to 4.0 (Veninga et al., 1981).

A few of the hematological effects observed in animals (i.e., decrease in GSH and AChE activity and the formation of Heinz bodies) following exposure to O_3 have also been seen following in vitro exposure of RBCs from humans (Freeman and Mudd, 1981; Menzel et al., 1975b; Verweij and Van Steveninck, 1981). A common effect observed by a number of investigators is that O_3 inhibits the membrane ATPase activity of RBCs (Koontz and Heath, 1979; Kesner et al., 1979;

Kindya and Chan, 1976; Freeman et al., 1979; Verweij and Van Steveninck, 1980). It has been postulated that this inhibition of ATPase could be related to the spherocytosis and increased fragility of RBCs seen in animal and human cells.

Although these in vitro data are useful in studying mechanisms of action, it is difficult to extrapolate these data to any effects observed in man. Not only is the method of exposure not physiological, but the actual concentration of O₃ reaching the RBC cannot be determined with any accuracy.

1.8.4.4 Cytogenetic and Teratogenic Effects. Uncertainty still exists regarding possible reproductive, teratogenic, and mutational effects of exposure to ozone. Based on various in vitro data, a number of chromosomal effects of ozone have been described for isolated cultured cell lines, human lymphocytes, and microorganisms (Fetner, 1962; Hamelin et al., 1977a,b, Hamelin and Chung, 1975a,b, 1978; Scott and Leshner, 1963; Erdman and Hernandez, 1982; Guerrero et al., 1979; Dubeau and Chung, 1979, 1982). The interpretation, relevance, and predictive values of such studies to human health are questionable since (1) the concentrations used were many-fold greater than what is found in the ambient air (see Chapter 10); (2) extrapolation of in vitro exposure concentrations to human exposure dose is not yet possible; and (3) direct exposure of isolated cells to ozone is highly artifactual since it bypasses all the defenses of the host that would normally be functioning in protecting the individual from the inhaled gas. Furthermore, the direct exposure of isolated cells in vitro to ozone may result in chemical reactions between ozone and culture media that might not occur in vivo.

Important questions still exist regarding in vivo cytogenetic effects of ozone in rodents and humans. Zelac et al. (1971a,b) reported chromosomal abnormalities in peripheral leukocytes of hamsters exposed to O₃ (0.2 ppm). Combined exposures to ozone and radiation (227-233 rads) produced an additive effect on the number of chromosome breaks in peripheral leukocytes. These specific findings were not confirmed by Gooch et al. (1976) or by Tice et al. (1978), but sufficient differences in the various experimental protocols make a direct comparison difficult. The latter group did report significant increases in the number of chromatid deletions and achromatic lesions resulting from exposure to 0.43 ppm ozone.

Because the volume of air inspired during pregnancy is significantly enhanced, the pregnant animal may be at greater risk to low levels of ozone

exposure. Early studies on the possible teratogenic effects of ozone have suggested that exposures as low as 0.2 ppm can reduce infant survival rate and cause unlimited incisor growth (Brinkman et al., 1964; Veninga, 1967). Kavlock et al. (1979, 1980) found that pregnant rats exposed to 1.0 and 1.49 ppm ozone showed a significant increase in embryo resorption rate, slower growth, slower development of righting reflexes, and delayed grooming and rearing behavior, but no increase in neonatal mortality was observed.

1.8.4.5 Other Extrapulmonary Effects. A series of studies was conducted to show that O_3 increases drug-induced sleeping time in a number of species of animals (Gardner et al., 1974; Graham, 1979; Graham et al., 1981, 1982a,b, 1983, 1985). At $1960 \mu\text{g}/\text{m}^3$ (1.0 ppm), effects were observed after 1, 2, and 3 days of exposure. As the concentration of O_3 was reduced, increasing numbers of daily 3-hr exposures were required to produce a significant effect. At the lowest concentration studied ($196 \mu\text{g}/\text{m}^3$, 0.1 ppm), the increase was observed at days 15 and 16 of exposure. It appears that this effect is not specific to the strain of mouse or to the three species of animals tested, but it is sex-specific, with females being more susceptible. Recovery was complete within 24 hr after exposure. Although a number of mechanistic studies have been conducted, the reason for this effect on pentobarbital-induced sleeping time is not known. It has been hypothesized that some common aspect related to liver drug metabolism is quantitatively reduced (Graham et al., 1983).

Several investigators have attempted to elucidate the involvement of the endocrine system in O_3 toxicity. Most of these studies were designed to investigate the hypothesis that the survival rate of mice and rats exposed to lethal concentrations of O_3 could be increased by use of various thyroid blocking agents or by thyroidectomy. To follow up these findings, Clemons and Garcia (1980a,b) and Clemons and Wei (1984) investigated the effects of a 24-hr exposure to $1960 \mu\text{g}/\text{m}^3$ (1.0 ppm) of O_3 on the hypothalamo-pituitary-thyroid system of rats. These three organs regulate the function of each other through various hormonal feedback mechanisms. Ozone caused decreases in serum concentration of thyroid stimulating hormone (TSH), in circulating thyroid hormones (T_3 and T_4) and in protein-bound iodine. No alterations were observed in many other hormone levels measured. Thyroidectomy prevented the effect of O_3 on TSH and T_4 and hypophysectomy prevented effects on T_4 , unless the animals were supplemented with T_4 in their drinking water. The thyroid gland itself was altered (e.g., edema) by O_3 . The authors hypothesized that O_3 alters serum binding of these hormones.

The extrapulmonary effects of ozone in experimental animals are summarized in Figure 1-12 and Table 1-16. Criteria used in developing the summary were presented in Section 1.8.1.

1.8.5 Interaction of Ozone With Other Pollutants

Combined exposure studies in laboratory animals have produced varied results, depending upon the pollutant combination evaluated and the measured variables. Additive and/or possibly synergistic effects of O_3 exposure in combination with NO_2 have been described for increased susceptibility to bacterial infection (Ehrlich et al., 1977, 1979; Ehrlich, 1980, 1983), morphological lesions (Freeman et al., 1974), and increased antioxidant metabolism (Mustafa et al., 1984). Additive or possibly synergistic effects from exposure to O_3 and H_2SO_4 have also been reported for host defense mechanisms (Gardner et al., 1977; Last and Cross, 1978; Grose et al., 1982), pulmonary sensitivity (Osebold et al. 1980), and collagen synthesis (Last et al., 1983), but not for morphology (Cavender et al., 1977; Moore and Schwartz, 1981). Mixtures of O_3 and $(NH_4)_2SO_4$ had synergistic effects on collagen synthesis and morphometry, including percentage of fibroblasts (Last et al., 1983, 1984a).

Combining O_3 with other particulate pollutants produces a variety of responses, depending on the endpoint measured. Mixtures of O_3 , $Fe_2(SO_4)_3$, H_2SO_4 , and $(NH_4)_2SO_4$ produced the same effect on clearance rate as exposure to O_3 alone. However, when measuring changes in host defenses, the combination of O_3 with NO_2 and $ZnSO_4$ or O_3 with SO_2 and $(NH_4)_2SO_4$ produced enhanced effects that can not be attributed to O_3 only.

However, since these issues are complex, they must be addressed experimentally using exposure regimens for combined pollutants that are more representative of ambient ratios of peak concentrations, frequency, duration, and time intervals between events.

The interactive effects of O_3 with other pollutants are summarized in Figure 1-13 and Table 1-17.

1.8.6 Effects of Other Photochemical Oxidants

There have been far too few controlled toxicological studies with the other oxidants to permit any sound scientific evaluation of their contribution to the toxic action of photochemical oxidant mixtures. When the effects seen after exposure to O_3 and PAN are examined and compared, it is obvious that the

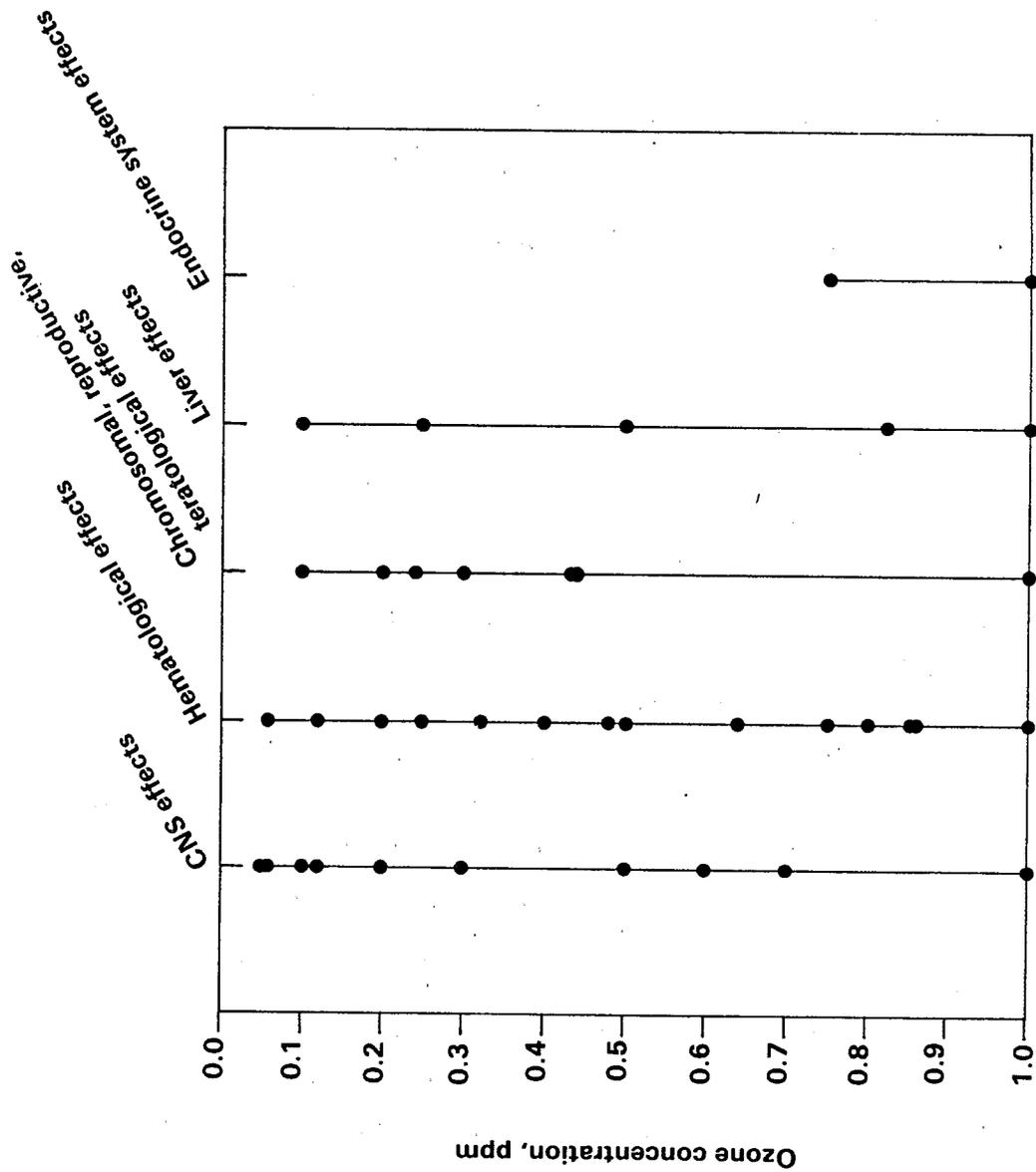


Figure 1-12. Summary of extrapulmonary effects of ozone in experimental animals. See Table 1-16 for reference citations of studies summarized here.

TABLE 1-16. SUMMARY TABLE: EXTRAPULMONARY EFFECTS OF OZONE
IN EXPERIMENTAL ANIMALS

Effect/response	O ₃ concentration, ppm	References
CNS effects	0.05, 0.5	Konigsberg and Bachman (1970)
	0.1 - 1.0	Weiss et al. (1981)
	0.12 - 1.0	Tepper et al. (1982)
	0.2, 0.3, 0.5, 0.7	Murphy et al. (1964)
	0.5	Tepper et al. (1983)
	0.5	Reynolds and Chaffee (1970)
	0.5	Xintaras et al. (1966)
	0.6	Peterson and Andrews (1963)
	1.0	Fletcher and Tappel (1973)
	1.0	Trams et al. (1972)
	Hematological effects	0.06, 0.12, 0.48
0.2		Brinkman et al. (1964)
0.2, 1.0		Veninga (1967, 1970)
		Veninga et al. (1981)
0.25, 0.32, 0.5		Moore et al. (1980; 1981a,b)
0.4		Jegier (1973)
0.4		P'an and Jegier (1972, 1976)
0.5		Menzel et al. (1972)
0.64		Larkin et al. (1983)
0.75		Clark et al. (1978)
0.8		Chow and Kaneko (1979)
0.8		Chow et al. (1974)
0.85		Menzel et al. (1975a)
0.86		Schlipkötter and Bruch (1973)
1.0		Dorsey et al. (1983)
1.0		Mizoguchi et al. (1973)
1.0	Christiansen and Giese (1954)	
Chromosomal, reproductive, teratological effects	0.1	Brinkman et al. (1964)
	0.2	Veninga (1967)
	0.24, 0.3	Zelac et al. (1971a)
	0.43	Tice et al. (1978)
	0.44	Kavlock et al. (1979)
	1.0	Kavlock et al. (1980)
Liver effects	0.1, 0.25, 0.5, 1.0	Graham (1979)
		Graham et al. (1981, 1982a,b)
	0.82	Veninga et al. (1981)
	1.0	Gardner et al. (1974)
Endocrine system effects	0.75	Atwal and Wilson (1974)
	0.75	Atwal et al. (1975)
	0.75	Atwal and Pemsingh (1981, 1984)
	0.75	Pemsingh and Atwal (1983)
	1.0	Clemons and Garcia (1980a,b)
	1.0	Clemons and Wei (1984)

Increased pulmonary lesions
 Increased pulmonary sensitivity
 and O₂ consumption
 Increased antioxidant metabolism
 Altered mucus secretion
 Increased collagen synthesis
 to respiratory irritability
 Altered upper respiratory
 clearance mechanisms

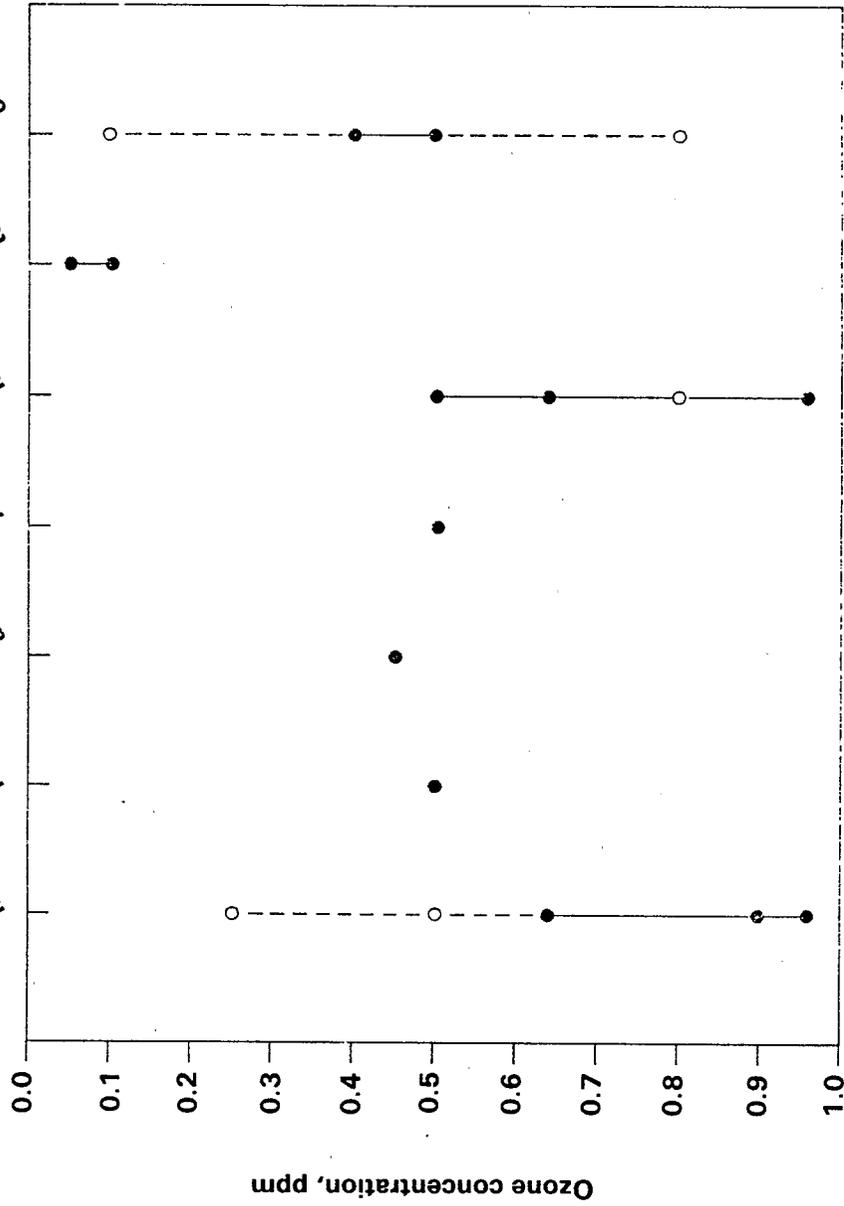


Figure 1-13. Summary of effects in experimental animals exposed to ozone combined with other pollutants. See Table 1-17 for reference citations of studies summarized here.

TABLE 1-17. SUMMARY TABLE: INTERACTION OF OZONE WITH OTHER POLLUTANTS IN EXPERIMENTAL ANIMALS

Effect/response	Pollutant concentrations	References
Increased pulmonary lesions	[0.25 ppm O ₃ + 2.5 ppm NO ₂]	Freeman et al. (1974)
	[0.5 ppm O ₃ + 1 mg/m ³ H ₂ SO ₄]	Moore and Schwartz (1981)
	[0.5 ppm O ₃ + 10 mg/m ³ H ₂ SO ₄	Cavender et al. (1978)
	0.64, 0.96 ppm O ₃ + 5 mg/m ³ (NH ₄) ₂ SO ₄	Last et al. (1984a)
	0.9 ppm O ₃ + 0.9 ppm NO ₂	Freeman et al. (1974)
Increased pulmonary sensitivity	1.2 ppm O ₃ + 5 mg/m ³ (NH ₄) ₂ SO ₄	Last et al. (1983)
	0.5 ppm O ₃ + 1 mg/m ³ H ₂ SO ₄	Osebold et al. (1980)
Increased anti-oxidant metabolism and O ₂ consumption	0.45 ppm O ₃ + 4.8 ppm NO ₂	Mustafa et al. (1984)
Altered mucus secretion	0.5 ppm O ₃ + 1.1 mg/m ³ H ₂ SO ₄	Last and Cross (1978); Last and Kaizu (1980)
Increased collagen synthesis	[0.5], [0.8], 1.5 ppm O ₃ + 5 mg/m ³ (NH ₄) ₂ SO ₄	Last et al. (1983)
	0.5 ppm O ₃ + 1 mg/m ³ H ₂ SO ₄	Last et al. (1983)
	0.64, 0.96 ppm O ₃ + 5 mg/m ³ (NH ₄) ₂ SO ₄	Last et al. (1984a)
Increased susceptibility to respiratory infections	0.05 ppm O ₃ + 3760 µg/m ³ (NH ₄) ₂ SO ₄	Ehrlich et al. (1977, 1979); Ehrlich (1980)
	0.05 ppm O ₃ + 100-400 µg/m ³ NO ₂ + 1.5 mg/m ³ ZnSO ₄	Ehrlich (1983)
	0.1 ppm O ₃ + 0.9 mg/m ³ H ₂ SO ₄ (sequential exposure)	Gardner et al. (1977)
	0.1 ppm O ₃ + 4.8 mg/m ³ H ₂ SO ₄	Grose et al. (1982)
	0.1 ppm O ₃ + 940 µg/m ³ NO ₂	Ehrlich (1980)
	0.1 ppm O ₃ + 13.2 mg/m ³ SO ₂ + 1.0 mg/m ³ (NH ₄) ₂ SO ₄	Aranyi et al. (1983)

TABLE 1-17 (continued). SUMMARY TABLE: INTERACTION OF OZONE WITH OTHER POLLUTANTS

Effect/response	Pollutant concentrations	References
Altered upper respiratory clearance mechanisms	[0.1 ppm O ₃ + 1.1 mg/m ³ H ₂ SO ₄] (sequential exposure)	Grose et al. (1980)
	0.4 ppm O ₃ + 7.0 ppm NO ₂	Goldstein et al. (1974)
	0.5 ppm O ₃ + 3 mg/m ³ H ₂ SO ₄	Last and Cross (1978)
	[0.8 ppm O ₃ + 3.5 mg/m ³ {Fe ₂ (SO ₄) ₃ + H ₂ SO ₄ + (NH ₄) ₂ SO ₄ }]	Phalen et al. (1980)

test animals must be exposed to concentrations of PAN much greater than those needed with O₃ to produce a similar effect on lethality, behavior modification, morphology, or significant alterations in host pulmonary defense system (Campbell et al., 1967; Dungworth et al., 1969; Thomas et al., 1979, 1981a). The concentrations of PAN required to produce these effects are many times greater than what has been measured in the atmosphere (0.047 ppm).

Similarly, most of the investigations reporting H₂O₂ toxicity have involved concentrations much higher than those found in the ambient air, or the investigations were conducted by using various in vitro techniques for exposure. Very limited information is available on the health significance of inhalation exposure to gaseous H₂O₂. Because H₂O₂ is highly soluble, it is generally assumed that it does not penetrate into the alveolar regions of the lung but is instead deposited on the surface of the upper airways (Last et al., 1982). Unfortunately, there have not been studies designed to look for possible effects in this region of the respiratory tract.

A few in vitro studies have reported cytotoxic, genotoxic, and biochemical effects of H₂O₂ when using isolated cells or organs (Stewart et al., 1981; Bradley et al., 1979; Bradley and Erickson, 1981; Speit et al., 1982; MacRae and Stich, 1979). Although these studies can provide useful data for studying possible mechanisms of action, it is not yet possible to extrapolate these responses to those that might occur in the mammalian system.

Field and epidemiological studies have shown that human health effects from exposure to ambient mixtures of oxidants and other airborne pollutants can produce human health effects (Chapter 12). Few such studies have been conducted with laboratory animals, because testing and measuring of such mixtures is not only complicated, but extremely costly. In these studies, the investigators attempted to simulate the photochemical reaction products produced under natural conditions and to define the cause-effect relationship.

Exposure to complex mixtures of oxidants plus the various components found in UV-irradiated auto exhaust indicates that certain effects, such as histopathological changes, increase in susceptibility to infection, a variety of altered pulmonary functional activities were observed in this oxidant atmosphere which was not reported in the nonirradiated exhaust (Murphy et al., 1963; Murphy, 1964; Nakajima et al., 1972; Hueter et al., 1966). Certain other biological responses were observed in both treatment groups, including a decrease in spontaneous activity, a decrease in infant survival rate, fertility, and certain pulmonary functional abnormalities (Hueter et al., 1966; Boche and Quilligan, 1960; Lewis et al., 1967).

Dogs exposed to UV-irradiated auto exhaust containing oxidants either with or without SO_x showed significant pulmonary functional abnormalities that had relatively good correlation with structural changes (Hyde et al., 1978; Gillespie, 1980; Lewis et al., 1974). There were no significant differences in the magnitude of the response in these two treatment groups, indicating that oxidant gases and SO_x did not interact in any synergistic or additive manner.

1.9 CONTROLLED HUMAN STUDIES OF THE EFFECTS OF OZONE AND OTHER PHOTOCHEMICAL OXIDANTS

A number of important controlled studies discussed in this chapter have reported significant decrements in pulmonary function associated with O_3 exposure (Table 1-18). In most of the studies reported, greatest attention has been accorded decrements in $FEV_{1.0}$, as this variable represents a summation of changes in both volume and resistance. While this is true, it must be pointed out that for exposure concentrations critical to the standard-setting process (i.e., ≤ 0.3 ppm O_3), the observed decrements in $FEV_{1.0}$ primarily reflect FVC decrements of similar magnitude, with little or no contribution from changes in resistance.

TABLE 1-18. SUMMARY TABLE: CONTROLLED HUMAN EXPOSURE TO OZONE

Ozone ^a concentration µg/m ³ ppm	Measurement ^{b,c} method	Exposure duration	Activity ^d level (V _E)	Observed effects(s)	No. and sex of subjects	Reference
HEALTHY ADULT SUBJECTS AT REST						
627 1960	MAST, NBKI	2 hr	R	Specific airway resistance increased with acetylcholine challenge; subjective symptoms in 3/14 at 0.32 ppm and 8/14 at 1.0 ppm.	13 male 1 female	König et al., 1980
980	CHEM, NBKI	2 hr	R (10)	Decrement in forced expiratory volume and flow.	40 male (divided into four exposure groups)	Folinsbee et al., 1978
980 1470	CHEM, NBKI	2 hr	R (8)	Decrement in forced expiratory volume and flow.	8 male 7 female	Horvath et al., 1979
EXERCISING HEALTHY ADULTS						
235 353 470 588 784	CHEM, UV	2.5 hr	IE (65) @ 15-min intervals	Decrement in forced expiratory volume and flow suggested at 0.12 ppm with larger decrements at ≥ 0.18 ppm; respiratory frequency and specific airway resistance increased and tidal volume decreased at ≥ 0.24 ppm; coughing reported at all concentrations, pain and shortness of breath at ≥ 0.24 ppm.	135 male (divided into six exposure groups)	McDonnell et al., 1983
314 470 627	UV, UV	1 hr	CE (57)	Small decrements in forced expiratory volume at 0.16 ppm with larger decrements at ≥ 0.24 ppm; lower-respiratory symptoms increased at ≥ 0.16 ppm.	42 male 8 female (competitive bicyclists)	Avol et al., 1984
353 470 588 784	CHEM, UV	2.5 hr	IE (65) @ 15-min intervals	Individual responses to O ₃ were highly reproducible for periods as long as 10 months; large intersubject variability in response due to intrinsic responsiveness to O ₃ .	32 male	McDonnell et al., 1985a
392 686	UV, UV	1 hr (mouth-piece)	IE (77.5) @ variable competitive intervals CE (77.5)	Decrement in forced expiratory volume and flow with IE and CE; subjective symptoms increased with O ₃ concentration and may limit performance; respiratory frequency increased and tidal volume decreased with CE.	10 male (distance runners)	Adams and Schelegle, 1983

TABLE 1-18 (continued). SUMMARY TABLE: CONTROLLED HUMAN EXPOSURE TO OZONE

Ozone ^a concentration µg/m ³ ppm	Measurement ^{b,c} method	Exposure duration	Activity ^d level (V _E)	Observed effects(s)	No. and sex of subjects	Reference
392 823 980	UV, UV	2 hr	IE (30 for male, 18 for female subjects) @ 15-min intervals	Repeated daily exposure to 0.2 ppm did not affect response at higher exposure concentrations (0.42 or 0.50 ppm); large inter-subject variability but individual pulmonary function responses were highly reproducible.	8 male 13 female	Gliner et al., 1983
392 490	UV, UV	2 hr	IE (68) (4) 14-min periods	Large intersubject variability in response; significant concentration-response relationships for pulmonary function and respiratory symptoms.	20 male	Kulle et al., 1985
412	UV, UV	1 hr	CE (81)	Decrement in forced expiratory volume and flow; subjective symptoms may limit performance.	6 male 1 female (distance cyclists)	Folinsbee et al., 1984
490	UV, UV	1 hr	CE (63)	Increased responsiveness to O ₃ lasts for 24 hr, may persist in some subjects for 48 hr, but is generally lost within 72 hr.	19 male 7 female	Folinsbee and Horvath, 1986
588 980	CHEM, NBKI	2 hr	R (10), IE (31, 50, 67) @ 15-min intervals	Decrement in forced expiratory volume and flow; the magnitude of the change was related to O ₃ concentration and V _E . Total lung capacity and inspiratory capacity decreased with IE (50, 67); no change in airway resistance or residual volume even at highest IE (67). No significant changes in pulmonary function were observed at 0.1 ppm.	40 male (divided into four exposure groups)	Folinsbee et al., 1978
725 980 1470	MAST, NBKI	2 hr	R (11) & IE (29) @ 15-min intervals	Good correlation between dose (concentration x V _E) and decrement in forced expiratory volume and flow.	20 male 8 female (divided into six exposure groups)	Silverman et al., 1976
784	UV, NBKI	2 hr	IE (2xR) @ 15-min intervals	Specific airway resistance increased with histamine challenge; no changes were observed at concentrations of 0.2 ppm.	12 male 7 female (divided into three exposure groups)	Dimeo et al., 1981
784	CHEM, NBKI & MAST, NBKI	3 hr	IE (4-5xR)	Decrement in forced expiratory volume and SG was greatest on the 2nd of 5 exposure days; attenuated response by the 4th day of exposure.	10 male 4 female	Färrell et al., 1979

TABLE 1-18 (continued). SUMMARY TABLE: CONTROLLED HUMAN EXPOSURE TO OZONE

Ozone ^a concentration $\mu\text{g}/\text{m}^3$ ppm	Measurement ^{b,c} method	Exposure duration	Activity ^d level (V_E)	Observed effects(s)	No. and sex of subjects	Reference
784 0.4	CHEM, UV	3 hr	IE (4-5xR) for 15 min	Decrement in forced expiratory volume was greatest on the 2nd of 5 exposure days; attenuation of response occurred by the 5th day and persisted for 4 to 7 days. Enhanced bronchoreactivity with methacholine on the first 3 days; attenuation of response occurred by the 4th and 5th day and persisted for > 7 days.	13 male 11 female (divided into two exposure groups)	Kulle et al., 1982
784 0.4	CHEM, UV	2.5 hr	IE (7I) @ 15-min intervals	Atropine pretreatment prevented the increased R_{E} observed with O_3 exposure, partially blocked the decreased forced expiratory flow, but did not prevent the O_3 -induced decreases in FVC and TLC, changes in exercise ventilation, or respiratory symptoms.	8 male	Beckett et al. 1985
823 0.42	UV, UV	2 hr	IE (30)	Decrement in forced expiratory volume and flow greatest on the 2nd of 5 exposure days; attenuation of response occurred by the 5th day and persisted for < 14 days with considerable intersubject variability.	24 male	Horvath et al., 1981
882 0.45	UV, UV	2 hr	IE (27) @ 20-min intervals	Increased responsiveness to O_3 was found with a 2nd O_3 challenge given 48 hr after the initial exposure.	1 male 5 female	Bedi et al., 1985
921 0.47	UV, NBKI	2 hr	IE (3xR)	Decrement in forced expiratory volume and flow greatest on the 2nd of 4 exposure days; attenuation of response occurred by the 4th day and persisted for 4 days.	8 male 3 female	Linn et al., 1982b
980 0.5	MAST, NBKI	6 hr	IE (44) for two 15-min periods	Small decrements in forced expiratory volume and specific airway conductance.	19 male 1 female	Kerr et al., 1975
1176 0.6	UV, NBKI	2 hr (noseclip)	IE (2xR) @ 15-min intervals	Specific airway resistance increased in 7 nonatopic subjects with histamine and methacholine and in 9 atopic subjects with histamine.	11 male 5 female (divided by history of atopy)	Holtzman et al., 1979
1470 0.75	MAST, NBKI	2 hr	IE (2xR) @ 15-min intervals	Decrements in spirometric variables (20%-55%); residual volume and closing capacity increased.	12 male	Hazucha et al., 1973

TABLE 1-18. (continued) SUMMARY TABLE: CONTROLLED HUMAN EXPOSURE TO OZONE

Ozone ^a concentration µg/m ³ ppm	Measurement ^{b,c} method	Exposure duration	Activity ^d level (V _E)	Observed effects(s)	No. and sex of subjects	Reference
EXERCISING HEALTHY CHILDREN						
235	CHEM, UV	2.5 hr	IE (39) @15-min intervals	Small decrements in forced expiratory volume, persisting for 24 hr. No subjective symptoms.	23 male (8-11 yrs)	McDonnell et al., 1985b,c
ADULT ASTHMATICS						
392	CHEM, NBKI	2 hr	IE (2xR) @ 15-min intervals	No significant changes in pulmonary function. Small changes in blood biochemistry. Increase in symptom frequency reported.	20 male 2 female	Linn et al., 1978
490	CHEM, NBKI	2 hr	R	No significant changes in pulmonary function.	5 males 12 female	Silverman, 1979
ADOLESCENT ASTHMATICS						
235	UV	1 hr (mouthpiece)	R	No significant changes in pulmonary function or symptoms.	4 male 6 female (11-18 yrs)	Koenig et al., 1985
SUBJECTS WITH CHRONIC OBSTRUCTIVE LUNG DISEASE						
235	UV, NBKI	1 hr	IE (variable) @ 15-min intervals	No significant changes in forced expiratory performance or symptoms. Decreased arterial oxygen saturation during exercise was observed.	18 male 7 female	Linn et al., 1982a
353 490	UV, NBKI	1 hr	IE (variable) @ 15-min intervals	No significant changes in forced expiratory performance or symptoms. Group mean arterial oxygen saturation was not altered by O ₃ exposure.	15 male 13 female	Linn et al., 1983
392 588	CHEM, NBKI	2 hr	IE (28) for 7.5 min each half hour	No significant changes in pulmonary function or symptoms. Decreased arterial oxygen saturation during exposure to 0.2 ppm.	13 male	Solic et al., 1982 Kehrl et al., 1983, 1985
784	UV, UV	3 hr	IE (4-5xR) for 15 min	Small decreases in FVC and FEV _{3.0} .	17 male 3 female	Kulle et al., 1984

^aRanked by lowest observed effect level.

^bMeasurement method: MAST = KI-Coulometric (Mast meter); CHEM = gas phase chemiluminescence; UV = ultraviolet photometry.

^cCalibration method: NBKI = neutral buffered potassium iodide; UV = ultraviolet photometry.

^dMinute ventilation reported in L/min or as a multiple of resting ventilation. R = rest; IE = intermittent exercise; CE = continuous exercise.

Results from studies of at-rest exposures to O_3 have demonstrated decrements in forced expiratory volumes and flows occurring at and above $980 \mu\text{g}/\text{m}^3$ (0.5 ppm) of O_3 (Folinsbee et al., 1978; Horvath et al., 1979). Airway resistance is not clearly affected at these O_3 concentrations. At or below $588 \mu\text{g}/\text{m}^3$ (0.3 ppm) of O_3 , changes in pulmonary function do not occur during at rest exposure (Folinsbee et al., 1978), but the occurrence of some O_3 -induced pulmonary symptoms has been suggested (König et al., 1980).

With moderate intermittent exercise at a \dot{V}_E of 30 to 50 L/min, decrements in forced expiratory volumes and flows have been observed at and above $588 \mu\text{g}/\text{m}^3$ (0.30 ppm) of O_3 (Folinsbee et al., 1978). With heavy intermittent exercise ($\dot{V}_E = 65$ L/min), pulmonary symptoms are present and decrements in forced expiratory volumes and flows are suggested to occur following 2-hr exposures to $235 \mu\text{g}/\text{m}^3$ (0.12 ppm) of O_3 (McDonnell et al., 1983). Symptoms are present and decrements in forced expiratory volumes and flows definitely occur at 314 to $470 \mu\text{g}/\text{m}^3$ (0.16 to 0.24 ppm) of O_3 following 1 hr of continuous heavy exercise at a \dot{V}_E of 57 L/min (Avol et al., 1984) or very heavy exercise at a \dot{V}_E of 80 to 90 L/min (Adams and Schelegle, 1983; Folinsbee et al., 1984) and following 2 hr of intermittent heavy exercise at a \dot{V}_E of 65 to 68 L/min (McDonnell et al., 1983; Kulle et al., 1985). Airway resistance is only modestly affected with moderate exercise (Kerr et al., 1975; Farrell et al., 1979) or even with heavy exercise while exposed at levels as high as $980 \mu\text{g}/\text{m}^3$ (0.50 ppm) O_3 (Folinsbee et al., 1978; McDonnell et al., 1983). Increased f_R and decreased V_T , while maintaining the same \dot{V}_E , occur with prolonged heavy exercise when exposed at 392 to $470 \mu\text{g}/\text{m}^3$ (0.20 to 0.24 ppm) of O_3 (McDonnell et al., 1983; Adams and Schelegle, 1983). While an increase in RV has been reported to result from exposure to $1470 \mu\text{g}/\text{m}^3$ (0.75 ppm) of O_3 (Hazucha et al., 1973), changes in RV have not been observed following exposures to $980 \mu\text{g}/\text{m}^3$ (0.50 ppm) of O_3 or less, even with heavy exercise (Folinsbee et al., 1978). Decreases in TLC and IC have been observed to result from exposures to $980 \mu\text{g}/\text{m}^3$ (0.50 ppm) of O_3 or less, with moderate and heavy exercise (Folinsbee et al., 1978).

Recovery of the lung from the effects of O_3 exposure consists of return of pulmonary function (FVC, FEV_1 , and SR_{aw}) to preexposure levels. The time course of this recovery is related to the magnitude of the O_3 -induced functional decrement (i.e., recovery from small decrements is rapid). Despite apparent functional recovery of most subjects within 24 hr, an enhanced responsiveness

to a second O_3 challenge may persist in some subjects for up to 48 hr (Bedi et al., 1985; Folinsbee and Horvath, 1986).

