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NOTICE

The project that is the subject of this report was approved by the Governing Board of the National Research Council, whose members are drawn from the Councils of the National Academy of Sciences, the National Academy of Engineering, and the Institute of Medicine. The members of the Committee responsible for the report were chosed for their special competences and with regard for apropriate balance.

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

FOREWORD

The many benefits of our modern, developing, industrial society are accompanied by certain hazards. Careful assessment of the relative risk of existing and new man-made environmental hazards is necessary for the establishment of sound regulatory policy. These regulations serve to enhance the quality of our environment in order to promote the public health and welfare and the productive capacity of our Nation's population.

The Health Effects Research Laboratory, Research Triangle Park, conducts a coordinated environmental health research program in toxicology, epidemiology, and clinical studies using human volunteer subjects. These studies address problems in air pollution, non-ionizing radiation, environmental carcinogenesis and the toxicology of pesticides as well as other chemical pollutants. The Laboratory develops and revises air quality criteria documents on pollutants for which national ambient air quality standards exist or are proposed, provides the data for registration of new pesticides or proposed suspension of those already in use, conducts research on hazardous and toxic materials, and is preparing the health basis for non-ionizing radiation standards. Direct support to the regulatory function of the Agency is provided in the form of expert testimony and preparation of affidavits as well as expert advice to the Administrator to assure the adequacy of health care and surveillance of persons having suffered imminent and substantial endangerment of their health.

To aid the Health Effects Research Laboratory to fulfill the functions listed above, the National Academy of Sciences (NAS) under EPA Contract No. 68-02-1226 prepares evaluative reports of current knowledge of selected atmospheric pollutants. These documents serve as background material for the preparation or revision of criteria documents, scientific and technical assessment reports, partial bases for EPA decisions and recommendations for research needs. "Nitrogen Oxides" is one of these reports.

John H. Knelson, M.D.
Director
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ABSTRACT

This report is a review of current knowledge of the environmental and health basis for control of manmade sources of nitrogen oxide emissions. The literature review covered the period through 1974. The principal subject areas considered in the report include: sources and control of atmospheric nitrogen oxides; analytical methodology; concentrations and chemical reactions in the atmosphere; and the effects of nitrogen oxides on human health, materials, vegetation, light transmission, and natural ecosystems. Emphasis is primarily on nitric oxide (NO) and nitrogen dioxide (NO_2), designated by the composite formula NOx for nitrogen oxides. The major manmade source is the combustion of fossil fuel. Highest atmospheric concentrations are found in heavily populated, industrialized urban areas. Both acute and chronic health effects resulting from short-term and long-term exposures, are discussed in the report. Effects range from slight increases in airway resistance to death depending upon exposure concentrations.

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References

ACKNOWLEDGMENTS

This document was prepared by the Subcommittee on Nitrogen Oxides. Dr. T. Timothy Crocker served as Chairman and Dr. Carl M. Shy, as Vice Chairman. Although the initial drafts of the various sections were prepared by individuals, the entire document was extensively reviewed by the entire subcommittee and represents a group effort.

The original draft of the Introduction was written by Dr. Carl M. Shy. Chapter 2, describing the properties of nitrogen oxides and their effects on atmospheric light transmission, was the responsibility of Dr. Jack G. Calvert. Dr. Craig T. Bowman prepared Chapter 3, which focuses on the sources and control of atmospheric nitrogen oxides. The author of Chapter 4, concerning the analytical methodology for the determination of nitrogen oxides in air, was Dr. Evaldo L. Kothny. Atmospheric levels of nitrogen dioxide are described in Chapter 5. This was the responsibility of Mr. George Turner. Chapter 6, in which the chemical interactions of nitrogen oxides in the atmosphere are discussed, was prepared by Dr. Jack G. Calvert with the assistance of Dr. Jerome F. Thomas.

Dr. Martin Alexander prepared Chapter 7, which concerns effects on natural ecosystems. Dr. Charles R. Frink provided Dr. Alexander with material on soils; Dr. Boyd R. Strain on plants; Dr. Charles R. Goldman on aquatic life; and Dr. Tony J. Peterle on animals. Effects on materials are covered in Chapter 8, which was authored by Dr. Victor S. Salvin and Dr. Norman Bornstein, with assistance from Dr. Craig T. Bowman. Dr. David C. MacLean provided the material for Chapter 9, which deals with effects

on vegetation. Chapter 10, on health effects, was prepared by Drs. T. Timothy Crocker, Elliot Goldstein, Richard Ehrlich, and Carl M. Shy. The Summary, Conclusions, and Recommendations were a subcommittee effort.

The preparation of the document was assisted by the comments of anonymous reviewers designated by both the Assembly of Life Sciences and the Report Review Committee. The Committee on Medical and Biologic Effects of Environmental Pollutants was very helpful. The subcommittee is particularly indebted to Dr. Ralph G. Smith who served as Associate Editor.

Dr. Robert J.M. Horton of the Environmental Protection Agency gave invaluable assistance by providing the subcommittee with various documents and translations. Informational assistance was obtained from the National Research Council Advisory Center on Toxicology, The National Academy of Sciences Library, the National Library of Medicine, the National Agricultural Library, The Library of Congress, the Department of Commerce Library, and the Air Pollution Technical Information Center.

Initially, the staff officer for the Subcommittee on Nitrogen Oxides was Dr. Elizabeth Force. After her departure from the National Research Council, Mr. John Redmond, Jr. was appointed staff officer and continued in that capacity through completion of this report. The document was edited by Mrs. Frances M. Peter.

CHAPTER 1

INTRODUCTION

Nitrogen oxide formation is an inherent consequence of fossil fuel combustion. Over the next several decades, such combustion will continue to be our major source of energy for electricity generation and motor vehicle propulsion. Unlike many other atmospheric pollutants, more nitrogen oxides are formed at higher combustion efficiencies. Fixation of atmospheric nitrogen and oxygen, and oxidation of nitrogen compounds in fuel are the two processes by which fuel combustion results in nitrogen oxide formation. Nitric oxide (NO) is the dominant oxide released initially; however, the photochemical interaction between nitrogen oxides, hydrocarbons, and various intermediary compounds generated in a sunlight-irradiated atmosphere results in conversion of nitric oxide to nitrogen dioxide (NO_2) and ultimately to further oxidation products. Among the various atmospheric oxides of nitrogen, nitric oxide and nitrogen dioxide, designated by the composite formula, NO_x, are the most important in relation to photochemical reactions, and their known effects on materials, vegetation, and health.

In this monograph, a subcommittee of the National Academy of Sciences, National Research Council has reviewed and summarized a large amount of literature published since the 1971 report of the U.S. Environmental Protection Agency entitled Air Quality Criteria for Nitrogen Oxides.[563] The majority of the subcommittee's work was performed in 1974. Consequently, data from 1975 and 1976 publications could not be included.

The purpose of the monograph is twofold: one, to provide the U.S. Environmental Protection Agency with an assessment of current literature in preparation for that agency's own reevaluation of the scientific basis for the

nitrogen dioxide air quality standard; and, second, to present an independent assessment of the environmental and health bases for control of manmade sources of nitrogen oxide emissions.

Chapters 2 and 3 provide a straightforward presentation of the physical and chemical properties and the sources and control of atmospheric emissions of NO_x. Since the 1971 publication, Air Quality Criteria for Nitrogen Oxides, considerable advances have been made in methodologies to monitor atmospheric NO_x. Existing analytical methodologies are reviewed and evaluated in Chapter 4. The conclusions reached in this chapter may elicit controversy because of disagreements concerning the merits of several basic methods of NO_x monitoring.

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Chapter 5 contains considerable data on atmospheric concentrations of nitrogen oxides. The tables in this chapter will be useful because they compile, in one publication, monitoring data that exclude results obtained by the discredited Jacobs-Hochheiser method. Their estimates of atmospheric NO_x concentrations in the United States are therefore more accurate than were possible in the 1971 air quality criteria document.

In Chapter 6, atmospheric reactions of nitrogen oxides with gaseous organic molecules are discussed. These reactions produce a variety of short-lived intermediary and end products which cannot as yet be identified by conventional analytical techniques. An attempt is made to identify these secondary section products by relating chemical and kinetic laboratory models to atmospheric observations. While it is impossible to make quantitative predictions concerning concentrations of these reaction products, enough information exists to identify the main qualitative features of the reaction model. Attention is focused on new avenues of research on the atmospheric chemistry, and the environmental and health effects of the atmospheric reaction products of nitrogen oxides.

