AIR QUALITY CRITERIA FOR NITROGEN OXIDES

ENVIRONMENTAL PROTECTION AGENCY

AIR QUALITY CRITERIA

FOR

NITROGEN OXIDES

ENVIRONMENTAL PROTECTION AGENCY
Air Pollution Control Office
Washington, D. C.
January 1971

Air Pollution Control Office Publication No. AP-84

For sale by the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C., 20402 - Price $1.50

PREFACE

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

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

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

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

Briefly, the Act calls for the Administrator, Environmental Protection Agency to define the broad atmospheric areas of the Nation in which climate, meteorology, and topography, all of which influence the capacity of air to dilute and disperse pollution, are generally homogeneous.

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

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

The Air Pollution Control Office of EPA will review, evaluate, and approve these standards and plans; and, once they are approved, the States will be expected to take action to control pollution sources in the manner outlined in their plans.

At the direction of the Administrator of the Environmental Protection Agency, the Air Pollution Control Office has established appropriate programs to carry out the several

Federal responsibilities specified in the legislation. Air Quality Criteria information has now been published on several pollutants: particulate matter, sulfur oxides, carbon monoxide, photochemical oxidants, and hydrocarbons.

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

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

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

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

The Administration is pleased to acknowledge the efforts of each of the persons specifically named, as well as the many not named, who contributed to the publication of this volume. Their participation does not necessarily imply their complete endorsement of all the conclusions presented in the document, however, for in the last analysis APCO alone retains full responsibility for its content.

JOHN T. MIDDLETON
Acting Commissioner
Air Pollution Control Office
Environmental Protection Agency

NATIONAL AIR QUALITY CRITERIA ADVISORY COMMITTEE

Chairman
Dr. Delbert S. Barth, Director
Bureau of Criteria and Standards
Air Pollution Control Office
Environmental Protection Agency

Dr. Seymour Calvert*
Dean
School of Engineering
University of California at Riverside
Riverside, California

Dr. Adrian Ramond Chamberlain*
President
Colorado State University
Fort Collins, Colorado

Dr. James P. Dunn
Staff Medical Director
Western Electric Company, Inc.
New York, New York

Dr. David M. Gates*
Director
Missouri Botanical Gardens
St. Louis, Missouri

Dr. Neil V. Hakala
President, Esso Research and
Engineering Company
Linden, New Jersey

Dr. Ian T. Higgins
Professor, School of Public Health
Department of Community Health Services
The University of Michigan
Ann Arbor, Michigan

Dr. Donald A. Jensen*
Director, Automotive Emissions Office
Engineering Staff
Ford Motor Company
Dearborn, Michigan

Dr. Herbert E. Klarman*
Department of Environmental Medicine
and Community Health
Downstate Medical Center
State University of New York
Brooklyn, New York

Frank J. Massey, Jr.
Professor, School of Public Health
University of California
Los Angeles, California

Dr. George L. McNew
Director, Boyce Thompson
Institute for Plant Research, Inc.
Yonkers, New York

Mr. Alexander Rihm, Jr.
Assistant Commissioner
Division of Air Resources
Department of Health
State of New York
Albany, New York

Dr. Frederick Sargent II
Provost
Western Washington State College
Bellingham, Washington

Dr. Vincent J. Schaefer
Director, Atmospheric Sciences
Research Center
State University of New York
Schenectady, New York

Dr. Warren Winkelstein, Jr.
Professor and Head
Division of Epidemiology
School of Public Health
University of California at Berkeley
Berkeley, California

*Term expired June 30, 1970.

CONTRIBUTORS AND REVIEWERS

Dr. David M. Anderson
Assistant Manager, Environmental Quality Control
Bethlehem Steel Corporation
Bethlehem, Pennsylvania

Mr. Roynal W. Andrews
Chief Environmental Engineer
Aluminum Company of America
Pittsburgh, Pennsylvania

Dr. Mario C. Battigelli
Department of Medicine
School of Medicine
University of North Carolina
Chapel Hill, North Carolina

Mr. Francis E. Blacet
Department of Chemistry
University of California at Los Angeles
Los Angeles, California

Dr. Fred W. Bowditch
Director, Emission Control
General Motors Technical Center
Warren, Michigan

Dr. Leslie A. Chambers
Director, Institute of Environmental Health
School of Public Health of Houston
University of Texas
Houston, Texas

Mr. Robert L. Chass
Chief, Deputy Air Pollution Control Officer
Los Angeles County Air Pollution Control District
Los Angeles, California

Dr. R. S. Danchik
Analytical Chemistry Division
Alcoa Research Laboratories
New Kensington, Pennsylvania

Dr. Richard Ehrlich
Director, Life Sciences Research
Illinois Institute of Technology Research Institute
Chicago, Illinois

Dr. W. L. Faith
Consulting Chemical Engineer
San Marino, California

Dr. Hans Falk
Associate Director for Laboratory Research
National Institute of Environmental Health Sciences
Research Triangle Park, North Carolina

Dr. Gustave Freeman, Jr.
Life Sciences Division
Stanford Research Institute
Menlo Park, California

Mr. James R. Garvey
President and Director of Research
Bituminous Coal Research, Inc.
Pittsburgh, Pennsylvania

Mr. J. L. Gilliland
Technical Director
Ideal Cement Company
Denver, Colorado

Dr. John R. Goldsmith
Chief, Environmental Epidemiology Unit
California Department of Public Health
Berkeley, California

Mr. Walter J. Hamming
Chief Air Pollution Analyst
Los Angeles County Air Pollution Control District
Los Angeles, California

Mr. Charles M. Heinen
Chief Engineer, Materials Laboratories
Product Planning & Development Staff
Chrysler Corporation
Detroit, Michigan

Dr. Charles H. Hine
Clinical Professor of Occupational & Environmental Medicine
University of California Medical School
San Francisco, California

Dr. Kenneth D. Johnson
Staff Engineer, Air Quality Program
Manufacturing Chemists' Association
Washington, D. C.

Dr. Charles G. Kramer
Associate Medical Director, Midland Division
The Dow Chemical Company
Midland, Michigan

Dr. Lawrence D. Kornreich
Director, Chemistry Division
The Research Corporation of New England
Hartford, Connecticut

Dr. Paul Kotin
Director, National Institute of Environmental Health Sciences
Research Triangle, North Carolina

Dr. Philip A. Leighton
Professor Emeritus
Stanford University

Mr. Arthur Levy
Chief, Air Water and Solid Waste Chemistry Division
Batelle Memorial Institute
Columbus, Ohio

Dr. Joseph A. Lieberman
Assistant Administrator for Research and Development
Environmental Health Service
Department of Health, Education, and Welfare
Washington, D. C.

Dr. H. N. MacFarland
Professor and Director, Centre of Research on Environmental Quality
York University
Downsview, Ontario, Canada

Dr. Herbert C. McKee
Assistant Director, Department of Chemistry and Chemical Engineering
Southwest Research Institute
Houston, Texas

Dr. Elmer Robinson
Chairman, Environmental Research Department
Stanford Research Institute
Menlo Park, California

Dr. Lawrence M. Roslinski
Professional Associate
Advisory Center on Toxicology
National Research Council

Mr. Francis Scofield
Vice President, Technical Affairs
National Paint, Varnish, and Lacquer Association, Inc.
Washington, D. C.

Dr. R. W. Scott
Coordinator for Conservation Technology
Esso Research and Engineering Company
Linden, New Jersey

Mr. William J. Stanley
Director, Chicago Department of Air Pollution Control
Chicago, Illinois

Dr. Edgar R. Stephens
Research Chemist, Statewide Air Pollution Research Center
University of California at Riverside
Riverside, California

Mr. Morton Sterling
Air Pollution Control Division
Wayne County Department of Health
Detroit, Michigan

Dr. Herbert E. Stokinger
Chief, Laboratory of Toxicology and Pathology
Bureau of Occupational Safety and Health
Environmental Control Administration
Department of Health, Education, and Welfare
Cincinnati, Ohio

Dr. E. Suriani
Research Scientist, Division of Research
Cottrell Environmental Systems, Inc.
Bound Brook, New Jersey

Dr. O. Clifton Taylor
Associate Director, Statewide Air Pollution Research Center
University of California at Riverside
Riverside, California

Dr. Ralph C. Wands
Director, Advisory Center on Toxicology
National Research Council
Washington, D. C.

Dr. Peter O. Warner
Laboratory Supervisory, Air Pollution Control Division
Wayne County Department of Health
Detroit, Michigan

Dr. Lowell G. Wayne
Consultant, Air Pollution Chemistry
Los Angeles, California

Dr. Eugene E. Weaver
Product Development Group
Ford Motor Company
Detroit, Michigan

Mr. Melvin Weisburd
Manager, Environmental Health Systems
System Development Corporation
Santa Monica, California

Dr. Bernard Weinstock
Manager and Senior Scientist, Fuel Sciences Department
Ford Motor Company
Dearborn, Michigan

Dr. John E. Yocom
Director, Engineering and Technical Programs
The Travelers Research Corporation
Hartford, Connecticut

FEDERAL AGENCY LIAISON REPRESENTATIVES

Department of Agriculture
Dr. Theodore C. Byerly
Assistant Director of Science and Education
Office of the Secretary

Department of Commerce
Dr. James R. Hibbs
Advisor on Environmental Quality

Department of Defense
Lt. Col. Herbert E. Bell
Staff Assistant, BCS
Office of the Assistant Secretary of Defense

Department of Housing and Urban Development
Mr. Samuel C. Jackson
Assistant Secretary for Metropolitan Development

Department of Interior
Mr. William L. Crentz
Director, Energy
Bureau of Mines

Department of Justice
Mr. Walter Kiechel, Jr.
Assistant Chief, General Litigation Section
Land & Natural Resources Division

Department of Labor
Mr. Robert D. Gidel
Deputy Director, Bureau of Labor Standards
Railway Labor Building

Post Office Department
Mr. W. Norman Meyers
Environmental Coordinator
Bureau of Facilities

Department of Transportation
Mr. William H. Close
Chief, Research Division
Office of Noise Abatement

Department of Treasury
Mr. Gerald M. Brannon
Director, Office of Tax Analysis

U. S. Atomic Energy Commission
Dr. Martin B. Biles
Director of Division of Operational Safety

Federal Power Commission
Mr. George Tomlinson
Acting Chief, Bureau of Power

General Services Administration
Mr. Thomas E. Crocker
Director of Repair & Improvement Division
Public Buildings Service

National Aeronautics & Space Administration
Maj. General R. H. Curtin, USAF (Ret.)
National Aeronautics & Space Administration

National Science Foundation
Dr. Eugene W. Bierly
Program Director for Meteorology
Division of Environmental Sciences

Tennessee Valley Authority
Dr. F. E. Gartrell
Assistant Director of Health

Veterans Administration
Mr. Gerald M. Hollander
Director of Architecture & Engineering

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

CHAPTER 1.

INTRODUCTION

Pursuant to authority delegated to the Commissioner, Air Pollution Control Office* of the Environmental Protection Agency, *Air Quality Criteria for Nitrogen Oxides* is issued in accordance with Section 107(b) of the Clean Air Act (42 U.S.C. 1857-18571).

Air quality *criteria* are an expression of the scientific knowledge of the relationship between various concentrations of pollutants in the air and their adverse effects on man and his environment. They are issued to assist the States in developing air quality standards. Air quality criteria are descriptive; that is, they describe the effects that have been observed to occur when the ambient air level of a pollutant has reached or exceeded a specific figure for a specific time period. In developing criteria, many factors have to be considered. The chemical and physical characteristics of the pollutants and the techniques available for measuring these characteristics must be considered, along with exposure time, relative humidity, and other conditions of the environment. The criteria must consider the contribution of all such variables to the effects of air pollution on human health, agriculture, materials, visibility, and climate. Further, the individual characteristics of the receptor must be taken into account. Table 1-1 is a listing of the major factors that need to be considered in developing criteria.†

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

Several oxides of nitrogen are found in the atmosphere, but only nitric oxide (NO) and nitrogen dioxide (NO_2) are important as air pollutants. This document focuses on them and uses the symbol NO_x to represent the composite of the two. The others seldom occur in appreciable quantities and then, only under special conditions. A complex relationship that exists among the oxides of nitrogen, photochemical oxidants, and hydrocarbons is discussed here primarily in terms of the nitrogen oxides. Photochemical oxidants and hydrocarbons are discussed in companion documents: AP-63, *Air Quality Criteria for Photochemical Oxidants,* and AP-64, *Air Quality Criteria for Hydrocarbons.*

The Air Pollution Control Office (APCO) of EPA is currently advocating the use of the mass per unit volume measure to express atmospheric concentrations of air pollutants (e.g., micrograms per cubic meter, $\mu g/m^3$). In most instances, gaseous pollutants have been reported on a volume-ratio basis, that is, parts per million (ppm); almost all data for atmospheric oxides of nitrogen were originally recorded as ppm. Conversion from volume (ppm) to mass ($\mu g/m^3$) units requires a knowledge of the gas density, which varies with the temperature and pressure of measurement. In this document 25° C (77° F) has been taken as standard temperature, and 760 mm Hg (atmospheric pressure at sea level) as standard pressure. It should be remembered

*Formerly the National Air Pollution Control Administration.

†Adapted from:

S. Calvert. Statement for air quality criteria hearings held by the Subcommittee on Air and Water Pollution of the U.S. Senate Committee on Public Works, July 30, 1968, published in "Hearings Before the Subcommittee on Air and Water Pollution of the Committee on Public Works, United States Senate (Air Pollution-1968, Part 2.)"

Table 1–1. FACTORS TO BE CONSIDERED IN DEVELOPING AIR QUALITY CRITERIA

Factor	Components
Properties of pollution	Adsorbed gases Coexisting pollutants Kinetics of formation Residence time
Properties of pollutant	Concentration Chemistry Physical state
Methods of measurement	Chemical analysis Colorimetric Coulometric Spectroscopic Gas chromatographic Sampling techniques
Exposure parameters	Time Temperature Pressure Humidity
Characteristics of receptor	Physical Susceptibility State of health Rate, site, and agent of transfer to
Responses (effects)	
Health	Type of illness Diagnosable Latent Increase in general susceptibility Victim Animals Man
Human welfare	Economics Sociology Epidemiology
Vegetation	Crops Ornamental plants
Material	Objectionable surface decomposition Corrosion Deterioration
Atmospheric	Radiation Temperature Diffusion Dispersion

that ambient concentrations of nitrogen oxides expressed in mass units ($\mu g/m^3$) will therefore vary depending on the ratios and densities of the specific oxides involved. For purposes of consistency and readability, all references to nitrogen oxides in this document are expressed in terms of NO_2 mass per unit volume, unless otherwise specified. Similarly, references to hydrocarbon (HC) and oxidant concentrations will be in terms of mass of methane and ozone (O_3) per unit volume, respectively.

This publication reviews the chemical and physical characteristics of the nitrogen oxides and considers the relative merit of various analytical methods for measuring them in the atmosphere. It also discusses their effects on visibility, vegetation, and materials; their toxicological effects on animals and on man; and epidemiological studies that assess the general population dose response and the specific response of children to nitrogen oxides.

In general, the terminology employed follows usage recommended in the publications style guide of the American Chemical Society. A conversion table for mass and volume units of measurement, and a subject index are provided.

This document is not intended as a complete, detailed literature review, and it does not cite every published article relating to nitrogen oxides in the ambient atmosphere. The literature has been reviewed thoroughly, however, for information related to the development of criteria through April 1970, and the document not only summarizes the current scientific knowledge of nitrogen oxides air pollution, but also points up the major deficiencies in that knowledge and the needs for further research. The results and conclusions of foreign investigations were evaluated for their possible application to the air pollution problem in the United States and relevant ones are cited.

The technological and economic aspects of air pollution control are considered in companion volumes to criteria documents. The best methods and techniques for controlling the sources of nitrogen oxide emissions, as well as the costs of applying these techniques, are described in: AP-67, *Control Techniques for Nitrogen Oxide Emissions from Stationary Sources,* and AP-66, *Control Techniques for Carbon Monoxide, Nitrogen Oxide, and Hydrocarbon Emissions from Mobile Sources.*

CHAPTER 2.

PROPERTIES OF NITROGEN OXIDES AND PHYSICAL EFFECTS ON LIGHT TRANSMISSION

A. INTRODUCTION

Of the various oxides of nitrogen, the most important as air pollutants are nitric oxide (NO) and nitrogen dioxide (NO_2). Other known oxides of nitrogen include nitrous oxide (N_2O), nitrogen sesquioxide (N_2O_3), nitrogen tetroxide (N_2O_4), and nitrogen pentoxide (N_2O_5), but only N_2O is present in the atmosphere in appreciable concentrations.

The term NO_x is used to represent the composite atmospheric concentration of nitric oxide and nitrogen dioxide throughout this document.

B. OCCURRENCE

Nitric oxide (NO) and a comparatively small amount of NO_2 are formed under high-temperature conditions such as those that accompany the burning of fossil fuels. They are emitted to the atmosphere from automobile exhausts, furnace stacks, incinerators, and vents from certain chemical processes. Although vast quantities are produced by natural biological reactions, the resultant concentrations of atmospheric NO_x are low because these reactions take place over wide areas. Most of the NO_x produced technologically is in the form of NO, which is subsequently oxidized in the atmosphere to the more toxic and irritant NO_2. Normally, at low-NO concentrations of 1.2 mg/m^3 (1 ppm) or less, the direct reaction with oxygen of the air proceeds slowly. The oxidation of NO to NO_2 is speeded up enormously, however, by photochemical processes involving reactive hydrocarbons.

Nitrous oxide (N_2O) is not considered an air contaminant. There is no evidence to suggest N_2O is involved in photochemical reactions in the lower atmosphere. Formation of N_2O is largely due to the decomposition of nitrogen compounds by soil bacteria and to reactions between nitrogen and atomic oxygen or ozone in the upper atmosphere.[1] The mean atmospheric concentration of N_2O is about 449 μg/m^3 (0.25 ppm).[2] The oxides, N_2O_4, N_2O_3, and N_2O_5, are not found in appreciable concentrations under urban atmospheric conditions. Nevertheless, it is feasible that even in small concentrations, N_2O_3 and N_2O_5 could be involved in the reactions leading to photochemical air pollution.[3]

C. PROPERTIES OF NITROGEN OXIDES

1. Nitric Oxide (NO)

Nitric oxide is a colorless, odorless gas, slightly soluble in water. It is the primary product formed during high-temperature combustion processes when atmospheric oxygen and nitrogen combine according to the following endothermic reaction:

$$N_2 + O_2 \rightleftarrows 2\,NO \qquad (1)$$

This reaction continues to an equilibrium level, dependent upon variables such as the temperature of the flame, the concentration of each gas, and the movement of the gases through different zones of temperatures, pressures, and concentrations. When rapid cooling follows combustion, time is inadequate for equilibrium to develop, and so NO persists in the flame products.

2. Nitrogen Dioxide (NO_2)

Nitrogen dioxide is a reddish-orange-brown gas with a characteristic pungent odor. Though its boiling point is 21.2°C, the low partial pressure of NO_2 in the atmosphere restricts it to the gas phase at usual atmospheric temperatures. NO_2 is corrosive and highly oxidizing, and may be physiologically irritating and toxic. Although NO_2 reacts with water to form nitric acid and either nitrous acid or NO:

$$2\ NO_2 + H_2O \rightarrow HNO_3 + HNO_2$$
$$\text{or} \qquad (2)$$
$$3\ NO_2 + H_2O \rightarrow 2HNO_3 + NO,$$

the reaction of NO_2 with H_2O has not been shown to have any significance in polluted air.[3]

Although NO_2 exists in equilibrium with its dimer, nitrogen tetroxide (N_2O_4):

$$2\ NO_2 \rightleftarrows N_2O_4 \qquad (3)$$

the fraction present in dimer form is negligible at atmospheric concentrations of NO_2.

NO_2 is a product of the termolecular reaction of nitric oxide with oxygen:

$$2\ NO + O_2 \rightarrow 2\ NO_2 \qquad (4)$$

Since two molecules of NO are involved, the NO_2-formation rate varies with the square of the NO concentration. Doubling the NO concentration increases the reaction rate four times. A generally accepted mechanism to explain this NO_2 formation was suggested by Trautz[4]:

$$NO + O_2 \underset{b}{\overset{a}{\rightleftarrows}} NO_3 \qquad (5)$$

$$NO_3 + NO \rightarrow 2\ NO_2 \qquad (6)$$

Thus, NO in the presence of O_2 produces the highly reactive and unstable nitrogen trioxide molecule (reaction 5a), which has two possible fates, i.e., decomposition back to NO and O_2 (reaction 5b), or combination with another NO molecule (reaction 6) to produce two molecules of stable NO_2. Because of the very short half-life of NO_3 the overall reaction on a time-sequence basis may be viewed as the collision of three molecules. It is technically more correct, however, to view NO_2 formation as the result of two bimolecular reactions, i.e., reaction (5) followed by reaction (6). The overall effect of high temperatures is to decrease the equilibrium concentration of NO_3, and thus decrease the rate of reaction (6) and the formation of NO_2.

The rate constants applicable to reactions (5) and (6) permit significant formation of NO_2 only when the NO concentration is in the range of 100 ppm and above, and when sufficient concentrations of O_2 are present. Both NO and NO_2 are formed at combustion temperatures above 1093°C (2000°F), but the amount of NO_2 formed is usually well under 0.5 percent of the total NO_x. Exhaust gases ejected into the ambient air are thought to be diluted by a factor of 100 in a matter of seconds. During this short interval, a fraction (approximately 10 percent) of the NO does react to form NO_2. Ejection into the air promotes conversion of NO to NO_2 because the diluent air increases the availability of oxygen.[5] Once the NO has been diluted to 1230 $\mu g/m^3$ (1 ppm) or below, its direct reaction with O_2, as in reactions (5) and (6), does not contribute significantly to NO_2 formation.

It is possible to explain NO_2 formation by an alternative mechanism. NO_2 formation may also result from reaction of O_2 with the dimer of the NO molecule.

$$(NO)_2 + O_2 \rightarrow 2NO_2 \qquad (7)$$

At present, it is not possible to prove which of the proposed schemes more correctly

describes the mechanism of NO_2 formation in the atmosphere.

During daylight hours, atmospheric NO may be quantitatively converted to NO_2 by reactions that involve absorption of sunlight energy by NO_2 and subsequent interaction with certain hydrocarbons and oxygen. A detailed description of the nitrogen dioxide photolytic cycle and photochemical interaction with hydrocarbons is given in two companion documents: AP-63, *Air Quality Criteria for Photochemical Oxidants,*[6] and AP-64, *Air Quality Criteria for Hydrocarbons.*[7]

Schematically, the NO_2 photolytic cycle has the form shown in Figure 2-1. The wavelengths of light responsible for NO_2 photolysis are the ultraviolet portions of sunlight energy reaching the earth's surface, i.e., 3000 to 4000 Å. The NO_2 absorption spectrum is shown in Figure 2-2. Wavelengths longer than 4000 Å are efficiently absorbed by NO_2, but such absorption does not add sufficient energy to break bonds. Ultraviolet absorption, on the other hand, adds enough energy to break one of the N-O bonds in the NO_2 molecule. The released oxygen atom reacts rapidly with atmospheric oxygen to form ozone, which in turn, reacts rapidly with the NO formed in the breakup of the NO_2 molecule. The result in the atmosphere is the rapid cycling of the NO_2. With the possible exception of minor secondary reactions, Figure 2-1 describes the NO_2 photolytic cycle. If there were no competing reactions, the photolytic cycle would have no overall net effect; that is,

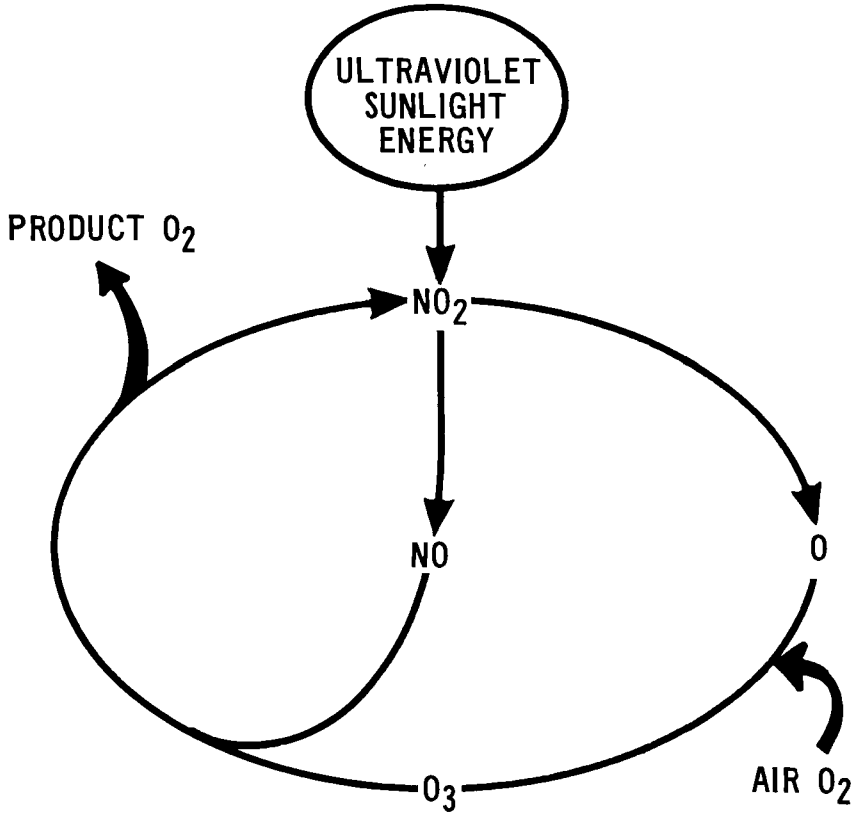


Figure 2-1. Atmospheric NO_2 photolytic cycle.

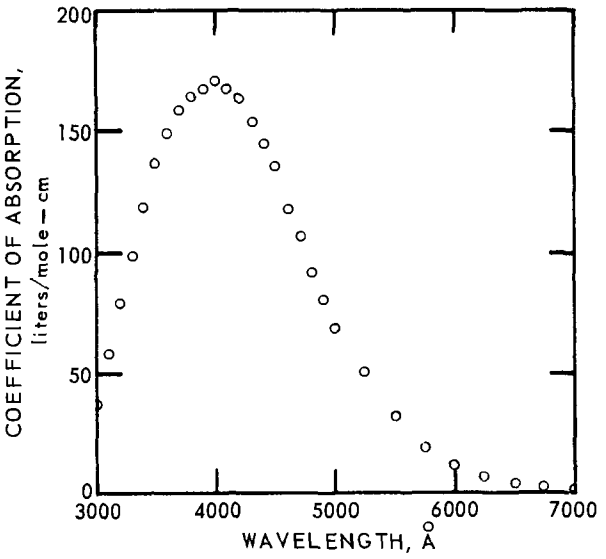


Figure 2-2. NO_2 absorption spectrum (developed from reference 3).

ambient NO and NO_2 concentrations would not change as a result of NO_2 photolysis. The same pollutant sources that emit NO_x, however, also emit hydrocarbons. Interaction of HC with the NO_2 photolytic cycle increases the speed of the NO to NO_2 conversion and thus unbalances the cycle in favor of NO_2 formation.

3. Nitrous Oxide (N_2O)

Nitrous oxide is a colorless and odorless gas used as an anesthetic and is familiarly known as "laughing gas" at concentrations in the range of 10^6 mg/m^3. N_2O is present in the atmosphere, but is chemically inert at usual atmospheric temperatures and is, therefore, not considered an air pollutant. N_2O may be a minor product of the atmospheric interactions of NO_2 in the presence of hydrocarbons and sunlight.

4. Nitrogen Sesquioxide (N_2O_3)

N_2O_3 may be involved as an intermediate in atmospheric reactions.[3] It is the anhydride of nitrous acid and produces this acid when dissolved in water. In the laboratory, it can be obtained as a blue liquid by cooling a mixture of nitric oxide and nitrogen dioxide.

5. Nitrogen Tetroxide (N_2O_4)

Nitrogen tetroxide is the colorless dimer of nitrogen dioxide. Its formation is favored by high NO_2 concentrations. At the dilute NO_2 concentrations found in ambient atmospheres, however, N_2O_4 cannot be present in more than trace amounts and therefore is probably of minor importance in atmospheric reactions.

6. Nitrogen Pentoxide (N_2O_5)

Like N_2O_3, N_2O_5 may be an intermediate in atmospheric reactions.[3] N_2O_5 is the anhydride of nitric acid and presumably reacts with atmospheric moisture to form nitric acid.

7. Other Oxides of Nitrogen

Nitrogen trioxide (NO_3) is thought to be an important intermediate in atmospheric reactions, but equilibrium and reaction kinetics factors do not favor accumulation of significant (or measurable) levels. Both NO_3 and $(NO)_2$ have been suggested as intermediates during the formation of NO_2 when NO reacts with O_2.

A summary of the physical properties of the oxides of nitrogen is presented in Table 2-1.

D. EFFECTS ON ATMOSPHERIC LIGHT TRANSMISSION

Visibility reduction is one of the most common manifestations of urban air pollution. It is caused by the scattering and absorption of light by particles or gases in the atmosphere and depends in a complicated way on the concentration and properties of the gases and particles present. This subject is discussed in AP-49, *Air Quality Criteria for Particulate Matter.*[9]

NO_2 is intensely colored and absorbs light over the entire visible spectrum, but primarily in the shorter wavelengths, violet, blue, and green. In the atmosphere it reduces the brightness and contrast of distant objects, and causes the horizon sky and white objects to

Table 2-1. PHYSICAL PROPERTIES OF NITROGEN OXIDES[8]

Oxide	Molecular weight	Melting point, °C	Boiling point, °C	Density, g/liter	Specific gravity[a]
NO	30.01	-163.6	-151.8	1.3204	–
NO_2	46.01	-11.2	21.2	–	1.4494^{20}_{20}
N_2O	44.01	-90.8	-88.5	1.977	–
N_2O_3	76.01	-102	3.5[b]	–	1.477^{2}
N_2O_4	92.02	c	c	c	c
N_2O_5	108.01	30	47 [b]	–	1.642^{18}

[a]Superscript: temperature of liquid, °C. Subscript: temperature of water, °C.

[b]Decomposes.

[c]Value uncertain, dimer of NO_2.

appear pale yellow to reddish-brown. A token amount of light is attenuated by the molecular scattering effect of NO_2.

The additional presence of particulate matter tends to mask the coloration effect of NO_2, but the two combined markedly reduce the visibility, contrast, and brightness of distant objects. Particulate matter and aerosols are present in the atmosphere as primary contaminants from urban sources such as industrial combustion and vehicular transportation, and from natural sources such as the sea, soil, and fog. They are also formed through photochemical reactions and are considered to be the major cause of the reduced visibility associated with photochemical smog.

Figure 2-3 shows the calculated percent of visible light transmitted as a function of wavelength for different atmospheric NO_2 concentrations and viewing distances in an aerosol-free atmosphere. Each curve is based on the concentration (ppm)-distance (mile) product; thus, 3 ppm-mile could be 1 ppm at 3 miles, or 3 ppm at 1 mile, or any combination of concentration and distance whose product is 3. A more normal picture of conditions actually existing in the atmosphere, with both NO_2 and aerosols present, is shown in Figure 2-4. These data are given in terms of calculated attenuation coefficients.[10] Here, the effect of visible light absorption is accompanied by transmitted light attenuation due to the light scattering by the aerosol. The photochemical system involves NO_x and HC in the formation of visibility-reducing aerosols. Light scattering associated with the presence of aerosols is the primary cause of visibility reduction in photochemical smog; absorption of light by NO_2 makes a minor contribution.

E. SUMMARY

By convention, the term NO_x represents the sum of NO and NO_2, the only significant nitrogen oxide air pollutants. They are chiefly emitted from combustion processes in which the nitrogen and oxygen in the combustion air are subjected to temperatures in excess of 1093° C (2000° F). The major oxide in combustion emissions is NO. The rapid cooling of the gases in the combustion chamber prevents dissociation back to nitrogen and oxygen. A small fraction of NO is converted to NO_2 by reaction with oxygen during the exhaust dilution process; however, the major pathway leading to formation of NO_2 from NO is the photochemical interaction between NO_x and hydrocarbons.

NO_2 causes reduction of visibility and coloration of the horizon sky in degrees dependent on its concentration, the viewing

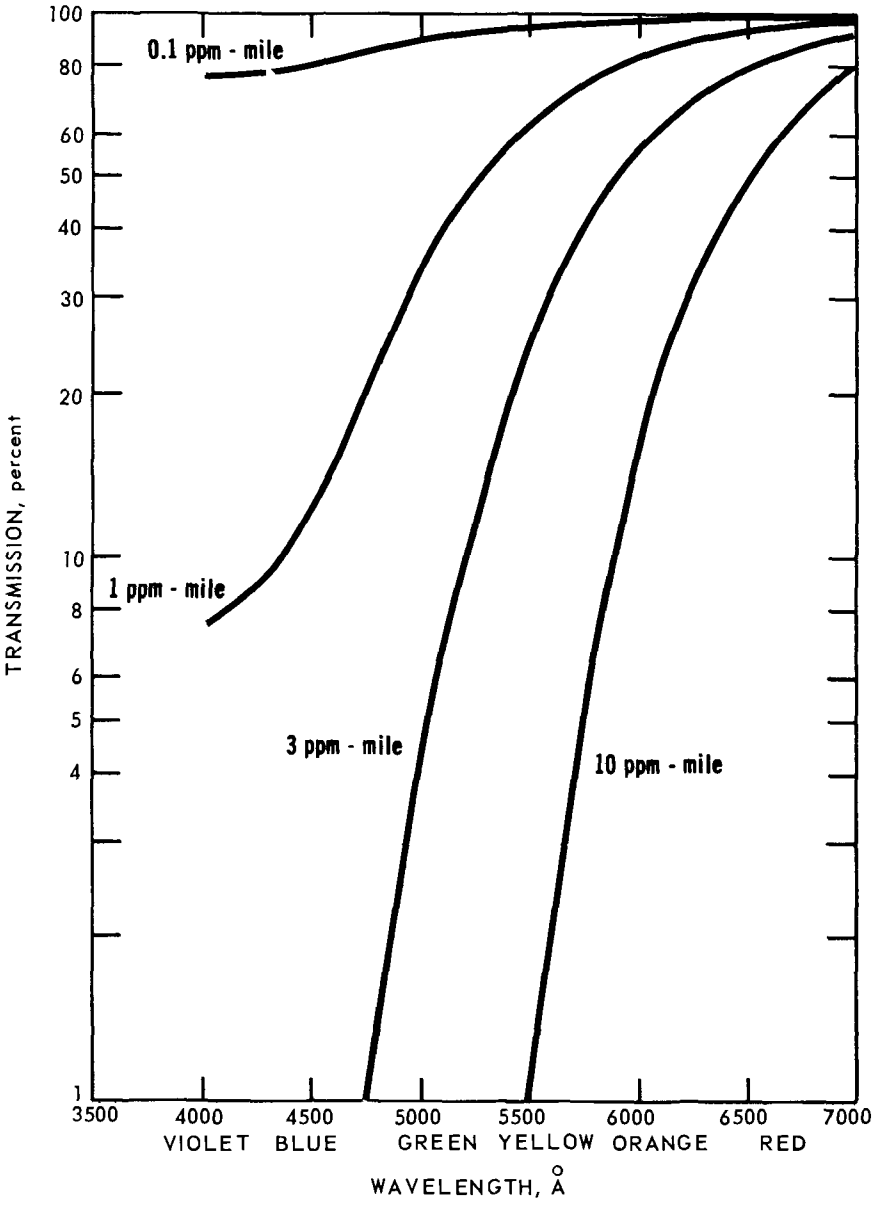


Figure 2-3. Transmittance of visible light at different NO_2 concentrations and viewing distances.[10]

distance, and the accompanying aerosol concentration. The presence of photochemical aerosol or other particulate matter suppresses the coloration effect of NO_2 and increases the visibility-reduction effect.

F. REFERENCES

1. Altshuller, A. P. Thermodynamic Consideration in the Interactions of Nitrogen Oxides and Oxy-Acids in the Atmosphere. J. Air Pollut. Contr. Assoc. *6*:97-100, September 1956.
2. Goody, R. M. and C. D. Walshaw. The Origin of Atmospheric Nitrous Oxide. Quart. J. Roy. Meteorol. Soc. *342*:496-500, October 1953.
3. Leighton, P. A. Photochemistry of Air Pollution. N.Y. and London, Academic Press, 1961. p. 184-200.
4. Trautz, M. Reaktionskinetik and Additivität der Inneren Atomwarmen bei Idealen Gasen. Z. Elektrochem. *22* (5/6):104-107, March 1916.

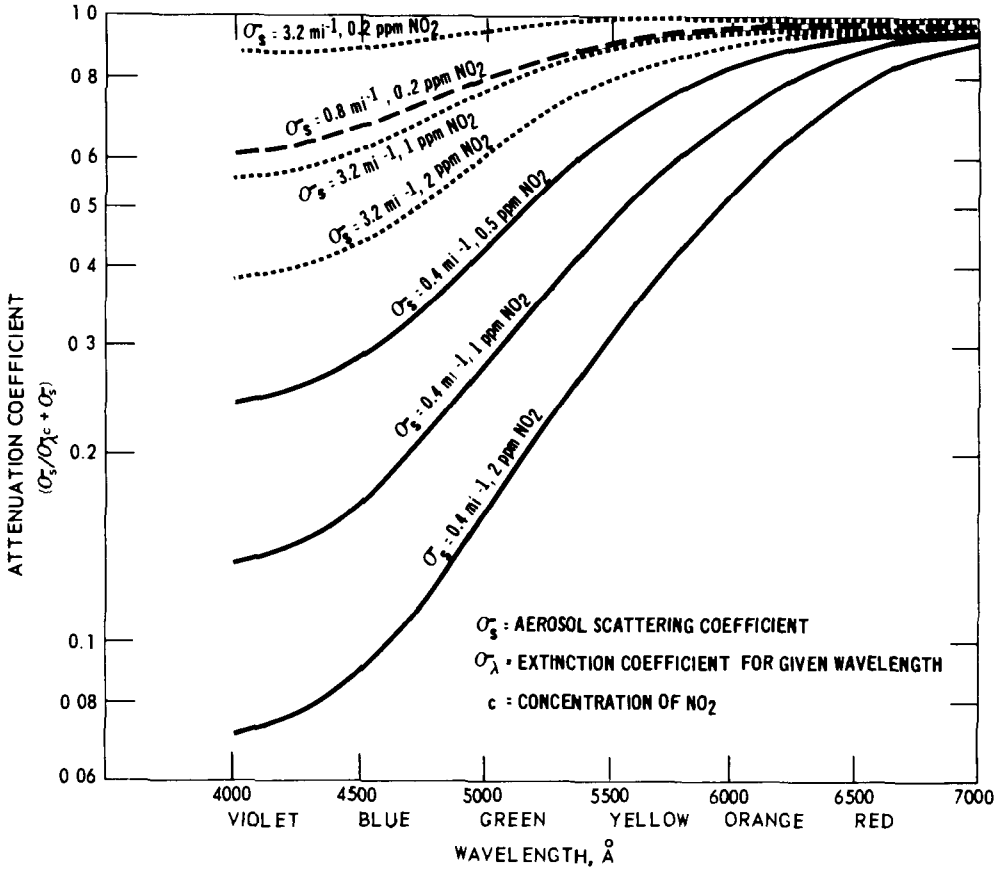


Figure 2-4. Aerosol attenuation in spectral luminance of horizon sky and distant objects at different NO_2 and aerosol concentrations.

5. Schuck, E. A. and E. R. Stephens. Oxides of Nitrogen In: Advances in Environmental Sciences, Vol. 1, Pitts, J. N., Jr. and R. L. Metcalf (Eds.). New York, Wiley-Interscience. 1969. p. 73-118.

6. Air Quality Criteria for Photochemical Oxidants. National Air Pollution Control Administration. Washington, D.C. Publication No. AP-63. March 1970.

7. Air Quality Criteria for Hydrocarbons. National Air Pollution Control Administration. Washington, D.C. Publication No. AP-64. March 1970.

8. Handbook of Chemistry and Physics, 49th Edition. Cleveland, The Chemical Rubber Co. 1968-1969. p. B-224.

9. Air Quality Criteria for Particulate Matter. National Air Pollution Control Administration. Washington, D.C. Publication No. AP-49. January 1969.

10. The Oxides of Nitrogen in Air Pollution. State of California, Department of Public Health, Bureau of Air Sanitation. Berkeley, California. January 1966. p. 53-63.

11. Carlson, R. J. and N. C. Ahlquist. Brown Haze: NO_2 or Aerosol? Atmos. Environ. *3*:653-656, November 1969.

CHAPTER 3.

SOURCES AND CONTROL OF ATMOSPHERIC NITROGEN OXIDES

A. INTRODUCTION

The major component of worldwide atmospheric nitrogen oxides (NO_X) is biologically produced nitric oxide (NO).[1] Natural sources produce about 50×10^7 tons of NO per year; man-made sources emit 5×10^7 tons per year of NO_X, which include NO and nitrogen dioxide (NO_2).

In general, NO_X concentrations in urban atmospheres are 10 to 100 times higher than those in nonurban atmospheres. Problems associated with NO_X consequently reflect the importance of technological sources over natural sources. Recent estimates indicate that total emission rates of NO_X from man-made sources in the United States have increased from approximately 1.7×10^7 tons in 1966 to 2.1×10^7 tons in 1968,[2] nearly one-half of the world production.

B. NATURAL SOURCES

On a global basis the major proportion of NO_X is NO produced by bacterial action. Relatively little is known about the global circulation of atmospheric NO_X, and only a few data on background concentration of NO_X are available from scattered nonurban areas.

Lodge and Pate[3] found average values of NO_2 ranging from 1.8 $\mu g/m^3$ (0.9 ppb) during the dry season to 7.1 $\mu g/m^3$ (3.6 ppb) during rainy periods in Panama. Individual samples were as high as 12 $\mu g/m^3$ (6 ppb). Junge[4] reported NO_2 concentrations averaging 1.8 $\mu g/m^3$ (0.9 ppb) in Florida and 2.5 $\mu g/m^3$ (1.3 ppb) in Hawaii.

At Pikes Peak in the Rocky Mountain area of Colorado, Hamilton[5] reported an average NO_2 concentration of 8.0 $\mu g/m^3$ (4.1 ppb) and an average NO level of 3.3 $\mu g/m^3$ (2.7 ppb). Ripperton et al.[6] found an average NO_2 concentration of 9.0 $\mu g/m^3$ (4.6 ppb) in the Appalachian area of North Carolina. From these data, Robinson and Robbins[1] have concluded that North American continental average levels of NO_2 are about 8 $\mu g/m^3$ (4 ppb) and those of NO are about 2 $\mu g/m^3$ (2 ppb).[2] They charted a tentative simplified global nitrogen circulation pattern. Based upon the estimated global background level and the annual emission rates, the average residence time of NO_2 is about 3 days and for NO, about 4 days. Residence times reflect the action of natural scavenging processes including photochemical reactions. Although scientists tend to emphasize the role of photochemical processes in urban areas, these processes occur during daylight in any area where the ambient air contains NO_X and reactive hydrocarbons.

C. MAN-MADE SOURCES

Fuel combustion is the major cause of technology-associated NO_X emissions. In 1968 coal, oil, natural gas, and motor-vehicle fuel combustion accounted for over 18 out of an estimated 20.6 million tons of man-made NO_X in the United States. Of the 10 million tons generated by stationary combustion sources, power plants emitted 4 million tons; industries, 4.8 million tons; and home and office heating plants, the remaining 1.2 million tons. Natural-gas-burning sources made the largest contribution of any fuel in the stationary source group. An estimated 8 million tons was emitted from transportation sources, 7 million tons of which was from motor vehicles. Industrial processes, solid waste disposal, and other miscellaneous

Table 3-1. SUMMARY OF NATIONWIDE NITROGEN OXIDES EMISSIONS, 1968[2]

Source category	Emissions	
	10^6 tons/year	percent
Transportation	8.1	39.3
Motor vehicles	7.2	34.9
Gasoline	6.6	32.0
Diesel	0.6	2.9
Aircraft[a]	N[b]	N
Railroads	0.4	1.9
Vessels	0.2	1.0
Nonhighway	0.3	1.5
Fuel combustion in stationary sources	10.0	48.5
Coal	4.0	19.4
Fuel oil	1.0	4.8
Natural gas[c]	4.8	23.3
Wood	0.2	1.0
Industrial processes	0.2	1.0
Solid waste disposal	0.6	2.9
Miscellaneous	1.7	8.3
Forest fires	1.2	5.8
Structural fires	N	N
Coal refuse	0.2	1.0
Agricultural	0.3	1.5
	20.6	100.0

[a]Emissions below 3000 feet.

[b]N=Not reported. Estimated less than 0.05 x 10^6 tons/year.

[c]Includes LPG and kerosene.

sources accounted for about 2.5 million tons of NO_X. The distribution of NO_X emissions by major source categories is indicated in Table 3-1.

Relatively small quantities of NO_X are emitted from noncombustion industrial processes, mainly the manufacturing and use of nitric acid.[7] Even though total quantities may be small, high concentrations of NO_X can be emitted from some of these chemical processes. Electroplating, engraving, welding, metal cleaning, and explosive detonation can also be responsible for industrial NO_X emissions. The same is true with regard to the manufacture and use of liquid-NO_2-based rocket propellants.

NO_X emissions from 22 cities are summarized by source category in Table 3-2.

Over 60 percent of the total NO_X emissions occur in highly populated areas; 60 percent of stationary source emissions and 45 percent of motor-vehicle emissions occur in urban areas.[2]

Table 3-2. NITROGEN OXIDE EMISSION SOURCES FOR 22 SELECTED CITIES[8]

Source category	Emissions, percent of total	
	Average	Range
Transportation	42.6	23-74
Motor vehicles	36.3	
Other	6.3	
Fuel combustion in stationary sources	50.7	10-79
Power plants	23.0	
Industrial	23.8	
Domestic	3.9	
Process losses	5.2	1-21
Refuse disposal	1.5	0.1-5.8

Estimated NO_X emissions increased 3.9 million tons from 1966 to 1968 (Table 3-3). This increase was largely due to: (1) 2.9-million-ton apparent increase from the burning of natural gas–apparent because these sources have existed but have not been included in previous emission estimates (most of the increase attributed to the inclusion of fuel used to power the transmission of fluids through pipelines); (2) 0.5-million-ton actual increase in emissions from utility power plants, mainly from increased use of coal; and (3) 0.6-million-ton increase in emissions from motor vehicles because of the increased number and use of vehicles and as a result of CO and HC exhaust controls, which have led to increased NO_X emissions.

D. PRINCIPLES OF FORMATION AND CONTROL

1. Formation

Nitric oxide is formed during fossil-fuel combustion according to the reaction:

$$N_2 + O_2 \rightleftarrows 2\,NO$$

The equilibrium level depends upon variables such as flame temperature, pressure, con-

Table 3-3. SUMMARY OF ANNUAL NATIONWIDE EMISSIONS OF NITROGEN OXIDES, 1966 THROUGH 1968[2]
(10^6 tons)

Source category	1966	1967	1968	Change from 1966 to 1968
Transportation	7.6	7.6	8.1	+0.5
Motor vehicles	6.6	6.7	7.2	+0.6
Other	1.0	0.9	0.9	-0.1
Fuel combustion	6.7	9.5	10.0	+3.3
Coal	4.0	3.8	4.0	N[a]
Fuel oil	0.9	1.0	1.0	+0.1
Natural gas	1.6	4.2	4.5	+2.9[b]
Wood	0.2	0.2	0.2	N
LPG and kerosene	--	0.3	0.3	+0.3[b]
Industrial processes	0.2	0.2	0.2	N
Solid waste disposal	0.5	0.6	0.6	+0.1
Miscellaneous	1.7	1.7	1.7	N
Man-made	0.5	0.5	0.5	N
Forest fires	1.2	1.2	1.2	N
Total	16.7	19.6	20.6	+3.9

[a]N = Negligible.

[b]Apparent change. (Emission estimates added in 1968 from sources not included in 1966).

centration of each gas, retention time in zones of different temperatures, and rates of cooling. The NO thus formed can react with more oxygen to form NO_2. Equilibrium concentrations of the NO_x mixture constituents are a function of the variables encountered during combustion and the subsequent extraction of heat from the gases. The formation of NO and achievement of equilibrium is favored by high temperatures whereas oxidation to NO_2 is an exothermic, second-order reaction favored at lower temperatures. Consequently, high combustion temperatures, rapid cooling, and instantaneous dilution of exhaust gases promote the emission of high concentrations of NO and low concentrations of NO_2.

2. Control

Techniques for controlling NO_x from large oil- and gas-fired boilers include two-stage combustion, low-excess-air firing, and furnace modifications. These approaches involve reduction of peak gas temperatures and changes in the time-temperature history of the combustion gases. Feasibility of these control methods has been commercially demonstrated; research is now under way to adapt them or others to the reduction of NO_x emissions from coal-fired boilers.

Control techniques directed at reducing the temperature in the combustion chamber of the internal combustion engine—such as exhaust-gas recirculation, higher-than-stoichiometric air-fuel ratios, water injection, spark retardation during high-speed-acceleration, and low-compression ratios – have proved to be effective in reducing NO_x emissions. It should be noted, however, that some of these methods may lead to increased HC and CO emissions while reducing NO_x.

Catalytic methods have been used to control NO_x emissions from chemical process industries, notably nitric acid manufacturing. One of the major problems is reduction of catalyst life by contamination with other chemical compounds. Another control technique employed in the chemical industries is caustic scrubbing. Still other techniques are under active investigation. Control of emissions from stationary sources is described in AP-67, *Control Techniques for Nitrogen Oxide Emissions from Stationary Sources.*[9]

Catalytic control approaches applicable to motor-vehicle-exhaust emissions are the subject of extensive research by catalyst manufacturers and the motor vehicle industry. Simultaneously controlling NO, CO, and HC is a complex, inter-related problem. One approach to NO_x emission control involves catalytic reaction of CO with NO to form elemental nitrogen and carbon dioxide (CO_2). This approach is complicated by current motor-vehicle-exhaust control methods aimed at the elimination of CO.

More elaborate discussion of the technology applicable to control of emissions from mobile sources is presented in AP-66, *Control Techniques for Carbon Monoxide, Nitrogen Oxide, and Hydrocarbon Emissions from Mobile Sources.*[10]

E. SUMMARY

The quantity of NO_x generated by natural sources greatly exceeds the man-made quantity on a worldwide basis. Man's contribution is a cause for concern, however, because the emissions are concentrated in urban areas. Fuel combustion, for transportation, power, and heating, is the major source of man-made NO_x air pollution. Chemical processing is responsible for high, but localized emissions.

Control of nitrogen oxide emissions has been directed at both combustion sources and chemical processes. For stationary combustion sources, the control principle has been based on reducing either the flame temperature or the availability of oxygen, both of which prevent NO formation. Similar principles of control are applicable to motor vehicles. Catalytic principles, which have been applied to reduce NO_x from chemical proc-

esses, may also be applicable to the control of NO_x in motor-vehicle exhaust.

F. REFERENCES

1. Robinson, E. and R. C. Robbins. Gaseous Atmospheric Pollutants from Urban and Natural Sources. J. Air Pollut. Contr. Ass. *20*: 303-306, May 1970.
2. Nationwide Inventory of Air Pollutant Emissions, 1968. National Air Pollution Control Administration. Raleigh, N.C. Publication No. AP-73, August 1970. p. 14-16.
3. Lodge, James P., Jr. and J. B. Pate. Atmospheric Gases and Particulate in Panama. Science *153*: 408-410, July 22, 1966.
4. Junge, C. E. Recent Investigations in Air Chemistry. Tellus *8*: 127-139, May 1956.
5. Hamilton, H. L. et al. An Atmospheric Physics and Chemistry Study on Pikes Peak in Support of Pulmonary Edema Research. Research Triangle Institute for Army Research Office. Research Triangle Park, N.C. Contract No. DA-HC19-67-C-0029. 1968.
6. Ripperton, L. A., L. Kornreich, and J. J. B. Worth. Nitrogen Dioxide and Nitric Oxide in Non-Urban Air. J. Air Pollut. Contr. Ass. *20*: 589-592, September 1970.
7. Atmospheric Emissions from Nitric Acid Manufacturing Processes. Manufacturing Chemists' Association and U.S. Department of Health, Education, and Welfare. Cincinnati. Public Health Service Publication No. 999-AP-27. 1966.
8. Mason, D. V., G. Ozolins, and C. B. Morita. Sources and Air Pollutant Emission Patterns in Major Metropolitan Areas. (62nd Annual Meeting, Air Pollution Control Association. June 1969.) Paper 69-101.
9. Control Techniques for Nitrogen Oxides from Stationary Sources. National Air Pollution Control Administration. Washington, D.C. Publication No. AP-67. March 1970.
10. Control Techniques for Carbon Monoxide, Nitrogen Oxide, and Hydrocarbon Emissions from Mobile Sources. National Air Pollution Control Administration. Washington, D.C. March 1970.

CHAPTER 4.
CHEMICAL INTERACTIONS OF NITROGEN OXIDES IN THE ATMOSPHERE

A. INTRODUCTION

This chapter examines the chemical relationship of nitrogen oxides (NO_x) to photochemical oxidants (OX) and to other products of atmospheric reactions, a relationship that encompasses several interrelated factors.[1]

The most important aspect of this relationship with respect to air pollution is associated with the generation of oxidants. The generation of oxidants is a complex function of the interaction of certain hydrocarbons and hydrocarbon oxidation products with the nitrogen dioxide photolytic cycle (See chapter 2). Nitrogen dioxide (NO_2), which initiates the process, is an efficient absorber of both the ultraviolet and visible portions of solar radiation (chapter 2, Figure 2-2). The ultraviolet portion of the radiation has sufficient energy to disrupt bonds in NO_2 and form two new species, nitric oxide (NO) and atomic oxygen (O). The O atoms then react with air oxygen (O_2) to produce ozone (O_3), the principal oxidant in ambient air. The O atoms and to a lesser extent the O_3 react with atmospheric hydrocarbons, especially the olefins and certain aromatics to form other pollutant products.

The NO formed in the initial NO_2 photolysis acts as a natural ozone regulator and reforms oxygen and nitrogen dioxide in the reaction:

$$NO + O_3 \rightarrow NO_2 + O_2$$

Since this reaction occurs almost instantaneously, appreciable concentrations of O_3 do not appear until NO has virtually disappeared from the ambient atmosphere. Hydrocarbon interaction with the NO_2 photolytic cycle leads to the disappearance of NO with subsequent formation of NO_2, and to the accumulation of hydrocarbon oxidation products and residual ozone (chapter 2. Figure 2-1).

The rates of formation and the steady-state concentrations of oxidants are a function of light intensity, the concentrations of HC and NO_x, the HC-NO_x ratio, and temperature. The nature of the HC molecule is an additional factor since individual hydrocarbons differ in their rate of interaction with the NO_2 photolytic cycle. Some hydrocarbons, such as methane and benzene, do not interact to form photochemical oxidants, at least not in synthetic systems. Others, certain olefins and substituted aromatics, for example, interact readily.[2] The onset of the reactions is regulated by NO_2 concentrations. Minimal NO_2 concentrations delay, whereas higher concentrations expedite the cycle.

This description of atmospheric reactions has been greatly simplified and mentions only the major known reactions that account for the formation of photochemical oxidants. There are other reactions known to occur in the photochemical system; however, their roles are considered less important. Certainly, their involvement with NO_x is less clearly understood, and discussion of them is omitted in this document. They are covered, in part, in AP-63, *Air Quality Criteria for Photochemical Oxidants.*[1]

Subsequent, more detailed research may yet show that some of what are now considered minor reactions have essential roles in the regulation of atmospheric phenomena. These so-called minor reactions may also become very important as the nature of

ambient atmospheres change in response to HC - and NO_X - control programs. This chapter does not discuss products of the photochemical reaction system, other than oxidants, in detail.

Earlier publications[1,2] have dealt with photochemical oxidant relationships to eye irritants and visibility-reducing aerosols, and they suggest that the magnitude of effects varies directly with the oxidant formed, at least in the controlled, experimental system. Such an association, however, although typical of present photochemical smog systems, must be recognized as a generalization to which there are specific exceptions. Such exceptions sustain the need for continuous atmospheric studies in order to reassess the relationships between pollutant and effects, particularly as they may be affected by precursor-control strategies.

The major known variables that contribute to oxidant formation have been investigated extensively under controlled laboratory conditions.[3-7] On the basis of such experimentation, it is possible to predict the effect of varied inputs on oxidant concentration and rates of formation. Extrapolation of laboratory results to ambient atmospheres, however, involves certain problems: (1) The mixing volume in laboratory simulations is a constant and favors a direct relationship between input concentrations of the precursors and the maximum concentration of resultant oxidant, which occurs several hours later. In contrast, the concentrations of both precursors and oxidants in ambient atmospheres are dominated by dispersion factors, which in turn alter the input/output relationship. (2) The definition of the variables is much more precise in laboratory simulations than in routine atmospheric monitoring. In the laboratory, for example, the identity and concentration of each hydrocarbon in the system can be determined by gas chromatographic techniques. Routine atmospheric monitoring techniques have been largely restricted to measurements of total HC concentrations and, only recently, to measurements of total HC minus non-reactive methane. In addition, laboratory simulations have been largely carried out at concentrations higher than those observed in ambient atmospheres. (3) All laboratory simulations are, to some degree, affected by the experimental system. Thus, interaction of the gases with contamination on the walls of the testing chamber may affect the observed oxidant results.

In defining the OX-NO_X relationship, the approach was the same as the one used to define the relationship of ambient HC concentrations to maximum daily oxidant values in an earlier publication.[8] The same patterns of chemical reactions and time-concentration relationships observed in the laboratory were assumed to apply in the atmospheric system and to affect oxidant levels in the same way. The approach, which can be termed an *observational hypothesis,* is based on several assumptions that are covered, in limited detail, in the companion document, AP-64, *Air Quality Criteria for Hydrocarbons.*[2]

The basic assumption in the observational approach is that early morning HC and NO_X levels are indicators of the oxidant levels that will occur later in the day. Specifically, the 6- to 9-a.m. HC and NO_X levels are compared to the daily 1-hour maximum oxidant levels that normally occur between 10 a.m. and 2 p.m. Because the time-concentration relationships are influenced by atmospheric dispersion factors, a specific ambient combination of HC and NO_X will not always result in a specific maximum oxidant value. The hypothesis provides rather for a wide range of maximum daily oxidant values to be associated with any given early-morning HC-NO_X combination. The precise oxidant concentration within that range will be regulated by the atmospheric stability factors for the day. For any concentration of precursors the hypothesis predicts maximum oxidant values near zero, if substantial atmospheric instability follows the 6- to 9-a.m. period. Such instability causes the precursors as well as the products to disperse before they can interact and yield any appre-

ciable amount of oxidants. Conversely, the hypothesis allows for elevated oxidant values when the dispersion factors are more restrictive or when the atmosphere is stable following the 6- to 9-a.m. period. In those cases, precursors are trapped long enough for formation and accumulation of oxidants to occur. Between maximum and minimum atmospheric stability is a large range of dispersion factor combinations, which lead to *intermediate* maximum daily oxidant concentrations under the terms of the model.

Information from laboratory studies, however, suggests that the maximum attainable oxidant concentration will be limited by the available precursors regardless of how stable the atmosphere becomes. It is this upper-limit oxidant concentration that the *observational* approach attempts to define.

B. OBSERVATIONAL MODEL FOR HC-NO_X - OX RELATIONSHIPS

The following sections examine the functional relationships observed between HC and OX, NO_X and OX, and the combined relationship among HC, NO_X, and OX. Since OX concentrations are also a direct function of light intensity, the discussion concentrates on those portions of the year in which sunlight intensities are at or near maximum, thus emphasizing the days with maximum potential for OX formation, that is, the months of May through October.

1. Early Morning HC Relationship to Maximum Daily Oxidant

The generation of a model based on the observational approach is limited by the number of days for which pollutant measurements are available. Oxidant values for any given combination of HC and NO_X precursors can range from zero to the upper limit. Since the upper limit is attained on only about 1 percent of the days in a year, many days' measurements are needed in order to provide reasonable assurance that an upper-limit point has actually been observed. The small number of data points in the vicinity of the upper-limit line shown in Figure 4-1 for the relationship between total hydrocarbons and the maximum daily OX observed in several United States cities, illustrates this point. (In this figure and throughout the chapter, hydrocarbons are measured in parts per million carbon, ppm C).

The problem with the data in Figure 4-1 is that total HC are reported. This includes a large fraction of non-reactive methane, which tends to mask the effect of the smaller fraction of HC known to be important to OX formation. A recent publication [2] presents a limited data base that relates the more important, nonmethane HC values to maximum daily oxidant concentrations. Figure 4-2 shows the 125 days of data. Again, one becomes acutely aware of the limited number of points that define the upper-limit oxidant line. Since they are limited in number, these points presumably define the minimum upper-limit-oxidant line, and more extensive observations would undoubtedly include still higher oxidant concentrations. It seems reasonable to conclude, then, that the upper-limit curve in Figure 4-2, is the most valid relationship at this time.

The curve can be used to predict the maximum 1-hour-average oxidant concentration from a measured 6- to 9-a.m. - average HC concentration. Likewise, the minimal level of HC that will produce a given OX level can also be predicted. For example, a reference concentration of 200 $\mu g/m^3$ (0.1 ppm) OX is associated with 200 $\mu g/m^3$ (0.3 ppm C) 6- to 9-a.m. - average nonmethane HC.