Group mean decrements in pulmonary function can be predicted with some degree of accuracy when expressed as a function of effective dose of O_3 , the simple product of O_3 concentration, \dot{V}_E , and exposure duration (Silverman et al., 1976). The relative contribution of these variables to pulmonary decrements is greater for O_3 concentration than for \dot{V}_E . A greater degree of predictive accuracy is obtained if the contribution of these variables is appropriately weighted (Folinsbee et al., 1978). However, several additional factors make the interpretation of prediction equations more difficult. There is considerable intersubject variability in the magnitude of individual pulmonary function responses to O_3 (Horvath et al., 1981; Gliner et al., 1983; McDonnell et al., 1983; Kulle et al., 1985). Individual responses to a given O_3 concentration have been shown to be quite reproducible (Gliner et al., 1983; McDonnell et al., 1985a), indicating that some individuals are consistently more responsive to O_3 than are others. No information is available to account for these differences. Considering the great variability in individual pulmonary responses to O_3 exposure, prediction equations that only use some form of effective dose are not adequate for predicting individual responses to O_3 .

In addition to overt changes in pulmonary function, enhanced nonspecific bronchial reactivity has been observed following exposures to O_3 concentrations $\geq 588 \mu\text{g}/\text{m}^3$ (0.3 ppm) (Holtzman et al., 1979; König et al., 1980; Dimeo et al., 1981). Exposure to $392 \mu\text{g}/\text{m}^3$ (0.2 ppm) of O_3 with intermittent light exercise does not affect nonspecific bronchial reactivity (Dimeo et al., 1981).

Changes in forced expiratory volumes and flows resulting from O_3 exposure reflect reduced maximal inspiratory position (inspiratory capacity) (Folinsbee et al., 1978). These changes, as well as altered ventilatory control and the occurrence of respiratory symptoms, most likely result from sensitization or stimulation of airway irritant receptors (Folinsbee et al., 1978; Holtzman et al., 1979; McDonnell et al., 1983). The increased airways resistance observed following O_3 exposure is probably initiated by a similar mechanism. Different efferent pathways have been proposed (Beckett et al., 1985) to account for the lack of correlation between individual changes in SR_{aw} and FVC (McDonnell et al., 1983). The increased responsiveness of airways to histamine and methacholine following O_3 exposure most likely results from an O_3 -induced increase in airways permeability or from an alteration of smooth muscle characteristics.

Decrements in pulmonary function were not observed for adult asthmatics exposed for 2 hours at rest (Silverman, 1979) or with intermittent light exercise (Linn et al., 1978) to O_3 concentrations of $490 \mu\text{g}/\text{m}^3$ (0.25 ppm) and less. Likewise, no significant changes in pulmonary function or symptoms were found in adolescent asthmatics exposed for 1 hr at rest to $235 \mu\text{g}/\text{m}^3$ (0.12 ppm) of O_3 (Koenig et al., 1985). Although these results indicate that asthmatics are not more responsive to O_3 than are healthy subjects, experimental-design considerations in reported studies suggest that this issue is still unresolved. For patients with COLD performing light to moderate intermittent exercise, no decrements in pulmonary function are observed for 1- and 2-hr exposures to O_3 concentrations of $588 \mu\text{g}/\text{m}^3$ (0.30 ppm) and less (Linn et al., 1982a, 1983; Solic et al., 1982; Kehrl et al., 1983, 1985) and only small decreases in forced expiratory volume are observed for 3-hr exposures of chronic bronchitics to $804 \mu\text{g}/\text{m}^3$ (0.41 ppm) (Kulle et al., 1984). Small decreases in SaO_2 have also been observed in some of these studies but not in others; therefore, interpretation of these decreases and their clinical significance is uncertain.

Many variables have not been adequately addressed in the available clinical data. Information derived from O_3 exposure of smokers and nonsmokers is sparse and somewhat inconsistent, perhaps partly because of undocumented variability in smoking histories. Although some degree of attenuation appears to occur in smokers, all current results should be interpreted with caution. Further and more precise studies are required to answer the complex problems associated with personal and ambient pollutant exposures. Possible age differences in response to O_3 have not been explored systematically. Young adults usually provide the subject population, and where subjects of differing age are combined, the groups studied are often too small in number to make adequate statistical comparisons. Children (boys, aged 8 to 11 yr) have been the subjects in only one study (McDonnell et al., 1985b) and nonstatistical comparison with adult males exposed under identical conditions has indicated that the effects of O_3 on lung spirometry were very similar (McDonnell et al., 1985c). While a few studies have investigated sex differences, they have not conclusively demonstrated that men and women respond differently to O_3 , and consideration of differences in pulmonary capacities have not been adequately taken into account. Environmental conditions such as heat and relative humidity may enhance subjective symptoms and physiological impairment following O_3 exposure, but the results so far indicate that the effects are no more than additive.

In addition, there may be considerable interaction between these variables that may result in modification of interpretations made based on available information.

During repeated daily exposures to O_3 , decrements in pulmonary function are greatest on the second exposure day (Farrell et al., 1979; Horvath et al., 1981; Kulle et al., 1982; Linn et al., 1982b); thereafter, pulmonary responsiveness to O_3 is attenuated with smaller decrements on each successive day than on the day before until the fourth or fifth exposure day when small decrements or no changes are observed. Following a sequence of repeated daily exposures, this attenuated pulmonary responsiveness persists for 3 (Kulle et al., 1982; Linn et al., 1982b) to 7 (Horvath et al., 1981) days. Repeated daily exposures to a given low effective dose of O_3 does not affect the magnitude of decrements in pulmonary function resulting from exposure at a higher effective dose of O_3 (Gliner et al., 1983).

There is some evidence suggesting that exercise performance may be limited by exposure to O_3 . Decrements in forced expiratory flow occurring with O_3 exposure during prolonged heavy exercise ($\dot{V}_E = 65$ to 81 L/min) along with increased f_R and decreased V_T might be expected to produce ventilatory limitations at near maximal exercise. Results from exposure to ozone during high exercise levels (68 to 75 percent of max $\dot{V}O_2$) indicate that discomfort associated with maximal ventilation may be an important factor in limiting performance (Adams and Schelegle, 1983; Folinsbee et al., 1984). However, there is not enough data available to adequately address this issue.

No consistent cytogenetic or functional changes have been demonstrated in circulating cells from human subjects exposed to O_3 concentrations as high as 784 to $1176 \mu\text{g}/\text{m}^3$ (0.4 to 0.6 ppm). Chromosome or chromatid aberrations would therefore be unlikely at lower O_3 levels. Limited data have indicated that O_3 can interfere with biochemical mechanisms in blood erythrocytes and sera but the physiological significance of these studies is questionable.

No significant enhancement of respiratory effects has been consistently demonstrated for combined exposures of O_3 with SO_2 , NO_2 , and sulfuric acid or particulate aerosols or with multiple combinations of these pollutants. Most of the available studies with other photochemical oxidants have been limited to studies on the effects of peroxyacetyl nitrate (PAN) on healthy young and middle-aged males during intermittent moderate exercise. No significant effects were observed at PAN concentrations of 0.25 to 0.30 ppm, which are

higher than the daily maximum concentrations of PAN reported for relatively high oxidant areas (0.047 ppm). One study (Drechsler-Parks et al., 1984) suggested a possible simultaneous effect of PAN and O_3 ; however, there are not enough data to evaluate the significance of this effect. Further studies are also required to evaluate the relationships between O_3 and the more complex mix of pollutants found in the natural environment.

1.10 FIELD AND EPIDEMIOLOGICAL STUDIES OF THE EFFECTS OF OZONE AND OTHER PHOTOCHEMICAL OXIDANTS

Field and epidemiological studies offer a unique view of health effects research because they involve the real world, i.e., the study of human populations in their natural setting. These studies have attendant limitations, however, that must be considered in a critical evaluation of their results. One major problem in singling out the effects of one air pollutant in field studies of morbidity in populations has been the interference of other environmental variables that are critical. Limitations of epidemiological research on the health effects of oxidants include: interference by other air pollutants or interactions between oxidants and other pollutants; meteorological factors such as temperature and relative humidity; proper exposure assessments, including determination of individual activity patterns and adequacy of number and location of pollutant monitors; difficulty in identifying oxidant species responsible for observed effects; and characteristics of the populations such as smoking habits and socioeconomic status.

The most quantitatively useful information of the effects of acute exposure to photochemical oxidants presented in this chapter comes from the field studies of symptoms and pulmonary function. These studies offer the advantage of studying the effects of naturally-occurring, ambient air on a local subject population using the methods and better experimental control typical of controlled-exposure studies. In addition, the measured responses in ambient air can be compared to clean, filtered air without pollutants or to filtered air containing artificially-generated concentrations of O_3 that are comparable to those found in the ambient environment. As shown in Table 1-19, studies by Linn et al. (1980, 1983) and Avol et al. (1983, 1984, 1985a,b,c) have demonstrated that respiratory effects in Los Angeles area residents are related to O_3 concentration and level of exercise. Such effects include: pulmonary

TABLE 1-19. SUMMARY TABLE: ACUTE EFFECTS OF OZONE AND OTHER PHOTOCHEMICAL OXIDANTS IN FIELD STUDIES WITH A MOBILE LABORATORY^a

Mean ozone concentration $\mu\text{g}/\text{m}^3$ ppm	Measurement ^{b,c} method	Exposure duration	Activity ^d level (V_E)	Observed effect(s)	No. of subjects	Reference
282 0.144	UV, UV	1 hr	CE(32)	Small significant decreases in FVC (-2.1%), FEV _{0.75} (-4.0%), FEV _{1.0} (-4.2%), and PEFR (-4.4%) relative to control with no recovery during a 1-hr post-exposure rest; no significant increases in symptoms.	59 healthy adolescents (12-15 yr)	Avol et al., 1985a,b
300 0.153	UV, UV	1 hr	CE(53)	Mild increases in lower respiratory symptom scores and significant decreases in FEV ₁ (-5.3%) and FVC; mean changes in ambient air were not statistically different from those in purified air containing 0.16 ppm O ₃ .	50 healthy adults (competitive bicyclists)	Avol et al., 1984, 1985c
306 0.156	UV, NBKI	1 hr	CE(38)	No significant changes for total symptom score or forced expiratory performance in normals or asthmatics; however, FEV ₁ remained low or decreased further (-3%) 3 hr after ambient air exposure in asthmatics.	48 healthy adults 50 asthmatic adults	Linn et al., 1983; Avol et al., 1983
323 0.165	UV, NBKI	1 hr	CE(42)	Small significant decreases in FEV ₁ (-3.3%) and FVC with no recovery during a 1-hr postexposure rest; TLC decreased and ΔN_2 increased slightly.	60 "healthy" adults (7 were asthmatic)	Linn et al., 1983; Avol et al., 1983
341 0.174	UV, NBKI	2 hr	IE(2 x R) @ 15-min intervals	Increased symptom scores and small significant decreases in FEV ₁ (-2.4%), FVC, PEFR, and TLC in both asthmatic and healthy subjects however; 25/34 healthy subjects were allergic and "atypically" reactive to O ₃ .	34 "healthy" adults 30 asthmatic adults	Linn et al., 1980, 1983

^aRanked by lowest observed effect level for O₃ in ambient air.

^bMeasurement method: UV = ultraviolet photometry.

^cCalibration method: UV = ultraviolet photometry standard; NBKI = neutral buffered potassium iodide.

^dMinute ventilation reported in L/min or as a multiple of resting ventilation. CE = continuous exercise, IE = intermittent exercise.

function decrements seen at O_3 concentrations of $282 \mu\text{g}/\text{m}^3$ (0.144 ppm) in exercising healthy adolescents; and increased respiratory symptoms and pulmonary function decrements seen at O_3 concentrations of $300 \mu\text{g}/\text{m}^3$ (0.153 ppm) in heavily exercising athletes and at O_3 concentrations of $341 \mu\text{g}/\text{m}^3$ (0.174 ppm) in lightly exercising normal and asthmatic subjects. The light exercise level is probably the type most likely to occur in the exposed population of Los Angeles. The observed effects are typically mild, and generally no substantial differences were seen in asthmatics versus persons with normal respiratory health, although symptoms lasted for a few hours longer in asthmatics. Many of the normal subjects, however, had a history of allergy and appeared to be more sensitive to O_3 than "non-allergic" normal subjects. Concerns raised about the relative contribution to untoward effects in these field studies by pollutants other than O_3 have been diminished by direct comparative findings in exercising athletes (Avol et al., 1984, 1985c) showing no differences in response between chamber exposures to oxidant-polluted ambient air with a mean O_3 concentration of $294 \mu\text{g}/\text{m}^3$ (0.15 ppm) and purified air containing a controlled concentration of generated O_3 at $314 \mu\text{g}/\text{m}^3$ (0.16 ppm). The relative importance of exercise level, duration of exposure, and individual variations in sensitivity in producing the observed effects remains to be more fully investigated, although the results from field studies relative to those factors are consistent with results from controlled human exposure studies (Chapter 10).

Studies of the effects of both acute and chronic exposures have been reported in the epidemiological literature on photochemical oxidants. Investigative approaches comparing communities with high O_3 concentrations and communities with low O_3 concentrations have usually been unsuccessful, often because actual pollutant levels have not differed enough during the study, or other important variables have not been adequately controlled. The terms "oxidant" and "ozone" and their respective association with health effects are often unclear. Moreover, information about the measurement and calibration methods used is often lacking. Also, as epidemiological methods improve, the incorporation of new key variables into the analyses is desirable, such as the use of individual exposure data (e.g., from the home and workplace). Analyses employing these variables are lacking for most of the community studies evaluated.

Studies of effects associated with acute exposure that are considered to be qualitatively useful for standard-setting purposes include those on irritative symptoms, pulmonary function, and aggravation of existing respiratory disease. Reported effects on the incidence of acute respiratory illness and on physician, emergency room, and hospital visits are not clearly related with acute exposure to ambient O_3 or oxidants and, therefore, are not useful for deriving health effects criteria for standard-setting purposes. Likewise, no convincing association has been demonstrated between daily mortality and daily oxidant concentrations; rather, the effect correlates most closely with elevated temperature.

Studies on the irritative effects of O_3 have been complicated by the presence of other photochemical pollutants and their precursors in the ambient environment and by the lack of adequate control for other pollutants, meteorological variables, and non-environmental factors in the analysis. Although O_3 does not cause the eye irritation normally associated with smog, several studies in the Los Angeles basin have indicated that eye irritation is likely to occur in ambient air when oxidant levels are about 0.10 ppm. Qualitative associations between oxidant levels in the ambient air and symptoms such as eye and throat irritation, chest discomfort, cough, and headache have been reported at >0.10 ppm in both children and young adults (Hammer et al., 1974; Makino and Mizoguchi, 1975). Discomfort caused by irritative symptoms may be responsible for the impairment of athletic performance reported in high school students during cross-country track meets in Los Angeles (Wayne et al., 1967; Herman, 1972) and is consistent with the evidence from field studies (Section 11.2.1) and from controlled human exposure studies (Section 10.4) indicating that exercise performance may be limited by exposure to O_3 . Although several additional studies have shown respiratory irritation apparently related to exposure to ambient O_3 or oxidants in community populations, none of these epidemiological studies provide satisfactory quantitative data on acute respiratory illnesses.

Epidemiological studies in children and young adults suggest an association of decreased peak flow and increased airway resistance with acute ambient air exposures to daily maximum 1-hr O_3 concentrations ranging from 20 to $274 \mu\text{g}/\text{m}^3$ (0.01 to 0.14 ppm) over the entire study period (Lippmann et al., 1983; Lebowitz et al., 1982, 1983, 1985; Lebowitz, 1984; Bock et al., 1985; Liroy et al., 1985). None of these studies by themselves can provide satisfactory

quantitative data on acute effects of O_3 because of methodological problems along with the confounding influence of other pollutants and environmental conditions in the ambient air. The aggregation of individual studies, however, provides reasonably good evidence for an association between ambient O_3 exposure and acute pulmonary function effects. This association is strengthened by the consistency between the findings from the epidemiological studies and the results from the field studies in exercising adolescents (Avol et al., 1985a,b) which have shown small decreases in forced expiratory volume and flow at $282 \mu\text{g}/\text{m}^3$ (0.144 ppm) of O_3 in the ambient air; and with the results from the controlled human exposure studies in exercising children which have shown small decrements in forced expiratory volume at $235 \mu\text{g}/\text{m}^3$ (0.12 ppm) of O_3 (Section 10.2.9.2).

In studies of exacerbation of asthma and chronic lung diseases, the major problems have been the lack of information on the possible effects of medications, the absence of records for all days on which symptoms could have occurred, and the possible concurrence of symptomatic attacks resulting from the presence of other environmental conditions in ambient air. For example, Whittemore and Korn (1980) and Holguin et al. (1985) found small increases in the probability of asthma attacks associated with previous attacks, decreased temperature, and with incremental increases in oxidant and O_3 concentrations, respectively. Lebowitz et al. (1982, 1983, 1985) and Lebowitz (1984) showed effects in asthmatics, such as decreased peak expiratory flow and increased respiratory symptoms, that were related to the interaction of O_3 and temperature. All of these studies have questionable effects from other pollutants, particularly inhalable particles. There have been no consistent findings of symptom aggravation or changes in lung function in patients with chronic lung diseases other than asthma.

Only a few prospective studies have been reported on morbidity, mortality, and chromosomal effects from chronic exposure to O_3 or other photochemical oxidants. The lack of quantitative measures of oxidant exposures seriously limits the usefulness of many population studies of morbidity and mortality for standards-setting purposes. Most of these long-term studies have employed average annual levels of photochemical oxidants or have involved broad ranges of pollutants; others have used a simple high-oxidant/low-oxidant dichotomy. In addition, these population studies are also limited by their inability to control for the effects of other factors that can potentially contribute to

the development and progression of respiratory disease over long periods of time. Thus, insufficient information is available in the epidemiological literature on possible exposure-response relationships between ambient O_3 or other photochemical oxidants and the prevalence of chronic lung disease or the rates of chronic disease mortality. None of the epidemiological studies investigating chromosomal changes have found any evidence that ambient O_3 or oxidants affect the peripheral lymphocytes of the exposed population.

1.11 EVALUATION OF HEALTH EFFECTS DATA FOR OZONE AND OTHER PHOTOCHEMICAL OXIDANTS

1.11.1 Health Effects in the General Human Population

Controlled human studies of at-rest exposures to O_3 lasting 2 to 4 hr have demonstrated decrements in forced expiratory volume and flow occurring at and above 0.5 ppm of O_3 (Chapter 10). Airway resistance was not significantly changed at these O_3 concentrations. Breathing O_3 at rest at concentrations < 0.5 ppm did not significantly impair pulmonary function although the occurrence of some O_3 -related pulmonary symptoms has been suggested in a number of studies.

One of the principal modifiers of the magnitude of response to O_3 is minute ventilation (\dot{V}_E), which increases proportionately with increases in exercise work load. Adjustment by the respiratory system to an increased work load is characterized by increased frequency and depth of breathing. Consequent increases in \dot{V}_E not only increase the overall volume of inhaled pollutant, but the increased tidal volume may lead to a higher concentration of ozone in the lung regions most sensitive to ozone. These processes are further enhanced at high work loads ($\dot{V}_E > 35$ L/min), since the mode of breathing changes at that \dot{V}_E from nasal to oronasal.

Statistically significant decrements in forced expiratory volume and flow are generally observed in healthy adult subjects (18 to 45 yr old) after 1 to 3 hr of exposure as a function of the level of exercise performed and the ozone concentration inhaled during the exposure. Group mean data pooled from numerous controlled human exposure (Chapter 10) and field (Chapter 11) studies indicate that, on average, pulmonary function decrements occur:

1. At ≥ 0.37 ppm O_3 with light exercise ($\dot{V}_E \leq 23$ L/min);
2. At ≥ 0.30 ppm O_3 with moderate exercise ($\dot{V}_E = 24-43$ L/min);

3. At ≥ 0.24 ppm O_3 with heavy exercise ($\dot{V}_E = 44-63$ L/min); and
4. At ≥ 0.18 ppm O_3 with very heavy exercise ($\dot{V}_E \geq 64$ L/min).

Note, however, that data from specific individual studies indicate that pulmonary function decrements occur with very heavy exercise in healthy adults at 0.15 to 0.16 ppm O_3 (Avol et al., 1984) and suggest that such effects may occur in healthy adults at levels as low as 0.12 ppm O_3 (McDonnell et al., 1983). Also, pulmonary function decrements have been observed in children and adolescents at concentrations of 0.12 and 0.14 ppm O_3 with heavy exercise (McDonnell et al., 1985b; Avol et al., 1985a). At the lower concentrations (0.12 to 0.15 ppm), the average changes in lung function are generally small (≤ 5 percent) and are a matter of controversy in regard to their medical significance.

In the majority of the studies reported, 15-min intermittent exercise alternated with 15-min rest was employed for the duration of the exposure. Figure 1-14 uses the pulmonary function measurement FEV_1 to illustrate the effects of intermittent exercise and O_3 concentration during 2-hr exposures. As noted above, larger decrements in lung function occur at higher exercise levels and at higher O_3 concentrations. The maximum reponse to O_3 exposure can be observed within 5 to 10 min following the end of each exercise period. Other measures of spirometric pulmonary function (e.g., FVC and $FEF_{25-75\%}$) are consistent with FEV_1 and, therefore, are not depicted here. It is important to note, however, that any predictions of average pulmonary function responses to O_3 only apply under the specific set of exposure conditions at which these data were derived.

Continuous exercise equivalent in duration to the sum of intermittent exercise periods at comparable ozone concentrations (0.2 to 0.4 ppm) and minute ventilation (60 to 80 L/min) seems to elicit greater changes in pulmonary function (Folinsbee et al., 1984; Avol et al., 1984, 1985c) but the differences between intermittent and continuous exercise are not clearly established. More experimental data are needed to make any quantitative evaluation of the differences in effects induced by these two modes of exercise.

Functional recovery, at least from a single exposure with exercise, appears to progress in two phases: during the initial rapid phase, lasting between 1 and 3 hr, pulmonary function improves more than 50 percent; this is followed by a much slower recovery that is usually completed in most subjects

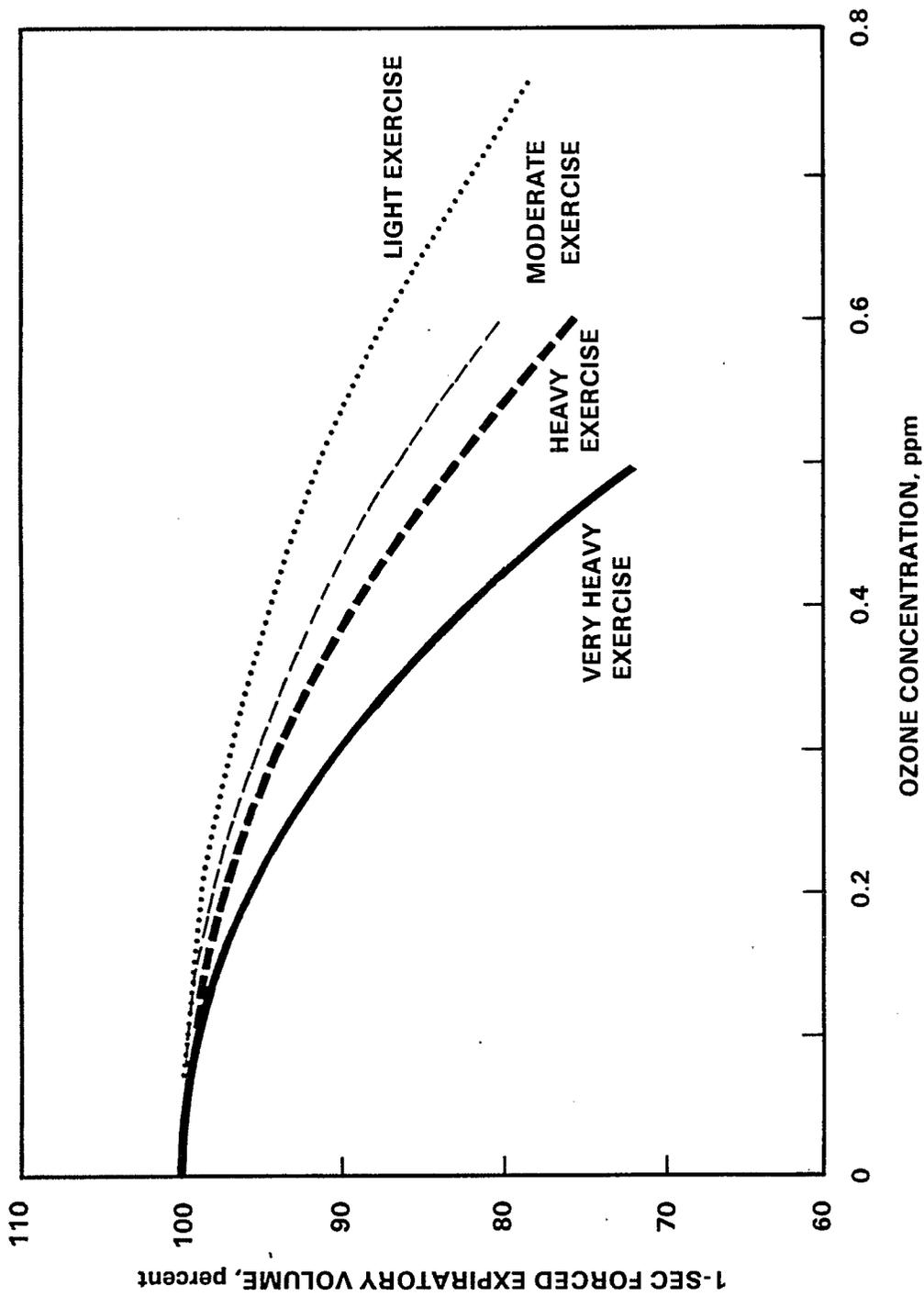


Figure 1-14. Group mean decrements in 1-sec forced expiratory volume during 2-hr ozone exposures with different levels of intermittent exercise: light ($\dot{V}_E \leq 23$ L/min); moderate ($\dot{V}_E = 24-43$ L/min); heavy ($\dot{V}_E = 44-63$ L/min); and very heavy ($\dot{V}_E \geq 64$ L/min). (Concentration-response curves are taken from Figures 12-2 through 12-5 in Chapter 12, Volume V.)

within 24 hr. In some individuals, an enhanced responsiveness to a second O_3 challenge may persist for up to 48 hr (Bedi et al., 1985; Folinsbee and Horvath, 1986). In addition, despite apparent functional recovery, other regulatory systems may still exhibit abnormal responses when stimulated; e.g., airway hyperreactivity may persist for days.

Group mean changes may be useful for making statistical inferences about homogeneous populations, but they are not adequate for describing difference in responsiveness to O_3 among individuals. Even in well-controlled experiments on an apparently homogeneous group of healthy subjects, physiological responses to the same work and pollutant loads will vary widely among individuals (Horvath et al., 1981; Gliner et al., 1983; McDonnell et al., 1983; Kulle et al., 1985). Despite large intersubject variability, individual responsiveness to a given O_3 concentration is quite reproducible (Gliner et al., 1983; McDonnell et al., 1985a). Some individuals, therefore, are consistently more responsive to O_3 than are others. The term "responders" has been used to describe the 5 to 20 percent of the studied population that is most responsive to O_3 exposure. There are no clearly established criteria to define this group of subjects. Likewise, there are no known specific factors responsible for increased or decreased responsiveness to O_3 . Characterization of individual responses to O_3 , however, is pertinent since it permits the assessment of a segment of the general population that is potentially at-risk to O_3 exposure (see Section 12.7.3) although statistical treatment of these data is still rudimentary and their validity is open to question.

A close association has been observed between the occurrence of respiratory symptoms and changes in pulmonary function in adults acutely exposed in environmental chambers to O_3 (Chapter 10) or to ambient air containing O_3 as the predominant pollutant (Chapter 11). This association holds for both the time-course and magnitude of effects. Studies on children and adolescents exposed to O_3 or ambient air containing O_3 under similar conditions have found no significant increases in symptoms despite significant changes in pulmonary function (Avol et al., 1985a,b; McDonnell et al., 1985b,c). Epidemiological studies of exposure to ambient photochemical pollution are of limited use for quantifying exposure-response relationships for O_3 because they have not adequately controlled for other pollutants, meteorological variables, and non-environmental factors in the data analysis. Eye irritation, for example, one of the most common complaints associated with photochemical pollution, is

not characteristic of clinical exposures to O_3 , even at concentrations several times higher than any likely to be encountered in ambient air. There is limited qualitative evidence to suggest that at low concentrations of O_3 , other respiratory and nonrespiratory symptoms, as well, are more likely to occur in populations exposed to ambient air pollution than in subjects exposed in chamber studies (Chapter 11).

Discomfort caused by irritative symptoms may be responsible for the impairment of athletic performance reported in high school students during cross-country track meets in Los Angeles (Chapter 11). Only a few controlled-exposure studies, however, have been designed to examine the effects of O_3 on exercise performance (Chapter 10). In one study, light intermittent exercise ($\dot{V}_E = 20-25$ L/min) at a high O_3 concentration (0.75 ppm) reduced postexposure maximal exercise capacity by limiting maximal oxygen consumption; submaximal oxygen consumption changes were not significant. The extent of ventilatory and respiratory metabolic changes observed during or following the exposure appears to have been related to the magnitude of pulmonary function impairment. Whether such changes are consequent to respiratory discomfort (i.e., symptomatic effects) or are the result of changes in lung mechanics or both is still unclear and needs to be elucidated.

Environmental conditions such as heat and relative humidity may alter subjective symptoms and physiological impairment associated with O_3 exposure. Modification of the effects of O_3 by these factors may be attributed to increased ventilation associated with elevated body temperature but there may also be an independent effect of elevated body temperature on pulmonary function (e.g., VC).

Numerous additional factors have the potential for altering responsiveness to ozone. For example, children and older individuals may be more responsive than young adults. Other factors such as gender differences (at any age), personal habits such as smoking, nutritional deficiencies, or differences in immunologic status may predispose individuals to susceptibility to ozone. In addition, social, cultural, or economic factors may be involved. Those actually known to alter sensitivity, however, are few, largely because they have not been examined adequately to determine definitively their effects on sensitivity to O_3 . The following briefly summarizes what is actually known from the data regarding the importance of these factors (see Section 12.3.3 for details):

1. Age. Although changes in growth and development of the lung with age have been postulated as one of many factors capable of modifying responsiveness to O_3 , sufficient numbers of studies have not been performed to provide any sound conclusions for effects of different age groups on responsiveness to O_3 .

2. Sex. Sex differences in responsiveness to ozone have not been adequately studied. Lung function of women, as assessed by changes in $FEV_{1.0}$, might be affected more than that of men under similar exercise and exposure conditions, but the possible differences have not been tested systematically.

3. Smoking Status. Differences between smokers and nonsmokers have been studied often, but the smoking histories of subjects are not documented well. There is some evidence, however, to suggest that smokers may be less responsive to O_3 than nonsmokers.

4. Nutritional Status. Antioxidant properties of vitamin E in preventing ozone-initiated peroxidation in vitro are well demonstrated and their protective effects in vivo are clearly demonstrated in rats and mice. No evidence indicates, however, that man would benefit from increased vitamin E intake relative to ambient ozone exposures.

5. Red Blood Cell Enzyme Deficiencies. There have been too few studies performed to document reliably that individuals with a hereditary deficiency of glucose-6-phosphate dehydrogenase may be at-risk to significant hematological effects from O_3 exposure. Even if O_3 or a reactive product of O_3 -tissue interaction were to penetrate the red blood cell after in vivo exposure, it is unlikely that any depletion of glutathione or other reducing compounds would be of functional significance for the affected individual.

Successive daily brief exposures of healthy human subjects to O_3 (<0.7 ppm for approximately 2 hr) induce a typical temporal pattern of response (Chapter 10, Section 10.3). Maximum functional changes that occur after the first or second exposure day become progressively attenuated on each of the subsequent days. By the fourth day of exposure, the average effects are not different from those observed following control (air) exposure. Individuals need between 3 and 7 days of exposure to develop full attenuation, with more sensitive subjects requiring more time. The magnitude of a peak response to O_3 appears to be directly related to O_3 concentration. It is not known how variations in the length or frequency of exposure will modify the time course of this altered

responsiveness. In addition, concentrations of O_3 that have no detectable effect appear not to invoke changes in response to subsequent exposures at higher O_3 concentrations. Full attenuation, even in ozone-sensitive subjects, does not persist for more than 3 to 7 days after exposure in most individuals, while partial attenuation might persist for up to 2 weeks. Although the severity of symptoms is generally related to the magnitude of the functional response, partial attenuation of symptoms appears to persist longer, for up to 4 weeks after exposure.

Whether populations exposed to photochemical air pollution develop at least partial attenuation is unknown. No epidemiological studies have been designed to test this hypothesis and additional information is required from controlled laboratory studies before any sound conclusions can be made.

Ozone toxicity, in both humans and laboratory animals, may be mitigated through altered responses at the cellular and/or subcellular level. At present, the mechanisms underlying altered responses are unclear and the effectiveness of such mitigating factors in protecting the long-term health of the individual against ozone is still uncertain. A growing body of experimental evidence suggests the involvement of vagal sensory receptors in modulating the acute responsiveness to ozone. It is highly probable that most of the decrements in lung volume reported to result from exposures of greatest relevance to standard-setting (≤ 0.3 ppm O_3) are caused by the inhibition of maximal inspiration rather than by changes in airway diameter. None of the experimental evidence, however, is definitive and additional research is needed to elucidate the precise mechanism(s) associated with ozone exposure.

1.11.2 Health Effects in Individuals with Preexisting Disease

Currently available evidence indicates that individuals with preexisting disease respond to O_3 exposure to a similar degree as normal, healthy subjects. Patients with chronic obstructive lung disease and/or asthma have not shown increased responsiveness to O_3 in controlled human exposure studies, but there is some indication from epidemiological studies that asthmatics may be symptomatically and possibly functionally more responsive than healthy individuals to ambient air exposures. Appropriate inclusion and exclusion criteria for selection of these subjects, however, especially better clinical diagnoses validated by pulmonary function, must be considered before their responsiveness to O_3 can be adequately determined. None of these factors has been sufficiently studied in relation to O_3 exposures to give definitive answers.

1.11.3 Extrapolation of Effects Observed in Animals to Human Populations

Animal experiments on a variety of species have demonstrated increased susceptibility to bacterial respiratory infections following O_3 exposure. Thus, it could be hypothesized that humans exposed to O_3 could experience decrements in their host defenses against infection. At the present time, however, these effects have not been studied in humans exposed to O_3 .

Animal studies have also reported a number of extrapulmonary responses to O_3 , including cardiovascular, reproductive, and teratological effects, along with changes in endocrine and metabolic function. The implications of these findings for human health are difficult to judge at the present time. In addition, central nervous system effects, alterations in red blood cell morphology and enzymatic activity, as well as cytogenetic effects on circulating lymphocytes, have been observed in laboratory animals following exposure to O_3 . While similar effects have been described in circulating cells from human subjects exposed to high concentrations of O_3 , the results were either inconsistent or of questionable physiological significance (Section 12.3.8). It is not known, therefore, if extrapulmonary responses would be likely to occur in humans when exposure schedules are used that are representative of exposures that the population at large might actually experience.

Despite wide variations in study techniques and experimental designs, acute and subchronic exposures of animals to levels of ozone < 0.5 ppm produce remarkably similar types of responses in all species examined. A characteristic ozone lesion occurs at the junction of the conducting airways and the gas-exchange regions of the lung after acute O_3 exposure. Dosimetry model simulations predict that the maximal tissue dose of O_3 occurs in this region of the lung. Continuation of the inflammatory process during longer O_3 exposures is especially important since it appears to be correlated with increased airway resistance, increased lung collagen content, and remodeling of the centriacinar airways, suggesting the development of distal airway narrowing. No convincing evidence of emphysema in animals chronically exposed to O_3 has yet been published, but centriacinar inflammation has been shown to occur.

Since substantial animal data exist for O_3 -induced changes in lung structure and function, biochemistry, and host defenses, it is conceivable that man may experience more types of effects from exposure to ozone than have been established in human clinical studies. It is important to note, however, that the risks to man from breathing ambient levels of ozone cannot fully be determined until quantitative extrapolations of animal results can be made.

1.11.4 Health Effects of Other Photochemical Oxidants and Pollutant Mixtures

Controlled human studies have not consistently demonstrated any modification of respiratory effects for combined exposures of O_3 with SO_2 , NO_2 , CO, or H_2SO_4 and other particulate aerosols. Ozone alone is considered to be responsible for the observed effects of those combinations or of multiple mixtures of these pollutants. Combined exposure studies in laboratory animals have produced varied results, depending upon the pollutant combination evaluated, the exposure design, and the measured variables (Section 12.6.3). Thus, no definitive conclusions can be drawn from animal studies of pollutant interactions. There have been far too few controlled toxicological studies with other oxidants, such as peroxyacetyl nitrate or hydrogen peroxide, to permit a sound scientific evaluation of their contribution to the toxic action of photochemical oxidant mixtures. There is still some concern, however, that combinations of oxidant pollutants with other pollutants may contribute to the symptom aggravation and decreased lung function described in epidemiological studies on individuals with asthma and in children and young adults. For this reason, the effects of interaction between inhaled oxidant gases and other environmental pollutants on the lung need to be systematically studied using exposure regimens that are more closely representative of ambient air ratios of peak concentrations, frequency, duration, and time intervals between events.

1.11.5 Identification of Potentially At-Risk Groups

Despite uncertainties that may exist in the data, it is possible to identify the groups that may be at potential risk from exposure to ozone, based on known health effects, activity patterns, personal habits, and actual or potential exposures to ozone.

The first group that appears to be at potential risk from exposure to ozone is that group of the general population characterized as having pre-existing respiratory disease. Available data on actual differences in responsiveness between these and healthy members of the general population indicate that under the exposure conditions studied to date, individuals with preexisting disease are as responsive to ozone as healthy individuals. Nevertheless, two primary considerations place individuals with preexisting respiratory disease among groups at potential risk from exposure to ozone. First, it must be noted that concern with triggering untoward reactions has necessitated the use of low concentrations and low exercise levels in most studies on subjects

with mild, but not severe, preexisting disease. Therefore, few or no data on responses at higher concentrations, at higher exercise levels, and in subjects with more severe disease states are available for comparison with responses in healthy subjects. Thus, definitive data on the modification by preexisting disease of responses to ozone are not available. Second, however, it must be emphasized that in individuals with already compromised pulmonary function, the decrements in function produced by exposure to ozone, while similar to or even the same as those experienced by normal subjects, represent a further decline in volumes and flows that are already diminished. It is possible that such declines may impair further the ability to perform normal activities. In individuals with preexisting diseases such as asthma or allergies, increases in symptoms upon exposure to ozone, above and beyond symptoms seen in the general population, may also impair or further curtail the ability to function normally.