Chapters 7 to 10 deal with the environmental and health effects of nitrogen oxides. Existing evidence implicates nitrogen dioxide as the most reactive and important of the atmospheric nitrogen oxides. However, some of the intermediary and end products of the photochemical conversion of NO_x, identified in Chapter 6, are potentially as important in their environmental and health consequences. By analogy with the emerging concern over reaction products of sulfur dioxide (SO_2), we may anticipate an increasing focus of research on various NO_x reaction products. Unfortunately, insufficient data on these compounds prevented the reviewing subcommittee from making more than a passing reference to them in the chapters concerning environmental and health effects.

This monograph brings together an array of information, not previously reviewed in one report, on material and health effects of nitrogen dioxide. The subcommittee has attempted to place these data in the perspective of the U.S. air quality standard for nitrogen oxides. While finding no basis in this evidence for altering the existing standard, the subcommittee reemphasizes the need for a standard controlling short-term nitrogen dioxide exposures of 24 hr or less. This need is demonstrated in the body of evidence showing acute effects of single or repeated nitrogen dioxide exposures of six or less hours.

CHAPTER 2

NITROGEN OXIDES--THEIR PROPERTIES AND EFFECTS ON ATMOSPHERIC LIGHT TRANSMISSION

Nitrogen and oxygen combine to form several stable oxides of different molecular composition. The two oxides identified as important pollutants in the lower atmosphere are nitric oxide (NO) and nitrogen dioxide (NO_2). Their interconvertibility in photochemical smog has led to their combined designation as NO_x. Nitrous oxide (N_2O) occurs in significant concentrations even in the natural unpolluted atmosphere. It arises from natural biological processes that occur in the soil, and is not classified as an air pollutant. Other oxides of nitrogen, which can occur in polluted atmospheres at very low concentrations are: symmetrical nitrogen trioxide (NO_3), unsymmetrical nitrogen trioxide (O-O-N-O), dinitrogen trioxide (N_2O_3), dinitrogen tetroxide (N_2O_4), and dinitrogen pentoxide (N_2O_5).

OCCURRENCE

Significant concentrations of nitric oxide (several hundred ppm) and smaller concentrations of nitrogen dioxide are formed at the high temperatures accompanying the burning of fossil fuels in mixtures with air. These pollutants are emitted to the atmosphere from auto exhausts, power plant and furnace stacks, incinerators, and vents from certain chemical processes.

Natural biological reactions also generate large quantities of nitric oxide and nitrogen dioxide. However, since the sources of these natural reactions are diffuse and the rate of natural removal processes significant, very low ambient levels of NO_x result.

Most of the NO_x produced in combustion processes is initially in the form of nitric oxide. This is subsequently oxidized in the atmosphere to the more toxic and irritating compound, nitrogen dioxide.

Nitrous oxide is not considered an air contaminant since there is no evidence involving it in the series of complex chemical reactions producing photochemical smog. Nitrous oxide is a product of natural chemical reactions that occur in the soil. Its concentration in the lower atmosphere ranges from 0.47-0.55 mg/m^3 (0.25-0.29 ppm) with only slight seasonal and geographical variations.[470]

The nitrogen oxides, symmetrical nitrogen trioxide, dinitrogen trioxide, dinitrogen tetroxide, and dinitrogen pentoxide have not been identified as trace components of the polluted atmosphere, although their presence at trace levels is inferred from the thermodynamics and kinetics of their reactions as studied in the laboratory. Table 2-1 shows the theoretical concentrations of the different oxides and acids of nitrogen which would be present at equilibrium with molecules of nitrogen, oxygen, and water present in air at 1 atm, 25°C, and 50% RH. Thermodynamic equilibrium is not maintained between most of the components in a polluted, sunlight-irradiated atmosphere. The concentrations of the oxides and acids of nitrogen are controlled largely by the reaction rates and are usually much greater than those expected at equilibrium. Compare column 2 with column 1 in Table 2-1. Theoretically dinitrogen pentoxide, symmetrical nitrogen trioxide, nitrous acid (HONO), and possibly other reactive transient species, are involved significantly in the reactions producing photochemical smog.

TABLE 2-1

Theoretical Concentrations of Nitrogen Oxides and Nitrogen Acids which would be Present at Equilibrium with Molecular Nitrogen, Molecular Oxygen, and Water in Air at 25°C, 1 Atm, 50% RH

	Concentrations in Hypothetical Atmosphere, ppm	
Compound	At Equilibrium	In Typical Sunlight-Irradiated, Smoggy Atmosphere[a]
O_2	2.06×10^{5}	2.06×10^{5}
N_2	7.69×10^{5}	7.69×10^{5}
H_2O	1.56×10^{4}	1.56×10^{4}
NO_2	1.91×10^{-4}	10^{-1}
NO	2.69×10^{-10}	10^{-1}
NO_3	3.88×10^{-16}	$10^{-8} - 10^{-9}$
N_2O_3	2.96×10^{-20}	$10^{-8} - 10^{-9}$
N_2O_4	2.48×10^{-13}	$10^{-7} - 10^{-8}$
N_2O_5	3.16×10^{-17}	$10^{-3} - 10^{-5}$
HONO(cis)	7.02×10^{-9}	10^{-3}
HONO(trans)	1.60×10^{-8}	10^{-3}
$HONO_2$	1.33×10^{-3}	$10^{-2} - 10^{-3}$

[a]Theoretical estimates made using computer simulations of the chemical reactions rates in a synthetic smog mixture. From Demerjian *et al.*[105]

THE PROPERTIES OF THE NITROGEN OXIDES

Nitric Oxide

Because nitric oxide does not absorb visible light, it is colorless. The first electronic absorption band of nitric oxide occurs at wavelengths less than 2,300 Å (Figure 2-1). Nitric oxide is odorless and only slightly soluble in water (0.006 g/100 g of water at 25°C). It is the major oxide of nitrogen formed during high temperature combustion processes resulting primarily from the interaction of the nitrogen content of the fuel with the nitrogen and oxygen present in the air during combustion. The net result is the near establishment of the nitrogen-oxygen-nitric oxide and nitrogen-oxygen-nitrogen dioxide equilibria at the high temperatures of the flame.

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

$$2NO + O_2 \rightleftarrows 2NO_2 \qquad (2)$$

Nitric oxide formation is favored at high temperatures. The concentrations of nitric oxide and nitrogen dioxide are limited by the thermodynamic and kinetic properties of nitrogen, oxygen, nitric oxide, and nitrogen dioxide molecules, and nitrogen and oxygen atoms, the flame temperature, the NO_x, nitrogen, and oxygen molecule concentrations, and the rate gases are transported through the different temperature zones in the combustion chamber. Several hundred to several thousand ppm of nitric oxide may be formed through Reaction (1) during combustion. The effects of increased temperature on the equilibrium concentrations of nitric oxide and nitrogen dioxide formed in heated air are illustrated in Table 2-2.

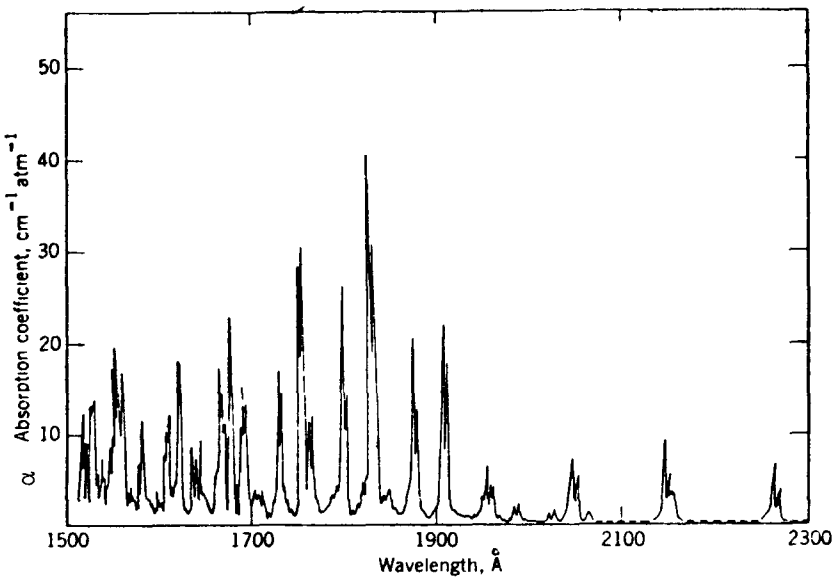


FIGURE 2-1. Absorption spectrum of nitric oxide; the form of the absorption law employed is $I = I_o \exp(-\alpha p l)$, where $\underline{p}$ is in atm at 0°C.

Data from McNesby and Okabe.