Oxidant values below 140 $\mu g/m^3$ (0.07 ppm) are omitted in Figures 4-1 and 4-2 because several factors indicate these values may be subject to measurement errors; however, they have no bearing on the upper-limit values. The HC values in Figure 4-2 are not reported below 200 $\mu g/m^3$ (0.3 ppm) even though they may have a bearing on the upper-limit oxidant level, because they are subject to even greater measurement error.[2]

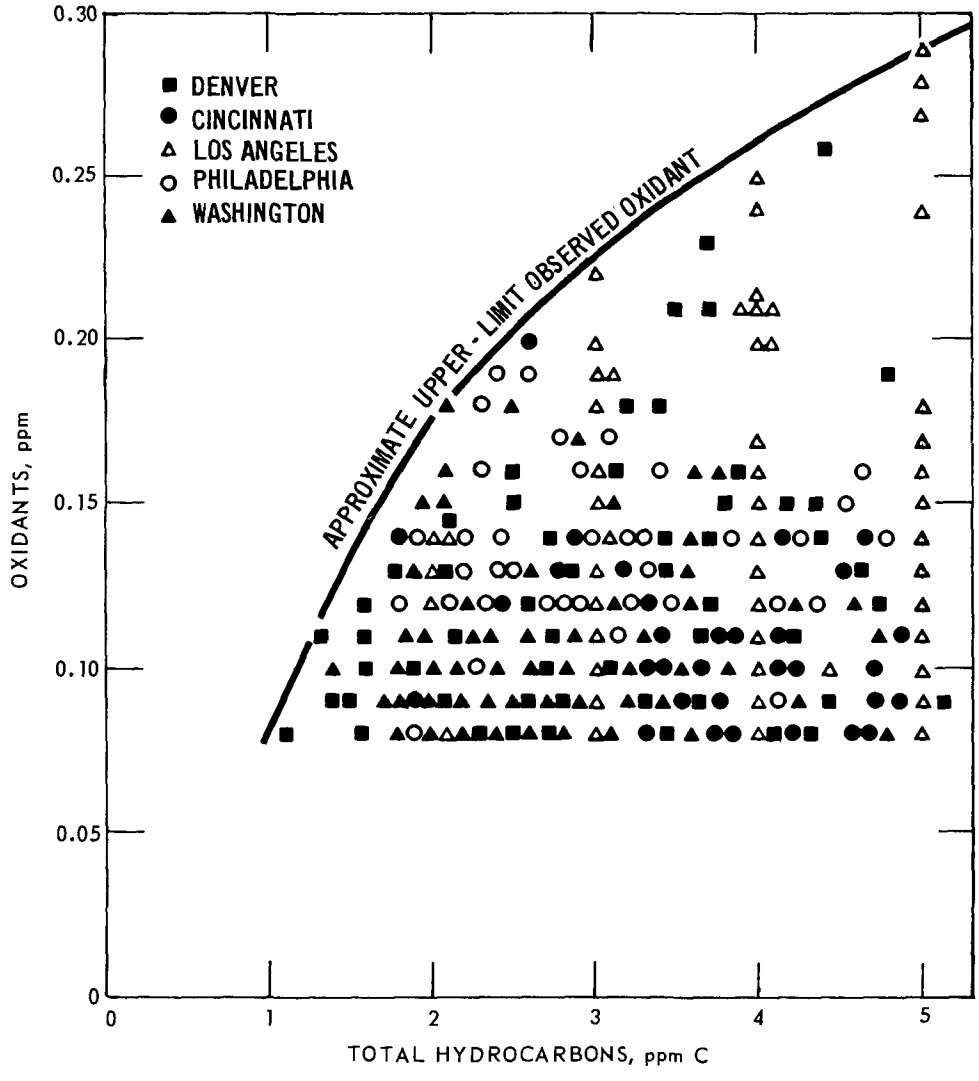


Figure 4-1. Maximum daily 1-hour-average oxidant concentrations as a function of 6-to 9-a.m. averages of total hydrocarbon concentrations at CAMP stations, June through September, 1966 through 1968 and in Los Angeles, May through October 1967.[2,8]

2. Early Morning NO_X Relationship to Maximum Daily Oxidant

An observational approach similar to the one described for the HC-OX relationship was applied to NO_X levels[8] with one important difference in regard to definition of the specific precursors. It was not possible to define the reactive hydrocarbon mixture contributing to OX concentrations[2] precisely, because present air-monitoring systems provide only gross measurements of ambient hydrocarbons (i.e., concentrations of total HC or total HC less methane). In contrast, it is believed that only two species of NO_X (NO and NO_2) are necessary to define the NO_X-OX

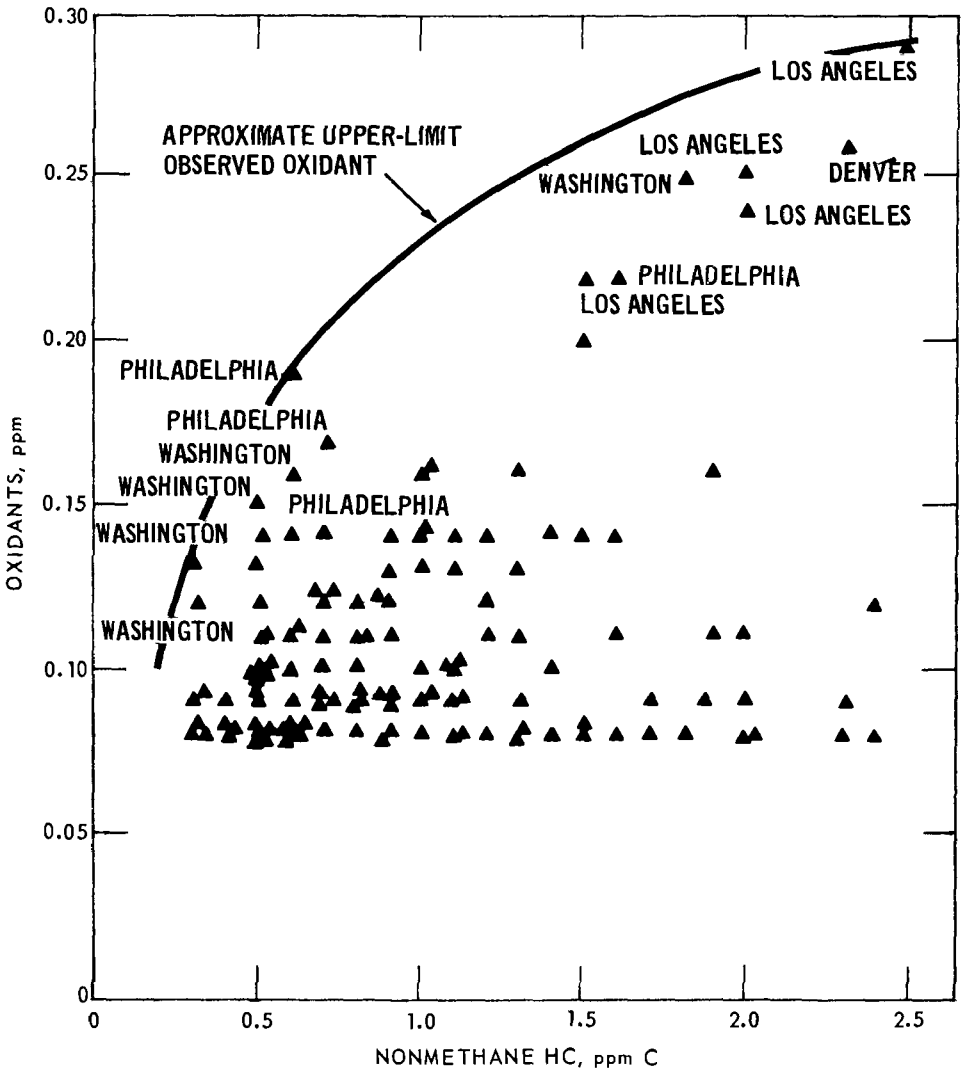


Figure 4-2. Maximum daily 1-hour-average oxidants as a function of 6-to 9-a.m. averages of nonmethane hydrocarbons at CAMP stations, June through September, 1966 through 1968, Los Angeles, May through October 1967. (Based on references 2 and 8).

relationship, and both of these are measured routinely. The initial ratio of NO_2 to NO will influence the rate of formation of oxidant. In terms of the oxidant maximum, however, the primary consideration is the total NO_X concentration, since NO is rapidly converted to NO_2 during the photolytic reactions.

Measurement problems do exist, particularly in regard to low NO_X concentrations. Unfortunately, the NO_X measuring instruments, like the HC and oxidant instruments, are primarily designed to measure values in the 0.1- to 1-ppm range, whereas much of the area of interest is below 0.1 ppm.

The daily-maximum 1-hour-average oxidant levels and the 6- to 9-a.m. - average NO_X concentrations[9] were recorded from June through September, 1965 through 1968, at three Continuous Air Monitoring Program (CAMP) stations in the downtown areas of

Washington, D.C., Philadelphia, and Denver. These are shown in Figures 4-3, 4-4, and 4-5. Careful evaluation of the data base has shown that about 1 percent of the oxidant values fall near the upper limit.[8]

If we accept all data points as equally valid, a reference value of 200 $\mu g/m^3$ (0.10 ppm) OX would be associated with as little as 20 $\mu g/m^3$ (0.01 ppm) NO_X. Because of the analytical uncertainties in the low concentration region a more rational approach is to locate an NO_X level below which OX can be expected to exceed the reference concentration, i.e., 200 $\mu g/m^3$ (0.10 ppm) oxidant, on 1 percent of the days. Figures 4-3, 4-4, and 4-5 show only seven occasions when the oxidant equalled or exceeded 0.10 ppm and the NO_X was less than 0.04 ppm. These events occurred twice per smog season in Denver, once in four smog seasons in Philadelphia, and not at all during three smog seasons in Washington, D. C. This frequency represents about 1 percent of the combined data base.

Although the curves that define the maximum oxidant-forming potential in Figures 4-1 through 4-5 were drawn to include all the data points, they were not based on a statistical approach. The data were too limited in number to justify a statistical analysis; however, strict adherence to the limits set by the few applicable data points would associate an even lower value of NO_X with 200 $\mu g/m^3$ (0.10 ppm) oxidant. Current analytical uncertainties in

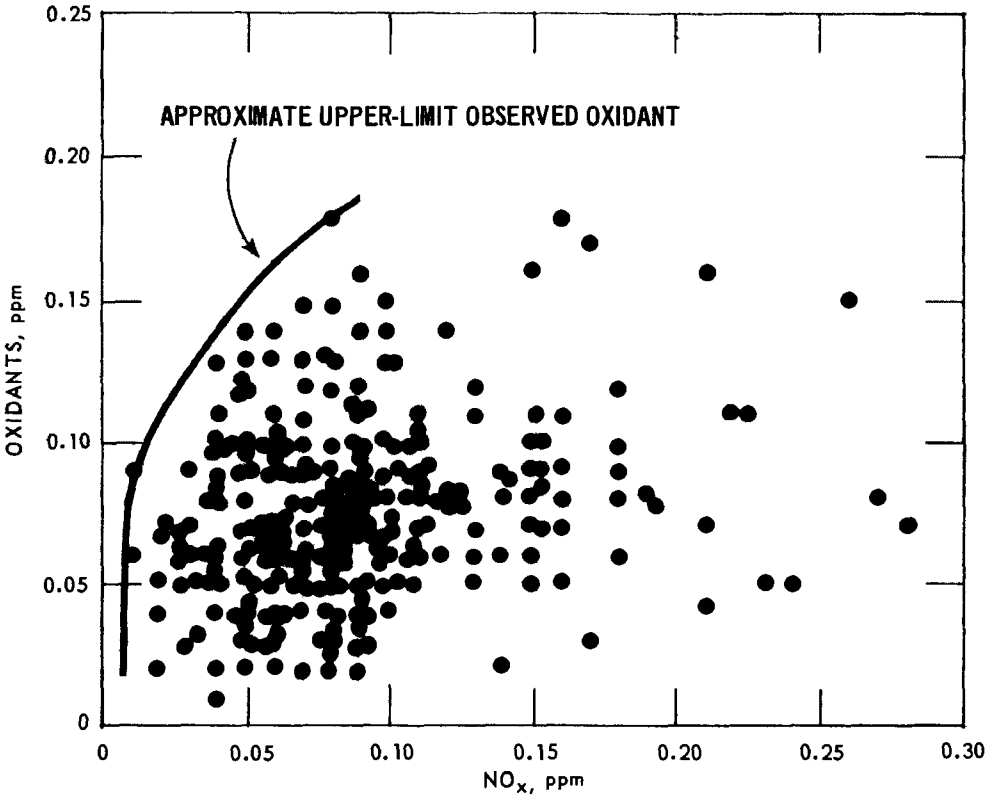


Figure 4-3. Maximum daily 1-hour-average oxidant concentrations as a function of 6- to 9-a.m. averages of total nitrogen oxides in Washington, D.C., June through September, 1966 through 1968.

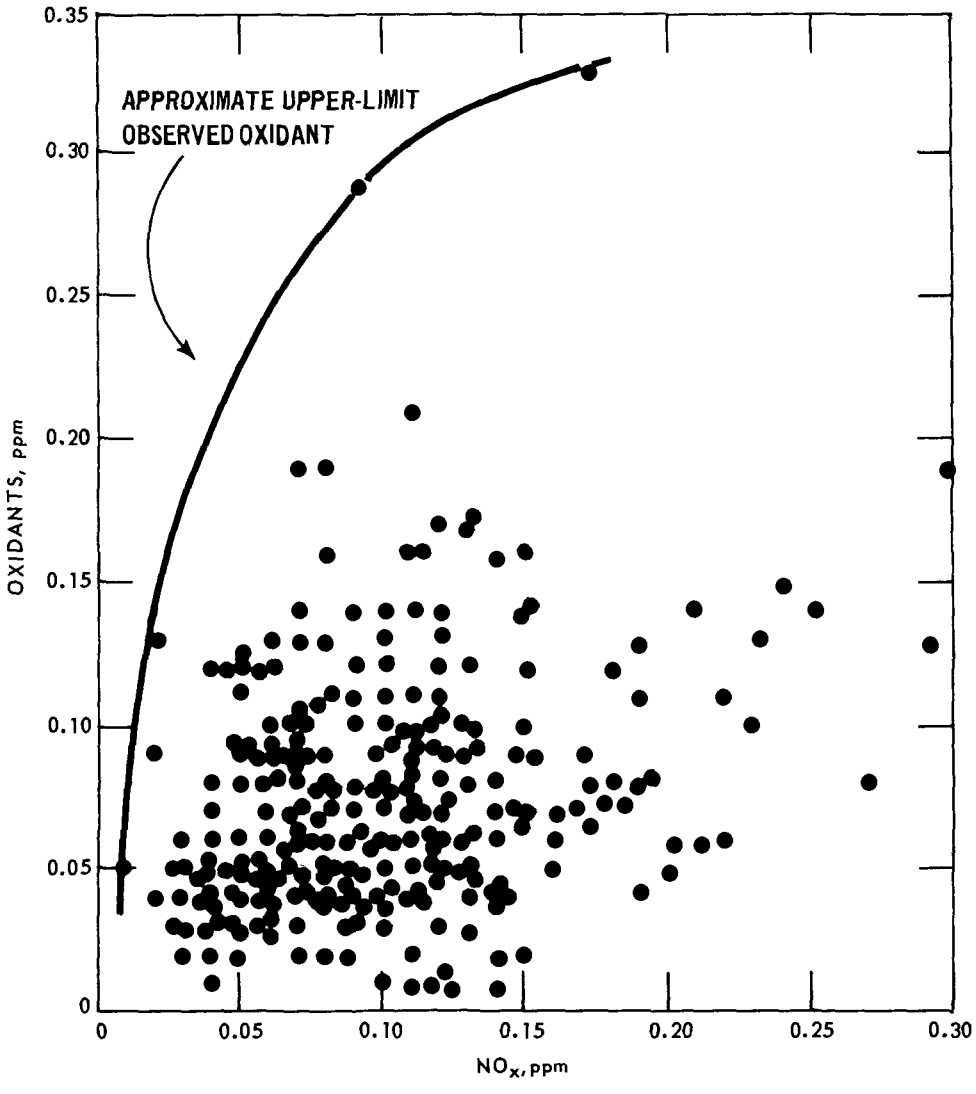


Figure 4-4. Maximum daily 1-hour-average oxidant concentrations as a function of 6-to 9-a.m. average total nitrogen oxides in Philadelphia, June through September, 1965 through 1968.

the low-range measurements make such a conclusion unwise. From the relationships in Figures 4-3, 4-4, and 4-5, it appears that the 6- to 9-a.m.-average NO_X levels must be kept below 80 $\mu g/m^3$ (0.04 ppm) in order to prevent the maximum daily 1-hour OX concentration from reaching 200 $\mu g/m^3$ (0.1 ppm) or more.

The reference concentration of 200 $\mu g/m^3$ OX used here was selected on the basis of convenience and does not represent the lowest health-related value (130 $\mu g/m^3$ OX) published in document AP-63, *Air Quality Criteria for Photochemical Oxidants.*

Data from three Los Angeles locations frequently affected by elevated oxidant levels are shown in Figure 4-6 for a calculated[2] nonmethane HC level of 1 mg/m^3 (1.5 ppm C).[2] At two of these stations a 6- to 9-a.m. value of 80 $\mu g/m^3$ (0.04 ppm) NO_X and at a third, 90 $\mu g/m^3$ (0.05 ppm) NO_X, is associated with a daily-maximum 1-hour-average oxidant concentration of 200 $\mu g/m^3$ (0.10 ppm). These Los Angeles results are similar to those obtained in Washington, D. C., Philadelphia, and Denver.

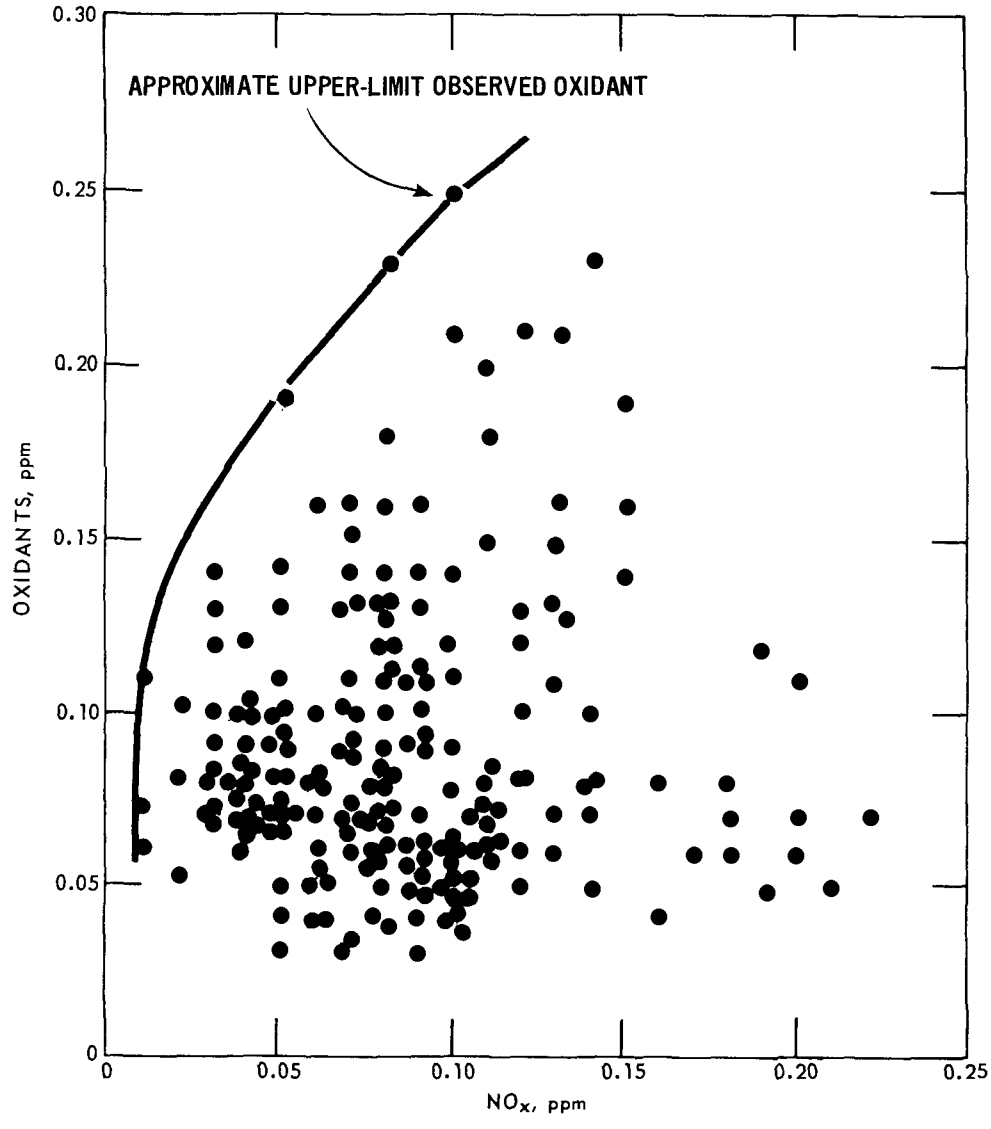


Figure 4-5. Maximum daily 1-hour-average oxidant concentrations as a function of 6-to9-a.m. averages of total nitrogen oxides in Denver, June through September, 1965 through 1968.

For these reasons the NO_X-OX relationship cited is considered the most reasonable that can be made at this time. It will be noted that the relationship states only that the 6- to 9-a.m. - average NO_X must be *below* 80 $\mu g/m^3$ (0.04 ppm) to prevent oxidant levels greater than 200 $\mu g/m^3$ (0.1 ppm) from occurring later in the day more frequently than 1 percent of the time. It does not attempt to specify the *exact* NO_X concentration. Whether this or an even more stringent limitation of NO_X is required, can be assessed only after further observations have been made of ambient atmospheres and the manner in which ambient HC-control affects ambient oxidant levels. Laboratory results indicate that HC control,

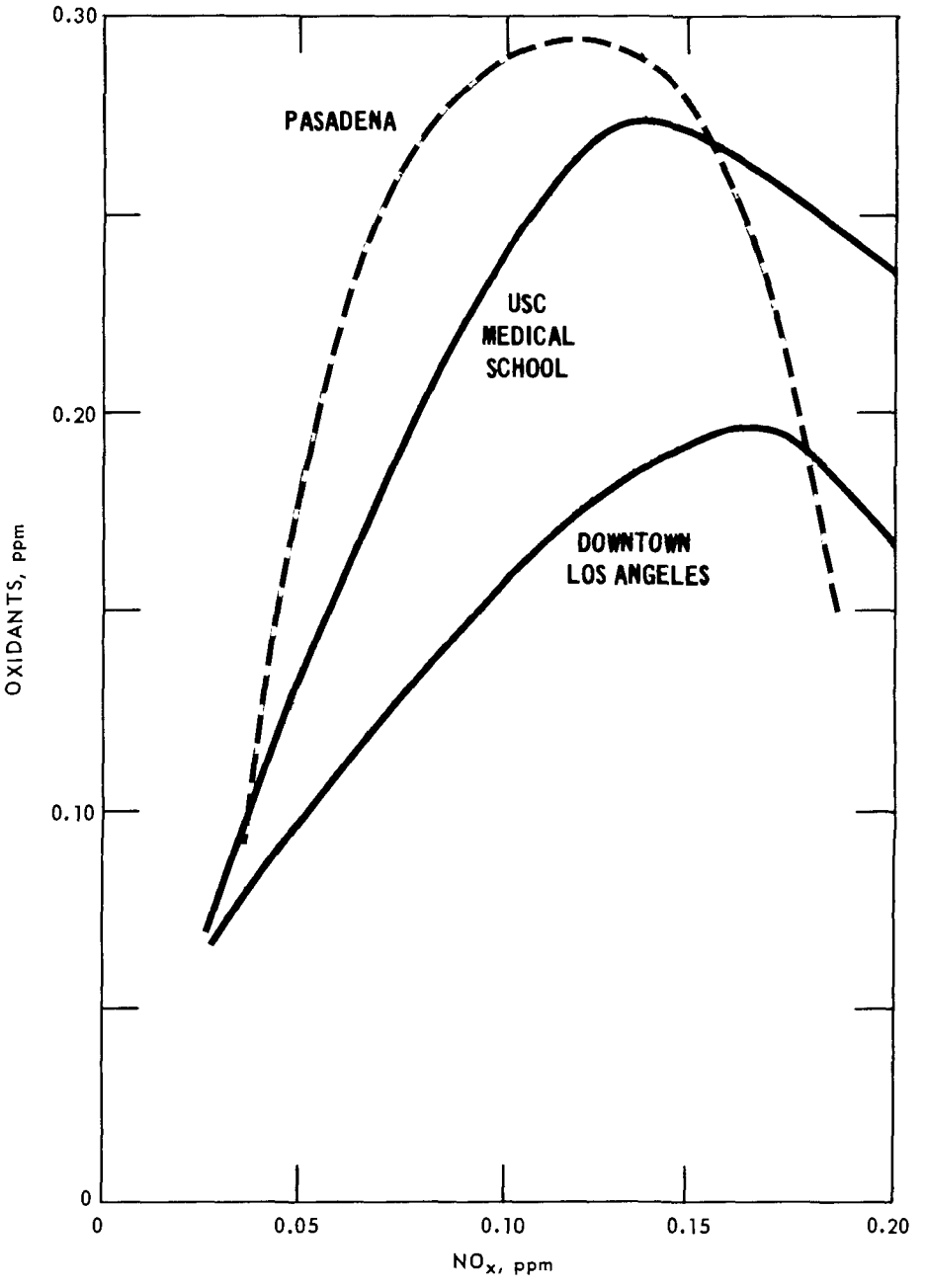


Figure 4-6. Upper limit of maximum daily 1-hour average oxidant concentrations, calculated nonmethane hydrocarbon concentration of 1.5 ppm C, as a function of average total nitrogen oxides from 6-to 9-a.m. at three Los Angeles stations, May through October 1967.

even in the absence of NO_X control, will definitely lead to reductions in ambient oxidant levels.

3. HC-NO_X - Oxidant Relationships

The previous sections dealt with the relationships of each individual precursor to maximum oxidant levels, without considering combined effects. An initial attempt to explore the possible combined effects is shown in Figure 4-7 for Pasadena, California. In this figure, upper-limit envelopes of HC levels as a function of oxidant and NO_X are superimposed on a graph showing the relationships of maximum oxidant levels to NO_X concentrations.

The interdependent relationship among HC, NO_X, and oxidant concentrations in Figure 4-7 is in complete agreement with

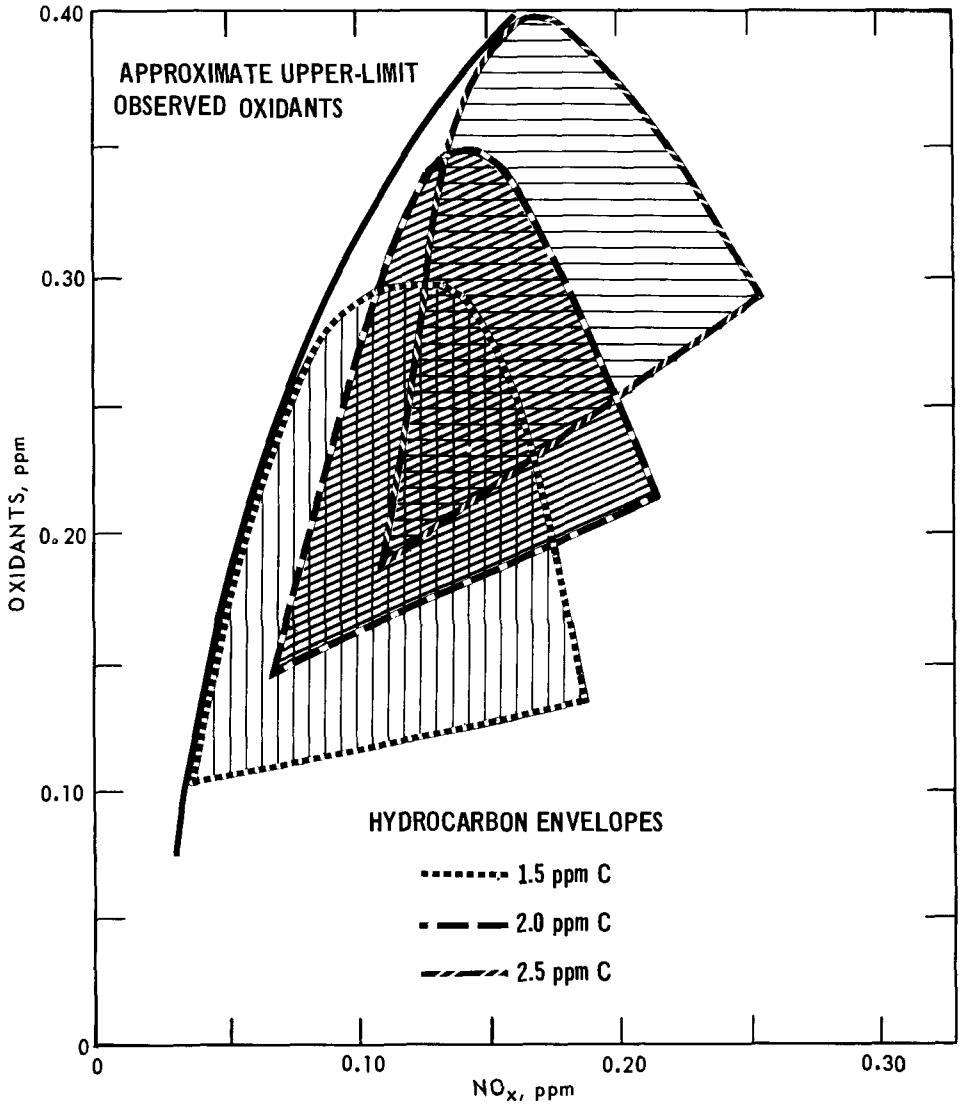


Figure 4-7. Hydrocarbon-oxidant envelopes superimposed on maximum daily 1-hour-average oxidant concentrations as a function of 6- to 9-a.m. average of total nitrogen oxides in Pasadena, California, May through October 1967.

laboratory simulations.[3-6] Ambient data available are too limited to clearly define these combinational effects. Furthermore, the HC values in Figure 4-7 represent calculated,[2] rather than measured, nonmethane hydrocarbon concentrations. As in the previously discussed HC-oxidant studies, data were taken at CAMP stations on the days when nonmethane hydrocarbons were being measured. A base from these, involving 125 days of validated data, was used to construct the daily-maximum-oxidant isopleth lines as a function of the early-morning HC and NO_X values, which are shown in Figure 4-8. The dotted lines in Figure 4-8 are the levels of HC and NO_X associated with the reference 1-hour maximum oxidant concentration of 200 $\mu g/m^3$ (0.1 ppm) in the previous HC-oxidant

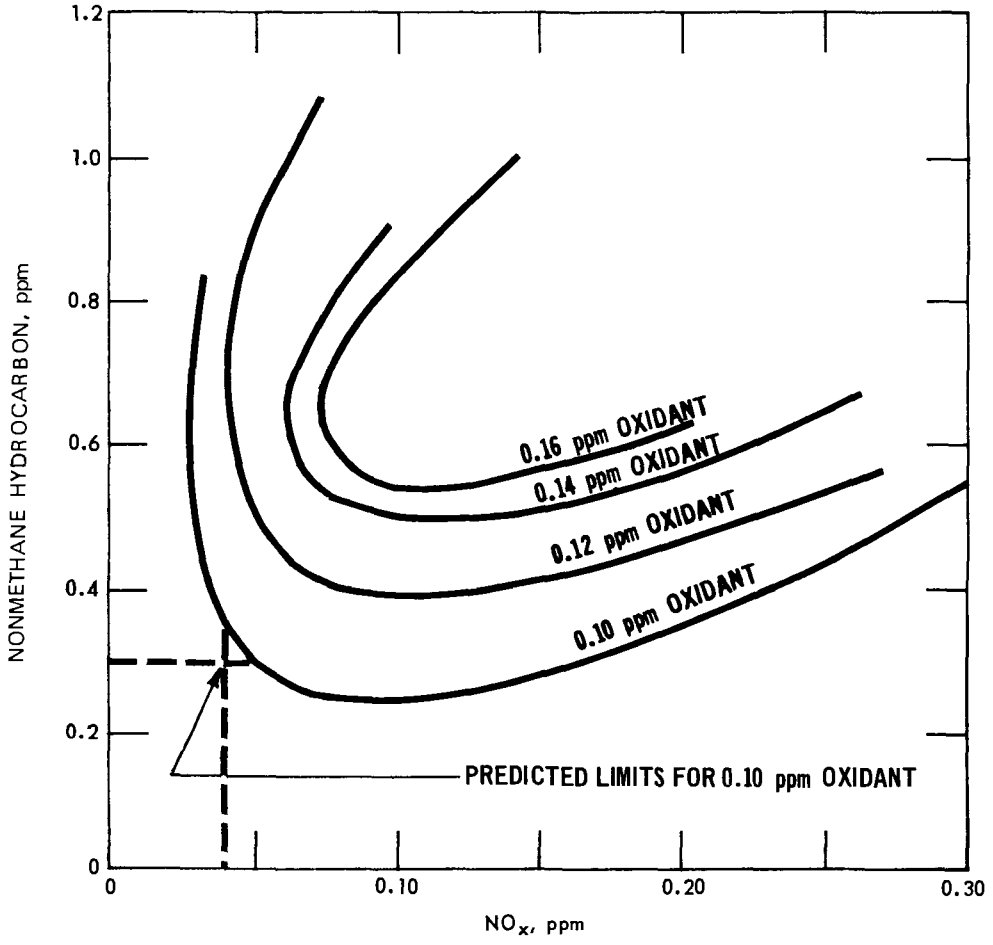


Figure 4-8. Approximate isopleths for selected upper-limit maximum daily 1-hour-average oxidant concentrations, as a function the 6-to 9-a.m. averages of nonmethane hydrocarbons and total nitrogen oxides in Philadelphia, Washington, D.C., and Denver, June through August, 1966 through 1968.

and NO_X-oxidant analyses (see Figures 4-2 through 4-5).

Since concentrations of HC and NO_X are apparently interdependent, it is not appropriate to make a separate analysis of oxidant for each, individually. This fact is illustrated in the contrast between Figures 4-3, 4-4, and 4-5, which treat the NO_X-oxidant relationship as if it were independent of HC level, and Figure 4-7, which shows the interdependence. Any relationship of one precursor to oxidant determines, to a degree, the relationship of the other precursor to oxidant.

The data in Figures 4-3, 4-4, and 4-5 are summarized in Table 4-1. Although the precursor effects are not completely separable, it is clear that increases in both precursors lead to increases in the maximum daily oxidant level.

Further observation of the data base shows that 80 $\mu g/m^3$ (0.04 ppm) NO_X occurs simultaneously with calculated nonmethane HC concentrations ranging from 200 to 930 $\mu g/m^3$ (0.3 to 1.4 ppm C). Similarly, observation of the 200 $\mu g/m^3$ (0.3 ppm C) nonmethane HC level shows NO_X in the range of 80 to 320 $\mu g/m^3$ (0.04 to 0.16 ppm).

It is also clear from the hydrocarbon envelopes shown in Figure 4-7 and the HC values in Table 4-1 that each HC level is associated with a wide *range* of NO_X values. For example, at a 1.0 mg/m^3 (1.5 ppm) HC level in Figure 4-7, the associated NO_X concentrations range from 80 to 340 $\mu g/m^3$ (0.04 to 0.18 ppm). Both of the atmospheric relationships (Figures 4-7 and 4-8) suggest a complex dependence of oxidant level on the ratio of HC to NO_X in addition to a dependence on the absolute concentrations of the two. The two figures also suggest the existence of an optimum HC-to-NO_X ratio with regard to maximum attainable oxidant.

The results of a series of laboratory photooxidation experiments,[5] designed to determine precursor effects on peak oxidant values, are shown in Figure 4-9. The oxidant values have been converted to ozone by correcting for the effect of NO_2 on the oxidant instrument.[1] Comparison of observed ambient oxidant isopleths in Figure 4-8 with the experimentally determined isopleths in Figure 4-9 indicates that the two observations are, qualitatively, quite similar.

This similarity between laboratory results and the data from ambient atmospheres (Figure 4-7) lends validity to the measurements obtained with current air monitoring instruments. Furthermore, it suggests that the measured total hydrocarbon and the calculated nonmethane hydrocarbons are indeed proportional to the reactive HC fraction taking part in atmospheric oxidant formation.

Table 4-1. CHANGES IN MAXIMUM DAILY 1-HOUR AVERAGE OXIDANT CONCENTRATION AS A FUNCTION OF 6- to 9 -a.m. AVERAGE HC AND NO_X CONCENTRATIONS[9,a]

No. of days	6- to 9- a.m. precursor concentrations				% of days oxidant 1-hour daily maximum[b] equals or exceeds:		
	Total HC, ppm C		NO_X, ppm				
	Median	Range	Median	Range	0.06 ppm	0.10 ppm	0.20 ppm
84	1.7	1.5-1.9	0.05	0.01-0.15	42	12	1
167	2.2	2.0-2.4	0.08	0.02-0.16	64	17	2
138	2.7	2.5-2.9	0.10	0.02-0.17	67	19	3
83	3.2	3.0-3.4	0.11	0.05-0.24	68	35	10

[a]Combined data from Washington D.C., June through September 1965, 1966, 1968; Philadelphia, June through September 1965 through 1968; and Denver, June through September 1965 through 1968.

[b]Oxidant data were corrected for effect of NO_2.[1]

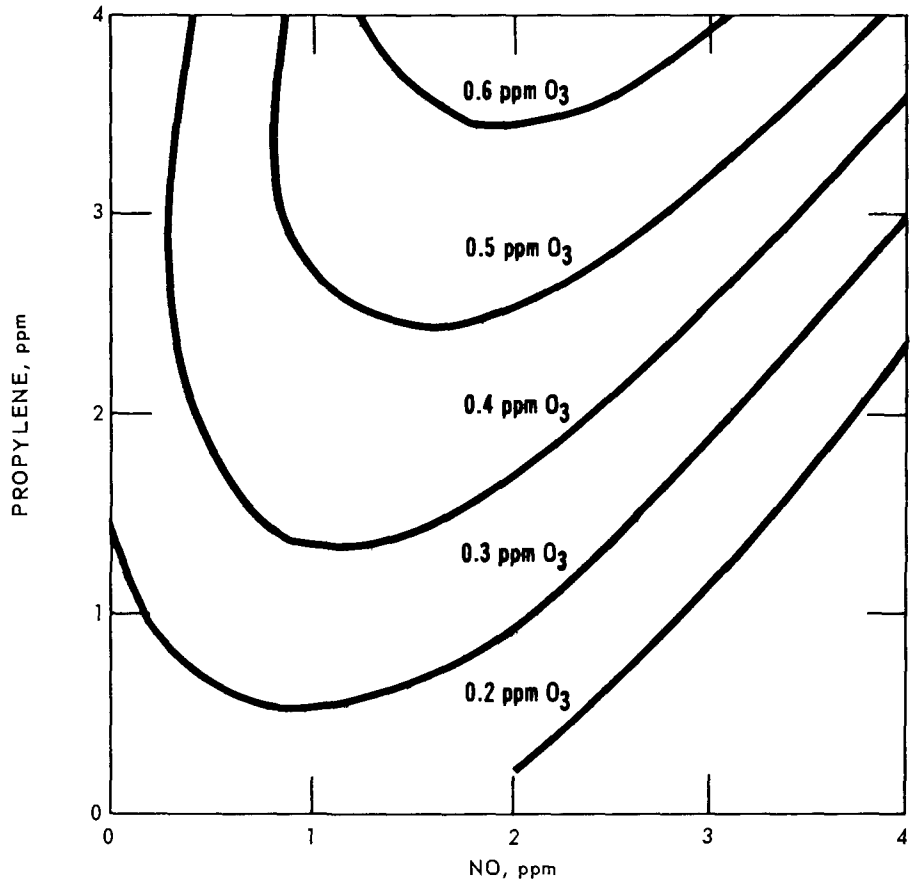


Figure 4-9. Oxidant isopleths from laboratory experiments showing effect of varying initial precursor hydrocarbon (propylene) and NO concentrations on maximum ozone concentrations.[5]

In lieu of adequate atmospheric sampling data, laboratory simulations must be used as broad guidelines in discussing the interactions of the HC-NO_X system that produce ambient OX concentrations. Qualitatively, the precursor-oxidant relationship shown in Figure 4-9 is a typical laboratory finding with certain noteworthy features. At practically every HC-NO_X concentration, reductions in hydrocarbons without NO_X reduction result in a reduction in O_3, whereas NO_X reduction without HC reduction does not always lead to a reduction in O_3.

Sources that emit different proportions of HC and NO_X likewise make different contributions to ambient levels of oxidants. Although the contributing factors are not yet identified, it appears that HC-NO_X ratios do vary in ambient atmospheres. Differing HC to NO_X ratios could arise from the range of operating conditions in automobile engines that result from variations in intake-air temperature and humidity.

4. Limitations and Interpretation of Observational Approach

The ideal approach to defining the HC-NO_X-OX interrelationships would take the form of a comprehensive model, which would take into account the interplay of emission

rates, chemical reactions, and dispersion variables in space and time.

The absence of a fully validated model of this type required the development of the observational model discussed above.

In order to prevent misinterpretation of this observational approach, it is necessary to consider in detail the various factors involved.

a. Assumptions

One of the major assumptions is that the parameters affecting ambient oxidant concentrations are the same as the ones determined in laboratory systems. This does not mean that all known parameters have been delineated. In terms of certain major items, however, the laboratory results and ambient observations are in agreement; in the atmosphere as in the laboratory, the major oxidant formed is ozone. Similarly, the conversion of NO to NO_2, the disappearance of certain reactive hydrocarbons, the appearance of aldehydes and peroxyacyl nitrates as intermediate products, and the dependence of the reactions on sunlight energy, are apparent in both. In addition to the gross similarities, the rates of reaction and relative product yields are consistent.

Another major assumption is that early-morning precursor levels measured at a specific location are proportional to the maximum oxidant values that will be measured at this same location 2 to 4 hours later. This assumption implies both that meteorological conditions are homogeneous and that the precursor emissions are similar over large metropolitan areas. Although this assumption may be open to question, it is necessary at this time because the ambient-air monitoring data presently available are generally restricted to a single station in any one metropolitan area. It has been justified by data from Los Angeles County, which has one of the few comprehensive air-monitoring systems that have been in operation for many years. It was possible to show that oxidant concentrations measured simultaneously at several locations in a 900-square-mile area were similar and related.[10]

Although evidence supporting the similarity of area-wide emissions is less substantial, the similarity of ambient concentrations of HC, NO_X, and oxidant for most days of the year over the entire Los Angeles Basin suggests that the activities leading to emissions are reasonably consistent within a metropolitan area. The similarity of the data also tends to confirm the fact that meteorological conditions are generally homogeneous throughout the area.

On weekends during the summer season, however, a shift in observed emission patterns and resulting maximum oxidant concentrations could be related to the recreational activities of the populace.[10] These alterations could only be demonstrated for stations separated by more than 20 miles, however. Otherwise, the assumption of the similarity between area-wide concentrations and subsequent maximum oxidant values appeared to be valid within a considerable distance of any one specific location.

The HC-NO_X-OX relationship in Figures 4-7 and 4-8 was obtained by comparing 6- to 9-a.m. precursor values to subsequent oxidant levels. The fact that such developed relationships show similar concentration and concentration-ratio effects in both field and laboratory observations (Figure 4-9) is strong support of both of the major assumptions made in the observational approach. If the 6- to 9-a.m. precursor values were not a measure of subsequent maximum oxidant, it would be impossible to obtain atmospheric confirmation of complex chemical interactions such as shown in Figures 4-7 and 4-8.

b. Weekday - Weekend Effect on Model Predictions

Although, as noted, the data in Figures 4-3, 4-4, and 4-5 are not adequate for a more exact delineation of the HC envelopes than is found in Figure 4-7, the effect of some HC-NO_X ratios on ambient oxidant levels can be observed by combining the data from the three cities and focusing attention on the HC range containing the most data. The oxidant

levels for the 2.0- to 2.4-ppm C HC range is shown in Figure 4-10 as a function of NO_X concentration. Although the HC range is narrow, the NO_X values have a rather wide range. The HC shows a 10 percent variation, but the associated NO_X values vary 60 percent.

Part of the wide variation in NO_X could be attributed to a weekday-weekend difference. During the week, NO_X values tended toward the upper range, whereas during the weekend, the values dominated the lower range, as is shown on Figure 4-10. This variation in NO_X, with HC held constant, suggests that the reduction in weekend emissions is not of the same magnitude for NO_X as for HC.

Further analysis of the data in Figures 4-3, 4-4, and 4-5 shows a 12 percent reduction in median total hydrocarbons and a 43 percent reduction in median NO_X on weekends, when the traffic patterns were similarly reduced.

It is not clearly understood why the percentage NO_X reduction was much greater than the percentage HC reduction on weekends. Part of the explanation is undoubtedly associated with the non-specificity of the total HC measurement, including, as it does, a large volume of background methane, which does not vary in proportion to motor-vehicle emissions. The calculated nonmethane HC drop, however, more closely approximates the NO_X drop.[2]

The significant downward shift in weekend emissions, and subsequent lower oxidant levels, in the Los Angeles area suggest that similar effects might occur in other metropolitan areas. To investigate the potential effect of such shifts in pollution patterns on the

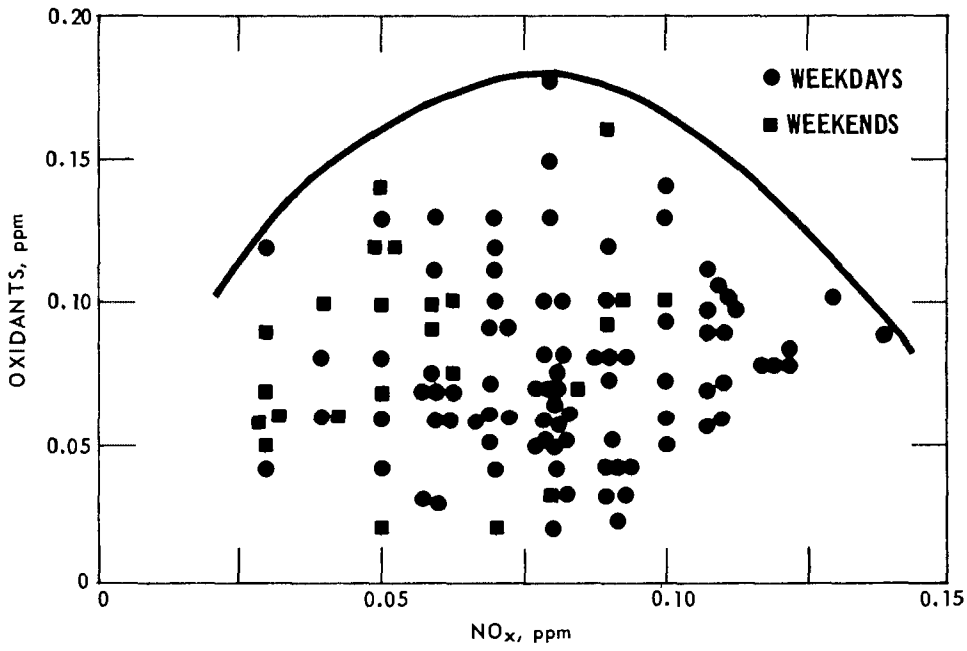


Figure 4-10. Maximum daily 1-hour-average oxidants as a function of 6-to 9-a.m. averages of total nitrogen oxides, for 2.0-to-2.4-ppm C total hydrocarbon in Washington, D.C., Philadelphia, and Denver, June through September, 1965 through 1968.

relationships charted in Figures 4-2 to 4-8, the data points in Figures 4-3, 4-4, and 4-5 were reexamined on a weekday versus weekend basis. In all three cases, the early-morning-weekend-NO_X values are significantly lower than weekday values. Approximately 75 percent of the data below 110 $\mu g/m^3$ (0.06 ppm) NO_X are weekend values, and 75 percent of the data above 110 $\mu g/m^3$ (0.06 ppm) NO_X are weekday values. With the model, therefore, the 80 $\mu g/m^3$ (0.04 ppm) early-morning-NO_X value associated with the maximum daily oxidant of 200 $\mu g/m^3$ (0.1 ppm) is predominantly a weekend observation. This raises the question of whether the weekend effect is due to an actual reduction in emissions or to a shift in the time of emissions, so that the 6- to 9-a.m. period does not include peak NO_X values on the weekend. An hourly check of the 6-a.m. to 12-noon-NO_X values on weekends revealed no consistent shift in the time of peak NO_X. It must be recalled that after 9 a.m. the NO_X can be incorporated into photochemical products, and so the ambient NO_X values measured during sunlight hours are no longer an accurate indicator of peak emissions. In this same study, the time of day for peak OX was examined for a weekend shift, but no consistent trend could be found.

The weekend effect is apparently due to emission reduction. Figure 6-3 (chapter 6) shows NO emission in Chicago during 1962 to 1964. Comparison of the weekend and weekday values in this figure indicates a substantial reduction of NO emissions on weekends.

Examination of carbon monoxide (CO) values provided additional information on the subject. This relatively inert substance may be used as an indicator of motor-vehicle emissions. The diurnal variation in CO on weekdays, Saturdays, and Sundays, in Chicago for the 1962 to 1964 period is shown in Figure 4-11. The most prominent feature of this graph is the substantial decrease in CO values on weekends, particularly during the morning hours, which suggests that the lower weekend emissions, and subsequent lower OX values, are predominantly the result of weekend reductions in vehicular traffic and other technology-related activities. Here again, the 6- to 9-a.m. period still appears to be the best choice for relating precursor emissions to subsequent maximum oxidant values. An extension of this time period to include later hours would only associate lower average precursor values with maximum daily oxidant concentrations.

The correlation between lower emissions and lower oxidant values provides further support for the observational model. The weekday-weekend difference is a direct measure of the improvement in air quality to be expected from a control method that reduces early-morning weekday emissions to the weekend level.

Although the foregoing analysis suggests the validity of relating precursor and oxidant values at a single point, it does not preclude the need for an adequate air monitoring network. The single-station approach is one born of necessity and should be replaced by better monitoring networks to support a more comprehensive emission-reaction model.

c. Effects of Reductions in Photochemical Precursors

As noted earlier, reduction in NO_X without simultaneous reduction in HC does not always lead to reduction in oxidants. Such laboratory findings indicate that if the objective is control of oxidant then control of HC is the most appropriate step to take. Unfortunately, oxidant control is not the only consideration. NO, itself, must be controlled or even reduced from present ambient levels, because its oxidation product, NO_2 is currently at or near levels that are associated with adverse health effects in over 50 percent of United States cities having a population greater than 50,000 (see chapter 10). The laboratory findings also raise the possibility that NO_X control by itself may, under certain circumstances, lead to increases in oxidant levels. Such increases, however, would only be characteristic of partial NO_X control since complete NO_X control, like complete HC control, would prevent formation of oxidant.

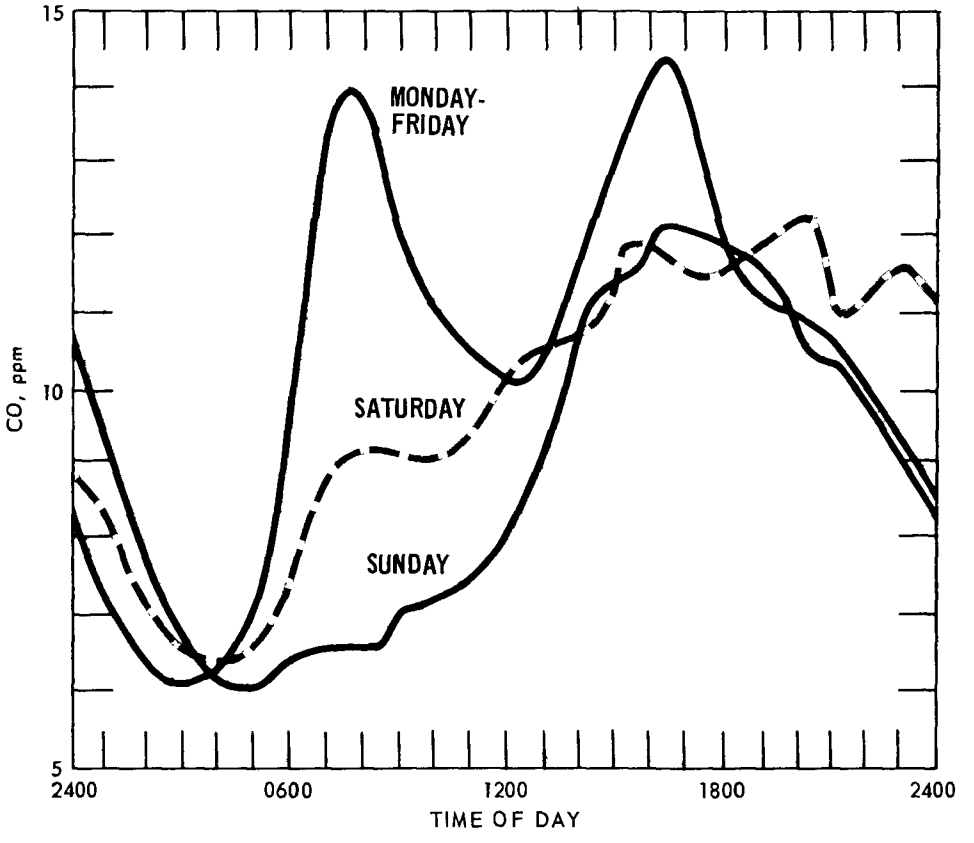


Figure 4-11. Diurnal variation of mean carbon monoxide levels on weekdays, Saturdays, and Sundays in Chicago, 1962 through 1964.[11]

An additional consideration is the effect of HC or NO_X reductions on other products of the photochemical complex, such as eye irritants and visibility-limiting aerosols, which are also related to oxidant concentrations. Although these products vary with oxidant levels, the HC-NO_X mixture having the greatest potential for oxidant production will not necessarily correspond to maximum potential for eye irritants.[5,12] The effect can be visualized in greatly simplified manner by examining known and expected reactions in a HC-NO_X system containing an excess of either HC or NO. Excess HC may act as a scavenger for oxidant and reduce oxidant levels with an accompanying increase in the eye irritant and other HC oxidation products. Excess NO acts as a scavenger for oxidant and thus reduces oxidant levels, increases NO_2 levels, and possibly decreases eye-irritant products. Unfortunately, much less is known about ambient levels and variations in eye irritants and areosols than about the ambient levels of oxidants and NO_2. For this reason, only generalized relationships of oxidant levels to eye irritation and visibility reduction can be defined. It is assumed that changes in all atmospheric pollutant concentrations in the years ahead will tend to modify these observed relationships. Such changes will most likely be small, and though demonstrable in the laboratory, may not be observable in the atmosphere, since all of the products are interdependent. Large reductions

of HC, NO_X, or both, however, should reduce all of the pollutants and the resulting manifestations.

Fortunately, APCO has a considerable body of ambient and laboratory information concerning the atmospheric relationships of oxidant and NO_2 that are most important from a health-related standpoint. What is needed in the atmospheric data is a determination of the most logical precursor-reduction method that will guarantee reductions in oxidant and NO_2 to levels consistent with known public health requirements. Only one reduction program promises to accomplish this goal, that is, a vigorous HC-reduction program coupled with NO_X reductions designed to reduce NO_2 levels below the health-related criteria level. The necessity for reducing NO_X below this suggested point cannot be ascertained at this time. Nonetheless, the use of less stringent NO_X reductions (coupled with vigorous HC reductions), the possibility of causing a slight increase in oxidant values in minimized and, at the same time, the scavenging effect of NO for oxidant is maximized.

C. ADDITIONAL REACTIONS AND PRODUCTS OF NO_X

In the previous sections, the major concern was the NO_2 photolytic cycle and its relationship to the major atmospheric oxidant, ozone. In addition to this major role the oxides also engage in reactions leading to formation of several nitrogen-containing compounds. Unfortunately, less is known about the fate of NO_X than is known about the fate of the hydrocarbons. In laboratory simulations, various investigators have been able to account for over 90 percent of the carbon atoms but only about 50 percent of the nitrogen.

One of the more important classes of nitrogen compounds resulting from atmospheric HC-NO_X interactions is known as the peroxy nitrates (peroxyacyl nitrates and peroxybenzoyl nitrates). Although these oxidants are minor from a concentration standpoint, peroxy nitrates are powerful eye irritants and phytotoxicants, and hence important contributors to photochemical air pollution. Peroxyacetyl nitrate (PAN), which has the highest atmospheric concentrations, has been extensively investigated. Pathways for formation of this class of compounds have been proposed, and several reaction systems have been used to produce these compounds in the laboratory; however, the specific mechanism involved in the atmosphere has not been established. Several other members of this class of peroxy nitrates that have been prepared and studied in the laboratory include peroxypropionyl nitrate, peroxybutyryl nitrate, peroxyisobutryl nitrate, and peroxybenzoyl nitrate,[13] but these have not been found in ambient atmospheres. Nor has the formation of the initial member of the peroxy nitrate series, peroxyformyl nitrate, been accomplished in the laboratory or observed in ambient atmospheres.

This class of peroxy nitrates causes eye irritation and vegetation damage. Recent investigations[14] suggest peroxyacetyl nitrate, and probably the other homologues, are important to the conversion of NO to NO_2 in ambient atmospheres.

Additional experimental evidence suggests that some undetermined fraction of the NO_X ends up as nitrous and nitric acid, and trace amounts of N_2O. Furthermore, nitrates have been identified as an important fraction of photochemically formed aerosols.

D. FUTURE RESEARCH

This examination of the role of NO_X in photochemical air pollution points out several areas requiring further investigation. The limitations of the observational hypothesis clearly indicate the need for: (1) a more comprehensive and definitive simulation model for NO_X, HC, and oxidants, (2) more precise and definitive air-monitoring instruments, and (3) an intensified effort to identify the mechanisms of reactions and the fate of NO_X.

The observational model, for example, can only relate average concentration of pre-

cursors during an arbitrarily chosen time period (6- to 9-a.m.) to the upper-limit of maximum observed oxidant levels that occur later in the day. The model cannot, in its present form, relate rates of precursor emissions to rates of oxidant formation, nor does it define the relative contribution of various sources, either near or distant, to any given oxidant maximum.

Coupled with the need for broader instrumentation is the need for better air monitoring networks. In many cities the description of air quality and changes in air quality are still dependent upon the data from a single station. Such a single station cannot adequately describe the variations in air quality that affect metropolitan areas which occupy several hundred square miles. The air-monitoring stations and networks that do exist provide only a limited description of air quality. There is a dearth of monitoring information concerning specialized community exposures. For example, the air quality regime to which the populace is exposed while traveling on, or living adjacent to, heavily trafficked roadways, cannot be adequately described.

Besides additional measurement of specific emissions and their products, the development of a more refined, detailed model requires more elaborate delineation of the meteorological parameters. At the present time, certain key factors, such as mixing height and sunlight intensity, are not measured on a routine basis in most polluted areas.

Better information is required to relate emission rates to air quality, as a function of source and time.

Although knowledge of the NO_2 photolytic mechanism and the ways in which this cycle interacts with reactive HC has a broad scope, the fate of up to 50 percent of the atmospheric NO_x is still not accounted for. Details regarding these unidentified products are required in order to clearly assess their impact on health and welfare.

E. SUMMARY

The relationships between early-morning ambient HC levels and the maximum oxidant level observed later in the day have been examined through an atmospheric *observational* approach,[1,2] which does not attempt to describe the complex relationship of oxidant to ambient NO_x and HC levels. This document is concerned with a study of the complete HC-NO_x-OX precursor - product relationship, and primarily with the changes in ambient oxidant and NO_x levels resulting from absolute and relative concentration variations of the HC-NO_x complex. The results of this examination are listed below.

1. The observational approach shows that the same reaction parameters that govern the HC-NO_x-OX system in the laboratory operate in ambient atmospheres.

2. The ambient levels of 6- to 9-a.m. precursors, HC and NO_x, are a reliable indicator of the maximum attainable 1-hour-average-oxidant concentration that occurs 2 to 4 hours later, between 10 a.m. and 2 p.m.

3. The atmospheric conditions that lead to maximum oxidant potential, i.e., low windspeeds, high temperature, intense sunlight, and surface inversions, occur on approximately 1 percent of the days.

4. Independent studies of the effect of variations in either HC or NO_x level are not possible in ambient atmospheres. Atmospheric observation shows, however, that when the calculated 6- to 9-a.m. nonmethane hydrocarbons are in the approximate range of 200 to 930 $\mu g/m^3$ (0.0 to 1.4 ppm C) and the 6- to 9-a.m. NO_x value is approximately 80 $\mu g/m^3$ (0.04 ppm), the maximum daily oxidant will reach 200 $\mu g/m^3$ (0.10 ppm) on only 1 percent of the days.

Further observation of the data base shows that 80 $\mu g/m^3$ (0.04 ppm) NO_x occurs simultaneously with calculated nonmethane HC concentrations ranging from 200 to 930 $\mu g/m^3$ (0.3 to 1.4 ppm C). Similarly, observation of the 200 $\mu g/m^3$ (0.3 ppm C) nonmethane HC level shows NO_x in the range of 80 to 320 $\mu g/m^3$ (0.04 to 0.16 ppm).

5. Laboratory studies do permit the independent evaluation of the effects of varying either HC or NO_X. Using such studies as guidelines, control agencies can consider a combinational HC and NO_X reduction program applicable to polluted ambient atmospheres. The data base suggests that reductions in HC should be the primary step for control of oxidants. Coupled with HC control, NO_X must be controlled at a level that will hold ambient NO_2 values below the level of adverse health effects.