The second group at potential special risk from exposure to ozone consists of the general population of normal, healthy individuals. Two specific factors place members of the general population at potential risk from exposure to ozone. First unusual responsiveness to ozone has been observed in some individuals ("responders"), not yet characterized medically except by their response to ozone, who experience greater decrements in lung function from exposure to ozone than the average response of the groups studied. It is not known if "responders" are a specific population subgroup or simply represent the upper 5 to 20 percent of the ozone response distribution. As yet no means of determining in advance those members of the general population who are "responders" has been devised. Second, data presented in this chapter underscore the importance of exercise in the potentiation of effects from exposure to ozone. Thus, the general population potentially at risk from exposure to ozone includes those individuals whose activities out of doors, whether vocational or avocational, result in increases in minute ventilation, which is the most prominent modifier of response to ozone.

Other biological and nonbiological factors have the potential for influencing responses to ozone. Data remain inconclusive at the present, however, regarding the importance of age, gender, and other factors in influencing response to ozone. Thus, at the present time, no other groups are thought to be biologically predisposed to increased sensitivity to ozone. It must be emphasized, however, that the final identification of those effects that are considered "adverse" and the final identification of "at-risk" groups are both the domain of the Administrator of the U.S. Environmental Protection Agency.

1.12 REFERENCES

1.12.1 References for Introduction

- U.S. Environmental Protection Agency (1982a) Air quality criteria for oxides of nitrogen. Research Triangle Park, NC: U.S. Environmental Protection Agency; EPA report no. EPA-600/8-82-026.
- U.S. Environmental Protection Agency (1982b) Air quality criteria for particulate matter and sulfur oxides. Research Triangle Park, NC: U.S. Environmental Protection Agency; EPA report no. EPA-600/8-82-029a,b,c,d.
- U.S. Code (1982) Clean Air Act, §108, air quality criteria and control techniques. U.S.C. 42: §7408.

1.12.2 References for Properties, Chemistry, and Transport of Ozone and Other Photochemical Oxidants and Their Precursors

- Aikin, A. C.; Herman, J. R.; Maier, E. J. R.; McQuillan, C. J. (1983) Influence of peroxyacetyl nitrate (PAN) on odd nitrogen in the troposphere and lower stratosphere. *Planet. Space Sci.* 31: 1075-1082.
- Altshuller, A. P. (1975) Evaluation of oxidant results at CAMP sites in the United States. *J. Air Pollut. Control Assoc.* 25: 19-24.
- Altshuller, A. P. (1983) Review: natural volatile organic substances and their effect on air quality in the United States. *Atmos. Environ.* 17: 2131-2165.
- Altshuller, A. P. (1986) Review paper: the role of nitrogen oxides in nonurban ozone formation in the planetary boundary layer over N America, W Europe and adjacent areas of ocean. *Atmos. Environment.* 20: 245-268.
- Altshuller, A. P.; Bufalini, J. J. (1971) Photochemical aspects of air pollution: a review. *Environ. Sci. Technol.* 5: 39-64.
- Atkinson, R. (1985) Kinetics and mechanisms of the gas phase reactions of the hydroxyl radical with organic compounds under atmospheric conditions. *Chem. Rev.*: in press.
- Atkinson, R.; Aschmann, S. M. (1984) Rate constants for the reactions of O_3 and OH radicals with a series of alkynes. *Int. J. Chem. Kinet.* 16: 259-268.
- Atkinson, R.; Carter, W. P. L. (1984) Kinetics and mechanisms of the reactions of ozone with organic compounds in the gas phase. *Chem. Rev.* 84: 437-470.
- Atkinson, R.; Darnall, K. R.; Lloyd, A. C.; Winer, A. M.; Pitts, J. N., Jr. (1979) Kinetics and mechanisms of the reactions of the hydroxyl radical with organic compounds in the gas phase. In: Pitts, J. N., Jr.; Hammond, G. S.; Gollnick, K.; Grosjean, D., eds. *Advances in photochemistry*, v. 11. New York, NY: John Wiley and Sons. pp. 375-488.

References for Properties, Chemistry, Transport (cont'd.)

- Atkinson, R.; Plum, C. N.; Carter, W. P. L.; Winer, A. M.; Pitts, J. N., Jr. (1984a) Rate constants for the gas-phase reactions of NO_3 radicals with a series of organics in air at 298 ± 1 K. *J. Phys. Chem.* 88: 1210-1215.
- Atkinson, R.; Pitts, J. N., Jr.; Aschmann, S. M. (1984b) Tropospheric reactions of dimethyl sulfide with NO_3 and OH radicals. *J. Phys. Chem.* 88: 1584-1587.
- Atkinson, R.; Aschmann, S. M.; Winer, A. M.; Pitts, J. N., Jr. (1984c) Kinetics of the gas-phase reactions of NO_3 radicals with a series of dialkenes, cycloalkenes, and monoterpenes at 295 ± 1 K. *Environ. Sci. Technol.* 18: 370-375.
- Atkinson, R.; Carter, W. P. L.; Plum, C. N.; Winer, A. M.; Pitts, J. N., Jr. (1984d) Kinetics of the gas-phase reactions of NO_3 radicals with a series of aromatics at 296 ± 2 K. *Int. J. Chem. Kinet.* 16: 887-898.
- Atkinson, R.; Plum, C. N.; Carter, W. P. L.; Winer, A. M.; Pitts, J. N., Jr. (1984e) Kinetics of the gas phase reactions of NO_3 radicals with a series of alkanes at 296 ± 2 K. *J. Phys. Chem.* 88: 2361-2364.
- Atkinson, R.; Aschmann, S. M.; Winer, A. M.; Pitts, J. N., Jr. (1985) Kinetics and atmospheric implications of the gas-phase reactions of NO_3 radicals with a series of monoterpenes and related organics at 294 ± 2 K. *Environ. Sci. Technol.* 19: 159-163.
- Bach, W. D., Jr. (1975) Investigation of ozone and ozone precursor concentrations in nonurban locations in the eastern United States; phase II. meteorological analyses. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards; EPA report no. EPA-450/3-74-034a. Available from NTIS, Springfield, VA; PB-246899.
- Bell, G. B. (1960) Meteorological conditions during oxidant episodes in coastal San Diego County in October and November, 1959. Sacramento, CA: Department of Public Health.
- Bollinger, M. J.; Parrish, D. D.; Hahn, C.; Albritton, D. L.; Fehsenfeld, F. C. (1982) NO_x measurements in clean continental air. In: Proceedings of 2nd symposium on composition of the nonurban troposphere; May; Williamsburg, VA.
- Calvert, J. G.; Stockwell, W. R. (1983) Acid generation in the troposphere by gas-phase chemistry. *Environ. Sci. Technol.* 17: 428A-443A.
- Calvert, J. G.; Stockwell, W. R. (1984) The mechanism and rates of the gas-phase oxidation of sulfur dioxide and nitrogen oxides in the atmosphere. In: Calvert, J. G., ed., Acid precipitation: SO_2 , NO and NO_2 oxidation mechanisms: atmospheric considerations. Boston, MA: Butterworth Publishers. (Teasley, J. I., ed., Acid precipitation series v. 3)
- Carter, W. P. L.; Winer, A. M.; Darnall, K. R.; Pitts, J. N., Jr. (1979) Smog chamber studies of temperature effects in photochemical smog. *Environ. Sci. Technol.* 13: 1094-1100.

References for Properties, Chemistry, Transport (cont'd.)

- Carter, W. P. L.; Winer, A. M.; Pitts, J. N., Jr. (1981a) Major atmospheric sink for phenol and the cresols: reaction with the nitrate radical. *Environ. Sci. Technol.* 15: 829-831.
- Carter, W. P. L.; Winer, A. M.; Pitts, J. N., Jr. (1981b) Effect of peroxyacetyl nitrate on the initiation of photochemical smog. *Environ. Sci. Technol.* 15: 831-834.
- Chameides, W. L.; Davis, D. D. (1982) The free radical chemistry of cloud droplets and its impact upon the composition of rain. *JGR J. Geophys. Res.* 87: 4863-4877.
- Chock, D. P.; Dunker, A. M.; Kumar, S.; Sloane, C. S. (1981) Effect of NO_x emission rates on smog formation in the California South Coast Air Basin. *Environ. Sci. Technol.* 15: 933-939.
- Clark, T. L.; Clarke, J. F. (1982) Boundary layer transport of NO_x and O_3 from Baltimore, Maryland--a case study. Presented at: 75th annual meeting of the Air Pollution Control Association; June; New Orleans, LA. Pittsburgh, PA: Air Pollution Control Association; paper no. 82-24.3.
- Clarke, J. F.; Ching, J. K. S.; Brown, R. M.; Westberg, J.; White, J. H. (1982) Regional transport of ozone. Presented at: Third conference on air pollution meteorology; January. Boston, MA: American Meteorological Society; paper no. 1.1.
- Cleveland, W. S.; Guarino, R.; Kleiner, B.; McRae, J. E.; Warner, J. L. (1976a) The analysis of the ozone problem in the northeast United States. In: Specialty conference on ozone/oxidants--interactions with the total environment. Pittsburgh, PA: Air Pollution Control Association; pp. 109-120.
- Cleveland, W. S.; Kleiner, B.; McRae, J. E.; Warner, J. L. (1976b) Photochemical air pollution: transport from New York City area into Connecticut and Massachusetts. *Science (Washington, DC)* 191: 179-181.
- Coffey, P. E.; Stasiuk, W. N. (1975) Evidence of atmospheric transport of ozone into urban areas. *Environ. Sci. Technol.* 9: 59-62.
- Countess, R. J.; Wolff, G. T.; Whitbeck, M. R. (1981) The effect of temperature on ozone formation in the propene/nitrogen dioxide/air system. *J. Environ. Sci. Health.* A16: 1-8.
- Cox, R. A.; Burrows, J. P. (1979) Kinetics and mechanism of the disproportionation of HO_2 in the gas phase. *J. Phys. Chem.* 83: 2560-2568.
- Cox, R. A.; Roffey, M. J. (1977) Thermal decomposition of peroxyacetyl nitrate in the presence of nitric oxide. *Environ. Sci. Technol.* 11: 900-906.
- Cronn, D. R. (1982) Monitoring of ambient halocarbons and natural hydrocarbons in the Smoky Mountains. In: Proceedings from 2nd symposium on composition of the nonurban troposphere; May; Williamsburg, VA.

References for Properties, Chemistry, Transport (cont'd.)

- Crutzen, P. J.; Fishman, J. (1977) Average concentrations of OH in the troposphere, and the budgets of CH₄, CO, H₂, and CH₃CCl₃. *Geophys. Res. Lett.* 4: 321-324.
- Danielsen, E. F. (1968) Stratospheric-tropospheric exchange based on radioactivity, ozone and potential vorticity. *J. Atmos. Sci.* 25: 502-518.
- Danielsen, E. F. (1980) Stratospheric source for unexpectedly large values of ozone measured over the Pacific Ocean during Gametag, August 1977. *JGR J. Geophys. Res.* 85: 401-412.
- Danielsen, E. F.; Mohnen, V. A. (1977) Project duststorm report: ozone transport, in situ measurements, and meteorological analyses of tropopause folding. *JGR J. Geophys. Res.* 82: 5867-5877.
- Darnall, K. R.; Lloyd, A. C.; Winer, A. M.; Pitts, J. N., Jr. (1976) Reactivity scale for atmospheric hydrocarbons based on reaction with hydroxyl radical. *Environ. Sci. Technol.* 10: 692-696.
- Demerjian, K. L.; Schere, K. L. (1979) Applications of a photochemical box model for O₃ air quality in Houston, Texas. *Proceedings of Air Pollution Control Association Specialty Conference on Ozone/Oxidants: Interactions with the Total Environment.* pp. 329-352.
- Demerjian, K. L.; Kerr, J. A.; Calvert, J. G. (1974) The mechanism of photochemical smog formation. In: Pitts, J. N., Jr.; Metcalf, R. L., eds. *Advances in environmental science and technology: v. 4.* New York, NY: John Wiley and Sons, Inc.; pp. 1-262.
- Demerjian, K. L.; Schere, K. L.; Peterson, J. T. (1980) Theoretical estimates of actinic (spherically integrated) flux and photolytic rate constants of atmospheric species in the lower troposphere. In: Pitts, J. N., Jr.; Metcalf, R. L.; Grosjean, D., eds. *Advances in environmental science and technology: volume 10.* New York, NY: John Wiley and Sons; pp. 369-459.
- Derwent, R. G.; Höv, O. (1980) Computer modeling studies of the impact of vehicle exhaust emission controls on photochemical air pollution formation in the United Kingdom. *Environ. Sci. Technol.* 14: 1360-1366.
- Dignon, J.; Hameed, S. (1985) A model investigation of the impact of increases in anthropogenic NO_x emissions between 1967 and 1980 on tropospheric ozone. *J. Atmos. Chem.* 3: 491-506.
- Dimitriades, B. (1970) On the function of hydrocarbon and nitrogen oxides in photochemical smog formation. Washington, DC: U.S. Department of Commerce, Bureau of Mines. Report of Investigations, RI 7433.
- Dimitriades, B. (1972) Effects of hydrocarbon and nitrogen oxides on photochemical smog formation. *Environ. Sci. Tech.* 6: 253-260.

References for Properties, Chemistry, Transport (cont'd.)

- Dimitriades, B. (1977a) An alternative to the Appendix-J method for calculating oxidant- and NO_2 -related control requirements. In: International Conference on Photochemical Oxidant Pollution and Its Control. Proceedings, Vol. II. Research Triangle Park, NC: U.S. Environmental Protection Agency, EPA-600/3-77-001b.
- Dimitriades, B. (1977b) Oxidant control strategies. Part I. Urban oxidant-control strategy derived from existing smog chamber data. Environ. Sci. Technol. 11: 80-88.
- Dodge, M. C. (1977a) Combined use of modeling techniques and smog chamber data to derive ozone-precursor relationships. In: International Conference on Photochemical Oxidant Pollution and Its Control. Proceedings, Vol. II. Research Triangle Park, NC: U.S. Environmental Protection Agency. EPA-600/3-77-001b.
- Dodge, M. C. (1977b) Effect of selected parameters on predictions of a photochemical model. Research Triangle Park, NC: U.S. Environmental Protection Agency. EPA-600/3-77-048.
- Dodge, M. C.; Arnts, R. R. (1979) A new mechanism for the reaction of ozone with olefins. Int. J. Chem. Kinet. 11: 399-410.
- Eaton, W. C.; Decker, C. E.; Tommerdahl, J. B.; Dimmock, F. E. (1979) Study of the nature of ozone, oxides of nitrogen, and nonmethane hydrocarbons in Tulsa, Oklahoma; v. 1: project description and data summaries. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards; EPA report no. EPA-450/4-79-008a. Available from: NTIS, Springfield, VA; PB-300481.
- F.R. (1979, November 14) 44: 65667-65670.
- Ferman, M. Z. A. (1981) Rural nonmethane hydrocarbon concentrations and composition. In: Bufalini, J. J.; Arnts, R. R., eds. Atmospheric biogenic hydrocarbons. Ann Arbor, MI: Ann Arbor Science Publishers, Inc.
- Ferman, M. Z. A.; Monson, P. R. (1978) Comparison of rural and urban air quality. Paper presented at 71st annual meeting of the Air Pollution Control Association; Houston, June.
- Fishman, J.; Carney, T. A. (1984) A one-dimensional photochemical model of the troposphere with planetary boundary-layer parameterization. J. Atmos. Chem. 1: 351-376.
- Fishman, J.; Seiler, W. (1983) Correlative nature of ozone and carbon monoxide in the troposphere: Implications for the tropospheric ozone budget. J. Geophys. Res. 88: 3662-3670.
- Fishman, J.; Vukovich, F. M.; Browell, E. V. (1985) The photochemistry of synoptic-scale ozone synthesis: Implications for the global tropospheric ozone budget. J. Atmos. Chem. 3: 299-320.

References for Properties, Chemistry, Transport (cont'd.)

- Gelinas, R. J.; Vajk, J. P. (1979) Systematic sensitivity analysis of air quality simulation models. Research Triangle Park, NC: U.S. Environmental Protection Agency. EPA-600/4-49-035.
- Gertler, A. W.; Miller, D. F.; Lamb, D.; Katz, U. (1984) Studies of sulfur dioxide and nitrogen dioxide reactions in haze and cloud. In: Durham, J. L., ed. Chemistry of particles, fogs and rain. Boston, MA: Butterworth Publishers; pp. 131-160. (Teasley, J. I., ed. Acid precipitation series, v. 2).
- Glasson, W. A.; Tuesday, C. S. (1970) Inhibition of atmospheric photooxidation of hydrocarbons by nitric oxide. Environ. Sci. Technol. 4: 37-44.
- Hanna, S. R. (1973) A simple dispersion model for the analysis of chemically reactive pollutants. Atmos. Environ. 7: 803-817.
- Herron, J. T.; Huie, R. E. (1977) Stopped-flow studies of the mechanism of ozone-alkene reactions in the gas phase. Ethylene. J. Am. Chem. Soc. 99: 5430-5435.
- Herron, J. T.; Huie, R. E. (1978) Stopped flow studies of mechanisms of ozone-alkene reactions in gas phase propene + isobutene. Int. J. Chem. Kinet. 10: 1019-1040.
- Hoffman, M. R.; Edwards, J. O. (1975) Kinetics of the oxidation of sulfite by hydrogen peroxide in acidic solution. J. Phys. Chem. 79: 2096-2098.
- Holdren, M. W.; Westberg, H. H.; Zimmerman, P. R. (1979) Analysis of monoterpene hydrocarbons in rural atmospheres. JGR J. Geophys. Res. 84: 5083-5088.
- Holdren, M. W.; Spicer, C. W.; Hales, J. M. (1984) Peroxyacetyl nitrate solubility and decomposition rate in acidic water. Atmos. Environ. 18: 1171-1173.
- Holzworth, G. C. (1964) Estimates of mean maximum mixing depths in the contiguous United States. Mon. Weather Rev. 92: 235-242.
- Holzworth, G. C. (1972) Mixing heights, wind speeds, and potential for urban air pollution throughout the contiguous United States. Research Triangle Park, NC: U.S. Environmental Protection Agency; publication no. AP-101. Available from: NTIS, Springfield, VA; PB-207103.
- Holzworth, G. C.; Fisher, R. W. (1979) Climatological summaries of the lower few kilometers of rawinsonde observations. Research Triangle Park, NC: U.S. Environmental Protection Agency, Environmental Sciences Research Laboratory; EPA report no. EPA-600/4-79-026.
- Hosler, C. R. (1961) Low-level inversion frequency in the contiguous United States. Mon. Weather Rev. 89: 319-339.
- Husain, L.; Coffey, P. E.; Meyers, R. E.; Cederwall, R. T. (1977) Ozone transport from stratosphere to troposphere. Geophys. Res. Lett. 4: 363-365.

References for Properties, Chemistry, Transport (cont'd.)

- Jaffee, R. J.; Smith, F. C., Jr.; West, K. W. (1974) Study of factors affecting reactions in environmental chambers: final report on phase III. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Research and Development; EPA report no. EPA-650/3-74-004b. Available from: NTIS, Springfield, VA PB-256252.
- Japar, S. M.; Niki, H. (1975) Gas-phase reactions of the nitrate radical with olefins. *J. Phys. Chem.* 79: 1629-1632.
- Jeffries, H.; Fox, D.; Kamens, R. (1975) Outdoor smog chamber studies. Effect of hydrocarbon reduction on nitrogen dioxide. Research Triangle Park, NC: U.S. Environmental Protection Agency; EPA report no. EPA-650/3-75-011. Available from: NTIS, Springfield, VA; PB-245829.
- Jeffries, H.; Fox, D.; Kamens, R. (1976) Outdoor smog chamber studies: light effects relative to indoor chambers. *Environ. Sci. Technol.* 10: 1006-1011.
- Johnson, W. B.; Viezee, W. (1981) Stratospheric ozone in the lower troposphere--I. Presentation and interpretation of aircraft measurements. *Atmos. Environ.* 15: 1309-1323.
- Junge, C. E. (1963) Air chemistry and radioactivity. New York, NY: Academic Press. (van Mieghen, J.; Hales, A. L., eds. International geophysics series v. 4).
- Kamens, R. M.; Gery, M. W.; Jeffries, H. E.; Jackson, M.; Cole, E. I. (1982) Ozone-isoprene reactions: product formation and aerosol potential. *Int. J. Chem. Kinet.* 14: 955-975.
- Kelly, N. A. (1985) Ozone/precursor relationships in the Detroit metropolitan area derived from captive-air irradiations and an empirical photochemical model. *J. Air Pollut. Control Assoc.* 35: 27-34.
- Kelly, N. A.; Wolff, G. T.; Ferman, M. Z. A. (1982) Background pollutant measurements in air masses affecting the eastern half of the United States--I. Air masses arriving from the northwest. *Atmos. Environ.* 16: 1077-1088.
- Kelly, N. A.; Wolff, G. T.; Ferman, M. A. (1984) Sources and sinks of ozone in rural areas. *Atmos. Environ.* 18: 1251-1266.
- Kelly, N. A.; Ferman, M. A.; Wolff, G. T. (1986) The chemical and meteorological conditions associated with high and low ozone concentrations in southeastern Michigan and nearby areas of Ontario. *J. Air Pollut. Control Assoc.* 36: 150-158.
- Killus, J. P.; Whitten, G. Z. (1984) Isoprene: a photochemical kinetic mechanism. *Environ. Sci. Technol.* 18: 142-148.
- Korshover, J. (1967) Climatology of stagnating anticyclones east of the Rocky Mountains, 1936-1965. Cincinnati, Ohio: U.S. Department of Health, Education and Welfare, National Center for Air Pollution Control; Public Health Service publication no. 999-AP-34. Available from: NTIS, Springfield, VA; PB-174709.

References for Properties, Chemistry, Transport (cont'd.)

- Korshover, J. (1975) Climatology of stagnating anticyclones east of the Rocky Mountains, 1936-1975. Washington, DC: U.S. Department of Commerce, National Oceanic and Atmospheric Administration; NOAA technical memorandum ERL AR-55. Available from: NTIS, Springfield, VA; PB-257368.
- Lee, Y.-N.; Senum, G. I.; Gaffney, J. S. (1983) Peroxyacetyl nitrate (PAN) stability, solubility, and reactivity--implications for tropospheric nitrogen cycles and precipitation chemistry. Presented at CACGP Symposium, Oxford, England; September.
- Levy, H. II; Mahlman, J. D.; Moxim, W. J. (1985) Tropospheric ozone: The role of transport. *J. Geophys. Res.* 90: 3753-3772.
- Liu, C. S.; Grisinger, J. E. (1981) Review of SAI Airshed Model Sensitivity Study Conducted for the South Coast Air Basin by California Air Resources Board. AQMP Technical Paper No. 5. South Coast Air Quality Management District; El Monte, CA.
- Liu, M.-K.; Morris, R. E.; Killus, J. P. (1984) Development of a regional oxidant model and application to the northeastern United States. *Atmos. Environ.* 18: 1145-1161.
- Lloyd, A. C.; Atkinson, R.; Lurmann, F. W.; Nitta, B. (1983) Modeling potential ozone impacts from natural hydrocarbons. I. Development and testing of a chemical mechanism for the NO_x-air photooxidations of isoprene and α -pinene under ambient conditions. *Atmos. Environ.* 17: 1931-1950.
- Logan, J. A. (1983) Nitrogen oxides in the troposphere: global and regional budgets. *JGR J. Geophys. Res.* 88: 10785-10807.
- Lynn, D. A.; Steigerwald, B. J.; Ludwig, J. H. (1964) The November-December 1962 air pollution episode in the eastern United States. Cincinnati, OH: U.S. Department of Health, Education and Welfare, Division of Air Pollution; Public Health Service publication no. 999-AP-77. Available from: NTIS, Springfield, VA; PB-168878.
- Lyons, W. A.; Olsson, L. E. (1972) Mesoscale air pollution transport in the Chicago lake breeze. *J. Air Pollut. Control Assoc.* 22: 876-881.
- Martin, L. R.; Damschen, D. E. (1981) Aqueous oxidation of sulfur dioxide by hydrogen peroxide at low pH. *Atmos. Environ.* 15: 1615-1621.
- Martinez, J. R.; Singh, H. B. (1979) Survey of the role of NO_x in nonurban ozone formation. SRI International, Menlo Park, CA. Final report to U.S. Environmental Protection Agency, SRI Project 6780-8. Research Triangle Park, NC: U.S. Environmental Protection Agency.
- Mohnen, V. A. (1977) Review and analysis. In: International conference on oxidants, 1976 -- Analysis of evidence and viewpoints. Part III. The issue of stratospheric ozone intrusion. Research Triangle Park, NC: U.S. Environmental Protection Agency. Report no. EPA-600/3-77-115.

References for Properties, Chemistry, Transport (cont'd.)

- Mueller, P. K.; Hidy, G. M. (1983) The sulfate regional experiment (SURE): report of findings. Palo Alto, CA: Electric Power Research Institute; report no. EA-1901.
- National Research Council (1977) Ozone and other photochemical oxidants. Washington, DC: National Academy of Sciences.
- Neiburger, M.; Johnson, D. S.; Chen, C. -W. (1961) Studies of the structure of the atmosphere over the eastern Pacific Ocean in the summer: I. the inversion over the eastern north Pacific Ocean. Berkeley, CA: University of California Press; publications in meteorology, v. 1, no. 1.
- Nicksic, S. W.; Harkins, J.; Mueller, P. K. (1967) Some analyses for PAN and studies of its structure. *Atmos. Environ.* 1: 11-18.
- Niki, H.; Maker, P. D.; Savage, C. M.; Breitenbach, L. P. (1981) An FT-IR study of a transitory product in the gas-phase ozone-ethylene reaction. *J. Phys. Chem.* 85: 1024-1027.
- Niki, H.; Maker, P. D.; Savage, C. M.; Breitenbach, L. P. (1983) An FT-IR study of the mechanism for the reaction $\text{HO} + \text{CH}_3\text{SCH}_3$. *Int. J. Chem. Kinet.* 15: 647-654.
- Peterson, J. T. (1976) Calculated actinic fluxes (290-700 nm) for air pollution photochemistry applications. Research Triangle Park, NC: U.S. Environmental Protection Agency, Environmental Sciences Research Laboratory; EPA report no. EPA-600/4-76-025. Available from: NTIS, Springfield, VA; PB-240125.
- Pitts, J. N., Jr.; Winer, A. M.; Darnall, K. R.; Lloyd, A. C.; Doyle, G. J. (1977) Hydrocarbon reactivity and the role of hydrocarbons, oxides of nitrogen, and aged smog in the production of photochemical oxidants. In: Dimitriadis, B., ed. International conference on photochemical oxidant pollution and its control; September 1976; Raleigh, NC. Proceedings: v. II. Research Triangle Park, NC: U.S. Environmental Protection Agency; EPA-600/3-77-001b.
- Reiter, E. R. (1963) A case study of radioactive fallout. *J. Appl. Meteorol.* 2: 691-705.
- Reiter, E. R. (1975) Stratospheric-tropospheric exchange processes. *Rev. Geophys. Space Phys.* 13: 459-473.
- Reiter, E. R.; Mahlman, J. D. (1965) Heavy radioactive fallout over the southern United States, November 1962. *JGR J. Geophys. Res.* 70: 4501-4519.
- Richards, L. W.; Anderson, J. A.; Blumenthal, D. L.; McDonald, J. A.; Kok, G. L.; Lazrus, A. L. (1983) Hydrogen peroxide and sulfur (IV) in Los Angeles cloud water. *Atmos. Environ.* 17: 911-914.

References for Properties, Chemistry, Transport (cont'd.)

- Richter, H. G. (1983) Analysis of organic compound data gathered during 1980 in northeast corridor cities. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards; EPA report no. EPA-450/4-83-017. Available from: NTIS, Springfield, VA; PB84-116052.
- Robinson, E. (1952) Some air pollution aspects of the Los Angeles temperature inversion. *Bull. Am. Meteorol. Soc.* 33: 247-250.
- Scheutle, D.; Rasmussen, R. A. (1978) The molecular composition of secondary aerosol particles formed from terpenes. *J. Air Pollut. Control Assoc.* 28: 236-240.
- Schwartz, S. E. (1984) Gas-aqueous reactions of sulfur and nitrogen oxides in liquid-water clouds. In: Calvert, J. G., ed., *SO₂, NO and NO₂ Oxidation Mechanisms: Atmospheric Considerations*, Chapter 4. Ann Arbor Science Publishers, Acid Precipitation Series, v. 3. Boston, MA: Butterworth Publishers, pp. 173-208.
- Seila, R. L. (1981) Nonurban hydrocarbon concentrations in ambient air north of Houston, TX. In: Bufalini, J. J.; Arnts, R. R., eds. *Atmospheric biogenic hydrocarbons*. Ann Arbor, MI: Ann Arbor Science Publishers, Inc.
- Sexton, K. (1982) Evidence of an additive effect for small city plumes. Presented at: the 75th annual meeting of the Air Pollution Control Association; June; New Orleans, LA. Pittsburgh, PA: Air Pollution Control Association; paper no. 82-31.4.
- Sexton, K.; Westberg, H. (1980) Elevated ozone concentrations measured downwind of the Chicago-Gary urban complex. *J. Air Pollut. Control Assoc.* 30: 911-914.
- Sexton, K.; Westberg, H. (1984) Nonmethane hydrocarbon composition of urban and rural atmospheres. *Atmos. Environ.*: in press.
- Shreffler, J. H.; Evans, R. B. (1982) The surface ozone record from the regional air pollution study, 1975-1976. *Atmos. Environ.* 16: 1311-1321.
- Singh, H. B.; Ludwig, F. L.; Johnson, W. B. (1977) Ozone in clean remote atmospheres: Concentrations and variabilities. SRI Project 5661. Menlo Park, CA: Stanford Research Institute (SRI).
- Singh, H. B.; Viezee, W.; Johnson, W. B.; Ludwig, F. L. (1980) The impact of stratospheric ozone on tropospheric air quality. *J. Air Pollut. Control Assoc.* 30: 1009-1017.
- Slade, D. H., ed. (1968) *Meteorology and atomic energy 1968*. Washington, DC: U.S. Atomic Energy Commission. Available from: NTIS, Springfield, VA; TID-24190.

References for Properties, Chemistry, Transport (cont'd.)

- Stephens, E. R. (1967) The formation of molecular oxygen by alkaline hydrolysis of peroxyacetyl nitrate. *Atmos. Environ.* 1: 19-20.
- Tiao, G. C.; Box, G. E. P.; Hamming, W. J. (1975) Analysis of Los Angeles photochemical smog data: a statistical overview. *J. Air Pollut. Control Assoc.* 25: 260-268.
- Tuazon, E. C.; Winer, A. M.; Pitts, J. N., Jr. (1981) Trace pollutant concentrations in a multiday smog episode in the California South Coast Air Basin by long-pathlength Fourier-transform infrared spectroscopy. *Environ. Sci. Technol.* 15: 1232-1237.
- U.S. Environmental Protection Agency (1977) Uses, limitations and technical basis of procedures for quantifying relationships between photochemical oxidants and precursors. Research Triangle Park, NC: Office of Air Quality Planning and Standards. Report no. EPA-450/2-77-021a.
- U.S. Environmental Protection Agency (1978) Air quality criteria for ozone and other photochemical oxidants. Research Triangle Park, NC: U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office; pp. 116-135; EPA report no. EPA-600/8-78-004. Available from: NTIS, Springfield, VA; PB80-124753.
- U.S. Environmental Protection Agency (1980a) Procedures for the preparation of emission inventories for volatile organic compounds: v. 1. 2nd ed. Research Triangle Park, NC: U.S. Environmental Protection Agency; EPA report no. EPA-450/2-77-028. Available from: NTIS, Springfield, VA; PB-275292.
- U.S. Environmental Protection Agency (1980b) Final emission inventory requirements for 1982 ozone state implementation plans. Research Triangle Park, NC: U.S. Environmental Protection Agency; EPA report no. EPA-450/4-80-016. Available from: NTIS, Springfield, VA; PB81-060434.
- U.S. Environmental Protection Agency (1982) Air quality criteria for oxides of nitrogen. Research Triangle Park, NC: U.S. Environmental Protection Agency; EPA-600/8-82-026. Available from: NTIS, Springfield, VA; PB83-131011.
- U.S. Environmental Protection Agency (1984) National air pollutant emission estimates, 1940-1983. Research Triangle Park, NC: U.S. Environmental Protection Agency. EPA-450/4-84-028.
- U.S. Environmental Protection Agency (1986) National air pollutant emission estimates, 1940-1984. Research Triangle Park, NC: U.S. Environmental Protection Agency. Report no. EPA-450/4-85-014.
- Vaughan, W. M.; Chan, M.; Cantrell, B.; Pooler, F. (1982) A study of persistent elevated pollution episodes in the Northeastern United States. *Bull. Am. Meteorol. Soc.* 63: 258-266.

References for Properties, Chemistry, Transport (cont'd.)

- Viezee, W.; Singh, H. B. (1982) Contribution of stratospheric ozone to ground level ozone concentrations -- a scientific review of existing evidence. Final report (revised draft). EPA grant CR 809330010. Menlo Park, CA: SRI International.
- Viezee, W.; Johnson, W. B.; Singh, H. B. (1979) Airborne measurements of stratospheric ozone intrusions into the troposphere over the United States: final report. Atlanta, GA: Coordinating Research Council; SRI project 6690.
- Viezee, W.; Johnson, W. B.; Singh, H. B. (1983) Stratospheric ozone in the lower troposphere-II. Assessment of downward flux and ground-level impact. Atmos. Environ. 17: 1979-1993.
- Wallington, T. J.; Atkinson, R.; Winer, A. M. (1984) Rate constants for the gas phase reaction of OH radicals with peroxyacetyl nitrate (PAN) at 273 and 296 K. Geophys. Res. Lett. 11: 861-864.
- Weast, R. C., ed. (1977) CRC handbook of chemistry and physics. 57th ed. Boca Raton, FL: CRC Press; p. D-194.
- Westberg, H.; Lamb, B. (1983) Ozone production and transport in the Atlanta, GA, region. Final report; EPA grant no. CR 809221. Research Triangle Park, NC: U.S. Environmental Protection Agency, Atmospheric Sciences Research Laboratory.
- Westberg, H.; Sexton, K.; Roberts, E. (1981) Transport of pollutants along the western shore of Lake Michigan. J. Air Pollut. Control Assoc. 31: 385-388.
- Whitten, G. Z. (1983) The chemistry of smog formation: a review of current knowledge. Environ. Int. 9: 447-463.
- Whitten, G. Z.; Hogo, H. (1977) Mathematical modeling of simulated photochemical smog. Research Triangle Park, NC: U.S. Environmental Protection Agency. Report no. EPA-600/3-77-011.
- Whitten, G. Z.; Hogo, H. (1978) User's manual for kinetics model and ozone isopleth plotting package. Research Triangle Park, NC: U.S. Environmental Protection Agency; EPA report no. EPA-600/8-78-014a.
- Winer, A. M.; Breuer, G. M.; Carter, W. P. L.; Darnall, K. R.; Pitts, J. N., Jr. (1979) Effects of ultraviolet spectral distribution on the photochemistry of simulated polluted atmospheres. Atmos. Environ. 13: 989-998.
- Winer, A. M.; Atkinson, R.; Pitts, J. N., Jr. (1984) Gaseous nitrate radical: possible nighttime atmospheric sink for biogenic organic compounds. Science (Washington, DC) 224: 156-159.
- Wolff, G. T.; Lioy, P. J. (1978) An empirical model for forecasting maximum daily ozone levels in the northeastern U.S. J. Air Pollut. Control Assoc. 28: 1034-1038.

References for Properties, Chemistry, Transport (cont'd.)

- Wolff, G. T.; Lioy, P. J. (1980) Development of an ozone river associated with synoptic-scale episodes in the eastern United States. *Environ. Sci. Technol.* 14: 1257-1260.
- Wolff, G. T.; Lioy, P. J.; Meyers, R. E.; Cederwall, R. T.; Wight, G. D.; Pasceri, R. E.; Taylor, R. S. (1977a) Anatomy of two ozone transport episodes in the Washington, D.C., to Boston, Mass., corridor. *Environ. Sci. Technol.* 11: 506-510.
- Wolff, G. T.; Lioy, P. J.; Wight, G. D.; Pasceri, R. E. (1977b) Aerial investigation of the ozone plume phenomenon. *J. Air Pollut. Control Assoc.* 27: 460-463.
- Wolff, G. T.; Ferman, M. A.; Monson, P. R. (1979) The distribution of beryllium-7 within high-pressure systems in the eastern United States. *Geophys. Res. Lett.* 6: 637-639. (See also "Correction," *Geophys. Res. Lett.* 6: 816, 1979.)
- Wolff, G. T.; Kelly, N. A.; Ferman, M. A. (1982) Source regions of summertime ozone and haze episodes in the eastern United States. *Water, Air, Soil Pollut.* 18: 65-81.