TABLE 2-2

Theoretical Equilibrium Concentrations of Nitric Oxide and Nitrogen Dioxide in air (50% RH) at Various Temperatures

	Concentration, mg/m^3 (ppm)	
Temperature °K (°C)	NO	NO_2
298 (24.85)	3.29×10^{-10}	3.53×10^{-4}
	(2.63×10^{-10})	(1.88×10^{-4})
500 (226.85)	8.18×10^{-4}	7.26×10^{-2}
	(6.54×10^{-4})	(3.86×10^{-2})
1,000 (726.85)	43	3.38
	(34.4)	(1.80)
1,500 (1,226.85)	1,620	12.35
	(1,296)	(6.57)
2,000 (1,726.85)	9,946.25	23.88
	(7,957)	(12.70)

Oxygen, as a reactant in combustion processes, is present in the flame region well below its concentration in air. In addition, the products of the combustion [carbon dioxide (CO_2), water, etc.] dilute the nitrogen and remaining oxygen molecules. These factors cause the equilibrium concentrations of nitric oxide formed in a combustion at atmospheric pressure to be somewhat lower than those shown at each temperature in Table 2-2. Nitric oxide and nitrogen dioxide gases produced in a flame-maintained equilibrium would revert largely to nitrogen and oxygen gases when cooled to ambient atmospheric temperatures. However, most combustion equipment quickly channels the thermal energy in the product gases to perform such work as turning a turbine to generate electricity, moving a piston to ultimately propel an automobile, etc.

The chemical equilibrium between products is not maintained during the rapid cooling of the gases that occurs in these circumstances. The product nitric oxide and nitrogen dioxide gases are "frozen out" at concentrations typical of those present at equilibrium near the flame temperature. Nitrogen contributing to NO_x formed in combustion can come from the molecular nitrogen of the air or from the nitrogen-containing organic compounds in fossil fuels.

Nitrogen Dioxide

Nitrogen dioxide gas absorbs light over a wide range of the visible wavelengths (Figure 2-2) causing the characteristic light yellowish-orange to reddish brown colors of gaseous nitrogen dioxide seen at relatively low and high concentrations, respectively. Nitrogen dioxide has a very pungent odor, a high oxidation rate, and is extremely corrosive. It may be physiologically irritating and toxic (see Chapter 11).

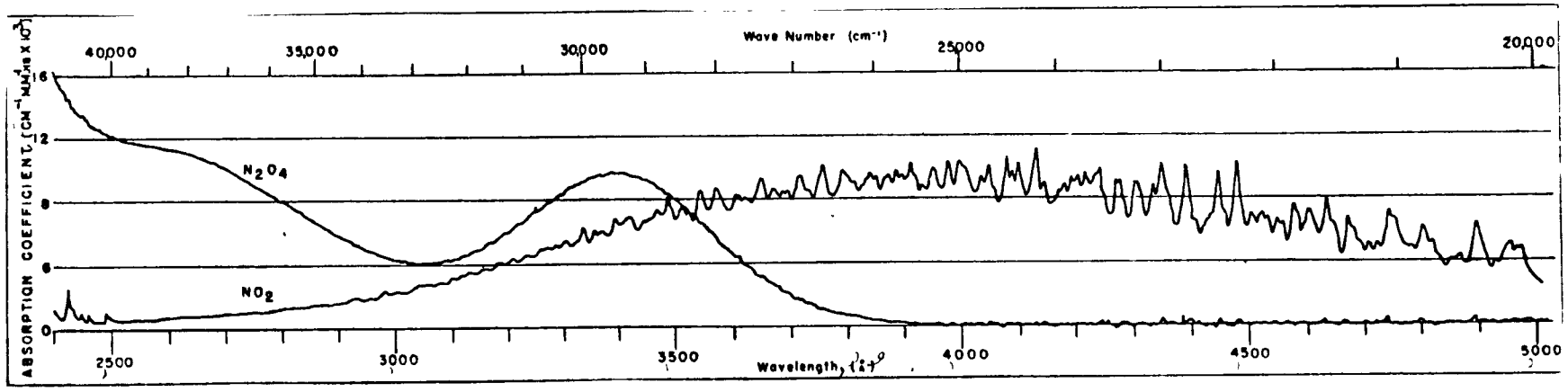


FIGURE 2-2. Absorption spectra of nitrogen dioxide and dinitrogen tetroxide at 25°C corrected to pure compound spectra; the form of the absorption law employed is $I = I_o 10^{-\alpha p l}$, where p is in mm of Hg at 25°C. Data from Hall and Blacet.[202]

Nitrogen dioxide may react with water to generate nitric acid ($HONO_2$) and nitrous acid:

$$2NO_2 + H_2O \rightleftarrows HONO_2 + HONO \tag{3}$$

and with nitric oxide and water to give nitrous acid:

$$NO_2 + NO + H_2O \rightleftarrows 2HONO \tag{4}$$

The homogeneous gas phase reactions of nitrogen dioxide and nitric oxide with water, forming nitric and nitrous acids, appear to be very slow at ambient temperatures. Therefore, the possible significance of Reactions (3) and (4) as NO_x removal paths remains uncertain.[314]

The major source of nitrogen dioxide in the atmosphere is the oxidation of the primary air pollutant, nitric oxide, through one of several pathways. During the first few minutes a nitric oxide-rich exhaust gas is mixed with air, oxidation may proceed significantly through the elementary Reaction (5):

$$2NO + O_2 \rightarrow 2NO_2 \tag{5}$$

The rate of nitrogen dioxide formation in Reaction (5) is given by the following rate law:

$$\frac{d[NO_2]}{dt} = [NO]^2[O_2]\ 2k_5 \tag{6}$$

The dependence of the rate on the square of the nitric oxide concentration results in a marked change in the rate with change in nitric oxide concentration. Thus, when the concentration of nitric oxide in exhaust gases is

1,250 mg/m^3 (1,000 ppm), the rate of nitrogen dioxide formation can be large; it maximizes at about 80.8 mg/m^3 (43 ppm) min^{-1} during the dilution caused by exhaust gases mixing with about 35% air by volume at 25°C.[467] When nitric oxide-rich exhaust gas is mixed with air, about 25% of the nitric oxide may be converted to nitrogen dioxide through Reaction (5). Once the nitric oxide concentration has reached its normal, low ambient level [less than 1.88 mg/m^3 (1 ppm)], Reaction (5) is very slow [less than (1.5 x 10^{-4} ppm) min^{-1}]. However, in a sunlight-irradiated, polluted atmosphere, nitric oxide may be oxidized to nitrogen dioxide rather quickly by mechanisms involving the hydrocarbons, aldehydes, carbon monoxide, and other compounds. The oxidizing agents in this case are believed to be the transient hydroperoxy (HO_2), alkylperoxy (RO_2), and acylperoxy ($RCOO_2$) free radicals.

$$NO + HO_2 \rightarrow NO_2 + HO \tag{6}$$

$$NO + RO_2 \rightarrow NO_2 + RO \tag{7}$$

$$NO + RCOO_2 \rightarrow NO_2 + RCO_2 \tag{8}$$

Here R represents methyl (CH_3), ethyl (C_2H_5), and higher alkyl groups. The hydroperoxy, alkylperoxy, and acylperoxy radicals are formed in chain reactions initiated by several active free radicals--i.e., hydroxy (HO), singlet D oxygen atoms [$O(^1D)$], ozone (O_3)--radicals interacting with the hydrocarbons, aldehydes, and carbon monoxide. (See the more detailed discussion in Chapter 6.)

Nitrogen dioxide photolysis in sunlight is believed to be largely responsible for the generation of ozone in the sunlight-irradiated, polluted atmosphere.[105,314,467] When a quantum of sunlight of wavelength less than

4,300 Å is absorbed by a nitrogen dioxide molecule, the excited molecule formed is of sufficient energy to dissociate into ground state oxygen atoms and nitric oxide:

$$NO_2 + h\nu \ (\lambda<4,300 \text{ Å}) \rightarrow O + NO \tag{9}$$

Oxygen atoms in air react predominantly with molecular oxygen to form ozone:

$$O + O_2 \ (+M) \rightarrow O_3 \ (+M) \tag{10}$$

The M represents a third molecular species (nitrogen, oxygen, water, etc.) that removes a fraction of the energy released during the interaction of the oxygen atom with the oxygen molecule. This energy removal stabilizes the ozone product. At the usual levels of common impurities present in urban air, ozone reacts most rapidly with nitric oxide to regenerate nitrogen dioxide:

$$O_3 + NO \rightarrow NO_2 + O_2 \tag{11}$$

The combination of the results of Reactions (9), (10), and (11) causes generation of a small concentration of ozone directly related to the ratio of the concentration of nitrogen dioxide to the concentration of nitric oxide and the intensity of the sunlight absorbed by the nitrogen dioxide. As the intermediates hydroperoxy, alkylperoxy, and acylperoxy radicals form during the complex interactions of the hydroxy radical, oxygen atom, ozone, etc., with hydrocarbons, aldehydes, and carbon monoxide, they react in part by Reactions (6), (7), and (8) and drive nitric oxide to nitrogen dioxide. This in turn builds the ozone concentration through the rapid

interplay of Reactions (9), (10), and (11). (See the more detailed discussion in Chapter 6.)