6. NO_X have also been implicated in the formation of peroxy nitrates and aerosols. The precise mechanism of formation has yet to be elucidated, but, to the extent that oxidant levels are a measure of the intensity of the effects of the photochemical complex, the control of NO_X and HC to the suggested levels can be expected to control all undesirable aspects of NO_X and its products.

These results are largely based on examination of current ambient atmospheric data as defined by current monitoring techniques. It can be expected that these techniques will be modified to provide improved information. Furthermore, the application of emission-control measures can be expected to change the relative, as well as the absolute, concentrations of atmospheric contaminants. All of these factors dictate the need for a continual reexamination and updating of the observations in this chapter.

H. REFERENCES

1. Air Quality Criteria for Photochemical Oxidants. National Air Pollution Control Administration. Washington, D. C. Publication No. AP-63. March 1970.

2. Air Quality Criteria for Hydrocarbons. National Air Pollution Control Administration. Washington, D. C. Publication No. AP-64. March 1970.

3. Altshuller, A.P., et al. Chemical Aspects of the Photooxidation of the Propylene-Nitrogen Oxide System. Environ. Sci. Technol. *1*: 899-914, November 1967.

4. Schuck, E. A. and G. J. Doyle. A Study of Irradiated Auto Exhaust. Stanford Research Institute. Report No. 9, Technical Report II. February 1958.

5. Romanovsky, J. C., R. M. Ingels, and R. J. Gordon. Estimation of Smog Effects in the Hydrocarbon-Nitric Oxide System. J. Air Pollut. Contr. Ass. *17*:454-459, July 1967.

6. Korth, M. W., A. H. Rose, and R. C. Stahman. Effects of HC/NO_X on Irradiated Auto Exhausts. Part I. J. Air Pollut. Contr. Ass. *14*:168-175, May 1964.

7. Dimitriades, Basil. On the Function of Hydrocarbons and Nitrogen Oxides in Photochemical Smog Formation. U. S. Bureau of Mines Report of Investigations, RI 7 and 33, September 1970.

8. Schuck, E. A., et al. Relationship of Hydrocarbons to Oxidants in Ambient Atmospheres. J. Air Pollut. Contr. Ass. *20*:297-302, May 1970.

9. 1965 Through 1968 Data Tabulations and Summaries (Denver, Philadelphia, Washington). National Air Pollution Control Administration, Arlington, Virginia. Data on file in APCO Division of Air Quality Emissions Data. Durham, N.C.

10. Schuck, E. A., J. N. Pitts, and J. K. S. Wan. Relationships Between Certain Meteorological Factors and Photochemical Smog. Air Water Pollut. *10*:689-711, 1966.

11. The Automobile and Air Pollution, A Program for Progress, Part II. U. S. Department of Commerce. Washington, D. C. December 1967.

12. Hamming, W. J. and J. E. Dickinson. Control of Photochemical Smog by Alteration of Initial Reactant Ratios. J. Air Pollut. Contr. Ass. *16*:317-323, June 1966.

13. Stephens, E. R. The Formation, Reactions, and Properties of Peroxyacyl Nitrates (PANS) in Photochemical Air Pollution. In: Advances in Environmental Sciences and Technology, Vol. I., Pitts, J.N. and R.L. Metcalf (eds.). New York, Wiley - Interscience, 1969, p. 119-146.

14. Schuck, E. A. and E. R. Stephens, Oxides of Nitrogen, In: Advances in Environmental Sciences and Technology, Vol. I., Pitts, J. N. and R. L. Metcalf (eds.). New York, Wiley-Interscience, 1969. p. 73-118.

CHAPTER 5.

METHODS FOR MEASUREMENT OF NITROGEN OXIDES

A. INTRODUCTION

The most accurate, reliable, and useful methods for measurement of the atmospheric nitric oxide (NO), nitrogen dioxide (NO_2), and nitrogen oxides (NO_x) employ standard techniques to collect the sample in an absorbing solution. The analytical determination is made colorimetrically or spectrophotometrically. Analytical methods based on physical properties such as absorption or emission of radiation, oxidation, or reduction and methods based on gas-chromatographic separation and detection are not yet in common use but will probably receive more attention in the future. Measurement techniques for other oxides of nitrogen will not be discussed in this chapter.

B. NITROGEN DIOXIDE

1. Manual Methods

a. Griess-Saltzman Method

The colorimetric Griess-Saltzman method[1] is deemed the most suitable manual method generally applicable to the measurement of NO_2 in the atmosphere.* This method is based on the reaction of NO_2 with sulfanilic acid to form a diazonium salt, which couples with N-(1-naphthyl)-ethylenediamine dihydrochloride to form a deeply colored azo dye. Air is sampled through a fritted bubbler into a solution of the Griess-Saltzman reagent for periods of 30 minutes or less, and the color is allowed to develop for an additional 15 minutes after completion of sampling. The color produced, which is proportional to the amount of NO_2 sampled, is measured at 550 nanometers (nm). The method can be used to determine concentrations of NO_2 in the air from 40 to 1500 $\mu g/m^3$ (0.02 to 0.75 ppm).

Ordinarily, interferences are not a problem, although high ratios of sulfur dioxide to nitrogen dioxide (about 30:1) can cause bleaching and give misleadingly low values.[1] Interference from other oxides of nitrogen or ozone are negligible at concentrations found in polluted air. Peroxyacetylnitrate (PAN) can give a response of up to 35 percent of an equivalent molar concentration of NO_2, but in ordinary ambient air PAN concentrations are too low to cause significant error.[1] The color formed is reasonably stable, but samples should be read within 1 hour of completion of sampling.

In the original method Saltzman[3] used solutions of sodium nitrite as calibration standards. He reported that, under laboratory conditions, 0.72 mole of nitrite produces the same color as 1 mole of NO_2 gas and incorporated this factor into his calculations. In recent years this stoichiometric factor has been the source of considerable controversy. Values ranging from 0.5 to 1.00 have been reported,[4-10] and one recent work[11] reconfirmed Saltzman's original findings. The method can be standardized by using an accurately known concentration of NO_2 gas and thus eliminate the stoichiometric factor from the calculations.

b. Jacobs-Hochheiser Method

The Griess-Saltzman method for NO_2 measurement cannot be used successfully when the delay between sample collection and color measurement is more than 4 to 6 hours or when sampling periods of longer

*This method has been adopted as a tentative method by the Intersociety Committee on Manual of Methods for Ambient Air Sampling and Analysis.[2]

than 1 hour are required. In such situations the Jacobs-Hochheiser method[12] is preferred. With this method, NO_2 can be measured in a network where a central laboratory is used for analyzing field samples. With a later modification[12-14] of the procedure, NO_2 sampling periods can be as long as 24 hours, and the delay of analysis can be at least 2 weeks, if necessary.

In the Jacobs-Hochheiser procedure, polluted air is passed through aqueous sodium hydroxide, in which nitrite ion is formed from the NO_2. The resulting solution, which contains sodium nitrate in addition to sodium nitrite, is treated with hydrogen perioxide to remove possible sulfur dioxide interference, and acidified. The nitrous acid produced is measured by the Griess-Saltzman diazotizing-coupling procedure, except that sulfanilamide is used in place of sulfanilic acid. In the development of the Jacobs-Hochheiser procedure, the ratio of nitrite to nitrate[14] was determined and an average stoichiometric factor of 0.63 was selected for converting nitrite ion to NO_2 concentrations.

Because the sampler adopted by the National Air Surveillance Network (NASN) of A P C O employs a 24-hour sampling period, it precludes the use of the Griess-Saltzman method and all data obtained by this network since its inception have been derived by the Jacobs-Hochheiser method. Because of discrepancies concerning the NO_2-nitrite stoichiometric factor, all NASN computations are now based on the assumption that one molecule of NO_2 produces one molecule of nitrite and no nitrate.

The absorption efficiency for NO_2 varies with the sampling system used. Detailed studies have shown that, under laboratory conditions, the NASN sampling system has an analytical efficiency of approximately 35 percent, a correction that is applied in NASN calculations of NO_2 concentrations. Other investigators[13,15] have found different efficiencies.

2. Continuous Method

Most continuous NO_2 analyzers use the Griess-Saltzman method or a slight modification of it. The sample air is drawn through an absorber at an accurately determined flow rate countercurrent to a controlled flow of absorbing reagent. The absorber must be carefully designed and properly sized because NO_2 is somewhat difficult to absorb. Sufficient time is allowed for full color development, and then the colored solution is passed to a colorimeter or spectrophotometer where the intensity is measured continuously at 550 nm.

3. Miscellaneous Methods

Correlation spectrometry,[16,17] gas chromatography,[18] long-path infrared spectroscopy, and electrochemical oxidation and reduction[19] have been applied to NO and NO_2 analysis, but have not found wide application in atmospheric sampling and measurement. Correlation spectrometry, a promising new technique for remote sensing, requires further field trials before it can be adopted for air pollution measurement.[20] Gas chromatography is potentially a useful and versatile method for measuring NO_2.[2] Even though it is intermittent by nature and, in the strictest sense, does not lend itself to continuous monitoring, gas chromatography can nonetheless analyze sequential samples rapidly enough to approximate a continuous measurement. Long-path infrared spectroscopy uses large-volume cells, which make rapid response difficult to achieve in a continuous-monitoring instrument. Long-path infrared also has the disadvantage that water vapor interferes with the measurements. Electrochemical transducers, which operate on the principle of oxidation or reduction of pollutant absorbed on a sensing electrode, show excellent potential as simple, compact means of analyzing for NO_2 and NO_x in sources such as automobile exhaust. At present, however, these sensors lack adequate sensitivity for ambient air analysis.

4. Calibration

Any measurement method should be calibrated to assure accuracy. The preferred procedure for calibrating a method for measuring NO_2 is to use the entire system, including the sampling line and its components, to analyze an atmosphere containing accurately known amounts of NO_2 in the concentration range to be studied. These calibration gases can be generated by simple-gas-dilution with check-analysis,[21] by precision-gas-dilution without check-analysis,[9] by a gravimetric procedure,[22] by an electrolysis system,[23] or by use of a permeation tube.[24] The permeation-tube procedure is by far the most accurate and simplest of these methods. In another less complex procedure for calibration[25] accurately known concentrations of sodium nitrite solutions are used to calibrate the analytical portion of the method. Since the sampling efficiency of the method being calibrated is not taken into account, the procedure is considered inferior to the permeation-tube method.

Permeation tubes deserve special mention because of their accuracy, simplicity, and growing use. A permeation tube is a device used to generate artifical atmospheres with known NO_2 concentrations.[11] Constructed of Teflon, it contains a liquified gas such as NO_2, which permeates through the walls at a slow, but constant rate, as long as the temperature of the device is held constant. The permeation rate of the gas can be accurately determined.[26] One suitable system using a permeation tube is shown schematically in Figure 5-1. The temperature of the bath containing the permeation device must be held constant, preferably within 0.1° C, and the air or nitrogen passed across the permeation tube must be dry. This technique has been used for both field and laboratory calibrations. NO_2 permeation tubes are moisture-sensitive, and care must be taken to protect them from atmospheric moisture during use and storage.

After any method has been calibrated under closely controlled laboratory conditions, preferably using the permeation-tube technique, the problem of interferences in the ambient air at the sampling site should be considered. One method for dealing with the interference problem is to use local ambient air for which NO_2 background is determined concurrently, as dilution air. Such a procedure will be satisfactory if all of the precautions in the use of permeation tubes, particularly that of allowing only dry air or nitrogen to pass over the tube, are observed.

C. NITRIC OXIDE

1. Oxidation to NO_2

Because no appropriate analytical techniques for direct measurement of NO exist, NO is generally determined by oxidizing it to NO_2, measuring the NO_2 and then converting the values to NO concentrations. The oxidation step is as yet poorly understood. Some workers find it unreliable and report conversion efficiencies ranging from 40 to 110 percent.[25,27] The oxidizers used are aqueous[28] or solid[29] potassium permanganate, and dichromate or chromium trioxide in various formulations[27, 30, 31] all of which have uncertain lifetimes at peak efficiency. In addition, the dichromate and chromium troxide preparations are extremely sensitive to high humidity. Aqueous permanganate seems to have the fewest limitations; hence, it is the best current choice.

It is generally believed that NO concentrations are underestimated as a result of the poor conversion efficiency, a factor that should be kept in mind when studying NO data. Steps can be taken to minimize the problem, but oxidation of NO to NO_2 must always be considered a potential source of error.

a. Series Mode

The measurement of NO by the oxidation method can be done in either a series or a parallel mode. In the series mode (Figure 5-2A), which some workers[32,33] prefer, the sample air passes through an NO_2 analyzer for removal and measurement of NO_2, then through an oxidizer where NO is converted to NO_2, and finally through a second NO_2

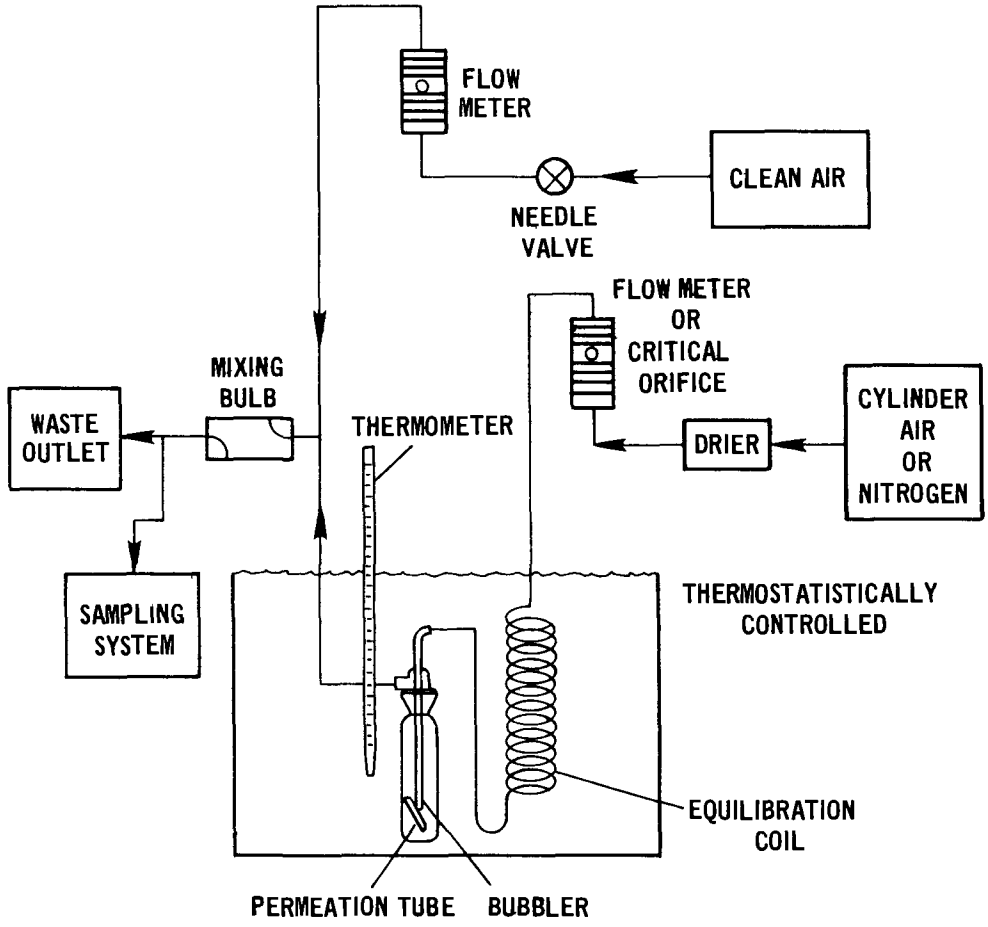


Figure 5-1. Permeation tube system.

analyzer. The response from the second NO_2 analyzer is a measure of the NO in the sample air.

b. Parallel Mode

The parallel mode (Figure 5-2B), requires two separate analyses. In one, sample air is analyzed for NO_2; and in the other, sample air is analyzed for total NO_x. The NO concentration is the difference between the total NO_x and the value for NO_2. In order to carry out the analysis for total NO_x, the sample air is passed through an oxidizing scrubber to convert NO into NO_2. In addition to the usual problem of uncertain efficiency in NO to NO_2 conversion, the problem of possible capture of NO_2 by the scrubber adds to any basic unreliability in the procedure.

2. Chemiluminescent Methods

Recently, a device that operates on the principle of chemiluminescence has been developed.[34] A detector measures the luminescence produced during the gas-phase reaction between NO and ozone. During the operation, ambient air flows continuously through a calibrated orifice into a vessel, where it is mixed with ozone and gives rise to

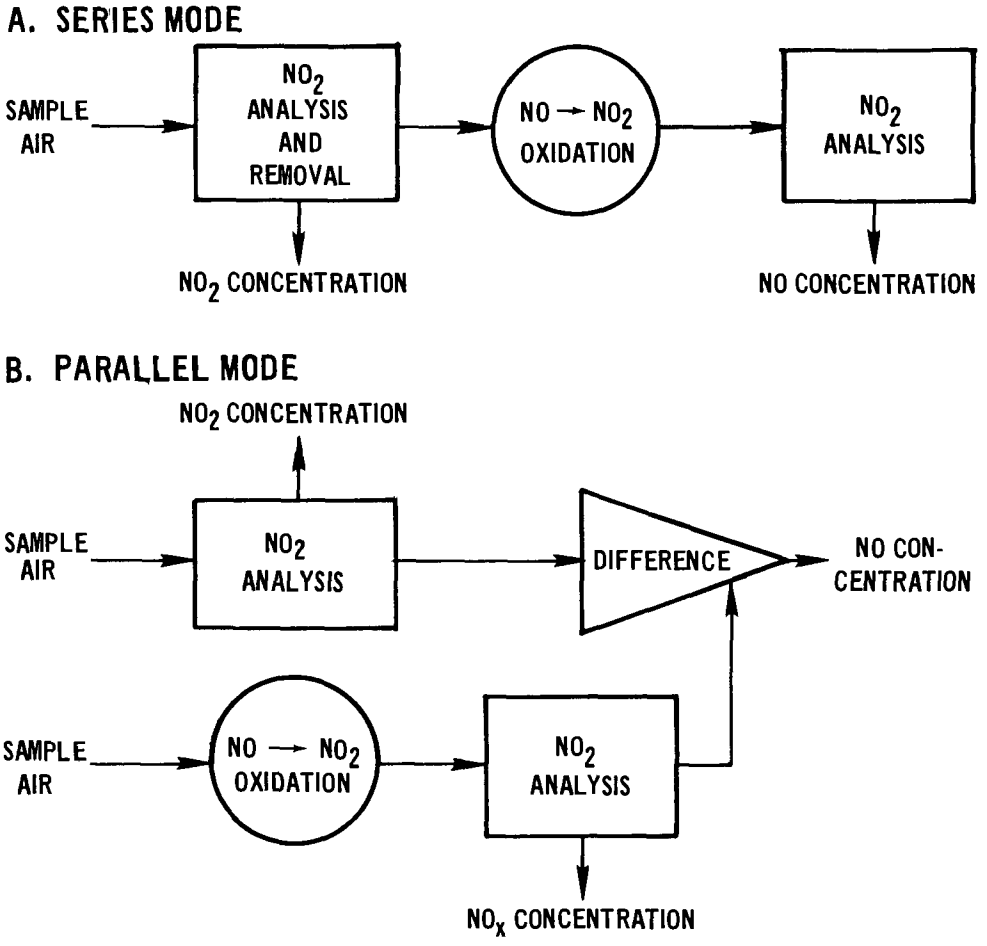


Figure 5-2. Oxidation schemes for analyzing NO and NO_x.

measurable luminescense. This approach represents a promising development in direct NO-measurement methododology.

3. Calibration

Artifical atmospheres containing known concentrations of NO are not easily prepared. The extreme ease with which NO at high concentrations is oxidized to NO_2 makes a dilution system impractical and the very low boiling point (−151.8° C) of NO makes preparation and calibration of permeation devices extremely difficult. Since most NO analyzers are, in reality, NO_2 analyzers preceded by an oxidizer, NO_2 can be used for calibration. Removal of the oxidizer before calibration is recommended to avoid the problem of NO_2 capture.

D. TOTAL NITROGEN OXIDES

1. Oxidation of NO to NO_2

At some point in the analysis all of the methods in general use for measuring total NO_x involve the oxidation of NO to NO_2, with the attendant problems of this step. Either the series or the parallel method in Figure 5-2 can be adapted to give the NO_x concentration, the former by summing NO and NO_2 concentrations, the latter by on-line reading.

2. Chemiluminescent Method

Total NO_x have been measured by a chemiluminescent reaction involving oxygen atoms and NO_x[35] in a gas-phase reaction similar to the one used for NO measurement. In this

system NO_x is mixed with oxygen atoms in a chamber where the oxygen atoms rapidly and quantitatively convert NO_2 to NO in the gas phase, and excess oxygen atoms react with NO to produce chemiluminescence, which is then measured by appropriate means. Though the method holds much promise, it has not yet been widely tested in the field.

E. SUMMARY

Nitrogen dioxide is the only atmospheric oxide of nitrogen that can be measured directly with currently used techniques. All of the procedures for measuring atmospheric NO and NO_x rely on some type of converter to oxidize NO to NO_2, which is then measured and arithmetically converted to respective values.

Any method used for measuring NO_2 in the ambient air should be calibrated before use with sample atmospheres containing known amounts of NO_2. Permeation tubes are recommended for generating such test atmospheres.

For sampling periods of 30 minutes or less, the most suitable method for measuring NO_2 is the manual colorimetric Griess-Saltzman method[1] in which the color depends on conversion of NO_2 to nitrite ion. The degree of conversion of NO_2 gas into nitrite ion remains a point of controversy in the Griess-Saltzman and other methods. Nonetheless, this method is relatively free of interferences and may be employed to determine concentrations of NO_2 ranging from 40 to 1500 $\mu g/m^3$ (0.02 to 0.75 ppm).

For periods from 30 minutes to 3 hours, sampling can be done either by taking the appropriate number of discrete 30-minute samples with the manual Griess-Saltzman method or by using the continuous Griess-Saltzman method for the entire period.

For sampling periods of 3 to 24 hours or for situations where analysis must be delayed more than 4 hours after sampling, the most suitable currently available method for measuring NO_2 is the Jacobs-Hochheiser method.[1,2] Because the collection efficiency of the Jacobs-Hochheiser method is low and variable, the method must be carefully calibrated before use.

The Griess-Saltzman and the Jacobs-Hochheiser methods are not interchangeable and can yield different results; therefore, the appropriate one must be carefully chosen according to the purpose of the sampling to be done.

For continuous NO_2 measurements, the most suitable method currently available is based on the Griess-Saltzman procedure. When used in conjunction with an oxidizing scrubber for conversion of NO to NO_2, the continuous Griess-Saltzman method can also be used, in either the series or parallel mode, for measuring NO in ambient air. Researchers disagree as to which of the two modes is more satisfactory, but the major problem with NO measurement lies in completely oxidizing NO to NO_2.

Total nitrogen oxides can be determined by summing NO and NO_2 concentrations from separate samples, or by oxidizing NO to NO_2 and then measuring NO_2.

New approaches for measuring NO and NO_x based on chemiluminescence are promising, but have yet to be adequately field-tested.

Research is still needed to develop and thoroughly evaluate sensitive, reliable, and practical methods for determining NO, NO_2, and NO_x.

F. REFERENCES

1. Standardization Advisory Committee, National Air Pollution Control Administration. Tentative Method for Determination of Nitrogen Dioxide in the Atmosphere (Griess-Saltzman Reaction) (in press).
2. Intersociety Committee on Manual of Methods for Ambient Air Sampling and Analysis, Subcommittee 3. Tentative Method of Analysis for Nitrogen Dioxide Content of the Atmosphere (Griess-Saltzman Reaction). Health Lab. Sci. *6*: 106-113, April 1969.
3. Saltzman, B. E. Colorimetric Microditermination of Nitrogen Dioxide in the Atmosphere. Anal. Chem. *26*: 1949-1955, December 1954.

4. Stratmann, H. and M. Buck. Messung von Stickstoffdioxid in der Atmosphere. Air Water Pollut. *10*: 313-326, May 1966.
5. Buck, M. and H. Stratmann. The Joint and Separate Determination of Nitrogen Monoxide and Nitrogen Dioxide in the Atmosphere. Staub *27*: 11-15, June 1967.
6. Huygen, I. C. Reaction of Nitrogen Dioxide with Griess Type Reagents. Anal. Chem. *42*:407-409, March 1970.
7. Gill, W. E. Determination of NO and NO_2 in Air. Am. Ind. Hygiene Ass. J. *21*(1): 87-96, February 1960.
8. Kooiker, R. H., L. M. Schuman, and Y. K. Chan. Nitrogen Dioxide Poisoning. Arch. Environment Health *7*(1): 13-32, July 1963.
9. Saltzman, B. E. and A. F. Wartburg, Jr. Precision Flow Dilution System for Standard Low Concentrations of Nitrogen Dioxide, Anal. Chem. *37*: 1261-1264, September 1965.
10. Shaw, J. T. The Measurement of Nitrogen Dioxide in Air. Atmos. Environment *1*(2): 81-85, March 1967.
11. Scaringelli, F. P., E. Rosenberg, and K. A. Rehme. Comparison of Permeation Devices and Nitrite Ion as Standards in the Colorimetric Determination of Nitrogen Dioxide. Environ. Science Technol. *1970 (in press)*.
12. Standardization Advisory Committee, National Air Pollution Control Administration. Tentative Method for Determination of Nitrogen Dioxide in the Atmosphere (24-Hour Sampling Method) (in press).
13. Jacobs, M. B., and S. Hochheiser. Continuous Sampling and Ultramicrodetermination of Nitrogen Dioxide in Air. Anal. Chem. *30*: 426-428, 1958.
14. Morgan, G. B., C. Golden, and E. C. Tabor. New and Improved Procedures for Gas Sampling and Analysis in the National Air Sampling Network. J. Air Pollut. Contr. Ass., *17*: 300-304, May 1967.
15. Meadows, F. L. and W. W. Stalker. The Evaluation of Efficiency and Variability of Sampling the Atmosphere for Nitrogen Dioxide. Am. Ind. Hygiene Ass. J. *27*: 559-566, November-December 1966.
16. Barringer, A. R. and B. C. Newburg. Remote Sensing Correlation Spectrometry for Pollution Measurement. (9th Conference on Methods in Air Pollution, Pasadena, California, February, 1968).
17. Kay, R. B. Absorption Spectra Apparatus Using Optical Correlation for the Detection of Trace Amounts of SO_2. J. Appl. Optics. *6*: 776-778, 1967.
18. Morrison, M. E. and W. H. Corcoran. Optimum Conditions and Variability in Use of Pulsed Voltage in Gas-Chromatographic Determination of Part-Per-Million Quantities of Nitrogen Dioxide. Anal. Chem. *39*: 255-258, 1967.
19. Shaw, M. Electrochemical Tranducers. (Paper presented at the Symposium on Advances in Instrumentation for Air Pollution Control, Cincinnati, Ohio, 1969.)
20. Remote Monitoring Instrumentation for Source Emissions and Urban Atmospheres. Clean Air Quarterly (State of California), *11*: 9-12, December 1967.
21. Chrisman, K. F. and K. E. Foster. Calibration of Automatic Analyzers in a Continuous Air Monitoring Program. (56th Annual Meeting of the Air Pollution Control Association, Detroit, 1963.)
22. Thomas, M. D. and R. E. Amtower. Gas Dilution Apparatus for Preparing Reproducible Dynamic Gas Mixtures in Any Desired Concentration and Complexity. J. Air Pollut. Contr. Ass., *16*: 618-623, November 1966.
23. Hersch, P. and R. Deuringer. Trace Addition of Nitric Oxide and Nitrogen Dioxide to Air by Electrolysis. (56th Annual Meeting, Air Pollution Control Association, Detroit, 1963.) Paper 63-36.
24. O'Keeffe, A. E. and G. C. Ortman. Primary Standards for Trace Gas Analysis. Anal. Chem. *38*: 760-763, 1966.
25. Interbranch Chemical Advisory Committee. Selected Methods for the Measurement of Air Pollutants. Public Health Service, Cincinnati. Publication No. 999-AP-11, 1965.
26. Scaringelli, F. P., et al. Preparation of Known Concentrations of Gases and Vapors with Permeation Devices Calibrated Gravimetrically. Anal. Chem. *42*: 871-876, July 1970.
27. Repley, D. L., J. M. Clingenpeel, and R. W. Hurn. Continuous Determination of Nitrogen Oxides in Air and Exhaust Gases. Air Water Pollut. *8*: 455-463, 1964.
28. Thomas, M. D., et al. Automatic Apparatus for Determination of Nitric Oxide and Nitrogen Dioxide in the Atmosphere. Anal. Chem. *28*: 1810-1816, December 1956.
29. Calhoun, J. D. and C. R. Brooks. A Solid Oxidant for Oxides of Nitrogen Analyzer. (7th Conference on Methods in Air Pollution, Los Angeles, California, January 1965.)
30. Wilson, D., and S. L. Kopczynski. Laboratory Experiences in Analysis of Nitric Oxide with "Dichromate" Paper. J. Air Pollut. Contr. Ass. *18*: 160-161, March 1968.

31. Jones, E. E., L. B. Pierce, and P. K. Mueller. Evaluation of a Solid Oxidant System. (7th Conference on Methods in Air Pollution Studies, Los Angeles, January 25, 1965.)
32. Mueller, P. K. and Y. Tokiwa. Series vs. Parallel Continuous Analysis for NO, NO_2 and NO_x. (8th Conference on Methods in Air Pollution and Industrial Hygiene Studies, Oakland, California. February 1967.)
33. Mueller, P. K., et al. Series vs. Parallel Continuous Analysis for NO, NO_2 and NO_x II. Laboratory Data. (9th Conference on Methods in Air Pollution and Industrial Hygiene Studies, Pasadena, California, 1968.)
34. Fontijn, A., A. J. Sabadell, and R. J. Ronco. Homogeneous Chemilumenescent Measurement of Nitric Oxide with Ozone. Anal. Chem., *42*: 575-579, May 1970.
35. Snyder, A. D. and G. W. Wooten. Feasibility Study for the Development of a Multifunctional Emission Detector for NO, CO, and SO_2. Monsanto Research Corporation. Dayton, Ohio. Final Report under National Air Pollution Control Administration Contract No. CPA-22-69-8. 1969.

CHAPTER 6.
ATMOSPHERIC LEVELS OF NITROGEN OXIDES

A. INTRODUCTION

The ambient concentration of oxides of nitrogen (NO_X) varies greatly with time and place. Values reported by the National Air Pollution Control Administration Continuous Air Monitoring Program (CAMP), the State of California's Statewide Cooperative Air Monitoring Network (SCAN), and other special studies since 1961, appear to have several characteristic concentration patterns. The data, which include both day-mean values for different time periods and frequency distributions of individual concentrations, have been evaluated in terms of both temporal and meteorological variables, since these factors, as well as the measurement variables must be considered in a complete description of atmospheric levels of NO_X.

B. TEMPORAL VARIATIONS IN NO_X CONCENTRATIONS

1. Diurnal Patterns

On a normal day in a city, ambient NO_X levels follow a regular pattern with the sun and traffic. Before daylight, nitric oxide (NO) and nitrogen dioxide (NO_2) remain relatively stable at concentrations somewhat higher than the daily minimum. As human activity, especially automotive traffic, increases in the hours just after dawn (6 to 8 a.m.), the concentration of the primary contaminant, NO, increases. Then, as ultraviolet energy from the sun becomes available, NO_2 concentration generally increases until nearly all of the primary NO is converted to secondary NO_2. As the NO concentration approaches very low levels (<0.1 ppm), photochemical oxidants begin to accumulate and reach a peak about midday. Concurrent with NO_2 and oxidant formation, both NO and NO_2 become incorporated into products such as the peroxyacyl nitrates and other nitrated compounds.

As solar intensity decreases, atmospheric stability increases, and winds decrease. In urban areas the increased automobile traffic in late afternoon and evening (5 to 8 p.m.) also increases concentrations of primary NO, but solar energy is generally unavailable to convert NO to NO_2 or to cause formation of oxidants. The absence of sunlight does not completely halt NO_2 formation, however. The principal oxidant present, ozone (O_3), will continue to react rapidly with NO to form NO_2, until the O_3 supply is exhausted, and thus the evening NO_X concentrations may continue to rise. This condition may be ascribed to meteorological factors and, on cold evenings, to increased emissions from stationary sources.

Table 3-1 (chapter 3) shows that transportation sources contribute 39 percent of the national NO_X emissions. This table does not account for the spatial distribution of NO_X sources. In the center of major cities, where most of the air monitoring stations are located, the contribution of mobile sources to NO_X levels may be considerably higher than the reported 39 percent. For this reason, traffic volume, which is highly variable and easily documented, becomes an important tool in identifying and relating NO_X pollution to a recognized emission - automotive exhaust.

The diurnal variations of NO, NO_2, carbon monoxide (CO), and O_3 in Los Angeles, on July 19, 1965, are shown in Figure 6-1. This concentration profile graphically depicts the NO peak and the associated lag in NO_2 peak as the NO is converted. It also illustrates the

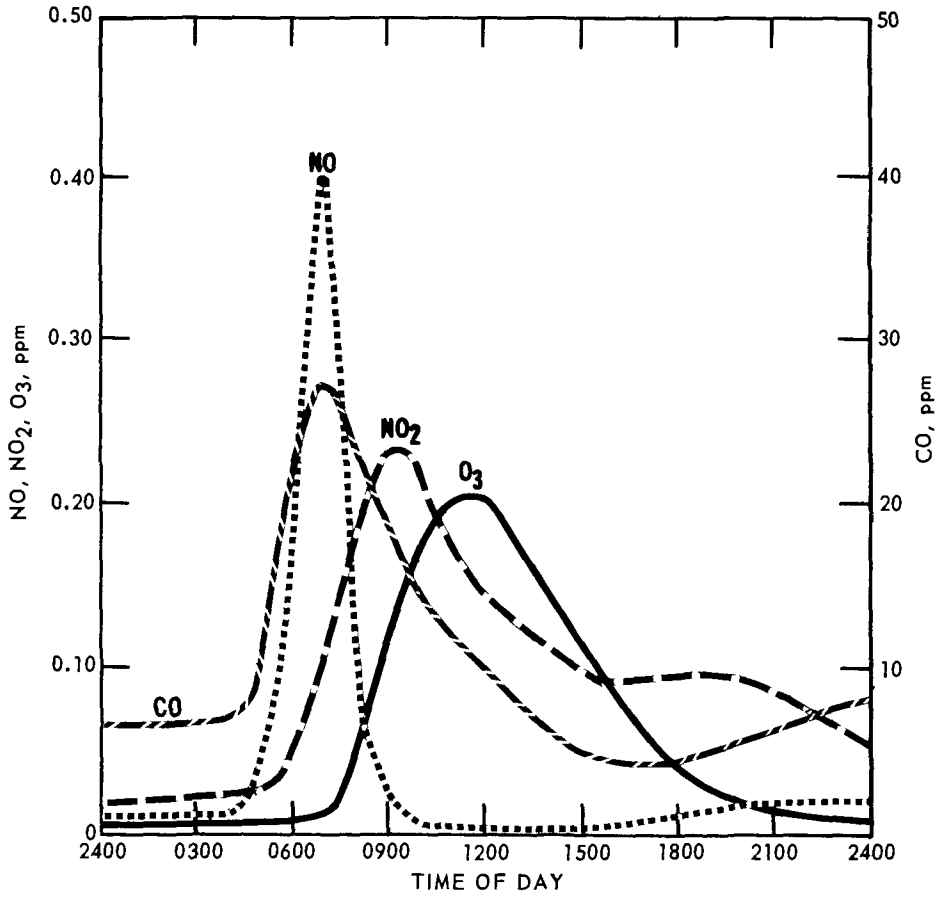


Figure 6-1. Average daily 1-hour concentrations of selected pollutants in Los Angeles, California, July 19, 1965.[8]

subsequent increase in O_3 concentration. The data in Figure 6-1 do not show the generalized increase in evening NO_X levels described in the previous paragraph. On this particular day, evening dispersion factors apparently prevented any substantial buildup of the NO_X.

Other measurements display a similar characteristic profile. Figure 6-2 shows diurnal variations in NO_2 concentration for St. Louis, Philadelphia, and Bayonne, N.J., taken from data gathered by personnel of the Air Pollution Control Office of EPA.

The hours at which the peak concentrations of NO_X usually occur either coincide with, or take place shortly after, the hours of peak automotive traffic. The diurnal pattern, therefore, shows little day-to-day variation except for weekends and holidays, when traffic differs from weekday patterns. The diurnal variations of NO on weekdays, Saturdays, and Sundays are shown for the Chicago CAMP station in Figure 6-3.[1] The Sunday 8-a.m. peak concentrations of NO are about one-third of the weekday peak concentrations. This is but one example of weekend changes. On some weekends, some locations have peak NO concentrations equal to weekday peak values, but occurring 1 to 3 hours later. Furthermore, weekend NO concentrations in certain recreational areas often exceed weekday values.

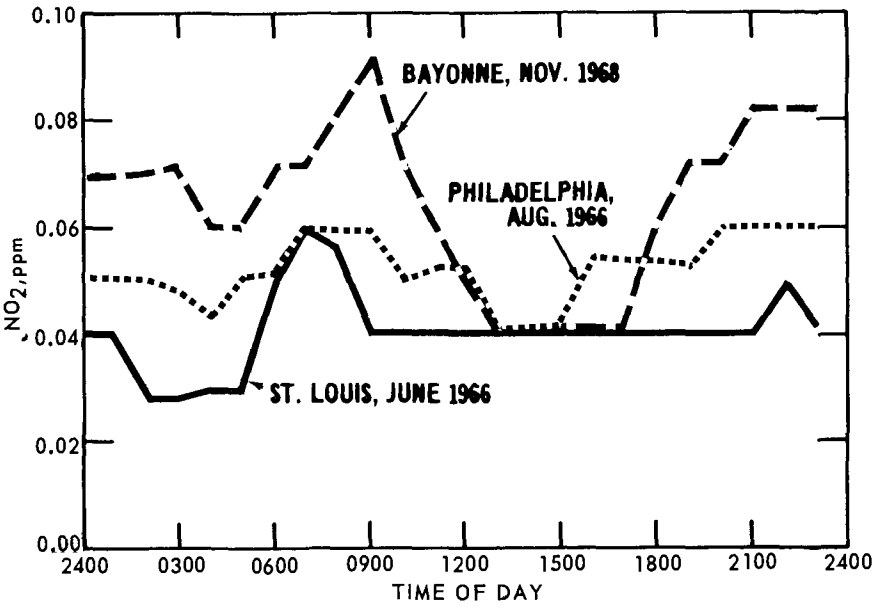


Figure 6-2. Diurnal variation in monthly mean 1-hour-average NO_2 concentrations from three urban stations.[8]

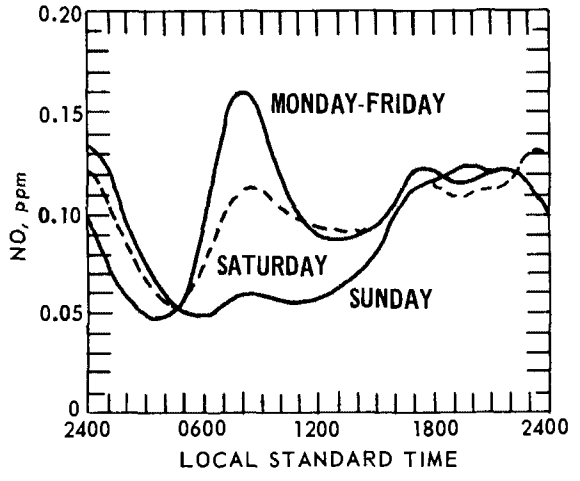


Figure 6-3. Weekday and weekend 1-hour average NO levels in Chicago, 1962 through 1964.[1]

2. Seasonal Patterns

Whereas NO displays a marked seasonal variation, NO_2 does not. For NO, as with most primary contaminants, higher mean values are observed during late fall and winter months, when there is less overall atmospheric mixing and generally less ultraviolet energy available for forming secondary products.

Figure 6-4 shows the seasonal patterns in NO, by presenting the mean values by month of the year, during the time period indicated, for the cities of Chicago, Denver, Bayonne, and Los Angeles. The distinctly higher levels during winter are evident.

The pattern for NO_2 is less distinct, and shows much less variation from month to month. Even though a greater amount of NO is converted to NO_2 during the summer months, the actual concentration of NO_2 may be greatest during other seasons of the year when the NO_2 disappearance rate is slower. The seasonal variation of monthly mean NO_2 values for four cities is shown in Figure 6-5.

The changes in NO_x and NO_2 average-1-hour maximum daily concentrations as a function of the month of the year are shown in Figure 6-6 for the Los Angeles Basin during 1962. Total NO_x varies by a factor of three and is highest during the winter and lowest in

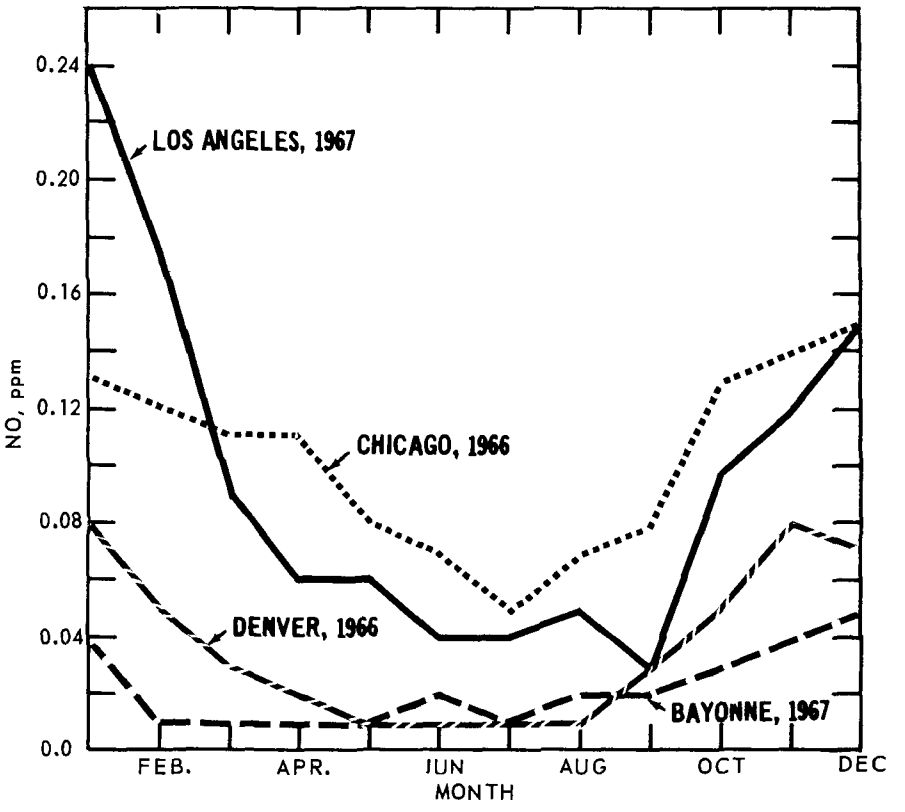


Figure 6-4. Monthly mean NO concentrations at four urban sites.[8]

summer. The high wintertime NO_x values correspond in part to increased NO emissions from power and heating sources. The ratio of NO_2 to NO_x is at a maximum during the summer because of the high conversion rate of NO to NO_2. Variations in dispersion factors further influence NO_2 and NO_x concentrations.

Study of California data has indicated that the same seasonal patterns are observed when the concentrations are expressed as: (1) monthly peak, the highest peak concentration observed during a month; (2) monthly average, the average of all hourly averages during a month; and (3) the monthly average of maximum hourly averages for each day of the month.[2]

3. Annual Trends

The ability to determine possible year-to-year trends in air quality depends upon the extent to which cyclical, seasonal, and random variations are considered. A study by Brier[3] indicates that no adequate long-term data base is presently available for computing cyclical variations. Seasonal factors, on the other hand, are distinct and can be recorded. When data for several years are available, an average "seasonal index" can be calculated and applied to monthly means to obtain monthly values that are corrected for seasonal variations.

The remaining and most troublesome difficulty in determining trends is random variation. Evidence of this is shown in several figures reproduced from a report by Ingels, et al.[4] on atmospheric trends of NO_2. Figure 6-7a shows a least-squares linear-trend line fitted to raw monthly data from downtown Los Angeles, and Figure 6-7b shows a subjectively fitted trend curve applied to monthly data

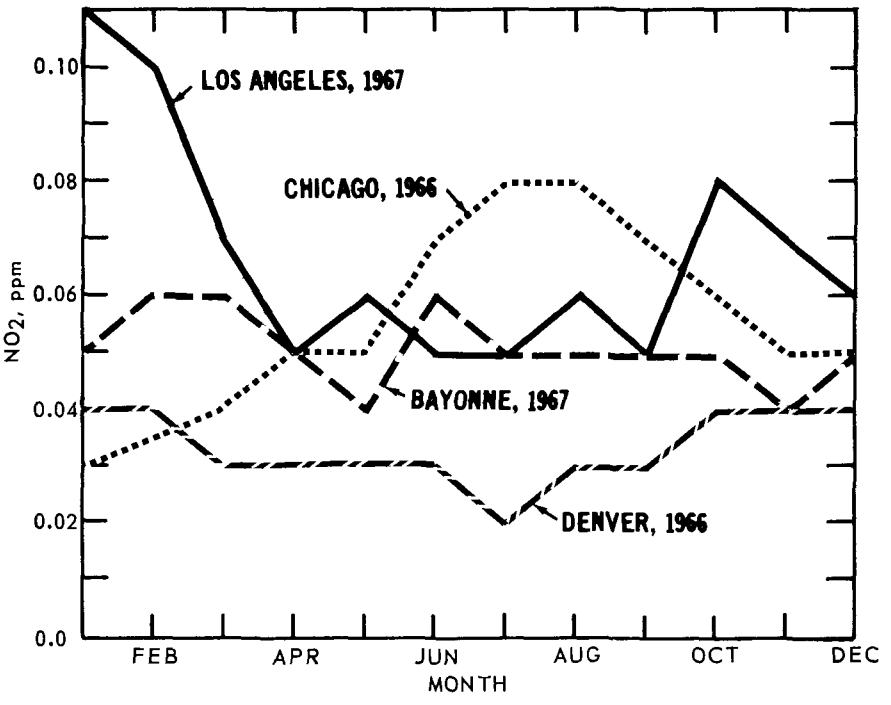


Figure 6-5. Monthly mean NO_2 concentrations at four urban sites.[8]

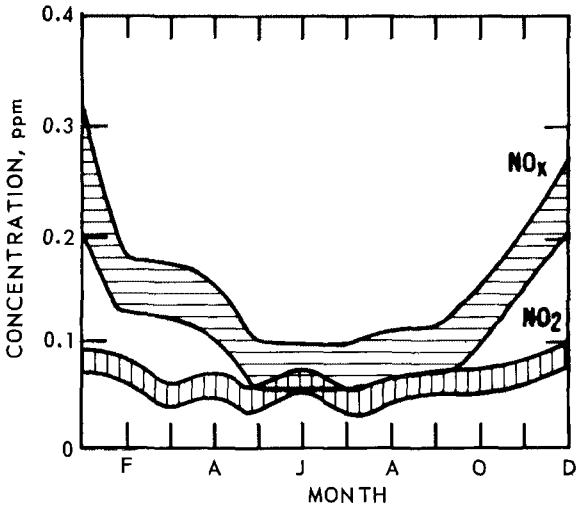


Figure 6-6. Average and standard deviation of daily maximum oxides of nitrogen and nitrogen dioxide concentrations for seven locations in Los Angeles Basin, 1962.[9]

corrected for seasonal variation. Even though NO_X emissions in Los Angeles County increased slightly during the periods considered, no easy interpretation of these data is possible. At this time, there is no clear way to determine the percent of this increase that was due to increased NO_X emissions and the percent due to long-term changes in the meteorological factors that affect ambient concentrations.

Nitrogen dioxide concentrations in several New Jersey cities are shown for the period 1966 through 1968 in Figure 6-8.[5] No significant trends are apparent.

4. Model for Relating Temporal Maxima

It is possible to convert the maximum concentration for one averaging time to that for another time. Larsen[6] has worked out formulas in which he computes the expected maximum concentration (C) for a selected averaging

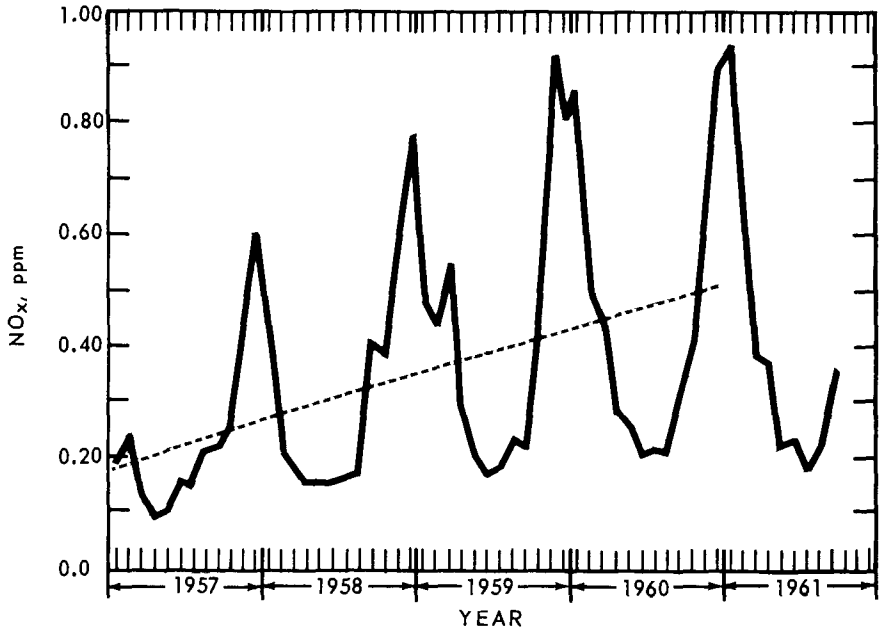


Figure 6-7a. Monthly means of daily maximum NO_X concentrations at Los Angeles Civic Center, 1957 through 1961.[4]

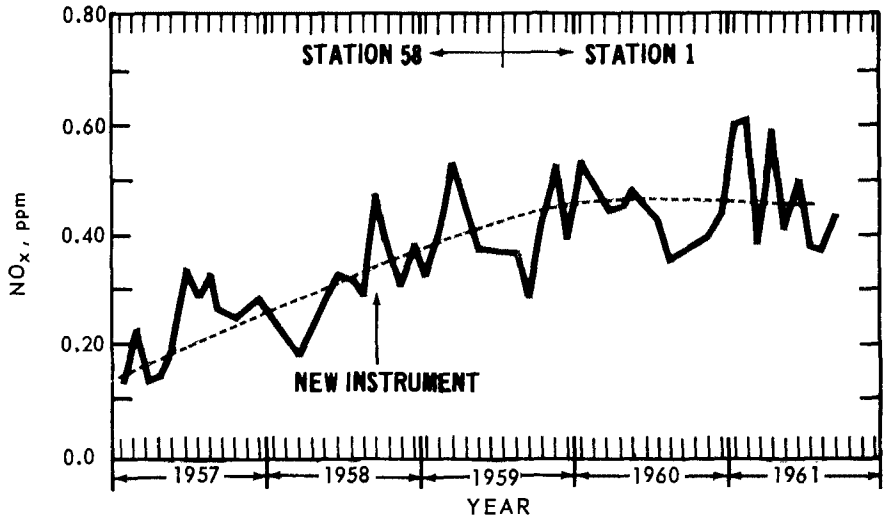


Figure 6-7b. Trend curves fitted to monthly means of daily maximum NO_X concentrations, corrected for seasonal variation, Los Angeles Civic Center, stations 1 and 58, 1957 through 1961.[4]

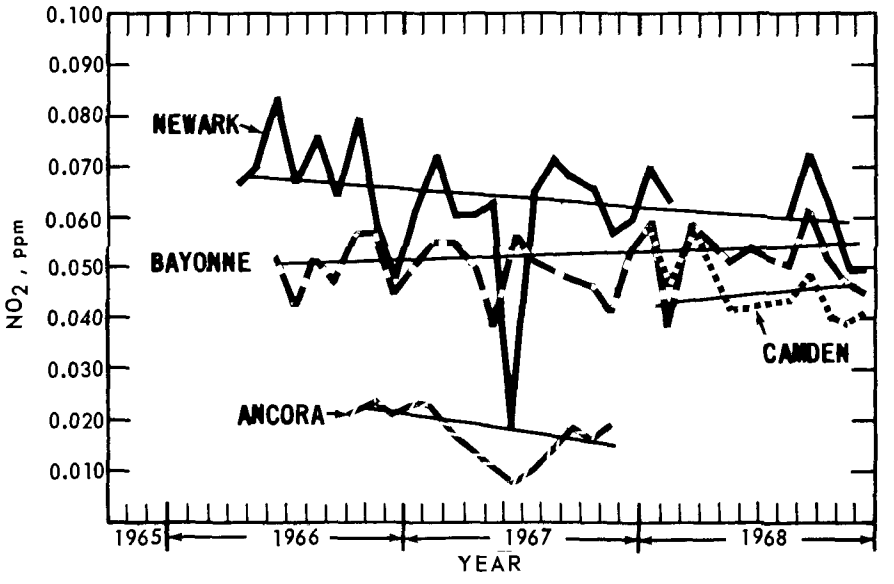


Figure 6-8. Average monthly NO_2 concentrations in four New Jersey communities, 1965 through 1968.[5]

time by using the geometric mean concentration (M_g) for the area in question with its geometric standard deviation (σ_g), and the standard normal deviate (Z) for no more than one occurrence in 1.67 N trials. His formula is:

$$C = M_g(\sigma_g)^Z$$

This formula and the associated mathematical model are based on the assumption that aerometric data are lognormally distributed. The model generally gives good estimates of expected maximum concentrations for averaging times of 1 hour to 1 day, even if aerometric data depart substantially from lognormal. The values of observed-hourly-average concentrations at the 0.1 and 30 percentile levels are used to calculate maximum concentrations for other averaging times. In addition, the model provides predictions of the 1-year concentration. The fit for averaging times from 1 day to about 6 months depends on how well actual aerometric data at a site fit the lognormal assumption. When the aerometric data fit lognormality well, then the model fits well for all averaging times from 1 hour to 1 year. Experience has shown that the model tends to overestimate expected concentrations for averaging times shorter than 1 hour.

C. EFFECT OF METEOROLOGICAL FACTORS

At any given location, the concentration of NO_X is dependent on many factors. Among them are the emission rate; the diffusion rate, which is meteorologically determined; and the reaction rate, which is both meteorologically and chemically determined. All of these factors have an important effect on the distribution of pollution over a city. As with any primary pollutant, concentrations of NO (and therefore, subsequent concentrations of NO_2) on the upwind side of an area of sources are different from concentrations downwind. Spatial variations in concentration make the location of air-monitoring stations important, if representative air quality data are to be collected.

As discussed earlier, the meteorology of an area also influences diurnal and seasonal variations; for example, daily minimum concentrations of NO_X usually occur during daylight

hours, at times of maximum ventilation and mixing. The amount of ultraviolet energy from the sun also governs the conversion rate of NO to NO_2.

From a photochemical standpoint, the highest concentrations of NO_2 should occur during late spring and summer months. In most regions of the United States, radiation inversions during this period are often quite pronounced in the morning hours, but usually break by midday. On the other hand, during the fall and winter, when low sunlight intensity limits maximum photochemical processes, the atmosphere may remain stable for longer periods when the general circulation is weak. Such counteractive effects account for the relatively constant level of NO_2 throughout the year. An additional factor in moderating NO_2 levels is the incorporation of NO_2 into photochemical products. This has the effect of lowering daily average NO_2 values during periods of intense photochemical activity.

The effect of a stagnation period on the peak concentrations of NO and NO_2 is shown in Figures 6-9 and 6-10. The NO rise occurs when minimal dispersion of pollutants, due to stagnating meteorological conditions, coincide with maximum traffic conditions. The sharp midday drop in NO coincides with the NO_2 peak; the sharp NO_2 drop coincides with a total oxidant peak (Figure 6-11). The rapid afternoon rise in both NO and NO_2 is, again, due to stagnating conditions coupled with increases in vehicular traffic.

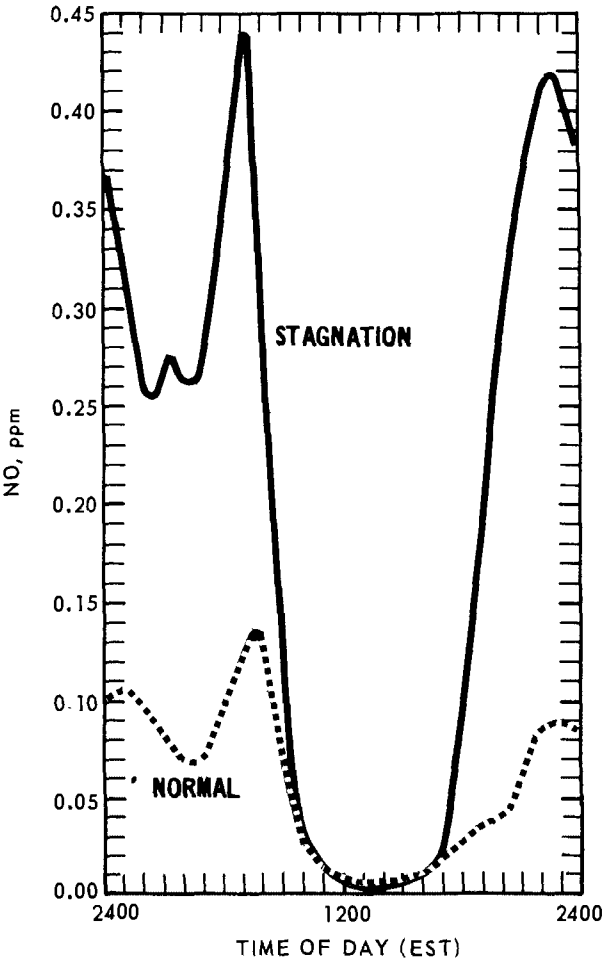


Figure 6-9. Diurnal variation of NO levels during 1963 stagnation in Washington, D.C., October 15 to 19, 1963.[1]

D. OBSERVED URBAN NO_x CONCENTRATIONS

The majority of available continuous air quality data for oxides of nitrogen is less than 15 years old. In 1956, the Los Angeles County Air Pollution Control District (LACAPCD) began monitoring NO and NO_2 continuously. In 1961, the State of California Department of Public Health organized a Statewide Cooperative Air Monitoring Network (SCAN) of 13 stations. Six of these were equipped and operated by the Department; the remaining seven stations were selected from the existing Los Angeles County Network. As of 1968 there were 20 SCAN stations in operation in areas outside the Los Angeles County Air Pollution Control District.

The Public Health Service opened the first station of its Continuous Air Monitoring Program (CAMP) in Cincinnati in October 1961. Subsequently, additional stations equipped to monitor NO and NO_2 have been added throughout the country.

Tables 6-1, 6-2, and 6-3 show NO, NO_2, and total NO_x data from the CAMP network.

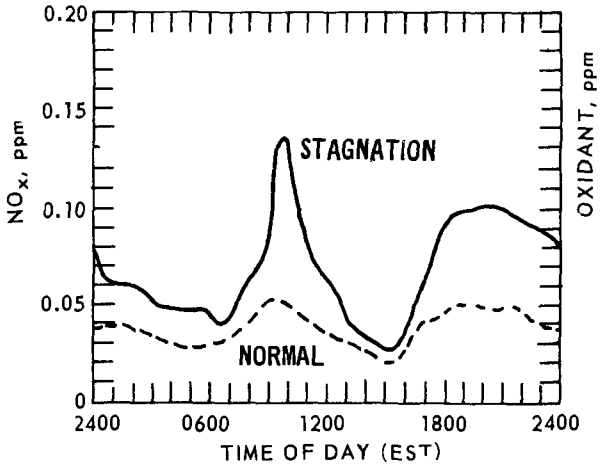


Figure 6-10. Diurnal variation of NO_2 levels during stagnation in Washington, D.C., October 15 through 19, 1963.[1]

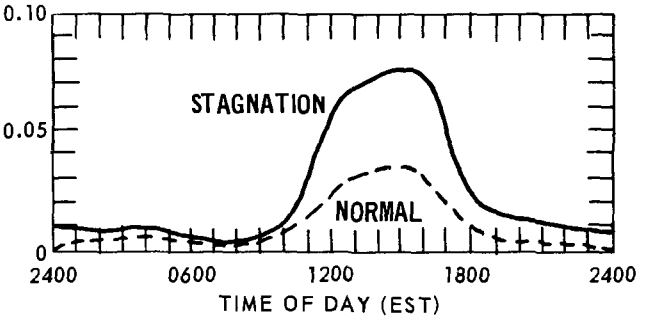


Figure 6-11. Diurnal variation of oxidant levels during stagnation in Washington, D.C., October 15 through 19, 1963.[1]

Similar data for 1963 through 1967 from selected cities in the California SCAN network and the LACAPCD network are in Tables 6-4 through 6-9. The maximum concentrations for each year and the frequency distribution for all years combined are shown for various averaging times.

Peak values (5-minute averages) of NO above 1 ppm are widespread (Table 6-1). Nitrogen dioxide concentrations have rarely been measured at this level. In most major urban areas, peak NO_2 concentrations are under 0.5 ppm (Table 6-2).