1.12.3 References for Sampling and Measurement of Ozone and Other Photochemical Oxidants and Their Precursors

- Allen, A. O.; Hochanadel, C. J.; Ghormley, J. A.; Davis, T. W. (1952) Decomposition of water and aqueous solutions under mixed fast neutron and gamma-radiation. *J. Phys. Chem.* 56: 575-586.
- Altshuller, A. P.; McPherson, S. P. (1963) Spectrophotometric analysis of aldehydes in the Los Angeles atmosphere. *J. Air Pollut. Control Assoc.* 13: 109-111.
- Andreae, W. A. (1955) A sensitive method for the estimation of hydrogen peroxide in biological materials. *Nature (London)* 175: 859-860.
- Armstrong, W. A.; Humphreys, W. G. (1965) A L.E.T. independent dosimeter based on the chemiluminescent determination of H₂O₂. *Can. J. Chem.* 43: 2576-2584.
- Bass, A. M.; Ledford, A. E., Jr.; Whittaker, J. K. (1977) Ultraviolet photometer for ozone calibration. In: Dimitriadis, B., ed. International conference on photochemical oxidant pollution and its control; September 1976; Raleigh, NC. Proceedings: v. I. Research Triangle Park, NC: U.S. Environmental Protection Agency; pp. 13-17; EPA report no. EPA-600/3-77-001a. Available from: NTIS, Springfield, VA; PB-264232.
- Bowman, L. D.; Horak, R. F. (1972) A continuous ultraviolet absorption ozone photometer. *Anal. Instrum.* 10: 103-108.

References for Sampling and Measurement (cont'd.)

- Brewer, A. W.; Milford, J. R. (1960) The Oxford-Kew ozone sonde. Proc. R. Soc. London Ser. A 256: 470-495.
- Bruckmann, P. W.; Willner, H. (1983) Infrared spectroscopic study of peroxyacetyl nitrate (PAN) and its decomposition products. Environ. Sci. Technol. 17: 352-357.
- Bufalini, J. J.; Brubaker, K. L. (1969) Symposium on chemical reactions in urban atmospheres; October. Warren, MI: General Motors Research Laboratories.
- Bufalini, J. J.; Gay, B. W., Jr.; Brubaker, K. L. (1972) Hydrogen peroxide formation from formaldehyde photooxidation and its presence in urban atmospheres. Environ. Sci. Technol. 6: 816-821.
- Burton, C. S.; Bekowies, P. J.; Pollack, R. I.; Connell, P. (1976) Oxidant/ozone ambient measurement methods: an assessment and evaluation. San Rafael, CA: Systems Applications, Inc.; EF76-111R.
- California Air Resources Board. (1976) A study of the effect of atmospheric humidity on analytical oxidant measurement methods. Presented at: 15th conference [on] methods in air pollution studies; January; Long Beach, CA. Berkeley, CA: California Air Resources Board, Air and Industrial Hygiene Laboratory.
- California Air Resources Board. (1978) California air quality data: July-August-September. 10(13): 2-3. Available from: Technical Service Div., California Air Resources Board, Sacramento, CA.
- Chameides, W.L.; Tan, A. (1981). The two-dimensional diagnostic model for tropospheric OH: An uncertainty analysis. J. Geophys. Res. 86: 5209-5223.
- Chock, D. P.; Kumar, S.; Herrmann, R. W. (1982) An analysis of trends in oxidant air quality in the South Coast Air Basin of California. Atmos. Environ. 16: 2615-2624.
- Cohen, I. R.; Purcell, T. C.; Altshuller, A. P. (1967) Analysis of the oxidant in photooxidation reactions. Environ. Sci. Technol. 1: 247-252.
- Darley, E. F.; Kettner, K. A.; Stephens, E. R. (1963) Analysis of peroxyacyl nitrates by gas chromatography with electron capture detection. Anal. Chem. 35: 589-591.
- Das, T.N.; Moorthy, P.N.; Rao, K.N. (1982). Chemiluminescent method for the determination of low concentrations of hydrogen peroxide. J. Indian Chem. Soc. 59: 85-89.
- Das, T.N.; Moorthy, P.N.; Rao, K.N. (1983). Chemiluminescent measurement of hydrogen peroxide in the Bombay area. Atmos. Environ. 17: 79-82.

References for Sampling and Measurement (cont'd.)

- Dasgupta, P. K. (1980a) Discussion on "The importance of atmospheric ozone and hydrogen peroxide in oxidizing sulphur dioxide in cloud and rainwater [on Penkett et al., 1979]." *Atmos. Environ.* 14: 272-275.
- Dasgupta, P. K. (1980b) The importance of atmospheric ozone and hydrogen peroxide in oxidizing sulfur dioxide in cloud and rainwater--further discussion [on Penkett et al., 1979]. *Atmos. Environ.* 14: 620-621.
- Dasgupta, P. K.; Hwang, H. (1985) Application of a nested loop system for the flow injection analysis of trace aqueous peroxide. *Anal. Chem.* In press.
- Demerjian, K. L.; Kerr, J. A.; Calvert, J. G. (1974) The mechanism of photochemical smog formation. In: Pitts, J. N., Jr.; Metcalf, R. L., eds. *Advances in environmental science and technology: v. 4.* New York, NY: John Wiley and Sons, Inc.; pp. 1-262.
- DeMore, W. B.; Patapoff, M. (1976) Comparison of ozone determinations by ultraviolet photometry and gas-phase titration. *Environ. Sci. Technol.* 10: 897-899.
- DeMore, W. B.; Romanovsky, J. C.; Feldstein, M.; Hamming, W. J.; Mueller, P. K. (1976) Interagency comparison of iodometric methods for ozone determination. In: *Calibration in air monitoring: a symposium; August 1975; Boulder, CO.* Philadelphia, PA: American Society for Testing and Materials; pp. 131-143; ASTM special technical publication 598.
- Dietz, W. A. (1967) Response factors for gas chromatographic analyses. *J. Gas Chromatogr.* 5: 68-71.
- F. R. (1971, April 30) 36: 8186-8201. National primary and secondary air quality standards.
- F.R. (1975, February 18) 40: 7042-7070. Ambient air monitoring reference and equivalent methods.
- F.R. (1976, December 1) 41: 52686-52692. National primary and secondary ambient air quality standards: nitrogen dioxide measurement principle and calibration procedure.
- F.R. (1979, February 8) 44: 8221-8233. Calibration of ozone reference methods.
- Flamm, D. L. (1977) Analysis of ozone at low concentrations with boric acid buffered KI. *Environ. Sci. Technol.* 11: 978-983.
- Fontijn, A.; Sabadell, A. J.; Ronco, R. J. (1970) Homogeneous chemiluminescent measurement of nitric oxide with ozone: implications for continuous selective monitoring of gaseous air pollutants. *Anal. Chem.* 42: 575-579.
- Fried, A.; Hodgeson, J. (1982) Laser photoacoustic detection of nitrogen dioxide in the gas-phase titration of nitric oxide with ozone. *Anal. Chem.* 54: 278-282.

References for Sampling and Measurement (cont'd.)

- Gay, B. W., Jr.; Bufalini, J. J. (1972a) Hydrogen peroxide in the urban atmosphere. In: Gould, R. F., ed., Photochemical smog and ozone reactions. Washington, DC: American Chemical Society; pp. 225-263. (Advances in chemistry series: 113.)
- Gay, B. W., Jr.; Bufalini, J. J. (1972b) Hydrogen peroxide in the urban atmosphere. Environ. Lett. 3: 21-24.
- Gay, B. W.; Noonan, R. C.; Bufalini, J. J.; Hanst, P. L. (1976) Photochemical synthesis of peroxyacyl nitrates in gas phase via chlorine-aldehyde reaction. Environ. Sci. Technol. 10: 82-85.
- Grosjean, D. (1983) Distribution of atmospheric nitrogenous pollutants at a Los Angeles area smog receptor site. Environ. Sci. Technol. 17: 13-19.
- Grosjean, D.; Fung, K.; Collins, J.; Harrison, J.; Breitung, E. (1984) Portable generator for on-site calibration of peroxyacetyl nitrate analyzers. Anal. Chem. 56: 569-573.
- Hanst, P. L.; Wong, N. W.; Bragin, J. (1982) A long-path infrared study of Los Angeles smog. Atmos. Environ. 16: 969-981.
- Hauser, T. R.; Cummins, R. L. (1964) Increasing sensitivity of 3-methyl-2-benzothiazolone hydrazone test for analysis of aliphatic aldehydes in air. Anal. Chem. 36: 679-681.
- Heikes, B.G. (1984) Aqueous H_2O_2 production from O_3 in glass impingers. Atmos. Environ. 18: 1433-1445.
- Heikes, B. G.; Lazrus, A. L.; Kok, G. L.; Kunen, S. M.; Gandrud, B. W.; Gitlin, S. N.; Sperry, P. D. (1982) Evidence for aqueous phase hydrogen peroxide synthesis in the troposphere. JGR J. Geophys. Res. 87: 3045-3051.
- Hendry, D. G.; Kenley, R. A. (1977) Generation of peroxy radicals from peroxy nitrates (RO_2NO_2). Decomposition of peroxyacyl nitrates. Atmos. Environ. 99: 3198-3199.
- Hodgeson, J. A.; Krost, K. J.; O'Keeffe, A. E.; Stevens, R. K. (1970) Chemiluminescent measurement of atmospheric ozone: response characteristics and operating variables. Anal. Chem. 42: 1795-1802.
- Hodgeson, J. A.; Stevens, R. K.; Martin, B. E. (1972) A stable ozone source applicable as a secondary standard for calibration of atmospheric monitors. ISA Trans. 11: 161-167.
- Hodgeson, J. A.; Hughes, E. E.; Schmidt, W. P.; Bass, A. M. (1977) Methodology for standardization of atmospheric ozone measurements. In: Dimitriades, B., ed. International conference on photochemical oxidant pollution and its control; September 1976; Raleigh, NC. Proceedings: v. I. Research Triangle Park, NC: U.S. Environmental Protection Agency; pp. 3-12; EPA report no. EPA-600/3-77-001a. Available from: NTIS, Springfield, VA; PB-264232.

References for Sampling and Measurement (cont'd.)

- Holdren, M. W.; Spicer, C. W. (1984) Field compatible calibration procedure for peroxyacetyl nitrate. *Environ. Sci. Technol.* 18: 113-116.
- Hughes, E. E. (1975) Development of standard reference materials for air quality measurement. *ISA Trans.* 14: 281-291.
- Jayanty, R. K. M.; Blackard, A.; McElroy, F. F.; McClenny, W. A. (1982) Laboratory evaluation of nonmethane organic carbon determination in ambient air by cryogenic preconcentration and flame ionization detection. Research Triangle Park, NC: U.S. Environmental Protection Agency; EPA report no. EPA-600/4-82-019. Available from: NTIS, Springfield, VA; PB82-224965.
- Johnson, D. F.; Kok, G. L.; Sonner, R. J. (1981) Improved chromatographic acid technique for the determination of formaldehyde. In: Grosjean, D.; Kok, G. L., eds. Interlaboratory comparison study of methods for measuring formaldehyde and other aldehydes in ambient air: final report. Atlanta, GA: Coordinating Research Council.
- Joshi, S. B.; Bufalini, J. J. (1978) Halocarbon interferences in chemiluminescent measurements of NO_x . *Environ. Sci. Technol.* 12: 597-599.
- Katz, M. (1976) Nitrogen compounds and oxidants. In: Stern, A. C., ed. Air pollution; v. III. measurement, monitoring and surveillance of air pollution. 3rd ed. New York, NY: Academic Press; pp. 259-305.
- Kok, G. L.; Darnall, K. R.; Winer, A. M.; Pitts, J. N., Jr.; Gay, B. W., Jr. (1978a) Ambient air measurements of hydrogen peroxide in the California South Coast Air Basin. *Environ. Sci. Technol.* 12: 1077-1080.
- Kok, G. L.; Holler, T. P.; Lopez, M. B.; Nachtrieb, H. A.; Yuan, M. (1978b) Chemiluminescent method for determination of hydrogen peroxide in the ambient atmosphere. *Environ. Sci. Technol.* 12: 1072-1076.
- Kravetz, T. M.; Martin, S. W.; Mendenhall, G. D. (1980) Synthesis of peroxyacetyl and peroxyaroyl nitrates: complexation of peroxyacetyl nitrate with benzene. *Environ. Sci. Technol.* 14: 1262-1264.
- Kunen, S. M.; Lazrus, A. L.; Kok, G. L.; Heikes, B. G. (1983) Aqueous oxidation of SO_2 by hydrogen peroxides. *JGR J. Geophys. Res.* 88: 3671-3674.
- Kuntz, R.; Lonneman, W.; Namie, G.; Hull, L. A. (1980) Rapid determination of aldehydes in air analyses. *Anal. Lett.* 13: 1409-1415.
- Lipari, F.; Swarin, S. (1982) Determination of formaldehyde and other aldehydes in automobile exhaust with an improved 2,4-dinitrophenylhydrazine method. *J. Chromatogr.* 247: 297-306.
- Littman, F. E.; Benoliel, R. W. (1953) Continuous oxidant recorder. *Anal. Chem.* 25: 1480-1483.

References for Sampling and Measurement (cont'd.)

- Logan, J.A.; Prather, M.J.; Wofsy, S.C.; McElroy, M.B. (1981) Tropospheric chemistry: A global perspective. *J. Geophys. Res.* 86: 7210-7254.
- Lonneman, W. A.; Bufalini, J. J.; Namie, G. R. (1982) Calibration procedure for PAN based on its thermal decomposition in the presence of nitric oxide. *Environ. Sci. Technol.* 16: 655-660.
- Maeda, Y.; Aoki, K.; Munemori, M. (1980) Chemiluminescence method for the determination of nitrogen dioxide. *Anal. Chem.* 52: 307-311.
- Martin, L. R.; Damschen, D. E. (1981) Aqueous oxidation of sulfur dioxide by hydrogen peroxide at low pH. *Atmos. Environ.* 15: 1615-1621.
- Mast, G. M.; Saunders, H. E. (1962) Research and development of the instrumentation of ozone sensing. *ISA Trans.* 1: 325-328.
- McClenny, W. A.; Pleil, J. D.; Holdren, M. W.; Smith, R. N. (1984) Automated cryogenic preconcentration and gas chromatographic determination of volatile organic compounds in air. *Anal. Chem.* 56: 2947-2951.
- McElroy, F. F. (1979) Transfer standards for the calibration of ambient air monitoring analyzers for ozone: technical assistance document. Research Triangle Park, NC: U.S. Environmental Protection Agency, Environmental Monitoring and Support Laboratory; EPA report no. EPA-600/4-79-056. Available from: NTIS, Springfield, VA; PB-146871.
- McElroy, F. F.; Thompson, V. L. (1975) Hydrocarbon measurement discrepancies among various analyzers using flame-ionization detectors. Research Triangle Park, NC: U.S. Environmental Protection Agency, Environmental Monitoring and Support Laboratory; EPA report no. EPA-600/4-75-010.
- Mottola, H. A.; Simpson, B. E.; Gorin, G. (1970) Absorptiometric determination of hydrogen peroxide in submicrogram amounts with leuco crystal violet and peroxidase as catalyst. *Anal. Chem.* 42: 410-411.
- National Research Council (1977) Ozone and other photochemical oxidants. Washington, DC: National Academy of Sciences; pp. 45-125 and chapters 4 and 6.
- Nederbragt, G. W.; van der Horst, A.; van Duijn, J. (1965) Rapid ozone determination near an accelerator. *Nature (London)* 206: 87.
- Nicksic, S. W.; Harkins, J.; Mueller, P. K. (1967) Some analyses for PAN and studies of its structure. *Atmos. Environ.* 1: 11-18.
- Nielsen, T.; Hansen, A. M.; Thomsen, E. L. (1982) A convenient method for preparation of pure standards of peroxyacetyl nitrate for atmospheric analyses. *Atmos. Environ.* 16: 2447-2450.

References for Sampling and Measurement (cont'd.)

- Ogle, L. D.; Hall, R. C.; Crow, W. L.; Jones, A. E.; Gise, J. P. (1982) Development of preconcentration and chromatographic procedures for the continuous and unattended monitoring of hydrocarbons in ambient air. Presented at: 184th national meeting of the American Chemical Society; September; Kansas City, MO. Austin, TX: Radian Corporation.
- O'Keefe, A. E.; Ortman, G. C. (1966) Primary standards for trace gas analysis. *Anal. Chem.* 38: 760-763.
- Osman, M.; Hill, H. H.; Holdren, M. W.; Westberg, H. H. (1979) Vapor-phase silylation of alcohols for air analysis. In: Zlatkis, A., ed. *Advances in chromatography, chromatography symposium*; Houston, TX.
- Overton, J. H., Jr.; Durham, J. L. (1982) Acidification of rain in the presence of SO_2 , H_2O_2 , O_3 , and HNO_3 . In: Keith, L. H., ed. *Energy and environmental chemistry, v. 2: acid rain*. Ann Arbor, MI: Ann Arbor Science; pp. 245-262.
- Paur, R. J.; McElroy, F. F. (1979) Technical assistance document for the calibration of ambient ozone monitors. Research Triangle Park, NC: U.S. Environmental Protection Agency, Environmental Monitoring and Support Laboratory; EPA final report no. EPA-600/4-79-057. Available from: NTIS, Springfield, VA; PB80-149552.
- Penkett, S. A.; Jones, B. M. R.; Brice, K. A.; Eggleton, A. E. J. (1979) The importance of atmospheric ozone and hydrogen peroxide in oxidising sulphur dioxide in cloud and rainwater. *Atmos. Environ.* 13: 123-137.
- Perschke, H.; Broda, E. (1961) Determination of very small amounts of hydrogen peroxide. *Nature (London)* 190: 257-258.
- Reckner, L. R. (1974) Survey of users of EPA Reference Method for measurement of non-methane hydrocarbon in ambient air. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Research and Development; EPA report no. EPA-650/4-75-008. Available from: NTIS, Springfield, VA; PB-247515.
- Regener, V. H. (1960) On a sensitive method for the recording of atmospheric ozone. *JGR J. Geophys. Res.* 65: 3975-3977.
- Regener, V. H. (1964) Measurement of atmospheric ozone with the chemiluminescent method. *JGR J. Geophys. Res.* 69: 3795-3800.
- Rehme, K.A.; Martin, B.E.; Hodgeson, J.A. (1974) Tentative method for the calibration of nitric oxide, nitrogen dioxide, and ozone analyzers by gas phase titration. U.S. Environmental Protection Agency, Research Triangle Park, N.C. EPA-R2-73-246.
- Rehme, K. A.; Puzak, J. C.; Beard, M. E.; Smith, C. F.; Paur, R. J. (1981) Evaluation of ozone calibration procedures. Research Triangle Park, NC: U.S. Environmental Protection Agency, Environmental Monitoring Systems Laboratory; EPA final report no. EPA-600/4-80-050. Available from: NTIS, Springfield, VA; PB81-118911.

References for Sampling and Measurement (cont'd.)

- Richter, H. G. (1983) Analysis of organic compound data gathered during 1980 in northeast corridor cities. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards; EPA report no. EPA-450/4-83-017. Available from: NTIS, Springfield, VA; PB84-116052.
- Sawicki, E.; Hauser, T. R.; Stanley, T. W.; Elbert, W. (1961) The 3-methyl-2-benzothiazolone hydrazone test: sensitive new methods for the detection, rapid estimation, and determination of aliphatic aldehydes. *Anal. Chem.* 33: 93-96.
- Scaringelli, F. P.; Rosenberg, P. E.; Rehme, K. A. (1970) Comparison of permeation devices and nitrite ion as standards for the colorimetric determination of nitrogen dioxide. *Environ. Sci. Technol.* 4: 924-929.
- Schiff, H. (1985) York University, Toronto, Canada. Personal communication: work in progress.
- Sevčík, J. (1975) Detectors in gas chromatography. Prague, Czechoslovakia: Elsevier Scientific Publishing Company.
- Sexton, F. W.; McElroy, F. F.; Michie, R. M., Jr.; Thompson, V. L. (1982) A comparative evaluation of seven automated ambient non-methane organic compound analyzers. Research Triangle Park, NC: U.S. Environmental Protection Agency; EPA report no. EPA-600/4-82-046. Available from: NTIS, Springfield, VA; PB82-230798.
- Singh, H. B.; Salas, L. J. (1983a) Peroxyacetyl nitrate in the free troposphere. *Nature (London)* 302: 326-328.
- Singh, H. B.; Salas, L. J. (1983b) Methodology for the analysis of peroxyacetyl nitrate (PAN) in the unpolluted atmosphere. *Atmos. Environ.* 17: 1507-1516.
- Stephens, E. R. (1964) Absorptivities for infrared determination of peroxyacyl nitrates. *Anal. Chem.* 36: 928-929.
- Stephens, E. R. (1969) The formation, reactions, and properties of peroxyacyl nitrates (PANs) in photochemical air pollution. In: Pitts, J. N., Jr.; Metcalf, R. L., eds. *Advances in environmental sciences v. 1*. New York, NY: Wiley-Interscience; pp. 119-146.
- Stephens, E. R.; Burleson, F. R.; Cardiff, E. A. (1965) The production of pure peroxyacyl nitrates. *J. Air Pollut. Control Assoc.* 15: 87-89.
- Stevens, R. K.; Hodgeson, J. A. (1973) Applications of chemiluminescent reactions to the measurement of air pollutants. *Anal. Chem.* 45: 443A-446A, 449A.
- Stevens, R. K.; Clark, T. A.; Decker, C. E.; Ballard, L. F. (1972a) Field performance of advanced monitors for oxides of nitrogen, ozone, sulfur dioxide, carbon monoxide, methane and nonmethane hydrocarbons. Presented at: 65th annual meeting of the Air Pollution Control Association; June; Miami, FL. Pittsburgh, PA: Air Pollution Control Association.

References for Sampling and Measurement (cont'd.)

- Stevens, R. K.; Hodgeson, J. A.; Ballard, L. F.; Decker, C. E. (1972b) Ratio of sulfur dioxide to total gaseous sulfur compounds and ozone to total oxidants in the Los Angeles atmosphere - an instrument evaluation study. In: Mamantov, G.; Shults, W. D., eds. Determination of air quality: proceedings of the ACS symposium on determination of air quality; April 1971; Los Angeles, CA. New York, NY: Plenum Press; pp. 83-108.
- Tokiwa, Y.; Twiss, S.; de Vera, E. R.; Mueller, P. K. (1972) Atmospheric ozone determination by amperometry and colorimetry. In: Mamantov, G.; Shults, W. D., eds. Determination of air quality: proceedings of the ACS symposium on determination of air quality; April 1971; Los Angeles, CA. New York, NY: Plenum Press; pp. 109-130.
- Tuazon, E. C.; Graham, R. A.; Winer, A. M.; Easton, R. R.; Pitts, J. N., Jr. Hanst, P. L. (1978) A kilometer pathlength Fourier-transform infrared system for the study of trace pollutants in ambient and synthetic atmospheres. *Atmos. Environ.* 12: 865-875.
- Tuazon, E. C.; Winer, A. M.; Graham, R. A.; Pitts, J. N., Jr. (1980) Atmospheric measurements of trace pollutants by kilometer-pathlength FTIR spectroscopy. *Adv. Environ. Sci. Technol.* 10: 259-300.
- Tuazon, E. C.; Winer, A. M.; Graham, R. A.; Pitts, J. N., Jr. (1981a) Atmospheric measurements of trace pollutants: long path Fourier-transform infrared spectroscopy. Research Triangle Park, NC: U.S. Environmental Protection Agency; EPA report no. EPA-600/3-81-026. Available from: NTIS, Springfield, VA; PB81-179848.
- Tuazon, E. C.; Winer, A. M.; Pitts, J. N., Jr. (1981b) Trace pollutant concentrations in a multiday smog episode in the California South Coast Air Basin by long path length Fourier transform infrared spectroscopy. *Environ. Sci. Technol.* 15: 1232-1237.
- U.S. Environmental Protection Agency. (1975) Technical assistance document for the chemiluminescence measurement of nitrogen dioxide. Research Triangle Park, NC: U.S. Environmental Protection Agency; EPA report no. EPA-600/4-75-003. Available from: NTIS, Springfield, VA.
- U.S. Environmental Protection Agency. (1977a) Air monitoring strategy for state implementation plans. Research Triangle Park, NC: Office of Air Quality Planning and Standards, Standing Air Monitoring Group; EPA report no. EPA-450/2-77-010. Available from: NTIS, Springfield, VA; PB-274014.
- U.S. Environmental Protection Agency. (1977b) Quality assurance handbook for air pollution measurement systems, volume II: ambient air specific methods. Research Triangle Park, NC: U.S. Environmental Protection Agency; EPA report no. EPA-600/4-77-027. Available from: NTIS, Springfield, VA; PB-273518.

References for Sampling and Measurement (cont'd.)

- U.S. Environmental Protection Agency. (1978) Air quality criteria for ozone and other photochemical oxidants. Research Triangle Park, NC: U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office; pp. 116-135; EPA report no. EPA-600/8-78-004. Available from: NTIS, Springfield, VA; PB80-124753.
- U.S. Environmental Protection Agency. (1981) Technical assistance document for the calibration and operation of automated ambient non-methane organic compound analyzers. Research Triangle Park, NC: U.S. Environmental Protection Agency; EPA report no. EPA-600/4-81-015. Available from: NTIS, Springfield, VA; PB82-147406.
- U.S. Environmental Protection Agency. (1982) Air quality criteria for oxides of nitrogen. Research Triangle Park, NC: Environmental Criteria and Assessment Office; EPA report no. EPA-600/8-82-026. Available from: NTIS, Springfield, VA; PB83-131011.
- Wendel, G. J.; Stedman, D. H.; Cantrell, C. A. (1983) Luminol-based nitrogen dioxide detector. *Anal. Chem.* 55: 937-940.
- Westberg, H. H.; Holdren, M. W.; Hill, H. H. (1980) Analytical methodology for the identification and quantification of vapor phase organic pollutants. New York, NY: Coordinating Research Council; CRC-APRAC project no. CAPA-11-71.
- Winer, A. M.; Peters, J. M.; Smith, J. P.; Pitts, J. N., Jr. (1974) Response of commercial chemiluminescent NO-NO₂ analyzers to other nitrogen-containing compounds. *Environ. Sci. Technol.* 8: 1118-1121.
- Yoshizumi, K.; Aoki, K.; Nouchi, I.; Okita, T.; Kobayashi, T.; Kamakura, S.; Tajima, M. (1984) Measurement of the concentration in rainwater and of the Henry's Law constant of hydrogen peroxide. *Atmos. Environ.* 18:395-401.
- Zaitso, K.; Okhura, Y. (1980) New fluorogenic substrates for horseradish peroxidase: rapid and sensitive assays for hydrogen peroxide and peroxidase. *Anal. Biochem.* 109: 109-113.
- Zika, R. G.; Saltzman, E. S. (1982) Interaction of ozone and hydrogen peroxide in water: implications for analysis of H₂O₂ in air. *Geophys. Res. Lett.* 9: 231-234.

1.12.4 References for Concentrations of Ozone and Other Photochemical Oxidants in Ambient Air

- Altshuller, A. P. (1983) Measurements of the products of atmospheric photochemical reactions in laboratory studies and in ambient air--relationships between ozone and other products. *Atmos. Environ.* 17: 2383-2427.

References for Ambient Air Concentrations (cont'd.)

- Berk, J. V.; Young, R. A.; Brown, S. R.; Hollowell, C. D. (1981) Impact of energy-conserving retrofits on indoor air quality in residential housing. Presented at: 74th annual meeting of the Air Pollution Control Association; June; Philadelphia, PA. Pittsburgh, PA: Air Pollution Control Association; paper no. 81-22.1
- Cleveland, W. S.; Graedel, T. E.; Kleiner, B. (1976a) Photochemical air pollution: transport from the New York City area into Connecticut and Massachusetts. *Science* (Washington, DC) 191: 179-181.
- Cleveland, W. S.; Guarino, R.; Kleiner, B.; McRae, J. E.; Warner, J. L. (1976b) The analysis of the ozone problem in the northeast United States. In: Specialty Conference on: Ozone/oxidants--interactions with the total environment. Pittsburgh, PA: Air Pollution Control Association; pp. 109-120.
- Coffey, P. E.; Stasiuk, W. N.; Mohnen, V. (1977) Ozone in rural and urban areas of New York State. In: Dimitriadis, B., ed. International conference on photochemical oxidant pollution and its control. Proceedings: v. I. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Research and Development; pp. 89-96; EPA report no. EPA-600/3-77-001a. Available from: NTIS, Springfield, VA; PB-624232.
- Contant, C. F.; Gehan, B. M.; Stock, T. H.; Holguin, A. H.; Buffler, P. A. (1985) Estimation of individual ozone exposures using microenvironment measurements. In: APCA Specialty Conference: Scientific Basis for the Ozone Standard, Houston, TX, November 28-December 1, 1984. Pittsburgh; APCA. In Press.
- Darley, E. F.; Kettner, K. A.; Stephens, E. R. (1963) Analysis of peroxyacyl nitrates by gas chromatography with electron capture detection. *Anal. Chem.* 35: 589-591.
- Davies, T. D.; Ramer, B.; Kaspyzok, G.; Delany, A. C. (1984) Indoor/outdoor ozone concentrations at a contemporary art gallery. *J. Air Pollut. Control Assoc.* 31: 135-137.
- Evans, G. F. (1985) The National Air Pollution Background Network: final project report. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Research and Development; EPA report no. EPA-600/4-85-038. Available from: NTIS, Springfield, VA; PB85-212413.
- Evans, G.; Finkelstein, P.; Martin, B.; Possiel, N.; Graves, M. (1983). Ozone measurements from a network of remote sites. *J. Air Pollut. Control Assoc.* 33: 291-296.
- Galbally, I. E.; Roy, C. R. (1980) Destruction of ozone at the earth's surface. *Quart. J. R. Met. Soc.* 106: 599-620.

References for Ambient Air Concentrations (cont'd.)

- Grosjean, D. (1981) Critical evaluation and comparison of measurement methods for nitrogenous compounds in the atmosphere. Final report, A 706-05, for Coordinating Research Council. Environmental Research and Technology. Westlake Village, CA.
- Grosjean, D. (1983) Distribution of atmospheric nitrogenous pollutants at a Los Angeles area smog receptor site. *Environ. Sci. Technol.* 17: 13-19.
- Hanst, P. L.; Wong, N. W.; Bragin, J. (1982) A long path infrared study of Los Angeles smog. *Atmos. Environ.* 16: 969-981.
- Heikes, B. G.; Lazrus, A. L.; Kok, G. L.; Kunen, S. M.; Gandrud, B. W.; Gitlin, S. N.; Sperry, P. D. (1982) Evidence of aqueous phase hydrogen peroxide synthesis in the troposphere. *JGR J. Geophys. Res.* 87: 3045-3051.
- Jorgen, R. T.; Meyer, R. A.; Hughes, R. A. (1978) Routine peroxyacetyl nitrate (PAN) monitoring applied to the Houston Area Oxidant Study. Presented at: 71st annual meeting of the Air Pollution Control Association; June; Houston, TX. Pittsburgh, PA: Air Pollution Control Association; paper no. 78-50.1.
- Kelly, N. A.; Ferman, M. A.; Wolff, G. T. (1986) The chemical and meteorological conditions associated with high and low ozone concentrations in south-eastern Michigan and nearby areas of Ontario. *J. Air Pollut. Control Assoc.* 36: 150-158.
- Lefohn, A. S. (1984) A comparison of ambient ozone exposures for selected nonurban sites. Proceedings of 77th Annual Meeting of the Air Pollution Control Association. Vol. 6. Paper no. 84-104.1. Pittsburgh, PA: Air Pollution Control Assoc.
- Lefohn, A. S.; Benedict, H. M. (1985) Exposure considerations associated with characterizing ozone ambient air quality monitoring data. In: Proceedings of the APCA Specialty Conference: Evaluation of the Scientific Basis for Ozone/Oxidants Standards, Houston, TX, November 28-30, 1984. Pittsburgh, PA: Air Pollution Control Assoc.
- Lewis, T. E.; Brennan, E.; Lonneman, W. A. (1983) PAN concentrations in ambient air in New Jersey. *J. Air Pollut. Control Assoc.* 33: 885-887.
- Logan, J. A.; Prather, M. J.; Wofsy, S. C.; McElroy, M. B. (1981) Tropospheric chemistry: a global perspective. *JGR J. Geophys. Res.* 86: 7210-7254.
- Lonneman, W. A.; Bufalini, J. J.; Seila, R. L. (1976) PAN and oxidant measurement in ambient atmospheres. *Environ. Sci. Technol.* 10: 374-380.
- Martinez, J. R.; Singh, H. B. (1979) Survey of the role of NO_x in nonurban ozone formation. Research Triangle Park, NC: prepared by SRI International for U.S. Environmental Protection Agency. EPA report no. EPA-450/4-79-035. Available from: NTIS, Springfield, VA; PB80-122815.

References for Ambient Air Concentrations (cont'd.)

- Mayrsohn, H.; Brooks, C. (1965) The analysis of PAN by electron capture gas chromatography. Presented at: Western regional meeting of the American Chemical Society; November; Los Angeles, CA. Los Angeles, CA: California State Department of Public Health.
- Miller, P. R.; McCutchan, M. H.; Ryan, B. C. (1972) Influence of climate and topography on oxidant air pollution concentrations that damage conifer forests in southern California. Mitt. Forst. Bundesversuchsanst. Wien 97: 585-607.
- Mohnen, V. A. (1977) Review and analysis. In: International Conference on Oxidants, 1976 -- Analysis of evidence and viewpoints. Part III. The issue of stratospheric ozone intrusion. U.S. Environmental Protection Agency, Research Triangle Park, NC; Report no. EPA-600/3-77-115.
- Oltmans, S. J. (1981) Surface ozone measurements in clean air. J. Geophys. Res. 86: 1174-1180. Cited in: Logan et al., 1981.
- Peterson, G. A.; Sabersky, R. H. (1975) Measurements of pollutants inside an automobile. J. Air Pollut. Control Assoc. 25: 1028-1032.
- Pratt, G. C.; Hendrickson, R. C.; Chevone, B. I.; Christopherson, D. A.; O'Brien, M. V.; Krupa, S. V. (1983) Ozone and oxides of nitrogen in the rural upper-midwestern U.S.A. Atmos. Environ. 17: 2013-2023.
- Reiter, E. R. (1977) Review and analysis. In: International Conference on Oxidants 1976 -- Analysis of evidence and viewpoints. Part III. The issue of stratospheric ozone intrusion. U.S. Environmental Protection Agency, Research Triangle Park, NC; Report no. EPA-600/3-77-115.
- Renzetti, N. A.; Bryan, R. J. (1961) Atmospheric sampling for aldehydes and eye irritation in Los Angeles smog - 1960. J. Air Pollut. Control Assoc. 11: 421-424.
- Sabersky, R. H.; Sinema, D. A.; Shair, F. H. (1973) Concentrations, decay rates, and removal of ozone and their relation to establishing clean indoor air. Environ. Sci. Technol. 1: 347-353.
- SAROAD, Storage and Retrieval of Aerometric Data [data base]. (1985a) Data file for 1976. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Disc; ASCII.
- SAROAD, Storage and Retrieval of Aerometric Data [data base]. (1985b) Data file for 1979. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Disc; ASCII.
- SAROAD, Storage and Retrieval of Aerometric Data [data base]. (1985c) Data file for 1980. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Disc; ASCII.

References for Ambient Air Concentrations (cont'd.)

- SAROAD, Storage and Retrieval of Aerometric Data [data base]. (1985d) Data file for 1981. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Disc; ASCII.
- SAROAD, Storage and Retrieval of Aerometric Data [data base]. (1985e) Data file for 1982. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Disc; ASCII.
- SAROAD, Storage and Retrieval of Aerometric Data [data base]. (1985f) Data file for 1983. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Disc; ASCII.
- Seiler, W.; Fishman, J. (1981) The distribution of carbon monoxide and ozone in the free troposphere. *JGR J. Geophys. Res.* 86: 7255-7265.
- Singh, H. B.; Salas, L. J.; Smith, A. J.; Shigeishi, H. (1981) Measurements of some potentially hazardous organic chemicals in urban environments. *Atmos. Environ.* 15: 601-612.
- Singh, H. B., Salas, L. J.; Stiles, R.; Shigeishi, H. (1982) Measurements of hazardous organic chemicals in the ambient atmosphere. Research Triangle Park, NC: U.S. Environmental Protection Agency, Environmental Sciences Research Laboratory. EPA report no. EPA-600/3-83-002. Available from: NTIS, Springfield, VA; PB83-156935.
- Smith, W. J. (1981) New York State air monitoring data report for the Northeast Corridor Regional Modeling Project (NECRMP). Albany, NY: New York State Department of Environment and Conservation.
- Spicer, C. W.; Gemma, J. L.; Joseph, D. W.; Sticksel, P. R.; Ward, G. F. (1976) The transport of oxidant beyond urban areas. Columbus, OH: Battelle Columbus Laboratories; EPA report no. EPA-600/3-76-018a. Available from: NTIS, Springfield, VA; PB-253736.
- Stock, T. H.; Holguin, A. H.; Selwyn, B. J.; Hsi, B. P.; Contant, C. F.; Buffler, P. A.; Kotchmar, D. J. (1983). Exposure estimates for the Houston area asthma and runners studies. In: Lee, S. D.; Mustafa, M. G.; Mehlman, M. A. eds. *Adv. Modern Environ. Toxicol. Vol. V. International Symposium on the Biomedical Effects of Ozone and Related Photochemical Oxidants.* Pinehurst, NC; March 14-18, 1982. Princeton, NJ: Princeton Scientific Publishers, Inc.
- Temple, P. J.; Taylor, O. C. (1983) World-wide ambient measurements of peroxyacetyl nitrate (PAN) and implications for plant injury. *Atmos. Environ.* 17: 1583-1587.
- Thompson, C. R.; Hensel, E. G.; Kats, G. (1973) Outdoor-indoor levels of six air pollutants. *J. Air Pollut. Control Assoc.* 23: 881-886.

References for Ambient Air Concentrations (cont'd.)