Nitrous Oxide

Nitrous oxide is a colorless gas with a slightly sweet taste and odor at high concentrations. It has been used for years as an anesthetic in medicine and dentistry. Inhalation of small quantities in air often produces a type of hysteria. This phenomenon is the source of its trivial name "laughing gas." Nitrous oxide in the atmosphere is believed to be created by decomposition of nitrogen compounds in the soil by anaerobic bacterial action.[470] The possible homogeneous formation of nitrous oxide in atmosperic reactions, e.g., $O(^1D) + N_2\ (+M) \rightarrow N_2O\ (+M)$, appears to be negligible. The mole fraction of nitrous oxide present in the atmosphere decreases markedly above the tropopause,[470] presumably as a result of photochemical dissociation of the nitrous oxide molecule.

$$N_2O + h\nu\ (\lambda<2{,}200\ \mathring{A}) \rightarrow O(^1D) + N_2 \qquad (12)$$

Singlet-D oxygen atoms are electronically excited oxygen atoms. Absorbed light of wavelengths less than 2,200 Å, present in solar radiation above the tropopause, can initiate Reaction (12).

The Unsymmetrical Nitrogen Trioxide

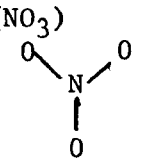
This oxide of nitrogen may be an intermediate in the kinetically third-order Reaction (5) which leads to oxidation of nitric oxide to nitrogen dioxide.

$$NO + O_2 \rightleftarrows O\text{-}O\text{-}N\text{-}O \qquad (13)$$

$$O\text{-}O\text{-}N\text{-}O + NO \rightarrow 2NO_2 \qquad (14)$$

The involvement of such an intermediate in equilibrium with oxygen and nitric oxide in a rate-determining step [Reaction (14)] would be consistent with the observed third order kinetics of the oxygen and nitric oxide observed in Reaction (5). No direct evidence supports the existence of the properties of this hypothetical transient species. Its significance as a participant in atmospheric reactions is unknown.

The Symmetrical Nitrogen Trioxide (NO_3)



Symmetrical nitrogen trioxide is a well established reactive transient. It is more important to air pollution than unsymmetrical nitrogen trioxide. Its well characterized absorption in the visible region leads to the blue color associated with this species at high concentrations. Much reliable laboratory evidence,[266] both kinetic and spectroscopic, identifies symmetrical nitrogen trioxide as an important transient in systems containing nitrogen dioxide-ozone, nitrogen dioxide-oxygen atom, and dinitrogen pentoxide. Symmetrical nitrogen trioxide can be formed as follows:

$$O_3 + NO_2 \rightarrow NO_3 + O_2 \tag{15}$$

$$O + NO_2\ (+M) \rightarrow NO_3\ (+M) \tag{16}$$

$$N_2O_5\ (+M) \rightarrow NO_3 + NO_2\ (+M) \tag{17}$$

Symmetrical nitrogen trioxide is highly reactive toward nitric oxide and nitrogen dioxide:

$$NO_3 + NO \rightarrow 2NO_2 \tag{18}$$

$$NO_3 + NO_2\ (+M) \rightarrow N_2O_5\ (+M) \tag{19}$$

It can be expected to attain only a very low concentration in the polluted atmosphere ($\sim 10^{-9}$ ppm). Its rate of reaction with acetaldehyde (CH_3CHO) and propene (C_3H_6) have been determined.[369] These reactions must occur in smog mixtures. The computer simulation of the rates of photochemical smog formation suggests that the hydrogen-atom abstraction reactions of symmetrical nitrogen trioxide such as:

$$NO_3 + CH_3CHO \rightarrow HONO_2 + CH_3CO \tag{20}$$

are probably much less important than the analogous reaction involving the hydroxyl radical.[105] However, the possible participation of symmetrical nitrogen trioxide in other types of reactions not now recognized (addition to olefinic hydrocarbons, etc.) remains open to question.

Dinitrogen Trioxide (or Nitrogen Sesquioxide)

The association of nitric oxide and nitrogen dioxide molecules to form dinitrogen trioxide and the dissociation of dinitrogen trioxide both occur rapidly establishing the equilibrium:

$$NO + NO_2 \rightleftarrows N_2O_3 \tag{21}$$

Dinitrogen trioxide is the anhydride of nitrous acid and reacts readily with water, at least in the condensed phase, to generate nitrous acid:

$$N_2O_3 + H_2O \rightarrow 2HONO \tag{22}$$

The equilibrium concentration of dinitrogen trioxide expected in the polluted atmosphere is very low compared with the common nitric oxide and

nitrogen dioxide concentrations encountered (Table 2-3). No important role for dinitrogen trioxide in atmospheric chemistry has been recognized to date.

Dinitrogen Tetroxide

In contrast to the brownish-red colored nitrogen dioxide gas, the dinitrogen tetroxide dimer of nitrogen dioxide is colorless. It absorbs only light outside the visible range (λ<4,000 Å) (Figure 2-1). Dinitrogen tetroxide, commonly called nitrogen tetroxide, is formed by the association of nitrogen dioxide molecules. It also readily dissociates to establish an equilibrium:

$$2NO_2 \rightleftarrows N_2O_4 \qquad (23)$$

The fraction of the nitrogen dioxide expected to associate at equilibrium is very small for the ambient concentrations and temperatures common to the urban atmosphere (Table 2-3). These low concentrations have never been observed directly, nor is there speculation suggesting that dinitrogen tetroxide offers any significant contribution to the chemical changes observed in the atmosphere.

Dinitrogen Pentoxide

The pure compound, dinitrogen pentoxide, commonly referred to as nitrogen pentoxide, occurs as a colorless gas at reduced pressures near 25°C. It is unstable toward decomposition [Reaction (17)]. In the atmosphere, it forms through the reaction of symmetrical nitrogen trioxide with nitrogen dioxide:

$$NO_3 + NO_2\ (+M) \rightarrow N_2O_5\ (+M) \qquad (19)$$

TABLE 2-3

Theoretical Concentrations of Dinitrogen Trioxide and Dinitrogen Tetroxide in Equilibrium with Various Levels of Gaseous Nitric Oxide and Nitrogen Dioxide in Air at 25°C

Concentration, ppm			
NO	NO_2	N_2O_3	N_2O_4
0.050	0.050	1.3×10^{-9}	1.7×10^{-8}
0.10	0.10	5.2×10^{-9}	6.8×10^{-8}
0.50	0.50	1.3×10^{-7}	1.7×10^{-6}
1.00	1.00	5.2×10^{-7}	6.8×10^{-6}

The dinitrogen pentoxide association product of nitrogen trioxide and nitrogen dioxide is more favored under atmospheric conditions than the dinitrogen tetroxide and dinitrogen trioxide association products of nitrogen dioxide-nitrogen dioxide and nitrogen dioxide-nitric oxide, respectively. Atmospheric simulations predict that about 10^{-4} ppm of dinitrogen pentoxide may accumulate in photochemical smog (Table 2-1).[470] Actual ambient levels of dinitrogen pentoxide have never been observed directly. Dinitrogen pentoxide is the anhydride of nitric acid:

$$H_2O + N_2O_5 \rightarrow 2HONO_2 \qquad (24)$$

The homogeneous rate of this reaction in the gas phase is uncertain.[368,470] The extent to which atmospheric dinitrogen pentoxide reacts with water to form nitric acid either homogeneously or heterogeneously, e.g., on a moist aerosol surface, has not been determined. The heterogeneous reaction pathway may be significant to nitric acid and nitrate salt formation in photochemical smog.

Some important physical properties of the major oxides of nitrogen are summarized in Table 2-4. Where no solubility constants are given, the species react with water. These reactions do not follow Henry's law which states that the weight of a gas dissolved by a liquid is proportional to the pressure of the gas. The actual amount of gas in solution at a given point in time is not only a function of the gas pressure and temperature, but also of the diffusion rate, the pH of the solution, and the interfacial surface area.