Care should be taken in analyzing these yearly data. For instance, the frequency distribution for any averaging time is for up to 7 years of data, thereby masking seasonal and diurnal effects. Figure 6-12 shows a 1964 frequency distribution of NO_x concentrations for a 3-hour averaging time at the CAMP station in Los Angeles. The measurements are differentiated by season. As discussed earlier, the concentrations of NO_x are lowest in the summer (June through August), when the photochemical reactions are the most acute. In the winter (December through February) during the period of stagnating weather and reduced photochemical activity, the concentrations are the highest. Similar CAMP data for other cities show the same pattern.

If yearly NO_x values are used as a basis for control, the degree of NO_x reduction necessary to keep oxidant concentrations below a specified value would be much greater than is justified by the established NO_x - oxidant relationship (chapter 4). When examining ambient NO_x values relative to oxidant values, it is, therefore, necessary to investigate the NO_x concentrations during the summer months, when the oxidant levels are potentially highest. As in the case of the hydrocarbon-oxidant relationship,[7] the time period for measuring NO_x that seems most appropriate is 6 to 9 a.m. This restriction of examination to the summer months' data only applies when the relationship of NO_x to oxidant is being considered. For ambient NO_2, the data from the entire year should be examined.

The seasonal variation in the 6- to 9-a.m. NO_x values is a result of several factors. It is a function of the more intense radiation inversions, which are characteristic of early morning hours during the wintertime. It is also a function of the increased combustion of fuels during the winter season. In Los Angeles, for example, this increased use of fuels produces

Table 6-1. NITRIC OXIDE CONCENTRATION[a] AT CAMP SITES, BY AVERAGING TIME AND FREQUENCY, 1962 THROUGH 1968
(ppm)

City and averaging time	Maximum for year							Data avail., %	% of time concentration is exceeded							
	62	63	64	65	66	67	68		0.01	0.1	1	10	30	50	70	90
Chicago																
5 min	0.66	0.63	0.97	0.67	0.74	0.69	0.66	79	0.67	0.52	0.33	0.18	0.11	0.08	0.05	0.02
1 hr	0.61	0.60	0.91	0.62	0.64	0.63	0.61	79	0.64	0.50	0.32	0.18	0.11	0.08	0.05	0.02
8 hr	0.41	0.39	0.49	0.34	0.42	0.41	0.40	83		0.40	0.27	0.17	0.11	0.08	0.06	0.03
1 day	0.36	0.35	0.35	0.28	0.34	0.26	0.21	86			0.23	0.15	0.11	0.09	0.07	0.04
1 mo	0.17	0.15	0.16	0.13	0.14	0.15	0.11	94				0.13	0.11	0.08	0.07	0.06
1 yr	0.10	0.10	0.10	0.10	0.10	0.08	0.07	100						0.10		
Cincinnati																
5 min	0.60	0.54	0.68	0.63	1.18	1.46	1.26	70	1.08	0.57	0.29	0.09	0.03	0.02	0.01	0.00
1 hr	0.58	0.50	0.65	0.62	1.00	1.38	1.02	71	0.98	0.55	0.29	0.09	0.03	0.02	0.01	0.00
8 hr	0.38	0.22	0.34	0.40	0.46	0.86	0.58	74		0.40	0.24	0.08	0.04	0.02	0.01	0.01
1 day	0.29	0.15	0.31	0.31	0.37	0.36	0.34	77			0.19	0.08	0.04	0.03	0.02	0.01
1 mo	0.06	0.06	0.10	0.07	0.07	0.06	0.08	80				0.06	0.04	0.03	0.02	0.02
1 yr	0.03	0.03	0.04	0.03	0.04	0.03		86						0.03		
Denver																
5 min				0.46	0.59	0.48	0.68	68	0.55	0.42	0.23	0.09	0.04	0.02	0.01	0.00
1 hr				0.40	0.54	0.36	0.61	68	0.52	0.39	0.22	0.09	0.04	0.02	0.01	0.00
8 hr				0.16	0.30	0.19	0.33	72		0.24	0.17	0.08	0.04	0.02	0.02	0.01
1 day				0.14	0.23	0.11	0.21	74			0.13	0.08	0.04	0.03	0.02	0.01
1 mo				0.05	0.08	0.07	0.07	79				0.07	0.05	0.03	0.02	0.01
1 yr				0.03	0.04	0.04	0.04	100						0.04		
Los Angeles																
5 min	2.11	0.97	1.32					72	1.23	0.85	0.54	0.23	0.08	0.03	0.01	0.00
1 hr	1.42	0.79	1.24					72	1.12	0.80	0.53	0.23	0.08	0.03	0.01	0.00
8 hr	0.60	0.49	0.60					78		0.55	0.41	0.21	0.09	0.04	0.02	0.01
1 day	0.46	0.38	0.44					81			0.36	0.19	0.10	0.05	0.03	0.01
1 mo	0.25	0.18	0.19					92				0.16	0.09	0.06	0.03	0.02
1 yr	0.08	0.08	0.07					100						0.07		

Philadelphia																
5 min	1.58	1.72	1.35	1.03	1.98	1.74	1.68	75	1.60	0.95	0.37	0.12	0.05	0.03	0.01	0.00
1 hr	1.57	1.51	1.15	0.89	1.87	1.49	1.44	76	1.49	0.93	0.36	0.12	0.05	0.03	0.01	0.00
8 hr	0.93	0.91	0.52	0.53	1.00	0.72	0.87	80		0.72	0.31	0.11	0.06	0.03	0.02	0.01
1 day	0.46	0.58	0.25	0.31	0.48	0.34	0.37	82			0.25	0.10	0.06	0.04	0.02	0.01
1 mo	0.08	0.08	0.09	0.07	0.13	0.10	0.12	87				0.09	0.06	0.05	0.03	0.02
1 yr	0.04	0.05	0.04	0.04	0.06	0.06	0.06	100						0.04		
St. Louis																
5 min			0.84	0.52	0.61	0.45	0.44	76	0.51	0.33	0.19	0.08	0.04	0.02	0.01	0.00
1 hr			0.75	0.37	0.57	0.41	0.41	76	0.41	0.31	0.18	0.08	0.04	0.02	0.01	0.00
8 hr			0.22	0.17	0.26	0.23	0.18	81		0.22	0.14	0.07	0.04	0.02	0.01	0.01
1 day			0.14	0.12	0.25	0.17	0.13	84			0.10	0.07	0.04	0.03	0.02	0.01
1 mo			0.05	0.06	0.05	0.07	0.05	87				0.05	0.04	0.03	0.03	0.02
1 yr			0.03	0.03	0.03	0.04	0.03	100						0.03		
San Francisco																
5 min	0.50	1.64	0.81					72	1.25	0.64	0.43	0.18	0.09	0.05	0.03	0.01
1 hr	0.42	1.30	0.63					72	0.87	0.60	0.42	0.18	0.09	0.05	0.03	0.01
8 hr	0.26	0.52	0.41					77		0.43	0.31	0.18	0.09	0.06	0.03	0.01
1 day	0.15	0.32	0.23					77			0.22	0.15	0.10	0.07	0.05	0.02
1 mo	0.06	0.11	0.13					83				0.11	0.09	0.08	0.06	0.05
1 yr		0.09	0.09					67						0.09		
Washington																
5 min	0.68	0.73	1.03	0.74	1.15	1.26	0.89	79			0.42	0.10	0.03	0.01	0.01	0.00
1 hr	0.65	0.68	0.88	0.62	1.02	1.14	0.69	80	0.88	0.62	0.33	0.09	0.03	0.01	0.01	0.00
8 hr	0.39	0.51	0.54	0.39	0.55	0.55	0.43	84		0.48	0.27	0.09	0.04	0.02	0.01	0.00
1 day	0.22	0.26	0.29	0.21	0.41	0.36	0.31	86			0.20	0.08	0.04	0.02	0.01	0.01
1 mo	0.07	0.08	0.07	0.06	0.09	0.08	0.10	93				0.07	0.04	0.03	0.02	0.01
1 yr	0.03	0.04	0.03	0.03	0.04	0.04	0.04	100						0.03		

[a]Determined by continuous Griess-Saltzman method.

Table 6-2. NITROGEN DIOXIDE CONCENTRATION[a] AT CAMP SITES, BY AVERAGING TIME AND FREQUENCY, 1962 THROUGH 1968
(ppm)

City and averaging time	Maximum for year							Data avail., %	% of time concentration is exceeded							
	62	63	64	65	66	67	68		0.01	0.1	1	10	30	50	70	90
Chicago																
5 min	0.26	0.23	0.79	0.21	0.35	0.29	0.29	79	0.28	0.18	0.13	0.07	0.05	0.04	0.03	0.02
1 hr	0.22	0.21	0.47	0.18	0.31	0.25	0.18	80	0.25	0.18	0.12	0.07	0.05	0.04	0.03	0.02
8 hr	0.16	0.17	0.22	0.15	0.20	0.15	0.12	84		0.16	0.12	0.07	0.05	0.04	0.04	0.03
1 day	0.12	0.13	0.15	0.09	0.15	0.11	0.10	87			0.10	0.07	0.05	0.04	0.04	0.03
1 mo	0.06	0.06	0.07	0.05	0.08	0.07	0.06	93				0.06	0.05	0.04	0.04	0.03
1 yr	0.04	0.04	0.05	0.04	0.06	0.05	0.05	100						0.04		
Cincinnati																
5 min	0.26	0.30	1.00	0.21	0.30	0.70	1.19	79	0.26	0.16	0.09	0.05	0.04	0.03	0.02	0.02
1 hr	0.23	0.25	0.34	0.17	0.24	0.24	0.56	79	0.24	0.15	0.09	0.05	0.04	0.03	0.02	0.02
8 hr	0.12	0.14	0.14	0.10	0.13	0.13	0.16	83		0.12	0.08	0.05	0.04	0.03	0.02	0.02
1 day	0.07	0.09	0.10	0.08	0.09	0.07	0.10	86			0.07	0.05	0.04	0.03	0.03	0.02
1 mo	0.04	0.04	0.05	0.04	0.05	0.04	0.05	94				0.04	0.04	0.03	0.03	0.02
1 yr	0.03	0.03	0.03	0.03	0.04	0.03	0.03	100						0.03		
Denver																
5 min				0.31	0.30	0.37	0.38	71	0.31	0.18	0.11	0.06	0.04	0.03	0.03	0.02
1 hr				0.28	0.27	0.33	0.25	72	0.27	0.17	0.10	0.06	0.04	0.03	0.03	0.02
8 hr				0.14	0.18	0.17	0.12	76		0.13	0.09	0.05	0.04	0.03	0.03	0.02
1 day				0.09	0.09	0.08	0.12	79			0.08	0.05	0.04	0.03	0.03	0.02
1 mo				0.04	0.04	0.04	0.05	81				0.04	0.04	0.03	0.03	0.03
1 yr				0.04	0.03	0.04	0.04	100						0.04		
Los Angeles																
5 min		0.57	1.27					63	0.66	0.44	0.25	0.12	0.06	0.04	0.03	0.01
1 hr		0.52	0.68					63	0.55	0.40	0.24	0.11	0.06	0.04	0.03	0.01
8 hr		0.37	0.27					67		0.27	0.23	0.11	0.06	0.05	0.03	0.02
1 day		0.20	0.26					71			0.18	0.10	0.07	0.05	0.04	0.02
1 mo		0.10	0.07					79				0.09	0.05	0.04	0.04	0.03
1 yr		0.06	0.05					100						0.05		

Philadelphia																
5 min	0.24	0.36	0.37	0.24	0.29	0.44	0.22	79	0.25	0.18	0.11	0.07	0.04	0.03	0.03	0.02
1 hr	0.22	0.32	0.26	0.20	0.23	0.23	0.20	79	0.23	0.18	0.11	0.07	0.04	0.04	0.03	0.02
8 hr	0.14	0.19	0.14	0.10	0.15	0.13	0.12	84		0.13	0.10	0.06	0.05	0.04	0.03	0.02
1 day	0.10	0.14	0.10	0.07	0.10	0.10	0.09	86			0.08	0.06	0.05	0.04	0.03	0.02
1 mo	0.05	0.06	0.05	0.04	0.05	0.05	0.05	90				0.05	0.04	0.04	0.03	0.03
1 yr	0.04	0.04	0.04	0.04	0.04	0.04	0.04	100						0.04		
St Louis																
5 min			0.31	0.28	0.21	0.17	0.20	81	0.21	0.13	0.08	0.05	0.03	0.03	0.02	0.01
1 hr			0.22	0.13	0.20	0.16	0.18	81	0.20	0.13	0.08	0.05	0.03	0.03	0.02	0.01
8 hr			0.14	0.08	0.14	0.09	0.09	87		0.11	0.07	0.05	0.03	0.03	0.02	0.01
1 day			0.12	0.05	0.10	0.06	0.05	89			0.07	0.04	0.03	0.03	0.02	0.01
1 mo			0.04	0.03	0.04	0.03	0.04	92				0.04	0.03	0.03	0.02	0.02
1 yr			0.04	0.03	0.03	0.03	0.02	100						0.03		
San Francisco																
5 min	0.50	0.38	0.52					70	0.43	0.31	0.18	0.08	0.06	0.04	0.03	0.01
1 hr	0.27	0.33	0.41					70	0.38	0.29	0.18	0.08	0.06	0.04	0.03	0.01
8 hr	0.16	0.23	0.31					75		0.23	0.16	0.08	0.06	0.04	0.03	0.01
1 day	0.10	0.13	0.18					75			0.12	0.07	0.06	0.04	0.03	0.02
1 mo	0.04	0.06	0.08					83				0.07	0.05	0.05	0.04	0.02
1 yr	0.03	0.05	0.06					100						0.03		
Washington																
5 min	0.37	0.24	0.24	0.25	0.19	0.24	0.26	79	0.22	0.16	0.10	0.06	0.04	0.03	0.03	0.02
1 hr	0.30	0.22	0.23	0.23	0.18	0.21	0.24	79	0.21	0.15	0.10	0.06	0.04	0.03	0.03	0.02
8 hr	0.10	0.11	0.14	0.10	0.09	0.14	0.13	84		0.11	0.09	0.06	0.04	0.03	0.03	0.02
1 day	0.07	0.09	0.10	0.07	0.07	0.09	0.08	86			0.08	0.06	0.04	0.04	0.03	0.02
1 mo	0.04	0.04	0.04	0.04	0.04	0.05	0.05	95				0.05	0.04	0.04	0.03	0.03
1 yr	0.03	0.03	0.04	0.03	0.03	0.04	0.05	100						0.03		

[a]Determined by continuous Griess-Saltzman method.

Table 6-3. NITROGEN OXIDES CONCENTRATION[a] AT CAMP SITES, BY AVERAGING TIME AND FREQUENCY, 1962 THROUGH 1968
(ppm)

City and averaging time	Maximum for year							Data avail, %	% of time concentration is exceeded							
	62	63	64	65	66	67	68		0.01	0.1	1	10	30	50	70	90
Chicago																
5 min	0.78	0.72	1.12	0.74	0.86	0.81	0.78	73	0.79	0.62	0.40	0.23	0.16	0.13	0.09	0.06
1 hr	0.69	0.68	1.06	0.68	0.75	0.74	0.73	74	0.75	0.59	0.39	0.23	0.17	0.13	0.09	0.06
8 hr	0.54	0.51	0.62	0.41	0.52	0.46	0.49	78		0.49	0.34	0.22	0.16	0.13	0.10	0.07
1 day	0.42	0.43	0.50	0.34	0.44	0.31	0.30	81			0.30	0.20	0.16	0.13	0.11	0.09
1 mo	0.21	0.18	0.22	0.17	0.19	0.19	0.15	89				0.17	0.15	0.13	0.12	0.10
1 yr	0.14	0.14	0.15	0.14	0.16	0.13	0.12	100						0.14		
Cincinnati																
5 min	0.69	0.49	1.02	0.74	1.35	1.51	1.26	65	1.20	0.66	0.35	0.13	0.07	0.05	0.04	0.02
1 hr	0.68	0.45	0.73	0.73	1.22	1.42	0.95	66	1.03	0.63	0.34	0.13	0.07	0.05	0.04	0.02
8 hr	0.50	0.29	0.51	0.51	0.67	0.90	0.46	69		0.49	0.29	0.12	0.07	0.05	0.04	0.03
1 day	0.37	0.21	0.39	0.39	0.46	0.39	0.32	72			0.23	0.12	0.07	0.06	0.04	0.03
1 mo	0.09	0.10	0.12	0.12	0.11	0.10	0.11	74				0.10	0.07	0.06	0.05	0.05
1 yr	0.06	0.06	0.07	0.07	0.08	0.06		86						0.06		
Denver																
5 min				0.61	0.68	0.62	0.80	61	0.64	0.53	0.31	0.14	0.08	0.05	0.04	0.02
1 hr				0.56	0.62	0.59	0.72	62	0.60	0.50	0.30	0.14	0.08	0.06	0.04	0.02
8 hr				0.27	0.36	0.33	0.44	65		0.33	0.24	0.13	0.08	0.06	0.05	0.03
1 day				0.22	0.28	0.17	0.31	68			0.20	0.13	0.08	0.06	0.05	0.04
1 mo				0.09	0.13	0.11	0.13	63				0.12	0.09	0.06	0.05	0.04
1 yr				0.06	0.07			50						0.06		
Los Angeles																
5 min		1.12	1.49					61	1.28	0.97	0.67	0.31	0.14	0.08	0.05	0.02
1 hr		1.07	1.39					61	1.09	0.97	0.67	0.31	0.14	0.08	0.05	0.02
8 hr		0.71	0.78					64		0.71	0.53	0.29	0.14	0.09	0.05	0.03
1 day		0.54	0.55					68			0.48	0.26	0.15	0.09	0.06	0.04
1 mo		0.28	0.23					79				0.23	0.11	0.09	0.08	0.05
1 yr		0.13	0.12					100						0.12		

Philadelphia																
5 min	1.81	1.93	1.50	1.14	2.16	1.87	1.82	70	1.81	1.04	0.42	0.17	0.10	0.07	0.05	0.03
1 hr	1.79	1.69	1.27	0.97	2.02	1.62	1.61	71	1.69	1.00	0.41	0.17	0.10	0.07	0.05	0.03
8 hr	1.06	1.06	0.45	0.60	1.11	0.79	0.98	75		0.80	0.36	0.16	0.10	0.07	0.05	0.03
1 day	0.56	0.70	0.28	0.38	0.58	0.42	0.44	76			0.29	0.15	0.10	0.08	0.06	0.04
1 mo	0.13	0.12	0.10	0.10	0.18	0.15	0.15	81				0.12	0.09	0.08	0.07	0.06
1 yr	0.08		0.08	0.08	0.10	0.10	0.10	83						0.08		
St Louis																
5 min			1.02	0.59	0.72	0.52	0.52	73	0.61	0.39	0.24	0.12	0.07	0.05	0.03	0.02
1 hr			0.92	0.44	0.66	0.48	0.46	73	0.55	0.37	0.23	0.12	0.07	0.05	0.03	0.02
8 hr			0.26	0.21	0.39	0.28	0.21	78		0.27	0.18	0.11	0.07	0.06	0.04	0.02
1 day			0.25	0.14	0.35	0.22	0.15	82			0.15	0.10	0.07	0.06	0.04	0.03
1 mo			0.08	0.07	0.09	0.08	0.08	85				0.07	0.07	0.06	0.05	0.04
1 yr			0.07	0.05	0.07	0.06	0.06	100						0.06		
San Francisco																
5 min	0.58	1.69	0.91					65	1.31	0.72	0.52	0.26	0.15	0.10	0.06	0.03
1 hr	0.49	1.35	0.76					65	0.92	0.68	0.51	0.26	0.15	0.10	0.06	0.03
8 hr	0.31	0.56	0.52					69		0.49	0.41	0.25	0.15	0.11	0.07	0.03
1 day	0.23	0.36	0.31					68			0.30	0.22	0.15	0.12	0.09	0.06
1 mo	0.10	0.16	0.20					75				0.16	0.14	0.13	0.10	0.07
1 yr		0.14	0.14					67						0.14		
Washington																
5 min	0.79	0.86	1.16	0.84	0.85	1.47	1.00	71	1.00	0.70	0.38	0.14	0.07	0.05	0.03	0.02
1 hr	0.74	0.76	1.00	0.71	0.68	1.30	0.78	71	0.97	0.71	0.38	0.14	0.07	0.05	0.04	0.02
8 hr	0.43	0.58	0.69	0.45	0.41	0.56	0.49	75		0.54	0.31	0.13	0.07	0.05	0.04	0.03
1 day	0.27	0.35	0.38	0.27	0.30	0.45	0.38	78			0.25	0.12	0.08	0.06	0.05	0.03
1 mo	0.11	0.13	0.12	0.10	0.12	0.11	0.14	86				0.10	0.08	0.06	0.05	0.04
1 yr	0.06	0.07	0.07	0.07	0.07	0.08	0.09	100						0.07		

[a]Determined by continuous Griess-Saltzman method.

Table 6-4. NITRIC OXIDE CONCENTRATION[a] IN CALIFORNIA BY AVERAGING TIME AND FREQUENCY, 1963 THROUGH 1967 (ppm)

Place, site No., averaging time	Maximum for year					% of time concentration equaled or exceeded							
	63	64	65	66	67	0.01	0.1	1	10	30	50	70	90
Anaheim–176													
1 hr	0.29	0.30	0.70	0.40	0.66	0.62	0.40	0.22	0.07	0.03	0.01	0.00	0.00
8 hr	0.18	0.19	0.29	0.24	0.41		0.28	0.18	0.07	0.03	0.02	0.01	0.00
1 day	0.11	0.09	0.17	0.15	0.18			0.12	0.06	0.03	0.02	0.01	0.00
1 mo	0.05	0.04	0.05	0.04	0.07				0.06	0.04	0.02	0.02	0.01
1 yr	0.02			0.02	0.04						0.02		
Bakersfield–201													
1 hr	0.34			0.44	0.56	0.55	0.40	0.25	0.09	0.03	0.01	0.00	0.00
8 hr	0.20			0.27	0.36		0.28	0.18	0.08	0.03	0.02	0.00	0.00
1 day	0.12			0.16	0.22			0.14	0.07	0.03	0.02	0.01	0.00
1 mo	0.05			0.07	0.09				0.06	0.04	0.02	0.02	0.01
1 yr				0.02	0.04						0.04		
Fresno–226													
1 hr	0.87	0.47	0.52	0.54	0.59	0.72	0.41	0.22	0.04	0.01	0.00	0.00	0.00
8 hr	0.49	0.22	0.25	0.24	0.30		0.26	0.16	0.04	0.01	0.00	0.00	0.00
1 day	0.21	0.15	0.14	0.12	0.14			0.11	0.04	0.01	0.00	0.00	0.00
1 mo	0.03	0.06	0.03	0.04	0.05				0.04	0.02	0.01	0.00	0.00
1 yr	0.01	0.02	0.02		0.02						0.02		
Oakland–327													
1 hr	0.93	0.93	0.66	0.68	0.91	0.92	0.70	0.41	0.15	0.05	0.02	0.01	0.00
8 hr	0.57	0.60	0.34	0.34	0.52		0.55	0.32	0.14	0.05	0.03	0.01	0.00
1 day	0.33	0.35	0.26	0.26	0.30			0.26	0.13	0.06	0.03	0.02	0.01
1 mo	0.02	0.14	0.11	0.13	0.11				0.11	0.07	0.04	0.02	0.02
1 yr		0.07	0.05	0.05	0.05						0.05		
Port Chicago–429													
1 hr				0.14	0.24	0.18	0.13	0.08	0.03	0.01	0.00	0.00	0.00
8 hr				0.07	0.12		0.10	0.06	0.03	0.01	0.01	0.00	0.00
1 day				0.06	0.07			0.05	0.02	0.01	0.01	0.00	0.00
1 mo				0.02	0.02				0.02	0.01	0.01	0.01	0.00
1 yr				0.01	0.01						0.01		

Richmond–428													
1 hr				0.74	0.58	0.74	0.49	0.27	0.10	0.04	0.02	0.01	0.00
8 hr				0.45	0.35		0.35	0.23	0.10	0.05	0.02	0.01	0.00
1 day				0.20	0.21			0.18	0.09	0.05	0.03	0.01	0.00
1 mo				0.07	0.08				0.08	0.07	0.04	0.02	0.01
1 yr					0.04						0.04		
Riverside–126													
1 hr		1.10	0.57	0.43	0.52	0.74	0.47	0.29	0.11	0.04	0.02	0.01	0.00
8 hr		0.59	0.36	0.26	0.26		0.38	0.21	0.10	0.04	0.02	0.01	0.00
1 day		0.28	0.24	0.20	0.20			0.18	0.09	0.05	0.03	0.01	0.00
1 mo		0.11	0.10	0.08	0.08				0.08	0.05	0.03	0.03	0.01
1 yr			0.04	0.04	0.04						0.04		
Sacramento–276													
1 hr	1.08	1.08	0.97	0.75	0.90	0.97	0.70	0.37	0.09	0.03	0.02	0.00	0.00
8 hr	0.60	0.53	0.62	0.49	0.58		0.54	0.29	0.09	0.03	0.02	0.01	0.00
1 day	0.35	0.26	0.28	0.23	0.33			0.22	0.09	0.04	0.02	0.01	0.00
1 mo	0.14	0.09	0.10	0.07	0.07				0.07	0.05	0.03	0.01	0.01
1 yr	0.04	0.04	0.03	0.04	0.03						0.04		
Salinas–536													
1 hr				0.45	0.29	0.45	0.24	0.15	0.05	0.01	0.00	0.00	0.00
8 hr				0.21	0.15		0.20	0.10	0.04	0.02	0.01	0.00	0.00
1 day				0.10	0.07			0.06	0.04	0.02	0.01	0.00	0.00
1 mo				0.04	0.03				0.03	0.02	0.01	0.01	0.00
1 yr					0.01						0.01		
San Bernadino–151													
1 hr	0.25	0.25	0.34	0.50	0.36	0.47	0.32	0.15	0.05	0.02	0.01	0.00	0.00
8 hr	0.12	0.12	0.16	0.26	0.15		0.25	0.12	0.05	0.02	0.01	0.01	0.00
1 day	0.06	0.06	0.10	0.20	0.11			0.09	0.05	0.03	0.02	0.01	0.00
1 mo	0.03	0.02	0.04	0.07	0.05				0.04	0.03	0.02	0.01	0.00
1 yr	0.01				0.03						0.03		
San Diego–101													
1 hr	0.74	1.10	0.90	1.20	0.80	1.00	0.65	0.38	0.10	0.02	0.01	0.00	0.00
8 hr	0.45	0.63	0.43	0.42	0.34		0.42	0.26	0.10	0.03	0.01	0.00	0.00
1 day	0.24	0.21	0.23	0.26	0.22			0.20	0.10	0.04	0.01	0.00	0.00
1 mo	0.14	0.08	0.09	0.12	0.10				0.09	0.05	0.02	0.01	0.00
1 yr	0.04	0.02	0.03	0.05	0.04						0.04		

Table 6-4 (continued). NITRIC OXIDE CONCENTRATION[a] IN CALIFORNIA BY AVERAGING TIME AND FREQUENCY, 1963 THROUGH 1967.
(ppm)

Place, site No., averaging time	Maximum for year					% of time concentration equaled or exceeded							
	63	64	65	66	67	0.01	0.1	1	10	30	50	70	90
San Diego–106													
1 hr		0.40	0.56	0.34		0.40	0.31	0.21	0.05	0.00	0.00	0.00	0.00
8 hr		0.26	0.22	0.15			0.22	0.13	0.06	0.01	0.00	0.00	0.00
1 day		0.12	0.12	0.08				0.10	0.05	0.02	0.00	0.00	0.00
1 mo		0.05	0.04						0.05	0.02	0.00	0.00	0.00
1 yr		0.02	0.01								0.02		
San Diego–108													
1 hr				0.52	0.60	0.60	0.40	0.20	0.07	0.03	0.01	0.00	0.00
8 hr				0.18	0.29		0.25	0.16	0.07	0.03	0.02	0.01	0.00
1 day				0.14	0.16			0.12	0.06	0.03	0.02	0.01	0.00
1 mo				0.07	0.08				0.05	0.03	0.02	0.02	0.00
1 yr				0.03	0.03						0.03		
San Jose–376													
1 hr	0.75	0.60	0.39	0.42	0.68	0.68	0.54	0.37	0.17	0.07	0.03	0.01	0.00
8 hr	0.48	0.46	0.22	0.25	0.38		0.46	0.29	0.15	0.08	0.04	0.02	0.01
1 day	0.26	0.27	0.17	0.15	0.21			0.25	0.15	0.08	0.04	0.02	0.01
1 mo	0.17	0.14	0.12		0.08				0.12	0.08	0.07	0.02	0.02
1 yr					0.04						0.04		
Santa Barbara–351													
1 hr		0.38	0.72	0.57		0.68	0.51	0.32	0.07	0.01	0.01	0.00	0.00
8 hr		0.19	0.45	0.29			0.40	0.22	0.07	0.02	0.01	0.00	0.00
1 day		0.10	0.21	0.18				0.18	0.08	0.02	0.01	0.00	0.00
1 mo		0.04	0.11	0.10					0.06	0.03	0.01	0.01	0.00
1 yr			0.03								0.03		
Stockton–252													
1 hr	0.38	0.50	0.48	0.87	0.36	0.68	0.47	0.27	0.08	0.02	0.01	0.00	0.00
8 hr	0.18	0.29	0.33	0.50	0.16		0.39	0.23	0.07	0.02	0.01	0.00	0.00
1 day	0.14	0.15	0.23	0.26	0.09			0.19	0.07	0.03	0.01	0.01	0.00
1 mo	0.04	0.06	0.11	0.11	0.02				0.06	0.03	0.02	0.01	0.00
1 yr		0.03	0.03	0.03	0.01						0.03		

[a]Determined by continuous Griess-Saltzman method.

Table 6-5. NITROGEN DIOXIDE CONCENTRATION[a] IN CALIFORNIA BY AVERAGING TIME AND FREQUENCY, 1963 THROUGH 1967
(ppm)

Place, site No., averaging time	Maximum for year					% of time concentration equaled or exceeded							
	63	64	65	66	67	0.01	0.1	1	10	30	50	70	90
Anaheim–176													
1 hr	0.20	0.22	0.23	0.27	0.27	0.25	0.21	0.15	0.07	0.05	0.03	0.02	0.01
8 hr	0.15	0.13	0.16	0.15	0.19		0.18	0.13	0.07	0.04	0.03	0.02	0.01
1 day	0.11	0.11	0.13	0.12	0.13			0.10	0.06	0.04	0.03	0.02	0.01
1 mo	0.06	0.04	0.05	0.05	0.07				0.06	0.05	0.04	0.03	0.02
1 yr	0.03			0.04	0.04						0.04		
Bakersfield–201													
1 hr	0.17			0.15	0.19	0.19	0.14	0.10	0.06	0.04	0.03	0.02	0.01
8 hr	0.10			0.10	0.14		0.13	0.09	0.06	0.04	0.03	0.02	0.01
1 day	0.07			0.08	0.09			0.08	0.05	0.04	0.03	0.03	0.02
1 mo	0.03			0.04	0.05				0.04	0.04	0.03	0.03	0.03
1 yr				0.03	0.04						0.04		
Fresno–226													
1 hr	0.10	0.17	0.25	0.14	0.17	0.22	0.14	0.09	0.04	0.03	0.02	0.01	0.01
8 hr	0.08	0.10	0.18	0.11	0.12		0.13	0.08	0.04	0.03	0.02	0.01	0.01
1 day	0.05	0.08	0.11	0.06	0.07			0.06	0.04	0.03	0.02	0.02	0.01
1 mo	0.03	0.04	0.04	0.04	0.05				0.03	0.03	0.02	0.02	0.02
1 yr	0.02	0.02	0.02	0.03	0.03						0.02		
Oakland–327													
1 hr	0.28	0.41	0.23	0.29	0.33	0.33	0.23	0.14	0.07	0.04	0.03	0.02	0.01
8 hr	0.19	0.25	0.15	0.18	0.23		0.20	0.13	0.06	0.04	0.03	0.02	0.01
1 day	0.10	0.16	0.10	0.13	0.15			0.10	0.06	0.04	0.03	0.02	0.02
1 mo	0.05	0.05	0.06	0.05	0.06				0.05	0.04	0.03	0.03	0.02
1 yr	0.03	0.03	0.03	0.03	0.04						0.03		
Port Chicago–429													
1 hr				0.17	0.14	0.15	0.11	0.08	0.04	0.03	0.02	0.02	0.01
8 hr				0.09	0.07		0.09	0.07	0.04	0.03	0.02	0.02	0.01
1 day				0.07	0.06			0.06	0.04	0.03	0.02	0.02	0.01
1 mo				0.03	0.04				0.03	0.03	0.03	0.02	0.02
1 yr				0.03	0.03						0.03		

[a]Determined by continuous Griess-Saltzman method.

Table 6-5 (continued). NITROGEN DIOXIDE CONCENTRATION[a] IN CALIFORNIA BY AVERAGING TIME AND FREQUENCY, 1963 THROUGH 1967
(ppm)

Place, site No., averaging time	Maximum for year					% of time concentration equaled or exceeded							
	63	64	65	66	67	0.01	0.1	1	10	30	50	70	90
Richmond–428													
1 hr				0.14	0.21	0.21	0.15	0.10	0.06	0.04	0.03	0.02	0.01
8 hr				0.08	0.14		0.10	0.08	0.06	0.04	0.03	0.02	0.01
1 day				0.07	0.10			0.07	0.05	0.04	0.03	0.02	0.02
1 mo				0.03	0.05				0.05	0.04	0.03	0.03	0.02
1 yr					0.03						0.03		
Riverside–126													
1 hr	0.27	0.56	0.49	0.25	0.31	0.49	0.32	0.18	0.09	0.05	0.03	0.02	0.01
8 hr	0.18	0.26	0.31	0.18	0.23		0.29	0.15	0.08	0.05	0.04	0.02	0.01
1 day	0.14	0.19	0.25	0.13	0.17			0.14	0.07	0.05	0.04	0.03	0.02
1 mo	0.06	0.05	0.08	0.05	0.05				0.05	0.05	0.04	0.04	0.03
1 yr		0.04	0.05	0.04	0.04						0.04		
Sacramento–276													
1 hr	0.29	0.32	0.30	0.27	0.30	0.30	0.22	0.14	0.07	0.04	0.03	0.02	0.01
8 hr	0.17	0.21	0.20	0.21	0.20		0.19	0.12	0.06	0.04	0.03	0.02	0.01
1 day	0.13	0.13	0.13	0.15	0.13			0.11	0.06	0.04	0.03	0.02	0.02
1 mo	0.07	0.07	0.07	0.05	0.05				0.05	0.04	0.03	0.03	0.02
1 yr	0.04	0.04	0.04	0.03	0.04						0.04		
Salinas–536													
1 hr				0.11	0.10	0.11	0.09	0.06	0.03	0.02	0.02	0.01	0.01
8 hr				0.07	0.08		0.07	0.05	0.03	0.02	0.02	0.01	0.01
1 day				0.05	0.05			0.04	0.03	0.02	0.02	0.01	0.01
1 mo				0.03	0.02				0.02	0.02	0.02	0.02	0.01
1 yr					0.02						0.02		
San Bernadino–151													
1 hr	0.14	0.06	0.11	0.25	0.22	0.23	0.19	0.13	0.07	0.04	0.02	0.01	0.00
8 hr	0.12	0.03	0.07	0.14	0.17		0.16	0.12	0.06	0.04	0.02	0.01	0.00
1 day	0.06	0.02	0.05	0.11	0.13			0.10	0.06	0.04	0.03	0.01	0.00
1 mo	0.03		0.03	0.06	0.07				0.05	0.04	0.03	0.01	0.00
1 yr	0.01			0.04	0.05						0.04		
San Diego–101													
1 hr	0.33	0.35	0.52	0.40	0.34	0.35	0.22	0.12	0.06	0.03	0.01	0.00	0.00

8 hr	0.18	0.15	0.22	0.19	0.17		0.18	0.11	0.06	0.03	0.02	0.01	0.00
1 day	0.12	0.09	0.12	0.12	0.08			0.09	0.05	0.03	0.02	0.01	0.00
1 mo	0.06	0.06	0.04	0.06	0.03				0.04	0.03	0.02	0.01	0.01
1 yr	0.03	0.02	0.02	0.03	0.02						0.02		
San Diego–106													
1 hr		0.20	0.17	0.15		0.17	0.14	0.10	0.04	0.01	0.00	0.00	0.00
8 hr		0.11	0.09	0.11			0.11	0.09	0.04	0.01	0.00	0.00	0.00
1 day		0.09	0.05	0.07				0.07	0.03	0.01	0.00	0.00	0.00
1 mo		0.05	0.02						0.02	0.02	0.01	0.00	0.00
1 yr		0.01	0.01								0.01		
San Diego–108													
1 hr				0.37	0.21	0.35	0.25	0.13	0.06	0.03	0.02	0.01	0.00
8 hr				0.25	0.13		0.18	0.12	0.05	0.03	0.02	0.01	0.00
1 day				0.14	0.08			0.10	0.05	0.03	0.02	0.02	0.01
1 mo				0.06	0.04				0.05	0.03	0.02	0.02	0.01
1 yr				0.03	0.02						0.03		
San Jose–376													
1 hr	0.30	0.39	0.23	0.27	0.27	0.34	0.29	0.18	0.09	0.05	0.04	0.03	0.01
8 hr	0.19	0.31	0.16	0.20	0.18		0.24	0.16	0.09	0.05	0.04	0.03	0.02
1 day	0.13	0.18	0.12	0.16	0.12			0.13	0.08	0.05	0.04	0.03	0.02
1 mo	0.07	0.07	0.07	0.06	0.07				0.07	0.05	0.04	0.04	0.03
1 yr		0.05		0.04	0.04						0.04		
Santa Barbara–351													
1 hr		0.18	0.31	0.13		0.28	0.14	0.08	0.05	0.03	0.02	0.02	0.01
8 hr		0.12	0.13	0.08			0.12	0.07	0.05	0.03	0.02	0.02	0.01
1 day		0.09	0.08	0.06				0.07	0.04	0.03	0.02	0.02	0.01
1 mo		0.03	0.04	0.03					0.03	0.03	0.03	0.02	0.02
1 yr		0.02	0.03								0.03		
Santa Cruz–841													
1 hr				0.12	0.09	0.12	0.08	0.06	0.03	0.02	0.01	0.01	0.01
8 hr				0.07	0.06		0.06	0.05	0.03	0.02	0.02	0.01	0.01
1 day				0.04	0.04			0.04	0.03	0.02	0.02	0.01	0.01
1 mo				0.02	0.02				0.02	0.02	0.02	0.02	0.01
1 yr					0.02						0.02		
Stockton–252													
1 hr	0.13	0.22	0.14	0.16	0.18	0.20	0.16	0.09	0.04	0.02	0.02	0.01	0.01
8 hr	0.08	0.15	0.11	0.11	0.10		0.12	0.07	0.04	0.03	0.02	0.01	0.01
1 day	0.06	0.08	0.07	0.07	0.06			0.06	0.04	0.03	0.02	0.02	0.01
1 mo	0.02	0.05	0.03	0.03	0.03				0.03	0.02	0.02	0.02	0.01
1 yr		0.03	0.02	0.02	0.02						0.02		

[a]Determined by continuous Griess-Saltzman method.

Table 6-6. NITROGEN OXIDES CONCENTRATION[a] IN CALIFORNIA, BY AVERAGING TIME AND FREQUENCY, 1963 THROUGH 1967.
(ppm)

Place, site No., averaging time	Maximum for year					% of time concentration equaled or exceeded							
	63	64	65	66	67	0.01	0.1	1	10	30	50	70	90
Anaheim–176													
1 hr	0.42	0.35	0.80	0.53	0.76	0.72	0.50	0.30	0.13	0.07	0.05	0.03	0.01
8 hr	0.30	0.23	0.36	0.37	0.50		0.38	0.26	0.12	0.07	0.05	0.04	0.02
1 day	0.22	0.17	0.22	0.24	0.27			0.20	0.12	0.07	0.05	0.04	0.02
1 mo	0.11	0.09	0.09	0.10	0.14				0.11	0.08	0.06	0.05	0.02
1 yr	0.05			0.06	0.08						0.06		
Bakersfield–201													
1 hr	0.42		0.32	0.52	0.64	0.59	0.48	0.30	0.13	0.07	0.04	0.03	0.01
8 hr	0.25		0.17	0.30	0.40		0.34	0.24	0.12	0.07	0.05	0.03	0.02
1 day	0.15		0.15	0.19	0.25			0.19	0.11	0.07	0.05	0.04	0.03
1 mo	0.08			0.10	0.13				0.10	0.07	0.05	0.05	0.04
1 yr				0.06	0.07						0.07		
Fresno–226													
1 hr	0.93	0.52	0.55	0.60	0.66	0.70	0.48	0.27	0.08	0.03	0.02	0.01	0.01
8 hr	0.54	0.27	0.30	0.31	0.38		0.30	0.22	0.08	0.03	0.02	0.02	0.01
1 day	0.24	0.19	0.16	0.17	0.21			0.16	0.08	0.04	0.03	0.02	0.01
1 mo	0.05	0.09	0.07	0.07	0.09				0.07	0.05	0.03	0.02	0.02
1 yr	0.03	0.04	0.04	0.04	0.04						0.04		
Oakland–327													
1 hr	0.95	1.26	0.74	0.79	1.06	1.03	0.82	0.49	0.20	0.09	0.05	0.04	0.02
8 hr	0.58	0.74	0.40	0.45	0.64		0.62	0.39	0.20	0.10	0.06	0.04	0.02
1 day	0.36	0.40	0.33	0.34	0.41			0.33	0.19	0.10	0.06	0.04	0.03
1 mo	0.23	0.19	0.15	0.17	0.15				0.15	0.12	0.07	0.05	0.04
1 yr		0.11	0.08	0.08	0.09						0.09		
Port Chicago–429													
1 hr				0.19	0.28	0.23	0.17	0.12	0.07	0.04	0.03	0.02	0.01
8 hr				0.12	0.15		0.15	0.10	0.06	0.04	0.03	0.02	0.01
1 day				0.10	0.11			0.09	0.06	0.04	0.03	0.03	0.02
1 mo				0.05	0.06				0.05	0.04	0.04	0.03	0.02
1 yr				0.04	0.04						0.04		

Richmond–428													
1 hr				0.80	0.90	0.90	0.59	0.33	0.15	0.08	0.05	0.03	0.02
8 hr				0.50	0.50		0.50	0.28	0.14	0.08	0.06	0.03	0.02
1 day				0.25	0.26			0.22	0.13	0.09	0.06	0.04	0.02
1 mo				0.10	0.14				0.12	0.10	0.07	0.05	0.02
1 yr					0.07						0.07		
Riverside–126													
1 hr		0.16	0.76	0.55	0.67	0.91	0.64	0.40	0.19	0.10	0.06	0.03	0.01
8 hr		0.65	0.48	0.38	0.41		0.56	0.31	0.17	0.10	0.06	0.04	0.02
1 day		0.36	0.39	0.24	0.28			0.29	0.15	0.10	0.07	0.05	0.03
1 mo		0.16	0.15	0.13	0.13				0.13	0.09	0.08	0.06	0.05
1 yr			0.08	0.07	0.09						0.08		
Sacramento–276													
1 hr	1.19	1.22	1.05	1.00	1.14	1.11	0.81	0.48	0.14	0.07	0.05	0.03	0.02
8 hr	0.70	0.64	0.68	0.62	0.78		0.67	0.38	0.14	0.07	0.05	0.04	0.02
1 day	0.43	0.34	0.38	0.38	0.34			0.30	0.14	0.08	0.05	0.04	0.03
1 mo	0.19	0.15	0.17	0.12	0.12				0.12	0.10	0.06	0.04	0.04
1 yr	0.08	0.08	0.07	0.06	0.07						0.07		
Salinas–536													
1 hr				0.51	0.32	0.51	0.27	0.18	0.07	0.03	0.02	0.01	0.01
8 hr				0.23	0.18		0.21	0.13	0.07	0.04	0.02	0.02	0.01
1 day				0.13	0.09			0.09	0.06	0.04	0.03	0.02	0.02
1 mo				0.06	0.05				0.05	0.04	0.04	0.02	0.02
1 yr					0.03						0.03		
San Bernadino–151													
1 hr	0.25	0.25	0.39	0.57	0.48	0.56	0.42	0.24	0.11	0.06	0.03	0.02	0.00
8 hr	0.12	0.12	0.21	0.37	0.28		0.33	0.20	0.10	0.06	0.04	0.02	0.00
1 day	0.07	0.06	0.13	0.28	0.22			0.17	0.10	0.06	0.04	0.02	0.00
1 mo	0.04	0.02	0.07	0.14	0.10				0.10	0.06	0.04	0.02	0.01
1 yr	0.02				0.08						0.08		
San Diego–101													
1 hr	0.96	1.18	0.98	1.42	0.84	1.05	0.75	0.45	0.15	0.05	0.02	0.01	0.00
8 hr	0.56	0.69	0.51	0.52	0.44		0.51	0.33	0.15	0.06	0.03	0.01	0.00
1 day	0.34	0.30	0.29	0.36	0.28			0.29	0.14	0.07	0.03	0.02	0.00
1 mo	0.21	0.12	0.13	0.19	0.14				0.13	0.08	0.05	0.03	0.01
1 yr	0.06	0.04	0.05	0.08	0.05						0.05		

[a]Determined by continuous Griess-Saltzman method.

Table 6-6 (continued). NITROGEN OXIDES CONCENTRATION[a] IN CALIFORNIA BY AVERAGING TIME AND FREQUENCY, 1963 THROUGH 1967.
(ppm)

Place, site No., averaging time	Maximum for year					% of time concentration equaled or exceeded							
	63	64	65	66	67	0.01	0.1	1	10	30	50	70	90
San Diego–106													
1 hr		0.44	0.97	0.42		0.59	0.40	0.27	0.09	0.02	0.00	0.00	0.00
8 hr		0.29	0.24	0.20			0.24	0.19	0.09	0.03	0.01	0.00	0.00
1 day		0.18	0.13	0.14				0.16	0.09	0.03	0.01	0.00	0.00
1 mo		0.10	0.05						0.09	0.05	0.01	0.00	0.00
1 yr		0.03	0.02								0.03		
San Diego–108													
1 hr				0.63	0.63	0.63	0.52	0.29	0.12	0.06	0.04	0.02	0.01
8 hr				0.31	0.30		0.30	0.24	0.12	0.06	0.04	0.02	0.01
1 day				0.24	0.21			0.19	0.12	0.06	0.04	0.03	0.01
1 mo				0.14	0.13				0.09	0.07	0.05	0.04	0.02
1 yr				0.06	0.05						0.06		
San Jose–376													
1 hr	0.89	0.89	0.52	0.50	0.87	0.89	0.70	0.46	0.22	0.12	0.08	0.05	0.03
8 hr	0.65	0.77	0.29	0.30	0.45		0.65	0.38	0.21	0.12	0.08	0.05	0.03
1 day	0.39	0.43	0.24	0.21	0.29			0.33	0.20	0.13	0.08	0.06	0.04
1 mo	0.24	0.21	0.17	0.09	0.15				0.17	0.14	0.10	0.07	0.05
1 yr					0.09						0.09		
Santa Barbara–351													
1 hr		0.47	0.76	0.65		0.74	0.56	0.37	0.11	0.05	0.03	0.02	0.01
8 hr		0.23	0.49	0.34			0.41	0.27	0.11	0.05	0.03	0.02	0.01
1 day		0.13	0.24	0.23				0.22	0.11	0.05	0.04	0.02	0.02
1 mo		0.06	0.14	0.13					0.09	0.06	0.04	0.03	0.02
1 yr			0.05								0.05		
Santa Cruz–841													
1 hr				0.17	0.20	0.20	0.16	0.10	0.05	0.02	0.02	0.01	0.01
8 hr				0.09	0.12		0.11	0.08	0.04	0.03	0.02	0.01	0.01
1 day				0.06	0.06			0.05	0.04	0.03	0.02	0.02	0.01
1 mo				0.03	0.03				0.03	0.03	0.03	0.02	0.01
1 yr					0.02						0.02		

Stockton–252													
1 hr	0.45	0.68	0.53	0.90	0.42	0.71	0.52	0.31	0.10	0.04	0.03	0.02	0.01
8 hr	0.23	0.39	0.36	0.52	0.21		0.43	0.25	0.11	0.05	0.03	0.02	0.01
1 day	0.17	0.18	0.24	0.28	0.12			0.21	0.10	0.05	0.03	0.03	0.02
1 mo	0.06	0.09	0.12	0.13	0.05				0.09	0.05	0.04	0.03	0.02
1 yr		0.06	0.05	0.05	0.04						0.05		
Ventura–401													
1 hr	0.26	0.28	0.45	0.39		0.42	0.26	0.17	0.09	0.05	0.03	0.02	0.01
8 hr	0.15	0.16	0.18	0.21			0.18	0.13	0.08	0.05	0.03	0.02	0.01
1 day	0.11	0.11	0.13	0.12				0.11	0.07	0.05	0.04	0.03	0.02
1 mo		0.05	0.06	0.06					0.06	0.04	0.04	0.03	0.03
1 yr		0.04	0.04								0.04		

[a]Determined by continuous Griess-Saltzman method.

Table 6-7. NITRIC OXIDE CONCENTRATION[a] IN LOS ANGELES COUNTY BY AVERAGING TIME AND FREQUENCY, 1963 THROUGH 1967
(ppm)

Place, site No., averaging time	Maximum for year					% of time concentration equaled or exceeded							
	63	64	65	66	67	0.01	0.1	1	10	30	50	70	90
Azusa–60													
1 hr	0.42	0.21	0.24	0.26	0.40	0.36	0.20	0.11	0.04	0.01	0.01	0.01	0.01
8 hr	0.14	0.13	0.13	0.17	0.17		0.15	0.08	0.04	0.02	0.01	0.01	0.01
1 day	0.07	0.07	0.08	0.08	0.12			0.06	0.03	0.02	0.01	0.01	0.01
1 mo	0.03	0.03	0.02	0.03	0.03				0.03	0.02	0.02	0.02	0.01
1 yr	0.02	0.02	0.02	0.02	0.02						0.02		
Burbank–69													
1 hr	1.14	0.72	1.02	0.90	0.90	0.94	0.75	0.56	0.29	0.10	0.03	0.01	0.01
8 hr	0.55	0.49	0.72	0.56	0.60		0.56	0.45	0.26	0.11	0.05	0.02	0.01
1 day	0.42	0.37	0.49	0.43	0.42			0.37	0.24	0.12	0.06	0.03	0.01
1 mo	0.26	0.16	0.21	0.22	0.23				0.21	0.14	0.08	0.04	0.03
1 yr	0.10	0.08	0.10	0.09	0.10						0.10		
Hywd Freeway–73													
1 hr	0.81	1.15	0.95	0.95	0.83	0.95	0.80	0.62	0.35	0.17	0.08	0.04	0.01
8 hr	0.57	0.72	0.71	0.62	0.63		0.64	0.48	0.31	0.18	0.10	0.05	0.03
1 day	0.39	0.49	0.51	0.43	0.38			0.42	0.30	0.18	0.11	0.07	0.04
1 mo	0.28	0.29	0.30	0.27	0.29				0.28	0.18	0.13	0.07	0.05
1 yr	0.12	0.14	0.14	0.14							0.14		
Los Angeles–1													
1 hr	0.85	1.30	1.15	0.83	1.15	1.10	0.80	0.55	0.26	0.09	0.03	0.01	0.01
8 hr	0.44	0.62	0.48	0.51	0.69		0.56	0.40	0.22	0.10	0.05	0.02	0.01
1 day	0.41	0.45	0.34	0.34	0.42			0.34	0.21	0.11	0.06	0.03	0.02
1 mo	0.27	0.22	0.17	0.19	0.24				0.18	0.11	0.07	0.04	0.03
1 yr	0.10	0.08	0.08	0.09	0.10						0.09		
Los Angeles–70													
1 hr	0.57	0.81	0.60	0.69	0.61	0.68	0.48	0.32	0.15	0.07	0.03	0.01	0.01
8 hr	0.34	0.45	0.37	0.44	0.31		0.36	0.24	0.13	0.07	0.04	0.02	0.01
1 day	0.21	0.29	0.24	0.26	0.21			0.20	0.12	0.07	0.05	0.03	0.01
1 mo	0.11	0.13	0.12	0.12	0.13				0.11	0.07	0.05	0.04	0.03
1 yr	0.06	0.06	0.06	0.06	0.06						0.06		

N Long Beach–72													
1 hr	0.93	1.25	0.75	0.80	1.05	1.05	0.77	0.53	0.27	0.12	0.06	0.03	0.01
8 hr	0.65	0.83	0.44	0.54	0.61		0.64	0.44	0.25	0.12	0.07	0.04	0.02
1 day	0.47	0.59	0.33	0.37	0.43			0.40	0.25	0.13	0.07	0.04	0.03
1 mo	0.31	0.28	0.21	0.20	0.23				0.21	0.14	0.08	0.05	0.03
1 yr	0.11	0.11	0.09	0.10	0.11						0.11		
Pasadena–64													
1 hr	0.69	0.62	0.42	0.50	0.53	0.62	0.49	0.33	0.13	0.05	0.02	0.01	0.01
8 hr	0.39	0.37	0.32	0.28	0.32		0.33	0.24	0.11	0.05	0.03	0.02	0.01
1 day	0.25	0.23	0.16	0.17	0.20			0.19	0.11	0.06	0.04	0.03	0.01
1 mo	0.15	0.13	0.09	0.10	0.10				0.10	0.06	0.04	0.03	0.02
1 yr	0.06	0.05	0.04	0.05	0.05						0.05		
Pomona–75													
1 hr			0.47	0.45	0.62	0.60	0.45	0.33	0.16	0.08	0.04	0.02	0.01
8 hr			0.29	0.31	0.34		0.32	0.25	0.15	0.08	0.04	0.02	0.01
1 day			0.18	0.19	0.24			0.20	0.14	0.08	0.05	0.03	0.02
1 mo			0.10	0.12	0.15				0.12	0.07	0.06	0.04	0.03
1 yr				0.06	0.08						0.08		
Lennox–76													
1 hr			1.02	1.75	1.80	1.65	1.29	0.80	0.36	0.12	0.04	0.02	0.01
8 hr			0.68	1.06	1.40		1.06	0.66	0.31	0.14	0.06	0.03	0.02
1 day			0.55	0.70	0.94			0.55	0.29	0.14	0.08	0.04	0.02
1 mo			0.26	0.30	0.31				0.26	0.14	0.09	0.06	0.04
1 yr			0.10	0.12	0.15						0.12		
Reseda–74													
1 hr			0.91	0.78	0.84	0.84	0.70	0.47	0.18	0.05	0.01	0.01	0.01
8 hr			0.59	0.53	0.50		0.51	0.38	0.16	0.06	0.02	0.01	0.01
1 day			0.41	0.31	0.32			0.29	0.16	0.06	0.04	0.02	0.01
1 mo			0.15	0.14	0.14				0.14	0.07	0.04	0.03	0.02
1 yr			0.05	0.06	0.07						0.06		
West LA–71													
1 hr	0.86	0.95	1.05	1.06	1.08	1.02	0.80	0.52	0.21	0.07	0.02	0.01	0.01
8 hr	0.66	0.50	0.73	0.63	0.57		0.58	0.37	0.19	0.08	0.04	0.01	0.01
1 day	0.44	0.33	0.41	0.36	0.36			0.32	0.18	0.09	0.05	0.02	0.01
1 mo	0.20	0.16	0.19	0.14	0.19				0.16	0.09	0.06	0.03	0.02
1 yr	0.08	0.07	0.07	0.07	0.07						0.07		

[a]Determined by continuous Griess-Saltzman method.

Table 6-8. NITROGEN DIOXIDE CONCENTRATION[a] IN LOS ANGELES COUNTY BY AVERAGING TIME AND FREQUENCY, 1963 THROUGH 1967
(ppm)

Place, site No., averaging time	Maximum for year					% of time concentration equaled or exceeded							
	63	64	65	66	67	0.01	0.1	1	10	30	50	70	90
Azusa–60													
1 hr	0.36	0.21	0.30	0.31	0.32	0.32	0.23	0.16	0.09	0.06	0.04	0.02	0.01
8 hr	0.22	0.15	0.24	0.20	0.25		0.20	0.14	0.08	0.06	0.04	0.03	0.01
1 day	0.11	0.12	0.16	0.14	0.15			0.12	0.08	0.05	0.04	0.03	0.02
1 mo	0.05	0.07	0.06	0.06	0.07				0.06	0.05	0.04	0.04	0.03
1 yr	0.04	0.05	0.04	0.05	0.05						0.05		
Burbank–69													
1 hr	0.39	0.30	0.47	0.40	0.56	0.51	0.38	0.26	0.13	0.08	0.06	0.04	0.02
8 hr	0.33	0.23	0.34	0.36	0.42		0.36	0.23	0.13	0.08	0.06	0.04	0.02
1 day	0.24	0.17	0.23	0.23	0.31			0.21	0.12	0.08	0.06	0.04	0.03
1 mo	0.11	0.08	0.10	0.10	0.13				0.10	0.08	0.06	0.06	0.04
1 yr	0.06	0.06	0.07	0.07	0.09						0.07		
Hywd Freeway–73													
1 hr	0.39	0.60	0.60	0.48	0.42	0.55	0.36	0.25	0.14	0.08	0.06	0.05	0.03
8 hr	0.26	0.36	0.32	0.28	0.30		0.30	0.22	0.14	0.09	0.07	0.05	0.03
1 day	0.22	0.23	0.20	0.19	0.25			0.18	0.12	0.09	0.07	0.06	0.04
1 mo	0.11	0.10	0.11	0.10	0.11				0.10	0.08	0.07	0.07	0.06
1 yr	0.07	0.08	0.08	0.08							0.08		
Los Angeles–1													
1 hr	0.55	0.70	0.70	0.80	0.54	0.70	0.42	0.26	0.13	0.08	0.05	0.04	0.02
8 hr	0.30	0.33	0.35	0.33	0.33		0.33	0.23	0.13	0.08	0.06	0.04	0.03
1 day	0.22	0.26	0.24	0.26	0.29			0.19	0.12	0.08	0.06	0.04	0.03
1 mo	0.11	0.09	0.11	0.11	0.11				0.10	0.08	0.06	0.05	0.04
1 yr	0.06	0.06	0.07	0.08	0.07						0.07		
Los Angeles–70													
1 hr	0.50	0.44	0.57	0.48	0.48	0.50	0.40	0.26	0.12	0.07	0.06	0.04	0.02
8 hr	0.37	0.29	0.36	0.35	0.39		0.36	0.23	0.12	0.08	0.06	0.04	0.02
1 day	0.24	0.19	0.23	0.19	0.28			0.19	0.11	0.08	0.06	0.05	0.03
1 mo	0.12	0.09	0.12	0.10	0.13				0.10	0.07	0.06	0.06	0.05
1 yr	0.07	0.07	0.07	0.06	0.07						0.07		

N Long Beach–72													
1 hr	0.57	0.59		0.42	0.46	0.50	0.35	0.24	0.13	0.07	0.05	0.03	0.02
8 hr	0.38	0.43		0.28	0.33		0.29	0.21	0.12	0.07	0.05	0.04	0.02
1 day	0.28	0.27		0.17	0.21			0.19	0.11	0.08	0.05	0.04	0.03
1 mo	0.14	0.10		0.09	0.13				0.09	0.08	0.06	0.05	0.04
1 yr	0.06	0.06		0.06	0.08						0.06		
Pasadena–64													
1 hr	0.33	0.36	0.34	0.33	0.37	0.34	0.28	0.20	0.11	0.07	0.05	0.04	0.02
8 hr	0.23	0.22	0.27	0.27	0.27		0.23	0.18	0.10	0.07	0.05	0.04	0.02
1 day	0.15	0.15	0.15	0.16	0.16			0.13	0.09	0.07	0.05	0.04	0.03
1 mo	0.08	0.08	0.07	0.07	0.09				0.08	0.06	0.06	0.05	0.04
1 yr	0.05	0.06	0.06	0.06	0.06						0.06		
Pomona–75													
1 hr			0.29	0.29	0.37	0.36	0.27	0.20	0.12	0.08	0.06	0.04	0.03
8 hr			0.24	0.22	0.22		0.22	0.17	0.11	0.08	0.06	0.05	0.03
1 day			0.16	0.16	0.16			0.14	0.10	0.08	0.06	0.05	0.04
1 mo			0.08	0.07	0.10				0.08	0.07	0.07	0.06	0.05
1 yr				0.06	0.07						0.07		
Lennox–76													
1 hr			0.43	0.40	0.67	0.65	0.51	0.28	0.13	0.07	0.05	0.04	0.02
8 hr			0.26	0.28	0.54		0.50	0.27	0.13	0.08	0.06	0.04	0.03
1 day			0.19	0.20	0.42			0.24	0.12	0.07	0.06	0.04	0.03
1 mo			0.11	0.10	0.16				0.10	0.07	0.06	0.05	0.04
1 yr			0.06	0.06	0.08						0.06		
Reseda–74													
1 hr			0.33	0.35	0.44	0.42	0.33	0.21	0.10	0.06	0.04	0.03	0.01
8 hr			0.25	0.25	0.34		0.28	0.19	0.10	0.06	0.05	0.03	0.02
1 day			0.17	0.15	0.23			0.15	0.09	0.06	0.05	0.04	0.02
1 mo			0.06	0.06	0.09				0.07	0.06	0.05	0.05	0.04
1 yr			0.05	0.05	0.06						0.05		
West LA–71													
1 hr	0.41	0.40	0.53	0.47	0.50	0.49	0.37	0.23	0.11	0.06	0.04	0.03	0.02
8 hr	0.28	0.25	0.36	0.29	0.36		0.28	0.20	0.11	0.06	0.05	0.03	0.02
1 day	0.18	0.18	0.25	0.19	0.24			0.16	0.10	0.06	0.05	0.04	0.03
1 mo	0.09	0.07	0.11	0.08	0.09				0.08	0.07	0.05	0.05	0.04
1 yr	0.05	0.05	0.06	0.06	0.06						0.06		

[a]Determined by continuous Griess-Saltzman method.