- Tuazon, E. C.; Winer, A. M.; Graham, R. A.; Pitts, J. N., Jr. (1981a) Atmospheric measurements of trace pollutants: long path Fourier transform infrared spectroscopy. Research Triangle Park, NC: U.S. Environmental Protection Agency, Environmental Sciences Research Laboratory. Available from: NTIS, Springfield, VA; PB81-179848.
- Tuazon, E. C.; Winer, A. M.; Pitts, J. N., Jr. (1981b) Trace pollutant concentrations in a multiday smog episode in the California South Coast Air Basin by long path length Fourier transform infrared spectrometry. Environ. Sci. Technol. 15: 1232-1237.
- U.S. Department of Commerce, Bureau of the Census (1982) Statistical Abstract of the United States, 1982-1983. 103rd Ed. Washington, DC.
- U.S. Environmental Protection Agency (1978) Air quality criteria for ozone and other photochemical oxidants. Research Triangle Park, NC: U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office; EPA report no. EPA-600/8-78-004. Available from: NTIS, Springfield, VA; PB80-124 753.
- U.S. Environmental Protection Agency (1980) Air quality data--1979 annual statistics. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards; EPA report no. EPA-450/4-80-014.
- U.S. Environmental Protection Agency (1981) Air quality data--1980 annual statistics. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards; EPA report no. EPA-450/4-81-027.
- U.S. Environmental Protection Agency (1982) Air quality data--1981 annual statistics. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards; EPA report no. EPA-450/4-82-007.
- U.S. Environmental Protection Agency (1984) National air quality and emission trends report, 1983. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. EPA report no. EPA-450/4-84-029.
- U.S. Environmental Protection Agency (1986) National air quality and emissions trends report, 1984. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. EPA report no. EPA-450/4-86-001.
- Viezee, W.; Johnson, W. B.; Singh, H. B. (1979) Airborne measurements of stratospheric ozone intrusions into the troposphere over the United States. Final report. SRI Proj. 6690, CRC-APRAC Proj. no. CAPA-15-76 (1-77), Coordinating Research Council, Atlanta, GA.

- Westberg, H.; Allwine, K.; Robinson, E. (1978) Measurement of light hydrocarbons and oxidant transport: Houston area 1976. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Research and Development; EPA report no. EPA-600/3-78-662. Available from: NTIS, Springfield, VA; PB-285891.
- Wolff, G. T.; Stasiuk, W. N.; Coffey, P. E.; Pasceri, R. E. (1975) Aerial ozone measurements over New Jersey, New York, and Connecticut. Presented at the 68th Annual Meeting of the Air Pollution Control Association, Boston, MA: June. Pittsburgh, PA: Air Pollution Control Association. Paper no. 75-58.6.
- Wolff, G. T.; Lioy, P. J.; Taylor, R. S. (1986) The diurnal variations of ozone at different altitudes on a rural mountain in the eastern United States. *J. Air Pollut. Control Assoc.* 36: (In Press)
- 1.12.5 References for Effects of Ozone and Other Photochemical Oxidants on Vegetation
- Adams, R. M.; Crocker, T. D. (1984) Economically relevant response estimation and the value of information: acid deposition. In: Crocker, T. D., ed. *Economic perspectives on acid deposition control*. Boston, MA: Butterworth Publishers; pp. 35-64. (Teasley, J. I., ed. *Acid precipitation series -v. 8.*)
- Adams, R. M.; McCarl, B. A. (1985) Assessing the benefits of alternative oxidant standards on agriculture: the role of response information. *J. Environ. Econ. Manage.*: in press.
- Adams, R. M.; Crocker, R. M.; Katz, R. W. (1984a) The adequacy of natural science information in economic assessments of pollution control.: a Bayesian methodology. *Rev. Econ. Stat.* 66: 568-575.
- Adams, R. M.; Hamilton, S. A.; McCarl, B. A. (1984b) The economic effects of ozone on agriculture. Corvallis, OR: U.S. Environmental Protection Agency; EPA report no. EPA-600/3-84-090. Available from: NTIS, Springfield, VA; PB85-168441/XAB.
- Adams, R. M.; Crocker, T. D.; Thanavibulchai, N. (1982) An economic assessment of air pollution damages to selected annual crops in southern California. *J. Environ. Econ. Manage.* 9: 42-58.
- Adedipe, N. O.; Ormrod, D. P. (1974) Ozone induced growth suppression in radish plants in relation to pre- and post-fumigation temperatures. *Z. Pflanzenphysiol.* 71: 281-287.
- Adedipe, N. O.; Barrett, R. E.; Ormrod, D. P. (1972) Phytotoxicity and growth responses of ornamental bedding plants to ozone and sulfur dioxide. *J. Am. Soc. Hortic. Sci.* 97: 341-345.
- Amiro, B. D.; Gillespie, T. J.; Thurtell, G. W. (1984) Injury response of *Phaseolus vulgaris* to ozone flux density. *Atmos. Environ.* 18: 1207-1215.

References for Vegetation Effects (cont'd.)

- Ashmore, M. R. (1984) Effects of ozone on vegetation in the United Kingdom. Proceedings of the OECD workshop on ozone; Gothenburg, Sweden.
- Benedict, H. M.; Miller, C. J.; Olson, R. E. (1971) Economic impact of air pollutants on plants in the United States. Research Triangle Park, NC: U.S. Environmental Protection Agency; report no. APTD-0953. Available from: NTIS, Springfield, VA; PB-209265.
- Bennett, J. H. (1979) Foliar exchange of gases. In: Heck, W. W.; Krupa, S. V.; Linzon, S. N., eds. Methodology for the assessment of air pollution effects on vegetation: a handbook from a specialty conference; April 1978; Minneapolis, MN. Pittsburgh, PA: Air Pollution Control Association; ch. 10.
- Bennett, J. H.; Hill, A. C. (1974) Acute inhibition of apparent photosynthesis. In: Dugger, J., ed. Air pollution effects on plant growth. Washington, DC: American Chemical Society. (ACS symposium series 3.)
- Bennett, J. P.; Oshima, R. J.; Lippert, L. F. (1979) Effects of ozone on injury and dry matter partitioning in pepper plants. Environ. Exp. Bot. 19: 33-39.
- Benson, E. J.; Krupa, S.; Teng, P. S.; Welsch, P. E. (1982) Economic assessment of air pollution damages to agricultural and silvicultural crops. In: Minnesota final report to Minnesota Pollution Control Agency. St. Paul, MN: University of Minnesota.
- Bisessar, S. (1982) Effect of ozone, antioxidant protection, and early blight on potato in the field. J. Am. Soc. Hortic. Sci. 107: 597-599.
- Bisessar, S.; Palmer, K. T. (1984) Ozone, antioxidant spray and Meloidogyne hapla effects on tobacco. Atmos. Environ. 18: 1025-1027.
- Black, V. J.; Ormrod, D. P.; Unsworth, M. H. (1982) Effects of low concentration of ozone, singly, and in combination with sulphur dioxide on net photosynthesis rates of Vicia faba L. J. Exp. Bot. 33: 1302-1311.
- Blum, U. T.; Smith, G. R.; Fites, R. C. (1982) Effects of multiple O₃ exposures on carbohydrate and mineral contents of Ladino clover. Environ. Exp. Bot. 22: 143-154.
- Clarke, B. B.; Henninger, M. R.; Brennan, E. (1983) An assessment of potato losses caused by oxidant air pollution in New Jersey. Phytopathology 73: 104-108.
- Coyne, P. E.; Bingham, G. E. (1978) Photosynthesis and stomatal light responses in snap beans exposed to hydrogen sulfide in ozone. J. Air Pollut. Control Assoc. 28: 1119-1123.
- Davis, D. D. (1975) Resistance of young ponderosa pine seedlings to acute doses of PAN. Plant Dis. Rep. 59: 183-184.

References for Vegetation Effects (cont'd.)

- Davis, D. D. (1977) Response of ponderosa pine primary needles to separate and simultaneous ozone and PAN exposures. *Plant Dis. Rep.* 61: 640-644.
- Dochinger, L. S.; Townsend, A. M. (1979) Effects of roadside deicer salts and ozone on red maple progenies. *Environ. Pollut.* 19: 229-237.
- Duchelle, S. F.; Skelly, J. M.; Sharick, T. L.; Chevone, B. I.; Yang, Y.-S.; Nellessen, J. E. (1983) Effects of ozone on the productivity of natural vegetation in a high meadow of the Shenandoah National Park of Virginia. *J. Environ. Manage.* 17: 299-308.
- Elkley, T.; Ormrod, D. P. (1981) Absorption of ozone, sulphur dioxide, and nitrogen dioxide by petunia plants. *Environ. Exp. Bot.* 21: 63-70.
- Ensing, J.; Hofstra, G. (1982) Impact of the air pollutant ozone on acetylene reduction and shoot growth of red clover. *Can. J. Plant Pathol.* 4: 237-242.
- Feder, W. A. (1968) Reduction in tobacco pollen germination and tube elongation, induced by low levels of ozone. *Science (Washington, DC)* 160: 1122.
- Feder, W. A.; Campbell, F. J. (1968) Influence of low levels of ozone on flowering of carnations. *Phytopathology* 58: 1038-1039.
- Flagler, R. B.; Youngner, V. B. (1982) Ozone and sulfur dioxide effects on tall fescue: I. Growth and yield responses. *J. Environ. Qual.* 11: 386-389.
- Foster, K. W.; Timm, H.; Labanauskas, C. K.; Oshima, R. J. (1983) Effects of ozone and sulfur dioxide on tuber yield and quality of potatoes. *J. Environ. Qual.* 12: 75-80.
- Fukuda, H.; Terakado, K. (1974) On the damage of plants due to peroxyacyl nitrates (PAN). *Tokyo Nogyo Shikenjo Sokuho*: 35-36 [English abstract].
- Guderian, R. (1977) Discussion of the suitability of plant responses as a basis for air pollution control measures. In: *Air pollution: phytotoxicity of acidic gases and its significance in air pollution control*. Berlin, West Germany: Springer Verlag; pp. 75-97. (Ecological studies: analysis and synthesis v. 22).
- Heagle, A. S. (1982) Interactions between air pollutants and parasitic plant diseases. In: Unsworth, M. H.; Ormrod, D. P., eds. *Effects of gaseous air pollution in agriculture and horticulture*. London, United Kingdom: Butterworth Scientific; pp. 333-348.
- Heagle, A. S.; Heck, W. W. (1974) Predisposition of tobacco to oxidant air pollution injury by a previous exposure to oxidants. *Environ. Pollut.* 7: 247-251.
- Heagle, A. S.; Heck, W. W. (1980) Field methods to assess crop losses due to oxidant air pollutants. In: Teng, P. S.; Krupa, S. V., eds. *Crop loss assessment: proceedings of E. C. Stakman commemorative symposium*. University of Minnesota, Agricultural Experimental Station; miscellaneous publication no. 7; pp. 296-305.

References for Vegetation Effects (cont'd.)

- Heagle, A. S.; Johnston, J. W. (1979) Variable responses of soybean to mixtures of ozone and sulfur dioxide. *J. Air Pollut. Control Assoc.* 29: 729-732.
- Heagle, A. S.; Body, D. E.; Pounds, E. K. (1972) Effect of ozone on yield of sweet corn. *Phytopathology* 62: 683-687.
- Heagle, A. S., Body, D. E.; Neely, G. E. (1974) Injury and yield responses of soybean to chronic doses of ozone and sulfur dioxide in the field. *Phytopathology* 64: 132-136.
- Heagle, A. S.; Philbeck, R. B.; Knott, W. M. (1979a) Thresholds for injury growth, and yield loss caused by ozone on field corn hybrids. *Phytopathology* 69: 21-26.
- Heagle, A. S.; Philbeck, R. B.; Letchworth, M. B. (1979b) Injury and yield response of spinach cultivars to chronic doses of ozone in open-top field chambers. *J. Environ. Qual.* 8: 368-273.
- Heagle, A. S.; Letchworth, M. B.; Mitchell, C. A. (1983a) Effects of growth medium and fertilizer rate on the yield response of soybeans exposed to chronic doses of ozone. *Phytopathology* 73: 134-139.
- Heagle, A. S.; Heck, W. W.; Rawlings, J. O.; Philbeck, R. B. (1983b) Effects of chronic doses of ozone and sulfur dioxide on injury and yield of soybeans in open-top field chambers. *Crop Sci.* 23: 1184-1191.
- Heagle, A. S.; Cure, W. W.; Rawlings, J. O. (1985) Response of turnips to chronic doses of ozone in open-top field chambers. *Environ. Poll. (Series A)* 38: 305-320.
- Heck, W. W.; Tingey, D. T. (1971) Ozone time-concentration model to predict acute foliar injury. In: Englund, H. M.; Beery, W. T., eds. *Proceedings of the second international clean air congress; December 1970; Washington, DC.* New York, NY: Academic Press; pp. 249-255.
- Heck, W. W.; Dunning, J. A.; Hindawi, I. J. (1966) Ozone: nonlinear relation of dose and injury to plants. *Science (Washington, DC)* 151: 511-515.
- Heck, W. W.; Taylor, O. C.; Adams, R.; Bingham, G.; Miller, J.; Preston, E.; Weinstein, L. (1982) Assessment of crop loss from ozone. *J. Air Pollut. Control Assoc.* 32: 353-361.
- Heck, W. W.; Adams, R. M.; Cure, W. W.; Heagle, A. S.; Heggstad, H. E.; Kohut, R. J.; Kress, L. W.; Rawlings, I. O.; Taylor, O. C. (1983a) A reassessment of crop loss from ozone. *Environ. Sci. Technol.* 17: 573A-581A.
- Heck, W. W.; Taylor, O. C.; Adams, R. M.; Miller, J. E.; Weinstein, L. (1983b) National Crop Loss Assessment Network (NCLAN) 1982 Annual Report. Report to U.S. Environmental Protection Agency, Corvallis Environmental Research Laboratory, Corvallis, OR.

References for Vegetation Effects (cont'd.)

- Heck, W. W.; Cure, W. W.; Rawlings, J. O.; Zaragoza, L. J.; Heagle, A. S.; Heggstad, H. E.; Kohut, R. J.; Kress, L. W.; Temple, P. J.; (1984) Assessing impacts of ozone on agricultural crops: II. Crop yield functions and alternative exposure statistics. *J. Air Pollut. Control Assoc.* 34: 810-817.
- Heggstad, H. E. (1973) Photochemical air pollution injury to potatoes in the Atlantic coastal states. *Am. Potato J.* 50: 315-328.
- Heggstad, H. E.; Bennett, J. H. (1981) Photochemical oxidants potentiate yield losses in snap beans attributable to sulfur dioxide. *Science* (Washington, DC) 213: 1008-1010.
- Henderson, W. R.; Reinert, R. A. (1979) Yield response of four fresh market tomato cultivars after acute ozone exposure in the seedling stage. *J. Am. Soc. Hortic. Sci.* 104: 754-759.
- Hoffman, G. J.; Maas, E. V.; Rawlins, S. L. (1975) Salinity-ozone interactive effects on alfalfa yield and water relations. *J. Environ. Qual.* 4: 326-331.
- Hofstra, G.; Littlejohns, D. A.; Wukasch, R. T. (1978) The efficacy of the antioxidant ethylene-diurea (EDU) compared to carboxin and benomyl in reducing yield losses from ozone in navy bean. *Plant. Dis. Rep.* 62: 350-352.
- Hogsett, W. E.; Tingey, D. T.; Holman, S. R. (1985) A programmable exposure control system for determination of the effects of pollutant exposure regimes on plant growth. *Atmos. Environ.* 19: 1135-1145.
- Horsman, D. C.; Nicholls, A. O.; Calder, D. M. (1980) Growth responses of Dactylis glomerata, Lolium perenne and Phalaris aquatica to chronic ozone exposure. *Aust. J. Plant Physiol.* 7: 511-517.
- Howell, R. K.; Rose, L. P., Jr. (1980) Residual air pollution effects on soybean seed quality. *Plant Dis.* 64: 385-386.
- Howell, R. K.; Koch, E. J.; Rose, L. P., Jr. (1979) Field assessment of air pollution-induced soybean yield losses. *Agron. J.* 71: 285-288.
- Howitt, R. E.; Grossard, T. E.; Adams, R. M. (1984a) Effects of alternative ozone levels and response data on economic assessments: The case of California corps. *J. Air Pollut. Control Assoc.* 34: 1122-1127.
- Howitt, R. E.; Gossard, T. E.; Adams, R. M. (1984b) *Applied Welfare Economics and Public Policy*. New York: Prentice-Hall.
- Jacobson, J. S. (1977) The effects of photochemical oxidants on vegetation. In: *Ozon und Begleitsubstanzen im photochemischen Smog*. [Ozone and other substances in photochemical smog]: VDI colloquium; 1976; Düsseldorf, West Germany. Düsseldorf, West Germany: Verein deutscher Ingenieure (VDI) GmbH; pp. 163-173. (VDI-Berichte no. 270).

References for Vegetation Effects (cont'd.)

- Jacobson, J. S. (1982) Ozone and the growth and productivity of agricultural crops. In: Unsworth, M. H.; Ormrod, D. P., eds. Effects of gaseous air pollution in agriculture and horticulture. London, United Kingdom: Butterworth Scientific; pp. 293-304.
- Jacobson, J. S.; Colavito, L. J. (1976) The combined effect of sulfur dioxide and ozone on bean and tobacco plants. *Environ. Exp. Bot.* 16: 277-285.
- James, R. L.; Cobb, F. W., Jr.; Miller, P. R.; Parmeter, J. R., Jr. (1980) Effects of oxidant air pollution on susceptibility of pine roots to Fomes annosus. *Phytopathology* 70: 560-563.
- Johnston, J. W., Jr.; Heagle, A. S. (1982) Response of chronically ozonated soybean plants to an acute ozone exposure. *Phytopathology* 72: 387-389.
- Kopp, R. J.; Vaughan, W. J.; Hazilla, M. (1984) Agricultural sector benefits analysis for ozone: methods evaluation and demonstration. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards; EPA report no. EPA-450/5-84-003. Available from: NTIS, Springfield, VA; PB85-119477/XAB.
- Kress, L. W.; Miller, J. E. (1983) Impact of ozone on soybean yield. *J. Environ. Qual.* 12: 276-281.
- Kress, L. W.; Skelly, J. M. (1982) Response of several eastern forest tree species to chronic doses of ozone and nitrogen dioxide. *Plant Dis.* 66: 1149-1152.
- Kress, L. W., Miller, J. E.; Smith, H. J. (1985) Impact of ozone on winter wheat yield. *Environ. Exp. Bot.* 25: 211-228.
- Larsen, R. I.; Heck, W. W. (1976) An air quality data analysis system for interrelating effects, standards, and needed source reductions: Part 3. Vegetation injury. *J. Air Pollut. Control Assoc.* 26: 325-333.
- Larsen, R. I.; Heck, W. W. (1984) An air quality data analysis system for interrelating effects, standards, and needed source reductions: Part 8. An effective mean O₃ crop reduction mathematical model. *J. Air Pollut. Control Assoc.* 34: 1023-1034.
- Larsen, R. I.; Heagle, A. S.; Heck, W. W. (1983) An air quality data analysis system for interrelating effects, standards, and needed source reductions: Part 7. An O₃-SO₂ leaf injury mathematical model. *J. Air Pollut. Control Assoc.* 33: 198-207.
- Laurence, J. A. (1981) Effects of air pollutants on plant-pathogen interactions. *Z. Pflanzenkr. Pflanzenschutz* 87: 156-172.
- Lefohn, A. S.; Benedict, H. M. (1982) Development of a mathematical index that describes ozone concentration, frequency, and duration. *Atmos. Environ.* 16: 2529-2532.

References for Vegetation Effects (cont'd.)

- Lefohn, A. S.; Tingey, D. T. (1984) The co-occurrence of potentially phytotoxic concentrations of various gaseous air pollutants. *Atmos. Environ.* in press.
- Legassicke, B. C.; Ormrod, D. P. (1981) Suppression of ozone-injury on tomatoes by ethylene diurea in controlled environments and in the field. *HortScience* 16: 183-184.
- Leung, S. K.; Reed, W.; Geng, S. (1982) Estimations of ozone damage to selected crops grown in southern California. *J. Air Pollut. Control Assoc.* 32: 160-164.
- Loucks, O. L.; Armentano, T. V. (1982) Estimating crop yield effects from ambient air pollutants in the Ohio River Valley. *J. Air Pollut. Control Assoc.* 32: 146-150.
- MacLean, D. C.; Schneider, P. E. (1976) Photochemical oxidants in Yonkers, New York: effects on yield of bean and tomato. *J. Environ. Qual.* 5: 75-78.
- Male, L.; Preston, E.; Neely, G. (1983) Yield response curves of crops exposed to SO₂ time series. *Atmos. Environ.* 17: 1589-1593.
- Mann, L. K.; McLaughlin, S. B.; Shriner, D. S. (1980) Seasonal physiological responses of white pine under chronic air pollution stress. *Environ. Exp. Bot.* 20: 99-105.
- Manning, W. J.; Feder, W. A.; Perkins, I. (1970a) Ozone and infection of geranium flowers by Botrytis cinerea. *Phytopathology* 60: 1302.
- Manning, W. J.; Feder, W. A.; Perkins, I. (1970b) Ozone injury increases infection of geranium leaves by Botrytis cinerea. *Phytopathology* 60: 669-670.
- Manning, W. J.; Feder, W. A.; Vardaro, P. M. (1974) Suppression of oxidant injury by benomyl: effects on yields of bean cultivars in field. *J. Environ. Qual.* 2: 1-3.
- McLaughlin, S. B.; Taylor, G. E. (1981) Relative humidity: important modifier of pollutant uptake by plants. *Science (Washington, DC)* 22: 167-169.
- McLaughlin, S. B.; Shriner, D. S.; McConathy, R. K.; Mann, L. K. (1979) The effects of SO₂ dosage kinetics and exposure frequency on photosynthesis and transpiration of kidney beans (Phaseolus vulgaris L.). *Environ. Exp. Bot.* 19: 179-191.
- Menser, H. A.; Heggstad, H. E. (1966) Ozone and sulfur dioxide synergism: injury to tobacco plants. *Science (Washington, DC)* 153: 424-425.
- Menser, H. A.; Street, O. E. (1962) Effects of air pollution, nitrogen levels, supplemental irrigation, and plant spacing on weather fleck and leaf losses of Maryland tobacco. *Tobacco* 155: 192-196.

References for Vegetation Effects (cont'd.)

- Middleton, J. T.; Kendrick, J. B., Jr.; Schwalm, H. W. (1950) Injury to herbaceous plants by smog or air pollution. *Plant Dis. Rep.* 34: 245-252.
- Miller, P. R.; Taylor, O. C.; Wilhour, R. G. (1982) Oxidant air pollution effects on a western coniferous forest ecosystem. Corvallis, OR: Corvallis Environmental Research Laboratory; EPA report no. EPA-600/D-82-276. Available from: NTIS, Springfield, VA; PB83-189308.
- Mjelde, J. W.; Adams, R. M.; Dixon, B. L.; Garcia, P. (1984) Using farmers' actions to measure crop loss due to air pollution. *J. Air Pollut. Control Assoc.* 31: 360-364.
- Mukammal, E. I. (1965) Ozone as a cause of tobacco injury. *Agricultural Meteorology* 2: 145-165.
- Mumford, R. A.; Lipke, H.; Laufer, D. A.; Feder, W. A. (1972) Ozone-induced changes in corn pollen. *Environ. Sci. Technol.* 6: 427-430.
- Musselman, R. C.; Oshima, R. J.; Gallavan, R. E. (1983) Significance of pollutant concentration distribution in the response of 'red kidney' beans to ozone. *J. Am. Soc. Hortic. Sci.* 108: 347-351.
- Neely, G. E.; Tingey, D. T.; Wilhour, R. G. (1977) Effects of ozone and sulfur dioxide singly and in combination on yield, quality and N-fixation of alfalfa. In: Dimitriadis, B., ed. *International conference on photochemical oxidant pollution and its control: proceedings, volume II*; September 1976; Raleigh, NC. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Research and Development; pp. 663-673; EPA report no. EPA-600/3-77-0016b. Available from: NTIS, Springfield, VA; PB-264233.
- Nouchi, I.; Mayumi, H.; Yamazoe, F. (1984) Foliar injury response of petunia and kidney bean to simultaneous and alternate exposure to ozone and PAN. *Atmos. Environ.* 18: 453-460.
- Ogata, G.; Maas, E. V. (1973) Interactive effects of salinity and ozone on growth and yield of garden beet. *J. Environ. Qual.* 2: 518-520.
- Olszyk, D. M.; Tibbitts, T. W. (1981) Stomatal response and leaf injury of Pisum sativum L. with SO₂ and O₃ exposures. *Plant Physiol.* 67: 539-544.
- Ormrod, D. P. (1982) Air pollutant interactions in mixtures. In: Unsworth, M. H.; Ormrod, D. P., eds. *Effects of gaseous air pollution in agriculture and horticulture*. London, United Kingdom: Butterworth Scientific; pp. 307-331.
- Oshima, R. J. (1973) Effect of ozone on a commercial sweet corn variety. *Plant Dis. Rep.* 57: 719-723.
- Oshima, R. J. (1978) The impact of sulfur dioxide on vegetation: a sulfur dioxide-ozone response model. Sacramento, CA: California Air Resources Board; final report; agreement no. A6-162-30.

References for Vegetation Effects (cont'd.)

- Oshima, R. J.; Taylor, O. C.; Braegelmann, P. K.; Baldwin, D. W. (1975) Effect of ozone on the yield and plant biomass of a commercial variety of tomato. *J. Environ. Qual.* 4: 463-464.
- Oshima, R. J.; Braegelmann, P. K.; Baldwin, D. W.; Van Way, V.; Taylor, O. C. (1977a) Reduction of tomato fruit size and yield by ozone. *J. Am. Soc. Hortic. Sci.* 102: 289-293.
- Oshima, R. J.; Braegelmann, P. K.; Baldwin, D. W.; Van Way, V.; Taylor, O. C. (1977b) Responses of five cultivars of fresh market tomato to ozone: a contrast of cultivar screening with foliar injury and yield. *J. Am. Soc. Hortic. Sci.* 102: 286-289.
- Oshima, R. J.; Bennett, J. P.; Braegelmann, P. K. (1978) Effect of ozone on growth and assimilate partitioning in parsley. *J. Am. Soc. Hortic. Sci.* 103: 348-350.
- Oshima, R. J.; Braegelmann, P. K.; Flagler, R. B.; Teso, R. R. (1979) The effects of ozone on the growth, yield, and partitioning of dry matter in cotton. *J. Environ. Qual.* 8: 474-479.
- Page, W. P.; Arbogast, G.; Fabian, R. G.; Ciecka, J. (1982) Estimation of economic losses to the agricultural sector from airborne residuals in the Ohio River Basin region. *J. Air Pollut. Control Assoc.* 32: 151-154.
- Patton, R. L. (1981) Effects of ozone and sulfur dioxide on height and stem specific gravity of *Populus* hybrids. Broomall, PA: Northeastern Forest Experiment Station; FSRP-NE-471. Available from: NTIS, Springfield, VA; PB81-171274.
- Pell, E. J.; Pearson, N. S. (1983) Ozone-induced reduction in quantity of ribulose-1,5-biphosphate carboxylase in alfalfa foliage. *Plant Physiol.* 73: 185-187.
- Pell, E. J.; Weissberger, W. C.; Speroni, J. J. (1980) Impact of ozone on quantity and quality of greenhouse-grown potato plants. *Environ. Sci. Technol.* 14: 568-571.
- Rawlings, J. O.; Cure, W. W. (1985) The Weibull function as a dose-response model to describe ozone effects on crop yields. *Crop Science* (in press).
- Reich, P. B.; Amundson, R. G. (1984) Low level O_3 and/or SO_2 exposure causes a linear decline in soybean yield. *Environ. Pollut. (Series A)* 34: 345-355.
- Reinert, R. A.; Nelson, P. V. (1979) Sensitivity and growth of twelve elatior begonia cultivars to ozone. *HortScience* 14: 747-748.
- Reinert, R. A.; Heagle, A. S.; Heck, W. W. (1975) Plant responses to pollutant combinations. Mudd, B.; Kozłowski, T. T., eds. *Responses of plants to air pollution*. New York, NY: Academic Press, Inc.; pp. 159-177.

References for Vegetation Effects (cont'd.)

- Rowe, R. P.; Chestnut, L. G.; Miller, C.; Adams, R. M.; Thresher, M.; Mason, H. O.; Howitt, R. E.; Trijonis, J. (1984) Economic assessment of the effect of air pollution in the San Joaquin Valley: draft report to the Research Division, California Air Resources Board. Boulder, CO: Energy and Resource Consultants, Inc.
- Runeckles, V. C.; Rosen, P. M. (1977) Effects of ambient ozone pretreatment on transpiration and susceptibility to ozone injury. *Can. J. Bot.* 55: 193-197.
- Ryan, J. W.; Loehman, E.; Lee, W.; Trondsen, E.; Bland, M.; Goen, R.; Ludwig, F.; Eger, T.; Eigsti, S.; Conley, D.; Cummings, R. (1981) An estimate of the nonhealth benefits of meeting the secondary national ambient air quality standards: final report. Menlo Park, CA: SRI International; project no. 2094.
- Shannon, J. G.; Mulchi, C. L. (1974) Ozone damage to wheat varieties at anthesis. *Crop Sci.* 14: 335-337.
- Shriner, D. S.; Cure, W. W.; Heagle, A. S.; Heck, W. W.; Johnson, D. W.; Olson, R. J.; Skelly, J. M. (1982) An analysis of potential agriculture and forestry impacts of long-range transport air pollutants. Oak Ridge, TN: Oak Ridge National Laboratory; ORNL report no. 5910.
- Stan, H-J.; Schicker, S. (1982) Effect of repetitive ozone treatment on bean plants-stress ethylene production and leaf necrosis. *Atmos. Environ.* 16: 2267-2270.
- Taylor, O. C. (1969) Importance of peroxyacetyl nitrate (PAN) as a phytotoxic air pollutant. *J. Air Pollut. Control Assoc.* 19: 347-351.
- Taylor, O. C. (1974) Air pollutant effects influenced by plant-environmental interaction. In: Dugger, W. M., ed. *Air pollution effects on plant growth.* ACS Symp. Ser. 3: 1-7.
- Taylor, O. C.; Dugger, W. M.; Cardiff, E. A.; Darley, E. F. (1961) Interaction of light and atmospheric photochemical products ("smog") within plants. *Nature (London)* 192: 814-816.
- Taylor, O. C.; Temple, P. J.; Thill, A. J. (1983) Growth and yield responses of selected crops to peroxyacetyl nitrate. *HortScience* 18: 861-863.
- Temple, P. J.; Bisessar, S. (1979) Response of white bean to bacterial blight, ozone, and antioxidant protection in the field. *Phytopathology* 69: 101-103.
- Thomas, M. D.; Hendricks, R. H.; Hill, G. R. (1950) Sulfur metabolism of plants: effects of sulfur dioxide on vegetation. *Indust. Eng. Chem.* 42: 2231-2235.
- Thompson, C. R.; Kats, G.; Cameron, J. W. (1976a) Effects of photochemical air pollutants on growth, yield, and ear characteristics of two sweet corn hybrids. *J. Environ. Qual.* 5: 410-412.

References for Vegetation Effects (cont'd.)

- Thompson, C. R.; Kats, G.; Pippen, E. L.; Isom, W. H. (1976b) Effect of photochemical air pollution on two varieties of alfalfa. *Environ. Sci. Technol.* 10: 1237-1241.
- Tingey, D. T. (1977) Ozone-induced alterations in plant growth and metabolism. In: Dimitriadis, B., ed. International conference on photochemical oxidant pollution and its control: proceedings, v. II; Research Triangle Park, NC: U.S. Environmental Protection Agency; pp. 601-609; Report no. EPA-600/3-77-001b.
- Tingey, D. T.; Reinert, R. A. (1975) The effect of ozone and sulfur dioxide singly and in combination on plant growth. *Environ. Pollut.* 9: 117-125.
- Tingey, D. T.; Taylor, G. E., Jr. (1982) Variation in plant response to ozone: a conceptual model of physiological events. In: Unsworth, M. H.; Ormrod, D. P., eds. Effects of gaseous air pollution in agriculture and horticulture. London, United Kingdom: Butterworth Scientific; pp. 113-138.
- Tingey, D. T.; Heck, W. W.; Reinert, R. A. (1971a) Effect of low concentrations of ozone and sulfur dioxide on foliage, growth and yield of radish. *J. Am. Soc. Hortic. Sci.* 96: 369-371.
- Tingey, D. T.; Thutt, G. L.; Gumpertz, M. L.; Hogsett, W. E. (1982) Plant water status influences ozone sensitivity of bean plants. *Agric. Environ.* 7: 243-254.
- Tonneijck, A. E. G. (1984) Effects of peroxyacetyl nitrate (PAN) and ozone on some plant species. In: Proceedings of the OECD workshop on ozone; Gothenburg, Sweden.
- U.S. Environmental Protection Agency. (1978) Air quality criteria for ozone and other photochemical oxidants. Research Triangle Park, NC: U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office; EPA report no. EPA-600/8-78-004. Available from: NTIS, Springfield, VA; PB80-124753.
- Walmsley, L.; Ashmore, M. L.; Bell, J. N. B. (1980) Adaptation of radish Raphanus sativus L. in response to continuous exposure to ozone. *Environ. Pollut.* 23: 165-177.
- Wilhour, R. G.; Neely, G. E. (1977) Growth response of conifer seedlings to low ozone concentrations. In: Dimitriadis, B., ed. International conference on photochemical oxidant pollution and its control: proceedings, vol. II; January; Research Triangle Park, NC. Research Triangle Park, NC: U.S. Environmental Protection Agency; pp. 635-645; EPA report no. EPA-600/3-77-001b. Available from: NTIS, Springfield, VA; PB-264233.
- Wukasch, R. T.; Hofstra, G. (1977a) Ozone and Botrytis interactions in onion-leaf dieback: open-top chamber studies. *Phytopathology* 67: 1080-1084.

References for Vegetation Effects (cont'd.)

- Wukasch, R. T.; Hofstra, G. (1977b) Ozone and *Botrytis* spp. interaction in onion-leaf dieback: field studies. *J. Am. Soc. Hortic. Sci.* 102: 543-546.
- Yang, Y.-S.; Skelly, J. M.; Chevone, B. I.; Birch, J. B. (1983) Effects of long-term ozone exposure on photosynthesis and dark respiration of eastern white pine. *Environ. Sci. Technol.* 17: 371-373.

1.12.6 References for Effects of Ozone on Natural Ecosystems and Their Components

- Adams, H. S.; Stephenson, S. L.; Blasing, T. J.; Duvick, D. N. (1985) Growth-trend declines of spruce and fir in mid-Appalachian subalpine forests. *Environ. and Exptl. Bot.* 25: 315-325.
- Barnes, R. L. (1972) Effects of chronic exposure to ozone on photosynthesis and respiration of pines. *Environ. Pollut.* 3: 133-138.
- Benoit, L. F.; Skelly, J. M.; Moore, L. D.; Dochinger, L. S. (1982) Radial growth reductions of *Pinus strobus* L. correlated with foliar ozone sensitivity as an indicator of ozone-induced losses in eastern forests. *Can. J. For. Res.* 12: 673-678.
- Bormann, F. H. (1985) Air pollution and forests: an ecosystem perspective. *BioScience* 35: 434-441.
- Botkin, D. B.; Smith, W. H.; Carlson, R. W.; Smith, T. L. (1972) Effects of ozone on white pine saplings: variation in inhibition and recovery of net photosynthesis. *Environ. Pollut.* 3: 273-289.
- Carlson, R. W. (1979) Reduction in the photosynthetic rate of *Acer*, *Quercus*, and *Fraxinus* species caused by sulfur dioxide and ozone. *Environ. Pollut.* 18: 159-170.
- Cowling, E. B. (1985) Effects of air pollution on forests: Critical review discussion papers. *J. Air Pollut. Control Assoc.* 35: 916-919.
- Coyne, P. E.; Bingham, G. E. (1981) Comparative ozone dose response of gas exchange in a ponderosa pine stand exposed to long-term fumigations. *J. Air Pollut. Control Assoc.* 31: 38-41.
- Dahlsten, D. L.; Rowney, D. L. (1980) Influence of air pollution on population dynamics of forest insects and on tree mortality. In: Miller, P. R., ed. *Proceedings of the symposium on effects of air pollutants on Mediterranean and temperate forest ecosystems*; June; Riverside, CA. Berkeley, CA: U.S. Department of Agriculture; Forest Service general technical report no. PSW-43; pp. 125-130. Available from: NTIS, Springfield, VA; PB81-133720.
- Dochinger, L. S.; Townsend, A. M. (1979) Effects of roadside deicer salts and ozone on red maple progenies. *Environ. Pollut.* 19: 229-237.

References for Ecosystem Effects (cont'd.)