TABLE 2-4

Some Physical and Thermodynamic Properties of the Nitrogen Oxides

Oxide	Molecular weight, g/mol	Melting point, °C[a,b]	Boiling point, °C[a,b]	Solubility in H_2O(0°C), cm^3 (STP)/100 g[a]	Thermodynamic functions (Ideal gas, 1 atm, 25°C)[c] Enthalpy of Formation, kcal/mol	Entropy, cal/mol-deg
NO	30.01	-163.6	-151.7	7.34	21.58	50.347
NO_2	46.01	liquid, solid forms largely associated as N_2O_4		Reacts with H_2O forming $HONO_2$ and HONO	7.91	57.34
N_2O_4	92.02	- 11.3	21.2		2.17	72.72
N_2O	44.02	-102.4	- 89.5	130.52	19.61	52.55
N_2O_3	76.02	-102	3.5 (decomposes)	Reacts with H_2O forming HONO	19.80	73.91
N_2O_5	108.01	30	32.4 (decomposes)	Reacts with H_2O forming $HONO_2$	2.7	82.8

[a] Matheson Gas Data Book. 333

[b] Handbook of Chemistry and Physics. 591

[c] JANAF Thermochemical Tables. 553

EFFECTS ON ATMOSPHERIC LIGHT TRANSMISSION

Visibility reduction, common to urban air pollution, 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.[555]

Nitrogen dioxide absorbs light over the entire visible spectrum. This absorption is strongest at the shorter wavelengths--violet, blue, and green (Figure 2-2) reducing the brightness and contrast of distant objects and causing the horizon sky and white objects to appear pale yellow to reddish-brown. A token amount of light is attenuated by the molecular scattering effect of nitrogen dioxide.

Figure 2-3 shows the calculated percent of visible light transmitted as a function of wavelength for different atmospheric nitrogen dioxide concentrations and viewing distances in an aerosol-free atmosphere. Each curve is based on the concentration [mg/m^3 (ppm)]-distance (km) product; thus, 9.02 mg/m^3 (4.8 ppm)-km could be 1.88 mg/m^3 (1 ppm) at 4.8 km, or 9.02 mg/m^3 (4.8 ppm) at 1 km, or any combination of concentration and distance whose product is 9.02 mg/m^3 (4.8 ppm). A more normal picture of conditions actually existing in the atmosphere, with both nitrogen dioxide and aerosols present, is shown in Figure 2-4. These data are given in terms of calculated attenuation coefficients.[72] Here, the effect of visible light absorption is accompanied by transmitted light attenuation because of light scattering by the aerosol. The photochemical system may involve NO_x, hydrocarbons, sulfur dioxide, and other molecules in the formation of visibility-reducing aerosols.

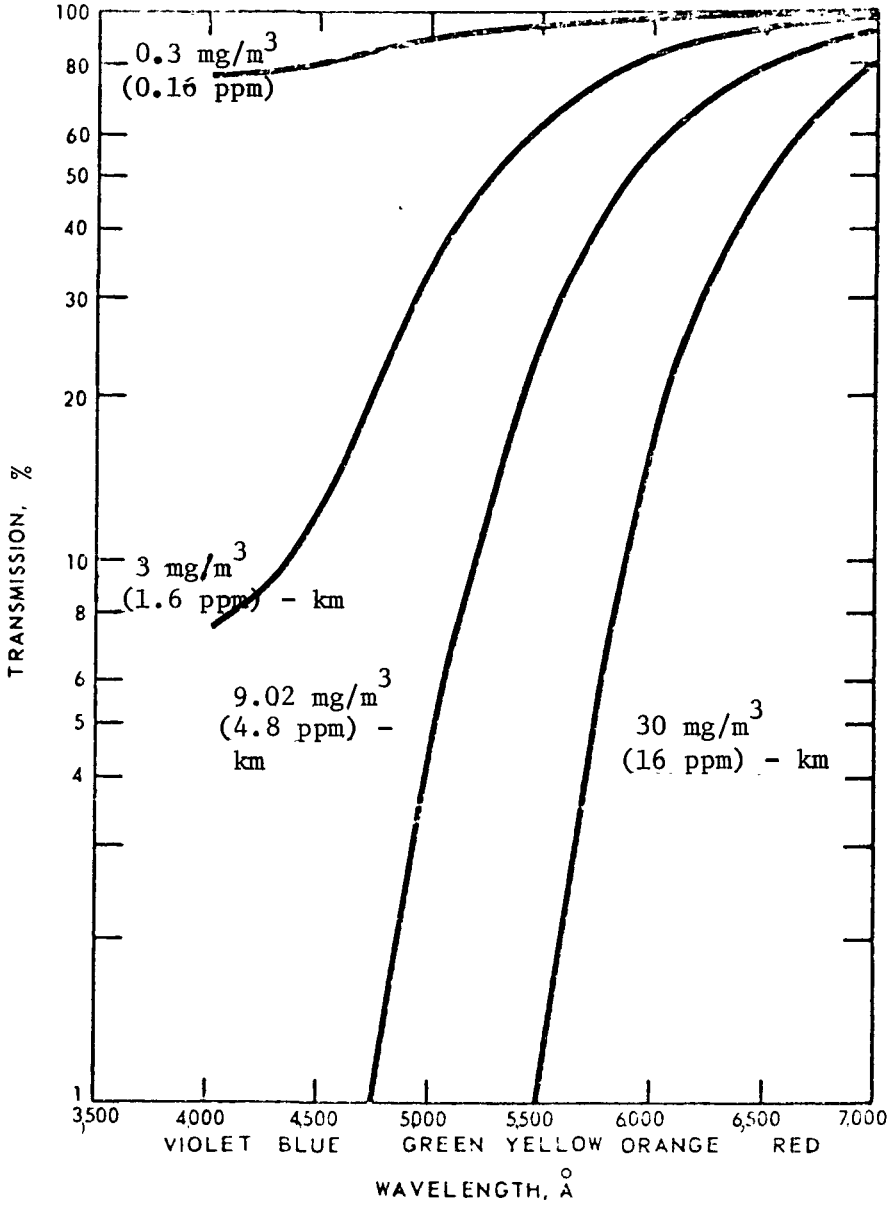


FIGURE 2-3. Transmittance of visible light at different NO_2 concentrations and viewing distances. Data from State of California.[72]

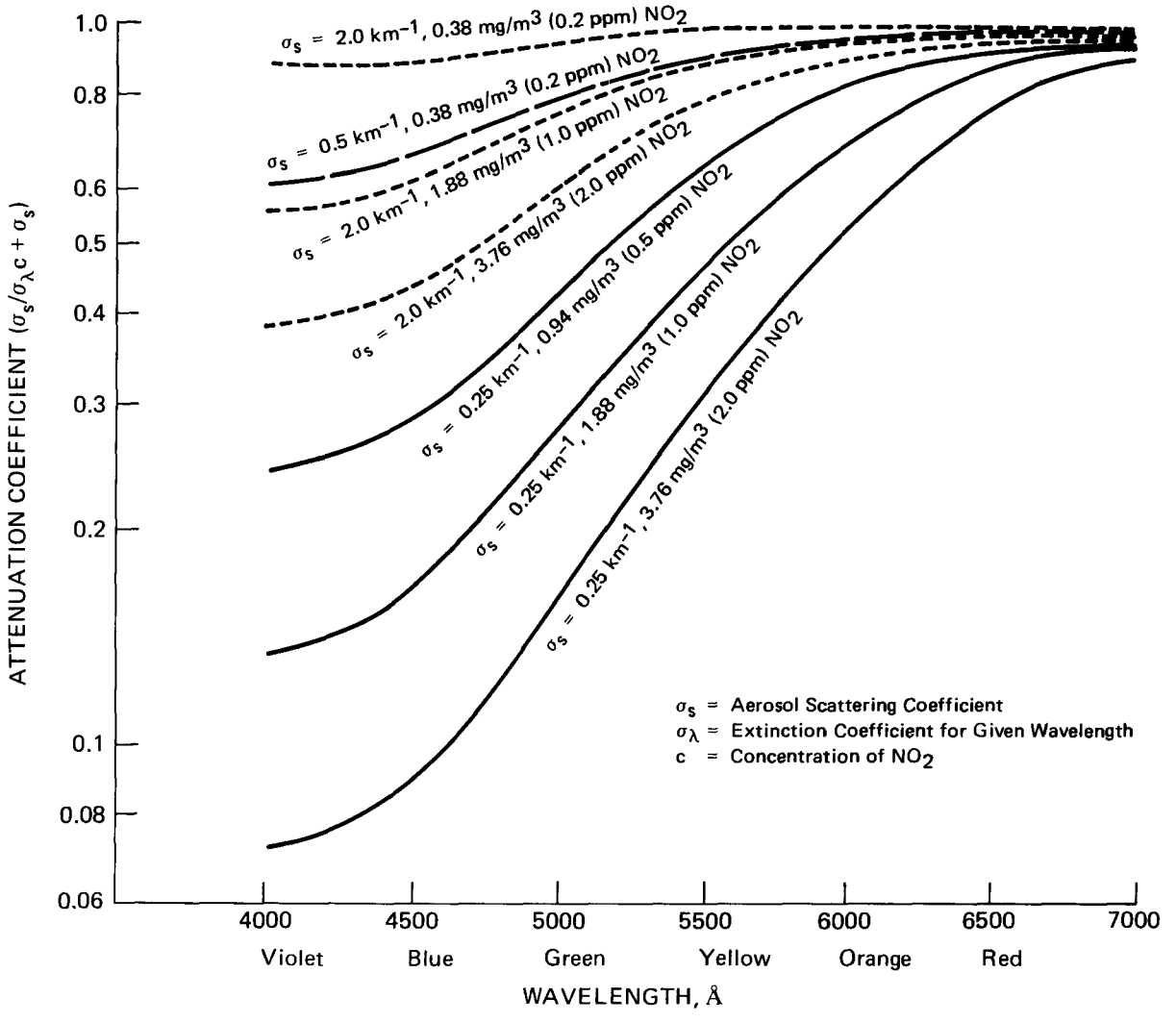


FIGURE 2-4 Aerosol attenuation in spectral luminance of horizon sky and distant objects at different NO_2 and aerosol concentrations. Data from State of California.[72]