Table 6-9. NITROGEN OXIDES CONCENTRATION[a] IN LOS ANGELES COUNTY BY AVERAGING TIME AND FREQUENCY, 1963 THROUGH 1967 (ppm)

Place, site No., averaging time	Maximum for year					% of time concentration equaled or exceeded							
	63	64	65	66	67	0.01	0.1	1	10	30	50	70	90
Azusa–60													
1 hr	0.48	0.28	0.35	0.48	0.52	0.51	0.33	0.22	0.12	0.08	0.05	0.03	0.02
8 hr	0.30	0.19	0.26	0.33	0.37		0.28	0.19	0.11	0.08	0.05	0.04	0.02
1 day	0.17	0.14	0.19	0.21	0.24			0.16	0.10	0.08	0.06	0.04	0.03
1 mo	0.09	0.09	0.08	0.08	0.09				0.08	0.07	0.06	0.06	0.05
1 yr	0.06	0.07	0.06	0.06	0.07						0.06		
Burbank–69													
1 hr	1.20	0.83	1.18	1.02	1.08	1.14	0.95	0.71	0.39	0.19	0.11	0.07	0.04
8 hr	0.70	0.59	0.90	0.75	0.89		0.77	0.60	0.36	0.19	0.12	0.08	0.04
1 day	0.53	0.44	0.60	0.56	0.71			0.51	0.35	0.19	0.13	0.09	0.05
1 mo	0.35	0.22	0.31	0.32	0.36				0.31	0.21	0.14	0.11	0.08
1 yr	0.16	0.14	0.17	0.16	0.19						0.16		
Hywd Freeway–73													
1 hr	0.96	1.28	1.12	1.05	1.10	1.12	0.95	0.76	0.45	0.25	0.16	0.11	0.07
8 hr	0.72	0.81	0.88	0.71	0.78		0.78	0.61	0.41	0.26	0.17	0.12	0.09
1 day	0.53	0.61	0.69	0.53	0.63			0.54	0.40	0.26	0.17	0.13	0.10
1 mo	0.39	0.37	0.40	0.36	0.40				0.37	0.27	0.20	0.15	0.12
1 yr	0.19	0.21	0.22	0.22							0.22		
Los Angeles–1													
1 hr	1.11	1.46	1.35	0.98	1.34	1.22	0.99	0.73	0.36	0.18	0.10	0.06	0.04
8 hr	0.68	0.83	0.68	0.69	0.83		0.77	0.56	0.34	0.18	0.12	0.07	0.04
1 day	0.57	0.57	0.54	0.49	0.60			0.50	0.31	0.19	0.12	0.08	0.05
1 mo	0.37	0.28	0.27	0.30	0.34				0.27	0.20	0.13	0.10	0.08
1 yr	0.16	0.14	0.15	0.17	0.17						0.16		
Los Angeles–70													
1 hr	0.88	0.89	0.82	0.86	0.84	0.84	0.68	0.48	0.25	0.14	0.09	0.07	0.04
8 hr	0.57	0.52	0.50	0.60	0.62		0.57	0.40	0.23	0.15	0.10	0.07	0.05
1 day	0.37	0.37	0.35	0.35	0.45			0.33	0.22	0.15	0.11	0.08	0.05
1 mo	0.23	0.19	0.20	0.21	0.23				0.19	0.15	0.11	0.10	0.08
1 yr	0.12	0.12	0.13	0.12	0.13						0.12		

N Long Beach–72													
1 hr	1.14	1.36	0.85	0.85	1.24	1.19	0.93	0.67	0.37	0.19	0.12	0.08	0.04
8 hr	0.82	0.91	0.58	0.61	0.73		0.78	0.59	0.35	0.19	0.13	0.08	0.05
1 day	0.69	0.67	0.45	0.54	0.60			0.53	0.35	0.20	0.13	0.09	0.06
1 mo	0.45	0.36	0.30	0.29	0.32				0.29	0.21	0.14	0.10	0.07
1 yr	0.17	0.17	0.15	0.16	0.19						0.17		
Pasadena–64													
1 hr	0.79	0.70	0.60	0.78	0.71	0.76	0.63	0.45	0.22	0.12	0.08	0.06	0.03
8 hr	0.52	0.45	0.39	0.53	0.49		0.50	0.37	0.19	0.12	0.09	0.07	0.04
1 day	0.35	0.30	0.28	0.33	0.31			0.28	0.18	0.13	0.10	0.08	0.05
1 mo	0.23	0.18	0.14	0.16	0.18				0.16	0.12	0.09	0.08	0.07
1 yr	0.11	0.11	0.10	0.11	0.12						0.11		
Pomona–75													
1 hr			0.59	0.59	0.67	0.66	0.57	0.43	0.26	0.16	0.11	0.07	0.04
8 hr			0.35	0.43	0.44		0.43	0.36	0.23	0.15	0.11	0.08	0.05
1 day			0.27	0.29	0.33			0.28	0.23	0.16	0.12	0.09	0.06
1 mo			0.15	0.19	0.23				0.20	0.15	0.12	0.11	0.09
1 yr				0.12	0.15						0.15		
Lennox–76													
1 hr			1.21	1.95	2.10	1.89	1.57	1.00	0.46	0.20	0.11	0.07	0.03
8 hr			0.78	1.21	1.66		1.27	0.83	0.42	0.22	0.12	0.08	0.05
1 day			0.67	0.83	1.13			0.79	0.40	0.22	0.13	0.09	0.06
1 mo			0.34	0.40	0.42				0.37	0.22	0.15	0.11	0.08
1 yr			0.17	0.18	0.23						0.18		
Reseda–74													
1 hr			1.01	0.86	1.00	1.00	0.83	0.59	0.26	0.13	0.07	0.04	0.02
8 hr			0.69	0.60	0.65		0.65	0.48	0.24	0.13	0.08	0.05	0.03
1 day			0.52	0.40	0.47			0.39	0.24	0.13	0.09	0.06	0.04
1 mo			0.21	0.19	0.22				0.20	0.14	0.09	0.08	0.06
1 yr			0.10	0.12	0.13						0.12		
West LA–71													
1 hr	1.18	1.05	1.15	1.18	1.23	1.18	0.95	0.65	0.31	0.14	0.08	0.05	0.03
8 hr	0.81	0.58	0.88	0.76	0.71		0.71	0.50	0.28	0.15	0.09	0.06	0.04
1 day	0.55	0.46	0.53	0.53	0.53			0.46	0.27	0.15	0.10	0.07	0.04
1 mo	0.29	0.23	0.27	0.22	0.28				0.23	0.17	0.11	0.09	0.06
1 yr	0.13	0.12	0.14	0.13	0.13						0.13		

[a]Determined by continuous Griess-Saltzman method.

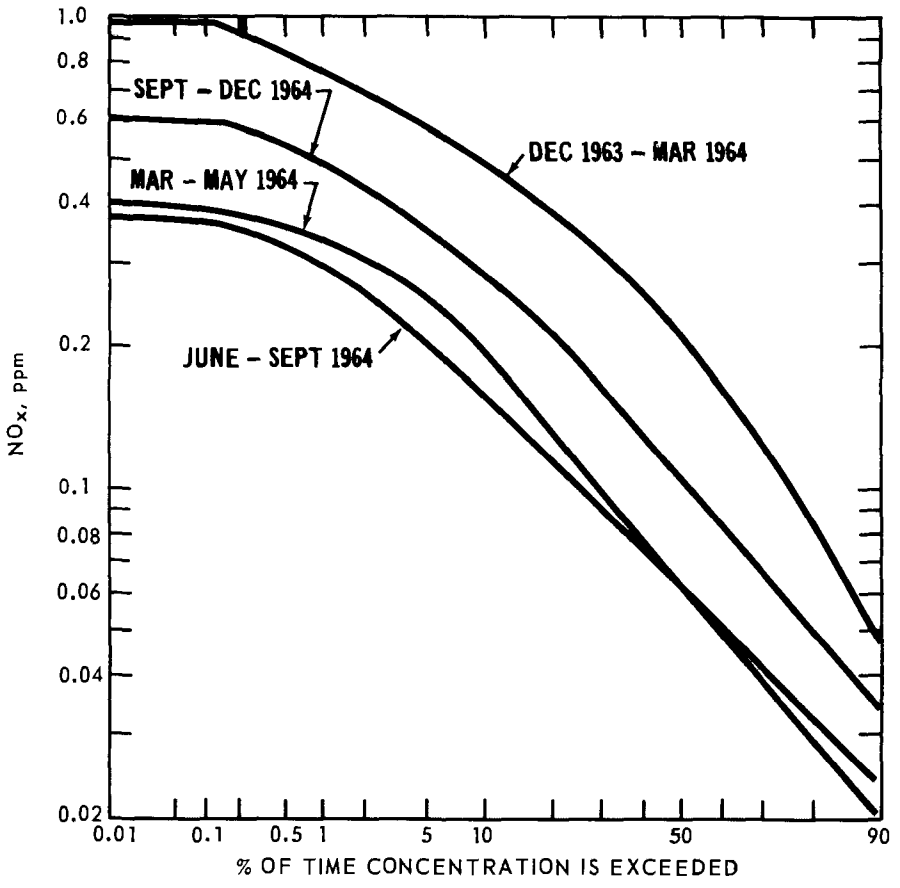


Figure 6-12. Frequency distribution of 3-hour-average concentrations of NO_X at Los Angeles CAMP Station, December 1, 1963, to December 1, 1964.

a 17 percent increase in NO_X emissions during the winter months. NO_X values are also affected by ambient temperatures and by photochemical reactions. Comparison of the NO and NO_X values by season clearly demonstrates the influence of the photochemical reactions that convert NO to NO_2. The NO median value in the summer season (Figure 6-12) represents 50 percent of the NO_X median, whereas in the winter season the NO median represents 80 percent of the NO_X median. This comparison of seasonal NO and NO_X values demonstrates the increased conversion of NO to NO_2 during the summer due to the presence of increased sunlight.

E. EFFECTS OF MEASUREMENT SYSTEMS ON DATA

In addition to the continuous monitoring of pollutants at CAMP stations, the Air Pollution Control Office of EPA obtains data from its Gas-Sampling Network in the National Air Surveillance Networks (NASN). This latter network, a cooperative effort with local health and air pollution agencies, determines 24-hour concentrations of certain pollutants on a biweekly schedule at approximately 150 sites. The yearly average 24-hour NO_2 concentrations are shown in Table 6-10 for the NASN network for the years 1967, 1968, and 1969.

Table 6-10. AVERAGE 24-HOUR NO_2 CONCENTRATION[a] AT NATIONAL AIR SAMPLING NETWORK SITES, 1967 THROUGH 1969
(ppm)

State	City	Station number	1967	1968	1969
Alabama	Birmingham	3			0.093[b]
	Mobile	1			0.025
	Montgomery	1			0.016
Alaska	Fairbanks	1	0.046	0.052	0.045
Arizona	Grand Canyon Park	1			0.012[b]
	Phoenix	2		0.117[b]	0.089
	Tucson	1			0.028[b]
Arkansas	El Dorado	1		0.057	0.047
	Little Rock	1			0.012
California	Anaheim	1		0.159	0.148
	Berkeley	1			0.027[b]
	Fresno	1			0.042
	Glendale	1			0.081[b]
	Humboldt County	1			0.012
	Long Beach	1		0.216[b]	0.182[b]
	Oakland	1			0.053[b]
	Sacramento	1			0.019[b]
	San Bernardino	1		0.131	0.106
	San Diego	1		0.113	0.106
	San Francisco	1		0.110	0.095
	San Jose	2		0.123	0.116
	Santa Ana	1			0.059
Colorado	Denver	1			0.032
	Denver CAMP	2	0.070	0.092[b]	0.076[b]
Connecticut	Bridgeport	1		0.112	0.106[b]
	Hartford	1	0.109	0.071	0.077
	New Haven	–	0.094	0.107	0.072
	Waterbury	1			0.037
Delaware	Kent County	1		0.047[b]	0.042
	Newark	1	0.045	0.075	0.054[b]
	Wilmington	1	0.096	0.104	0.071[b]
District of Columbia	Washington	1			0.040
	Washington CAMP	3			0.069
	Washington CAMP	2	0.132	0.101	
Florida	Jacksonville	2			0.038
	Miami	2		0.072	0.061
	St. Petersburg	2			0.020
	Tampa	2		0.080	0.079[b]
Georgia	Atlanta	1		0.118	0.096
	Columbus	1			0.025[b]
	Savannah	1			0.031
Illinois	Chicago	1			0.054
	Chicago CAMP	2	0.144	0.155	0.160
	Peoria	–			0.050[b]
	Rockford	1			0.036

Table 6-10 (continued). AVERAGE 24-HOUR NO_2 CONCENTRATION[a] AT NATIONAL AIR SAMPLING NETWORK SITES, 1967 THROUGH 1969
(ppm)

State	City	Station number	1967	1968	1969
Indiana	East Chicago	1	0.081	0.081	0.086
	Evansville	1	0.044	0.032	0.031
	Gary	1			0.045
	Hammond	1			0.054
	Indianapolis	1	0.051	0.099	0.079
	Monroe	1	0.028[b]	0.032	0.034
	New Albany	1	0.086[b]		
	New Albany	2		0.081[b]	0.072[b]
	South Bend	2			0.031
Iowa	Des Moines	1	0.051	0.070	0.033
	Dubuque	1	0.030	0.091	0.072
Kansas	Topeka	1			0.023
	Wichita	1		0.057	0.064
Kentucky	Covington	1	0.088	0.110	0.096
	Lexington	1		0.077	0.062[b]
	Louisville	1		0.115	0.096
Louisiana	Carville	1		0.053	0.043
	New Orleans	2		0.080[b]	0.061
Maine	Acadia National Park	1		0.024[b]	0.020
Maryland	Baltimore	1		0.095[b]	0.099
Massachusetts	Boston	1	0.063[b]	0.055[b]	0.040[b]
	Springfield	2		0.115[b]	0.086
	Worcester	1		0.090[b]	0.087
Michigan	Detroit	1	0.100	0.130	0.119
	Flint	1		0.093	0.085
	Grand Rapids	1		0.093	0.090
	Lansing	1			0.038
	Saginaw	1			0.031
Minnesota	Minneapolis	1	0.071	0.076	0.076
Missouri	Kansas City	1	0.076	0.081[b]	0.045
	St. Louis	1	0.094	0.116[b]	0.135
	St. Louis CAMP	2	0.116	0.115	0.108
Montana	Glacier National Park	1			0.012[b]
Nebraska	Omaha	1	0.076	0.072	0.075
New Jersey	Burlington County	2	0.097	0.091	0.062
	Camden	1	0.106	0.121	0.128
	Glassboro	1	0.071	0.059	0.020
	Jersey City	1	0.131	0.066[b]	0.065
	Newark	1	0.116	0.137	0.092
	Patterson	1		0.129	0.099
New Mexico	Albuquerque	1		0.060	0.048
New York	Albany	1		0.084[b]	0.071
	Buffalo	1		0.079	0.029
	Buffalo	3	0.082[b]		
	New York City	1	0.179	0.148[b]	0.142[b]

Table 6-10 (continued). AVERAGE 24-HOUR NO_2 CONCENTRATION[a] AT NATIONAL AIR SAMPLING NETWORK SITES, 1967 THROUGH 1969
(ppm)

State	City	Station number	1967	1968	1969
	Rochester	1		0.096[b]	0.086
	Syracuse	1		0.093[b]	0.074
	Utica	1		0.062	0.070
North Carolina	Durham	1		0.102	0.076[b]
	Greensboro	1			0.076
	Greensboro	2		0.099	
Ohio	Akron	1		0.107	0.038
	Canton	1		0.103	0.092
	Cincinnati	1		0.102	0.099
	Cincinnati CAMP	3	0.087	0.096	0.091
	Cleveland	1	0.072	0.119	0.099
	Columbus	1		0.105	0.087
	Dayton	1	0.032	0.043	0.057
	Toledo	1		0.093	0.096[b]
	Youngstown	1	0.088	0.096	0.083[b]
Oklahoma	Oklahoma City	1	0.074	0.090	0.048[b]
	Tulsa	1	0.039	0.056	0.033
Oregon	Portland	1	0.027[b]	0.074	0.056[b]
Pennsylvania	Allentown	1		0.077	0.090
	Clearfield County	1			0.033
	Indiana County	1			0.040
	Johnstown	1			0.080
	Lancaster	1		0.101[b]	
	Philadelphia	1		0.088[b]	0.024
	Philadelphia	2	0.125		
	Pittsburgh	1	0.079	0.132	0.113
	Reading	1		0.112	0.081
	Warminster	1	0.075	0.066	0.054
	West Chester	1	0.049	0.066	0.042
	York	2		0.096	0.076
Puerto Rico	Bayamon	2		0.047	0.041
	Bayamon	1	0.040[b]		
	Guayanilla	1	0.029	0.031[b]	
	Guayanilla	2			0.034
Rhode Island	Providence	1	0.051	0.100	0.087
South Dakota	Custer	1		0.025	0.015[b]
Tennessee	Chattanooga	1	0.076	0.089[b]	0.042
	Memphis	1		0.090[b]	0.078
	Nashville	1	0.084	0.101	0.068[b]
Texas	Austin	2			0.030
	Beaumont	1			0.042[b]
	Corpus Christi	1			0.026[b]
	Dallas	2		0.100	0.074
	El Paso	1	0.043	0.071	

Table 6-10 (continued). AVERAGE 24-HOUR NO_2 CONCENTRATION[a] AT NATIONAL AIR SAMPLING NETWORK SITES, 1967 THROUGH 1969

(ppm)

State	City	Station number	1967	1968	1969
	El Paso	2			0.048[b]
	Fort Worth	1		0.083	0.071
	Houston	1		0.116[b]	0.105
	Lubbock	1			0.021[b]
	Pasadena	1	0.035	0.074[b]	
	Pasadena	2			0.050
	San Antonio	1		0.068	0.065
	Tom Green County	1			0.015
Utah	Salt Lake City	1	0.048	0.084	0.060
Virginia	Norfolk	1		0.087	0.077
	Page	1		0.035	
	Shenandoah National Park	1			0.015
	Richmond	2		0.102	0.088
Washington	Seattle	1	0.052	0.096	0.095[b]
	Tacoma	1			0.023
West Virginia	Charleston	1	0.076	0.109	0.092
Wisconsin	Milwaukee	1	0.093	0.109	0.090
Wyoming	Casper	1	0.035	0.032	0.025

[a]Determined by integrated Jacobs-Hochheiser method.

[b]The number and distribution of individual values making up this average do not meet the NASN criteria for calculating a yearly average. Nevertheless, it is felt that the average is adequate for drawing the general relationships reported in this chapter.

Since there is an NASN station at each of the CAMP stations, the NO_2 values determined by the CAMP continuous instrument with the Griess-Saltzman method of analysis can be readily compared with the NASN 24-hour-integrated-value based on the Jacobs-Hochheiser method.

Table 6-11 presents the ratios of NASN to CAMP NO_2 values for the years 1967, 1968, and 1969. The table shows that NASN NO_2 values are three times greater, on the average, than the corresponding CAMP values. There is no satisfactory explanation for the discrepancy at this time. Both methods have been carefully checked under laboratory conditions and are internally consistent. In the field, however, it is possible that other factors, such as difference in type and age of sampling-line particulate filters, may be affecting the measurements.

Limited laboratory testing suggests that the specific particulate filter employed in the sampling line and the age of the filter may exert a major influence on measurement values. Frequent in-field calibration and servicing of the instruments is, therefore, essential.

The results are further complicated by the fact that the CAMP continuous instruments cannot be expected to give accurate or precise results for 1-hour NO_2 concentrations below 190 $\mu g/m^3$ (0.10 ppm). CAMP instruments

Table 6-11. RATIO OF AVERAGE YEARLY NASN[a] TO CAMP[b] NO_2 MEASUREMENTS

City	NO_2 ratio: NASN/CAMP		
	1967	1968	1969
Chicago	3.2	3.0	3.0
Cincinnati	3.3	3.2	–
Denver	2.0	2.7	2.3
Philadelphia	3.2	–	–
St. Louis	5.5	4.3	4.5
Washington	3.0	2.9	1.5
Median	3.2	3.0	2.7
Minimum	2.0	2.7	1.5
Maximum	5.5	4.3	4.5

[a]National Air Sampling Network.

[b]Continuous Air Monitoring Program.

are designed to measure NO_2 concentrations from 0 to 1.9 mg/m^3 (0 to 1 ppm), but are calibrated by the use of NO_2 mixtures in the range of 0.2 to 1.9 mg/m^3 (0.1 to 1 ppm). Many of the days during which comparisons in Table 6-11 were made, had 24-hour CAMP values of 20 to 130 μg/m^3 (0.01 to 0.07 ppm), however; and since these 24-hour values are established by a process involving summation of the hourly values, a large proportion of the included hourly values were in the 20- to 60- μg/m^3 (0.01 to 0.03 ppm) concentration range. Under these circumstances large errors (100 to 200%) would not be uncommon. By contrast, the NASN technique has been calibrated in the 0- to 190- μg/m^3 (0 to 0.1 ppm) range, as well as at higher concentrations, hence should be more trustworthy.

F. SUMMARY

Continuous measurement of the oxides of nitrogen by various monitoring networks has made it possible to compile selected-time-averages of mean concentrations for selected time periods. From these values, various temporal patterns have been analyzed and related to photochemical and meteorological parameters.

Both NO and NO_2 concentrations display distinct diurnal variations dependent on the intensity of the solar ultraviolet energy and the amount of atmospheric mixing. These concentrations also vary with the traffic patterns in the sampling area.

Nitric oxide concentration shows a seasonal variation, with higher values occurring during the late fall and winter months. Nitrogen dioxide, however, does not display such distinct seasonal patterns. An analysis of limited air-quality data for total NO_x concentrations has not clearly indicated any yearly trends.

The effect of meteorological factors on NO and NO_2 concentrations has been well documented. Periods of stagnation in urban areas have resulted in high peak levels of NO_x and resultant high-oxidant levels.

Continuous measurement has indicated that peak values of NO above 1.23 mg/m^3 (1 ppm) are common, but NO_2 concentrations have rarely been measured at this level. Most NO_2 concentrations measured in urban areas have been under 0.94 mg/m^3 (0.5 ppm).

Methods for measuring atmospheric concentrations of NO_x are still in need of improvement. In one instance, measurements of NO_2 taken at the same site by different methods were found to differ by a factor of 3, but there is no satisfactory explanation for the discrepancy at this time.

G. REFERENCES

1. The Automobile and Air Pollution, A Program for Progress Part II. U.S. Department of Commerce. Washington, D.C. December 1967.
2. Nitrogen Oxides and Air Pollution, California Air Resources Board. Sacramento. January 1966.
3. Brier, G.W. Some Statistical Aspects of Long-Term Fluctuations in Solar and Atmospheric Phenomena. Ann. N.Y. Acad. Sci. *95*: 173-187, 1961.
4. Ingels, R.M., et al. Trends in Atmospheric Concentrations of Oxides of Nitrogen, 1957-1961. Los Angeles County Air Pollution Control District. August 1962.

5. Green, M.H. New Jersey Air Monitoring Systems and Air Quality Data, October 1965 through December 1968. N.J. Department of Health, Division of Clean Air and Water. Trenton. Technical Bulletin No. A-69-1. July 1969. 216 p.
6. Larsen, R.I. A New Mathematical Model of Air Pollutant Concentration Averaging Time and Frequency. J. Air Pollut. Contr. Ass. *19*: 24-30. January 1969.
7. Air Quality Criteria for Hydrocarbons. National Air Pollution Control Administration. Washington, D.C. Publication No. AP-64. March 1970. p. 5-1 to 5-13.
8. Comprehensive Technical Report on All Atmospheric Contaminants Associated with Photochemical Air Pollution. System Development Corporation. Santa Monica, California. Report No. TM-(L)-4411/002/01. June 1970.
9. Schuck E. A., J.N. Pitts, Jr., J.K.S. Wan. Relationships Between Certain Meteorological Factors and Photochemical Smog. Air and Water Pollut. *10*: 689-711, 1966.

CHAPTER 7.

EFFECTS OF NITROGEN OXIDES ON MATERIALS

A. INTRODUCTION

Field studies and laboratory research have successfully linked nitrogen oxides (NO_X) to problems regarding effects on textile dyes and additives, natural and synthetic textile fibers, and metals. While not as well known as those caused by sulfur dioxide or ozone, these effects, nevertheless, are important and represent a significant economic burden.

Nitric oxide (NO) and its principal reaction product, nitrogen dioxide (NO_2), participate in a number of complex atmospheric reactions. Eventually, atmospheric processes bring the NO_X to the nitric acid stage where it rapidly reacts to produce various particulate nitrates. This chapter discusses only the material effects that are attributed to NO_X and particulate nitrates, but does not preclude the possibility that other reactants such as nitric acid (HNO_3) can cause damage. One must not, however, lose sight of the role of NO_X in the photochemical formation and buildup of ozone and other oxidants, as well as in the photo-oxidation of sulfur dioxide in the presence of reactive hydrocarbons to produce sulfuric acid aerosols. The photochemical reaction products are believed to cause more damage than NO_X directly; thus, control and reduction of NO_X should also be valuable in reducing damage from these products. AP-63, *Air Quality Criteria for Photochemical Oxidants,* discusses the effects of photochemical pollutants on materials.

B. EFFECTS ON TEXTILE DYES

1. Acetate Rayon Fading

a. Historical Background

Just prior to World War I, a German dye manufacturer investigated some unusual cases of dye fading on stored wool goods.[1] Fading was most noticeable on the edges of the goods, and the primary cause was traced to NO_X in the air. Open electric-arc lamps and incandescent gas mantles were major sources of these pollutants by virtue of a process of high-temperature fixation. The investigators found that all the susceptible dyes contained free or substituted amino groups, and they suggested that these might become either diazotized or nitrosated by the NO_X.

During and following the war years, increased replacement of older forms of lighting with electric filament lamps led to a general decline of the wool-fading problem. In the mid-1920's, however, researchers developed and introduced a new fiber, cellulose acetate rayon. Traditional dyes were of little use on this fiber, but chemists soon developed disperse dyes that were effective. Some of these dyes, however, contained amino-groups that the Germans had previously found susceptible to NO_X fading.

Shortly thereafter a puzzling type of fading began to occur increasingly on dyed acetate. Blue, green, and violet shades of fabrics, either stored or in use, faded mysteriously. Because this fading was frequently observed in rooms heated by gas heaters, it was called "gas-fume fading" or "gas fading."

During the 1930's, acetate fading became a serious problem. Dye and fiber chemists, apparently unaware of the earlier German work, devoted considerable efforts toward a solution. These efforts culminated in 1937 when Rowe and Chamberlain[2] systematically investigated the fundamental chemistry of dye degradation and independently reached the same conclusions as the earlier German team. Since then, much research has been

carried out in an effort to understand gas fading and to develop ways of preventing it.[3-5]

b. Gas-Fading Characteristics

Gas fading is marked by a definite and characteristic reddening of the fabric. This reddening effect took on special importance in the mid-1950's when investigators discovered that ozone also fades many of the same dyes found to be sensitive to NO_x. In many cases the characteristic reddening enables scientists to discriminate between gas fading and the bleached, washed-out fading that ozone produces.

c. Test for Sensitivity

Recognizing the importance of fading by NO_x, the American Association of Textile Chemists and Colorists (AATCC) developed a fading-test procedure that they tentatively adopted in 1941 and formally approved in 1957. It is presently designated as Standard Test Method 23-1962, Colorfastness to Oxides of Nitrogen in the Atmosphere.[6] This method calls for suspending test specimens along with a control sample in a chamber, and exposing them to combustion gases from a gas burner that has been adjusted so that the chamber temperature does not exceed 60°C. The resulting concentration of NO_x is about 38 mg/m^3 (20 ppm).[7] Specimens remain in the chamber until the control sample shows a change in shade corresponding to a fading standard. When this happens, the specimens are said to have had a treatment equivalent to 6 months of actual exposure to average air, as represented by three separate locations in southern New Jersey. If the test specimens do not fade appreciably after one exposure period, the procedure is repeated (using a fresh control sample) as many times as necessary to make an evaluation. Dyed fabrics are classified and rated according to the number of exposures necessary to produce appreciable changes in shade.

Despite an earlier research report that pure NO slightly fades some dyes,[4] current opinion[1,7] favors NO_2 as the fading agent. This is largely because (1) photochemical interactions convert NO to NO_2 and (2) controlled environment studies using concentrations of NO_2 below 188 mg/m^3 (<100 ppm) produce color changes similar to those found in field exposures. Since the test concentrations were so far above normal ambient levels, the validity of these controlled-environment studies is open to questions. Cellulose acetate fibers are excellent absorbers of NO_2,[7] and this property undoubtedly also plays an important role in dye-fading mechanisms.

The disperse dyes sensitive to gas fading have an amino anthraquinone structure. The reactions that cause fading include nitrosation, diazotization, and oxidation.[1] Blue dyes are especially susceptible; some violets and reds also fade. Even blends of the susceptible colors with resistant dyes are subject to gas fading. Nevertheless, these sensitive dyes are economically valuable because they are moderately priced and have desirable dyeing properties.

d. Attempts at Protection

To alleviate the fading problem, researchers have developed a number of chemical inhibitors that can be added to the dyed fabric and selectively react with the nitrogen oxides.[7,8] Inhibitors, however, are only effective temporarily, since they are eventually used up and the dye again becomes vulnerable to fading. Subsequently, researchers developed dyes that were resistant to fading; but those dyes had poor lightfastness. In the 1950's, chemists synthesized new dyes that resisted the fading effects of both NO_x and light.[7,9,10] These dyes however, are more expensive and have poor dyeing properties, which result in slower processing rates and require greater care during application. Other means to circumvent the fading problem include using alternative fibers, or fibers with colored pigments incorporated in them.

Notwithstanding, costly gas-fading incidents still persist.[11-13] Many incidents of fading have occurred in warehouses, and caused

whole truckloads of materials to be returned to manufacturers. Fading of acetate linings in men's suits has been a particularly vexing problem. Some fiber manufacturers do not allow labelling with their brand names unless the resulting fabrics meet established colorfastness standards against both pollutants and light.[13] Even though dyers are aware of this fading problem, many still use the sensitive dyes for economic reasons. Nevertheless, use of fibers vulnerable to fading is decreasing as both fabric producers and retailers become more sensitive to the public's demand for products of higher quality.

2. Cotton and Viscose Rayon Fading (Cellulosics)

While NO_x fading of certain disperse dyes on cellulose acetate fabrics is now well documented, the textile industry has paid little attention to the fading, caused by pollutants, on cotton and viscose rayon (collectively referred to as cellulosics). Consumers and retailers have registered color-change complaints, but textile people generally attribute this fading to light.

An early indication of the cotton problem occurred in the mid-1950's. Dye chemists investigated a series of complaints that some colored cotton fabrics were fading during the drying cycle in home gas-fired dryers.[14] The investigators traced the fading to oxides of nitrogen formed during the combustion of natural gas used to heat the dryers. Fading occurred only while the textile materials were moist. Subsequent research about 10 years later confirmed the gas-dryer fading problem and found that NO_x levels (expressed as NO_2) in such dryers ranged from 1.1 to 3.7 mg/m^3 (0.6 to 2 ppm).[15]

Within the last decade, additional evidence of this type of fading has emerged. When exposed to a number of different field environments in the absence of light, some dyed cellulosic fabrics showed fading after 2 to 3 months.[16,17] Fading occurred in certain blue and green shades, representing dyes from four major classes: direct, sulfur, vat, and reactive dyes. Laboratory exposure of these shades to the standard gas-fading test procedure did not produce fading. The test procedure, was designed to evaluate dyed acetate fabrics, and has no provisions for controlling relative humidity, because humidity is not a critical factor in acetate fading. The field exposures, however, indicated that relative humidity might be an important controlling factor in cellulosics fading. Subsequent laboratory exposures to the gas-fading test procedure were therefore conducted under high-humidity conditions (probably greater than 50 percent). The results were dramatic; the same concentrations of NO_x produced color changes that generally agreed with the field results. Presently, the textile-dye industry is considering modifying the gas-fading test procedure to call for humidity-controlled conditions.[18-20]

3. Yellowing of Whites

A new problem that has received little publicity, but is of considerable concern to the textile industry is the yellow discoloration of undyed-white or pastel-colored fabrics.[15,20] These fabrics may be woven from any number of common fibers, but most of the discoloration has occurred on Nylon, acetate, and permanent-press materials. Discoloration has usually occurred on items stored or on display, including dresses, shirts, curtains, and lingerie. Returned items have represented major losses to some textile companies.

Since most discoloration occurs on whites, dyes were ruled out as a source of the problem. Investigators turned to various additives, including optical brighteners; cationic, antistatic, and soil-release finishers; softeners; and resinous processing agents, which are applied to fibers and fabrics to enhance certain properties. When tested by standard laboratory procedures, including the gas-fading procedure, many of these additives yellowed on exposure to NO_x. Washing the fabrics sometimes removed the yellow discoloration, but this is impractical for items that yellow in warehouses or on display. Today, the problem

is best solved by selecting resistant additives. Such selection may result in diseconomies, but the textile industry and retailers recognize that some action must be taken to avoid an increasing number of complaints about the discoloration of whites.

C. EFFECTS ON TEXTILE FIBERS

1. Cellulosic Fibers

Researchers have not studied the direct effect of NO_x on cellulosic fibers, but Morris et al.[21] did conduct a field study of cellulosics from which they attributed damage to ambient levels of NO_x in Berkeley, California. They exposed combed cotton yarn samples at a 45-degree angle in cabinets facing south. Polyvinyl fluoride film, rather than glass, was used to cover the cabinets in order to allow a greater amount of sunlight to enter. One chamber in each cabinet was set up as a control, in which entering air was filtered through carbon canisters. Ambient air was circulated through the other chamber. (The investigators did not note the rate of air change.) Some samples were exposed directly to daylight, while others were shaded. Samples were exposed for three separate 28-day periods (December through February), as well as for consecutive combinations of these three periods, as is shown in Table 7-1. During exposure, both air pollution and weather measurements were made (Table 7-2).

Table 7-1. BREAKING-STRENGTH OF COTTON YARN SAMPLES EXPOSED TO AIR AND SUNLIGHT IN BERKELEY, CALIFORNIA[21]

Exposure		Breaking-strength, % loss		Pollution effect, increase in % loss breaking-strength
Number of days	Period[a]	Filtered air	Unfiltered air	
28	I	11	15	4
28	II	15	18	3
28	III	15	16	1
56	I, II	20	24	4
56	II, III	19	29	10
84	I, II, III	26	32	6

[a]I - December, II - January, III - February.

Table 7-2. AIR POLLUTANT LEVELS[a] AND WEATHER MEASUREMENTS[21]

Exposure period	Total oxidant,		Nitric oxide ,		Nitrogen dioxide,		Temp, °F	Total sunshine, % days	Rain, in.
	ppm	$\mu g/m^3$	ppm	$\mu g/m^3$	ppm	$\mu g/m^3$			
I	0.03	60	0.19	230	0.08	150	50	100	0.5
II	0.03	60	0.23	280	0.08	150	50	100	6
III	0.03	60	0.07	90	0.05	90	50	72	10

[a]For clock hour with highest average value.

At least 20 yarn specimens were evaluated for each exposure period. The investigators assessed deterioration by measuring loss of breaking strength. Table 7-1 shows the mean-breaking-strength in percent losses, for unshaded samples, exposed to filtered and unfiltered ambient air, for the various exposure periods. An analysis of variance of these mean-loss values revealed that unfiltered air deteriorates cotton yarn to a significantly greater extent than filtered air. The shaded samples did not develop a corresponding difference between filtered and unfiltered air. While textile investigators are well aware of the pronounced deteriorating action of direct sunlight, the results of this study serve to emphasize the importance of sunlight in stimulating reactions between some air pollutants and materials. It is noteworthy that the smallest difference between breaking-strength losses for filtered and unfiltered air occurred during Period III—the period having the most rain (which cleans the air), the lowest levels of NO_X, and the lowest total hours of sunshine. While it is impossible to designate the aggressive pollutant in this study, the investigators proposed that NO_X, either directly or indirectly, were instrumental in causing damage. Sulfur dioxide was not measured, since levels were known to be low.

The investigators did not mention the fact that, although the filter material, activated carbon, absorbs NO_2 effectively, it absorbs NO poorly. Levels of NO in the filtered air chamber could, conceivably, have contributed to the decrease in breaking strength. If this were the case, cotton specimens exposed to *clean* air would show a smaller loss in breaking strength, and the relative pollution effect would increase.

2. Synthetic Fibers

Ambient levels of NO_X do not appear to cause noticeable damage to synthetic fibers. Several items, however, are worthy of mention

In March 1964, New York City newspapers[22] reported an episode of runs in Nylon stockings worn by women in the vicinity of a demolition project. Investigators identified the guilty agent as NO_2 gas released during dynamite blasting operations. Local weather conditions at that time were unfavorable—a temperature inversion existed along with wind stagnation and high humidity. The investigators proposed that the combination of these conditions in the presence of abnormally high levels of released NO_2 and dust had produced nitric acid aerosols that damaged the Nylon stockings.

Travnicek,[23] noting the New York City episode, suggested that "the corrosive effect of NO_X is rather strong, not only for Nylons, but for practically all other fiber-forming polymers, because it combines acid corrosion and oxidation."

Spandex is a synthetic, elastomeric material. On exposure to the standard gas-fading test procedure, it develops a yellow cast.[20] This is not a dye-fading problem, since the oxides of nitrogen react directly with the polymeric material.

D. EFFECTS ON NICKEL-BRASS ALLOYS

1. Stress-Corrosion

In 1959 the Pacific Telephone and Telegraph Company noticed considerable breakage of their nickel-brass (65 Cu–23 Zn–Ni) wire springs in some relays located in Los Angeles area central offices.[24-26] Failures often occurred within 2 years after installation. These failures were totally unexpected, since the wire springs had been used with excellent results for years, throughout the nation. Investigators found that breakage occurred on wires that were under moderate stress and a positive electrical potential, and it was concluded that the failure mechanism was a form of stress-corrosion-cracking. Bell Laboratories subsequently showed that high-nitrate concentrations in airborne dust, which had accumulated on surfaces adjacent to cracked areas, produced the failures. The nitrate content of dust from Los Angeles is from 5 to 15 times greater than from most eastern and midwestern cities. Furthermore, Los Angeles dust consists of light-colored, very fine, claylike materials and considerable

organic matter in a highly oxidized, polar condition. This combination results in dust that is more sensitive and reactive to moisture than the carbonaceous, oily, siliceous, high-sulfate content of most eastern dusts. Nitrates are also more hygroscopic than sulfate salts. Additional tests have shown that failures take place only when surface nitrate concentrations are above 2.4 $\mu g/cm^2$ and when the relative humidity, a very important controlling factor, is above 50 percent. Other salts will cause stress corrosion, but only when the relative humidity is greater than 75 percent; for sulfate salts it must exceed 95 percent.

Sometime after this stress-corrosion problem was first observed in Los Angeles, scattered failures were reported, not only in wire springs, but in other nickel-brass components elsewhere in California, and in New York City, and Philadelphia. Westinghouse[25] has reported failures in Texas and New Jersey.

Bell Laboratories[26] also reported a totally different type of corrosion problem that has been observed in such widely scattered locations as Cincinnati, Cleveland, Detroit, Los Angeles, New York, and Philadelphia. The nickel bases of palladium-topped contacts of crossbar switches corroded forming bright-greenish corrosion products that gradually crept up over the palladium cap of the contact, resulting in electrically open circuits. Investigators concluded that the "creeping green" corrosion was promoted by the presence of anions, principally nitrates, in accumulated dust.

Nitrates in urban atmospheres have been identified as one of the end products of the photochemical reactions between oxides of nitrogen and hydrocarbons (see chapter 4). In Cincinnati and Philadelphia, the cities for which data are available, the 1965 average airborne nitrate particulate concentrations were 3.4 and 3.0 $\mu g/m^3$ respectively. The corresponding average gaseous NO_x levels were 124 and 158 $\mu g/m^3$ (0.066 and 0.084 ppm).

2. Protection

The telephone company took several measures to correct the stress-corrosion problem. Researchers found that when zinc is left out of the nickel-brass alloy, stress-corrosion no longer occurs. To prevent future problems, a copper-nickel material was specified for subsequently manufactured wire-spring relays. In high-nitrate areas, local central offices protected existing nickel-brass relays by installing high-efficiency filters in outside-air intakes of ventilating systems, and by redesigning their cooling systems to keep relative humidity below 50 percent.

E. FUTURE RESEARCH NEEDS

At present, the accepted procedure for evaluating the fading characteristics of dyed fabrics uses NO_x generated from a natural-gas flame. This produces levels of NO_x that, while of low magnitude, vary considerably. In addition, since temperature and relative humidity are not controlled, experimental results do not correspond, absolutely, to practical environmental conditions.

Although dye chemists use this procedure to assess the vulnerability of dyes and additives to NO_x, they have no sound idea of the dose-response relationships. Reported complaints and field studies have been the principal means for establishing the actual existence of atmospheric fading problems.

To estimate costs resulting from NO_x damage, investigators must determine reliable dose-response relationships for vulnerable materials. Such research would also delineate the influence of temperature, relative humidity, sunlight, and other possible parameters, including interaction with other pollutants.

F. SUMMARY

Investigators have found that oxides of nitrogen and their corresponding reaction products cause certain textile dyes to fade, cause some textile additives to yellow, deteriorate cotton fabrics, and accelerate corrosion of certain metals. The most serious problems concern textile dyes and additives.

Many years ago, complaints by retailers lead to the discovery that NO_x fades a number of sensitive, disperse dyes used on cellulose acetate fibers. The sensitive colors were

mainly shades of blue. To correct this fading problem, dye chemists developed both dye-additive inhibitors that provide temporary protection and fade-resistant dyes. In both cases, especially in the latter, the end result is a more expensive fabric.

During the last decade or so, investigators found that certain blue and green shades of dyed cellulosics faded under high-humidity conditions. They first encountered this problem when investigating complaints that cotton fabrics faded during the drying cycle in home gas-fired dryers. They found that oxides of nitrogen, which are among the products of combustion of natural gas, readily reacted with some dyes under moist conditions. NO_2 levels in the dryers ranged from 1.1 to 3.7 mg/m^3 (0.6 to 2 ppm). Later, field exposures, in the absence of light, showed fading occurred in certain dyed cellulosic fabrics. Previous laboratory exposures to NO_x had not faded these fabrics. Subsequent laboratory exposure to NO_x, under conditions of high humidity did produce fading in these fabrics and confirmed the importance of atmospheric moisture in the fading reactions.

A more recent problem concerns the yellow discoloration of undyed-white and pastel-colored fabrics. Chemists have traced this yellowing to the action of NO_x on certain additives applied to fabrics to enhance their marketing properties. These additives include optical brighteners, softeners, antistatic and soil-release finishes, and resinous processing agents. To prevent this yellow discoloration, textile processors must be critical in selecting NO_x-resistant additives, which are generally more costly.

Information concerning the damaging effects of NO_x on textile fibers is meager. One study has been reviewed here in which investigators exposed cotton fabrics to ambient air containing above-average levels of NO_x. The exposed yarns showed increased losses in fiber strength.

New York City scientists who investigated an episode of runs in Nylon stockings traced the problem to abnormal levels of NO_2 released during dynamite blasting operations. The combination of unfavorable weather conditions and NO_2 produced acid aerosols that damaged the Nylon.

Nitrogen oxides react with spandex, a synthetic elastomeric fiber, producing a yellow discoloration. This is an inherent property of the fiber rather than a dye-fading phenomenon.

High-particulate-nitrate levels have caused stress-corrosion failures of nickel-brass wire springs on relays used by telephone companys. Failures take place when surface nitrate concentrations exceed 2.4 $\mu g/cm^2$ and the relative humidity is above 50 percent. This problem was first noticed in the Los Angeles area, where airborne nitrate levels are 5 to 15 times greater than in most eastern and midwestern cities.

Another type of this corrosion has been associated with annual average particulate nitrate concentrations of 3.0 and 3.4 $\mu g/m^3$ with corresponding NO_x levels of 124 and 158 $\mu g/m^3$ (0.066 and 0.084 ppm).

The apparent lack of dose-response relationships for the various materials sensitive to NO_x points the way for future research. Until these relationships can be developed, economic estimates will be, at the very best, gross projections.

G. REFERENCES

1. Giles, C. H. The Fading of Colouring Matter. J. Appl. Chem., *15*: 541-550, December 1965.
2. Rowe, F. M. and K. A. J. Chamberlain. The "Fading" of Dyeings on Cellulose Acetate Rayon - The Action of "Burnt Gas Fumes" (Oxides of Nitrogen, etc. in the Atmosphere) on Cellulose Acetate Rayon Dyes. J. Soc. Dyers Colour., *53*: 268-278, July 1937.
3. Seibert, C. A. Atmospheric (Gas) Fading of Colored Cellulose Acetate. Amer. Dyest. Rep., *29*: 363-374, July 1940.
4. Greenspan, F. P. and P. E. Spoerri. A Study of Gas Fading of Acetate Rayon Dyes. Amer. Dyest. Rep., *30*: 645-665, November 1941.
5. Ray, T. K., et al. A Comparison of the Effect on Rayon Fabrics of Various Gases Under Controlled Conditions. Amer. Dyest. Rep., *37*: 391-396, June 1948.

6. 1969 Technical Manual of the American Association of Textile Chemists and Colorist, Volume *45*. 391-396, June 1948.
7. Salvin, V. S., W. D. Paist and W. J. Myles. Advances in Theoretical and Practical Studies of Gas Fading. Amer. Dyest. Rep., *14*: 297-304. May 1952.
8. Mousalli, F. S. and W. J. Myles. Gas Fading of Acetate and Triacetate Prints. Amer. Dyest. Rep., *54*: 1136-1140. December 1965.
9. Salvin, V. S. and R. A. Walker. Relation of Dye Structure to Properties of Disperse Dyes - Part I Anthraquinone Blues. Amer. Dyest. Rep., *48*: 35-37, July 1959.
10. Salvin, V. S. and R. A. Walker. Correlation Between Colorfastness and Structure of Anthraquinone Blue Disperse Dyes. Text. Res., *30*: 381-388, May 1960.
11. Gas Fume Fading. Dyer, Text. Printer, *128*: 89-90, July 1962.
12. Fume Fading. National Institute of Drycleaning, Silver Spring, Maryland, FF-141, 2 pgs., June 1966.
13. Moreley, D. J. Upholstery Fabric Fading by Impurities Present in the Air. Bull. Furniture Ind. Res. Assoc., 2-3, March 1967.
14. A Study of the Destructive Action of Home Gas Fired Dryers on Certain Dyestuffs. Amer. Dyest. Rep., *45*: 471, July 1956.
15. McLendon, V. and F. Richardson. Oxides of Nitrogen as a Factor in Color Changes of Used and Laundered Cotton Articles. Amer. Dyest. Rep., *54*: 305-311, April 1965.
16. Salvin, V. S. Effect of Atmospheric Contaminants on Light-Fastness Testing. Amer. Dyest. Rep., *47*: 450-451, June 1958.
17. Salvin, V. S. Relation of Atmospheric Contaminants and Ozone to Lightfastness. Amer. Dyest. Rep., *53*: 12-20, January 1964.
18. Rabe, P., and R. Dietrich. A Comparison of Methods for Testing the Fastness to Gas Fading of Dyes on Acetate. Amer. Dyest. Rep., *45*: 737-740, September 1956.
19. Salvin, V. S. Testing Atmospheric Fading of Dyed Cotton and Rayon Amer. Dyest. Rep., *58*: 28-29, October 1969.
20. Salvin, V. S. Effect of Atmospheric Contaminants on Fabrics - Dyed and Undyed. Test. Qual. Contr. Pap., *14*: 56-64, 1969.
21. Morris, M. A., M. A. Young and T. Molvig. The Effect of Air Pollutants on Cotton. Text. Res., *34*: 563-564, June 1964.
22. City Finds Nylon Culprit: Blasting Gas. The New York Times, March 11, 1964.
23. Travnicek, Z. Effects of Air Pollution on Textiles, Especially Synthetic Fibers. Int. Clean Air Congr. Proc., I: 224-226, London, Eng., October 1966.
24. Hermance, H. W. Combatting the Effects of Smog on Wire-Spring Relays. Bell Lab. Rec., 48-52, February 1966.
25. McKinney, N. and H. W. Hermance. Stress Corrosion Cracking Rates of a Nickel-Brass Alloy Under Applied Potential, Stress Corrosion Testing. ASTM STP *452*: 274-291, 1967.
26. Hermance, H. W., C. A. Russell, E. J. Bauer, T. F. Egan and H. V. Wadlow. Relation of Air-Borne Nitrate to Telephone Equipment Damage. For publication in "Environmental Science and Technology", 1970.

CHAPTER 8.

EFFECTS OF NITROGEN OXIDES ON VEGETATION

A. INTRODUCTION

The primary importance of nitrogen oxides (NO_X) as phytotoxicants was first inferred 20 years ago in California when photochemical oxidants were shown to have adverse effects on vegetation. The essential role of NO_X in the production of atmospheric oxidants, including peroxyacyl nitrates (PAN) and research activities in vegetative effects of these pollutants are thoroughly reviewed in A P C O Publication AP-63, *Air Quality Criteria for Photochemical Oxidants.* Although the importance of NO_X in photochemical reactions is well recognized, the separate effects on vegetation attributable to the oxides of nitrogen *per se* are difficult to assess. Nevertheless, plant scientists agree that indirect exposure of sensitive plants to NO_X, through the photochemical reactions producing oxidants, constitutes the most significant and widespread mechanism for NOx phytotoxicity. These effects, however, have yet to be thoroughly examined.

Evidence of damage to plants resulting from direct exposures of NO_X in the atmosphere is usually confined to the proximity of specific industrial sources. For example, damage from high ambient levels of nitrogen dioxide (NO_2) has been observed near nitric acid plants. Direct effects due to nitric oxide (NO) and other NO_X components or derivitives have not been delineated in the field.

In view of the foregoing, this chapter is limited to a discussion of the direct effects of NO_X on vegetation determined principally from the results of laboratory studies. The bulk of discussion is focused on the effects of NO_2, but very recent, limited evidence of NO effects are also treated.

Evidence of plant response to phytotoxic levels of NO_X can be divided into three major categories: (1) acute injury, (2) chronic injury, and (3) physiological effects. *Acute injury* is manifested by collapse of cells with subsequent development of identifiable necrotic patterns. Symptoms usually result from short exposures (hours) to varying levels of NO_2, and appear within 2 to 48 hours after exposure.

Chronic injury is caused by intermittent exposure, over long periods, to low concentrations of gas. It results in chlorotic or other pigmented patterns in leaf tissue and may be accompanied by loss of leaves (leaf-drop).

Growth alterations, reduced yields, and changes in quality of plant products are among the *physiological effects* frequently associated with pollutant exposure. At present atmospheric NO_X exposure has not been associated with any of these effects. In the laboratory physiological effects are often measured in terms of more subtle responses such as reduced photosynthesis or changes in rates of transpiration and enzymatic processes. Although most research concerning the physiological effects of NO_X has dealt with laboratory exposures to NO_2, recently the effect of NO exposure has been measured in terms of the reduction in *apparent photosynthesis.* Apparent photosynthesis is measured by the amount of carbon dioxide (CO_2) absorbed by the plant.

Descriptions of plant injury from NO_2 are found in two recent publications containing color plates.[4,18]

B. ACUTE INJURY

Historically, vegetation in the vicinity of nitric acid plants has been injured by gases that escape during plant operations.[1] Leaves develop brown or black spots, especially on the margins. The levels of exposure at which these effects become apparent are unknown, but probably vary widely. Experimental studies have not revealed any direct acute effect of NO exposure on vegetation.

Benedict and Breen[2] exposed ten selected annual and perennial weeds to a mixture of NO and NO_2 for 4 hours. These plants were chosen as indicators of plant injury from air pollutants. Two types of markings developed: (1) a discoloration associated with collapse of cells and necrosis and (2) a general, overall waxy appearance of the leaf. With broad-leaved plants the collapsed, irregular-shaped necrotic markings were intercostal, but nearer the margins of the leaf. Middle-aged leaves usually developed the most markings; but with sunflower, annual bluegrass, and nettleleaf goosefoot, the middle-aged and old leaves were equally sensitive. On mustard plants, the oldest leaves were the most sensitive. In all species, the young leaves were injured least. Mustard was the most sensitive indicator tested, but all plants were somewhat resistant.

Heck[3] fumigated cotton, pinto bean, and endive plants with 1.9 mg/m^3 (1.0 ppm) NO_2 for 48 hours and observed slight, but definite, spotting on the leaves. When concentrations of 6.6 mg/m^3 (3.5 ppm) were used for 21 hours, mild necrotic spots appeared on cotton and bean, but endive leaves were completely necrotic.

Van Haut and Stratmann[4] grouped plants in relation to their sensitivity or resistance to injury by NO_2 and NO. When 60 species of plants were fumigated with a 1:1 mixture of NO and NO_2, they developed gray-green or brownish spots on the leaves, which later became necrotic. Leaf injury reported was similar to that caused by SO_2, except that the NO_x were 2 to 5 times less toxic. Very young and mature leaves were less sensitive than rapidly growing ones. The authors made no mention of duration or concentration of exposures.

MacLean et al.[5] exposed 14 ornamental species and 6 citrus varieties to high concentrations of NO_2 for short periods and classified them according to their susceptibility to injury. The fumigations caused marginal and intercostal necrosis, which was often visible within 1 hour. Collapsed tissues on the upper leaf surface suggested an initial injury to palisade cells. The necrosis later spread throughout the leaf. Citrus responded with the wilting of young leaves and leaf-drop. Older leaves developed marginal and intercostal necrosis. The varieties, listed in the order of decreasing sensitivity, were Marsh seedless grapefruit, pineapple orange, Valencia orange, Tangelo orange, Hamlin orange, and Temple orange. Ornamentals represented a wide range of susceptibility. Tissue collapse and subsequent leaf-drop occurred rapidly in azaleas. Croton plants exposed to 282 mg/m^3 (150 ppm) for 4 hours showed slight intercostal necrosis, but Carissa given a like treatment showed no observable effects. Although defoliation and/or necrosis were complete in several instances, all species survived and axillary buds developed within 2 to 6 weeks.

C. CHRONIC INJURY

Evidence of chronic NO_x injury is limited. No evidence is available that would indicate a chronic effect of NO on plants. Leaf-drop and chlorosis were observed in navel orange trees after 35 days of fumigation with 940 μg/m^3 (0.5 ppm) NO_2 and leaf-drop after 8 months exposure to 470 μg/m^3 (0.25 ppm).[6] Similar effects were found on tobacco, tomato, spinach, and soybean plants exposed to the Los Angeles Basin smog; on the same plants raised in greenhouses equipped with carbon filters; and in irradiation chambers supplied with automobile exhaust from motors equipped with afterburners. Since leaf lesions typical of ozone injury were not observed, Glater postulated that low, constant levels of NO_x were the cause of injury.[7]

D. PHYSIOLOGICAL EFFECTS

1. Observed Responses

All of the evidence of *physiological* effects is based on controlled experiments. Heck, et al.[8] recently fumigated bean and tomato plants with 12.3 mg/m^3 (10.0 ppm) NO and observed an immediate 60 to 70 percent reduction in apparent photosynthesis. An immediate reduction in photosynthesis also occurred with concentrations above 5.6 mg/m^3 (>3 ppm). The rate of CO_2 absorption returned to normal as soon as the fumigations were discontinued. No visible injury developed after exposure.

Hill and Bennett[9] have compared the effect of NO with the effect of NO_2 on the rate of apparent photosynthesis of alfalfa and oats. A *threshold concentration* of about 0.7 mg/m^3 (0.6 ppm) NO or 1.1 mg/m^3 (0.6 ppm) NO_2 was required to reduce CO_2 assimulation. Combining the two gases gave an additive physiological effect. Nitric oxide produced a more rapid reduction in apparent photosynthesis than NO_2, and recovery was more rapid when the fumigation was stopped. Concentrations causing up to 50 percent reduction in apparent photosynthesis after 1 to 2 hours did not induce leaf injury.

Yields of the navel oranges exposed to 470 μg/m^3 (0.25 ppm) for 8 months were reduced.[6]

Taylor and Eaton[10] exposed pinto bean plants to 560 μg/m^3 (0.3 ppm) NO_2 for 10 to 19 days and reported a decrease in dry weight and an increase in unit-weight chlorophyll content. Similar studies with tomato plants exposed to concentrations ranging from 280 to 490 μg/m^3 (0.15 to 0.26 ppm) showed a decrease in dry weight and leaf area, a darker green color, and a strong tendency for the leaves to curl downward.

2. Biochemical Mechanisms

Little work has been done on the biochemical mechanism(s) by which oxides of nitrogen cause direct injury to plants. When NO_2 reacts with water, it forms a stoichiometric mixture of nitrous and nitric acids. This reaction probably occurs as the gas reaches the wet surfaces of the spongy parenchyma in the leaves of plants. *In vitro* studies by Weill and Caldwell[11] on the effect of 1 molar nitrous acid on beta-amylase from barley showed the enzyme to be slowly inactivated. They concluded that easily oxidized groups such as sulfhydryls could be affected. DiCarlo and Redfern[12] conducted more detailed studies with alpha-amylase obtained from *Bacillus subtilis* and concluded that the nitrite was reacting with an essential amino group in proteins. It reacts with two oxidation states of catalase, a hemoprotein from mammalian tissues, and with a peroxidase obtained from horseradish.[13] To date, all enzymatic studies have used one molar nitrate. The low concentrations of nitrate that would occur in plant tissues after exposure to a few parts per million of NO_2 in air have yet to be examined.

E. FACTORS AFFECTING RESPONSE TO NITROGEN DIOXIDE

Several factors such as species of plant, stage of plant development, plant environment (temperature, light, humidity, soil moisture, mineral nutrition), and variable susceptibility within species (variety or clone) influence the degree of injury to vegetation by air pollutants. Depending upon the kind of plant and its environment, one factor may be of much greater importance than another.

Benedict and Breen[2] reported that moist soil conditions caused several times as much injury to their ten test species as did dry conditions.

Taylor and MacLean[14] recognized the increased sensitivity of plants to NO_2 caused by low light intensity and reported that 5.6 mg/m^3 (3.0 ppm) NO_2 in darkness caused as much injury as 11.3 mg/m^3 (6.0 ppm) in light. Czech and Nothdurft,[15] and Van Haut and Stratmann[4] also reported that night fumigations may cause more injury than daytime exposures. Van Haut and Stratmann,[4] reporting on the effect of time of day, found that NO_2 caused more leaf injury during

certain periods than at others. Rye plants were most sensitive from noon to 4 p.m. Oats had a bimodal sensitivity, with more injury occurring from midnight to 2 a.m. than from noon to 2 p.m.

Of special interest is the study by Dunning, et al., which showed that leaf injury occurred on tobacco after a 4-hour exposure to a mixture of 188 $\mu g/m^3$ (0.10 ppm) NO_2 + 260 $\mu g/m^3$ (0.10 ppm) SO_2.[17] If these pollutants act synergistically, perhaps very low concentrations of each in combination with the other will cause plant injury.

F. DOSE-INJURY RELATIONSHIP BETWEEN NITROGEN DIOXIDE AIR POLLUTION AND VEGETATION

Dosage is a measure of the combination of duration of exposure and pollutant concentration; therefore, it is an essential element of air quality criteria. The general types of information needed and problems involved have been thoroughly discussed in a preceding air quality criteria document.[16]

Presently, it is possible to cite only cursory results concerning the effects of variable dosages of NO_x on the physiology of plants. Results available are summarized in Table 8-1.

The acute effects of NO_2 have not been widely studied. Threshold levels of injury reported are tabulated in Table 8-2. The *threshold injury* level is defined as the exposure necessary to injure 5 percent of the area of the leaf (upper surface). Preliminary time-concentration curves for several sensitivity groupings have been developed. Based on these curves, Table 8-3 suggests exposure durations and concentrations necessary to produce injury in sensitive, low-sensitive, and resistant plants at the threshold-injury level. Table 8-4 gives a complete list of plants that have been studied, and places them in three NO_2-susceptibility classes on the basis of the projections in Table 8-3.

Heck, et al.[6] present a modified model for reporting injury to vegetation from a group of common air pollutants, including nitrogen dioxide. The model recognizes the separate influence of time and concentration and permits the development of a three-dimensional injury-response surface:

$$C = A_0 + A_1 I + A_2/T$$

C = Concentration, ppm

A_0, A_1, A_2 = Constants (partial regression coefficients) specific for pollutant plant species, and environmental conditions.

I = Percent injury

T = Time, hours

Constants developed for selected test species are shown in Table 8-5. These species are classed in susceptibility groupings based on Table 8-3 projections. The values of the constants are dependent upon the prevailing environmental conditions. The experimental design suggests that these equations can be used by control agencies to predict injury to vegetation under environmental conditions when vegetation would be most sensitive (high humidity and temperatures between 75° and 90° F). These equations are preliminary attempts to place usable tools in the hands of control agencies. They should not be applied to time periods less than 30 minutes or over 8 hours, or for concentration averages below 0.5 or above 25.0 ppm.