- Duchelle, S. F.; Skelly, J. M.; Sharick, T. L.; Chevone, B. I.; Yang, Y.-S.; Nellesen, J. E. (1983) Effects of ozone on the productivity of natural vegetation in a high meadow of the Shenandoah National Park of Virginia. *J. Environ. Manage.* 17: 299-308.
- Ehrlich, P. R.; Mooney, H. A. (1983) Extinction, substitution, and ecosystem services. *BioScience* 33: 248-254.
- Hacsckaylo, E. (1972) Mycorrhizae: the ultimate in reciprocal parasitism? *BioScience* 22: 577-583.
- Hacsckaylo, E. (1973) The Torrey symposium on current aspects of fungal development; IV. Dependence of mycorrhizal fungi on hosts. *Bull. Torrey Bot. Club* 100: 217-223.
- Hogsett, W. E.; Plocher, M.; Wildman, V.; Tingey, D. T.; Bennett, I. P. (1985) Growth response of two varieties of slash pine seedlings to chronic ozone exposures. *Can. J. Bot.* 63: 2369-2376.
- James, R. L.; Cobb, F. W., Jr.; Wilcox, W. W.; Rowney, D. L. (1980) Effects of photochemical oxidant injury of ponderosa and Jeffrey pines on susceptibility of sapwood and freshly cut stumps to Fomes annosus. *Phytopathology* 70: 704-708.
- Johnson, A. H.; Siccama, T. G. (1983) Acid deposition and forest decline. *Environ. Sci. Technol.* 17: 294A-305A.
- Krause, G. H. M.; Prinz, B.; Jung, K. D. (1984) Forest effects in West Germany. In: Davis, D. D.; Millen, A. A.; Dochinger, L., eds. *Air pollution and the productivity of the forest: proceedings of the symposium; October 1983; Washington, DC.* Arlington, VA: Izaak Walton League of America; pp. 297-332.
- Kress, L. W.; Skelly, J. M. (1982) Response of several eastern forest tree species to chronic doses of ozone and nitrogen dioxide. *Plant Dis.* 66: 1149-1152.
- Kress, L. W.; Skelly, J. M.; Hinkelmann, K.H. (1982) Growth impact of O₃, NO₂, and/or SO₂ on Platanus occidentalis. *Agric. Environ.* 7: 265-274.
- Laurence, J. A.; Weinstein, L. H. (1981) Effects of air pollutants on plant productivity. *Annu. Rev. Phytopathol.* 19: 257-271.
- Mahoney, M. J. (1982) An analysis of the potential effects of air pollutants emitted during coal combustion on yellow poplar and loblolly pine and influences on mycorrhizal associations of loblolly pine. Unpublished. Blacksburg, VA: Virginia Polytechnical Institute and State University; Ph.D. Thesis.
- Manion, P. D. (1985) Effects of air pollution on forests: Critical review discussion papers. *J. Air Pollut. Control Assoc.* 35: 919-922.

References for Ecosystem Effects (cont'd.)

- Mann, L. K.; McLaughlin, S. B.; Shriner, D. S. (1980) Seasonal physiological responses of white pine under chronic air pollution stress. *Environ. Exp. Bot.* 20: 99-105.
- McBride, J. R.; Semion, V.; Miller, P. R. (1975) Impact of air pollution on the growth of ponderosa pine. *Calif. Agric.* 29: 8-10.
- McCool, P. M.; Menge, J. A.; Taylor, O. C. (1979) Effects of ozone and HCl gas on the development of the mycorrhizal fungus Glomus fasciculatus and growth of 'Troyer' citrange. *J. Amer. Soc. Hort. Sci.* 104: 151-154.
- McLaughlin, S. B.; McConathy, R. K.; Duvick, D.; Mann, L. K. (1982) Effects of chronic air pollution stress on photosynthesis, carbon allocation, and growth of white pine trees. *For. Sci.* 28: 60-70.
- Miller, P. L. (1973) Oxidant-induced community change in a mixed conifer forest. In: Naegele, J. A., ed. *Air pollution damage to vegetation*. Washington, DC: American Chemical Society; pp. 101-117. (Advances in chemistry series: no. 122.)
- Miller, P. R.; Elderman, M. J., eds. (1977) Photochemical oxidant air pollutant effects on a mixed conifer forest ecosystem: a progress report, 1976. Corvallis, OR: U.S. Environmental Protection Agency; EPA report no. EPA-600/3-77-104. Available from: NTIS, Springfield, VA; PB-274531.
- Miller, P. R.; Parmeter, J. R., Jr.; Taylor, O. C.; Cardiff, E. A. (1963) Ozone injury to the foliage of Pinus ponderosa. *Phytopathology* 53: 1072-1076.
- Miller, P. R.; Parmeter, J. R., Jr.; Flick, B. H.; Martinez, C. W. (1969) Ozone dosage response of ponderosa pine seedlings. *J. Air Pollut. Control Assoc.* 19: 435-438.
- Miller, P. R.; Taylor, O. C.; Wilhour, R. G. (1982) Oxidant air pollution effects on a western coniferous forest ecosystem. Corvallis, OR: Corvallis Environmental Research Laboratory; EPA report no. EPA-600/D-82-276. Available from: NTIS, Springfield, VA; PB83-189308.
- Mooi, J. (1980) Influence of ozone on the growth of two poplar cultivars. *Plant Dis.* 64: 772-773.
- National Park Service. (1985) Testimony of the National Park Service before the Subcommittee on Parks and Recreation. Washington, D.C.: U.S. House of Representatives, Committee on Interior and Insular Affairs; 99th Congress, 1st session.
- National Research Council. (1977) Ozone and other photochemical oxidants. Washington, D.C.: National Academy of Sciences; pp. 437-585.
- Odum, E. P. (1985) Trends expected in stressed ecosystems. *BioScience*. 35: 419-422.

References for Ecosystem Effects (cont'd.)

- Parmeter, J. R., Jr.; Bega, R. V.; Neff, T. (1962) A chlorotic decline of ponderosa pine in southern California. *Plant Dis. Rep.* 46: 269-273.
- Patton, R. L. (1981) Effects of ozone and sulfur dioxide on height and stem specific gravity of Populus hybrids. *For. Sci. Res. Pop.* NE-471.
- Price, H.; Treshow, M. (1972) Effects of ozone on the growth and reproduction of grasses. In: *Proceedings of the international air pollution conference; Melbourne, Australia.* pp. 275-280.
- Reich, P. B.; Amundson, R. G. (1985) Ambient levels of ozone reduce net photosynthesis in tree and crop species. *Science.* 230: 566-570.
- Sigal, L. L.; Nash, T. H. (1983) Lichen communities on conifers in southern California mountains: an ecological survey relative to oxidant air pollution. *Ecology* 64: 1343-1354.
- Stark, R. W.; Cobb, F. W., Jr. (1969) Smog injury, root diseases and bark beetle damage in ponderosa pine. *Calif. Agric.* 23: 13-15.
- Tingey, D. T.; Wilhour, R. G.; Standley, E. (1976) The effect of chronic ozone exposures on the metabolite content of ponderosa pine seedlings. *For. Sci.* 22: 234-241.
- Treshow, M.; Stewart, D. (1973) Ozone sensitivity of plants in natural communities. *Biol. Conserv.* 5: 205-214.
- U.S. Environmental Protection Agency (1978) Air quality criteria for ozone and other photochemical oxidants. Research Triangle Park, NC: U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office; Report no. EPA-600/8-78-004. Available from: NTIS, Springfield, VA; PB83-163337.
- Woodwell, G. M. (1970) Effects of pollution on the structure and physiology of ecosystems. *Science (Washington, DC)* 168: 429-433.
- Yang, Y.-S.; Skelly, J. M.; Chevone, B. I.; Birch, J. B. (1983) Effects of long-term ozone exposure on photosynthesis and dark respiration of eastern white pine. *Environ. Sci. Technol.* 17: 371-373.

1.12.7 References for Effects of Ozone and Other Photochemical Oxidants on Nonbiological Materials

- Beloin, N. J. (1972) Fading of dyed fabrics by air pollution: a field study. *Text. Chem. Color.* 4: 77-78.
- Beloin, N. J. (1973) Fading of dyed fabrics exposed to air pollutants: a chamber study. *Text. Chem. Color.* 5: 128-133.

References for Nonbiological Materials (cont'd.)

- Bogaty, H.; Campbell, K. S.; Appel, W. D. (1952) The oxidation of cellulose by ozone in small concentrations. Text. Res. J. 22: 81-83.
- Bradley, C. E.; Haagen-Smit, A. J. (1951) The application of rubber in the quantitative determination of ozone. Rubber Chem. Technol. 24: 750-775.
- Edwards, D. C.; Storey, E. B. (1959) A quantitative ozone test for small specimens. Chem. Can. 11: 34-38.
- Haylock, J. C.; Rush, J. L. (1976) Studies on the ozone fading of anthraquinone dyes on nylon fibers. Text. Res. J. 46: 1-8.
- Haylock, J. C.; Rush, J. L. (1978) Studies on the ozone fading of anthraquinone dyes on nylon fibers; part II: in-service performance. Text. Res. J. 48: 143-149.
- Haynie, F. H.; Spence, J. W.; Upham, J. B. (1976) Effects of gaseous pollutants on materials--a chamber study. Research Triangle Park, NC: U.S. Environmental Protection Agency, Environmental Sciences Research Laboratory; EPA report no. EPA-600/3-76-015. Available from: NTIS, Springfield, VA; PB-251580.
- Heuvel, H. M.; Huisman, R.; Schmidt, H. M. (1978) Ozone fading of disperse blue 3 on nylon 6 fibers. The influence of physical fiber properties. Text. Res. J. 48: 376-384.
- Kamath, Y. K.; Ruetsch, S. B.; Weigmann, H.-D. (1982) Microspectrophotometric study of ozone fading of disperse dyes in nylon. Text. Res. J.: 53: 391-402.
- Kerr, N.; Morris, M. A.; Zeronian, S. H. (1969) The effect of ozone and laundering on a vat-dyed cotton fabric. Am. Dyest. Rep. 58: 34-36.
- McCarthy, E. F.; Stankunas, A. R.; Yocom, J. E.; Rae, D. (1983) Damage cost models for pollution effects on material. Research Triangle Park, NC: U.S. Environmental Protection Agency, Environmental Sciences Research Laboratory; EPA report no. EPA-600/3-84-012. Available from: NTIS, Springfield, VA; PB-140342.
- Nipe, M. R. (1981) Atmospheric contaminant fading. Text. Chem. Color. 13: 18-28.
- Salvin, V. S. (1969) Ozone fading of dyes. Text. Chem. Color. 1: 245-251.
- Salvin, V. S.; Walker, R. A. (1955) Service fading of disperse dyestuffs by chemical agents other than the oxides of nitrogen. Text. Res. J. 25: 571-585.
- Schmitt, C. H. A. (1960) Lightfastness of dyestuffs on textiles. Am. Dyest. Rep. 49: 974-980.

References for Nonbiological Materials (cont'd.)

- Schmitt, C. H. A. (1962) Daylight fastness testing by the Langley system. *Am. Dyest. Rep.* 51: 664-675.
- Upham, J. B.; Haynie, F. H.; Spence, J. W. (1976) Fading of selected drapery fabrics by air pollutants. *J. Air Pollut. Control Assoc.* 26: 790-792.
- Veith, A. G.; Evans, R. L. (1980) Effect of atmospheric pressure on ozone cracking of rubber. *Polym. Testing* 1: 27-38.
- Wenghoefer, H. M. (1974) Environmental effects on RFL adhesion. *Rubber Chem. Technol.* 47: 1066-1073.
- Yocom, J. E.; Kawicki, J. M.; Hoffnagle, G. F. (1985) Estimating materials damage from oxidant pollutants. In: *Proceedings of the APCA Specialty Conference: evaluation of the scientific Basis for Ozone/Oxidants Standards*, Houston, TX, November 28-30, 1984. Pittsburgh, PA: Air Pollution Control Association.

1.12.8 References for Toxicological Effects of Ozone and Other Photochemical Oxidants

- Abraham, W. M.; Januszkiewicz, A. J.; Mingle, M.; Welker, M.; Wanner, A.; Sackner, M. A. (1980) Sensitivity of bronchoprovocation and tracheal mucous velocity in detecting airway responses to O₃. *J. Appl. Physiol.: Respir. Environ. Exercise Physiol.* 48: 789-793.
- Abraham, W. M.; Lauredo, I.; Sielczak, M.; Yerger, L.; King, M. M.; Ratzan, K. (1982) Enhancement of bacterial pneumonia in sheep by ozone exposure. *Am. Rev. Respir. Dis. Suppl.* 125(4 pt. 2): 148.
- Abraham, W.; Chapman, G. A.; Marchette, B. (1984a) Differences between inhaled and intravenous carbachol in detecting O₃-induced airway effects. *Environ. Res.* 35: 430-438.
- Abraham, W. M.; Delehunt, J. C.; Yerger, L.; Marchette, B.; Oliver, W., Jr. (1984b) Changes in airway permeability and responsiveness after exposure to ozone. *Environ. Res.* 34: 110-119.
- Aharonson, E. F.; Menkes, H.; Gurtner, G.; Swift, D. L.; Proctor, D. F. (1974) Effect of respiratory airflow rate on removal of soluble vapors by the nose. *J. Appl. Physiol.* 37: 654-657.
- Alpert, S. M.; Schwartz, B. B.; Lee, S. D.; Lewis, T. R. (1971a) Alveolar protein accumulation: a sensitive indicator of low level oxidant toxicity. *Arch. Intern. Med.* 128: 69-73.
- Alpert, S. M.; Gardner, D. E.; Hurst, D. J.; Lewis, T. R.; Coffin, D. L. (1971b) Effects of exposure to ozone on defensive mechanisms of the lung. *J. Appl. Physiol.* 31: 247-252.

References for Toxicological Effects (cont'd.)

- Amdur, M. O.; Ugro, V.; Underhill, D. W. (1978) Respiratory response of guinea pigs to ozone alone and with sulfur dioxide. *Am. Ind. Hyg. Assoc. J.* 39: 958-961.
- Amoruso, M. A.; Witz, G.; Goldstein, B. D. (1981) Decreased superoxide anion radical production by rat alveolar macrophages following inhalation of ozone or nitrogen dioxide. *Life Sci.* 28: 2215-2221.
- Aranyi, C.; Vana, S. C.; Thomas, P. T.; Bradof, J. N.; Fenters, J. D.; Graham, J. A.; Miller, F. J. (1983) Effects of subchronic exposure to a mixture of O₃, SO₂, and (NH₄)₂SO₄ on host defenses of mice. *J. Toxicol. Environ. Health* 12: 55-71.
- Atwal, O. S.; Pemsingh, R. S. (1981) Morphology of microvascular changes and endothelial regeneration in experimental ozone-induced parathyroiditis. III. Some pathologic considerations. *Am. J. Pathol.* 102: 297-307.
- Atwal, O. S.; Pemsingh, R. S. (1984) Occurrence of mallery body-like inclusions in parathyroid chief cells of ozone-treated dogs. *J. Pathol.* 142: 169-174.
- Atwal, O. S.; Wilson, T. (1974) Parathyroid gland changes following ozone inhalation: a morphologic study. *Arch. Environ. Health* 28: 91-100.
- Atwal, O. S.; Samagh, B. S.; Bhatnagar, M. K. (1975) A possible autoimmune parathyroiditis following ozone inhalation. II. A histopathologic, ultrastructural, and immunofluorescent study. *Am. J. Pathol.* 80: 53-68.
- Barry, B. E.; Miller, F. J.; Crapo, J. D. (1983) Alveolar epithelial injury caused by inhalation of 0.25 ppm of ozone. In: Lee, S. D.; Mustafa, M. G.; Mehlman, M. A., eds. *International symposium on the biomedical effects of ozone and related photochemical oxidants; March 1982; Pinehurst, NC. Princeton, NJ: Princeton Scientific Publishers, Inc; pp. 299-309. (Advances in modern environmental toxicology: v. 5).*
- Bartlett, D., Jr.; Faulkner, C. S. II; Cook, K. (1974) Effect of chronic ozone exposure on lung elasticity in young rats. *J. Appl. Physiol.* 37: 92-96.
- Bergers, W. W. A.; Gerbrandy, J. L. F.; Stap, J. G. M. M.; Dura, E. A. (1983) Influence of air polluting components viz ozone and the open air factor on host-resistance towards respiratory infection. In: Lee, S. D.; Mustafa, M. G.; Mehlman, M. A., eds. *International symposium on the biomedical effects of ozone and related photochemical oxidants; March 1982; Pinehurst, NC. Princeton, NJ: Princeton Scientific Publishers, Inc.; pp. 459-467. (Advances in modern environmental toxicology: v. 5).*
- Berliner, J. A.; Kuda, A.; Mustafa, M. G.; Tierney, T. F. (1978) Pulmonary morphologic studies of ozone tolerance in the rat (a possible mechanism for tolerance). *Scanning Electron Microsc.* 2: 879-884.

References for Toxicological Effects (cont'd.)

- Bhatnagar, R. S.; Hussain, M. Z.; Sorensen, K. R.; Mustafa, M. G.; von Dohlen, F. M.; Lee, S. D. (1983) Effect of ozone on lung collagen biosynthesis. In: Lee, S. D.; Mustafa, M. G.; Mehlman, M. A., eds. International symposium on the biomedical effects of ozone and related photochemical oxidants; March 1982; Pinehurst, NC. Princeton, NJ: Princeton Scientific Publishers, Inc.; pp. 311-321. (Advances in modern environmental toxicology: v. 5).
- Boatman, E. S.; Sato, S.; Frank, R. (1974) Acute effects of ozone on cat lungs. II. Structural. *Am. Rev. Respir. Dis.* 110: 157-169.
- Boche, R. D.; Quilligan, J. J., Jr. (1960) Effects of synthetic smog on spontaneous activity of mice. *Science (Washington, DC)* 131: 1733-1734.
- Boorman, G. A.; Schwartz, L. W.; McQuillen, N. K.; Brummer, M. E. G. (1977) Pulmonary response following long-term intermittent exposure to ozone: structural and morphometric changes. *Am. Rev. Respir. Dis.* 115: 201.
- Boorman, G. A.; Schwartz, L. W.; Dungworth, D. L. (1980) Pulmonary effects of prolonged ozone insult in rats: morphometric evaluation of the central acinus. *Lab. Invest.* 43: 108-115.
- Bradley, M. O.; Erickson, L. C. (1981) Comparison of the effects of hydrogen peroxide and X-ray irradiation on toxicity, mutation, and DNA damage/repair in mammalian cells (V-79). *Biochim. Biophys. Acta* 654: 135-141.
- Bradley, M. O.; Hsu, I. C.; Harris, C. C. (1979) Relationships between sister chromatid exchange and mutagenicity, toxicity, and DNA damage. *Nature (London)* 282: 318-320.
- Brinkman, R.; Lamberts, H. B.; Veninga, T. S. (1964) Radiomimetic toxicity of ozonised air. *Lancet (7325)*: 133-136.
- Brummer, M. E. G.; Schwartz, L. W.; McQuillen, N. K. (1977) A quantitative study of lung damage by scanning electron microscopy: inflammatory cell response to high-ambient levels of ozone. *Scanning Electron Microsc.* 2: 513-518.
- Calabrese, E. J.; Moore, G. S.; Grunwald, E. L. (1983) Ozone-induced decrease of erythrocyte survival in adult rabbits. In: Lee, S. D.; Mustafa, M. G.; Mehlman, M. A., eds. International symposium on the biomedical effects of ozone and related photochemical oxidants; March 1982; Pinehurst, NC. Princeton, NJ: Princeton Scientific Publishers, Inc.; pp. 103-117. (Advances in modern environmental toxicology: v. 5).
- Campbell, K. I.; Hilsenroth, R. H. (1976) Impaired resistance to toxin in toxoid-immunized mice exposed to ozone or nitrogen dioxide. *Clin. Toxicol.* 9: 943-954.
- Campbell, K. I.; Clarke, G. L.; Emik, L. O.; Plata, R. L. (1967) The atmospheric contaminant peroxyacetyl nitrate. *Arch. Environ. Health* 15: 739-744.

References for Toxicological Effects (cont'd.)

- Castleman, W. L.; Dungworth, D. L.; Tyler, W. S. (1973a) Cytochemically detected alterations of lung acid phosphatase reactivity following ozone exposure. *Lab. Invest.* 29: 310-319.
- Castleman, W. L.; Dungworth, D. L.; Tyler, W. S. (1973b) Histochemically detected enzymatic alterations in rat lung exposed to ozone. *Exp. Mol. Pathol.* 19: 402-421.
- Castleman, W. L.; Tyler, W. S.; Dungworth, D. L. (1977) Lesions in respiratory bronchioles and conducting airways of monkeys exposed to ambient levels of ozone. *Exp. Mol. Pathol.* 26: 384-400.
- Castleman, W. L.; Dungworth, D. L.; Schwartz, L. W.; Tyler, W. S. (1980) Acute respiratory bronchiolitis: an ultrastructural and autoradiographic study of epithelial cell injury and renewal in rhesus monkeys exposed to ozone. *Am. J. Pathol.* 98: 811-840.
- Cavender, F. L.; Steinhagen, W. H.; Ulrich, C. E.; Busey, W. M.; Cockrell, B. Y.; Haseman, J. K.; Hogan, M. D.; Drew, R. T. (1977) Effects in rats and guinea pigs of short-term exposures to sulfuric acid mist, ozone, and their combination. *J. Toxicol. Environ. Health* 3: 521-533.
- Cavender, F. L.; Singh, B.; Cockrell, B. Y. (1978) Effects in rats and guinea pigs of six-month exposures to sulfuric acid mist, ozone, and their combination. *J. Toxicol. Environ. Health* 4: 845-852.
- Chow, C. K. (1976) Biochemical responses in lungs of ozone-tolerant rats. *Nature (London)* 260: 721-722.
- Chow, C. K. (1984) Glutathione peroxidase system in the lungs of ozone-tolerant and non-tolerant rats. In: Bors, W.; Saran, M.; Tait, D., eds. *Oxygen radicals in chemistry and biology; proceedings of the third international conference; July 1983; Neuherberg, West Germany. Berlin, West Germany: Walter de Gruyter & Co.; pp. 707-712.*
- Chow, C. K.; Kaneko, J. J. (1979) Influence of dietary vitamin E on the red cells of ozone-exposed rats. *Environ. Res.* 19: 49-55.
- Chow, C. K.; Tappel, A. L. (1972) An enzymatic protective mechanism against lipid peroxidation damage to lungs of ozone-exposed rats. *Lipids* 7: 518-524.
- Chow, C. K.; Tappel, A. L. (1973) Activities of pentose shunt and glycolytic enzymes in lungs of ozone-exposed rats. *Arch. Environ. Health* 26: 205-208.
- Chow, C. K.; Dillard, C. J.; Tappel, A. L. (1974) Glutathione peroxidase system and lysozyme in rats exposed to ozone or nitrogen dioxide. *Environ. Res.* 7: 311-319.
- Chow, C. K.; Mustafa, M. G.; Cross, C. E.; Tarkington, B. K. (1975) Effects of ozone exposure on the lungs and the erythrocytes of rats and monkeys: relative biochemical changes. *Environ. Physiol. Biochem.* 5: 142-146.

References for Toxicological Effects (cont'd.)

- Chow, C. K.; Hussain, M. Z.; Cross, C. E.; Dungworth, D. L.; Mustafa, M. G. (1976) Effect of low levels of ozone on rat lungs. I. Biochemical responses during recovery and reexposure. *Exp. Mol. Pathol.* 25: 182-188.
- Chow, C. K.; Cross, C. E.; Kaneko, J. J. (1977) Lactate dehydrogenase activity and isoenzyme pattern in lungs, erythrocytes, and plasma of ozone-exposed rats and monkeys. *J. Toxicol. Environ. Health* 3: 877-884.
- Chow, C. K.; Plopper, C. G.; Chiu, M.; Dungworth, D. L. (1981) Dietary vitamin E and pulmonary biochemical and morphological alterations of rats exposed to 0.1 ppm ozone. *Environ. Res.* 24: 315-324.
- Christensen, E.; Giese, A. C. (1954) Changes in absorption spectra of nucleic acids and their derivatives following exposure to ozone and ultraviolet radiation. *Arch. Biochem. Biophys.* 51: 208-216.
- Clark, K. W.; Posin, C. I.; Buckley, R. D. (1978) Biochemical response of squirrel monkeys to ozone. *J. Toxicol. Environ. Health* 4: 741-753.
- Clemons, G. K.; Garcia, J. F. (1980a) Endocrine aspects of ozone exposure in rats. *Arch. Toxicol. Suppl.* (4): 301-304.
- Clemons, G. K.; Garcia, J. F. (1980b) Changes in thyroid function after short-term ozone exposure in rats. *J. Environ. Pathol. Toxicol.* 4: 359-369.
- Clemons, G. K.; Wei, D. (1984) Effect of short-term ozone exposure on exogenous thyroxine levels in thyroidectomized and hypophysectomized rats. *Toxicol. Appl. Pharmacol.* 74: 86-90.
- Coffin, D. L.; Blommer, E. J. (1970) Alteration of the pathogenic role of streptococci group C in mice conferred by previous exposure to ozone. In: Silver, I. H., ed. *Aerobiology: proceedings of the third international symposium; September 1969; Sussex, United Kingdom.* New York, NY: Academic Press, Inc.; pp. 54-61.
- Coffin, D. L.; Gardner, D. E. (1972a) The role of tolerance in protection of the lung against secondary insults. In: *MEDICHEM: first international symposium of occupational physicians of the chemical industry; April; Ludwigshafen, West Germany.* Ludwigshafen, West Germany: Badische Anilin-und Soda-Fabrik (BASF); pp. 344-364.
- Coffin, D. L.; Gardner, D. E. (1972b) Interaction of biological agents and chemical air pollutants. *Ann. Occup. Hyg.* 15: 219-235.
- Coffin, D. L.; Blommer, E. J.; Gardner, D. E.; Holzman, R. (1967) Effect of air pollution on alteration of susceptibility to pulmonary infection. In: *Proceedings of the third annual conference on atmospheric contamination in confined spaces; May; Dayton, OH.* Wright-Patterson Air Force Base, OH: Aerospace Medical Research Laboratories; pp. 71-80; report no. AMRL-TR-67-200. Available from: NTIS, Springfield, VA; AD-835008.

References for Toxicological Effects (cont'd.)

- Coffin, D. L.; Gardner, D. E.; Holzman, R. S.; Wolock, F. J. (1968) Influence of ozone on pulmonary cells. Arch. Environ. Health 16: 633-636.
- Costa, D. L.; Kutzman, R. S.; Lehmann, J. R.; Popenoe, E. A.; Drew, R. T. (1983) A subchronic multi-dose ozone study in rats. In: Lee, S. D.; Mustafa, M. G.; Mehlmán, M. A., eds. International symposium on the biomedical effects of ozone and related photochemical oxidants; March 1982; Pinehurst, NC. Princeton, NJ: Princeton Scientific Publishers, Inc.; pp: 369-393. (Advances in modern environmental toxicology: v. 5).
- Crapo, J. D.; Barry, B. E.; Chang, L.-Y.; Mercer, R. R. (1984) Alterations in lung structure caused by inhalation of oxidants. J. Toxicol. Environ. Health 13: 301-321.
- DeLucia, A. J.; Hoque, P. M.; Mustafa, M. G.; Cross, C. E. (1972) Ozone interaction with rodent lung: effect on sulfhydryls and sulfhydryl-containing enzyme activities. J. Lab. Clin. Med. 80: 559-566.
- DeLucia, A. J.; Mustafa, M. G.; Cross, C. E.; Plopper, C. G.; Dungworth, D. L.; Tyler, W. S. (1975a) Biochemical and morphological alterations in the lung following ozone exposure. In: Rai, C.; Spielman, L. A., eds. Air: I. pollution control and clean energy; 1973; New Orleans, LA; Detroit, MI; Philadelphia, PA; Vancouver, British Columbia. New York, NY: American Institute of Chemical Engineers; pp. 93-100. (AIChE symposium series: v. 71, no. 147).
- DeLucia, A. J.; Mustafa, M. G.; Hussain, M. Z.; Cross, C. E. (1975b) Ozone interaction with rodent lung. III. Oxidation of reduced glutathione and formation of mixed disulfides between protein and nonprotein sulfhydryls. J. Clin. Invest. 55: 794-802.
- Dillard, C. J.; Urribarri, N.; Reddy, K.; Fletcher, B.; Taylor, S.; de Lumen, B.; Langberg, S.; Tappel, A. L. (1972) Increased lysosomal enzymes in lungs of ozone-exposed rats. Arch. Environ. Health 25: 426-431.
- Dorsey, A. P.; Morgan, D. L.; Menzel, D. B. (1983) Filterability of erythrocytes from ozone-exposed mice. In: Abstracts of the third international congress on toxicology; September; San Diego, CA. Toxicol. Lett. 18(suppl. 1): 146.
- Dowell, A. R.; Lohrbauer, L. A.; Hurst, D.; Lee, S. D. (1970) Rabbit alveolar macrophage damage caused by in vivo ozone inhalation. Arch. Environ. Health 21: 121-127.
- Dubeau, H.; Chung, Y. S. (1979) Ozone response in wild type and radiation-sensitive mutants of Saccharomyces cerevisiae. Mol. Gen. Genet. 176: 393-398.
- Dubeau, H.; Chung, Y. S. (1982) Genetic effects of ozone: induction of point mutation and genetic recombination in Saccharomyces cerevisiae. Mutat. Res. 102: 249-259.

References for Toxicological Effects (cont'd.)

- Dungworth, D. L.; Clarke, G. L.; Plata, R. L. (1969) Pulmonary lesions produced in A-strain mice by long-term exposure to peroxyacetyl nitrate. *Am. Rev. Respir. Dis.* 99: 565-574.
- Dungworth, D. L.; Castleman, W. L.; Chow, C. K.; Mellick, P. W.; Mustafa, M. G.; Tarkington, B.; Tyler, W. S. (1975) Effect of ambient levels of ozone on monkeys. *Fed. Proc. Fed. Am. Soc. Exp. Biol.* 34: 1670-1674.
- Easton, R. E.; Murphy, S. D. (1967) Experimental ozone preexposure and histamine. *Arch. Environ. Health* 15: 160-166.
- Ehrlich, R. (1980) Interaction between environmental pollutants and respiratory infections. *EHP Environ. Health Perspect.* 35: 89-100.
- Ehrlich, R. (1983) Changes in susceptibility to respiratory infection caused by exposures to photochemical oxidant pollutants. In: Lee, S. D.; Mustafa, M. G.; Mehman, M. A., eds. *International symposium on the biomedical effects of ozone and related photochemical oxidants; March 1982; Pinehurst, NC. Princeton, NJ: Princeton Scientific Publishers, Inc.; pp. 273-285. (Advances in modern environmental toxicology: v. 5).*
- Ehrlich, R.; Findlay, J. C.; Fenters, J. D.; Gardner, D. E. (1977) Health effects of short-term inhalation of nitrogen dioxide and ozone mixtures. *Environ. Res.* 14: 223-231.
- Ehrlich, R.; Findlay, J. C.; Gardner, D. E. (1979) Effects of repeated exposures to peak concentrations of nitrogen dioxide and ozone on resistance to streptococcal pneumonia. *J. Toxicol. Environ. Health* 5: 631-642.
- Elsayed, N. M.; Mustafa, M. G.; Postlethwait, E. M. (1982a) Age-dependent pulmonary response of rats to ozone exposure. *J. Toxicol. Environ. Health* 9: 835-848.
- Elsayed, N. M.; Hacker, A.; Mustafa, M.; Kuehn, K.; Schrauzer, G. (1982b) Effects of decreased glutathione peroxidase activity on the pentose phosphate cycle in mouse lung. *Biochem. Biophys. Res. Commun.* 104: 564-569.
- Elsayed, N. M.; Hacker, A. D.; Kuehn, K.; Mustafa, M. G.; Schrauzer, G. N. (1983) Dietary antioxidants and the biochemical response to oxidant inhalation. II. Influence of dietary selenium on the biochemical effects of ozone exposure in mouse lung. *Toxicol. Appl. Pharmacol.* 71: 398-406.
- Erdman, H. E.; Hernandez, T. (1982) Adult toxicity and dominant lethals induced by ozone at specific stages in spermatogenesis in Drosophila virilis. *Environ. Mutagen.* 4: 657-666.
- Eustis, S. L.; Schwartz, L. W.; Kosch, P. C.; Dungworth, D. L. (1981) Chronic bronchiolitis in nonhuman primates after prolonged ozone exposure. *Am. J. Pathol.* 105: 121-137.

References for Toxicological Effects (cont'd.)

- Evans, M. J.; Mayr, W.; Bils, R. F.; Loosli, C. G. (1971) Effects of ozone on cell renewal in pulmonary alveoli of aging mice. *Arch. Environ. Health* 22: 450-453.
- Evans, M. J.; Johnson, L. V.; Stephens, R. J.; Freeman, G. (1976a) Renewal of the terminal bronchiolar epithelium in the rat following exposure to NO₂ or O₃. *Lab. Invest.* 35: 246-257.
- Evans, M. J.; Johnson, L. V.; Stephens, R. J.; Freeman, G. (1976b) Cell renewal in the lungs of rats exposed to low levels of ozone. *Exp. Mol. Pathol.* 24: 70-83.
- Evans, M. J.; Stephens, R. J.; Freeman, G. (1976c). Renewal of pulmonary epithelium following oxidant injury. In: Bouhuys, A., ed. *Lung cells in disease: proceedings of a Brook Lodge conference; April; Augusta, MI.* New York, NY: Elsevier/North-Holland Biomedical Press; pp. 165-178.
- Evans, M. J.; Dekker, N. P.; Cabral-Anderson, L. J.; Shami, S. G. (1985) Morphological basis of tolerance to ozone. *Exp. Mol. Pathol.* 42: 366-376.
- Fabbri, L. M.; Aizawa, H.; Alpert, S. E.; Walters, E. H.; O'Byrne, P. M.; Gold, B. D.; Nadel, J. A.; Holtzman, M. J. (1984) Airway hyperresponsiveness and changes in cell counts in bronchoalveolar lavage after ozone exposure in dogs. *Am. Rev. Respir. Dis.* 129: 288-291.
- Fairchild, E. J., II. (1967) Tolerance mechanisms: determinants of lung responses to injurious agents. *Arch. Environ. Health* 14: 111-126.
- Fetner, R. H. (1962) Ozone-induced chromosome breakage in human cell cultures. *Nature (London)* 194: 793-794.
- Fletcher, B. L.; Tappel, A. L. (1973) Protective effects of dietary α -Tocopherol in rats exposed to toxic levels of ozone and nitrogen dioxide. *Environ. Res.* 6: 165-175.
- Frager, N. B.; Phalen, R. F.; Kenoyer, J. L. (1979) Adaptations to ozone in reference to mucociliary clearance. *Arch. Environ. Health* 34: 51-57.
- Freeman, B. A.; Mudd, J. B. (1981) Reaction of ozone with sulfhydryls of human erythrocytes. *Arch. Biochem. Biophys.* 208: 212-220.
- Freeman, B. A.; Sharman, M. C.; Mudd, J. B. (1979) Reaction of ozone with phospholipid vesicles and human erythrocyte ghosts. *Arch. Biochem. Biophys.* 197: 264-272.
- Freeman, G.; Stephens, R. J.; Coffin, D. L.; Stara, J. F. (1973) Changes in dogs' lungs after long-term exposure to ozone: light and electron microscopy. *Arch. Environ. Health* 26: 209-216.
- Freeman, G.; Juhos, L. T.; Furiosi, N. J.; Mussenden, R.; Stephens, R. J.; Evans, M. J. (1974) Pathology of pulmonary disease from exposure to interdependent ambient gases (nitrogen dioxide and ozone). *Arch. Environ. Health* 29: 203-210.

References for Toxicological Effects (cont'd.)

- Friberg, L.; Holma, B.; Rylander, R. (1972) Animal lung reactions after inhalation of lead and ozone. *Environ. Physiol. Biochem.* 2: 170-178.
- Friedman, M.; Gallo, J. M.; Nichols, H. P.; Bromberg, P. A. (1983) Changes in inert gas rebreathing parameters after ozone exposure in dogs. *Am. Rev. Respir. Dis.* 128: 851-856.
- Fujimaki, H.; Ozawa, M.; Imai, T.; Shimizu, F. (1984) Effect of short-term exposure to O₃ on antibody response in mice. *Environ. Res.* 35: 490-496.
- Fujinaka, L. E.; Hyde, D. M.; Plopper, C. G.; Tyler, W. S.; Dungworth, D. L.; Lollini, L. O. (1985) Respiratory bronchiolitis following long-term ozone exposure in Bonnet monkeys: a morphometric study. *Exp. Lung Res.* 8: 167-190.
- Fukase, O.; Isomura, K.; Watanabe, H. (1975) Effect of ozone on glutathione in vivo. *Taiki Osen Kenkyu* 10: 58-62.
- Fukase, O.; Watanabe, H.; Isomura, K. (1978) Effects of exercise on mice exposed to ozone. *Arch. Environ. Health* 33: 198-200.
- Gardner, D. E. (1984) Oxidant-induced enhanced sensitivity to infection in animal models and their extrapolation to man. In: Miller, F. J.; Menzel, D. B., ed. *Fundamentals of extrapolation modeling of inhaled toxicants*. Washington, DC: Hemisphere Publ. Co.; pp. 423-439.
- Gardner, D. E.; Graham, J. A. (1977) Increased pulmonary disease mediated through altered bacterial defenses. In: Sanders, C. L.; Schneider, R. P.; Dagle, G. E.; Ragen, H. A., eds. *Pulmonary macrophage and epithelial cells: proceedings of the sixteenth annual Hanford biology symposium; September 1976; Richland, WA*. Washington, DC: Energy Research and Development Administration; pp. 1-21. (ERDA symposium series: 43). Available from: NTIS, Springfield, VA; CONF-760927.
- Gardner, D. E.; Pfitzer, E. A.; Christian, R. T.; Coffin, D. L. (1971) Loss of protective factor for alveolar macrophages when exposed to ozone. *Arch. Intern. Med.* 127: 1078-1084.
- Gardner, D. E.; Lewis, T. R.; Alpert, S. M.; Hurst, D. J.; Coffin, D. L. (1972) The role of tolerance in pulmonary defense mechanisms. *Arch. Environ. Health* 25: 432-438.
- Gardner, D. E.; Illing, J. W.; Miller, F. J.; Coffin, D. L. (1974) The effect of ozone on pentobarbital sleeping time in mice. *Res. Commun. Chem. Pathol. Pharmacol.* 9: 689-700.
- Gardner, D. E.; Miller, F. J.; Illing, J. W.; Kirtz, J. M. (1977) Increased infectivity with exposure to ozone and sulfuric acid. *Toxicol. Lett.* 1: 59-64.

References for Toxicological Effects (cont'd.)