The additional presence of particulate matter masks the coloration effect of nitrogen dioxide, but causes markedly reduced visibility, contrast, and brightness of distant objects. Particulate matter and aerosols in the atmosphere are primary contaminants originating from urban sources (i.e., industrial combustion and vehicular transportation), natural sources (i.e., the sea, soil, and fog), and through photochemical reactions. Light scattering associated with the presence of aerosols is thought to be the primary cause of visibility reduction in photochemical smog and absorption of light by nitrogen dioxide a minor contributor.[84]

SUMMARY

Among the various oxides of nitrogen present in the sunlight-irradiated, polluted atmospheres, nitric oxide and nitrogen dioxide, designated by the composite formula NO_x, play the most important role in chemical and photochemical changes. A major source of these oxides is the combustion of fossil fuels, which releases a predominance of nitric oxide. The rapid cooling of the gases in the combustion chamber prevents the return of nitric oxide to molecular nitrogen and oxygen to the extent required to establish equilibrium at ambient temperatures. A fraction of the nitric oxide is converted to nitrogen dioxide by reaction with oxygen during the exhaust dilution process; however, the major pathway leading to formation of nitrogen dioxide from nitric oxide is the photochemical interaction between NO_x, hydrocarbons, and various other compounds and intermediate free radicals generated in the sunlight-irradiated polluted atmosphere.

The extent to which nitrogen dioxide reduces visibility and colors the horizon sky depends on the concentration of the pollutant, the viewing distance, and the accompanying aerosol concentration. The presence of photochemical aerosol or other particulate matter suppresses the coloration effect of NO_x and increases the visibility-reduction effect.

CHAPTER 3

SOURCES AND CONTROL OF ATMOSPHERIC NITROGEN OXIDES

Manmade sources account for only a small fraction of the total global nitrogen oxide emissions (see Table 3-1); however, in localized urban atmospheres, these sources can be significant.

In this chapter both natural and manmade sources of nitrogen oxide are discussed, and the principles governing nitrogen oxide formation and control of manmade sources are reviewed.

NATURAL SOURCES OF NITROGEN OXIDES

The most abundant nitrogen oxide in the atmosphere, nitrous oxide (N_2O), has an estimated annual global emission of 54×10^{10} kg. It is not classified as a significant atmospheric pollutant since ambient levels are assumed to be harmless and since it does not appear to interact with the nitrogen dioxide (NO_2) photolytic cycle.

Bacterial action on nitrogen compounds under anaerobic conditions is thought to be the major natural source of nitric oxide. The nitric oxide formed in this way enters the photolytic cycle and eventually is converted to nitrogen dioxide and nitrate aerosols. The estimated annual global nitric oxide emission from biologic sources is 45×10^{10} kg. Uncontrolled combustion, such as forest fires, contributes an estimated $0.2\text{-}1.1 \times 10^{9}$ kg of nitrogen dioxide annually to nitrogen oxide emissions in the United States.[80,560] This represents 1 to 5% of the total nationwide emissions from combustion sources. Atmospheric nitric oxide formed by lightning is thought to be negligible.[442]

TABLE 3-1

Estimated Annual Global Emissions of Nitrogen Oxides[a]

Compound	Source	Estimated emission, 10^{10} kg/yr
NO_x	Manmade	4.8[b]
Nitric oxide	Biological action	45
Nitrous oxide	Biological action	54

[a]From Robinson and Robbins.[441]

[b]Expressed as nitrogen dioxide (NO_2).

Ammonia (NH_3) plays a significant role in the atmospheric nitrogen cycle and may be a precursor to formation of nitrate aerosols.[442] The principal natural source of ammonia emissions in the atmosphere is thought to be the bacterial breakdown of amino acids in organic material.[442]

MANMADE SOURCES OF NO_x

Combustion provides the major manmade source of NO_x emissions. Minor contributions are made by industrial processes and agricultural operations, which produce not only NO_x but also nitrates and ammonia, a potential source of nitrate aerosols.

An inventory of estimated nationwide NO_x emissions for 1970 is presented in Table 3-2. Although useful in summarizing available source data, these inventories can be misleading since the relative importance of a particular source depends on many factors, including land use, climate, and topography.

Manmade sources of NO_x emissions fall into two major categories--mobile and stationary. In 1970, an estimated 106 x 10^8 kg/yr of NO_x, or about 51% of the total NO_x emissions, were emitted by mobile sources. Combustion in stationary sources, including waste disposal, produced approximately 95 x 10^8 kg/yr of NO_x, or 46% of the total. Industrial processes and agricultural operations accounted for the remaining 5.6 x 10^8 kg of NO_x.

Mobile Combustion Sources

Motor vehicles are the major mobile source of NO_x emissions, accounting for approximately 40% of all manmade NO_x emissions in the United States. The importance of this source varies greatly with location. For example, motor vehicles contribute an estimated 90% of the annual NO_x emissions in

TABLE 3-2

Estimated Annual NO_x Emissions in the United States in 1970[a]

Source	Estimated emission, 10^8 kg/yr[b]			Total, (%)		
Mobile	106.1			51.4		
Motor vehicles		82.5			40.0	
Gasoline			70.7			34.3
Diesel			11.8			5.7
Aircraft		3.3			1.6	
Railroads		1.3			0.6	
Marine use		1.5			0.7	
Nonhighway use		17.5			8.5	
Stationary	90.9			44.1		
Electric utilities		42.7			20.7	
Industrial combustion		41.1			19.9	
Commercial		2.0			1.0	
Residential		5.1			2.5	
Solid waste disposal	3.6			1.7		
Agricultural burning	2.5			1.2		
Industrial process loss	1.8			0.9		
Miscellaneous	1.3			0.6		
Total	206.2					

[a] From Cavender *et al.* 80

[b] Expressed as nitrogen dioxide.

Sacramento, California; an estimated 56% in San Francisco;[71] and an estimated 8% in Northwest Indiana.[406]

In 1970, aircraft contributed an estimated 3.3×10^8 kg of NO_x annually, about 1.5% of the total manmade NO_x emissions. Although aircraft are not a major contributor nationally, they are the major NO_x source in the vicinity of many airports;[171] furthermore, at high altitudes they could become a major source of NO_x in the stratosphere.[269]

The relative importance of different stationary and mobile NO_x sources varies greatly from one location to another. Therefore, nationwide emission inventories have limited value in assessing local problems. The importance of various NO_x sources to a particular region should be defined by making a local source inventory in order to evaluate effectively the control strategies for that region.

NO_x emissions from most mobile sources in the United States have shown a continuing upward trend from 1940 - 1970 (Table 3-3). During this period, NO_x emissions from motor vehicles increased nearly 300%--from an estimated 27×10^8 kg/yr to 83×10^8 kg/yr. This increase is attributed to the additional numbers of motor vehicles in operation and their increased use.

Nationwide data (Table 3-3) do not necessarily reflect local trends. For example, in Los Angeles County, NO_x emissions from motor vehicles increased more than sixfold between 1940 and 1970.[324] Standards for NO_x emissions from motor vehicles, first adopted in 1971, have been updated several times since then. By 1980, if implementation of existing standards proceeds on schedule, NO_x emissions from motor vehicles should be reduced to 30 or 40% of the 1970 figures, or approximately 25 to 33×10^8 kg/yr.[386]

TABLE 3-3

Nationwide NO_x Emission Trends, 1940 - 1970[a]

(10^8 kg/yr)[b]

Source	1940		1950		1960		1970	
Mobile	29.2		47.2		72.5		106.1	
Motor vehicles		26.6		40.8		66.0		82.5
Aircraft		neg.[c]		0.1		0.1		3.3
Railroads		0.1		1.9		1.4		1.3
Marine use		0.9		1.1		0.7		1.5
Nonhighway use		1.6		3.3		4.3		17.5
Stationary	32.1		39.3		46.8		90.9	
Electric utilities		5.4		11.1		20.8		42.7
Industrial combustion		17.6		18.4		16.2		41.1
Commercial		0.5		1.0		1.8		2.0
Residential		8.6		8.8		8.0		5.1
Solid waste disposal	1.2		1.7		2.2		3.6	
Agricultural burning	1.8		2.0		2.4		2.5	
Industrial process losses	0.3		0.5		0.7		1.8	
Miscellaneous	6.8		3.6		2.3		1.3	
Total	71.4		94.3		126.9		206.2	

80

[a] From Cavender *et al.*

[b] Expressed as nitrogen dioxide.