G. NEED FOR FUTURE RESEARCH

Plants vary in sensitivity to NO_x. Further testing in the high concentration range [28 to 47 mg/m^3 (15 to 25 ppm)] for more than 1 hour will yield little more information, except to show the exact kind of leaf lesions that will occur on a given species. This information would only be worthwhile for evaluating injury caused by spills, rocket fuels, accidental release of concentrated gas, or elevated atmospheric levels in the vicinity of poorly controlled, industrial use or production of NO_2.

Table 8-1. CHRONIC INJURY AND PHYSIOLOGICAL EFFECTS OF NO AND NO_2

Gas	Plant	Exposure: Concentration, ppm	Exposure: Concentration, mg/m^3	Exposure: Time,[a] days	Effects	Reference
NO	**Gramineae**					
	Oat (*Avena sativa*, L.)	0.6	0.7	V	Reduced rate of apparent photosynthesis	9
	Leguminosae					
	Alfalfa (*Medicago sativa*, L.)	0.6	0.7	V	Reduced rate of apparent photosynthesis	9
	Pinto Bean (*Phaseolus vulgaris*, L.)	3-10	3.7-12.3	V	Immediate reduction up to 70% in apparent photosynthesis	8
	Solanaceae					
	Tomato (*Lycopersicon esculentum* Mill)	3-10	3.7-12.3	V	Immediate reduction up to 70% in apparent photosynthesis	8
NO_2	**Gramineae**					
	Oat (*Avena,sativa*, L.)	0.6	1.1	V	Reduced rate of apparent photosynthesis	9
	Leguminosae					
	Alfalfa (*Medicago sativa*, L.)	0.6	1.1	V	Reduced rate of apparent photosynthesis	9
	Pinto Bean (*Phaseolus vulgaris*, L.)	0.3	0.6	10-19	Decrease in dry weight, increase in chlorophyll content per unit weight	10
	Rutaceae					
	Navel orange (*Citrus sinensis*, Osbeck)	0.5-1.0	0.9-1.9	35	Chlorosis of leaves, extensive leaf-drop	6
		0.25	0.47	240	Increased leaf-drop, reduced yield	
	Solanaceae					
	Tomato (*Lycopersicon esculentum*, Mill)	0.15-0.26	0.3-0.5	10-22	Decrease in dry weight and leaf area, increase in chlorophyll, downward curvature of leaves	10

[a]V = variable.

Table 8-2. PLANT THRESHOLD SUSCEPTIBILITY TO ACUTE INJURY FROM NO_2

Species	Variety	Threshold Concentration, ppm	Threshold Concentration, mg/m^3	Threshold Time, hr	Effects[a]	Reference
Compositae						
Endive (*Cichorium endivia*, L.)	Ruffee	1	1.9	12	0	3
		1	1.9	48	1	3
		3.5	6.6	21	3	3
Lettuce (*Lactuca sativa*, L.)	Ruby	4	7.5	1.5	1	8
Mustard (*Brassica arvensis*, Rabenh.)		20	37.6	4	2	2
Radish (*Raphanus sativus*, L.)	Cherry Belle	5	9.4	0.5	1	8
Gramineae						
Oats (*Avena sativa*, L.)	329-80	4	7.5	1	1	8
Wheat (*Triticum vulgare*, Vill)	Wells	1	1.9	7	1	8
Leguminosae						
Alfalfa (*Medicago sativa*, L.)		30	56.4	1	1	15
Bean (*Phaseolus vulgaris*, L.)	Pinto	10	18.8	4	1	10
		1	1.9	12	0	3
		1	1.9	48	1	3
		3.5	6.6	21	1	3
Malvaceae						
Cotton (*Gossypium hirsutum*, L.)	Rex	1	1.9	12	0	3
		1	1.9	48	1	3
		3.5	6.6	21	1	3
Cotton (*Gossypium sp.*, L.)	Paymaster	3	5.6	2	1	8
Rutaceae						
Navel orange (*Citrus sinensis*, Osbeck)	–	1	1.9	35	2	6
Solanaceae						
Tomato (*Lycopersicon esculentum* Mill)	–	8	15	1	1	8
Tobacco (*Nicotiana glutinosa*)	–	2.4	4.5	5	0	10
		2.3	4.3	8.66	3	10
Pepper (*Capsicum frutescens*, L.)	Cal chili 505	5.5	10.4	1.5	1	8

[a]Severity of injury: 1 = slight, 2 = moderate, and 3 = severe.

Table 8-3. PROJECTED NO_2 EXPOSURES FOR 5 PERCENT INJURY LEVELS ON SELECTED VEGETATION[a]

Time, hr	Concentrations producing injury					
	Sensitive,[b]		Low-sensitive,		Resistant,	
	ppm	mg/m^3	ppm	mg/m^3	ppm	mg/m^3
0.5	6-10	11.3-18.8	9-17	16.9-32.0	$\geq$16	$\geq$30.1
1.0	4-8	7.5-15.0	7-14	13.1-26.3	$\geq$13	$\geq$24.4
2.0	3-7	5.6-13.2	6-12	11.3-22.6	$\geq$11	$\geq$20.7
4.0	2-6	3.8-11.3	5-10	9.4-18.8	$\geq$ 9	$\geq$16.9
8.0	2-5	3.8-9.4	4-9	7.5-16.9	$\geq$ 8	$\geq$15.0

[a]Reference 6.

[b]Plant type.

Table 8-4. RELATIVE PLANT SENSITIVITY TO NO_2 INJURY

Species	Variety	Reference
Sensitive plants		
Apocynaceae		
Periwinkle (*Vinca minor*, L.)	Bright Eyes	8
Balsaminaceae		
Sultana (*Impatiens sultani*, Hook.)	White Impatiens	8
Begoniaceae		
Begonia (*Begonia Rex*, Putz.)	Thousand Wonders White	8
Chenopodiaceae		
Spinach (*Spinacia oleracea*, L.)	Bloomsdale Long Standing	8
Compositae		
Chrysanthemum (*Chrysanthemum*, sp.)	Oregon	8
Lettuce (*Lactuca sativa*, L.)	Ruby	8
	Early Prize Head	8
	Iceberg	8
	Grand Rapids	8
	Great Lakes	8
	Romaine	8
	Burpee Bibb	8
	Black Seeded Simpson	8
	Butter King	8
	Big Boston	8
	Butter Crunch	8
Cruciferae		
Broccoli (*Brassica oleracea botrytis*, L.)	Calabrese	8
Radish (*Raphanus sativus*, L.)	Cherry Belle	8
Radish (*Raphanus sativus*, L.)	–	8

Table 8-4 (continued). RELATIVE PLANT SENSITIVITY TO NO_2 INJURY

Species	Variety	Reference
Gramineae		
Bromegrass (*Bromus inermis*, L.)	Sac Smooth	8
Oats (Avena *Sativa*, L.)	Clintland 64	8
	329-80	8
	Pendek	8
Wheat (*Triticum vulgare*, V.11.)	Wells	8
Solanaceae		
Pepper (*Capsicum frutescens*, L.)	Cal Chili 505	8
Low-sensitive plants		
Chenopodiaceae		
Spinach (*Spinacia oleracea*, L.)	Noble	8
	Early Hybrid # 7	8
	American	8
Compositae		
Dahlia (*Dahlia variabilis*)	–	8
Cruciferae		
Mustard (*Brassica arvensis*, Rabenh)	–	8
Cucurbitaceae		
Cucumber (*Cucumis sativus*, L.)	Heinz Pickling	8
	Black Diamond	8
	London	8
Musk melon (*Cucumis melo*, L.)	–	8
Squash (*Cucurbita*, sp.)	Early White Bush	8
	Golden Summer Crookneck	8
Gramineae		
Barley (*Hordeum vulgare*, L.)	–	8
Leguminosae		
Bean (*Phaseolus vulgaris*, L.)	Pinto	10
Lima Bean (*Phaseolus lunatus*, L.)	Henderson	8
	B F	8
	Thaxter	8
Soybean (*Glycine max*, merr.)	Scott	8
	Bonsei	8
	Kanrich	8
Malvaceae		
Cotton (*Gossypium*, sp.)	–	8
Cotton (*Gossypium hirsutim*, L.)	Acala 4-42	8
Solanaceae		
Tobacco (*Nicotiana tabacum*, L.)	White Gold	8
	Bel - B	8
	Beltsville W3	8
	Beltsville C	8

Table 8-4 (continued). RELATIVE PLANT SENSITIVITY TO NO_2 INJURY

Species	Variety	Reference
Tomato (*Lycopersicon esculentum*, Mill.)	Roma	8
	A	8
	B	8
	C	8
	D	8
	Pearson Improved	8
Resistant plants		
Amaranthaceae		
Pigweed (*Amaranthus retroflexus*)	–	2
Caryophyllaceae		
Chickweed (*Stellaria media*, cyrill)	–	2
Chenopodiaceae		
Beet (*Beta vulgaris*, L.)	Perfected Detroit	8
Lamb's - quarters (*Chenopodium album*, L.)		2
Nettle-leaf goosefoot (*Chenopodium murale*)		2
Compositae		
Dandelion (*Taraxacum officinale*, Weber)	–	2
Sunflower (*Helianthus annuus*, C.)		2
Cucurbitacea		
Cucumber (*Cucymis salivus*, L.)	Long Marketeer	8
Ericaceae		
Azalea (*Rhododendron*, sp.)	Alaska	8
Euphorbiaceae		
Croton (*Codiaeum*, Juss)	–	5
Gramineae		
Annual bluegrass (*Poa annua*, L.)		2
Corn (*Zea mays*, L.)		8
Kentucky blue grass (*Poa pratensis*)	Pioneer and Golden Cross	2
Orchard grass (*Dactylis glomerata*, L.)	Potomac	8
Sorghum (*sorghum*, sp.)	Martin	8
Malvaceae		
Cheeseweed (*Malva parviflora*, L.)	–	2
Solanaceae		
Tobacco (*Nicotiana tabacum*, L.)	Beltsville B	8
	W3	8
	Burley 21	8

Table 8-5. NO_2 TIME-CONCENTRATION RESPONSE EQUATIONS (0.5 to 7 hours)[a]

$C = A_0 + A_1 I + A_2/T$

Species	A_0	A_1	A_2	R^2	D	E	D+E
Sensitive plants							
Oats (Clintland 64)	1.45	0.13	24	0.76	4.5	2.3	6.8
Radish (Cherry Belle)	2.40	0.14	1.02	0.83	4.1	3.2	7.3
Oats (329-80)	1.75	0.15	3.24	0.56	5.7	2.9	8.6
Bromeglass (Sac Smooth)	2.49	0.16	1.9	0.71	5.2	3.5	8.7
Begonia	2.45	0.15	2.99	0.63	6.2	3.5	9.7
Chrysanthemum	3.16	0.16	2.14	0.72	6.1	3.7	9.8
Oats (Pendek)	2.79	0.14	2.88	0.50	6.4	3.8	10.2
Wheat (wells)	2.80	0.13	2.94	0.52	6.4	3.8	10.2
Sultana	3.93	0.13	1.73	0.67	6.3	4.8	11.1
Broccoli	3.07	0.20	2.94	0.53	7.0	4.5	11.5
Periwinkle	2.92	0.23	3.02	0.55	7.1	4.5	11.6
Low-sensitive plants							
Cotton (Paymaster)	3.97	0.23	1.94	0.58	7.1	5.3	12.4
Cotton (Acala 4-42)	3.68	0.22	3.15	0.50	7.9	5.2	13.1
Tobacco (Bel B)	3.62	0.21	3.98	0.38	8.7	5.2	13.9
Tobacco (Bel W_3)	3.65	0.18	4.40	0.31	9.0	5.2	14.2
Tobacco (White Gold)	4.03	0.30	3.56	0.40	9.1	6.0	15.1
Resistant plants							
Azalea	3.79	1.90	3.39	0.33	16.7	13.8	30.5
Corn (Pioneer)	2.60	2.70	4.10	0.37	20.3	16.5	36.8

[a]See text for explanation of equation.

R^2 = coefficients of determination; represent percent variation in model.

D = concentrations that will cause 5 percent injury in 1 hour.

E = concentrations that will cause 5 percent injury in 8 hours.

D+E= Basis for placing the plant in a specific susceptibility category; exact cut-off sums not yet established.

To determine in detail how lower levels of NO_x affect vegetation will require much time and effort. Representative species must be grown under a variety of carefully controlled, reproducible, environmental conditions. The plants selected as standards would then have to be fumigated with specific concentrations of NO_2 and/or NO for specified times.

Evaluation would require preliminary *in vitro* work to determine the effect of NO_x gases on enzymes, growth regulators, plant pigments, and other metabolic systems. The same reactions could then be looked for in selected fractions of the treated plants and in the untreated, but similarly grown, controls. Enzymatic effects should be pursued intensively, so that the way in which the particular pollutant affects plant metabolism could be defined. As biochemical effects were understood, correlations could be made with reactions known to occur in other organisms such as mammals, insects, and birds.

The tentative findings by Heck involving synergism show that studies should be broadened to explore more fully the interactions between NO_x and other air pollutants.

H. SUMMARY

Many kinds of plants develop severe acute leaf injury (lesions) when exposed to concentrations of NO_2 greater than 47 mg/m^3 (25 ppm) for a 1-hour period. Though characteristic for each plant, the lesions are difficult to distinguish from similar injury caused by SO_2. The fact that very young and mature leaves are more resistent to NO_2 than those that are expanding rapidly may provide aid in identifying NO_2 injury.

Under controlled growth conditions, the injury-threshold value for NO_2 (level that injures 5 percent of the leaf area) for certain sensitive plants is 7.5 to 15.0 mg/m^3 (4 to 8 ppm) for 1 hour. Increasing the exposure-duration decreases this threshold concentration; with a rough time-concentration relationship in which 4.3 to 6.6 mg/m^3 (2.3 to 3.5 ppm) NO_2 administered to sensitive plant species for 8 to 21 hours, or 1.9 mg/m^3 (1 ppm) NO_2 for 48 hours, causes leaf injury. Continuous fumigation with 940 μg/m^3 (0.5 ppm) NO_2 for 35 days resulted in leaf drop and chlorosis in citrus, but no actual necrotic lesions developed.

The effects of exposure to low levels of NO_2 for extended periods are less evident. Recently completed studies suggest that 470 μg/m^3 (0.25 ppm) or less of NO_2 supplied continuously for 8 months increased leaf-drop and reduced yield of navel oranges. The degree of injury from lower, atmospheric concentrations of NO_2 remains to be determined. Very mild chronic effects, resulting from fumigation of pinto bean plants with 560 μg/m^3 (0.3 ppm) and of tomato plants with 280 to 490 μg/m^3 (0.15 to 0.26 ppm) NO_2, approximate the possible effects of persistently high ambient concentrations.

The limited information on the effectiveness of NO in reducing apparent photosynthesis indicates that it would reduce the growth of plants if concentrations in the range of 3.8 to 7.5 mg/m^3 (2 to 4 ppm) persisted continuously.

I. REFERENCES

1. Thomas M. D. Gas Damage to Plants. Ann. Rev. Plant Physiol.*2*:293-322, 1951.
2. Benedict, H. M. and W. H. Breen. The Use of Weeds as a Means of Evaluating Vegetation Damage Caused by Air Pollution. Proc. 3rd Nat. Air Pollut. Symp., Pasadena, Calif., April 18-20, 1955. 177-190
3. Heck, W. W. Plant Injury Induced by Photochemical Reaction Products of Propylene-Nitrogen Dioxide Mixtures. J. Air Pollut. Contr. Ass. *14*:255-261, July 1964.
4. Van Haut, H. and H. Stratmann. Experimental Investigations of the Effect of Nitrogen Dioxide on Plants. (Experimentelle untersuchungen uber die Winkunge von Stuckstoffdioxid auf Pflanzen). Transactions of the Land Inst. of Pollution Control and Soil Conservation of the Land of North Rhine-Westphalia, (Essen). No. 7:50-70, 1967.
5. MacLean, D. C., et al., Effects of Acute Hydrogen Fluoride and Nitrogen Dioxide Exposures on Citrus and Ornamental Plants of Central Florida. Environ. Sci. Technol. *2*:444-449, June 1968.
6. Thompson, C. R., et al., Effects of Continuous Exposure of Navel Oranges to NO_2. Atmos. Environ. In Press, 1970.
7. Glater, R. A. B. Smog and Plant Structure in Los Angeles County, March 1970. Reports Group, School of Engineering and Applied Science. University of California at Los Angeles, p. 1-39, Report No. 70-17.
8. Heck, W. W., O. C. Taylor, and D. T. Tingey, Response of Plants to Acute Doses of Nitrogen Dioxide. BioScience, *21* (In Press, 1971).
9. Hill, A. C. and J. H. Bennett. Inhibition of Apparent Photosynthesis by Nitrogen Oxides. Atmos. Environ. (In Press, 1970).
10. Taylor, O. C. and F. M. Eaton. Suppression of Plant Growth by Nitrogen Dioxide. Plant Physiol. *41*:132-135, January 1966.
11. Weill, E. C. and M. L. Caldwell. A Study of the Essential Groups of B-amylase. J. Amer. Chem. Soc. *67*:212-214, February 1945.
12. DiCarlo, F. J. and S. Redfern. α-Amylase from *B. subtilis* II. Essential Groups. Arch. Biochem. *15*:343-350, 1947.
13. Nicholls. P. and G. R. Schonbaum "Catalases" In: Enzymes *8*, Boyer, P. D. et al (eds.) Academic Press. 1963, 180-181.
14. Taylor, O. C., and D. C. MacLean. Nitrogen Oxides and the Peroxyacyl Nitrates. *In*: "Recognition of Air Pollution Safety to Vegetation: A Pictorial Atlas." J. S. Jacobson and A. C. Hill, eds. Air Pollution Control Association, Pittsburg, Pennsylvania, pp. E1-E14, 1970.

15. Czeck, M. and W. Nothdurft. Investigation of the Damage to Field and Horticultural Crops by Chlorine, Nitrous and Sulfur Dioxide Gases. (Untersuchungen uber Schadigungen landwirtschaftlicher und gartnerischer Kulturpflanzen durch Chlor-Nitrose-und Schwefeldioxydgase). Landwirtshaftliche Forschung. Darmstadt,, *4*: No. 1, 1-36, 1952.
16. Air Quality Criteria for Photochemical Oxidants. Nat. Air Pollut. Contr. Admin., Government Printing Office, Washington, D. C. AP-63, 6-1 to 6-23, March, 1970.
17. Dunning, J. A., D. T. Tingey, and R. A. Reinert. Nitrogen Dioxides and Sulfur Dioxide Interact to Injure Horticultural and Agronomic Crops. Hort. Sci. *5*:333, 1970.
18. Recognition of Air Pollution Injury to Vegetation: A Picturial Atlas. Jacobson, J. S. and A. C. Hill 1, Ed. Air Pollution Control Association, Publ. 1970.

CHAPTER 9.

TOXICOLOGICAL EFFECTS OF NITROGEN OXIDES

A. INTRODUCTION

Data from human and animal studies indicate that both nitric oxide (NO) and nitrogen dioxide (NO_2) have untoward effects on health. Animal mortality studies indicate that NO_2 is about four times more toxic than NO.[1] This chapter represents an attempt to evaluate health studies and to identify the probable hazardous doses of both pollutants.

The potential role of nitrogen oxides (NO_X) in atmospheric reactions that result in photochemical smog is discussed in chapter 4. Although considerable information is available regarding the toxicology of some of these photochemical products, they are not discussed in this chapter. Many are treated in AP-63, *Air Quality Criteria for Photochemical Oxidants,* and AP-64, *Air Quality Criteria for Hydrocarbons.*

B. NITRIC OXIDE

No cases of human poisoning due to NO have been reported in the scientific literature, although NO is undoubtedly included in occupational exposures to NO_X. When present in high concentrations, NO is rapidly converted to NO_2, a property that makes it difficult to control NO concentrations in laboratory experiments.

In animals, extremely high concentrations of NO produced central nervous system paralysis and convulsions.[2] Mice exposed to 3,075 mg/m^3 (2,500 ppm) were narcotized in 6 to 7 minutes and died within 12 minutes, but when the narcotized animals were returned to fresh air after 4 to 6 minutes of exposure, they recovered rapidly.

Data from limited experiments on guinea pigs indicate no effect on pulmonary function from 4-hour exposures to concentrations of 19.7 to 94.0 mg/m^3 (16 to 50 ppm) NO.[3]

1. Methemoglobin Increase

Methemoglobin (MeHb) does not bind oxygen; hence, it reduces the oxygen-carrying capacity of blood when it replaces normal hemoglobin. Low concentrations of MeHb ranging from 0.01 to 0.5 gram MeHb per 100 milliliters (g/100 cc) are usually present in normal human blood,[4] although the range in normal subjects has been reported to vary between 0 and 8 percent of total hemoglobin[5] (i.e., up to 1.2 g MeHb/100 cc, assuming a hemoglobin value of 15 g/100 cc). The earliest clinical evidence of MeHb in human blood is cyanosis, which begins when the concentration reaches 10 to 15 percent of the total hemoglobin, a concentration not likely to result from exposure to ambient concentrations of NO_X. Symptoms such as exertional dyspnea (labored breathing),which reflects hypoxia (low oxygen supply) are not likely to appear until blood levels of MeHb reach 30 to 45 percent of total hemoglobin.[4]

High quantities of MeHb and cyanosis were detected in the blood of animals that died as a result of poisoning from exposures to 1,500 mg/m^3 (1,200 ppm) NO.[6,7] Evidence concerning MeHb formation following exposure to lower concentrations of NO is conflicting.

The significance of low blood levels of MeHb is not well understood. In chapter 10, the use of blood levels of MeHb as an index of exposure to ambient NO_X is discussed,[8] but few epidemiological investigators actually use this parameter in their experimental design.

In vitro, NO combines with hemoglobin to form NO-hemoglobin (NO-Hb) and with

MeHb to form NO-methemoglobin (NO-MeHb), both of which have biological half-lives of about 2 days. A preliminary examination of the blood of rats exposed for 1 and 9 days to 12.3 mg/m^3 (10 ppm) NO in air by electron-spin resonance revealed neither NO-Hb nor NO-MeHb.[9]

2. Enzyme Inhibition

A concentration of about 24.6 mg/m^3 (20 ppm) NO inhibited hydrogenase activity of the bacteria *Proteus vulgaris*, both *in vivo* and *in vitro*.[10] This activity could be restored by the addition of $Na_2S_2O_4$, which reduces NO to nitrous oxide (N_2O), except at concentrations of 12.3 x 10^3 mg/m^3 (10,000 ppm) NO and above where inhibition was irreversible.

Although ambient levels of NO are not inherently toxic, toxicological potential lies in its relationship to NO_2 which is discussed in AP-63, *Air Quality Criteria for Photochemical Oxidants*.[11]

C. NITROGEN DIOXIDE

1. Effects in Animals

a. Mortality

Many early studies produced negative evidence of NO_2 toxicity in animals. This may have been due to the presence of large amounts of NO in the experimental mixtures, for recent studies have produced more reliable and positive evidence of toxicity. Several factors affecting the mortality due to NO_2 are discussed in the following section.

(1) Length of exposure and concentration (ct). Short exposures of rats to high concentrations are more toxic than equivalent exposures to low concentrations for longer times. This is illustrated in Table 9-1.[12]

Most investigations involving inhalation of high concentrations of NO_2 have implicated pulmonary edema as the major cause of death. When 112 animals of various species were exposed to 56.4 to 1,880 mg/m^3 (30 to 1,000 ppm) NO_2, 84 animals died, 74 from pulmonary edema, 5 from asphyxia, and 5 from pneumonitis (inflammation of lung tissues) (Table 9-2). Exposure to 56.4 mg/m^3 (30 ppm) NO_2 for 3 hours produced no apparent immediate or delayed harmful effects in guinea pigs. The harmful effects of 2- to 3-hour exposures of 103.4 mg/m^3 (55 ppm) NO_2 were questionable in experiments with rats and mice. None were found at the

Table 9-1. COMPARISON OF LETHAL LEVELS OF ACUTE EXPOSURE OF MALE RATS TO NO_2[12]

Number of experiments	Exposure time, min	LC_{50},[a,b] ppm	LC_{50},[a,b] mg/m^3	LCt_{50},[b,c] ppm x min
3	2	1,445[d]	2,715	2,890
2	5	833[d]	1,566	4,165
6	15	420 (362-487)	790 (680-916)	6,300 (5,430-7,305)
10	30	174 (154-197)	325 (290-370)	5,220 (4,620-5,910)
10	60	168 (153-185)	315 (290-350)	10,080 (9,180-11,100)
7	240	88 (79-99)	165 (140-185)	21,120 (18,960-23,760)

[a] LC_{50} represents the concentration lethal to 50 percent of the animals.

[b] 95 percent confidence limits in parenthesis.

[c] LCt_{50} represents the exposure (concentration x time) lethal to 50 percent of the animals.

[d] No confidence limits.

Table 9-2. TIME NECESSARY TO PRODUCE DEATH IN ANIMALS[a] EXPOSED TO HIGH CONCENTRATIONS OF NO_2[13]

Concentration, ppm	Concentration, mg/m^3	Time until death, min	% deaths
30	56.4	----	0
100	188.0	318	74
150	282.0	90	70
400	752.0	58	92
600	1,128.0	32	93
800	1,524.0	29	100
1,000	1,880.0	19	100

[a]Animals included the following species: cats, guinea pigs, mice, rats, and rabbits. Death occurred in 84 of 112 animals.

same concentration with rabbits, cats, and guinea pigs.[13]

It is rare that any species can withstand exposure to NO_2 concentrations of 188 to 1,880 mg/m^3 (100 to 1,000 ppm). In experiments with these concentrations, death occurred after 10 minutes to 21 hours of exposure, depending on the animal.[12] Four-hour exposure to 99.6 mg/m^3 (53 ppm) NO_2 was fatal to rats and 122.2 to 141 mg/m^3 (65 to 75 ppm) NO_2, to dogs.

(2) Temperature. An ambient temperature increase of about 11° C (20° F) increases the toxicity of NO_2 for rats by about 25 percent.[14]

(3) Presence of other irritants. The survival time for rabbits was increased from 60 to 140 minutes when nonlethal amounts of sulfur dioxide (SO_2) were added to the NO_2 dose that caused 50 percent mortality.[14]

b. Respiratory tract effects

(1) Changes in pulmonary function. Short-term (less than 4 hours) exposure to NO_2 produced reversible changes in pulmonary function.[3,15] In general, both short-term and continuous exposures caused respiratory rates to increase and tidal volumes (the volume of air inhaled in an average single breath) to decrease during exposures to concentrations ranging from 1.5 (0.8 ppm) to 94 mg/m^3 (50 ppm). The degree of response varied with the exposure and the animal (Table 9-3 and Figure 9-1).

Other pulmonary functions have been examined, but show no evidence of NO_2 effects. Guinea pigs exposed to 9.4 mg/m^3 (5 ppm) NO_2 for either 4 or 7.5 hours a day, 5 days a week, for periods up to 5.5 months had no changes in expiratory flow resistance.[16] Four rabbits exposed to 47.0 mg/m^3 (25 ppm) NO_2 continuously for 18 months had a transient increase in the rate of oxygen consumption. The rate reverted to normal within 48 to 72 hours after exposure. In 16 rabbits exposed to 1.9 and 9.4 mg/m^3 (1 and 5 ppm) NO_2 for the same length of time, no change in oxygen consumption occurred.[17]

Beagles exposed to 0.9 to 1.9 mg/m^3 (0.5 to 1.0 ppm) NO_2 plus 245 $\mu g/m^3$ (0.2 ppm) NO, and to 2.8 to 3.8 mg/m^3 (1.5 to 2.0 ppm) NO_2 plus 245 $\mu g/m^3$ (0.2 ppm) NO for 16 hours a day for 18 months, showed no changes in body weight, or in carbon monoxide (CO)-diffusing capacity, compliance, or total expiratory resistance of the lungs when compared with a control group of 20 breathing filtered air.[18]

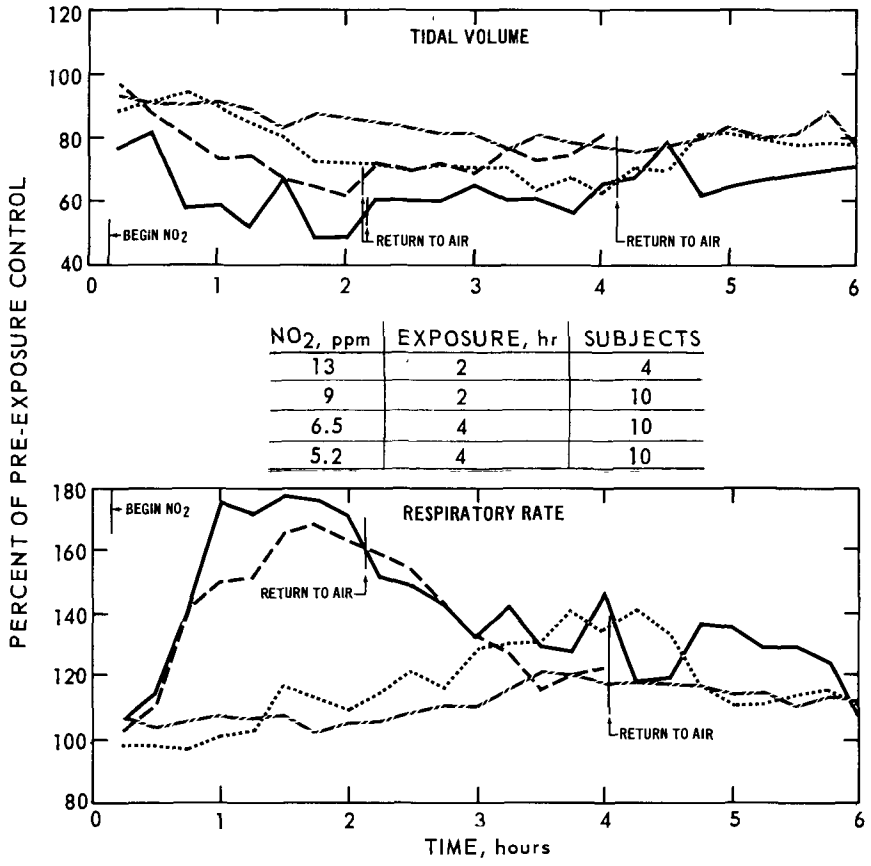
(2) Chemical effects.

(a) Structural proteins—NO_2 can alter the configuration of the lung tissue structural proteins, collagen and elastin.[19] One animal from each of five sets of four litter-mate female rabbits served as a control; one breathed 1.9 mg/m^3 (1 ppm) NO_2 for 1 hour; and the remaining two were exposed to 9.4 mg/m^3 (5 ppm) NO_2 for 1 hour. All the animals were sacrificed immediately after exposure, except one in the last group, which was sacrificed 24 hours after exposure. The lungs were excised and converted to a lipid-free powder from which collagen and elastin were isolated by a combination of solvent-extraction and enzymatic-hydrolysis methods. Differential ultraviolet spectrophotometry indicated that

Table 9-3. CHANGES IN PULMONARY FUNCTION IN ANIMALS EXPOSED TO SUB-LETHAL DOSES OF NO_2

Animal	Concentration, mg/m^3	Concentration, ppm	Exposure time	Respiratory rate	Tidal volume	Minute volume	Time for maximum effect, min	Recovery: Amount	Recovery: Time	References
Guinea pigs	9.8	5.2	2-4 hr	+[a]	—	0	180	NO_2	Within	3
	12.2	6.5					135		4 hr	
	16.9	9.0					90			
	24.4	13.0					60			
Squirrel monkeys	18.8 and	10 and								
	28.2	15	2 hr	0	—	—	n.a.	complete	24-48 hr	15
	65.8 and	35 and								
	94.0	50	2 hr	++	—	n.a.	n.a.	complete	up to 72 hr	
Rats	1.5	0.8	Continuous, lifetime	+(20%)	n.a.	n.a.	n.a.	none	Lifetime	75
Monkeys (M. *speciosa*)	3.8 and 16.9	2 and 9	Continuous	±	—	n.a.	n.a.	none	2 yr	37
Squirrel monkeys	9.4	5	2 mo	+	—	0	n.a.	n.a.		
	18.8	10	1 mo	+	+	+	n.a.	n.a.		55

[a]Key: + Increased
- Decreased
0 Unchanged
n.a. Not available



NO_2, ppm	EXPOSURE, hr	SUBJECTS
13	2	4
9	2	10
6.5	4	10
5.2	4	10

Figure 9-1. Effect of NO_2 on tidal volume and respiratory rate of guinea pigs.[10]

the molecular structures of both collagen and elastin were altered in both exposure groups. Similarity between the spectra of collagen and elastin from the animal sacrificed 24 hours after exposure and those from the control animal indicated that the changes were reversible under the conditions of the experiment. The authors suggested that metabolic activity of the lung tissue was reduced in the highly acidic environment produced by exposure to NO_2 and speculated that repeated exposure to NO_2 with concurrent repeated denaturation of collagen and elastin may be a factor in the etiology of pulmonary emphysema.

Rabbits exposed on a schedule of 470 $\mu g/m^3$ (0.25 ppm) NO_2, 4 hours a day for 6 days, and sacrificed immediately, developed structural changes in the lung collagen, as determined by electron microscopy. These changes were not reversible and were apparent in animals sacrificed 7 days after the final exposure.[20]

(b) Lipid peroxidation—Short-term low-level exposures [4 hours at 1.9 mg/m^3 (1 ppm)] produced evident peroxidation in extracts of lipids from rat lungs.[21] Peroxidation was maximum 24 hours after exposure, and lasted for at least 24 hours more. In six daily 4-hour exposures to 1.9 mg/m^3 (1ppm), the lipid peroxidation increased, possibly as a cumulative effect. The significance of this peroxidation is not yet established; however, rats fed a vitamin-E-deficient diet and exposed to NO_2 had more peroxidation in both surfactant and tissue lipids than did

those on vitamin-E-supplemented diets.[22] Vitamin E is an antioxidant.

(3) General Pathological Effects. Pathologically, all animal lungs demonstrate the same general pattern of response to NO_2, irrespective of the duration of exposure, but the severity tends to increase as the concentration increases. An inflammatory reaction characterized by macrophage infiltration and epithelial degeneration occurs during the 24-hour period immediately following exposure and can develop into pulmonary edema at sufficiently high concentrations. Subsequent regeneration follows. Such changes were described in rabbits exposed to 188 mg/m^3 (100 ppm) and in rats and guinea pigs exposed to 141 to 150 mg/m^3 (75 to 80 ppm) NO_2 for 2 hours.[23] In experiments in which rats and guinea pigs were exposed to 28.2 to 37.6 mg/m^3 (15 to 20 ppm) and rabbits to 47.0 mg/m^3 (25 ppm) NO_2 for 2 hours a day for 5 successive days, initial edema and inflammation were not as severe as they were in the single-dose experiments; but chronic peribronchial and perivascular inflammation followed later. On the fourth day after epithelium was destroyed by a solitary high dose, lung tissue did repair itself in both rabbits and guinea pigs. Regeneration became intense and appeared to be complete in 2 weeks; after a series of high intermittent doses repair was slower. Animals exposed intermittently during a period of 21 months required up to 3 weeks after the last exposure to recover.[23,24]

(4) Cellular Changes.

(a) Structural damage–short-term exposure–In short-term exposures to low concentrations (0.5 ppm for 4 hours or 1 ppm, 1 hour) rats sustained reversible lung-tissue change.[25] In tissues from animals sacrificed immediately after exposure, the mast cells were ruptured and disoriented, and showed loss of cytoplasmic granules. This occurred primarily in the pleura, bronchi, and surrounding tissues, but most markedly in the mediastinum. This response seemed reversible, since animals sacrificed 24 to 27 hours after exposure appeared to have only a few ruptured mast cells. The investigators considered that the release of granular material from the lung mast cells in response to NO_2 inhalation signified the potential onset of an acute inflammatory reaction.

After 4 hours exposure to 32.0 mg/m^3 (17 ppm) NO_2, the appearance of bits of free fibrin in both alveolar spaces and tissue indicated transient leakage of plasma from rat alveolar capillaries. With continued exposure in the same experiment, researchers noted that focal injury to type I alveolar epithelial cells was exaggerated within 48 hours, as bits of amorphous cellular debris appeared in ductal alveoli. The facts that fibrin and debris tended to disappear and that injured cells healed as exposure continued suggests the development of tolerance.[26]

Primary lesions appeared in lung alveoli of squirrel monkeys exposed for 2 hours to 18.8 to 94.0 mg/m^3 (10 to 50 ppm) NO_2. Progressive alveolar expansion occurred with increasing concentrations of NO_2. At 18.8 mg/m^3 (10 ppm) NO_2, many septal breaks appeared and the alveoli expanded markedly. In some areas, large air vesicles with extremely thin septal walls were seen. Other tissues appeared to be normal. At 28.2 mg/m^3 (15 ppm) NO_2 alveolar tissue was expanded with minimal wall thinning and patchy interstitial infiltration with lymphocytes. The bronchioles were normal. At 65.8 mg/m^3 (35 ppm) NO_2 areas of lung were collapsed and alveolar septa became very basophilic. In other areas, the alveoli were expanded and had thin septal walls. The bronchi were moderately inflamed; some showed epithelial proliferation.[15] Exposure to 94.0 mg/m^3 (50 ppm) NO_2 resulted in extreme vesicular dilatation or total collapse of alveoli, along with extensive edema and lymphocyte infiltration. The bronchi showed epithelial surface erosion and absence of cilia.

(b) Orientation changes–continuous exposure–After 3 days of continuous exposure to 3.8 mg/m^3 (2 ppm) NO_2, the terminal bronchiolar epithelium of rats changed from an active, inhomogeneous, lining layer, to a

uniform layer of enlarged cells. This subtle biochemical effect on epithelial cell metabolism was further documented by the demonstration of abnormal ciliogenesis. Although ciliary basal bodies developed normally, they failed to orient appropriately at the apical surfaces of the cells. They either formed no cilia or directed them intracytoplasmically into vacuoles. Intracytoplasmic, crystalloid inclusion bodies also developed in time.[26-28]

(c) Hyperplasia—In another case, continuous exposure to 32.0 mg/m^3 (17 ppm) NO_2 precipitated a wave of accelerated cellular replication (hyperplasia) of terminal-bronchiolar and type-II epithelial cells and macrophages in rat lungs. This response was measured both by nuclear autoradiography with tritiated thymidine incorporation and by mitotic-figure counts.[29] The activity peaked at 24 hours and returned to baseline values within a week, despite the continued exposure and without apparent vital injury to the epithelial cells involved.

In hamsters continuously exposed to higher NO_2 concentrations [84.6 to 103.4 mg/m^3 (45 to 55 ppm)] for 2.5 months, researchers noted transitory hyperplasia (increased cell production) of the respiratory bronchiolar epithelium. The lung volumes increased during exposure and reverted toward normal after a 2-week recovery.[30] In hamsters exposed to 188 mg/m^3 (100 ppm) NO_2 for 6 hours, tritiated thymidine uptake indicated an intense epithelial proliferation in the major bronchi, that was maximal 24 hours after exposure and normal again within 4 days. In the periphery, the response was delayed and less intense.[31]

(d) Emphysematous lesions—With exposures of 3 months or longer, 0.9 to 47.0 mg/m^3 (0.5 to 25 ppm) NO_2 caused changes in animal lung tissue similar to those seen in human emphysema. Pre-emphysematous lesions indicative of the development of early focal emphysema were observed in the lungs of mice exposed to 940 μg/m^3 (0.5 ppm) NO_2 for 6, 18, and 24 hours a day for periods from 3 to 12 months.[32] The data are given in Table 9-4. Alveoli, consistently increased in size by cell-distension rather than by septal breakage. The investigators also noted inflammation of the bronchiolar epithelium and reduction in distal airway size.

Continuous exposure of young rats to 18.8 to 47.0 mg/m^3 (10 to 25 ppm) NO_2 produced voluminous, heavy, dry, air-retaining lungs in expanded, kyphotic thoraces—a condition grossly resembling emphysema (see Table 9-5).[33-36] Such animals died of respiratory complications after several months of exposure. Pathological findings included narrow, occasionally fibrotic and occluded terminal bronchioles with no cilia; distended, ruptured peripheral alveoli; and alterations in the staining characteristics of both collagen and elastic tissue, particularly in tissue from the alveolar ducts.[37] Terminal bronchiolar, ductal, and adjacent alveolar epithelium was hypertrophic (enlarged).

When such lungs were allowed to recover, the hypertrophic epithelium receded and cilia reappeared. With the recession, the lung weights returned to normal. Additional pulmonary connective tissue usually appears in rats as a part of aging; however, the previously exposed, healing rats developed excessive lung weight, compared to controls of the same age. This suggested an "anamnestic" connective-tissue response in old age.[38] Abnormally huge collagen fibrils and overtly thick basement laminae were revealed by electron microscopy of the lungs from such rats, including those exposed to as little as 3.8 mg/m^3 (2 ppm) NO_2.[39]

Early, emphysema-like lesions could also be produced in larger animals by exposure to NO_2. Twelve pure-bred beagle dogs, divided into two equal groups, were exposed continuously for 6 months to either filtered air, or filtered air and approximately 47.0 mg/m^3 (25 ppm) NO_2. Autopsies on the NO_2-exposed dogs revealed bullous emphysema in the lungs of one animal and increased firmness with scattered, small bullae in each of the remaining five animals. A diffuse increase in collagen was noted in the lungs of all

Table 9-4. PATHOLOGICAL CHANGES IN LUNGS OF MICE EXPOSED DAILY TO 940 $\mu g/m^3$ (0.5 ppm) NO_2 ON VARIOUS SCHEDULES[32]

Length of total exposure, mo	Daily exposure, hr		
	6	18	24
3	Pneumonitis characterized by peribronchial and interstitial alveolar lymphocytic infiltration. Bronchiolar epithelium cuboid and appeared normal. Some alveolar expansion, but no bronchiolar obstruction. Alveolar septal breaks apparent.	Bronchiolar epithelium cuboid and appeared normal. Some alveolar expansion, but no bronchiolar obstruction. Alveolar septal breaks apparent.	Obstruction of some respiratory bronchioles with acidophilic substance. In these areas bronchiolar epithelium was columnar and devoid of cilia. Alveoli distal to the obstructed areas were expanded with concomitant thinning walls. Alveolar septal breaks apparent.
6	Pneumonitis like 3-month exposure. Bronchial mucosa markedly inflamed with surface erosion and loss of cilia. Bronchiole-alveolar junction, partially to totally blocked, with cellular debris in some areas. Bronchiolar epithelium cuboid. Alveolar expansion and septal breakage like 3-month exposure.	Pneumonitis. Bronchial mucosal and bronchiole-alveolar junction reactions similar to 6-hour exposure. Bronchiolar epithelium cuboid. Alveolar expansion and septal breakage like 3-month exposure.	Pneumonitis similar to that found in 6- and 18-hour exposure, but more severe. In some areas bronchiolar epithelium columnar in shape and coated with mucoid-like material. Alveolar expansion and septal breaks apparent.
9	Pneumonitis, less severe that shorter-term exposures. Alveolar septal walls thicker. Alveolar expansion not as severe as 3- and 6-month exposures.	Pneumonitis, less severe than in shorter-term exposures. Alveolar septal walls thicker. Alveolar expansion not as severe as 3- and 6-month exposure.	Pneumonitis, less severe than in shorter-term exposures. Alveolar expansion not as severe as in 3- and 6-month exposures.
12	Moderate pneumonitis. Evidence of bronchiolar epithelial erosion.	Severe pneumonitis characterized by widespread interstitial and peribronchial lymphocytic infiltration. Evidence of bronchiolar epithelial erosion.	Severe pneumonitis as in 18-hour exposure. Some lung lobes in state of gray hepatization of pneumonia with exudate in alveoli and infiltration of polymorphonuclear leucocytes. Evidence of bronchiolar epithelial erosion.

Table 9-5. PATHOLOGICAL CHANGES IN LUNGS OF RATS CONTINUOUSLY EXPOSED TO NO_2[33,34]

Concentration, ppm	Concentration, mg/m^3	Length of exposure	Lung-weight changes	Histology
25	47.0	37-41 days	No difference from control	At 41 days: moderate hypertrophy and hyperplasia of bronchiolar epithelium; columnar type cells, tall; increased numbers of active goblet cells in the major bronchi.
		146-157 days	Over double weight of control	At 157 days: advanced hyperplasia and hypertrophy of epithelial cells. Proliferation of connective tissue stroma, some free macrophages and desquamated cells in alveolar spaces. Considerable distension of alveolar ducts and alveoli, septal breaks-change considered emphysematous.
12.5	23.8	About 30 weeks	Increased up to double the weight of controls	Similar to effects at 47 mg/m^3 (25 ppm). In rats surviving long periods of exposure, one had focal nodular metaplasia in bronchial epithelium; two had focal adenomatosis in lung periphery.
4.0	7.5	16 weeks (Experiment stopped)	No difference from controls	Mild hyperplasia of bronchial epithelium in all lungs. Strands of mucous material in bronchial lumen in 2/9 animals. Alveolar ducts and some alveoli contained bits of proteinaceous material and scattered macrophages. Otherwise no difference from controls.
2	3.8	Natural lifetime	Increased to about 110% of control	Grossly, like controls. Microscopically, bronchiolar epithelial cells were more uniform with few remaining cilia and increased size (area one-third greater than controls). Cells suggested dormancy, lacking most exfoliative activity and cytoplasmic blebbing. Rod-like cytoplasmic inclusion bodies seen.
0.8	1.5	Natural lifetime	No difference from control	Microscopically, essentially unchanged except for occasional evidence of bronchiolar epithelial changes similar to those obtained with 3.8 mg/m^3 (2 ppm).

six animals, but no such changes were observed in the controls.[40] Further studies suggested that adding 1 $\mu g/m^3$ ferric oxide dust to air containing 48.9 mg/m^3 (26 ppm) NO_2 may protect dogs against the development of pathological changes. This was the case in a subsequent 6-month experiment.[41]

(5) Combined Effects of NO_2 With Tobacco Smoke. Exposure either for 2 hours to 28.2 mg/m^3 (15 ppm) NO_2 or for 1 hour to 3 percent (v/v) tobacco smoke had only a slight effect on the surface structure of hamster lungs. When hamsters were exposed to the same concentrations for the same times, but sequentially (NO_2 followed by tobacco smoke), the combination of exposures produced marked alterations in surface morphology. The structure of mucus-secreting cells was not typical, and a marked loss of cilia was noted 2 days after exposure. These changes were not reversible. Seven days after exposure, the surface structure of the main bronchi and secondary airways appeared to be even more disrupted; small patches of cilia and deep holes appeared in the bronchial mucosa. The authors concluded that NO_2 and tobacco smoke act synergistically on the bronchial epithelium, producing pathological changes much greater than those caused by either pollutant alone.[42]

c. Systemic Effects

(1) Tissue Changes. Kidney, liver, and heart tissues of squirrel monkeys given 2-hour exposures to NO_2 were damaged in proportion to the dosage. At 28.2 mg/m^3 (15 ppm) some tubular erosion appeared in the kidneys; liver cells ballooned and developed clear cytoplasm with displaced nuclei and congested interstitial spaces. At 65.8 mg/m^3 (35 ppm) renal glomerular tufts were swollen and heart tissue showed areas of interstitial fibrosis. At 94 mg/m^3 (50 ppm) the hearts developed interstitial edema and lymphocytes infiltrated kidney and liver tissue. Centrolobular necrosis also appeared in the liver.[15]

(2) Weight Loss. Studies on the effects of *long-term* exposure to NO_2 have produced conflicting results with respect to weight loss. No significant reduction in the rate of weight gain was noticed in rabbits, guinea pigs, rats, or hamsters exposed to 1.9, 9.4, or 47 mg/m^3 (1, 5, and 25 ppm) NO_2 and dogs to 1.9 and 9.4 mg/m^3 (1 and 5 ppm) NO_2, for 6 hours a day for 18 months.[17] Furthermore, no significant differences in weight gains appeared in mice exposed to 940 $\mu g/m^3$ (0.5 ppm) NO_2 for 6, 18, or 24 hours a day, 5 days a week, for up to 12 months.[43] Combined NO_x exposures of 940 to 1,880 $\mu g/m^3$ (0.5 to 1.0 ppm) NO_2 plus 245 $\mu g/m^3$ (0.2 ppm) NO or 2.8 to 3.8 mg/m^3 (1.5 to 2.0 ppm) NO_2 plus 245 $\mu g/m^3$ (0.2 ppm) NO 16 hours a day for 18 months did not change weight gain in the 12 beagles in either experiment.[18]

In contrast, rats exposed *continuously* to 22.6 mg/m^3 (12 ppm) NO_2 for 9 months continued to grow, but their body weights remained 20 percent below those of control animals.[31,34] One observer[44] reported a 10 percent weight loss in rabbits exposed to 5.6 $\mu g/m^3$ (3 ppm) NO_2 for 15 weeks, while controls added 11 percent to their weight. The effect was apparently dose dependent, for rabbits exposed to 2.5 mg/m^3 (1.3 ppm) NO_2 for 17 weeks increased their weight 2 percent compared to an 8 percent increase by controls.

(3) Voluntary Behavior. Six-hour exposures of 6.9 to 39.3 mg/m^3 (3.7 to 20.9 ppm) NO_2 depressed the voluntary running activity of male mice, when the concentration was 14.5 mg/m^3 (7.7 ppm) or greater. Although the threshold was not identified, it could be narrowed to between 6.9 and 14.5 mg/m^3. In all cases, activity returned to normal on the first post-exposure day.[9]

(4) Hematologic Effects. Studies of the effect of NO_2 on the circulating blood have included both short- and long-term exposures. In one *short-term* study, a group[45] exposed dogs to 73.3 and 99.6 mg/m^3 (39 and 53 ppm) for 60 minutes, 97.8 and 160 mg/m^3

(52 and 85 ppm) for 15 minutes, and 235 and 308 mg/m^3 (125 and 164 ppm) for 5 minutes, and found no changes in the hematocrits and blood platelet counts determined 4, 24, 48, and 72 hours after exposure. The same was true in dogs exposed to 1.9 and 47.0 mg/m^3 (1 and 25 ppm) NO_2 6 hours a day for 18 months.[17]

Freeman, et al.[37] found polycythemia in rats allowed to breathe 3.8 mg/m^3 (2 ppm) NO_2 or more, continuously. The concentration of erythrocytes rose within 2 to 3 weeks and achieved levels of about 40 to 100 percent above baseline values. After some delay, both hematocrit and hemoglobin levels rose, but to a lesser degree, so that the fully developed polycythemia was characterized by a reduction in mean corpuscular volume, mean corpuscular hemoglobin, and mean corpuscular hemoglobin concentration. Cellular diameters measured on the flat surface of stained smears were normal. The blood of monkeys (*M. speciosa*) exposed to 3.8 and 16.9 mg/m^3 (2 and 9 ppm) NO_2 followed a similar course.[37]

Leukocytosis occurred in the peripheral blood of rabbits exposed to both 2.5 and 5.6 mg/m^3 (1.3 and 3.0 ppm) NO_2 for 15 to 17 weeks but it receded with cessation of exposure. The leukocytic response was accelerated by the presence of SO_2. Phagocytic activity was depressed by NO_2 and both leukocytic and phagocytic alterations were greater at the higher NO_2 exposure.[44]

Methemoglobin (MeHb) has been detected in the blood of animals exposed to NO_2 as well as to NO. Cats, rabbits, guinea pigs, mice, and rats were exposed to 56.4 to 1,880 mg/m^3 (30 to 1,000 ppm) NO_2 for unspecified periods of time. MeHb was not detected in animals receiving up to 103 mg/m^3 (55 ppm) NO_2. Cats exposed to 188 mg/m^3 (100 ppm) showed detectable amounts of MeHb after approximately 1 hour of exposure, however. At concentrations of 282 mg/m^3 (150 ppm) NO_2 and above, the blood of all animals tested contained MeHb after 1 hour of exposure, but the MeHb was no longer measurable 1 to 2 hours after animals were returned to clean air. In these experiments the NO_2 values reflected the total oxides of nitrogen present in the exposure chamber; the concentration of NO was not evaluated separately.[46]

In other experiments, MeHb appeared in the blood of rabbits and rats exposed to fumes produced in the electric-arc welding process.[47] Rats exposed for 6 hours a day, 5 days a week, for 43 days, to fumes containing 45.1 mg/m^3 (24 ppm) NO_2 developed 4.5 to 21.7 percent MeHb (mean of 13.6 percent). Detectable levels were still present on the eleventh day of the post-exposure period. Rabbits similarly exposed for 45 days, formed an average of 2.8 percent MeHb (range 0.9 to 4.5 percent). On the sixth day of the post-exposure period, detectable amounts remained in male rabbits only. Male rats exposed to 132 mg/m^3 (70 ppm) NO_2 for 6 hours a day formed 2.6 percent MeHb after the first day of exposure and 3 percent by the third day. Three days after exposure, the levels were within the control range. In these experiments, it was estimated that between 12 and 17 percent of the nitrous fumes existed as NO.[47]

A mixture of 180 ppm NO_2 plus N_2O_4 caused death in 12 dogs within 55 to 285 minutes. MeHb concentrations increased significantly ($p < 0.01$), with an average of 325 mg per 100 g of blood before, and 700 mg per 100 g after, exposure.[48]

(5) Immunologic Effects. A circulating substance with properties similar to a lung antibody appeared in the serum of guinea pigs exposed to 9.4 mg/m^3 (5 ppm) NO_2 either 4 hours a day 5 days a week, or 7.5 hours a day 5 days a week, for up to 5.5 months. In a second group, exposed to 28.2 mg/m^3 (15 ppm) NO_2 continuously for 1 year, the antibody reacted *in vitro* with proteins extracted from the lung tissue of control animals. The titers of reactive substances increased with the intensity and duration of exposure, but no absolute values were

ascribed to the data because the latex agglutination method employed is not quantitative.[16]

(6) Effects on Enzyme Systems. The effect of exposure to 28.2 mg/m^3 (15 ppm) NO_2 continuously for 10 weeks; on oxygen consumption, lactic dehydrogenase, and aldolase; in guinea pig lung, liver, spleen, and serum was determined.[49] The following significant changes occurred: oxygen consumption increased in spleen and kidney; lactic dehydrogenase activity increased in lung, liver, and kidney; and aldolase activity increased in tissues. Short-term exposures also caused a significant increase in the activities of the same enzymes in some tissues. Exposure of guinea pigs to 75.2 mg/m^3 (40 ppm) for 30 minutes at 2-hour intervals for a total of 4.5 hours revealed the following significant changes: oxygen consumption increased in liver, spleen, and kidney; lactic dehydrogenase activity increased in liver, kidney, and serum; and aldolase activity increased in liver, spleen, kidney, and serum. The mechanism that causes these enzymatic changes is not known, but it might reflect a general response to stress.

Following a 2-hour exposure to 65.8 and 94.0 mg/m^3 (35 and 50 ppm) NO_2, lactic dehydrogenase isoenzymes from lung tissue of squirrel monkeys shifted to predominantly anaerobic band 5 when chromatographed, and after a 2-hour exposure to 18.8 mg/m^3 (10 ppm) NO_2, a similar increase in band 5 was noted in serum lactic dehydrogenase isoenzymes. This change in isoenzyme pattern was present at 2 days, but returned to normal 8 days after termination of the NO_2 exposure. A similar shift to anaerobic isoenzyme was observed in the heart tissue of hamsters exposed for 2 hours to 9.4 and 65.8 mg/m^3 (5 and 35 ppm) NO_2.[15,50]

d. Susceptibility to Respiratory Infection

(1) Bacteria and Influenza. Study of rabbits exposed to concentrations of NO_2 ranging from ambient Cincinnati levels to 113 mg/m^3 (60 ppm) revealed increased numbers of polymorphonuclear leukocytes (heterophiles) in lung washings; this condition persisted for more than 72 hours after a single 3-hour exposure. When streptococci were instilled into lungs of NO_2-exposed, anesthetized rabbits 30 minutes before lavage, a pronounced inhibition of phagocytic activity was observed, in comparison to controls.[51]

Exposure of mice, hamsters, and squirrel monkeys to NO_2 increases susceptibility to bacterial pneumonia and influenza infection. This susceptibility, observed in both *short-* and *long-term* exposures, is based on three parameters: (1) increased mortality rates, (2) reduced survival times, and (3) reduced ability to clear inhaled infectious agents from the lungs, as determined by the number of viable organisms that can be cultured. Mice in these experiments were exposed for 2 hours to NO_2 in concentrations ranging from 2.8 to 47.0 mg/m^3 (1.5 to 25 ppm), then challenged with an aerosol of *Klebsiella pneumonia* within 1, 6, and 27 hours after the NO_2 exposure.[52,53] The minimum NO_2 concentrations required to produce a statistically significant rise in subsequent mortality was 6.6 mg/m^3 (3.5 ppm) for 2 hours when the infectious challenge took place 1 hour after the NO_2 exposure. When the infectious challenge was delayed, a statistically significant effect was noted at 6, but not at 27 hours, at NO_2 concentrations of 9.4 mg/m^3 (5 ppm) and above. Exposure to 47.0 mg/m^3 (25 ppm) NO_2, 6 and 14 days prior to the challenge with *K. pneumoniae* did not increase mortality. The same authors reported that when mice were infected with *K. pneumoniae* first, and then exposed for 2 hours to 47.0 mg/m^3 (25 ppm) NO_2, within 1, 6, 27, 48, and 72 hours after the infectious challenge, the mortality increase was statistically significant. This effect was not observed at 4.7 mg/m^3 (2.5 ppm) NO_2.

A 2-hour exposure to 9.4 mg/m^3 (5 ppm) NO_2 in various inbred strains of mice (BDF_1, BALB/c, $C_{57}BL$, and LAF_1) either before or after infectious challenge with an aerosol of *K. pneumoniae* favored increased mortalities. A similar enhancement in mortality was

observed in hamsters exposed for 2 hours to 65.8 mg/m^3 (35 ppm) NO_2 and above, and challenged with *K. pneumoniae.*[50]

The rate of clearance of inhaled bacteria from the lungs of mice and hamsters was reduced upon *short-term* exposure to NO_2.[50] The animals were exposed to 9.4 mg/m^3 (5 ppm) NO_2 for 2 hours, and within 1 hour challenged with *K. pneumoniae* aerosols. Immediately, and at 1, 3, 5, 6, 7, and 8 hours after the infectious challenge, the animals were sacrificed and *K. pneumoniae* in lungs was quantitatively assayed. In control animals the bacterial population was markedly reduced during the 6 hours following the challenge. Thereafter, the population increased, reaching its initial concentration after approximately 8 hours. In mice and hamsters exposed to 9.4 mg/m^3 (5 ppm) NO_2, the period of initial clearance was reduced to 4.5 and 5 hours, respectively, and the original concentration was re-established in less than 7 hours.[48]

In a long-term study, Ehrlich and Henry[43,54] exposed four groups of mice to 940 μg/m^3 (0.5 ppm) NO_2 for 6, 18, and 24 hours a day, 7 days a week, for up to 12 months. Challenge with an aerosol of *K. pneumoniae* took place after 1, 3, 6, 9, and 12 months of exposure. Statistically significant increases in mortality were observed after continuous exposure to 940 μg/m^3 (0.5 ppm) for 3 months, and after 6- and 18-hour-a-day exposures for 6 months. After 12 months exposure, mortality could only be increased significantly when the NO_2 exposure was continuous. The clearance rate of viable bacteria from the lungs of mice was also affected by long-term exposure to 940 μg/m^3 (0.5 ppm) NO_2. In mice exposed to NO_2 for 6 and 18 hours a day for 9 months some reduced capacity to clear bacteria from lungs was observed, and after 12 months exposure a significant inhibition of bacterial clearance was apparent. Mice exposed to NO_2 for 24 hours a day showed significantly reduced capacity to clear viable bacteria after 6, 9, and 12 months of exposure.

Henry *et al.*[15] exposed squirrel monkeys to 18.8 to 94.0 mg/m^3 (10 to 50 ppm) NO_2 for 2 hours and challenged them by the intratracheal route with *K. pneumoniae.* Exposure to 94.0 mg/m^3 (50 ppm) NO_2 for 2 hours was not fatal, whereas the same exposure followed by challenge with *K. pneumoniae* was fatal to three out of three monkeys. Squirrel monkeys exposed to 18.8 mg/m^3 (10 ppm) NO_2 for 2 hours and then challenged with *K. pneumoniae* had bacteria present in their lungs 19 to 51 days after challenge. In many bronchioles, epithelial cilia were missing; cells had proliferated; and lymphocytes and polymorphonuclear cells had infiltrated the collapsed areas.

The same group[55] exposed male squirrel monkeys continuously to NO_2 and challenged them with *K. pneumoniae* aerosols. Out of four monkeys exposed to 18.8 mg/m^3 (10 ppm) NO_2 for 1 month, one died and two had the infectious agent present in the lungs at autopsy. Two out of seven monkeys exposed to 9.4 mg/m^3 (5 ppm) NO_2 for 2 months died, and five had the infectious agent present in the lungs at autopsy.