- Gershwin, L. J.; Osebold, J. W.; Zee, Y. C. (1981) Immunoglobulin E-containing cells in mouse lung following allergen inhalation and ozone exposure. *Int. Arch. Allergy Appl. Immunol.* 65: 266-277.
- Gertner, A.; Bromberger-Barnea, B.; Dannenberg, A. M., Jr.; Traystman, R.; Menkes, H. (1983a) Responses of the lung periphery to 1.0 ppm ozone. *J. Appl. Physiol. Respir. Environ. Exercise Physiol.* 55: 770-776.
- Gertner, A.; Bromberger-Barnea, B.; Traystman, R.; Berzon, D.; Menkes, H. (1983b) Responses of the lung periphery to ozone and histamine. *J. Appl. Physiol.: Respir. Environ. Exercise Physiol.* 54: 640-646.
- Gertner, A.; Bromberger-Barnea, B.; Traystman, R.; Menkes, H. (1983c) Effects of ozone in peripheral lung reactivity. *J. Appl. Physiol.: Respir. Environ. Exercise Physiol.* 55: 777-784.
- Gertner, A.; Bromberger-Barnea, B.; Kelly, L.; Traystman, R.; Menkes, H. (1984) Local vagal responses in the lung periphery. *J. Appl. Physiol.: Respir. Environ. Exercise Physiol.* 57: 1079-1088.
- Gillespie, J. R. (1980) Review of the cardiovascular and pulmonary function studies on beagles exposed for 68 months to auto exhaust and other air pollutants. In: Stara, J. F.; Dungworth, D. L.; Orthoefer, J. C.; Tyler, W. S., eds. Long-term effects of air pollutants in canine species. Cincinnati, OH: U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office; pp. 115-154; EPA report no. EPA 600/8-80-014. Available from: NTIS, Springfield, VA; PB81-144875.
- Goldstein, E.; Tyler, W. S.; Hoeprich, P. D.; Eagle, C. (1971a) Ozone and the antibacterial defense mechanisms of the murine lung. *Arch. Intern. Med.* 127: 1099-1102.
- Goldstein, E.; Tyler, W. S.; Hoeprich, P. D.; Eagle, C. (1971b) Adverse influence of ozone on pulmonary bactericidal activity of murine lungs. *Nature (London)* 229: 262-263.
- Goldstein, E.; Eagle, M. C.; Hoeprich, P. D. (1972) Influence of ozone on pulmonary defense mechanisms of silicotic mice. *Arch. Environ. Health* 24: 444-448.
- Goldstein, B. D.; Lai, L. Y.; Cuzzi-Spada, R. (1974) Potentiation of complement-dependent membrane damage by ozone. *Arch. Environ. Health* 28: 40-42.
- Goldstein, B. D.; Hamburger, S. J.; Falk, G. W.; Amoruso, M. A. (1977) Effect of ozone and nitrogen dioxide on the agglutination of rat alveolar macrophages by concanavalin A. *Life Sci.* 21: 1637-1644.
- Gooch, P. C.; Creasia, D. A.; Brewen, J. G. (1976) The cytogenetic effect of ozone: inhalation and in vitro exposures. *Environ. Res.* 12: 188-195.
- Gordon, T.; Amdur, M. O. (1980) Effect of ozone on respiratory response of guinea pigs to histamine. *J. Toxicol. Environ. Health* 6: 185-195.

References for Toxicological Effects (cont'd.)

- Gordon, T.; Taylor, B. F.; Amdur, M. O. (1981) Ozone inhibition of tissue cholinesterase in guinea pigs. *Arch. Environ. Health* 36: 284-288.
- Gordon, T.; Venugopalan, C. S.; Amdur, M. O.; Drazen, J. M. (1984) Ozone-induced airway hyperreactivity in the guinea pig. *J. Appl. Physiol.: Respir. Environ. Exercise Physiol.* 57: 1034-1038.
- Graham, J. A. (1979) Alteration of hepatic xenobiotic metabolism by ozone [dissertation]. Durham, NC: Duke University. Available from: University Microfilms, Ann Arbor, MI: publication no. 80-09646.
- Graham, J. A.; Menzel, D. B.; Miller, F. J.; Illing, J. W.; Gardner, D. E. (1981) Influence of ozone on pentobarbital-induced sleeping time in mice, rats, and hamsters. *Toxicol. Appl. Pharmacol.* 61: 64-73.
- Graham, J. A.; Menzel, D. B.; Miller, F. J.; Illing, J. W.; Gardner, D. E. (1982a) Effect of ozone on drug-induced sleeping time in mice pretreated with mixed-function oxidase inducers and inhibitors. *Toxicol. Appl. Pharmacol.* 62: 489-497.
- Graham, J. A.; Miller, F. J.; Gardner, D. E.; Ward, R.; Menzel, D. B. (1982b) Influence of ozone and nitrogen dioxide on hepatic microsomal enzymes in mice. *J. Toxicol. Environ. Health* 9: 849-856.
- Graham, J. A.; Menzel, D. B.; Miller, F. J.; Illing, J. W.; Ward, R.; Gardner, D. E. (1983) Influence of ozone on xenobiotic metabolism. In: Lee, S. D.; Mustafa, M. G.; Mehlman, M. A., eds. *International symposium on the biomedical effects of ozone and related photochemical oxidants; March 1982; Pinehurst, NC.* Princeton, NJ: Princeton Scientific Publishers, Inc.; pp. 95-117. (*Advances in modern environmental toxicology: v. 5*).
- Graham, J. A.; Menzel, D. B.; Mole, M. L.; Miller, F. J.; Gardner, D. E. (1985) Influence of ozone on pentobarbital pharmacokinetics in mice. *Toxicol. Lett.* 24: 163-170.
- Green, G. M. (1984) Similarities of host defense mechanisms against pulmonary disease in animals and man. *J. Toxicol. Environ. Health* 13: 471-478.
- Grose, E. C.; Gardner, D. E.; Miller, F. J. (1980) Response of ciliated epithelium to ozone and sulfuric acid. *Environ. Res.* 22: 377-385.
- Grose, E. C.; Richards, J. H.; Illing, J. W.; Miller, F. J.; Davies, D. W.; Graham, J. A.; Gardner, D. E. (1982) Pulmonary host defense responses to inhalation of sulfuric acid and ozone. *J. Toxicol. Environ. Health* 10: 351-362.
- Guerrero, R. R.; Rounds, D. E.; Olson, R. S.; Hackney, J. D. (1979) Mutagenic effects of ozone on human cells exposed in vivo and in vitro based on sister chromatid exchange analysis. *Environ. Res.* 18: 336-346.

References for Toxicological Effects (cont'd.)

- Hadley, J. G.; Gardner, D. E.; Coffin, D. L.; Menzel, D. B. (1977) Enhanced binding of autologous cells to the macrophage plasma membrane as a sensitive indicator of pollutant damage. In: Sanders, C. L.; Schneider, R. P.; Dagle, G. E.; Ragan, H. A., eds. Pulmonary macrophage and epithelial cells: proceedings of the sixteenth annual Hanford biology symposium; September 1976; Richland, WA. Washington, DC: Energy Research and Development Administration; pp. 1-21. (ERDA symposium series: v. 43). Available from: NTIS, Springfield, VA; CONF-760927.
- Hamelin, C.; Chung, Y. S. (1975a) The effect of low concentrations of ozone on Escherichia coli chromosome. *Mutat. Res.* 28: 131-132.
- Hamelin, C.; Chung, Y. S. (1975b) Characterization of mucoid mutants of Escherichia coli K-12 isolated after exposure to ozone. *J. Bacteriol.* 122: 19-24.
- Hamelin, C.; Chung, Y. S. (1978) Role of the *pol*, *rec*, and DNA gene products in the repair of lesions produced in Escherichia coli DNA by ozone. *Stud. Biophys.* 68: 229-335.
- Hamelin, C.; Sarhan, F.; Chung, Y. S. (1977a) Ozone-induced DNA degradation in different DNA polymerase I mutants of Escherichia coli K12. *Biochem. Biophys. Res. Commun.* 77: 220-224.
- Hamelin, C.; Sarhan, F.; Chung, Y. S. (1977b) DNA degradation caused by ozone in mucoid mutants of Escherichia coli K12. *FEMS Microbiol. Lett.* 2: 149-151.
- Hesterberg, T. W.; Last, J. A. (1981) Ozone-induced acute pulmonary fibrosis in rats: prevention of increased rates of collagen synthesis by methylprednisolone. *Am. Rev. Respir. Dis.* 123: 47-52.
- Holtzman, M. J.; Fabbri, L. M.; Skoogh, B.-E.; O'Byrne, P. M.; Walters, E. H.; Aizawa, H.; Nadel, J. A. (1983a) Time course of airway hyperresponsiveness induced by ozone in dogs. *J. Appl. Physiol.: Respir. Environ. Exercise Physiol.* 55: 1232-1236.
- Holtzman, M. J.; Fabbri, L. M.; O'Byrne, P. M.; Gold, B. D.; Aizawa, H.; Walters, E. H.; Alpert, S. E.; Nadel, J. A. (1983b) Importance of airway inflammation for hyperresponsiveness induced by ozone. *Am. Rev. Respir. Dis.* 127: 686-690.
- Hu, P. C.; Miller, F. J.; Daniels, M. J.; Hatch, G. E.; Graham, J. A.; Gardner, D. E.; Selgrade, M. K. (1982) Protein accumulation in lung lavage fluid following ozone exposure. *Environ. Res.* 29: 377-388.
- Huber, G. L.; Mason, R. J.; LaForce, M.; Spencer, N. J.; Gardner, D. E.; Coffin, D. L. (1971) Alterations in the lung following the administration of ozone. *Arch. Intern. Med.* 128: 81-87.
- Hueter, F. G.; Contner, G. L.; Busch, K. A.; Hinners, R. G. (1966) Biological effects of atmospheres contaminated by auto exhaust. *Arch. Environ. Health* 12: 553-560.

References for Toxicological Effects (cont'd.)

- Hurst, D. J.; Coffin, D. L. (1971) Ozone effect on lysosomal hydrolases of alveolar macrophages in vitro. Arch. Intern. Med. 127: 1059-1063.
- Hurst, D. J.; Gardner, D. E.; Coffin, D. L. (1970) Effect of ozone on acid hydrolases of the pulmonary alveolar macrophage. J. Reticuloendothel. Soc. 8: 288-300.
- Hussain, M. Z.; Mustafa, M. G.; Chow, C. K.; Cross, C. E. (1976a) Ozone-induced increase of lung proline hydroxylase activity and hydroxyproline content. Chest 69 (suppl. 2): 273-275.
- Hussain, M. Z.; Cross, C. E.; Mustafa, M. G.; Bhatnagar, R. S. (1976b) Hydroxyproline contents and prolyl hydroxylase activities in lungs of rats exposed to low levels of ozone. Life Sci. 18: 897-904.
- Hyde, D.; Orthoefer, J. G.; Dungworth, D.; Tyler, W.; Carter, R.; Lum, H. (1978) Morphometric and morphologic evaluation of pulmonary lesions in beagle dogs chronically exposed to high ambient levels of air pollutants. Lab. Invest. 38: 455-469.
- Ibrahim, A. L.; Zee, Y. C.; Osebold, J. W. (1980) The effects of ozone on the respiratory epithelium of mice. II. Ultrastructural alterations. J. Environ. Pathol. Toxicol. 3: 251-258.
- Illing, J. W.; Miller, F. J.; Gardner, D. E. (1980) Decreased resistance to infection in exercised mice exposed to NO₂ and O₃. J. Toxicol. Environ. Health 6: 843-851.
- Inoue, H.; Sato, S.; Hirose, T.; Kikuchi, Y.; Ubukata, T.; Nagashima, S.; Sasaki, T.; Takishima, T. (1979) A comparative study between functional and pathologic alterations in lungs of rabbits exposed to an ambient level of ozone: functional aspects. Nikkyo Shikkai-Shi 17: 288-296.
- Jegier, Z. (1973) L'ozone en tant que polluant atmosphérique [Ozone as an air pollutant]. Can. J. Public Health 64: 161-166.
- Kavlock, R.; Daston, G.; Grabowski, C. T. (1979) Studies on the developmental toxicity of ozone. I. Prenatal effects. Toxicol. Appl. Pharmacol. 48: 19-28.
- Kavlock, R. J.; Meyer, E.; Grabowski, C. T. (1980) Studies on the developmental toxicity of ozone: postnatal effects. Toxicol. Lett. 5: 3-9.
- Kenoyer, J. L.; Phalen, R. F.; Davis, J. R. (1981) Particle clearance from the respiratory tract as a test of toxicity: effect of ozone on short and long term clearance. Exp. Lung Respir. 2: 111-120.
- Kesner, L.; Kindya, R. J.; Chan, P. C. (1979) Inhibition of erythrocyte membrane (Na + K)-activated ATPase by ozone-treated phospholipids. J. Biol. Chem. 254: 2705-2709.

References for Toxicological Effects (cont'd.)

- Kindya, R. J.; Chan, P. C. (1976) Effect of ozone on erythrocyte membrane adenosine triphosphatase. *Biochim. Biophys. Acta* 429: 608-615.
- Konigsberg, A. S.; Bachman, C. H. (1970) Ozonized atmosphere and gross motor activity of rats. *Int. J. Biometeorol.* 14: 261-266.
- Koontz, A. E.; Heath, R. L. (1979) Ozone alteration of transport of cations and the Na⁺/K⁺-ATPase in human erythrocytes. *Arch. Biochem. Biophys.* 198: 493-500.
- Kotlikoff, M. I.; Jackson, A. C.; Watson, J. W. (1984) Oscillatory mechanics of the respiratory system in ozone-exposed rats. *J. Appl. Physiol.: Respir. Environ. Exercise Physiol.* 56: 182-186.
- Kyei-Aboagye, K.; Hazucha, M.; Wyszogrodski, I.; Rubinstein, D.; Avery, M. E. (1973) The effect of ozone exposure *in vivo* on the appearance of lung tissue lipids in the endobronchial lavage of rabbits. *Biochem. Biophys. Res. Commun.* 54: 907-913.
- Larkin, E. C.; Goheen, S. C.; Rao, G. A. (1983) Morphology and fatty acid composition of erythrocytes from monkeys exposed to ozone for one year. *Environ. Res.* 32: 445-454.
- Last, J. A.; Cross, C. E. (1978) A new model for health effects of air pollutants: evidence for synergistic effects of mixtures of ozone and sulfuric acid aerosols on rat lungs. *J. Lab. Clin. Med.* 91: 328-339.
- Last, J. A.; Greenberg, D. B. (1980) Ozone-induced alterations in collagen metabolism of rat lungs. II. Long-term exposure. *Toxicol. Appl. Pharmacol.* 55: 108-114.
- Last, J. A.; Kaizu, T. (1980) Mucus glycoprotein secretion by tracheal explants: effects of pollutants. *EHP Environ. Health Perspect.* 35: 131-138.
- Last, J. A.; Jennings, M. D.; Schwartz, L. W.; Cross, C. E. (1977) Glycoprotein secretion by tracheal explants cultured from rats exposed to ozone. *Am. Rev. Respir. Dis.* 116: 695-703.
- Last, J. A.; Greenberg, D. B.; Castleman, W. L. (1979) Ozone-induced alterations in collagen metabolism of rat lungs. *Toxicol. Appl. Pharmacol.* 51: 247-258.
- Last, J. A.; Dasgupta, P. K.; DeCesare, K.; Tarkington, B. K. (1982) Inhalation toxicology of ammonium persulfate, an oxidant aerosol, in rats. *Toxicol. Appl. Pharmacol.* 63: 257-263.
- Last, J. A.; Gerriets, J. E.; Hyde, D. M. (1983) Synergistic effects on rat lungs of mixtures of oxidant air pollutants (ozone or nitrogen dioxide) and respirable aerosols. *Am. Rev. Respir. Dis.* 128: 539-544.
- Last, J. A.; Hyde, D. M.; Chang, D. P. Y. (1984a) A mechanism of synergistic lung damage by ozone and a respirable aerosol. *Exp. Lung Res.* 7: 223-235.

References for Toxicological Effects (cont'd.)

- Last, J. A.; Reiser, K. M.; Tyler, W. S.; Rucker, R. B. (1984b) Long-term consequences of exposure to ozone. I. Lung collagen content. *Toxicol. Appl. Pharmacol.* 72: 111-118.
- Lee, L.-Y.; Bleecker, E. R.; Nadel, J. A. (1977) Effect of ozone on bronchomotor response to inhaled histamine aerosol in dogs. *J. Appl. Physiol.: Respir. Environ. Exercise Physiol.* 43: 626-631.
- Lee, L.-Y.; Dumont, C.; Djokic, T. D.; Menzel, T. E.; Nadel, J. A. (1979) Mechanism of rapid, shallow breathing after ozone exposure in conscious dogs. *J. Appl. Physiol.: Respir. Environ. Exercise Physiol.* 46: 1108-1114.
- Lee, L.-Y.; Djokic, T. D.; Dumont, C.; Graf, P. D.; Nadel, J. A. (1980) Mechanism of ozone-induced tachypneic response to hypoxia and hypercapnia in conscious dogs. *J. Appl. Physiol.: Respir. Environ. Exercise Physiol.* 48: 163-168.
- Lewis, T. R.; Hueter, F. G.; Busch, K. A. (1967) Irradiated automobile exhaust: its effects on the reproduction of mice. *Arch. Environ. Health* 15: 26-35.
- Lewis, T. R.; Moorman, W. J.; Yang, Y.; Stara, J. F. (1974) Long-term exposure to auto exhaust and other pollutant mixtures: effects on pulmonary function in the beagle. *Arch. Environ. Health* 29: 102-106.
- Lum, H.; Schwartz, L. W.; Dungworth, D. L.; Tyler, W. S. (1978) A comparative study of cell renewal after exposure to ozone or oxygen: response of terminal bronchiolar epithelium in the rat. *Am. Rev. Respir. Dis.* 118: 335-345.
- Lunan, K. D.; Short, P.; Negi, D.; Stephens, R. J. (1977) Glucose-6-phosphate dehydrogenase response of postnatal lungs to NO₂ and O₃. In: Sanders, C. L.; Schneider, R. P.; Dagle, G. E.; Ragan, H. A., eds. *Pulmonary macrophage and epithelial cells: proceedings of the sixteenth annual Hanford biology symposium; September 1976; Richland, WA.* Washington, DC: Energy Research and Development Administration; pp. 236-247. (ERDA symposium series: 43). Available from: NTIS, Springfield, VA; CONF-760927.
- MacRae, W. D.; Stich, H. F. (1979) Induction of sister chromatid exchanges in Chinese hamster ovary cells by thiol and hydrazene compounds. *Mutat. Res.* 68: 351-365.
- Martin, C. J.; Boatman, E. S.; Ward, G. (1983) Mechanical properties of alveolar wall after pneumonectomy and ozone exposure. *J. Appl. Physiol.: Respir. Environ. Exercise Physiol.* 54: 785-788.
- McAllen, S. J.; Chiu, S. P.; Phalen, R. F.; Rasmussen, R. E. (1981) Effect of in vivo ozone exposure on in vitro pulmonary alveolar macrophage mobility. *J. Toxicol. Environ. Health* 7: 373-381.
- McJilton, C.; Thielke, J.; Frank, R. (1972) Ozone uptake model for the respiratory system. In: *Abstracts of technical papers: American industrial hygiene conference; May; San Francisco, CA.* Am. Ind. Hyg. Assoc. J. 33: paper no. 45.

References for Toxicological Effects (cont'd.)

- Mellick, P. W.; Mustafa, M. G.; Tyler, W. S.; Dungworth, D. L. (1975) Morphologic and biochemical changes in primate lung due to low-level ozone exposure. In: Proceedings of the 23rd international tuberculosis conference; September; Mexico. Bull. Int. Union Tubercul. 51: 565-567.
- Mellick, P. W.; Dungworth, D. L.; Schwartz, L. W.; Tyler, W. S. (1977) Short term morphologic effects of high ambient levels of ozone on lungs of rhesus monkeys. Lab. Invest. 36: 82-90.
- Menzel, D. B.; Roehm, J. N.; Lee, S. D. (1972) Vitamin E: the biological and environmental antioxidant. J. Agric. Food Chem. 20: 481-486.
- Menzel, D. B.; Slaughter, R. J.; Bryant, A. M.; Jauregui, H. O. (1975a) Heinz bodies formed in erythrocytes by fatty acid ozonides and ozone. Arch. Environ. Health 30: 296-301.
- Menzel, D. B.; Slaughter, R. J.; Bryant, A.M.; Jauregui, H. O. (1975b) Prevention of ozonide-induced Heinz bodies in human erythrocytes by vitamin E. Arch. Environ. Health 30: 234-236.
- Miller, F. J.; Illing, J. W.; Gardner, D. E. (1978a) Effect of urban ozone levels on laboratory-induced respiratory infections. Toxicol. Lett. 2: 163-169.
- Miller, F. J.; Menzel, D. B.; Coffin, D. L. (1978b) Similarity between man and laboratory animals in regional deposition of ozone. Environ. Res. 17: 84-101.
- Miller, F. J.; Overton, J. H., Jr.; Jaskot, R. H.; Menzel, D. B.; (1985) A model of the regional uptake of gaseous pollutants in the lung. I. The sensitivity of the uptake of ozone in the human lung to lower respiratory tract secretions and to exercise. Toxicol. Appl. Pharmacol. 79: 11-27.
- Mizoguchi, I.; Osawa, M.; Sato, Y.; Makino, K.; Yagyu, H. (1973) Studies on erythrocyte and photochemical smog. I. Effects of air pollutants on erythrocyte resistance. Taiki Osen Kenkyu 8: 414.
- Moore, P. F.; Schwartz, L. W. (1981) Morphological effects of prolonged exposure to ozone and sulfuric acid aerosol on the rat lung. Exp. Mol. Pathol. 35: 108-123.
- Moore, G. S.; Calabrese, E. J.; Grinberg-Funes, R. A. (1980) The C57L/J mouse strain as a model for extrapulmonary effects of ozone exposure. Bull. Environ. Contam. Toxicol. 25: 578-585.
- Moore, G. S.; Calabrese, E. J.; Labato, F. J. (1981a) Erythrocyte survival in sheep exposed to ozone. Bull. Environ. Contam. Toxicol. 27: 126-138.
- Moore, G. S.; Calabrese, E. J.; Schulz, E. (1981b) Effect of *in vivo* ozone exposure to Dorset sheep, an animal model with low levels of erythrocyte glucose-6-phosphate dehydrogenase activity. Bull. Environ. Contam. Toxicol. 26: 273-280.

References for Toxicological Effects (cont'd.)

- Murlas, C. G.; Roum, J. H. (1985) Sequence of pathologic changes in the airway mucosa of guinea pigs during ozone-induced bronchial hyperractivity. *Am. Rev. Respir. Dis.* 131: 314-320.
- Murphy, S. D. (1964) A review of effects on animals of exposure to auto exhaust and some of its components. *J. Air Pollut. Control Assoc.* 14: 303-308.
- Murphy, S. D.; Leng, J. K.; Ulrich, C. E.; Davis, H. V. (1963) Effects on animals of exposure to auto exhaust. *Arch. Environ. Health* 7: 60-70.
- Murphy, S. D.; Ulrich, C. E.; Frankowitz, S. H.; Xintaras, C. (1964) Altered function in animals inhaling low concentrations of ozone and nitrogen dioxide. *Am. Ind. Hyg. Assoc. J.* 25: 246-253.
- Mustafa, M. G. (1975) Influence of dietary vitamin E on lung cellular sensitivity to ozone in rats. *Nutr. Rep. Int.* 11: 473-476.
- Mustafa, M. G.; Lee, S. D. (1976) Pulmonary biochemical alterations resulting from ozone exposure. *Ann. Occup. Hyg.* 19: 17-26.
- Mustafa, M. G.; Tierney, D. F. (1978) Biochemical and metabolic changes in the lung with oxygen, ozone, and nitrogen dioxide toxicity. *Am. Rev. Respir. Dis.* 118: 1061-1090.
- Mustafa, M. G.; DeLucia, A. J.; York, G. K.; Arth, C.; Cross, C. E. (1973) Ozone interaction with rodent lung. II. Effects on oxygen consumption of mitochondria. *J. Lab. Clin. Med.* 82: 357-365.
- Mustafa, M. G.; Hacker, A. D.; Ospital, J. J.; Hussain, M. Z.; Lee, S. D. (1977) Biochemical effects of environmental oxidants pollutants in animal lungs. In: Lee, S. D., ed. *Biochemical effects of environmental pollutants*. Ann Arbor, MI: Ann Arbor Science Publishers, Inc.; pp. 59-96.
- Mustafa, M. G.; Elsayed, N. M.; Quinn, C. L.; Postlethwait, E. M.; Gardner, D. E.; Graham, J. A. (1982) Comparison of pulmonary biochemical effects of low level ozone exposure on mice and rats. *J. Toxicol. Environ. Health* 9: 857-865.
- Mustafa, M. G.; Elsayed, N. M.; von Dohlen, F. M.; Hassett, C. M.; Postlethwait, E. M.; Quinn, C. L.; Graham, J. A.; Gardner, D. E. (1984) A comparison of biochemical effects of nitrogen dioxide, ozone, and their combination in mouse lung. I. Intermittent exposures. *Toxicol. Appl. Pharmacol.* 72: 82-90.
- Nakajima, T.; Kusumoto, S.; Tsubota, Y.; Yonekawa, E.; Yoshida, R.; Motomiya, K.; Ito, K.; Ide, G.; Otsu, H. (1972). Histopathological studies on the respiratory organs of mice exposed to photochemical oxidants and automobile exhaust. *Osaka-furitsu Koshu Eisei Kenkyusho Kenkyu Hokoku Rodo Eisei Hen* 10: 35-42.
- Nambu, Z.; Yokoyama, E. (1983) Antioxidant system and ozone tolerance. *Environ. Res.* 32: 111-117.

References for Toxicological Effects (cont'd.)

- O'Byrne, P. M.; Walters, E. H.; Gold, B. D.; Aizawa, H. A.; Fabbri, L. M.; Alpert, S. E.; Nadel, J. A.; Holtzman, M. J. (1984a) Neutrophil depletion inhibits airway hyperresponsiveness induced by ozone exposure. *Am. Rev. Respir. Dis.* 130: 214-219.
- O'Byrne, P. M.; Walters, E. H.; Aizawa, H.; Fabbri, L. M.; Holtzman, M. J.; Nadel, J. A. (1984b) Indomethacin inhibits the airway hyperresponsiveness but not the neutrophil influx induced by ozone in dogs. *Am. Rev. Respir. Dis.* 130: 220-224.
- Osebold, J. W.; Owens, S. L.; Zee, Y. C.; Dotson, W. M.; LaBarre, D. D. (1979) Immunological alterations in the lungs of mice following ozone exposure: changes in immunoglobulin levels and antibody-containing cells. *Arch. Environ. Health* 34: 258-265.
- Osebold, J. W.; Gershwin, L. J.; Zee, Y. C. (1980) Studies on the enhancement of allergic lung sensitization by inhalation of ozone and sulfuric acid aerosol. *J. Environ. Pathol. Toxicol.* 3: 221-234.
- P'an, A. Y. S.; Jegier, Z. (1971) The serum trypsin inhibitor capacity during ozone exposure. *Arch. Environ. Health* 23: 215-219.
- P'an, A. Y. S.; Jegier, Z. (1972) Trypsin protein esterase in relation to ozone-induced vascular damage. *Arch. Environ. Health* 24: 233-236.
- P'an, A. Y. S.; Jegier, Z. (1976) Serum protein changes during exposure to ozone. *Am. Ind. Hyg. Assoc. J.* 37: 329-334.
- P'an, A. Y. S.; Béland, J.; Jegier, Z. (1972) Ozone-induced arterial lesions. *Arch. Environ. Health* 24: 229-232.
- Pemsingh, R. S.; Atwal, O. S. (1983) Occurrence of APUD-type cells in the ciliated cyst of the parathyroid gland of ozone-exposed dogs. *Acta Anat.* 116: 97-105.
- Peterson, D. C.; Andrews, H. L. (1963) The role of ozone in radiation avoidance in the mouse. *Radiat. Res.* 19: 331-336.
- Phalen, R. F.; Kenoyer, J. L.; Crocker, T. T.; McClure, T. R. (1980) Effects of sulfate aerosols in combination with ozone on elimination of tracer particles inhaled by rats. *J. Toxicol. Environ. Health* 6: 797-810.
- Plopper, C. G.; Chow, C. K.; Dungworth, D. L.; Brummer, M.; Nemeth, T. J. (1978) Effect of low level of ozone on rat lungs. II. Morphological responses during recovery and re-exposure. *Exp. Mol. Pathol.* 29: 400-411.
- Plopper, C. G.; Chow, C. K.; Dungworth, D. L.; Tyler, W. S. (1979) Pulmonary alterations in rats exposed to 0.2 and 0.1 ppm ozone: a correlated morphological and biochemical study. *Arch. Environ. Health* 34: 390-395.

References for Toxicological Effects (cont'd.)

- Raub, J. A.; Miller, F. J.; Graham, J. A. (1983) Effects of low-level ozone exposure on pulmonary function in adult and neonatal rats. In: Lee, S. D.; Mustafa, M. G.; Mehlman, M. A., eds. International symposium on the biomedical effects of ozone and related photochemical oxidants; March 1982; Pinehurst, NC Princeton, NJ: Princeton Scientific Publishers, Inc.; pp. 363-367. (Advances in modern environmental toxicology: v. 5).
- Reasor, M. J.; Adams, G. K., III; Brooks, J. K.; Rubin, R. J. (1979) Enrichment of albumin and IgG in the airway secretions of dogs breathing ozone. J. Environ. Sci. Health C13: 335-346.
- Revis, N. W.; Major, T.; Dalbey, W. E. (1981) Cardiovascular effects of ozone and cadmium inhalation in the rat. In: Northrop Services, Inc., ed. Proceedings of the research planning workshop on health effects of oxidants; January 1980; Raleigh, NC. Research Triangle Park, NC: U.S. Environmental Protection Agency, Health Effects Research Laboratory; EPA-600/9-81-001; pp. 171-179. Available from: NTIS, Springfield, VA; PB81-178832.
- Reynolds, R. W.; Chaffee, R. R. J. (1970) Studies on the combined effects of ozone and a hot environment on reaction time in subhuman primates. In: Project clean air: v. 2. Santa Barbara, CA: University of California; research project S-6.
- Roehm, J. N.; Hadley, J. G.; Menzel, D. B. (1972) The influence of vitamin E on the lung fatty acids of rats exposed to ozone. Arch. Environ. Health 24: 237-242.
- Roum, J. H.; Murlas, C. (1984) Ozone-induced changes in muscarinic bronchial reactivity by different testing methods. J. Appl. Physiol.: Respir. Environ. Exercise Physiol. 57: 1783-1789.
- Schlipkötter, H.-W.; Bruch, J. (1973) Funktionelle und morphologische Veränderung bei Ozonexposition [Functional and morphological alterations caused by exposure to ozone]. Zentralbl. Bakteriol. Parasitenkd. Infektionskr. Hyg. Abt. 1: Orig. Reihe B 156: 486-499.
- Schwartz, L. W.; Christman, C. A. (1979) Alveolar macrophage migration: influence of lung lining material and acute lung insult. Am. Rev. Respir. Dis. 120: 429-439.
- Schwartz, L. W.; Dungworth, D. L.; Mustafa, M. G.; Tarkington, B. K.; Tyler, W. S. (1976) Pulmonary responses of rats to ambient levels of ozone: effects of 7-day intermittent or continuous exposure. Lab. Invest. 34: 565-578.
- Scott, D. B. M.; Leshner, E. C. (1963) Effect of ozone on survival and permeability of Escherichia coli. J. Bacteriol. 85: 567-576.
- Sherwood, R. L.; Kimura, A.; Donovan, R.; Goldstein, E. (1984) Effect of 0.64 ppm ozone on rats with chronic pulmonary bacterial infection. J. Toxicol. Environ. Health 13: 893-904.

References for Toxicological Effects (cont'd.)

- Shingu, H.; Sugiyama, M.; Watanabe, M.; Nakajima, T. (1980) Effects of ozone and photochemical oxidants on interferon production by rabbit alveolar macrophages. *Bull. Environ. Contam. Toxicol.* 24: 433-438.
- Sielczak, M. W.; Denas, S. M.; Abraham, W. M. (1983) Airway cell changes in tracheal lavage of sheep after ozone exposure. *J. Toxicol. Environ. Health* 11: 545-553.
- Speit, G.; Vogel, W.; Wolf, M. (1982) Characterization of sister chromatid exchange induction by hydrogen peroxide. *Environ. Mutagen.* 4: 135-142.
- Stephens, R. J.; Sloan, M. F.; Evans, M. J.; Freeman, G. (1974a) Early response of lung to low levels of ozone. *Am. J. Pathol.* 74: 31-58.
- Stephens, R. J.; Sloan, M. F.; Evans, M. J.; Freeman, G. (1974b) Alveolar type 1 cell response to exposure to 0.5 ppm O₃ for short periods. *Exp. Mol. Pathol.* 20: 11-23.
- Stephens, R. J.; Sloan, M. F.; Groth, D. G.; Negi, D. S.; Lunan, K. D. (1978) Cytologic response of postnatal rat lungs to O₃ or NO₂ exposure. *Am. J. Pathol.* 93: 183-200.
- Stewart, R. M.; Weir, E. K.; Montgomery, M. R.; Niewoehner, D. E. (1981) Hydrogen peroxide contracts airway smooth muscle: a possible endogenous mechanism. *Respir. Physiol.* 45: 333-342.
- Stokinger, H. E.; Wagner, W. D.; Wright, P. G. (1956) Studies on ozone toxicity. I. Potentiating effects of exercise and tolerance development. *AMA Arch. Ind. Health* 14: 158-162.
- Tepper, J. L.; Weiss, B.; Cox, C. (1982) Microanalysis of ozone depression of motor activity. *Toxicol. Appl. Pharmacol.* 64: 317-326.
- Tepper, J. L.; Weiss, B.; Wood, R. W. (1983) Behavioral indices of ozone exposure. In: Lee, S. D.; Mustafa, M. G.; Mehlman, M. A., eds. *International symposium on the biomedical effects of ozone and related photochemical oxidants; March 1982; Pinehurst, NC.* Princeton, NJ: Princeton Scientific Publishers, Inc.; pp. 515-526. (*Advances in modern environmental toxicology: v. 5*).
- Thomas, G.; Fenters, J. D.; Ehrlich, R. (1979) Effect of exposure to PAN and ozone on susceptibility to chronic bacterial infection. Research Triangle Park, NC: U.S. Environmental Protection Agency, Health Effects Research Laboratory; EPA report no. EPA-600/1-79-001. Available from: NTIS, Springfield, VA; PB-292267.
- Thomas, G. B.; Fenters, J. D.; Ehrlich, R.; Gardner, D. E. (1981a) Effects of exposure to peroxyacetyl nitrate on susceptibility to acute and chronic bacterial infection. *J. Toxicol. Environ. Health* 8: 559-574.
- Thomas, G. B.; Fenters, J. D.; Ehrlich, R.; Gardner, D. E. (1981b) Effects of exposure to ozone on susceptibility to experimental tuberculosis. *Toxicol. Lett.* 9: 11-17.

References for Toxicological Effects (cont'd.)

- Tice, R. R.; Bender, M. A.; Ivett, J. L.; Drew, R. T. (1978) Cytogenetic effects of inhaled ozone. *Mutat. Res.* 58: 293-304.
- Trams, E. G.; Lauter, C. J.; Brown, E. A. B.; Young, O. (1972) Cerebral cortical metabolism after chronic exposure to ozone. *Arch. Environ. Health* 24: 153-159.
- Tyson, C. A.; Lunan, K. D.; Stephens, R. J. (1982) Age-related differences in GSH-shuttle enzymes in NO₂- or O₃-exposed rat lungs. *Arch. Environ. Health* 37: 167-176.
- Veninga, T. S. (1967) Toxicity of ozone in comparison with ionizing radiation. *Strahlentherapie* 134: 469-477.
- Veninga, T. S. (1970) Ozone-induced alterations in murine blood and liver. Presented at: Second international clean air congress; December; Washington, DC. Washington, DC: International Union of Air Pollution Prevention Associations; paper no. MB-15E.
- Veninga, T. S.; Wagenaar, J.; Lemstra, W. (1981) Distinct enzymatic responses in mice exposed to a range of low doses of ozone. *EHP Environ. Health Perspect.* 39: 153-157.
- Verweij, H.; Van Steveninck, J. (1980) Effects of semicarbazide on oxidative processes in human red blood cell membranes. *Biochim. Biophys. Acta* 602: 591-599.
- Verweij, H.; Van Steveninck, J. (1981) Protective effects of semicarbazide and p-aminobenzoic acid against ozone toxicity. *Biochem. Pharmacol.* 30: 1033-1037.
- Warshauer, D.; Goldstein, E.; Hoepflich, P. D.; Lippert, W. (1974) Effect of vitamin E and ozone on the pulmonary antibacterial defense mechanisms. *J. Clin. Med.* 83: 228-240.
- Watanabe, S.; Frank, R.; Yokoyama, E. (1973) Acute effects of ozone on lungs of cats. I. Functional. *Am. Rev. Respir. Dis.* 108: 1141-1151.
- Wegner, C. D. (1982) Characterization of dynamic respiratory mechanics by measuring pulmonary and respiratory system impedances in adult bonnet monkeys (Macaca radiata): including the effects of long-term exposure to low-level ozone [dissertation]. Davis, CA: University of California. Available from: University Microfilms, Ann Arbor, MI; publication no. 82-27900.
- Weiss, B.; Ferin, J.; Merigan, W.; Stern, S.; Cox, C. (1981) Modification of rat operant behavior by ozone exposure. *Toxicol. Appl. Pharmacol.* 58: 244-251.
- Williams, S. J.; Charles, J. M.; Menzel, D. B. (1980) Ozone induced alterations in phenol red absorption from the rat lung. *Toxicol. Lett.* 6: 213-219.

References for Toxicological Effects (cont'd.)