[c] Negligible: $< 0.1 \times 10^8$ kg/yr.

Stationary Combustion Sources

Electric utilities are the most significant stationary source of atmospheric NO_x emissions in the United States, accounting for approximately 21% of manmade NO_x emissions. The amount of NO_x emitted by electricity generating plants varies with location and season. Electric utilities account for an estimated 9.5% of the annual NO_x emissions in Los Angeles County[324] and an estimated 26% in Northwest Indiana.[406] Seasonal emission variations of up to 60% have been reported,[71] reflecting fluctuations in the demand for electricity to meet changing heating and cooling requirements.

Industrial combustion provides an estimated 20% of annual U.S. NO_x emissions. Oil and gas pipelines, petroleum processing, and metallurgical refining are the principal sources of NO_x emissions in this category.

NO_x emissions from stationary sources continued their upward trend in the United States from 1940 - 1970 (Table 3-3). During this period, NO_x emissions from electric utilities increased nearly 800%--from an estimated 5.4×10^8 kg/yr to 43×10^8 kg/yr. This increase was attributed to greater electric power consumption. Again, nationwide trends do not necessarily reflect local trends. For example, in Los Angeles County, NO_x emissions from electric utilities decreased approximately 20% from 1960 to 1970 while consumption of electric power increased approximately 150%. This decrease resulted from the implementation of NO_x emission standards for stationary sources and a changeover to natural gas from fuel oil by the utility companies. During 1970 to 1980, U.S. consumption of electric power is expected to double and the availability of fuels, such as natural gas and low-nitrogen-content oil, to decrease. Since these fuels are used to reduce NO_x emissions, implementation of existing standards will become more

difficult. Because of these factors, a slowdown in the upward nationwide trend of NO_x emissions is unlikely and local downward trends, such as in Los Angeles County, will be difficult to maintain. The upward trend in NO_x emissions from industrial sources also is expected to continue during 1970 to 1980 due to increased industrial output and fuel problems similar to those affecting the electric utilities.

Noncombustion Sources

Industrial process loss refers to nitrogen oxide emissions from noncombustion industrial sources. Although nationally insignificant, these emissions can be important near metal processing plants using nitric acid and in the vicinity of facilities manufacturing chemical products such as nitric acid, explosives, and nitrate fertilizers. In 1970, an estimated 1×10^8 kg/yr of NO_x were emitted by nitric acid manufacturing facilities,[80] making them the most significant noncombustion NO_x emission source. Of secondary importance is the explosives industry which, in 1970, emitted an estimated 0.25×10^8 kg/yr of NO_x[80] during the manufacture of trinitrotoluene (TNT) and nitrocellulose.

As stated above, ammonia may play a role in the formation of nitrate aerosols in the atmosphere. Most ammonia produced from noncombustion sources is emitted during the manufacture of ammonia- and nitrogen-base fertilizers. On a local level, agricultural operations are another noncombustion source of nitrogen oxides; i.e., the nitrates in dust from feed lots is a significant local source of airborne nitrates.[621]

Limitations of Nitrogen Oxide Emission Inventories

The nitrogen oxide emission data discussed in the previous sections were obtained from the published emission inventories referenced above. These

inventories are useful summaries of available source data, but have the following limitations which should be considered during data interpretation:

- Since most emission inventories cover large geographic areas, they do not accurately reflect the distribution of sources on a local level.
- These inventories usually span an extended period. Therefore, the time-dependent nature of nitrogen oxide emissions from many sources is not reflected, e.g., the intermittent nature of the emissions from batch processes such as solid waste disposal, the diurnal variations of the emissions from commuter automobile traffic, and the seasonal variations of the emissions from stationary sources due to varying heating and cooling demands. During certain periods, the importance of these sources may be greater or less than indicated by a time-averaged emission inventory.
- Many emission inventories are estimates based on tabulated emission factors[574] and on a compilation of the number and size of various source types. Since published emission factors[201] may not be accurate for all cases, the inventories using these estimates may be in error.
- Emission inventories generally do not discriminate between nitric oxide and nitrogen dioxide, but report a combined NO_x emission. Such discrimination is desirable because each oxide interacts differently with the atmospheric photolytic cycle and because the ratio of nitric oxide to nitrogen dioxide can influence the selection of techniques for reducing the concentration of nitrogen oxides in the effluent gases.

PRINCIPLES OF NITROGEN OXIDE FORMATION AND CONTROL

Formation in Combustion Sources

Nitric oxide is the major oxide of nitrogen produced in the combustion process. Under certain conditions during combustion, significant amounts of nitrogen dioxide may also be produced;[462] but, for the most part, nitrogen dioxide in the effluent gases results from oxidation of nitric oxide after completion of combustion. The two principal sources of nitric oxide in the combustion of conventional fuels are oxidation of atmospheric (molecular) nitrogen and oxidation of nitrogen-containing compounds in the fuel (fuel nitrogen). In most combustion systems, the first process is the dominant source of nitric oxide. However, in combustion systems such as electric utility boilers which use distillate or crude oil, fuel nitrogen can be a significant source of nitric oxide. The nitrogen content of fossil fuels can vary considerably, depending on geographic origin of the fuel and on fuel processing techniques. Typical nitrogen contents of distillate oils, shale oil, and coal are given in Table 3-4.

The mechanism of nitric oxide formation from atmospheric nitrogen has been studied extensively. It is generally accepted that in combustion of fuel-air mixtures the principal reactions governing formation of nitric oxide from molecular nitrogen are:

$$O + N_2 \rightleftarrows NO + N \qquad (1)$$

$$N + O_2 \rightleftarrows NO + O \qquad (2)$$

Experimental studies[206,619] have shown that the nitric oxide formation is much slower than the combustion process so that most of the nitric oxide is formed after completion of combustion. The nitric oxide formation process

TABLE 3-4

Typical Nitrogen Content of Fossil Fuels[a]

Fuel	Average Nitrogen, wt%	Range, wt%
Distillate oil		
Crude	0.49	0.01 - 1.00
Asphaltenes	2.35	2.20 - 2.50
Heavy distillate	1.40	0.60 - 2.20
Light distillate	0.07	0 - 0.60
Crude shale oil	2.6	2.0 - 3.2
Coal	1.7	0.6 - 2.5

[a]From Ball and Rall,[28] Kirner,[285] and Dinneen *et al.*[116]

can therefore be decoupled from the combustion process. A simplified expression for the initial nitric oxide formation rate may be written[537]

$$dX_{NO}/dt = 1.5 \times 10^{17} T^{-\frac{1}{2}} X_{O_2}^{\frac{1}{2}} X_{N_2} \exp(-68{,}000/T) \quad \text{ppm NO/sec} \qquad (3)$$

where X_{O_2} and X_{N_2} are the equilibrium oxygen and nitrogen mole fractions at the temperature, T (°K), of the postcombustion gases. The strong dependence of the nitric oxide formation rate on temperature is evident from Equation (3). Elevated temperatures and high oxygen concentrations in the postcombustion gases result in relatively high nitric oxide formation rates. In certain situations, these rates exceed the rate predicted by Equation (3). Near the combustion zone, the concentration of oxygen atoms generally exceeds the equilibrium values in the postcombustion gases, resulting in an acceleration of the nitric oxide formation rate via Reaction (1). In this situation, observed nitric oxide formation rates exceed that given by Equation (3). Calculation of nitric oxide formation rates must consider the coupling of the nitric oxide formation reactions with the combustion process.[55]

In fuel-rich flames, nitric oxide formation rates significantly in excess of those predicted by Equation (3) have been observed.[141,258] While this may be due partially to excess oxygen atom concentration near the flame, Reactions (1) and (2) do not suffice as an explanation. Other reaction paths forming nitric oxide must therefore be postulated. For example, these paths may involve reactions between fuel fragments and molecular nitrogen to form nitrogen atoms, which in turn form nitric oxide [Reaction (2)]. Nitric oxide concentrations in the exhaust gases of most combustion devices are considerably higher than would be predicted from chemical equilibrium at exhaust gas temperatures, indicating that nitric oxide formed in the combustion process is removed only very slowly as the combustion gases are cooled. In most combustion devices, the perturbations of this nitric oxide formation process discussed

above are insignificant, and the rate nitric oxide forms from atmospheric nitrogen may be estimated using Equation (3).