Squirrel monkeys were also experimentally infected with influenza A/PR-8 virus, 24 hours before continuous exposure to 18.8 and 9.4 mg/m^3 (10 and 5 ppm) NO_2. All six monkeys exposed to 18.8 mg/m^3 (10 ppm) died within 3 days, while at 9.4 mg/m^3 (5 ppm) one out of three succumbed to the disease. There were no deaths in the control group challenged with influenza virus.[53]

When the squirrel monkeys were exposed to 9.4 mg/m^3 (5 ppm) NO_2 for 5 months and, during the exposure, challenged three times by the intractracheal route with the influenza A/PR-8 virus, the formation of protective serum neutralization antibody appeared to be depressed as evidenced by serum neutralization antibody titers and hemagglutination-inhibition titers.[56]

(2) Bacteria and Tobacco Smoke. Combined exposures to NO_2, tobacco smoke, and *K. pneumoniae* were reported for hamsters.[42,57] A 2-hour exposure to 28.2 mg/m^3 (15 ppm)

NO_2 followed by 1-hour exposure to 3 percent (v/v) tobacco smoke significantly decreased the resistance to bacterial pneumonia, as evidenced by enhanced mortalities. Furthermore the combined exposures reduced the rate of clearance of viable bacteria from the lungs of hamsters to a greater extent than exposures to the individual pollutants. The combined exposure produced irreversible, marked alterations in surface morphology of lung tissue. The typical structure of mucus-secreting cells was not apparent, and a marked loss of cilia was noted 2 days after exposure.

(3) Interferon Formation. Impairment of interferon formation by NO_2 exposure has been demonstrated by challenging rabbit alveolar monocytes with parainfluenza-3 virus.[58] Rabbits exposed to 49.0 mg/m^3 (25 ppm) NO_2 for 3 hours, either immediately after, or at 0, 3, 6, 12, or 24 hours before innoculation with rabbit pox virus, failed to develop the anticipated resistance to infection. This inhibition of resistance persisted for at least 96 hours after NO_2 exposures. During this period, alveolar monocytes from NO_2–exposed animals were unable to produce the antiviral substance, interferon, *in vitro* when innoculated with the parainfluenza-3 virus.

2. Effects in Man

a. Experimental Exposures

(1) Odor Perception. In a series of experiments with healthy male volunteers between the ages of 20 and 35 years, individuals were exposed to varying concentrations of NO_2 in a specially designed chamber, and the olfactory threshold was measured under various conditions (Table 9-6). At a concentration of 225 $\mu g/m^3$ (0.12 ppm) NO_2, only a few subjects perceived the odor immediately. Perception was immediate in over

Table 9-6. RECOGNITION OF NO_2 ODOR BY HEALTHY YOUNG ADULT MEN AFTER VARIED EXPOSURES IN AN EXPERIMENTAL CHAMBER[59]

Concentration,[a]		Subjects	Odor perception on entry to chamber	
mg/m^3	ppm		Number of subjects	Duration,[a] min
0.225	0.12 (0.1-0.15)	9	3	5 (0.5-10)
0.415	0.22 (0.16-0.28)	13	8	3 (1-10)
0.835	0.42 (0.39-0.46)	8	8	2.5 (1-5)
8.0	4.0 (3.0-4.2)	12	12	5.5 (3-10)
19.9	10.6 (10.1-11.2)	4	4	8 (6-13)
39.9	19.7 (19.3-20.1)	8	8	12 (5-24)
56.8	30.2 (29.1-31.4)	3	3	32 (25-40)

[a]The range of actual measured values is included in parentheses.

half of the subjects exposed to 415 $\mu g/m^3$ (0.22 ppm) NO_2. At higher concentrations, beginning with 835 $\mu g/m^3$ (0.42 ppm) NO_2, all subjects recognized the odor immediately. Persistent recognition of the odor varied considerably, ranging from a few minutes at the lower exposure levels to over 30 minutes at some of the higher concentrations.[59]

In another study[60] of 14 subjects, the NO_2 olfactory threshold was 230 $\mu g/m^3$ (0.12 ppm). The effect of NO_2 and SO_2 was additive, i.e., a lower concentration of each gas led to odor perception if both gases were present simultaneously.

Recently diesel exhaust odor has been the subject of considerable study and it has been found that NO_2 may contribute to the odor of diesel exhaust. An assessment of the major industrial odorants in diesel exhaust revealed that the mixture with the greatest odor contained about 940 $\mu g/m^3$ (0.5 ppm) NO_2.[61]

(2) Pulmonary Function. In one experiment, exposure to NO_2 was controlled in a special room equipped with an air filter.[62] Six normal subjects and four patients with "moderate to marked" pulmonary disease were exposed to concentrations of 940 to 5,640 $\mu g/m^3$ (0.5 to 3 ppm) NO_2 on several occasions for 2 to 3 hours at a time. Several physiological parameters were measured before, during, and after exposure, when the subjects were either at rest or exercising. Smokers were not excluded from the study, but were required to abstain from smoking for the 8-hour period prior to the chamber study. The data showed no consistent changes in airway resistance, pulse rate, respiratory rate, or subjective complaints that could be related to the NO_2 challenge.

NO_2 and SO_2 produced additive effects on pulmonary function in five healthy males, ages 21 to 40, who were judged to be free from respiratory disease.[63] One subject smoked 5 to 6 cigarettes a day. Each subject was exposed on separate occasions to 7.5 to 9.4 mg/m^3 (4 to 5 ppm) NO_2 and 10.5 to 13.1 mg/m^3 (4 to 5 ppm) SO_2. Each exposure was for 10 minutes, with 2-week intervals between exposures. The subjects wore nose clips and inhaled the gas-air mixtures through a mouthpiece. Inspiratory and expiratory flow resistance and pulmonary compliance were measured before, immediately after, and 10, 20, and 30 minutes after exposure. Vital capacity, FEV_1 (1 - second forced expiratory volume), maximal mid-expiratory flow rate, and peak flow rate were measured prior to and 30 minutes after exposure. Inhalation of NO_2 caused an increase in both inspiratory and expiratory flow resistance, the maximum recorded increase occurring 30 minutes after the end of exposure. No data were given for the recovery time. Mean pulmonary compliance was unchanged immediately after exposure, but was slightly decreased 30 minutes after exposure (significant at $p < 0.10$ but not at < 0.05). Lung volumes and peak flow rates did not change significantly. Inhalation of SO_2 caused an increase in both inspiratory and expiratory flow resistance that was maximal immediately after inhalation and restored to pre-exposure levels 30 minutes later.

Exposure to a mixture of 4.7 mg/m^3 (2.5 ppm) NO_2 and 6.6 mg/m^3 (2.5 ppm) SO_2 produced a bimodal increase in both inspiratory and expiratory flow resistance. The first increase, which corresponded to the effect of SO_2 alone, occurred immediately after exposure. The second increase in resistance corresponded to the effect of NO_2 alone, and was maximal 30 minutes after exposure. SO_2 is known to cause an immediate reflex increase in airway resistance along parasympathetic pathways.[64] The mechanism by which NO_2 induces resistance is unknown, but the delay suggests that it is different from that of SO_2.[63]

b. Occupational exposures

There are numerous possibilities for occupational exposure to NO_x. Hazardous occupations include the manufacture of NO, nitration of cellulose, and other organic

materials; electric-arc welding; and photo-engraving. Accidental exposures have resulted from the combustion of celluloid and nitro-cellulose films, and from inhalation of silage gas.

Four clinical types of poisonings due to occupational exposures[65] have been encountered: (1) irritant gas, (2) reversible, (3) shock, and (4) combinations of the other three. Irritant gas exposure is characterized initially by severe irritation that results in pain, burning, and choking in the throat and chest; violent cough; and the expectoration of yellow-tinged sputum. The reversible type is characterized by dyspnea, cyanosis, vomiting, vertigo, somnolence, a feeling of intoxication, fainting, loss of consciousness, and methemoglobinemia. Persons suffering this type of poisoning do not develop pulmonary edema, and if removed from the exposure early enough, may recover completely. Otherwise, the poisoning may rapidly become fatal. Shock-type patients immediately show severe symptoms of asphyxiation, convulsions, and respiratory arrest; death is presumably due to stasis in the pulmonary circulation. This form is exceptional and may result from the sudden inhalation of high concentrations. In the combined type the patient immediately shows symptoms referable to the central nervous system, such as vertigo, somnolence, and a staggering gait. There may be cyanosis. After apparent recovery, this stage may be followed some hours later by progressive dyspnea, marked cyanosis, and pulmonary edema.

Several accidental exposures to silage gas,[66,67] which may contain very high concentrations of NO_2, have been reported. In several instances the exposure proved fatal. One man died 20 hours after being exposed to this type of gas for 5 to 8 minutes.[64] One person exposed at the same time for 2 to 3 minutes survived, although he suffered acute pneumonitis. Exposure to NO_2 has been explained in six degrees, with corresponding clinical syndromes: (1) at 940mg/m^3 (500 ppm) or higher victims develop acute pulmonary edema and die within 48 hours; (2) at 564 to 752 mg/m^3 (300 to 400 ppm) pulmonary edema with broncho-pneumonia develops and death ensues in 2 to 10 days; (3) those exposed to 282 to 376 mg/m^3 (150 to 200 ppm) develop bronchiolitis fibrosa obliterans, which is fatal in 3 to 5 weeks; (4) exposure to 94 to 188 mg/m^3 (50 to 100 ppm) produces bronchiolitis with focal pneumonitis lasting 6 to 8 weeks, followed by spontaneous recovery; (5) individuals exposed to NO_2 in the range of 47 to 141 mg/m^3 (25 to 75 ppm) develop varying degrees of bronchitis and broncho-pneumonia, but recover completely; and (6) chronic intermittent exposure to concentrations of NO_2 in the order of 18.8 to 75.2 mg/m^3 (10 to 40 ppm) may produce chronic pulmonary fibrosis and emphysema.

Occupational exposure to NO_2 is frequently encountered in a variety of welding processes. Evaluation of responses to inhalation of such fumes is complicated by the presence of other contaminants. Predominant among these are NO, ozone, manganese, ferric oxide fumes, and particulates of various types. In a case of acute poisoning from NO_x from an oxyacetylene torch, a welder died from chemical pneumonitis 10 days later.[68] Other workers present during the exposure developed coughs and other respiratory symptoms. Random air samples obtained under simulated conditions revealed oxides of nitrogen in the range of 71.4 to 662 mg/m^3 (38 to 352 ppm), occurring as NO_2. After 3 minutes exposure to fumes containing 395 to 714 mg/m^3 (210 to 380 ppm) NO_2, welders developed a dry cough and tightness in their chests. These symptoms disappeared when they returned to clean air.

Pulmonary edema developed 18 hours after a 30-minute accidental exposure to the fumes of an oxyacetylene torch estimated to have produced approximately 169 mg/m^3 (90 ppm) NO_2. In pulmonary function tests conducted at that time the vital capacity was 50 percent of the expected level, but the 0.5-, 1.0-, and 2.0-second FEV's were not diminished.[69]

The effects of electric-arc welding exposures from both bare and coated rods have also been examined. In an experiment with bare rods, two men were exposed to the fumes of the electric-arc welding process for 190 minutes.[47,70] The average concentration of nitrous fumes, expressed as NO_2, was 158 mg/m^3 (84 ppm), with a maximum of 175 mg/m^3 (93 ppm) that lasted for 40 minutes and a peak of 194 mg/m^3 (103 ppm) lasting 5 minutes. These concentrations measured by the total nitrate method actually include an unspecified mixture of $NO\text{-}NO_2\text{-}N_2O_4$. Subjects were also exposed to approximately 785 $\mu g/m^3$ (0.4 ppm) ozone throughout the period. They reported no headache, eye irritation, throat irritation, or other ill effects, however.

In welders exposed to the fumes from electric-arc welding using coated rods[47] for the same length of time, the average MeHb concentration was 2.5 percent and the maximum was 3 percent. The concentration of "nitrous gas" in the working area did not exceed 25 mg/m^3 (13.3 ppm), expressed as NO_2, of which not more than 20 percent was estimated to be NO.

The possible long-term effects of occupational exposure to nitrous fumes was reported by Becklake et al.[71] in a study of seven miners who were accidentally exposed to nitrous fumes for periods of 5 to 75 minutes and who developed pulmonary edema 3 to 27 hours after exposure (see Table 9-7). The investigators studied pulmonary function at the time of each patient's discharge from the hospital and compared yearly follow-up examination data to the mean values for the same data from 16 normal persons.

Among the seven patients, the most common effects were a reduction in maximal breathing capacity and an increase in expiratory resistance; the latter functional impairment was common to all the patients who complained of exertional dyspnea. In the two patients who felt that their capacity for work was unaffected by the accident, tests showed that pulmonary function had returned to normal during the study period. Chest roentgenograms of those patients with more than 6 years of underground mining service, taken within a year prior to the exposure, had been normal. Following the accident, the arterial oxygen saturation was not affected either at rest or during exercise. The mixing index, a measure of efficiency in clearing nitrogen gas from the lungs, was significantly different from predicted values in four patients, although it returned to normal in the course of the study period in the three who could be followed. The investigators concluded that the exposed individuals sustained a degree of bronchial and bronchiolar narrowing due to fibrosis, secondary to various degrees of bronchiolitis obliterans.

A study of the survivors of the Cleveland Clinic fire in 1967, who also were exposed to high concentrations of nitrogen oxides, is discussed in chapter 10, Section C.1.

Multiple clinical symptoms, biochemical, and hematologic changes were described in workers engaged in the manufacture of sulfuric acid and hence exposed to an average of 4.9 mg/m^3 (2.6 ppm) NO_2 for 3 to 5 years.[72] Because the data are not supported by diagnostic criteria, this report cannot be evaluated. In contrast, Italian workers employed in the manufacture of nitric acid and exposed to an average of 56.4 to 65.8 mg/m^3 (30 to 35 ppm) NO_2 for an unspecified number of years exhibited no signs or symptoms of injury.[73]

D. OTHER OXIDES OF NITROGEN

Although several other oxides of nitrogen besides NO and NO_2 exist in the atmosphere, they are generally present in very small quantities and except for nitrous oxide (N_2O) have not been of toxicological interest. Information concerning their effects is likewise scarce.

Considerable attention has been focused on N_2O for the past century because of its anesthetic and analgesic properties. At concentrations of 80 percent (1.44×10^6 mg/m^3), N_2O is an effective general anesthetic. Inhalation of 10 to 20 percent N_2O (1.8×10^5 to 3.6×10^5 mg/m^3) provides effective analgesia.[74] Although extensive information relevant to

Table 9-7. RESPIRATORY EFFECTS OF EXPOSURE TO NITROUS FUMES[71]

(Summary of Clinical Data)

Subject age, years	Time after accident, months	Lung volume: Vital capacity, liters	Lung volume: Vital capacity, % of expected	Lung volume: Residual volume, liters	Lung volume: Residual volume, % of expected	Lung volume: FRC/TLC,[a] %	Lung volume: RV/TLC,[b] %	Maximal breathing capacity,[c] Mixing index	Maximal breathing capacity,[c] liters/min	Maximal breathing capacity,[c] % of expected	Compliance, liters/cm H_2O	Non-elastic resistance, cm/liter-sec: Inspiration	Non-elastic resistance, cm/liter-sec: Expiration	Respiratory rate, liters/sec	Dyspnea[d] after accident
33	8	2.56	53[e]	1.93	116	69[e]	42[e]	5.66[e]	71	44[e]					3
	55	3.48	74[e]	1.53	81	58	30	3.29	68	43[e]					
	64	3.98	85[e]	1.74	92	51	30	3.35	85	54[e]	0.173	1.84	4.04	26	
	65							2.74							
35	1	3.8	72[e]	1.79	87[e]	47	32[e]	7.44[e]	89	53[e]					0
	2	4.33	83[e]	1.77	86[e]	45	29	3.63[e]	141	84[e]					
	6	4.81	92	1.93	94	47	28	2.92[e]	158	95					
	24	4.80	93	1.55	74[e]	40	24	2.32	146	90[e]					
	37	4.80	93	2.41	114	40	33[e]	2.45	184	114	0.174	3.65	4.50	12	
21	1	5.09	100	1.69	106	61[e]	25	2.34	139	81[e]					
	32	5.66	114	1.46	88	59	20	1.90	186	113	0.158	2.09	2.40	20	
20	3	3.81	77[e]	2.28	154[e]	52	36[e]	2.03	75	42[e]					3
	15	4.59	94	1.86	124[e]	49	28	1.95	137	78[e]					
	26	4.48	92	1.41	107	63[e]	24	1.51	149	87[e]	0.249	6.76	5.89	22	
31	1	5.52	102	2.02	100	54	27	3.59[e]	130	75[e]					2
	4	5.51	102	1.80	89	57	24	2.46	152	87[e]					
	25	5.15	96	1.95	95	61	27	2.94	142	83[e]	0.283	2.75	3.27	22	
49	1	5.39	110	2.54	109	44	32	1.77	126	81[e]					3
	7	3.14	64[e]	2.42	104	56	42[e]	2.47	97	61[e]					
	18	4.86	101	3.22	137[e]	52	40[e]	1.88	89	57[e]					
	19										0.124	5.43	6.18	19	
39	9	6.06	106	2.86	121[e]	62[e]	32	1.31	133	74[e]	0.219	3.87	4.90	18	3
Mean value for 16 normal subjects											0.219	1.86	2.04	20	

[a]Functional Residual Capacity/Total Lung Capacity.

[b]Residual Volume/Total Lung Capacity.

[c]In liters/min at ambient pressure saturated with water vapor.

[d]At most recent examination.

[e]Significant difference from predicted value.

these properties of N_2O is available, its inclusion is beyond the scope of this review.

E. FUTURE RESEARCH NEEDS

To cope with the problem of NO_X pollution, future research should include:

1. Further delineation of *in vivo* biochemical and biophysical effects of nitrogen oxide exposures relative to: (a) oxidation of fatty acid double bonds in cellular lipids and in lung surfactants; (b) denaturation or alteration of lung proteins (collagen and elastin, enzymes, and cellular membranes).

2. A definition of the relationship of metabolic tissue changes to NO_2 dosage in terms of a concentration-time response. The relative importance of low-concentration, long-term exposures versus short-term peak concentration should be studied.

3. The determination of interaction of NO_X with particulate pollutants in terms of biochemical, biophysical, infectious, and ultrastructural responses.

4. The assessment of the biologic importance of tolerance and cross tolerance with other oxidant pollutants.

5. Expanded studies to determine the effects of NO_2 on the infectious defense mechanisms and immunological processes.

F. SUMMARY

The two oxides of nitrogen present in ambient air in greatest quantities, nitric oxide (NO) and nitrogen dioxide (NO_2), are potential health hazards. Summaries of laboratory studies of animal exposures are given in Tables 9-8, 9-9, and 9-10. The toxicology of nitrous oxide (N_2O) and other oxides of nitrogen does not appear to be relevant to the problems of ambient air pollution at the present time. N_2O is analgesic at concentrations of about 1.8×10^5 to 3.6×10^5 mg/m^3 (1.0 to 2.0×10^5 ppm) and a general anesthetic at concentrations of 1.4×10^6 mg/m^3 (8.0×10^6 ppm) and above.

Nitric Oxide

At concentrations found in the atmosphere, NO is not an irritant and is not considered to have adverse health effects. Its main toxic potential at ambient concentrations results from its oxidation to NO_2. A 12-minute exposure to 3,075 mg/m^3 (2,500 ppm) NO was lethal to mice. Lower doses, from 24.6 mg/m^3 (20 ppm) to 1.23×10^4 mg/m^3 (10^4 ppm), of NO produced reversible inhibition of bacterial hydrogenase activity.

Nitrogen Dioxide

Animal Studies

NO_2 exerts its primary toxic effect on the lungs. Most of the available information comes from studies with animals. Concentrations greater than 188 mg/m^3 (100 ppm) are lethal to most animal species, and 90 percent of the deaths are caused by pulmonary edema. The mortality rate may be modified by varying the exposure product concentration x time (Ct), the temperature, and the presence of other irritants.

Pulmonary function. Short-term exposures to nonlethal concentrations of NO_2 have produced transient pulmonary-function changes in the lungs of animals. Guinea pigs have shown increased respiratory rates and decreased tidal volumes after exposure to concentrations of 9.8 mg/m^3 (5.2 ppm) for 2 hours, and 24.4 mg/m^3 (13.0 ppm) for 1 hour. Pulmonary function returned to normal when animals were returned to clean air. Similar changes in pulmonary function were observed in squirrel monkeys exposed for 2 hours to 18.8 to 94.0 mg/m^3 (10 to 50 ppm) NO_2 and in other monkeys exposed continuously for 2 months to 9.4 mg/m^3 (5 ppm) NO_2. Monkeys (*M. speciosa*) continuously breathing 3.8 or 16.9 mg/m^3 (2 or 9 ppm) NO_2 developed tachypnea which has persisted for almost 2 years.

Rats continuously exposed to 1.5 mg/m^3 (0.8 ppm) NO_2 maintained a 20 percent increase in respiratory frequency throughout their lifetimes. Dogs exposed to 0.9 to 3.8 mg/m^3 (0.5 to 2.0 ppm) NO_2 plus 245 μg/m^3 (0.2 ppm) NO for 18 months, however, showed no signs of changes in pulmonary function.

Metabolism. The structure of lung collagen and elastin was altered in rabbits exposed to 1.9 mg/m^3 (1 ppm) NO_2 for 1 to 4 hours, but the alteration appeared to be reversible within 24 hours. Similar changes were observed in rabbits exposed to 470 μg/m^3 (0.25 ppm) NO_2, 4 hours a day, for 6 days, but recovery was delayed and some damage persisted after 7 days. It has been suggested that the metabolic activity of the lung tissue is reduced following exposure to NO_2, and that repeated exposure, with associated denaturation of collagen and elastin, may be a factor in the pathogenesis of pulmonary emphysema.

Lung lipids extracted from rats, exposed to 1.9 mg/m^3 (1 ppm) NO_2 for 4 hours were peroxidated. The process was delayed and sustained, reached a maximum at 24 hours after exposure, and was maintained for an additional 24 hours. Rats fed a vitamin E-deficient diet and exposed to NO_2 had more peroxidation of surfactant and tissue lipids than similarly exposed rats receiving vitamin E supplementation.

Pathology. Pathological lesions have been observed in the lungs of animals following both short- and long-term NO_2 exposures. At exposures to 141 to 188 mg/m^3 (75 to 100 ppm) for 2 hours, rabbits, rats, and guinea pigs developed acute inflammation in bronchiolar epithelium, but appeared to recover 2 weeks after exposure. At 1.9 mg/m^3 (1 ppm) for 1 hour, or 940 μg/m^3 (0.5 ppm) for 4 hours, mast cells of rat lungs became degranulated, possibly signifying the onset of an acute inflammatory reaction. These cells returned to normal 24 hours after the end of exposure. More serious damage was found in lungs of squirrel monkeys exposed for 2 hours to 18.8 to 94.0 mg/m^3 (10 to 50 ppm) NO_2; the primary lesions found at low concentrations became progressive alveolar expansion. Hyperplasia of the respiratory bronchiolar epithelium was seen in hamsters exposed to 84.6 to 103 mg/m^3 (45 to 55 ppm) for 10 weeks. A similar response was noted in the major bronchi and distal portions of the respiratory tract in hamsters exposed to 188 mg/m^3 (100 ppm) for 6 hours.

Chronic Changes. Since certain pathological changes seen in animals after experimental NO_2 exposure are similar to changes occurring in the pathogenesis of chronic obstructive pulmonary disease in man, it is suggested that long-term, low-level exposures to this pollutant may play a significant role in the development of chronic lung disease.

Improved histochemical and electron microscopic techniques have shown that long-term exposure of rats to NO_2 at concentrations that do not produce acute inflammatory responses have a cumulative and sustained effect. Emphysema-like lesions were produced in the rat lungs with concentrations of 18.8 to 47.0 mg/m^3 (10 to 25 ppm). Rats exposed to 3.8 mg/m^3 (2 ppm) for their natural lifetime had less cilia; less-than-normal bronchiolar epithelial blebbing; and crystalloid, rod-shaped, intracytoplasmic inclusion bodies in their bronchiolar epithelium. Similar effects have been seen occasionally in rats continuously exposed to 1.5 mg/m^3 (0.8 ppm). Mice exposed to 940 μg/m^3 (0.5 ppm) for 3 to 12 months on 6-, 18-, and 24-hour daily schedules have shown increase in the size of alveoli, due to alveolar distension rather than septal breakage. Additionally, inflammation of the bronchiolar epithelium with a reduction in distal airway size suggested the development of early focal emphysema. Rats exposed chronically to 18.8 to 47.0 mg/m^3 (10 to 25 ppm) NO_2 were observed to develop compensatory changes such as polycythemia, thoracic kyphosis, and a lateral flaring of the ribs. Early evidence of pulmonary emphysema was observed in dogs exposed continuously to 47.0 mg/m^3 (25 ppm) NO_2 for 6 months.

Susceptibility to Infection. Exposure of mice, hamsters, and squirrel monkeys to NO_2 causes increased susceptibility to bacterial pneumonia and influenza infections, demonstrated by increased mortality, decreased survival time, and a reduction in ability to clear infectious agents from the lungs.

Table 9-8. SUMMARY OF STUDIES OF TOXICOLOGICAL EFFECTS OF NITRIC OXIDE IN ANIMALS AND BACTERIA

Species	Concentration, ppm	Concentration, mg/m^3	Length of exposure	Effects	Comment	Reference
Proteus vulgaris	20	24.6	10 min	Reversible inhibition of hydrogenase activity		10
Rats	10	12.3	1 and 9 days	NO-hemoglobin and NO-methemoglobin not found in blood	Methemoglobin not examined	8
Guinea pigs	16-40	19.7	4 hr	No effect on respiratory rates and tidal volumes		3
Proteus vulgaris	10,000	12,300	10 min	Irreversible inhibition of hydrogenase activity		10
Mice	2,500	3,075	6-7 min	Death within 12 min. Recovered if returned to clean air after 4- to 6-min exposure		2

Table 9-9. SUMMARY OF STUDIES OF TOXICOLOGICAL EFFECTS IN ANIMALS OF SHORT-TERM NO_2 EXPOSURE

Species	Concentration, ppm	Concentration, mg/m^3	Exposure, hr	Effects	Comment	Reference
Rats	0.5	0.9	4	Lung: Degranulation in mast cell morphology	Possibly precedes onset of acute inflammatory reaction	25
Rats	1.0	1.9	1	Lung: Degranulation in mast cell morphology	Mostly reversible 24 hr after exposure	25
Rats	1.0	1.9	4	Lung: Lipid peroxidation	Delayed effect. Maximum response 24 hr after exposure	21
Rabbits	1.0	1.9	1	Lung: Structural changes in collagen (by spectroscopy) of animals sacrificed immediately after exposure; reduced in animals sacrificed 24 hr after exposure	Chemical changes suggest denaturation of structural protein, only partially reversed	19
Mice	3.5	6.6	2	Enhanced susceptibility to respiratory infection demonstrated by increased mortality after challenge with *K. pneumoniae*	Threshold for significant effect 6.6 mg/m^3 (3.5 ppm) for 2 hr when challenged 1 hr after exposure	53
Mice	5.0	9.4	2	Increased mortality when infected with *K. pneumoniae* either before or after NO_2 exposure; reduced bacterial clearance from lungs		50
Guinea pigs	5.2-13.0	9.8-24.4	2 - 4	Lung: Increased respiratory rates and decreased tidal volumes		3
Mice	7.7	14.5	6	20% decrease in voluntary running activity		3

Monkeys	10	18.8	2	Lung: Respiratory rate unchanged; tidal volume decreased; septal breaks, alveolar expansion, and some large air vesicles with thin septal walls observed	Tidal volume returned to pre-exposure levels within 48 hr	15
Monkeys	15	28.2	2	Lung: Respiratory rate unchanged; tidal volume decreased; moderate in alveoli expansion, minimal septal wall thinning, patchy lymphocytic interstitial infiltration Kidney: Some tubular erosion Liver: Cell ballooning with clear cytoplasm and nuclear displacement; interstitial congestion	Tidal volume returned to pre-exposure levels within 48 hr	15
Rabbits	25	47.0	3	Decreased interferon production and decreased resistance to viral infection		58
Monkeys	35-50	65.8-94.0	2	Lung: Marked increased in respiratory rate with marked decrease in tidal volume; large areas of total alveolar collapse, edema, and lymphocytic infiltration with other areas of alveolar expansion and septal wall thinning; bronchi inflamed, epithelial surface erosion, and loss of cilia Heart: Interstitial fibrosis and edema Kidney: Glomerular-tuft swelling and intercellular lymphocytic infiltration	Change in pulmonary function persisted up to 72 hr	15

Table 9-9 (continued). SUMMARY OF STUDIES OF TOXICOLOGICAL EFFECTS IN ANIMALS OF SHORT-TERM NO_2 EXPOSURE

Species	Concentration, ppm	Concentration, mg/m^3	Exposure, hr	Effects	Comment	Reference
				Liver: Lymphocytic infiltration, congestion, and centrolobular necrosis		
Guinea pigs	40.0	75.2	0.5 hr at 2-hr intervals, totaling 4.5 hr	Enzyme systems: Increased O_2 consumption–liver, spleen, kidney; increased lactic dehydrogenase–liver, kidney, serum; increased aldolase–liver, spleen, kidney, serum		49
Rats, guinea pigs, and rabbits	75-80	141.0-150.4	2	Lung: Inflammation of bronchiolar epithelium	Apparent recovery 2 wk after exposure	23
	100	188.0				
Hamsters	100	188.0	6	Lung: Epithelial proliferation in major bronchi and more distal respiratory tract; electron miscroscopic evidence of depressed mucopolysaccharide production, increased phospholipid production, and increased lysozymal enzyme content		30
Rats	100	188.0	1	Blood: Methemoglobin present	Disappeared 1 to 2 hr after return to clean air	46
Rabbits, guinea pigs	150	282.0	1	Blood: Methemoglobin present		

Table 9-10. SUMMARY OF STUDIES OF TOXICOLOGICAL EFFECTS IN ANIMALS OF LONG-TERM NO_2 EXPOSURE

Species	Concentration, ppm	Concentration, mg/m^3	Exposure	Effects	Comment	Reference
Rabbits	0.25	0.5	4 hr/day, 6 days	Lung: Structural changes in collagen (electron microscopy)	Still apparent 7 days after final exposure	20
Mice	0.5	0.9	6-24 hr/day, 3-12 mo	Lung: Pneumonitis; alveolar distension	Possibly emphysematous condition	32
Mice	0.5	0.9	6 - 24 hr/day, 7 days/wk up to 12 mo	Enhanced susceptibility to respiratory infection as demonstrated by increased mortality after challenge with *K. pneumoniae*	Significant increase after 3 mo exposure 24 hr/day and 6 mo at 6 and 18 hr/day. After 12 mo, increase significant at 24 hr/day only	43
Rats	0.8	1.5	Continuous, lifetime	Tachypnea and some terminal bronchiolar hypertrophy		75
Rabbits	1.3	2.5	Continuous, 17 wk	Reduction in rate of weight gain; 2% increase in exposed versus 8% in controls		44
Rats, monkeys	2.0	3.8	Continuous, 3 wk	Blood: Polycythemia	Approximate doubling of red cell number with lesser increases in hematocrit and hemoglobin	37
Rabbits	2.0	3.8	4 days	Morphological changes in terminal bronchiolar epithelium		39

Table 9-10 (continued). SUMMARY OF STUDIES OF TOXICOLOGICAL EFFECTS IN ANIMALS OF LONG-TERM NO_2 EXPOSURE

Species	Concentration, ppm	Concentration, mg/m^3	Exposure	Effects	Comment	Reference
Rats	2.0	3.8	Continuous, lifetime	Lung: Bronchiolar epithelial changes–loss of cilia and reduced cytoplasmic blebbing, crystalloid inclusion bodies	Possibly pre-emphysematous lesion. Effect occasionally seen at 1.5 mg/m^3 (0.8 ppm)	33
Rabbits	3.0	5.6	Continuous, 15 wk	Loss in weight; exposed group showed 10% decrease, 11% increase in control group		44
Guinea pigs	5.0	9.4	4 or 7.5 hr/day, 5 days/wk, 5.5 mo	Lung: Formation of circulating substance–possibly lung antibody; no change in expiratory flow resistance	Amounts increased with length of exposure	16
Monkeys	5.0	9.4	Continuous, 2 mo	Lung: Increased respiratory rate and decreased tidal volume	Minute volume unchanged. One of three monkeys exposed to influenza virus 24 hr before NO_2 died after 5 days	55
Monkeys	10.0	18.8	Continuous, 1 mo	Lung: Both respiratory rate and tidal volume increased	Minute volume increased. Three monkeys exposed to influenza virus 24 hr before NO_2 died within 3 days	55
Rats	10-25	18.8-47.0	Continuous, 4-12 mo	Lung: Voluminous emphysema-like lungs; distended and disrupted alveoli Blood: Polycythemia	Associated thoracic kyphosis and rib flaring	36
Rats	12.0	22.6	Continuous, 9 mo	Weight: Decrease in rate of gain; exposed 20% below controls		33,34

Guinea pigs	15.0	28.2	Continuous, 1 yr	Lung: Formation of circulating substance--possibly lung antibody		16
Guinea pigs	15.0	28.2	10 wk	Enzyme systems: Increased O_2 consumption--spleen, kidney; increased lactic dehydrogenase--lung, liver, kidney; increased aldolase--liver, spleen, kidney, serum		49
Guinea pigs	15-20	28.0-37.6	2 hr/day, 5 day/wk, for 21 mo	Lung: Inflammation of bronchiolar epithelium	Apparent recovery 21 days after final exposure	23
Rabbits	25	47.0				
Rats, rabbits	24.0	45.1	6 hr/day, 5 days/wk, for 43 days (rats) or 45 days (rabbits)	Blood: Methemoglobin formation; mean: rabbits, 2.8% and rats, 13.6%	Animals exposed to welding fumes, which contained 3,075 to 6,150 g/m^3 (2.5-5.0 ppm) NO as well as other contaminants; methemoglobin disappeared during post-exposure period	47
Dogs	25-26	47.0-48.9	6 mo	Lung: Bullous emphysema or scattered small bullae; diffuse increase in collagen	Early lesions of pulmonary emphysema produced; inclusion of ferric oxide with the NO_2 appeared to exert a protective effect	40,41
Hamsters	50	94.0	10 wk	Lung: Increase in volume, with a return to normal 2 wk after cessation of the exposure; transitory epithelial proliferation in respiratory bronchioles	No histological evidence of emphysema	30

In mice, the threshold of increased susceptibility to *Klebsiella pneumoniae* occurred at 6.6 mg/m^3 (3.5 ppm) NO_2 for 2 hours, if the infectious challenge was given within 1 hour after the NO_2 exposure. Mice infected with *K. pneumoniae* and exposed up to 72 hours later to 9.4 or 47.0 mg/m^3 (5 or 25 ppm) NO_2 for 2 hours exhibit similarly enhanced susceptibility to the respiratory infection. Squirrel monkeys exposed to 18.8 mg/m^3 (10 ppm) NO_2 for 2 hours, then challenged with *K. pneumoniae* aerosol, retained the infectious agent in the lungs for extended periods of time. Two-hour exposure to 94.0 mg/m^3 (50 ppm) NO_2 followed by infectious challenge, led to a markedly increased mortality in squirrel monkeys.

Continuous exposure reduces the NO_2 threshold concentration. In long-term studies of mice, significantly increased susceptibility to infection occurred after continuous daily exposure to 940 μg/m^3 (0.5 ppm) NO_2 for 3 months, or 6- or 18-hour daily exposure for 6 months. A significant increase in susceptibility to influenza virus or *K. pneumoniae* has also been found in squirrel monkeys continuously exposed to 18.8 and 9.4 mg/m^3 (10 and 5 ppm) NO_2 for 1 and 2 months, respectively.

Impairment of interferon formation and decreased resistance to viral infection was demonstrated in rabbits exposed to 47.0 mg/m^3 (25 ppm) NO_2 for a 3-hour period.

Systemic effects. Inhalation of NO_2 can produce systemic effects generally secondary to those on the lungs. Monkeys exposed to 28.2 to 94.0 mg/m^3 (15 to 50 ppm) NO_2 for 2 hours exhibited cellular changes in heart, liver, and kidney tissue. Long-term exposure of rats to 2.5 to 5.6 mg/m^3 (1.3 to 3.0 ppm) for 15 to 17 months has been associated with loss of weight and reduced rates of weight gain.

A circulating substance, possibly a lung antibody, has been detected in the blood of guinea pigs exposed to 9.4 mg/m^3 (5.0 ppm) for 4 hours daily, 5 days per week, for 5.5 months. Guinea pigs exposed to 28.2 mg/m^3 (15 ppm) NO_2 for 10 weeks showed an alteration in O_2 consumption and enzyme activities (aldolase and lactic dehydrogenase) in the serum, lung, kidney, liver, and spleen. Rats and monkeys continuously exposed to 3.8 mg/m^3 (2.0 ppm) NO_2 for 3 weeks exhibited marked polycythemia. An increase in circulating methemoglobin was detected in the blood of several species exposed to concentrations greater than 122 mg/m^3 (70 ppm) NO_2 for 1 hour.

Human Studies

The small amount of information available concerning the toxicological effects of the oxides of nitrogen in man pertains to levels of these compounds higher than those normally found in ambient air. Experimental exposure of volunteer subjects to 9.4 mg/m^3 (5 ppm) NO_2 for 10 minutes has produced a substantial, but transient, increase in airway resistance. Other information derived from occupational exposure to higher concentrations of NO/NO_2 mixtures is complicated by the presence of other pollutants. Impaired pulmonary function, indicated by reduced maximal breathing capacity, increased expiratory resistance, and occasional decreased vital capacity, has been observed in patients accidentally exposed to high concentrations of nitrous fumes for a few minutes. In some cases, the impairment has lasted for more than 2 years after the incident. Occupational exposure to 169 mg/m^3 (90 ppm) NO_2 for 30 minutes produced pulmonary edema and decreased vital capacity 18 hours later. Exposure to very high concentrations for about 5 minutes has produced death within 2 days to 5 weeks.

The human threshold for perceiving the odor of NO_2 appears to be about 225 μg/m^3 (0.12 ppm).

G. REFERENCES

1. Gray, E. Le B. Oxides of Nitrogen, Their Occurrence, Toxicity, Hazard. AMA Arch. of Ind. Health, *19*: 479-586, 1959.
2. Flury, F. and F. Zernik. Schadliche Gas. Berlin, Sprunger, 1931.
3. Murphy, S. D., et al. Altered Function in Animals Inhaling Low Concentrations of Ozone

and Nitrogen Dioxide. Amer. Ind. Hyg. Ass. J., *25*: 246-253, 1964.
4. Paul, W. D. and C. R. Kemp. Methemoglobin: A Normal Constituent of Blood. Proc. Soc. Exp. Biol. Med., *56*: 144-196, 1951.
5. Bodansky, M. Methemoglobinemia and Methemoglobin-Producing Compounds. Pharmacol. Rev., *3*: 144-196, 1951.
6. Flury, F. The Role of Nitrogen Monoxide in Poisoning from Nitrogen Fumes. Arch. Exp. Pathol. Pharmakol., *157*: 104-106, 1930.
7. Pflesser, G. The Importance of Nitrogen Monoxide in Poisoning by Nitrous Oxide Fumes. Arch. Exp. Pathol. Pharmakol., *179*: 545-553, 1935.
8. Petr, B. and P. Schmidt. The Influence of an Atmosphere Contaminated with Sulfur Dioxide and Nitrous Gases on the Health of Children. Z. Gesamte Hyg. Grenzgeb., *13*: 34-48, January 1967.
9. Sancier, K. M., G. Freeman, and J. S. Mills. Electron Spin Resonance of Nitric Oxide-Hemoglobin Complexes in Solutions. Science, *137*: 752-754, 1962.
10. Krasna, I. and D. Rittenberg. The Inhibition of Hydrogenase by Nitric Oxide. Proc. Nat. Acad. Sci., *40*: 225-227, 1954.
11. Air Quality Criteria for Photochemical Oxidants. National Air Pollution Control Administration, Washington, D. C. Publication Number AP-63, March 1970.
12. Gray, E. Le B., et al. Toxicity of the Oxides of Nitrogen. AMA Arch. Ind. Hyg. Occup. Med., *10*: 423-425, November 1954.
13. La Towsky, L. W., E. L. MacQuiddy, and J. P. Tollman. Toxicology of Oxides of Nitrogen: Toxic Concentrations. J. Ind. Hyg. Toxicol., *23*: 129-133, 1941.
14. Gray, E. Le B. Toxicity of the Oxides of Nitrogen II. Acute Inhalation Toxicity of Nitrogen Dioxide, Red Fuming Nitric Acid and White Fuming Nitric Acid. AMA Arch. Ind. Hyg. Occup. Med., *10*: 418-422, November 1954.
15. Henry, M. C., R. Ehrlich, and W. H. Blair. Effect of Nitrogen Dioxide on Resistance of Squirrel Monkeys to *Klebsiella pneumoniae* Infection. Arch. Environ. Health, *18*: 580-587, 1969.
16. Balchum, O. J., et al. Nitrogen Dioxide Inhalation and Lung Antibodies. Arch. Environ. Health, *10*: 274-277, 1965.
17. Wagner, W. D., et al. Experimental Study of Threshold Limit of NO_2. Arch. Environ. Health, *10*: 455-466, 1965.
18. Vaughan, T. R., L. F. Jennelle, and T. R. Lewis. Long-Term Exposure to Low Levels of Air Pollutants. Effects on Pulmonary Function in the Beagle. Arch. Environ. Health, *19*: 45-50, 1969.
19. Buell, G. C., Y. Tokiwa, and P. K. Mueller. Lung Collagen and Elastin Denaturation *in Vivo* Following Inhalation of Nitrogen Dioxide. California State Dept. of Public Health. (Presented at the 59th Annual Air Pollution Control Association Meeting. San Francisco. Paper No. 66-7, June 1966.)
20. Mueller, P. K. and M. Hitchcock. Air Quality Criteria-Toxicological Appraisal for Oxidants, Nitrogen Oxides, and Hydrocarbons. J. Air Pollut. Contr. Ass., *19*: 670-676, 1969.
21. Thomas, H. V., P. K. Mueller, and R. L. Lyman. Lipoperoxidation of Lung Lipids in Rats Exposed to Nitrogen Dioxide. Science, *159*: 532-534, 1968.
22. Roehm, J. N., J. G. Hadley, and D. B. Menzel. Antioxidants versus Lung Disease. (Presented at the 10th Annual Hanford Biology Symposium. Richland, Washington, June 2-5, 1970).
23. Kleinerman, J. and G. W. Wright. The Reparative Capacity of Animal Lungs After Exposure to Various Single and Multiple Doses of Nitrite. Amer. Rev. Resp. Dis., *83*: 423-424, 1961.
24. Kleinerman, J. and G. W. Wright. The Effects of Prolonged and Repeated Nitrogen Dioxide Inhalation on the Lungs of Rabbits and Guinea Pigs. (Read Before the Fifth Annual Air Pollution Medical Research Conference California State Department of Public Health. Los Angeles. December 1961.)
25. Thomas, H. V., P. K. Mueller, and G. Wright. Response of Rat Lung Mast Cells to Nitrogen Dioxide Inhalation. J. Air Pollut. Contr. Ass., *17*: 33-35, 1967.
26. Freeman, G. Discussion: Ultrastructural Early Lesion Induced in the Rat Lung by NO_2 in Inhalation Carcinogenesis. (Proc. of Oak Ridge National Laboratory Conference. Gatlinburg. October 8-11, 1969.) AEC Symp. Ser., *18*: 267-269, 1970.
27. Freeman, G., et al. Lesion of the Lung in Rats Continuously Exposed to Two Parts per Million of Nitrogen Dioxide. Arch. Environ. Health, *17*: 181-192, 1968.
28. Stephens, R. J., et al. Ultrastructural Changes in the Terminal Bronchiole of the Rat During Continuous Low-Level Exposure to Nitrogen Dioxide. Exp. Mol. Pathol., In Press, 1970.
29. Evans, M. J., R. J. Stephens, and G. Freeman. Effects of Subacute Concentrations of NO_2 on a Cell Renewal in the Lung. (Presented at 10th Annual Hanford Biology Symposium. Richland, Washington June 2-5, 1970.) Arch. of Intern. Med., In Press, 1970.
30. Kleinerman, J. and C. R. Cowdrey. The Effects of Continuous High Level Nitrogen Dioxide on Hamsters. Yale J. Biol. Med. *40*: 579-585, 1968.

31. Kleinerman, J. Effects of NO_2 in Hamsters: Autoradiographic and Electron Microscopic Aspects. (Inhalation Carcinogenesis, AEC Symposium Series 18. Gatlinburg. CONF-69 1001. October 1969.) 10 pages. National Technical Information Service. Springfield, Virginia, April 1970. 271-281.
32. Blair, W. H., M. C. Henry, and R. Ehrlich. Chronic Toxicity of Nitrogen Dioxide: II. Effects on Histopathology of Lung Tissue. Arch. Environ. Health, *18*: 186-192, 1969.
33. Freeman, G. and G. B. Haydon. Emphysema After Low-Level Exposure to NO_2. Arch. Environ. Health, *8*: 125-128, 1964.
34. Freeman, G. B., et al. Environmental Factors in Emphysema and a Model System with NO_2. Yale J. Biol. Med., *40*: 566-575, 1968.
35. Haydon, G. B., G. Freeman, and N. H. Furiosi. Covert Pathogenesis of NO_2 Induced Emphysema in the Rat. Arch. Environ. Health, *11*: 776-783, 1965.
36. Freeman, G., S. C. Crane, and R. J. Stephens. Pathogenesis of the Nitrogen Dioxide - Induced Lesion in the Rat Lung: A Review and Presentation of New Observations. Am. Rev. Resp. Dis., *98*: 429-443, 1968.
37. Freeman, G., et al. The Subacute Nitrogen Dioxide - Induced Lesion of the Rat Lung. Arch. Environ. Health, *18*: 609-612, 1969.
38. Freeman, G., S. C. Crane, and N. J. Furiosi. Healing in Rat Lung after Subacute Exposure to Nitrogen Dioxide. Amer. Rev. Resp. Dis., *100*: 662-676, 1969.
39. Stephens, R. J., G. Freeman, and M. J. Evans. Connective Tissue Changes in Lungs of Rats Exposed to NO_2 (Presented at 10th annual Hanford Biology Symposium. Richland, Washington. June 2-5, 1970.) Arch. of Intern. Med., In Press, 1970.
40. Riddick, J. H., K. I. Campbell, and D. L. Coffin. Histopathological Changes Secondary to Nitrogen Dioxide Exposure to Dog Lungs. (Presented at the Annual Scientific Assembly, American Society of Clinical Pathologists. Chicago. September 22-30, 1967.)
41. Riddick, J. H., K. I. Campbell, and D. L. Coffin. The Effects of Ferric Oxide Dust Exposure in Nitrogen Dioxide-Impaired Dog Lungs. (Presented at the Annual Scientific Assembly, American Society of Clinical Pathologists. Miami Beach. October 11-19, 1968.)
42. Henry, M. C., and C. Aranyi. Scanning Electron Microscopic Observations of the Effects of Atmospheric Pollutants. Arch. Environ. Health, To Be Published, 1970.
43. Ehrlich, R. and M. C. Henry. Chronic Toxicity of Nitrogen Dioxide: I. Effects on Resistance to Bacterial Pneumonia. Arch. Environ. Health, *17*: 860-865, 1968.
44. Mitina, L. S. The Combined Effects of Small Concentrations of Nitrogen Dioxide and Sulfur Dioxide Upon an Organism. Gig. Sanit., *27*: 3-8, October 1962.
45. Carson, T. R., et al. The Responses of Animals Inhaling Nitrogen Dioxide for Single, Short-Term Exposures. Aeronautical Systems Div., Tech. Report 61-525. October 1961. Amer. Ind. Hyg. Ass. J. *23*: 457-462, 1962.
46. MacQuiddy, E. L., et al. Toxicology of Oxides of Nitrogen: II. Physiological Effects and Symptomatology. J. Ind. Hyg. Toxicol., *23*: 134-140, 1941.
47. McCord, C. P., G. C. Harrold, and S. F. Meek. A Chemical and Physiological Investigation of Electric Arc Welding: III. Coated Welding Rods. J. Ind. Hyg. Toxicol., *23*: 200-215, 1941.
48. Pryor, W. H., H. L. Bitter, and R. J. Bertler. The Effect of Nitrogen Dioxide-Nitrogen Tetroxide on Oxyhemoglobin Dissociation. SAM-TR-67-33. U. S. A. F. Sch. Aerosp. Med., 1-5, April 1967.
49. Buckley, R. D., and O. J. Balchum. Acute and Chronic Exposures to Nitrogen Dioxide Effects on Oxygen Consumption and Enzyme Activity on Guinea Pig Tissues. Arch. Environ. Health, *10*: 220-223, 1965.
50. Ehrlich, R. Effect of Nitrogen Dioxide on Resistance to Respiratory Infection. Bacterial Rev., *30*: 604-614, 1966.
51. Gardner, D. E., R. C. Holyman, and D. L. Coffin. Effects of Nitrogen Dioxide on Pulmonary Cell Population. J. Bacteriol. *98*: 1041-1043, 1969.
52. Ehrlich, R. Effect of Air Pollutants on Respiratory Infection. Arch. Environ. Health, *6*: 638-742, 1963.
53. Purvis, M. R. and R. Ehrlich. Effect of Atmospheric Pollutants on Susceptibility to Respiratory Infection: II. Effect of Nitrogen Dioxide. J. Infec. Dis., *113*: 72-75, 1963.
54. Ehrlich, R., M. C. Henry, and J. Fenters. Influence of Nitrogen Dioxide on Resistance to Respiratory Infections. (Inhalation Carcinogenesis. AEC Symposium Series 18. CONF-691001. Gatlinburg. October 1969.) 14 pages. National Technical Information Service. Springfield, Virginia. April 1970. 243-257.
55. Henry, M. C., et al. Chronic Toxicity of NO_2 in Squirrel Monkeys III. Effect of Resistance to Bacterial and Viral Infection. Arch. Environ. Health, *20*: 566-570, 1970.
56. Fenters, J., et al. Chronic Toxicity of Nitrogen Dioxide. IV. Serologic Response in Squirrel Monkeys Exposed to 5 ppm NO_2 and Influenza

Virus. Arch. Environ. Health, To Be Published, 1970.
57. Ehrlich, R., et al. Effects of Nitrogen Dioxide and Tobacco Smoke on Retention of Inhaled Bacteria. (Third International Symposium on Inhaled Particles. London. September 16-23, 1970.) pp. 7.6.1-7.6.4.
58. Valand, S. B., J. D. Acton, and Q. N. Myrvik. Nitrogen Dioxide Inhibition of Viral Induced Resistance in Alveolar Monocytes. Arch. Environ. Health, *20*: 303-309, 1970.
59. Henschler, D., et al. Olfactory Threshold of Some Important Irritant Gases and Manifestations in Man by How Concentrations. Arch. Gewerbepathol. Gewerbehyg., Berlin, *17*: 547-570, 1960.
60. Shalamberidze, O. P. Reflex Effects of Mixtures of Sulfur and Nitrogen Dioxides. Hyg. Sanit., *32*: 10-14, 1967.
61. Vogh, J. W. Nature of Odor Components in Diesel Exhaust. J. Air Pollut. Contr. Ass., *19*: 773-777, 1969.
62. Rokaw, S. N., et al. Human Exposures to Single Pollutants - NO_2 in a Controlled Environment Facility. (Preprint of presentation at the Ninth AMA Air Pollution Medical Research Conference. Denver. July 24, 1968.)
63. Abe, M. Effects of Mixed NO_2 - SO_2 Gas on Human Pulmonary Functions. Bull. Tokyo Med. Dent. Univ., *14*: 415-433, 1967.
64. Nadel, J. A., et al. Mechanism of Bronchoconstriction During Inhalation of Sulfur Dioxide. J. Appl. Physiol., *20*: 164-167, 1965.
65. Von Oettingen, W. F. The Toxicity and Pollution Dangers of Nitrogen Fumes. U. S. Public Health Service. Washington, D. C. Public Health Bulletin No. 272. 1941. 34 pages.
66. Grayson, R. R. Silage Gas Poisoning: Nitrogen Dioxide Penumonia, a New Disease in Agricultural Workers. Ann. Intern. Med., *45*: 393-408, 1956.
67. Lowry, T. and L. M. Schuman. Silo-Filler's Disease. A Syndrome Caused by Nitrogen Dioxide. J. Amer. Med. Ass., *162*: 153-160, 1956.
68. Adley, F. E. Exposures to Oxides of Nitrogen Accompanying Shrinking Operations. J. Ind. Hyg. Toxicol., *28*: 17-20, 1946.
69. Norwood, W. D., et al. Nitrogen Dioxide Poisoning Due to Metal Cutting with Oxyacetylene Torch. J. Occup. Med., *8*: 201-305, 1966.
70. Harrold, G. C., S. F. Meek, and C. P. McCord. A Chemical and Physiological Investigation of Electric Arc Welding: Bare, Washed Welding Rods. J. Ind. Hyg. Toxicol., *22*: 347-378, 1940.
71. Becklake, M. R., et al. The Long-Term Effects of Exposure to Nitrogen Fumes. Amer. Rev. Tuberc. Pulm. Dis., *76*: 398-409, 1957.
72. Vigdortschik, N. A., et al. The Symptomatology of Chronic Poisoning with Oxides of Nitrogen. J. Ind. Hyg. Toxicol., *19*: 469-473, 1937.
73. Vigliana, B. C. and N. Zurlo. Experiences of the Clinical del Lavoro with Maximum Allowable Concentrations of Industrial Poisons. Arch. Gewerbepathol. Gewerbehyg., *13*: 528-535, 1955.
74. Goodman, L. S. and A. Gelman (eds.). The Pharmacological Basis of Therapeutics. New York: Macmillan, 1965.
75. Freeman, G., N. J. Furiosi, and G. B. Haydon. Effects of Continuous Exposure to 0.8 ppm NO_2 on Respiration of Rats. Arch. Environ. Health, *13*: 454-456, 1966.

CHAPTER 10.
EPIDEMIOLOGICAL APPRAISAL OF NITROGEN OXIDES

A. INTRODUCTION

In contrast to the large volume of toxicological data, epidemiological data on the effects of exposure to nitrogen oxides (NO_x) are meager. Epidemiological studies have been primarily concerned with nitrogen dioxide (NO_2) because it is the oxide of nitrogen that has been most closely associated with effects on health. Generalizations drawn from the toxicological data in chapter 9 form the basis for several of the epidemiology studies presented in this chapter; that is to say, NO_2 exerts its primary toxic effect on the lungs where, in animals, exposure can be associated with increased susceptibility to respiratory infection and emphysematous changes.

Exposure to other pollutants, such as ozone, SO_2, metal fumes, and aerosols, presents a major problem in the interpretation of all information collected. The studies reported here have incorporated some measures to minimize interference by these factors.

B. EPIDEMIOLOGIC EVIDENCE OF LONG-TERM EFFECTS

1. The Cleveland Clinic Study[1] – An Accidental High Exposure

On May 15, 1929, the X-ray room of the Cleveland Clinic was engulfed in flames; 50,000 nitrocellulose films were ignited; and three explosions occurred. The air was filled with concentrations of nitric oxide (NO), carbon monoxide (CO), and hydrocyanic acid (HCN) estimated at 63.3 g/m^3 (51,500 ppm), 45.9 g/m^3 (40,000 ppm), and 6 g/m^3 (5,400 ppm), respectively. Ninety-seven individuals succumbed within 2 hours. Neither CO nor HCN was considered the primary cause of death in the 26 additional individuals who died in less than 1 month, nor were either CO or HCN implicated in 92 nonfatal cases of serious physical injury.

Much later, an epidemiological study was conducted to determine whether survivors of the disaster incurred a risk of dying sooner than nonexposed individuals.

Study groups consisted of individuals who:

1. Were present in the building at the time of the explosion.
2. Entered the building that afternoon.
3. Were exposed to smoke in an adjacent building.
4. Assisted rescue and first-aid workers.

An estimated 98 to 99 percent of the individuals present at the fire were enumerated by searching records and 87 percent of these were actually studied. Individuals, who were present at the fire, but who were not in any of the four exposure groups were used as controls.

When cumulative observed survival rates and cumulative expected survival rates were compared, their ratios, calculated for the years 1929 to 1965, showed no statistically significant differences between mortality rates of exposed and nonexposed groups.

2. The Chattanooga Studies - Ambient NO_2 and Respiratory Illness

a. Community effects

Shy, et al.[2,3] studied the effects of community exposure to nitrogen dioxide in four residential areas in greater Chattanooga. One area, in close proximity to a large TNT plant, had high-NO_2 and low-particulate exposure. Another had high-suspended particulate and low-NO_2 exposure. The two other areas served as "clean" controls.

(1.) Pollutant monitoring. Each area contained three elementary schools. In the high-NO_2 area surrounding the TNT plant, monitoring sites were selected to represent the average pollution level near each of the three schools. Sites for pollution monitoring stations in the high-particulate and the control areas were selected to represent average pollutant exposure on an area-wide basis. The effects of wind, topography, vegetation, and local pollution sources were considered in selecting a site.

Twenty-four-hour total-suspended-particulate matter, suspended nitrates, suspended sulfates, and gaseous nitrogen dioxide concentrations were measured daily at each site, in November 1968 and November 1969. From December 1968 through February 1969, and again in April 1969, these 24-hour concentrations were measured once every 4 days on a systematic sampling schedule (Table 10-1). Twenty-four-hour sulfur dioxide concentrations were measured once weekly in November 1968, but measurements were discontinued when concentrations were found to be less than 2.8 $\mu g/m^3$ (0.015 ppm) at each site.

Integrated 24-hour NO_2 samples were collected by gas-bubbler techniques and

Table 10–1. ARITHMETIC MEAN AND 90TH PERCENTILE CONCENTRATIONS OF POLLUTANTS SAMPLED FOR 24 HOURS AT VARIOUS SITES[2]

Pollutant	Level of exposure	High NO_2 School 1	High NO_2 School 2	High NO_2 School 3	High particulate	Control 1	Control 2
NO_2, ppm	Mean[a]	0.109	0.078	0.062	0.055	0.063	0.043
	90 percentile[b]	0.242	0.141	0.098	0.087	0.096	0.069
	Standard deviation	0.098	0.054	0.040	0.024	0.030	0.021
Suspended nitrate, $\mu g/m^3$	Mean	7.2	6.3	3.8	2.4	2.6	1.6
	90 percentile	14.8	13.4	8.0	4.6	5.9	3.1
	Standard deviation	9.1	5.8	4.6	1.7	2.6	1.0
Suspended sulfate, $\mu g/m^3$	Mean	13.2	11.4	10.0	10.7	9.8	10.0
	90 percentile	22.6	19.2	19.5	17.3	15.8	15.6
	Standard deviation	6.8	6.4	4.9	4.6	4.5	4.5
Total suspended particulates, $\mu g/m^3$	Mean	96	83	63	99	72	62
	90 percentile	183	138	108	181	128	112
	Standard deviation	63	46	42	58	45	35
Soiling index,[c] Coh/1000 lineal ft	Mean	0.80	0.89	0.91	2.09	1.39	1.23
	90 percentile	1.46	1.73	1.84	4.37	3.29	2.53
	Standard deviation	0.51	0.64	0.68	1.67	1.20	0.89

[a]Mean = arithmetic mean of all samples collected.

[b]90 Percentile = concentration exceeded by only 10 percent of samples.

[c]4-hour measurements.

analyzed by the Jacobs-Hochheiser method. Total-suspended-particulate matter, nitrate, and sulfate were collected with a high-volume sampler and subsequently, analyzed.

(2.) Health-effect evaluation. Two possible health effects of NO_2 exposure were investigated in this community during the 1968-69 school year: (1) impaired ventilatory function in elementary school children and (2) increased frequency of acute respiratory illness in family groups. A total of 987 second-grade school children participated in the ventilatory testing. Weekly ventilatory function (Forced Expiratory Volume in 0.75 seconds, $FEV_{0.75}$) of each child was adjusted for differences in individual standing height, for boys and girls, separately. These tests were made during November 1968 and March 1969.

The socioeconomic factors considered were: house value or rent, education of the head of the household, and the number of people in a household (crowding). In each case, the high-NO_2–low-particulate exposure area exibited the highest socioeconomic level, followed in rank by control areas. The low-NO_2–high-particulate exposure area had a distinctly lower socioeconomic level. Home cigarette smoking in the control and high-NO_2 areas differed only by 1 percent. No differences in the duration of residence at the current address were found in any of the areas.

Four ventilatory tests with Stead-Wells spirometers were made weekly in November 1968 and again in March 1969. Analysis of variance was used to determine the statistical significance of factors such as sex, month of test, study area, schools within high-NO_2 area, and the concentration of NO_2 on day of test (see Tables 10-2 and 10-3).

At the beginning of the study, all members of the household of each participating second-grade child were asked to volunteer for the second phase of the study – a prospective study, designed to assess the frequency of acute respiratory disease. A total of 4,043 individuals in 871 families participated. At bi-weekly intervals from November 1968 through April 1969, each family was asked to report the incidence of new colds or sore throats, in terms of the age and sex of sick household members and the severity of the illness. Severity indices included: the presence of fever, length of home confinement, and consultation with a doctor for treatment.

Area differences in respiratory illness rates were analyzed in the following groupings: the entire 24-week period, the influenza-A interval, the interim interval, and the influenza-B interval.

(3.) Conclusions. The ventilatory performance of second-grade school children in the high-NO_2 exposure area was significantly lower than performance of children in the control areas.

Table 10–2. ANALYSIS OF VARIANCE OF EFFECTS ON HEIGHT-ADJUSTED $FEV_{0.75}$ OF SEX OF CHILD, MONTH OF TESTING, AND STUDY AREA [2]

Factor	Mean square	F value	Probability of significant difference
Sex of child	0.3852	189.3	$p<0.01$
Month of test	0.0318	15.6	$p<0.01$
Study area			
High-NO_2 vs. Controls 1 and 2	0.0098	4.8	$p<0.05$
High-TSP[a] vs. Controls 1 and 2	0.0011	0.6	not significant
Control 1 vs. Control 2	0.0004	0.2	not significant

[a]Total suspended particulate matter.