- Witz, G.; Amoruso, M. A.; Goldstein, B. D. (1983) Effect of ozone on alveolar macrophage function: membrane dynamic properties. In: Lee, S. D.; Mustafa, M. G.; Mehlman, M. A., eds. International symposium on the biomedical effects of ozone and related photochemical oxidants; March 1982; Pinehurst, NC. Princeton, NJ: Princeton Scientific Publishers, Inc.; pp. 263-272. (Advances in modern environmental toxicology: v. 5).
- Wolcott, J. A.; Zee, Y. C.; Osebold, J. W. (1982) Exposure to ozone reduces influenza disease severity and alters distribution of influenza viral antigens in murine lungs. *Appl. Environ. Microbiol.* 44: 723-731.
- Xintaras, C.; Johnson, B. L.; Ulrich, C. E.; Terrill, R. E.; Sobacki, M. F. (1966) Application of the evoked response technique in air pollution toxicology. *Toxicol. Appl. Pharmacol.* 8: 77-87.
- Yokoyama, E. (1969) A comparison of the effects of SO₂, NO₂ and O₃ on the pulmonary ventilation: guinea pig exposure experiments. *Sangyo Igaku* 11: 563-568.
- Yokoyama, E. (1974) On the maximal expiratory flow volume curve of rabbits exposed to ozone. *Nippon Kyobu Shikkan Gakkai Zasshi* 12: 556-561.
- Yokoyama, E.; Frank, R. (1972) Respiratory uptake of ozone in dogs. *Arch. Environ. Health* 25: 132-138.
- Yokoyama, E.; Ichikawa, I.; Nambu, Z.; Kawai, K.; Kyono, Y. J. (1984) Respiratory effects of intermittent exposure to ozone of rats. *Environ. Res.* 33: 271-283.
- Zelac, R. E.; Cromroy, H. L.; Bolch, W. E., Jr.; Dunavant, B. G.; Bevis, H. A. (1971a) Inhaled ozone as a mutagen. I. Chromosome aberrations induced in Chinese hamster lymphocytes. *Environ. Res.* 4: 262-282.
- Zelac, R. E.; Cromroy, H. L.; Bolch, W. E., Jr.; Dunavant, B. G.; Bevis, H. A. (1971b) Inhaled ozone as a mutagen. II. Effect on the frequency of chromosome aberrations observed in irradiated Chinese hamsters. *Environ. Res.* 4: 325-342.
- Zitnik, L. A.; Schwartz, L. W.; McQuillen, N. K.; Zee, Y. C.; Osebold, J. W. (1978) Pulmonary changes induced by low-level ozone: morphological observations. *J. Environ. Pathol. Toxicol.* 1: 365-376.

1.12.9 References for Controlled Human Studies of the Effects of Ozone and Other Photochemical Oxidants

- Adams, W. C.; Schelegle, E. S. (1983) Ozone and high ventilation effects on pulmonary function and endurance performance. *J. Appl. Physiol.: Respir. Environ. Exercise Physiol.* 55: 805-812.

References for Controlled Human Studies (cont'd.)

- Avol, E. L.; Linn, W. S.; Venet, T. G.; Shamoo, D. A.; Hackney, J. D. (1984) Comparative respiratory effects of ozone and ambient oxidant pollution exposure during heavy exercise. *J. Air Pollut. Control Assoc.* 34: 804-809.
- Beckett, W. S.; McDonnell, W. F.; Horstman, D. H.; House, D. E. (1985) Role of the parasympathetic nervous system in the acute lung response to ozone. *J. Appl. Physiol.* 59: 1879-1885.
- Bedi, J. F.; Drechsler-Parks, D. M.; Horvath, S. M. (1985) Duration of increased pulmonary function sensitivity to an initial ozone exposure. *Am. Ind. Hyg. Assoc. J.* 46: 731-734.
- Dimeo, M. J.; Glenn, M. G.; Holtzman, M. J.; Sheller, J. R.; Nadel, J. A.; Boushey, H. A. (1981) Threshold concentration of ozone causing an increase in bronchial reactivity in humans and adaptation with repeated exposures. *Am. Rev. Respir. Dis.* 124: 245-248.
- Drechsler-Parks, D. M.; Bedi, J. F.; Horvath, S. M. (1984) Interaction of peroxyacetyl nitrate and ozone on pulmonary functions. *Am. Rev. Respir. Dis.* 130: 1033-1037.
- Farrell, B. P.; Kerr, H. D.; Kulle, T. J.; Sauder, L. R.; Young, J. L. (1979) Adaptation in human subjects to the effects of inhaled ozone after repeated exposure. *Am. Rev. Respir. Dis.* 119: 725-730.
- Folinsbee, L. J.; Drinkwater, B. L.; Bedi, J. F.; Horvath, S. M. (1978) The influence of exercise on the pulmonary changes due to exposure to low concentrations of ozone. In: Folinsbee, L. J.; Wagner, J. A.; Borgia, J. F.; Drinkwater, B. L.; Gliner, J. A.; Bedi, J. F., eds. *Environmental stress: individual human adaptations*. New York, NY: Academic Press; pp. 125-145.
- Folinsbee, L. J.; Bedi, J. F.; Horvath, S. M. (1984) Pulmonary function changes after 1-hour continuous heavy exercise in 0.21 ppm ozone. *J. Appl. Physiol.: Respir. Environ. Exercise Physiol.* 57: 984-988.
- Folinsbee, L. J.; Horvath, S. M. (1986) Persistence of the acute effects of ozone exposure. *Aviat. Space Environ. Med.* (in press).
- Gliner, J. A.; Horvath, S. M.; Folinsbee, L. J. (1983) Pre-exposure to low ozone concentrations does not diminish the pulmonary function response on exposure to higher ozone concentration. *Am. Rev. Respir. Dis.* 127: 51-55.
- Hazucha, M.; Silverman, F.; Parent, C.; Field, S.; Bates, D. V. (1973) Pulmonary function in man after short-term exposure to ozone. *Arch. Environ. Health* 27: 183-188.
- Holtzman, M. I.; Cunningham, J. H.; Sheller, J. R.; Irsigler, G. B.; Nadel, J. A.; Boushey, H. A. (1979) Effect of ozone on bronchial reactivity in atopic and nonatopic subjects. *Am. Rev. Respir. Dis.* 120: 1059-1067.

References for Controlled Human Studies (cont'd.)

- Horvath, S. M.; Gliner, J. A.; Matsen-Twisdale, J. A. (1979) Pulmonary function and maximum exercise responses following acute ozone exposure. *Aviat. Space Environ. Med.* 50: 901-905.
- Horvath, S. M.; Gliner, J. A.; Folinsbee, L. J. (1981) Adaptation to ozone: duration of effect. *Am. Rev. Respir. Dis.* 123: 496-499.
- Kehr1, H. R.; Hazucha, M. J.; Solic, J.; Bromberg, P. A. (1983) The acute effects of 0.2 and 0.3 ppm ozone in persons with chronic obstructive lung disease (COLD). In: Mehlman, M. A.; Lee, S. D.; Mustafa, M. G., eds. International symposium on the biomedical effects of ozone and related photochemical oxidants; March 1982; Pinehurst, NC. Princeton, NJ: Princeton Scientific Publishers, Inc.; pp. 213-225. (Advances in modern environmental toxicology: v. 5).
- Kehr1, H. R.; Hazucha, M. J.; Solic, J. J.; Bromberg, P. A. (1985) Responses of subjects with chronic pulmonary disease after exposures to 0.3 ppm ozone. *Am. Rev. Respir. Dis.* 131: 719-724.
- Kerr, H. D.; Kulle, T. J.; McIlhany, M. L.; Swidersky, P. (1975) Effects of ozone on pulmonary function in normal subjects. *Am. Rev. Respir. Dis.* 111: 763-773.
- Koenig, J. Q.; Covert, D. S.; Morgan, M. S.; Horike, M.; Horike, N.; Marshall, S. G.; Pierson, W. E. (1985) Acute effects of 0.12 ppm ozone or 0.12 ppm nitrogen dioxide on pulmonary function in healthy and asthmatic adolescents. *Am. Rev. Respir. Dis.* 132: 648-651.
- König, G.; Römmelt, H.; Kienele, H.; Dirnagl, K.; Polke, H.; Fruhmann, G. (1980) Changes in the bronchial reactivity of humans caused by the influence of ozone. *Arbeitsmed. Sozialmed. Praeventivmed.* 151: 261-263.
- Kulle, T. J.; Sauder, L. R.; Kerr, H. D.; Farrell, B. P.; Bermel, M. S.; Smith, D. M. (1982) Duration of pulmonary function adaptation to ozone in humans. *Am. Ind. Hyg. Assoc. J.* 43: 832-837.
- Kulle, T. J.; Milman, J. H.; Sauder, L. R.; Kerr, H. D.; Farrell, B. P.; Miller, W. R. (1984) Pulmonary function adaptation to ozone in subjects with chronic bronchitis. *Environ. Res.* 34: 55-63.
- Kulle, T. J.; Sauder, L. R.; Hebel, J. R.; Chatham, M. D. (1985) Ozone response relationships in healthy nonsmokers. *Am. Rev. Respir. Dis.* 132: 36-41.
- Linn, W. S.; Buckley, R. D.; Spier, C. E.; Blessey, R. L.; Jones, M. P.; Fischer, D. A.; Hackney, J. D. (1978) Health effects of ozone exposure in asthmatics. *Am. Rev. Respir. Dis.* 117: 835-843.
- Linn, W. S.; Fischer, D. A.; Medway, D. A.; Anzar, U. T.; Spier, C. E.; Valencia, L. M.; Venct, T. G.; Hackney, J. D. (1982a) Short-term respiratory effects of 0.12 ppm ozone exposure in volunteers with chronic obstructive lung disease. *Am. Rev. Respir. Dis.* 125: 658-663.

References for Controlled Human Studies (cont'd.)

- Linn, W. S.; Medway, D. A.; Anzar, U. T.; Valencia, L. M.; Spier, C. E.; Tsao, F. S-O.; Fischer, D. A.; Hackney, J. D. (1982b) Persistence of adaptation to ozone in volunteers exposed repeatedly over six weeks. *Am. Rev. Respir. Dis.* 125: 491-495.
- Linn, W. S.; Shamoo, D. A.; Venet, T. G.; Spier, C. E.; Valencia, L. M.; Anzar, U. T.; Hackney, J. D. (1983) Response to ozone in volunteers with chronic obstructive pulmonary disease. *Arch. Environ. Health* 38: 278-283.
- McDonnell, W. F.; Horstmann, D. H.; Hazucha, M. J.; Seal, E., Jr.; Haak, E. D.; Salaam, S.; House, D. E. (1983) Pulmonary effects of ozone exposure during exercise: dose-response characteristics. *J. Appl. Physiol.: Respir. Environ. Exercise Physiol.* 54: 1345-1352.
- McDonnell, W. F., III; Hortsman, D. H.; Abdul-Salaam, S.; House, D. E. (1985a) Reproducibility of individual responses to ozone exposure. *Am. Rev. Respir. Dis.* 131: 36-40.
- McDonnell, W. F., III; Chapman, R. S.; Leigh, M. W.; Strobe, G. L.; Collier, A. M. (1985b) Respiratory responses of vigorously exercising children to 0.12 ppm ozone exposure. *Am. Rev. Respir. Dis.* 132: 875-879.
- McDonnell, W. F.; Chapman, R. S.; Horstman, D. H.; Leigh, M. W.; Abdul-Salaam, S. (1985c) A comparison of the responses of children and adults to acute ozone exposure. In: Lee, S. D., ed. *Evaluation of the scientific basis for ozone/oxidants standards*; November 1984; Houston, TX. Pittsburgh, PA: Air Pollution Control Association; pp. 317-328 (APCA international specialty conference transactions: TR-4).
- Silverman, F. (1979) Asthma and respiratory irritants (ozone). *EHP Environ. Health Perspect.* 29: 131-136.
- Silverman, F.; Folinsbee, L. J.; Barnard, J.; Shephard, R. J. (1976) Pulmonary function changes in ozone - interaction of concentration and ventilation. *J. Appl. Physiol.* 41: 859-864.
- Solic, J. J.; Hazucha, M. J.; Bromberg, P. A. (1982) The acute effects of 0.2 ppm ozone in patients with chronic obstructive pulmonary disease. *Am. Rev. Respir. Dis.* 125: 664-669.

1.12.10 References for Field and Epidemiological Studies of the Effects of Ozone and Other Photochemical Oxidants

- Avol, E. L.; Linn, W. S.; Shamoo, D. A.; Venet, T. G.; Hackney, J. D. (1983) Acute respiratory effects of Los Angeles smog incontinuously exercising adults. *J. Air Pollut. Control Assoc.* 33: 1055-1060.
- Avol, E. L.; Linn, W. S.; Venet, T. G.; Shamoo, D. A.; Hackney, J. D. (1984) Comparative respiratory effects of ozone and ambient oxidant pollution exposure during heavy exercise. *J. Air Pollut. Control Assoc.* 34: 804-809.

References for Field and Epidemiological Studies (cont'd.)

- Avol, E. L.; Linn, W. S.; Shamoo, D. A.; Valencia, L. M.; Anzar, U. T.; Hackney, J. D. (1985a) Respiratory effects of photochemical oxidant air pollution in exercising adolescents. *Am. Rev. Respir. Dis.* 132: 619-622.
- Avol, E. L.; Linn, W. S.; Shamoo, D. A.; Valencia, L. M.; Anzar, U. T.; Hackney, J. D. (1985b) Short-term health effects of ambient air pollution in adolescents. In: Lee, S. D., ed. Evaluation of the scientific basis for ozone/oxidants standards; November 1984; Houston, TX. Pittsburgh, PA: Air Pollution Control Association; pp. 329-336. (APCA international specialty conference transactions: TR-4).
- Avol, E. L.; Linn, W. S.; Venet, T. G.; Shamoo, D. A.; Spier, C. E.; Hackney, J. D. (1985c) Comparative effects of laboratory generated ozone and ambient oxidant exposure in continuously exercising subjects. In: Lee, S. D., ed. Evaluation of the scientific basis for ozone/oxidants standards; November 1984; Houston, TX. Pittsburgh, PA: Air Pollution Control Association; pp. 216-225. (APCA international specialty conference transactions: TR-4).
- Bock, N.; Lippmann, M.; Liroy, P.; Munoz, A.; Speizer, F. (1985) The effects of ozone on the pulmonary function of children. In: Lee, S. D., ed. Evaluation of the scientific basis for ozone/oxidants standards; November 1984; Houston, TX. Pittsburgh, PA: Air Pollution Control Association; pp. 297-308. (APCA international specialty conference transactions: TR-4).
- Hammer, D. I.; Hasselblad, V.; Portnoy, B.; Wehrle, P. F. (1974) Los Angeles student nurse study: daily symptom reporting and photochemical oxidants. *Arch. Environ. Health* 28: 255-260.
- Herman, D. R. (1972) The effect of oxidant air pollution on athletic performance [master's thesis]. Chapel Hill, NC: University of North Carolina.
- Holguin, A. H.; Buffler, P. A.; Contant, C. F., Jr.; Stock, T. H.; Kotchmar, D.; Hsi, B. P.; Jenkins, D. E.; Gehan, B. M.; Noel, L. M.; Mei, M. (1985) The effects of ozone on asthmatics in the Houston area. In: Lee, S. D., ed. Evaluation of the scientific basis for ozone/oxidants standards; November 1984; Houston, TX. Pittsburgh, PA: Air Pollution Control Association; pp. 262-280. (APCA international specialty conference transactions: TR-4).
- Lebowitz, M. D. (1984) The effects of environmental tobacco smoke exposure and gas stoves on daily peak flow rates in asthmatic and non-asthmatic families. *Eur. J. Respir. Dis.* 65 (suppl. 133): 90-97.
- Lebowitz, M. D.; O'Rourke, M. K.; Dodge, R.; Holberg, C. J.; Corman, G.; Hoshaw, R. W.; Pinnas, J. L.; Barbee, R. A.; Sneller, M. R. (1982) The adverse health effects of biological aerosols, other aerosols, and indoor microclimate on asthmatics and nonasthmatics. *Environ. Int.* 8: 375-380.
- Lebowitz, M. D.; Holberg, C. J.; Dodge, R. R. (1983) Respiratory effects on populations from low level exposures to ozone. Presented at: 76th annual meeting of the Air Pollution Control Association; June; Atlanta, GA. Pittsburgh, PA: Air Pollution Control Association; paper no. 83-12.5.

References for Field and Epidemiological Studies (cont'd.)

- Lebowitz, M. D.; Holberg, C. J.; Boyer, B.; Hayes, C. (1985) Respiratory symptoms and peak flow associated with indoor and outdoor air pollutants in the southwest. *J. Air Pollut. Control Assoc.* 35: 1154-1158.
- Linn, W. S.; Jones, M. P.; Bachmayer, E. A.; Spier, C. E.; Mazur, S. F.; Avol, E. L.; Hackney, J. D. (1980) Short-term respiratory effects of polluted ambient air: a laboratory study of volunteers in a high-oxidant community. *Am. Rev. Respir. Dis.* 121: 243-252.
- Linn, W. S.; Avol, E. L.; Hackney, J. D. (1983) Effects of ambient oxidant pollutants on humans: a movable environmental chamber study. In: Lee, S. D.; Mustafa, M. G.; Mehlman, M. A., eds. *International symposium on the biomedical effects of ozone and related photochemical oxidants; March 1982; Pinehurst, NC. Princeton, NJ: Princeton Scientific Publishers, Inc.; pp. 125-137. (Advances in modern toxicology: v. 5).*
- Lioy, P. J.; Vollmuth, T. A.; Lippman, M. (1985) Persistence of peak flow decrement in children following ozone exposures exceeding the National Ambient Air Quality Standard. *J. Air Pollut. Control Assoc.*: 35: 1068-1071.
- Lippmann, M.; Lioy, P. J.; Leikauf, G.; Green, K. B.; Baxter, D.; Morandi, M.; Pasternack, B. S. (1983) Effects of ozone on the pulmonary function of children. In: Lee, S. D.; Mustafa, M. G.; Mehlman, M. A., eds. *International symposium on the biomedical effects of ozone and related photochemical oxidants; March 1982; Pinehurst, NC. Princeton, NJ: Princeton Scientific Publishers, Inc.; pp. 423-446. (Advances in modern toxicology: v. 5).*
- Makino, K.; Mizoguchi, I. (1975) Symptoms caused by photochemical smog. *Nippon Koshu Eisei Zasshi* 22: 421-430.
- Wayne, W. S.; Wehrle, P. F.; Carroll, R. E. (1967) Oxidant air pollution and athletic performance. *J. Am. Med. Assoc.* 199: 901-904.
- Whittemore, A. S.; Korn, E. L. (1980) Asthma and air pollution in the Los Angeles area. *Am. J. Public Health* 70: 687-696.

1.12.11 References for Evaluation of Health Effects Data for Ozone and Other Photochemical Oxidants

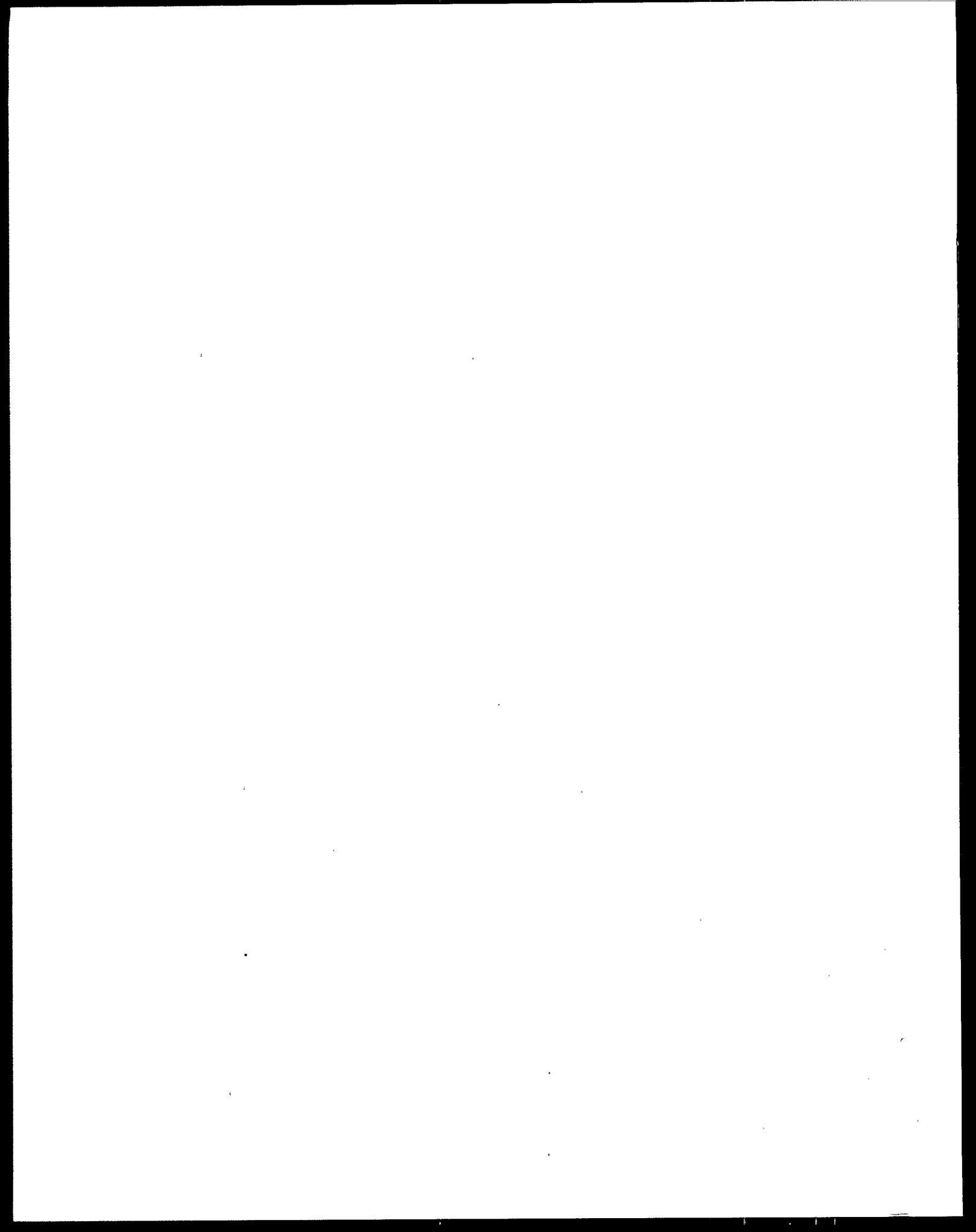
- Avol, E. L.; Linn, W. S.; Venet, T. G.; Shamoo, D. A.; Hackney, J. D. (1984) Comparative respiratory effects of ozone and ambient oxidant pollution exposure during heavy exercise. *J. Air Pollut. Control Assoc.* 34: 804-809.
- Avol, E. L.; Linn, W. S.; Shamoo, D. A.; Valencia, L. M.; Anzar, U. T.; Hackney, J. D. (1985a) Respiratory effects of photochemical oxidant air pollution in exercising adolescents. *Am. Rev. Respir. Dis.* 132: 619-622.

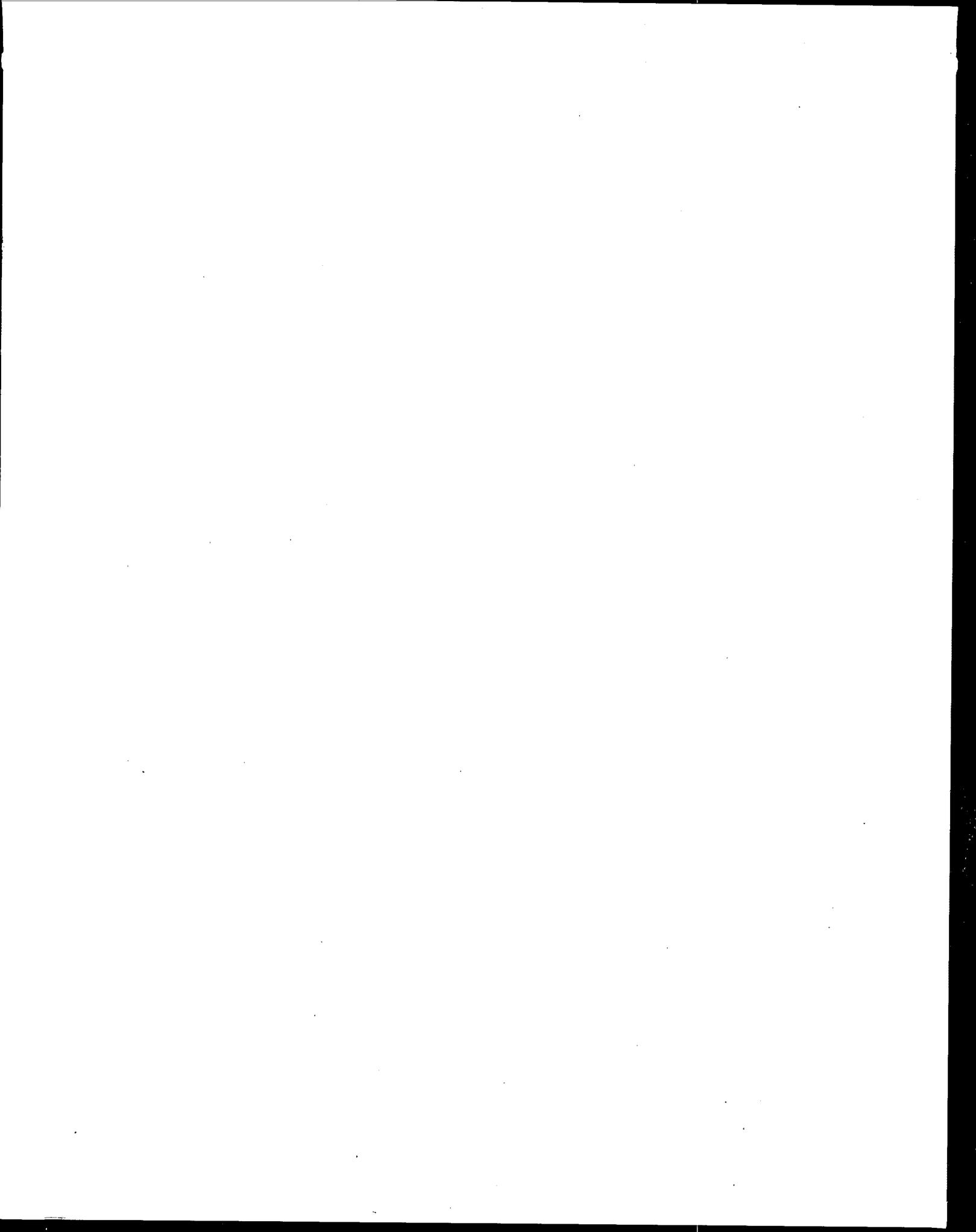
References for Evaluation of Health Effects Data (cont'd.)

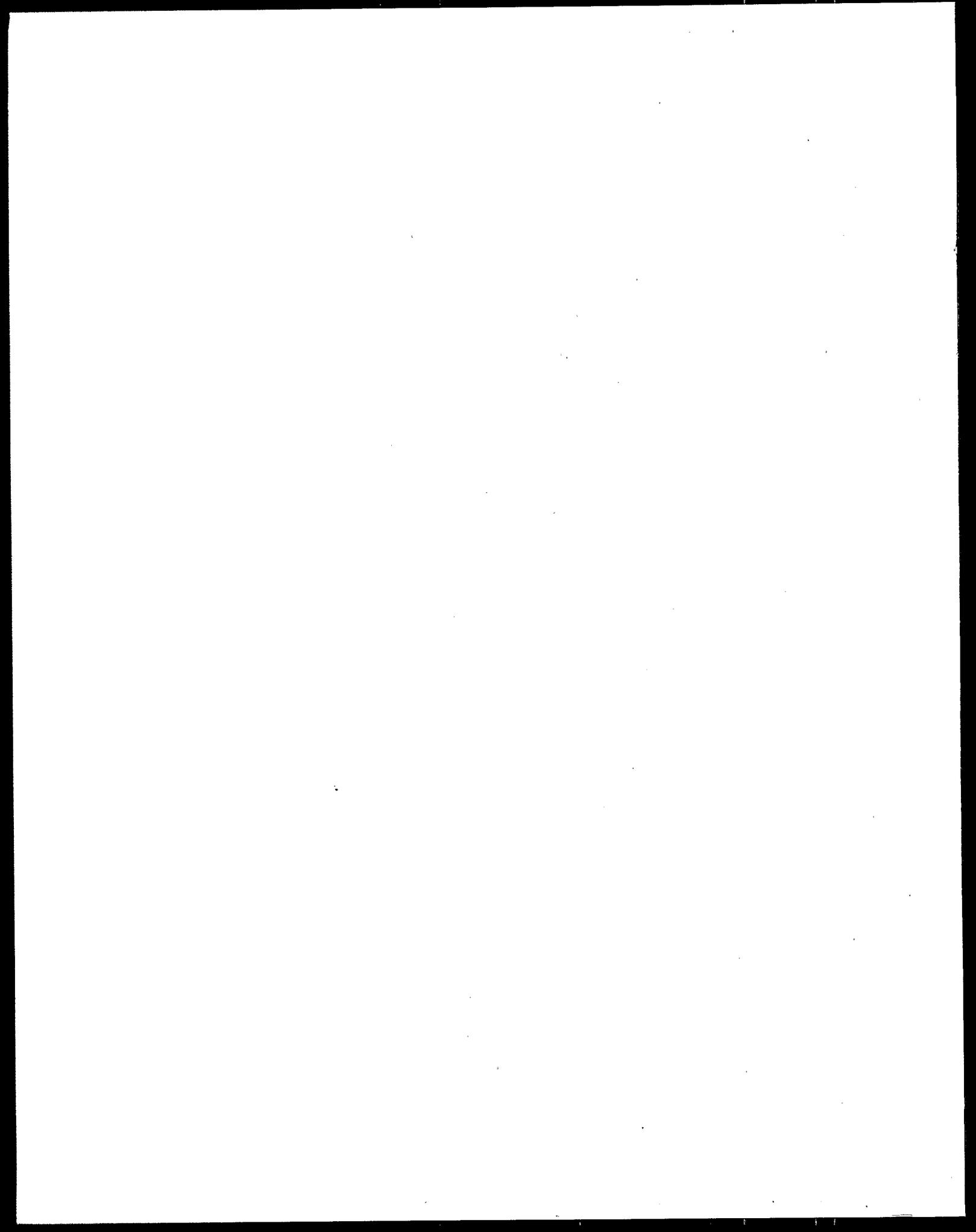
- Avol, E. L.; Linn, W. S.; Shamoo, D. A.; Valencia, L. M.; Anzar, U. T.; Hackney, J. D. (1985b) Short-term health effects of ambient air pollution in adolescents. In: Lee, S. D., ed. Evaluation of the scientific basis for ozone/oxidants standards; November 1984; Houston, TX. Pittsburgh, PA: Air Pollution Control Association; pp. 329-336 (APCA international specialty conference transactions: TR-4).
- Avol, E. L.; Linn, W. S.; Venet, T. G.; Shamoo, D. A.; Spier, C. E.; Hackney, J. D. (1985c) Comparative effects of laboratory generated ozone and ambient oxidant exposure in continuously exercising subjects. In: Lee, S. D., ed. Evaluation of the scientific basis for ozone/oxidants standards; November 1984; Houston, TX. Pittsburgh, PA: Air Pollution Control Association; pp. 216-225. (APCA international specialty conference transactions: TR-4).
- Bedi, J. F.; Dreschsler-Parks, D. M.; Horvath, S. M. (1985) Duration of increased pulmonary function sensitivity to an initial ozone exposure. *Am. Ind. Hyg. Assoc. J.* 46: 731-734.
- Folinsbee, L. J.; Horvath, S. M. (1986) Persistence of the acute effects of ozone exposure. *Aviat. Space Environ. Med.* (in press).
- Folinsbee, L. J.; Bedi, J. F.; Horvath, S. M. (1984) Pulmonary function changes after 1-hour continuous heavy exercise in 0.21 ppm ozone. *J. Appl. Physiol.: Respir. Environ. Exercise Physiol.* 57: 984-988.
- Gliner, J. A.; Horvath, S. M.; Folinsbee, L. J. (1983) Pre-exposure to low ozone concentrations does not diminish the pulmonary function response on exposure to higher ozone concentration. *Am. Rev. Respir. Dis.* 127: 51-55.
- Horvath, S. M.; Gliner, J. A.; Folinsbee, L. J. (1981) Adaptation to ozone: duration of effect. *Am. Rev. Respir. Dis.* 123: 496-499.
- Kulle, T. J.; Sauder, L. R.; Hebel, J. R.; Chatham, M. D. (1985) Ozone response relationships in healthy nonsmokers. *Am. Rev. Respir. Dis.* 132: 36-41.
- McDonnell, W. F.; Horstmann, D. H.; Hazucha, M. J.; Seal, E., Jr.; Haak, E. D.; Salaam, S.; House, D. E. (1983) Pulmonary effects of ozone exposure during exercise: dose-response characteristics. *J. Appl. Physiol.: Respir. Environ. Exercise Physiol.* 54: 1345-1352.
- McDonnell, W. F., III; Horstman, D. H.; Abdul-Salaam, S.; House, D. E. (1985a) Reproducibility of individual responses of ozone exposure. *Am. Rev. Respir. Dis.* 131: 36-40.
- McDonnell, W. F., III; Chapman, R. S.; Leigh, M. W.; Strobe, G. L.; Collier, A. M. (1985b) Respiratory responses of vigorously exercising children to 0.12 ppm ozone exposure. *Am. Rev. Respir. Dis.* 132: 875-879.

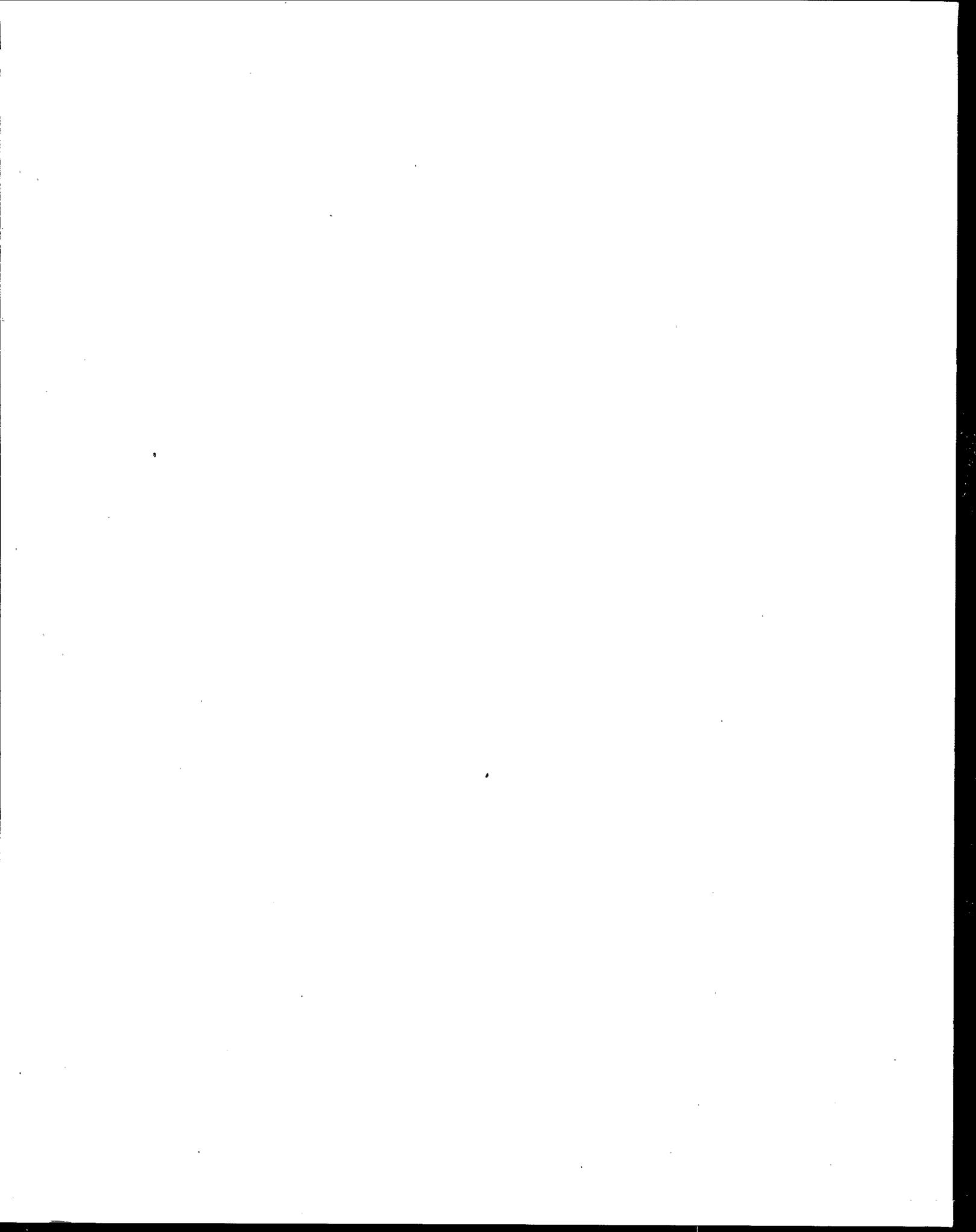
References for Evaluation of Health Effects Data (cont'd.)

McDonnell, W. F.; Chapman, R. S.; Horstman, D. H.; Leigh, M. W.; Abdul-Salaam, S. (1985c) A comparison of the responses of children and adults to acute ozone exposure. In: Lee, S. D., ed. Evaluation of the scientific basis for ozone/oxidants standards; November 1984; Houston, TX. Pittsburgh, PA: Air Pollution Control Association; pp. 317-328. (APCA international specialty conference transactions: TR-4).









United States
Environmental Protection
Agency

Center for Environmental Research
Information
Cincinnati OH 45268

Official Business
Penalty for Private Use, \$300

Please make all necessary changes on the above label,
detach or copy, and return to the address in the upper
left-hand corner.

If you do not wish to receive these reports CHECK HERE
detach, or copy this cover, and return to the address in the
upper left-hand corner.

EPA/600/8-84/020aF