Table 10-3. ANALYSIS OF VARIANCE OF EFFECTS ON HEIGHT-ADJUSTED $FEV_{0.75}$ OF NO_2 CONCENTRATIONS ON DAY OF TEST, SCHOOL WITHIN HIGH-NO_2 AREA, SEX OF CHILD, AND MONTH OF TEST[2]

Factor	Mean square	F value	Probability of significant difference
Sex of child	0.0952	90.3	$p<0.01$
Month of test	0.0178	16.9	$p<0.01$
NO_2 on day of test	0.0009	0.9	not significant
Schools within high-NO_2 area			
Schools 1 vs. 2 and 3	0.0019	1.8	not significant
School 3 vs. 1 and 2	0.0007	0.6	not significant
School 1 vs. 2	0.0030	2.8	not significant

Illness-incidence rates for each family segment in the high-NO_2 area were consistently and significantly higher than indicence rates in the two control areas throughout the entire study period. This increased incidence of acute respiratory disease was observed when the 24-hour NO_2 concentration, measured over a 6-month period, was between 117 and 205 $\mu g/m^3$ (0.062 and 0.109 ppm) and the mean suspended nitrate level was 3.8 $\mu g/m^3$ or greater (Tables 10-1 and 10-4).

School 3 in the high-NO_2/low-particulate area had consistently higher incidence rates than the control areas, even though the average NO_2 level was the same as in control 1 (Table 10-4). The NO_2 and suspended

Table 10–4. AVERAGE BIWEEKLY RESPIRATORY ILLNESS RATES PER 100 FOR EACH FAMILY SEGMENT ACCORDING TO EXPOSURE TO OXIDES OF NITROGEN[3]

Rank of Population by NO_2 exposure			Family segment				
Average 24-hour NO_2, ppm	Average 24-hour suspended nitrate, $\mu g/m^3$	Study population	All family members	Second graders	Siblings	Mothers	Fathers
0.109	7.2	School 1 (high-NO_2)	17.7	23.4	19.9	15.3	11.0
0.078	6.3	School 2 (high-NO_2)	17.5	23.4	18.0	14.4	12.8
0.062	3.8	School 3 (high-NO_2)	16.3	20.4	19.1	13.4	12.1
0.063	2.6	Control 1	13.9	18.0	15.6	11.8	8.8
0.043	1.6	Control 2	15.0	20.1	17.0	12.3	9.6

nitrate levels at school 3 were, however, more variable than control area levels. This variability was associated with higher peak-levels of these pollutants (Table 10-1).

Illness-incidence rates peaked during the A_2/Hong Kong-influenza epidemic and again during an influenza-B outbreak. Families residing in the two polluted areas reported a consistent excess of respiratory illness over the control area, particularly during the A_2 epidemic and the interim period between epidemics.

Control-2 area appeared to be hard hit by the influenza-B epidemic. During this time, illness rates among second graders and mothers in the Control-2 area were equal to rates of comparable family segments in the high-NO_2 area. Rates in Control-1 area, however, remained well below those of the high-NO_2 area in all intervals and in all family segments.

In the high-NO_2 area and in the high-particulate area excess illness rates occurred in all family segments: second-grade children, siblings, fathers, and mothers. The relative excess was 18.3 percent in the high-NO_2 area and 10.4 percent in the high-particulate area (Table 10-5). Area differences in illness rates could not be explained by difference in family composition, economic level, demographic characteristics, or prevalence of chronic conditions. Parental smoking habits did not appear to influence respiratory illness rates in second-grade school children. Exposure to NO_2 and to suspended particulates appear to be the most probable explanation for the observed excess in respiratory illness rates for the population. Severity of illness was essentially the same among all the areas; however, a precise NO_2 dose-response relationship cannot be established from the data.

b. Acute Lower Respiratory Illness in Children

Later, Pearlman, et al.[4] made a retrospective study of acute lower respiratory illness among infant cohorts and first and second graders living in three of the Chattanooga neighborhoods previously described, (all, except the high-particulate area). Pearlman felt, on the basis of aerometric observations, that one of the control areas (Control 1) in the Shy study could properly be designated an intermediate-NO_2-exposure area. The high-

Table 10–5. PERCENT RELATIVE *EXCESS*[a] OF RESPIRATORY ILLNESS AMONG FAMILY SEGMENTS IN EXPOSED VERSUS CONTROL AREAS DURING 24 WEEKS OF STUDY[3]

Family segment	Study areas	
	High-NO_2	High-particulate
All family members	18.8	10.4
Second graders	16.8	12.6
Siblings	16.0	-0.06
Mothers	18.3	34.2
Fathers	31.5	22.8

[a] $\left(\frac{\text{Illness rate of exposed group}}{\text{Illness rate of control groups}} - 1\right) \times 100.$

NO_2 exposure area and the other control area (Control 2) completed the pollutant-dose gradient for the Pearlman study.

Parents were asked to complete a questionnaire for the study-children, reporting the frequency with which a physician had diagnosed croup, bronchitis, pneumonia, and asthma, during the 3-year period from July 1966 through June 1969. Completed questionnaries were returned by 95 percent of the 1820 school children and 84 percent of the 1311 infants still residing in the study areas. Questionnaire responses were validated by physicians' office and hospital records. Sensitivity and specificity exceeded 67 percent for each clinical diagnosis. Parents could usually approximate the number of episodes of each illness but were often unable to date each, precisely, within the 3-year study period.

About 7 percent of the respondents in each area had a history of asthma and were excluded from the main analysis. There were too few asthmatics in each dose-duration category to permit firm conclusions about this subpopulation, but excess bronchitis was noted among the ones residing in the high-exposure area.

Exposure to intermediate and high levels of NO_2 was associated with a significant increase in acute bronchitis among the infant cohort, exposed for 3 years, and school children, exposed for 2 and 3 years. This greater frequency of acute bronchitis was observed when the mean 24-hour NO_2 concentration, measured over a 6-month period, was between 118 and 156 $\mu g/m^3$ (0.063 and 0.083 ppm) (Table 10-6).

Pearlman suggested a threshold relationship for NO_2 exposure and bronchitis since the morbidity excess followed the pollutant gradient only among school children exposed for 3 years. Area differences in total acute lower respiratory illness could be accounted for by bronchitis excess. Croup, pneumonia, and hospitalizations for acute lower respiratory illness did not show significant area differences.

Since the high-NO_2 exposure area of this study included a school with NO_2 levels about the same as the levels in the intermediate area, the difference in NO_2 exposure levels between the high and intermediate areas was diminished. This may explain why the illness rates of some intermediate exposure groups exceeded the rates of high exposure groups. Groups within each school of the high-NO_2 area were too small, however, to allow separate analysis of illness rates.

The Pearlman study replicated Shy's observation that excess acute respiratory illness can be found among children living in NO_2-pol-

Table 10–6. DISTRIBUTION OF CHILDREN REPORTING ONE OR MORE EPISODES OF BRONCHITIS BY LENGTH OF EXPOSURE[4]

6-month mean NO_2	School children exposure, yr			Infant exposure, yr		
	1	2	3	1	2	3
High-NO_2, 156 $\mu g/m^3$ (0.083 ppm)	20.9	34.7[a]	32.2[a]	33.3	37.5	46.8[a]
Intermediate-NO_2, 118 $\mu g/m^3$ (0.063 ppm)	31.6	45.5[a]	31.2[a]	26.2	29.5	50.5[a]
Low-NO_2 (control), 81 $\mu g/m^3$ (0.043 ppm)	25.1	20.3	23.2	21.1	34.0	36.3

[a]Differs significantly from low-NO_2(control) area.

luted areas. He suggested that acute lower-respiratory illness may be a more selective, sensitive indicator of pollutant effect upon host response than is acute general respiratory illness.

3. Czechoslovakian Study - Ambient NO_2 and SO_2 on Peripheral Blood

Studies of Czechoslovakian children[5] who lived near a factory that emits NO_x and SO_2 have produced some positive information not usually sought in the United States. Data are available from three towns: (1) Ohrazenice, which experiences air pollution predominately from SO_2 [concentrations ranging from 30 to 320 $\mu g/m^3$ (0.01 to 0.12 ppm)], with slight admixtures of NO_x [concentrations ranging from 5 to 50 $\mu g/m^3$*]; (2) Rosice, which experiences a larger amount of pollution by NO_x (concentrations ranging from 20 to 70 $\mu g/m^3$), and only a small amount of SO_2 pollution [concentrations up to 12 $\mu g/m^3$ (0.005 ppm)]; and (3) Bohdanec, which has no nearby air pollution sources.

Petr and Schmidt[5] observed a statistically significant difference in the configuration of lymphocytes and monocytes in the smears of peripheral blood from children in the three areas. Although they failed to indicate the size of the study group, they noted that the children did not differ from one another in age or socioeconomic grouping. The authors consider the results to be a sensitive indicator of the children's reaction to SO_2 and nitrous gases in the environment.

When they studied the blood of ten 8- to 10-year-old children from each of the three towns, Petr and Schmidt[5] observed relative increases in resistance to hemolysis and increases in number of immature red blood cells in children from Rosice (high-NO_2/low-SO_2) and Ohrazenice (high-SO_2/low-NO_2) as compared to children from Bohdanec (control). They interpreted this as a compensatory response of the children to noxious substances in the environment. Methemoglobin levels were determined in these same children, and the investigators observed an average of 2.5 percent methemoglobin in the blood of the Rosice (high-NO_2/low-SO_2) children, compared to an average of 0.86 percent in the children from Bohdanec (control). The difference was statistically significant ($p < 0.025$), but the possible contribution of large amounts of nitrates in the drinking water could not be overlooked. When the study was repeated in the Rosice (high-NO_2/low-SO_2) children at a later date, after the source of atmospheric nitrous gases had been controlled, they found no abnormal levels of methemoglobin. The authors implicated the atmospheric NO_x as an etiologic agent in methemoglobinemia.

It is difficult to interpret the significance of hematological changes. More experimental and epidemiological work are needed to confirm these data. For example, no information is given about antecedent viral or bacterial infections, which are known to infect children seasonally, and could explain peripheral blood changes. Controlled studies of red-cell age, and of lymphocyte and monocyte morphology are also needed.

4. Occupational Exposure

While occupational exposures to NO_x are fairly common, either in connection with the use of rocket fuel (nitrogen tetroxide) or dynamite, most occupational exposures include mixed pollutants and many are poorly documented. Among the frequently cited studies are those of Vigdortschik,[6] who presented evidence that nitrogen oxide exposure over many years is capable of producing pulmonary fibrosis; and of McCord,[7] who suggested that some methemoglobinemias occur as a result of nitrogen oxide exposure among welders. Such studies, although suggestive, need further confirmation.

C. IMPLICATIONS OF CHATTANOOGA STUDIES

The most substantial studies of ambient exposure to NO_2 in the United States, the

*It is not possible to calculate ppm from total oxides of nitrogen.

Chattanooga studies, have several implications in regard to respiratory illness, implications that can be extended to other cities.

The National Air Surveillance Network (NASN) has measured NO_2 levels for the years 1967, 1968, and 1969, by the Jacobs-Hochheiser 24-hour method (see chapter 6, Table 6-7), the same method that was employed in the Chattanooga studies. Although the Chattanooga analyses were based on a 6-month NO_2 average, the yearly average would not be substantially different, since NO_2 does not exhibit marked seasonal variations (See discussion chapter 6, Section B.2), and direct comparison of the NASN yearly averages with the lower limit at which health effects were noted in the Chattanooga studies is, therefore, possible. Any site that exhibits a concentration of 113 $\mu g/m^3$ (0.06 ppm) or greater exceeds the Chattanooga health-effect-related NO_2 value. As might be expected, an analysis of the NASN data shows that the yearly NO_2 averages reflect variations according to population densities. Ten percent of cities with populations of less than 50,000 show a yearly average equal to or exceeding 113 $\mu g/m^3$ (0.06 ppm). In the population range from 50,000 to 500,000, 54 percent of the cities equal or exceed a yearly average of 113 $\mu g/m^3$ (0.06 ppm). In the over-500,000 population class, 85 percent of the cities equal or exceed 113 $\mu g/m^3$ NO_2 (0.06 ppm) on a yearly average.

It is important to realize that the relationship to population is a very general one and that the exact location of the sampling site within each city or general area plays a dominant role in determining the concentrations measured. This is illustrated by an examination of those locations in Table 6-7 (chapter 6) that have more than one station in operation. The levels reported in 1969 by two Denver and two Chicago stations show that within any one city the NO_2 yearly averages can differ by factors of 2 to 3, depending on the site.

D. FUTURE RESEARCH NEEDS

The dearth of information relating community health effects to ambient concentration of NO_x is apparent, but the following studies are particularly needed:

1. Studies to determine which segment of the population is most susceptible to abnormal levels of NO_x.
2. Studies to precisely delineate the relationship among methemoglobin levels, peripheral blood alterations, and NO_x concentrations.
3. Replication of studies of the enhanced susceptibility to respiratory infection at various NO_x levels.
4. Studies to determine the relationship of other pollutants to the oxides of nitrogen and their combined effect on human health.

E. SUMMARY

There is a paucity of well-controlled epidemiological studies involving exposure of human populations to ambient levels of nitrogen oxides. The Cleveland Clinic Fire of 1929 exposed many employees to extremely high levels of HCN, CO, and NO_x. A follow-up study of survivors revealed no increase in mortality between the exposed group of employees and a control group.

Effects of community exposure to NO_2 in four residential areas of greater Chattanooga were studied. In one area, NO_2 concentrations were high and particulate matter was low; in another, NO_2 concentrations were low and particulate matter was high. Two other areas served as "clean" controls.

The ventilatory performance ($FEV_{0.75}$) of children in the high-NO_2 area was significantly reduced, when compared to the performance of children in the control areas. In addition, an 18.8 percent relative excess of respiratory illness occurred among families living in the elevated-NO_2 area. This increased incidence of acute respiratory disease in family groups was observed when the mean 24-hour NO_2 concentration, measured over a 6-month period, was between 117 and 205 $\mu g/m^3$ (0.062 and 0.109 ppm) and the mean suspended-nitrate level was 3.8 $\mu g/m^3$* or greater.

*Suspended nitrate is a solid, and solids are not reported in ppm.

In a retrospective study of the same Chattanooga area, exposure to intermediate and high levels of NO_2 in ambient air was associated with a significant increase in the frequency of acute bronchitis. This occurred among infants, exposed for 3 years, and school children, exposed for 2 and 3 years. This increased frequency of acute bronchitis was observed when the mean 24-hour NO_2 concentration, measured over a 6-month period, was between 118 and 156 $\mu g/m^3$ (0.063 and 0.083 ppm) and the mean suspended nitrate level was 2.6 $\mu g/m^3$ or greater.

A report from Czechoslovakia indicates that NO_x produced several alterations in the peripheral blood. Increased levels of methemoglobin were observed in school children residing in a town that had relatively high ambient levels of NO_x; however, the findings in this report require further clarifying investigation.

The Chattanooga studies have several implications in regard to respiratory illness–implications that can be extended to other cities. Since NO_2 does not exhibit marked seasonal variations, it is possible to make direct comparison of the NASN yearly averages with the lower limit at which health effects were noted in the Chattanooga studies. Any site where a concentration of 113 $\mu g/m^3$ (0.06 ppm) or greater is measured exceeds the Chattanooga health-effect-related NO_2 value. Ten percent of cities in the United States with populations of less than 50,000 have a yearly average equal to or exceeding 113 $\mu g/m^3$ (0.06 ppm). In the population range from 50,000 to 500,000, the yearly average NO_2 equals or exceeds 113 $\mu g/m^3$ (0.06 ppm) in 54 percent of the cities. In the over-500,000 population class, 85 percent of the cities equal or exceed 113 $\mu g/m^3$ (0.06 ppm) yearly average NO_2.

F. REFERENCES

1. Gregory, K. L., V. F. Malinoski, and C. R. Sharp. Cleveland Clinic Fire: Survivorship Study, 1929-1965. Arch. Environ. Health, *18*: 508-515, April 1969.
2. Shy, et al. The Chattanooga School Study: Effects of Community Exposure to Nitrogen Dioxide. Methods, Description of Pollutant Exposure and Results of Ventilatory Function Testing. To be Published in J. Air Pollut. Contr. Ass. (1970).
3. Shy, et al. The Chattanooga School Study: Effects of Community Exposure to Nitrogen Dioxide. Incidence of Acute Respiratory Illness. To be published in J. Air Pollut. Contr. Ass., (1970).
4. Pearlman, M. E., et al. Nitrogen Dioxide and Lower Respiratory Illness. Submitted to Pedatrics, 1970.
5. Petr, B. and P. Schmidt. The Influence of an Atmosphere Contaminated with Sulfur Dioxide and Nitrous Gases on the Health of Children. Z. Gesamte Hyg. Grenzgeb., *13*: 34-48, 1967.
6. Vigdortschik, N. A., et al. The Symptomatology of Chronic Poisoning with Oxides of Nitrogen. J. Ind. Hyg. Toxicol., *19*: 469-473, 1937.
7. McCore, C. P., G. C. Harrold, and S. F. Meek. A Chemical and Physiological Investigation of Electric Arc Welding. J. Ing. Hyg. Toxicol., *23*: 200-215, 1941.

CHAPTER 11.

SUMMARY AND CONCLUSIONS

A. INTRODUCTION

This document contains a consolidation and an assessment of the current state of knowledge regarding the group of air pollutants known as the oxides of nitrogen. This chapter provides a concise presentation of that information, including reasonable conclusions for evaluating the concentrations of nitrogen oxides (NO_X) and the accompanying situations that have an effect on either health or welfare. The studies and data cited comprise the best available basis for developing specific standards for NO_X in the ambient air, aimed at protecting public health and the environment.

Although the essential role of NO_X in the production of photochemical oxidants is treated from the physical-chemical standpoint in this document, little research has been done to demonstrate the significance of the indirect effects of NO_X on health, vegetation, and materials through the photochemical reaction mechanism; thus, only the direct effects of NO_X are treated here. A P C O publication AP-63, *Air Quality Criteria for Photochemical Oxidants*, provides a comprehensive review of photochemical oxidant effects.

Units of pollution concentration used in this document are expressed as both mass per unit volume (e.g., micrograms per cubic meter, $\mu g/m^3$) and as volume-ratios (e.g., parts per million, ppm). Conversion between these units requires a knowledge of the gas density, which varies with temperature and pressure measurement. In this document 25°C (77° F) has been taken as standard temperature, and 760 mm Hg (atmospheric pressure at sea level) as standard pressure. All references to NO_X are expressed in terms of NO_2 mass per unit volume on the basis of the conversion formula: ppm x 1880 = $\mu g/m^3$ at 25° C, 760 mm Hg, unless otherwise specified. Similarly, hydrocarbons and oxidant concentrations are expressed as mass of methane and ozone per unit volume, respectively.

B. PROPERTIES OF NITROGEN OXIDES AND PHYSICAL EFFECTS ON LIGHT TRANSMISSION

Of the oxides of nitrogen known to exist, only two, nitric oxide (NO) and nitrogen dioxide (NO_2) are emitted to the atmosphere in significant quantities. Nitric oxide is formed during all atmospheric combustion processes in a spontaneous chemical reaction between the nitrogen and oxygen in the air. The amount formed depends on the combustion temperature, the concentration of both reactants and products, and the length of time favorable conditions persist for the reaction.

Both NO and NO_2 are formed when combustion temperatures exceed approximately 1093°C (2000°F), but usually less than 0.5 percent is NO_2. More NO_2 is formed when atmospheric oxygen (O_2) reacts with NO, but at the dilute concentrations of NO characteristically found in ambient atmospheres, this reaction proceeds very slowly. During the initial phases of exhaust gas dilution, however, the concentration of NO is high, and forces the reaction to proceed more rapidly until the exhaust has been sufficiently diluted (to 1 ppm or less). At that time the major process for converting NO to NO_2 reverts to the photochemical cycle.

Visibility reduction is common in polluted atmospheres. Scattering and absorption of light rays by particles and gases reduce the

brightness and contrast of distant objects. The degree of reduction depends on the concentration and properties of the pollutants. Nitrogen dioxide absorbs light energy over the entire visible spectrum, although primarily in the shorter, blue-wavelength regions; thus, NO_2 can by itself reduce visibility. At present, however, under most ambient conditions, aerosols make the major contributions to visibility reduction.

C. SOURCES AND CONTROL OF ATMOSPHERIC NITROGEN OXIDES

On a global basis, the total amount of nitrogen oxides generated by natural sources exceeds the amount from man-made, technological sources. Natural scavenging processes keep background levels in nonurban areas low, on the order of 8 $\mu g/m^3$ (4 ppb) NO_2 and 2 $\mu g/m^3$ (2 ppb) NO. In urban areas, however, where 60 percent of the technological sources are located, the levels are frequently higher because pollutants are added faster than scavenging processes control them.

Fuel combustion is the major source of technological NO_x air pollution. Chemical processing is responsible for high, but localized emissions.

Control of NO_x emissions has been directed at both combustion sources and chemical processes. For stationary combustion sources, the control principle has been based on reducing either the flame temperature or the availability of oxygen, to prevent NO formation. Similar principles of control are applicable to motor vehicles. Catalytic principles, which have been applied to reduce NO_x emissions from chemical processes, are also being investigated for possible use in control of NO_x in motor-vehicle exhaust.

D. CHEMICAL INTERACTIONS OF NITROGEN OXIDES IN THE ATMOSPHERE

The role of NO_x in the generation of photochemical oxidants is a complex function of the interaction of certain hydrocarbons (HC) with the NO_2 photolytic cycle, which is discussed here as well as in the document AP-63, *Air Quality Criteria for Photochemical Oxidants* and the document AP-64, *Air Quality Criteria for Hydrocarbons.*

In order to fully describe the HC-NO_x-OX interrelationship a comprehensive simulation model that takes into account emission rates, chemical reactions, and atmospheric dispersion factors, is required. In the absence of such an applicable model an observation-based model was developed and applied to ambient aerometric data. This latter model is restricted to defining the maximum daily oxidant that may be reached from a given early-morning precursor level and, therefore, the model results in definition of the upper-level oxidant curve, as a function of precursor concentrations. The model for the NO_x-OX relationship indicates that an NO_x 6- to 9-a.m. value of 80 $\mu g/m^3$ (0.04 ppm) is associated with the reference concentration of 200 $\mu g/m^3$ (0.1 ppm) maximum daily 1-hour-average oxidant.

The reference concentration of 200 $\mu g/m^3$ OX used here was selected on the basis of convenience and does not represent the lowest health-related value (130 $\mu g/m^3$ OX) expressed in APCO publication AP-63, *Air Quality Criteria for Photochemical Oxidants.*

Application of the observation-based model to ambient NO_x, HC, and oxidant interrelationships showed that the peak oxidant level is dependent on the concentration of both reactants. Analysis of data from three urban areas indicates that a reference concentration of 200 $\mu g/m^3$ (0.1 ppm) maximum daily 1-hour-average oxidant is associated with an HC range of 200 to 930 $\mu g/m^3$ (0.3 to 1.4 ppm C) 6- to 9-a.m. nonmethane hydrocarbon, when the 6- to 9-a.m. average NO_x, expressed as NO_2, was below 80 $\mu g/m^3$ (0.04 ppm). Similarly, observation of the 200 $\mu g/m^3$ (0.3 ppm C) nonmethane HC level showed NO_x in the range of 80 to 320 $\mu g/m^3$ (0.04 to 0.16 ppm), expressed as NO_2. These conclusions are supported by the predominance of weekend data near the low-concentration end of the upper-limit oxidant curve, which reflects the lower oxidant values from lower emissions on weekends.

E. METHODS FOR MEASURING NITROGEN OXIDES

Research is still needed to develop and thoroughly evaluate more sensitive, reliable, and practical methods for measuring ambient levels of NO, NO_2, and NO_x. All of the field techniques in use at present can measure only NO_2 directly; NO must be oxidized to NO_2, then measured. NO_x can be determined either by summing NO and NO_2 concentrations that have been measured independently or by oxidizing NO to NO_2, then measuring the total as NO_2.

Any method used for measuring NO_2 in the ambient air should be calibrated against atmospheres containing known amounts of NO_2. The use of permeation tubes to generate the test atmospheres is recommended.

Two techniques are currently used in atmospheric monitoring programs. For sampling periods of 30 minutes or less, the most suitable currently available method for measuring NO_2 is the colorimetric Griess-Saltzman method. This method can also be automated for continuous measurement. The Jacobs-Hochheiser method is the most suitable of the available methods for long-term (up to 24 hours) sampling, or for situations requiring a delay of the analysis for more than 4 hours after sampling. The Griess-Saltzman and Jacobs-Hochheiser methods are not interchangeable, can yield different results, and must be chosen carefully, according to the purposes of the sampling to be done.

When used in conjunction with an oxidizing prescrubber to convert NO to NO_2, the continuous Griess-Saltzman method can be used to measure NO in ambient air in either a series or parallel mode, with the same or separate samples of air. Problems exist in obtaining complete NO to NO_2 oxidation, and researchers disagree as to which of the two modes is more satisfactory.

F. ATMOSPHERIC LEVELS OF NITROGEN OXIDES

Continuous measurement of the oxides of nitrogen by various monitoring networks has made it possible to compile tables of mean concentrations averaged over different time periods and to relate various temporal patterns to photochemical and meteorological parameters.

Both NO and NO_2 concentrations display distinct diurnal variations dependent on both the intensity of the solar ultraviolet energy and the amount of atmospheric mixing. In many sampling areas, these variations are also associated with the traffic patterns.

Nitric oxide shows an additional seasonal variation, with higher values occurring during the late fall and winter months. Nitrogen dioxide, however, does not display any distinct seasonal patterns.

The effect of meteorological parameters on NO and NO_2 concentrations has been reasonably well documented. As might be expected, periods of stagnation and high traffic volume in urban areas have resulted in high peak levels of NO_x.

Continuous measurement has indicated that peak values of NO above 1.2 mg/m^3 (1 ppm) are widespread, but NO_2 concentrations have rarely been measured at this level. Peak concentrations of NO_2 in urban areas rarely exceed 0.94 mg/m^3 (0.5 ppm).

Considerable differences were found among NO_2 data collected at the same site, at the same time, but by different methods. The methods of NO, NO_2, and NO_x measurement are still in need of refining and must be judged accordingly.

G. EFFECTS OF NITROGEN OXIDES ON MATERIALS

Significant effects of NO_x have been observed and studied on three classes of materials: textile dyes and additives, natural and synthetic textile fibers, and metals.

The most pronounced problem is associated with textile dyes and additives. Fading of sensitive disperse dyes used on cellulose acetate fibers has been attributed to NO_2 levels below 188 mg/m^3 (<100 ppm). Loss of color, particularly in blue- and green-dyed cotton and viscose rayon, has occurred in gas dryers where NO_x concentrations range from 1.1 to 3.7 mg/m^3 (0.6 to 2 ppm). Yellow discoloration in undyed white and pastel-colored

fabrics has recently been attributed to NO_x by controlled laboratory experiments.

Laboratory and field observations have shown that cotton and Nylon textile fibers can be deteriorated by the presence of NO_x, but specific reactants and threshold levels are undetermined at this time.

Failure of nickel-brass wire springs on relays has been related to high particulate nitrate levels. This type of stress corrosion has been observed when surface concentrations of particulate nitrates have exceeded 2.4 $\mu g/cm^2$ and relative humidity was greater than 50 percent. Another type of this corrosion has been associated with annual average particulate nitrate concentrations of 3.0 and 3.4 $\mu g/m^3$ with corresponding NO_x levels of 124 and 158 $\mu g/m^3$ (0.066 and 0.084 ppm).

H. EFFECTS OF NITROGEN OXIDES ON VEGETATION

The degree of injury occurring with the lower concentrations of NO_2 present in the atmosphere remains to be determined. Exposure of many kinds of plants to concentrations of NO_2 above 47 mg/m^3 (25 ppm) for any extended period causes acute necrotic leaf injury. Such lesions are usually characteristic for each plant, but their nonspecific character in relation to other toxicants renders these symptoms of little value in diagnosing NO_2 damage in the field.

The 1-hour visible-injury-threshold value for NO_2 can be achieved by exposing plants to 18.8 to 28.2 mg/m^3 (10 to 15 ppm). Increasing the exposure time, however, obviates the threshold level; 4.3 to 6.6 mg/m^3 (2.3 to 3.5 ppm) NO_2 administered for 8 to 21 hours and 1.9 mg/m^3 (1 ppm) NO_2 for 48 hours cause equivalent leaf injury. Continuous fumigation with 940 $\mu g/m^3$ (0.5 ppm) NO_2 for 35 days resulted in leaf drop and chlorosis in citrus, but no actual necrotic lesions developed.

The effects of exposure to low levels of NO_2 for extended periods are less evident. Recently completed studies suggested that 470 $\mu g/m^3$ (0.25 ppm) or less of NO_2, supplied continuously for 8 months will cause increased leaf drop and reduced yield in navel oranges.

The mechanism(s) by which NO_x causes direct injury to plants can only be postulated at this time. Evidence of diurnal fluctuation in sensitivity to NO_2 has been presented, and could indicate that the pollutant is reacting with a particular plant metabolite, which only accumulates at certain periods during the day. The absence of a protective metabolite within the plant at certain periods would also cause a diurnal sensitivity.

Limited information regarding the effect of nitric oxide on photosynthesis indicates that NO would reduce the growth of plants if concentrations in the range of 3.8 to 7.5 mg/m^3 (2.0 to 4.0 ppm) persisted continuously.

I. TOXICOLOGICAL EFFECTS OF NITROGEN OXIDES

Both of the prominent oxides of nitrogen present in ambient air are potential health hazards. At ambient concentrations, NO presents no direct threat to general health; NO_2 does, however. Effects of NO_2 determined in extensive studies are summarized in Table 11-1.

The toxicology of nitrous oxide (N_2O) and other oxides of nitrogen does not appear to be relevant to the problems of ambient air pollution at the present time.

1. Nitric Oxide

NO is not an irritant and is not considered to have adverse health effects at concentrations found in the atmosphere. Its greatest toxic potential at ambient concentrations is related to its tendency to undergo oxidation to NO_2. A 12-minute exposure to 3,075 mg/m^3 (2,500 ppm) NO has proved lethal to mice. In addition, NO has been observed to inhibit bacterial hydrogenase activity at lower concentrations–24.6 mg/m^3 (20 ppm). This inhibition was reversible, however, until the exposure reached about 12,300 mg/m^3 (10,000 ppm).

2. Nitrogen Dioxide

NO_2 exerts its primary toxic effect on the lungs. High concentrations, greater than 188 mg/m^3 (100 ppm), are lethal to most animal

Table 11–1. SUMMARY OF REPRESENTATIVE NO_2 EFFECTS

Effect	NO_2 concentration, ppm	NO_2 concentration, $\mu g/m^3$	Duration	Comment	Reference
Lowest level associated with reference oxidant production of 200 $\mu g/m^3$ (0.1 ppm)	0.04	80	3 hr (6 to 9 a.m.)		1
Increased incidence of acute respiratory disease in families	0.062 to 0.109	117 to 205	2 to 3 yr	Chattanooga study - 6-mo mean concentration range	2
Increased incidence of acute bronchitis in infants and school children	0.063 to 0.083	118 to 156	2 to 3 yr	Chattanooga study - 6-mo mean concentration range	3
Human olfactory threshold	0.12	225	---	Immediate perception	4
Rabbits - structural changes in lung collagen	0.25	470	4 hr/day for 6 days	Still apparent 7 days after final exposure	5
Navel orange - leaf abscission; decreased yield	0.25	470	8 mo, continuously		6
Rats - morphological changes in lung mast cells characterized by degranulation	0.5	940	4 hr	Possibly precedes onset of acute inflammatory reaction	7
	1.0	1880	1 hr		
Mice - pneumonitis; alveolar distension	0.5	940	6 to 24 hr/day for 3 to 12 mo	Possibly emphysematous condition	8
Mice - increased susceptibility to respiratory infection	0.5	940	6 to 24 hr/day up to 12 mo	Based on mortality following challenge with *K. pneumoniae*	9
Navel orange - leaf abscission, chlorosis	0.5	940	35 days, continuously		6
Rats - tachypnea, terminal bronchiolar hypertrophy	0.8	1504	Lifetime, continuously		10

Table 11–1 (Continued). SUMMARY OF REPRESENTATIVE NO_2 EFFECTS

Effect	NO_2 concentration, ppm	NO_2 concentration, $\mu g/m^3$	Duration	Comment	Reference
Rats - bronchiolar epithelial changes, loss of cilia, reduced cytoplasmic blebbing, crystalloid inclusion bodies	0.8 to 2.0	1504 to 3760	Lifetime, continuously	Possibly pre-emphysematous lesion	11
Rabbits - structural changes in lung collagen	1.0	1880	1 hr	Denaturation of structural protein suggested	12
Sensitive plants - visible leaf damage	1.0	1880	21 to 48 hr		13
Rats, monkeys - polycythemia	2.0	3760	3 wk, continuously	Approximate doubling of red cell number with lesser increases in hematocrit and hemoglobin	14
Man - increase in airway resistance	5	9,400	10 min	Transient	15
Monkeys - tissue changes in lungs, heart, liver, and kidneys	15 to 50	28,200 to 94,000	2 hr	Degree of damage directly related to concentration of NO_2	16

species; 90 percent of the deaths are caused by pulmonary edema.

The concentration time product determines nonlethal morbidity effects of NO_2 exposures. At 940 $\mu g/m^3$ (0.5 ppm) for 4 hours or 1.9 mg/m^3 (1.0 ppm) for 1 hour, mast cells of rat lungs became degranulated, possibly signifying the onset of an acute inflammatory reaction. These cells returned to normal 24 hours after exposure was terminated. Lung proteins, collagen and elastin, were found to be altered structurally in rabbits exposed to 1.9 mg/m^3 (1 ppm) NO_2 for 1 to 4 hours. The condition was also reversible within 24 hours. Similar changes were observed in rabbits exposed to 470 $\mu g/m^3$ (0.25 ppm) NO_2, 4 hours a day for 6 days, except that recovery was delayed and some denaturation was still apparent 7 days after the final exposure. Denaturation of collagen and elastin associated with repeated exposure to NO_2 has been suggested as a possible factor in the pathogenesis of pulmonary emphysema.

Early pulmonary emphysema-type lesions have been observed in dogs exposed continuously to 47.0 mg/m^3 (25 ppm) for 6 months. In lung tissue from monkeys exposed to 18.8 to 94.0 mg/m^3 (10 to 50 ppm) NO_2 for 2 hours, alveoli were expanded and had thin septal walls. This response involved increasing numbers of alveoli as the NO_2 concentration was increased. Hyperplasia has been observed in respiratory bronchiolar epithelium of hamsters exposed to 94.0 mg/m^3 (50 ppm) for 10 weeks, and a similar response was noted in major bronchi and distal portions of

the respiratory tract of hamsters exposed to 18.8 mg/m^3 (100 ppm) for 6 hours.

Long-term exposures to NO_2 concentrations that do not produce acute inflammatory responses have a cumulative, sustained effect, suggestive of a pre-emphysematous condition. Examination of lung tissue from rats exposed to 3.8 mg/m^3 (2 ppm) for their natural lifetimes showed loss of cilia; decreased bronchiolar blebbing; and intercellular, crystalloid, rod-shaped, inclusion bodies. Similar effects have been seen in lungs of rats continuously exposed to 1.5 mg/m^3 (0.8 ppm). Alveoli in lungs of mice exposed to 940 μg/m^3 (0.5 ppm) for 3 to 12 months on 6-, 18-, and 24-hour daily schedules have shown increase in size from distension rather than from septal breakage. The accompanying inflammation of the bronchiolar epithelium and reduction in distal airway size suggested the development of early focal emphysema.

Rats chronically exposed to 18.8 to 47.0 mg/m^3 (10 to 25 ppm) NO_2 developed compensatory changes, such as polycythemia and thoracic kyphosis, with lateral flaring of the ribs.

Since certain pathological changes seen in animals after experimental NO_2 exposure are similar to changes that occur in the pathogenesis of chronic obstructive pulmonary disease in man, it is suggested that long-term, low-level exposures to NO_2 may play a significant role in the development of chronic lung disease.

Exposure of mice, hamsters, and squirrel monkeys to NO_2 increased susceptibility to bacterial pneumonia and influenza infection. The susceptibility has been demonstrated by a significantly increased mortality, decreased survival time, and a reduction in ability to clear infectious agents from the lungs. In mice, threshold for increased susceptibility to *Klebsiella pneumoniae* occurred after exposure to 6.6 mg/m^3 (3.5 ppm) NO_2 for 2 hours, if the infectious challenge was given within 1 hour after the NO_2 exposure. Squirrel monkeys exposed to 18.8 mg/m^3 (10 ppm) NO_2 for 2 hours and then challenged with *K. pneumonia* aerosol retained the infectious agent in their lungs for extended periods of time.

In long-term studies of mice, significantly increased susceptibility to infection occurred after continuous daily exposure to 940 μg/m^3 (0.5 ppm) NO_2 for 3 months, and after 6- and 18-hour daily exposures for 6 months. A significant increase in susceptibility to influenza virus or *K. pneumoniae* was also seen in squirrel monkeys continuously exposed to 18.8 and 9.4 mg/m^3 (10 and 5 ppm) NO_2 for 1 and 2 months, respectively. In addition, interferon formation has been impaired and resistance to viral infection has decreased following exposure of rabbits to 47.0 mg/m^3 (25 ppm) NO_2 for 3 hours. Researchers conjecture that such increased susceptibility to infection may also be significant in the pathogenesis of human lung disease.

Inhalation of NO_2 can produce other systemic effects, although these are generally secondary to the effects on the lungs. In monkeys exposed to 28.2 to 94.0 mg/m^3 (15 to 50 ppm) NO_2 for 2 hours, cellular changes appeared in heart, liver, and kidney tissue. A circulating substance, possibly a lung antibody, has been detected in the blood of guinea pigs exposed to 9.4 mg/m^3 (5.0 ppm) for 4 hours daily, 5 days per week for 5.5 months. Rats and monkeys continuously exposed to 3.8 mg/m^3 (2.0 ppm) NO_2 for 3 weeks developed marked polycythemia. Methemoglobin has been detected in the blood of several species exposed to NO_2 concentrations greater than 122 mg/m^3 (70 ppm) for 1 hour.

The small amount of information available concerning the toxicological effects of the oxides of nitrogen in man pertains to levels higher than those found in ambient air. Experimental exposure of volunteer subjects to 9.4 mg/m^3 (5 ppm) NO_2 for 10 minutes has produced a substantial, but transient, increase in airway resistance. Other data, derived from occupational exposure to high-concentration mixtures of NO and NO_2,are complicated by the presence of other pollutants.

Impaired pulmonary function, evidenced by reduced maximal breathing capacity, increased expiratory resistance, and occasional decreased vital capacity, has been observed in patients accidentally exposed to high concentrations of nitrous fumes for a few minutes. Such evidence has persisted for more than 2 years after the exposure, in some cases. In one case, occupational exposure to 169 mg/m^3 (90 ppm) NO_2 for 30 minutes produced pulmonary edema and a vital capacity 50 percent lower than expected 18 hours later. Exposure to very high concentrations for about 5 minutes has produced death within 2 days to 5 weeks.

The threshold for odor perception of NO_2 is about 225 μg/m^3 (0.12 ppm).

J. EPIDEMIOLOGICAL APPRAISAL OF NITROGEN OXIDES

Nitrogen dioxide, the only oxide of nitrogen examined in epidemiological surveys, can be significantly correlated with increased respiratory disease at mean 24-hour concentrations between 117 and 205 μg/m^3 (0.062 and 0.109 ppm).

Effects of community exposure to NO_2 were studied in four residential areas of greater Chattanooga. The ventilatory performance ($FEV_{0.75}$) of children in a high-NO_2 area was significantly reduced, when compared to the performance of children in control areas. In addition, an 18.8 percent relative excess of respiratory illness occurred among families exposed to high NO_2 concentrations. A 10.4 percent excess occurred among families in an elevated-particulate area. The increased incidence of acute respiratory disease was observed when the mean 24-hour NO_2 concentration, measured over a 6-month period, was between 117 and 205 μg/m^3 (0.062 and 0.109 ppm) and the mean suspended nitrate level was 3.8 μg/m^3 or greater.

In a retrospective study of the same Chattanooga area, exposure to intermediate and high levels of NO_2 in ambient air was associated with a significant increase in the frequency of acute bronchitis among infants exposed for 3 years and school children exposed for 2 and 3 years. When increase was observed, the mean 24-hour NO_2 concentration, measured over a 6-month period, had ranged between 118 and 156 μg/m^3 (0.063 and 0.083 ppm) and the mean suspended nitrate level had been 2.6 μg/m^3 or greater.

A report from Czechoslovakia indicates that NO_x has produced several alterations in the peripheral blood. Increased levels of methemoglobin were observed in school children residing in a town that had relatively high ambient levels of nitrogen oxides. The findings in that report require further clarifying investigation, however, before conclusions can be drawn.

The Chattanooga studies have several implications in regard to respiratory illness – implications that can be extended to other cities. Since NO_2 does not exhibit marked seasonal variations (See discussion chapter 6, Section B,2), direct comparison of the NASN yearly averages with the lower limit at which health effects were noted in the Chattanooga studies is, therefore, possible. Any site that exhibits a concentration of 113 μg/m^3 (0.06 ppm) or greater exceeds the Chattanooga health-effect-related NO_2 value. Ten percent of cities with populations of less than 50,000 show a yearly average equal to or exceeding 113 μg/m^3 (0.06 ppm). In the population range from 50,000 to 500,000, 54 percent of the cities in the United States equal or exceed a yearly average of 113 μg/m^3 (0.06 ppm) NO_2. In the over-500,000 population class, 85 percent of the cities equal or exceed 113 μg/m^3 (0.06 ppm) NO_2 on a yearly average.

K. AREAS FOR FUTURE RESEARCH

1. Environmental Aspects of Oxides of Nitrogen

The fate of as much as 50 percent of the nitrogen oxides that become incorporated into the photochemical complex is still undetermined, for many of the nitrogen oxide end products remain unidentified.

Even for the identified nitrogen oxides the relationship between emissions and air quality

needs further definition through improved instrumentation, expansion of the number of monitoring stations, and more accurate determination of the location and distribution of sources.

A model for predicting upper limits of photochemical oxidant pollutants from observed HC and NO_X levels has been presented, but needs further definition, sophistication, and revision before it can be applied on a practical basis.

2. Effects on Vegetation and Materials

a. Materials

Further research is needed to define reliable dose-response relationships for vulnerable materials. The effects of variables such as temperature, relative humidity, sunlight, and other pollutants on the damage potential of the nitrogen oxides must also be determined.

b. Vegetation

The biochemical, enzymatic, and other metabolic responses of plants to ambient levels of the nitrogen oxides are in need of research-based delineation. Evidence of diurnal variations in sensitivity suggests the existence of either extra-sensitive or protective metabolites in some plants. Evidence of synergistic effects of NO_X in mixtures containing other air pollutants should be investigated further.

3. Toxicity of Oxides of Nitrogen

In order to ascribe toxicity to a specific concentration range of NO_X, the relation of metabolic tissue changes to NO_2 concentration-time responses and the relative importance of low-concentration, long-time exposures versus short-time, peak ambient concentrations should be studied. The interactions of the oxides of nitrogen with particulate pollutants in relation to biochemical, biophysical, infectious, immunological, and ultrastructural response parameters require further research aimed at elucidating possible synergistic damage or protection. Tolerance to NO_2 in the presence of oxidant pollutants has been suggested as a result of exploratory studies, but the biologic importance of such protection needs to be defined.

Further examination of *in vivo* biochemical and biophysical effects of exposure to typical ambient concentrations of the oxides of nitrogen relative to: (1) oxidation of fatty acid double bonds in lung surfactants; and (2) denaturation or alteration of lung proteins (collagen and elastin, enzymes, and cellular membranes) is needed before optimal treatment for, or protection from exposures can be developed.

4. Epidemiology of Oxides of Nitrogen

In order to determine the effect of NO_X on the health of the general population, epidemiological research must be expanded to include: (1) studies to determine which segments of the population are most susceptible to the oxides of nitrogen; (2) studies to precisely delineate the relationship between methemoglobin levels, peripheral blood alterations, and nitrogen oxide concentrations; (3) replication of studies of the enhanced susceptibility to respiratory infection that occurs with exposure to ambient levels of NO_X; and (4) studies to determine the relationship between other pollutants and the oxides of nitrogen and their material effect on human health.

L. CONCLUSIONS

Derived from a careful evaluation of the studies cited in this document, the conclusions given below represent the best judgment of the scientific staff of the Air Pollution Control Office of EPA regarding the effects that may occur when various levels of nitrogen oxides are reached in the ambient air. More detailed information from which the conclusions were derived, and the qualifications that entered into the considerations of these data, can be found in appropriate chapters of this document.

1. Nitric Oxide

a. Effects on Humans

No evidence shows that NO produces significant adverse health effects at the ambient

atmospheric concentrations thus far measured (chapter 9, section B.).

b. Effects on Materials and Vegetation

Damaging effects to materials at ambient pollutant levels of nitrogen oxides have been observed; however, concentrations of NO producing these effects have not been precisely determined (chapter 7, sections C and D).

When beans were exposed to concentrations of 12.3 mg/m^3 (10 ppm), apparent photosynthesis was reduced 50 to 70 percent; when exposed to 4.9 mg/m^3 (4 ppm), a 10 percent reduction occurred (chapter 8, section B).

c. Effects on Laboratory Animals

A concentration of 3,075 mg/m^3 (2,500 ppm) is lethal to mice after a 12-minute exposure. Fully reversible inhibition of bacterial hydrogenase activity occurs at a concentration of 24.6 mg/m^3 (20 ppm) (chapter 9, section B).

2. Nitrogen Dioxide

a. Effects on Humans

(1) Short-Term Exposure. Limited studies show that exposure to NO_2 for less than 24 hours continuously can have several concentration-dependent effects.

1. The olfactory threshold value of NO_2 is about 225 $\mu g/m^3$ (0.12 ppm) (chapter 9, section C.2.a.1).
2. Exposure to 9.4 mg/m^3 (5 ppm) for 10 minutes has produced transient increase in airway resistance (chapter 9, section C.2.a.2).
3. Occupational exposure to 162.2 mg/m^3 (90 ppm) for 30 minutes has produced pulmonary edema 18 hours later, accompanied by an observed vital capacity that was 50 percent of the value predicted for normal function (chapter 9, section C.2.b).

(2) Long-Term Exposure. An increased incidence of acute respiratory disease was observed in family groups when the mean range of 24-hour NO_2 concentrations, measured over a 6-month period, was between 117 and 205 $\mu g/m^3$ (0.062 and 0.109 ppm) and the mean suspended nitrate level during the same period was 3.8 $\mu g/m^3$ or greater.

The frequency of acute bronchitis increased among infants and school children when the range of mean 24-hour NO_2 concentrations, measured over a 6-month period, was between 118 and 156 $\mu g/m^3$ (0.063 and 0.083 ppm) and the mean suspended nitrate level during the same period was 2.6 $\mu g/m^3$ or greater (chapter 10, section C.1).

Yearly average NO_2 concentrations exceed the Chattanooga health-effect-related value of 113 $\mu g/m^3$ (0.06 ppm) in 10 percent of cities in the United States with populations of less than 50,000, 54 percent of cities with populations between 50,000 and 500,000, and 85 percent of cities with populations over 500,000 (chapter 10, section d.).

b. Effects on Materials and Vegetation

Although damage to materials has been attributed to the oxides of nitrogen in ambient atmospheres, the precise air-concentrations producing these effects have not been determined (chapter 7, sections C and D).

Crops and ornamental plants can be classified into three groups with respect to NO_x sensitivity: sensitive, low sensitive, and resistant. Several characteristic effects have been observed among the sensitive plants studied with regard to direct NO_2 exposure.

1. Exposure to 470 $\mu g/m^3$ (0.25 ppm) of NO_2 for 8 months caused leaf abscission and decreased yield among navel oranges (chapter 8, section G).

2. Exposure to NO_2 concentrations of 940 $\mu g/m^3$ (0.5 ppm) for 35 days resulted in leaf abscission and chlorosis on citrus fruit trees (chapter 8, section G).

3. Exposure to NO_2 concentrations of 1.9 mg/m^3 (1 ppm) for 1 day can cause overt leaf injury to sensitive plants (chapter 8, section G).

c. Effects on Laboratory Animals

(1) Short-Term Exposure. Short-term effects of NO_2 on animals can be summarized by the analyses of five salient experiments.

1. Exposure of rats to either 940 $\mu g/m^3$ (0.5 ppm) for 4 hours, or 1.9 mg/m^3 (1.0 ppm) for 1 hour has produced degranulation of lung mast cells (chapter 9, section C.1.b.3).
2. Structural changes in collagen were observed in rabbits exposed to 1.9 mg/m^3 (1.0 ppm) for 1 hour (chapter 9, section C.1.b.2).
3. The threshold for increased susceptibility of mice to respiratory infection by *K. pneumoniae* is 6.6 mg/m^3 (3.5 ppm) for 2 hours (chapter 9, section C.1.d).
4. Exposure of monkeys to 28.2 to 94.0 mg/m^3 (15 to 50 ppm) for 2 hours has produced damage to their lungs, heart, liver, and kidneys and pulmonary changes that resemble those seen in human emphysema (chapter 9, sections C.1.b.3 and C.1.c.1).
5. In rabbits exposed to 47.0 mg/m^3 (25 ppm) for 3 hours interferon formation and resistance to viral infection decreased (chapter 9, section C.1.d).

(2) Long-Term Exposure. Long-term exposure to NO_2 altered several functions in animal circulatory and respiratory systems.

1. Structural changes were found in lung tissue collagen from rabbits exposed to 470 $\mu g/m^3$ (0.25 ppm) 4 hours a day for 6 days (chapter 9, section C.1.b.2).
2. Enhanced susceptibility of mice to respiratory infection by *K. pneumoniae* was observed after 3 months of continuous exposure to 940 $\mu g/m^3$ (0.5 ppm) (chapter 9, section C.1.d).
3. Polycythemia has been reported in rats and monkeys exposed continuously to 3.8 mg/m^3 (2.0 ppm) for 3 weeks (chapter 9, section C.1.c.3).
4. Changes resembling those seen in human emphysema were reported in the following: mice exposed 6 to 24 hours daily, for a period of 3 to 12 months to 940 $\mu g/m^3$ (0.5 ppm) (chapter 9, section C.1.b.3); rats continuously exposed to 18.8 to 47.0 mg/m^3 (10 to 25 ppm) for 4 to 12 months (chapter 9, section C.1.b.3); and dogs continuously exposed to 47.0 mg/m^3 (25 ppm) for 6 months (chapter 9, section C.1.b.3).

3. Other Nitrogen Oxide Effects

a. Photochemical Relationships

An observation-based model applied to ambient NO_X, HC, and oxidant interrelationships showed that peak oxidant yield was dependent on the concentration of both reactants. Analysis of data from three urban areas indicated that a reference concentration of 200 $\mu g/m^3$ (0.1 ppm) maximum daily 1-hour-average OX could be associated with a hydrocarbon range of 200 to 930 $\mu g/m^3$ (0.3 to 1.4 ppm C) 6- to 9-a.m. nonmethane hydrocarbon, expressed as methane, when the 6- to 9-a.m. average NO_X, expressed as NO_2, was below 80 $\mu g/m^3$ (0.04 ppm). A similar observation related an NO_X range of 80 to 320 $\mu g/m^3$ (0.04 to 0.16 ppm), expressed as NO_2, with 200 $\mu g/m^3$ (0.3 ppm C) nonmethane hydrocarbon.

b. Stress Corrosion

Nitrogen oxide reaction products have been associated with corrosion and failure of electrical components. In two cities where this problem has been observed, the 1965 average airborne particulate nitrate concentration were 3.0 and 3.4 $\mu g/m^3$ with associated average NO_X levels of 124 and 158 $\mu g/m^3$ (0.066 to 0.084 ppm).

M. RESUME

Adverse health effects, as evidenced by a greater incidence of acute bronchitis among infants and school children, have been observed, under the conditions prevailing in the areas where studies were conducted, when the mean 24-hour NO_2 concentration, measured by the Jacobs-Hochheiser method, over a 6-month period, varied from 118 to 156 $\mu g/m^3$ (0.063 to 0.083 ppm). On an annual basis, a maximum 24-hour average as low as 284 $\mu g/m^3$ (0.15 ppm) would be expected to be associated with a 6-month mean of 118

$\mu g/m^3$. Adverse health effects, as evidenced by an increased incidence of acute respiratory disease, have been observed in family groups when the mean 24-hour NO_2 concentration measured over a 6-month period was between 117 and 205 $\mu g/m^3$ (0.062 and 0.109 ppm) and the mean suspended nitrate level was 3.8 $\mu g/m^3$ or greater.

An analysis of 3 years of data collected in three American cities shows that on those several days a year when meteorological conditions are most conducive to the formation of photochemical oxidant, and the 6- to 9-a.m. nonmethane hydrocarbon concentration is 200 $\mu g/m^3$ (0.3 ppm C), a 6- to 9-a.m. NO_x concentration (measured by the continuous Saltzman Method and expressed as NO_2) that ranges between 80 and 320 $\mu g/m^3$ (0.04 and 0.16 ppm) would be expected to produce a 1-hour photochemical oxidant level of 200 $\mu g/m^3$ (0.1 ppm) 2 to 4 hours later. If this same functional relationship exists at the lowest levels at which photochemical oxidant has been observed to adversely affect human health, the corresponding nonmethane hydrocarbon concentration would be approximately 130 $\mu g/m^3$ (0.2 ppm C) and the 6- to 9-a.m. NO_x level would be as high as 214 $\mu g/m^3$ (0.11 ppm).

Adverse effects on vegetation such as leaf abscission and decreased yield of navel oranges have been observed during fumigation studies when the NO_2 concentration (measured by the continuous Saltzman Method) was 470 $\mu g/m^3$ (0.25 ppm) during an 8-month period.

Nitrate compounds have been identified with corrosion and failure of electrical components. In two cities where these effects were observed, the average airborne nitrate particulate concentrations were 3.0 and 3.4 $\mu g/m^3$ with associated average NO_x levels of 124 and 158 $\mu g/m^3$ (0.066 and 0.084 ppm).

It is reasonable and prudent to conclude that, when promulgating ambient air quality standards, consideration should be given to requirements for margins of safety that would take into account possible effects on health, vegetation, and materials that might occur below the lowest of the above levels.

N. REFERENCES

1. Air Quality Criteria for Photochemical Oxidants. National Air Pollution Control Administration. Washington, D.C. Publication No. AP-63. March 1970.

2. Shy, et al. The Chattanooga School Study: Effects of Community Exposure to Nitrogen Dioxide. Incidence of Acute Respiratory Illness. To be published in J. Air Pollut. Contr. Ass., 1970.

3. Pearlman, M. E., et al. Nitrogen Dioxide and Lower Respiratory Illness. Submitted to Pediatrics, 1970.

4. Henschler, P, et al. Olfactory Threshold of Some Important Irritant Gases and Manifestations in Man by Low Concentrations. Arch. Gewerbepathol. Gewerbehgy., Berlin, *17*: 547-570, 1960.

5. Mueller, P. K. and M. Hitchcock. Air Quality Criteria-Toxicological Appraisal for Oxidants, Nitrogen Oxides, and Hydrocarbons. J. Air Pollut. Control Ass., *19*: 670-676, 1969.

6. Thompson, C. R., et al. Effects of Continuous Exposure of Navel Oranges to NO_2. Atmos. Environ. In Press, 1970.

7. Thomas, H. V., P. K. Mueller, and G. Wright. Response of Rat Lung Mast Cells to Nitrogen Dioxide Inhalation. J. Air Pollut. Contr. Ass., *17*: 33-35, 1967.

8. Blair, W. H., M. C. Henry, and R. Ehrlich. Chronic Toxicity of Nitrogen Dioxide: II. Effects on Histopathology of Lung Tissue. Arch. Environ. Health, *18*: 186-192, 1969.

9. Ehrlich, R. and M. C. Henry. Chronic Toxicity of Nitrogen Dioxide: I. Effects on Resistance to Bacterial Pneumonia. Arch. Environ. Health, *17*: 860-865, 1968.

10. Freeman, G., N..J. Furiosi and G. B. Haydon. Effects of Continuous Exposure to 0.8 ppm NO_2 on Respiration of Rats. Arch. Environ. Health, *13*: 454-456, 1966.

11. Freeman, G. and G. B. Haydon. Emphysema After Low-Level Exposure to NO_2. Arch. Environ. Health, *8*: 125-128, 1964.

12. Buell, G. C., Y. Tokiwa, and P. K. Mueller. Lung Collagen and Elastin Denaturation *In vivo* Following Inhalation of Nitrogen Dioxide. California State Dept. of Public Health. (Presented at the Annual Air Pollution Control Association Meeting.) San Francisco. Paper No. 66-7, June 1966.

13. Heck., W. W. Plant Injury Induced by Photochemical Reaction Products of Propylene-Nitrogen Dioxide Mixtures. J. Air Pollut. Contr. Ass. *14*: 255-261, July 1964.

14. Freeman, G., et al. The Subacute Nitrogen Dioxide - Induced Lesion of the Rat Lung. Arch. Environ. Health, *18*: 609-612, 1969.

15. Abe, M. Effects of Mixed NO_2 - SO_2 Gas on Human Pulmonary Functions. Bull. Tokyo Med. Dent. Univ., *14*: 415-433, 1967.

16. Henry, M. C., R. Ehrlich, and W. H. Blair. Effects of Nitrogen Dioxide on Resistance of Squirrel Monkeys to *Klebsiella pneumoniae* Infection. Arch. Environ. Health, *18*: 580-587, 1969.

APPENDIX.

VOLUME TO MASS CONVERSION TABLES

VOLUME (ppm) TO MASS ($\mu g/m^3$) CONVERSION TABLE FOR NO
(ppm x 1230 = $\mu g/m^3$ at 25° C, 760 mm Hg)

ppm	$\mu g/m^3$	ppm	$\mu g/m^3$	ppm	$\mu g/m^3$
0.01	10	0.36	440	0.71	870
0.02	20	0.37	460	0.72	890
0.03	40	0.38	470	0.73	900
0.04	50	0.39	480	0.74	910
0.05	60	0.40	490	0.75	920
0.06	70	0.41	500	0.76	930
0.07	90	0.42	520	0.77	950
0.08	100	0.43	530	0.78	960
0.09	110	0.44	540	0.79	970
0.10	120	0.45	550	0.80	980
0.11	140	0.46	570	0.81	1000
0.12	150	0.47	580	0.82	1010
0.13	160	0.48	590	0.83	1020
0.14	170	0.49	600	0.84	1030
0.15	180	0.50	620	0.85	1050
0.16	200	0.51	630	0.86	1060
0.17	210	0.52	640	0.87	1070
0.18	220	0.53	650	0.88	1080
0.19	230	0.54	660	0.89	1090
0.20	250	0.55	680	0.90	1110
0.21	260	0.56	690	0.91	1120
0.22	270	0.57	700	0.92	1130
0.23	280	0.58	710	0.93	1140
0.24	300	0.59	730	0.94	1160
0.25	310	0.60	740	0.95	1170
0.26	320	0.61	750	0.96	1180
0.27	330	0.62	760	0.97	1190
0.28	340	0.63	770	0.98	1210
0.29	360	0.64	790	0.99	1220
0.30	370	0.65	800	1.00	1230
0.31	380	0.66	810		
0.32	390	0.67	820		
0.33	410	0.68	840		
0.34	420	0.69	850		
0.35	430	0.70	860		

VOLUME (ppm) TO MASS ($\mu g/m^3$) CONVERSION TABLE FOR NO_2
(ppm x 1880 = $\mu g/m^3$ at 25° C, 760 mm Hg)

ppm	$\mu g/m^3$	ppm	$\mu g/m^3$	ppm	$\mu g/m^3$
0.01	20	0.36	680	0.71	1330
0.02	40	0.37	700	0.72	1350
0.03	60	0.38	710	0.73	1370
0.04	80	0.39	730	0.74	1390
0.05	90	0.40	750	0.75	1410
0.06	110	0.41	770	0.76	1430
0.07	130	0.42	790	0.77	1450
0.08	150	0.43	810	0.78	1470
0.09	170	0.44	830	0.79	1490
0.10	190	0.45	850	0.80	1500
0.11	210	0.46	860	0.81	1520
0.12	230	0.47	880	0.82	1540
0.13	240	0.48	900	0.83	1560
0.14	260	0.49	920	0.84	1580
0.15	280	0.50	940	0.85	1600
0.16	300	0.51	960	0.86	1620
0.17	320	0.52	980	0.87	1640
0.18	340	0.53	1000	0.88	1650
0.19	360	0.54	1020	0.89	1670
0.20	380	0.55	1030	0.90	1690
0.21	390	0.56	1050	0.91	1710
0.22	410	0.57	1070	0.92	1730
0.23	430	0.58	1090	0.93	1750
0.24	450	0.59	1110	0.94	1770
0.25	470	0.60	1130	0.95	1790
0.26	490	0.61	1150	0.96	1800
0.27	510	0.62	1170	0.97	1820
0.28	530	0.63	1180	0.98	1840
0.29	550	0.64	1200	0.99	1860
0.30	560	0.65	1220	1.00	1880
0.31	580	0.66	1240		
0.32	600	0.67	1260		
0.33	620	0.68	1280		
0.34	640	0.69	1300		
0.35	660	0.70	1320		

SUBJECT INDEX

N

O

P

R

S

T

U

V

W

Z