Recent experiments involving nitric oxide emitted from stationary combustion devices[332,479] revealed that organic nitrogen compounds in fossil fuels (fuel nitrogen) were an important nitric oxide source. Although the combustion of many organic nitrogen compounds has been studied extensively, scant information on the mechanism of nitric oxide formation from these compounds has been obtained. Existing data indicate that oxidation of many organic compounds is rapid occuring on a time scale comparable to that of the combustion process. Conversion of fuel nitrogen to nitric oxide can occur at temperatures much lower than those required to oxidize atmospheric nitrogen to nitric oxide. In lean mixtures, measured nitric oxide concentrations in the postcombustion gases indicate a nearly complete conversion of the organic nitrogen compound to nitric oxide. Although the mechanism by which fuel nitrogen converts to nitric oxide is uncertain, several empirical correlations are available for estimating nitric oxide yields.[142,148]

Control Methods for Combustion Sources

There are two basic approaches to control NO_x emissions from mobile and stationary combustion sources. One is to modify the combustion process either through changes in operating conditions, the fuel, or the design of the device to decrease nitric oxide formation. A second approach is chemical or physical removal of nitrogen oxides from the effluent gas.

Mobile Sources. NO_x emissions from mobile sources result almost exclusively from oxidation of atmospheric nitrogen during combustion. Control

methods either prevent the formation of nitric oxide in the combustion chamber or remove it from the effluent gas. Since elevated temperatures in the presence of oxygen favor nitric oxide formation [Equation (3)], modifications to reduce this formation rate generally involve reductions of peak temperatures and oxygen concentrations in the combustion chamber.

NO_x emissions from conventional spark ignition internal combustion engines have been effectively decreased by spark retardation, lean combustion, low-compression ratio, water injection, and exhaust gas recirculation. However, these modifications may reduce performance and increase carbon monoxide and hydrocarbon emissions. Fuels such as alcohol, hydrogen, or fuel blends may also reduce NO_x emissions from conventional engines. They not only can provide lower combustion temperatures but also may permit operation at lean mixtures, both factors contributing to decreased nitric oxide formation in the engine. Stratified-charge engines, diesel engines, gas turbines, and Wankel rotary engines are alternative power sources capable of reducing NO_x emissions from mobile sources.

Several catalytic methods are used to remove nitric oxide from the effluent gases from mobile sources. A major disadvantage of these methods is deterioration of the catalyst material. NO_x reductions obtained by these control techniques may be limited by considerations of fuel economy.

Comprehensive discussions and evaluations of the various techniques for controlling NO_x emissions from mobile sources are presented in a 1973 report by the Committee on Motor Vehicle Emissions, National Academy of Sciences,[386] and a 1970 publication of the National Air Pollution Control Administration.[558]

Stationary Sources. Oxidation of atmospheric nitrogen accounts for most of the NO_x emitted from stationary sources. In those sources using crude oil or coal, fuel nitrogen can also oxidize producing significant amounts of nitric oxide. In selecting methods to reduce nitric oxide formation in the combustion chamber, the nitric oxide source should be considered since mechanisms leading to oxidation of atmospheric nitrogen and fuel nitrogen differ.

Operating conditions in stationary sources have been modified to reduce NO_x emissions through the introduction of low-excess-air firing, staged combustion, over-fire air, flue gas recirculation, and water injection. Variations in burner design, i.e., burner repositioning and tangential, rather than horizontal, firing have also reduced NO_x emissions. Premixing of fuel and air combined with off-stoichiometric combustion has reduced NO_x emissions from gas-fired combustion devices. All of these techniques cause peak temperature reduction in the combustion chamber, thereby decreasing the rate at which nitric oxide is formed from atmospheric nitrogen.

Temperature reduction does not significantly affect the rate fuel nitrogen oxidizes to form nitric oxide; however, the burning of a mixture which is fuel rich in the primary combustion, can result in reduced nitric oxide emissions. The two control methods with the potential to reduce NO_x emissions from fuel nitrogen are therefore low-excess-air firing and staged combustion.

A change in fuel type may also result in reduced NO_x emissions. For example, substitution of natural gas for fuel oil, reduction of the organic nitrogen content of coal or oil, fuel processing, vaporization of liquid fuels and gasification of coal, and catalytic processing of fuels for

selective removal of organic nitrogen compounds are all potential NO_x control techniques.

Combustion flue gas treatment processes (aqueous scrubbing, selective catalytic reduction or decomposition of NO_x, various adsorption processes, and afterburning) also reduce NO_x emissions from stationary sources.[33,387,559]

Control Methods for Noncombustion Sources

Control of nitrogen oxide emissions from industrial process losses generally involves removal of the emissions from the effluent gas. Since the concentration of these oxides and the ratio of nitrogen dioxide to nitric oxide can differ markedly from composition of flue gas from combustion sources, the treatment methods for each effluent are also different. The reader is referred to a 1970 publication of the National Air Pollution Control Administration entitled "Control Techniques for Nitrogen Oxide Emissions from Stationary Sources" which contains a comprehensive discussion of these various treatments.[559]

SUMMARY AND RECOMMENDATIONS

Annual global emissions of nitrogen oxides from manmade sources are substantially less than those from natural sources; however, manmade sources play a very significant role in atmospheric pollution in localized areas. The principal manmade source of nitrogen oxides is combustion. Although industrial process losses contribute only a small amount to the total manmade nitrogen oxide emissions, they can be important locally.

Existing methods for obtaining emission inventories have limited application in local evaluations of NO_x sources since the methods generally use data from large geographic areas covering extended periods. Further,

inaccuracies result from differences between actual NO_x emissions from specific sources and tabulated emission factors. Source inventories should discriminate between nitric oxide and nitrogen dioxide emissions since each interacts with the atmospheric photolytic cycle in different ways.

Nitrogen oxides are produced in combustion of conventional fuels through oxidation of both atmospheric (molecular) nitrogen and nitrogen compounds in the fuel (fuel nitrogen) via different mechanisms. Nitric oxide is formed from atmospheric nitrogen in the high-temperature regions in the combustion chamber; fuel nitrogen oxidizes at lower temperatures and can be a major source of nitrogen oxide emissions in some combustion devices.

Two basic approaches to control NO_x emissions from combustion sources are modification of the combustion process and exhaust gas treatment. Present understanding of the principles of nitric oxide formation in combustion is sufficient for development of techniques to reduce these emissions. However, implementation of these techniques may be hampered by excessive cost, various operational problems, and loss in combustion efficiency.

Treatment of effluent gas is the principal technique for reducing nitrogen oxide emissions from noncombustion sources.

CHAPTER 4

ANALYTICAL METHODOLOGY FOR THE DETERMINATION OF NITROGEN OXIDES IN AIR

The 1967 Amendments to the Clean Air Act required that: "From time to time, but as soon as practicable, [the Administrator of EPA] shall develop and issue to the States such criteria of air quality, ... [that] reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on health...."[552] This statement was responsible for the publication of several reviews of methodologies for measuring the oxides of nitrogen.[134,561,563] Unfortunately, since most epidemiological studies do not provide enough information to compare older and newer methods, assessment of one method over another is difficult, if not impossible.

Chemical methods for measuring pollutants have been used for almost 100 years. One example is the Griess-Ilsovay Method in which nitrous acid (HNO_2) is reacted with an aromatic amine to form diazonium salts. These salts are then coupled with an organic substance to produce an azo-dye.

These chemical methods can be divided into two general categories: sampling and analysis. Since these categories are interchangeable in many methods described in the literature, countless variations in effective methods are possible. Chemical methods can be further classified as direct and indirect. In direct chemical methods a single solution is used to sample the gas and measure the produced color. In indirect

chemical methods, a separate sampling solution is required and the color is developed later by addition of certain chemicals.

Physical methods have been developed recently, especially as applied to ambient air measurements. Both chemical and physical methods are discussed below.

MANUAL METHODS FOR CHEMICAL ANALYSIS OF NITROGEN DIOXIDE

Direct Methods

Absorbing Solutions. An early (1939) chemical method applied to atmospheric levels[186] required that the contaminant be sampled in an impinger containing a reagent mixture composed of 0.17% sulfanilic acid ($H_2NC_6H_4SO_3H$) and 0.033% 1-naphthylamine ($C_{10}H_7NH_2$) in 14% acetic acid (CH_3COOH). This resembles the method proposed originally by Ilosvay (quoted by Treadwell and Hall in 1935).[542] To avoid the handling of the highly carcinogenic and volatile naphthylamines,[67] other coupling substances were subsequently substituted. For instance, in 1939 Bratton and Marshall[57] proposed the use of 1-(N-1-naphthyl) ethylenediamine dihydrochloride ($C_{10}H_7NHCH_2CH_2NH_2 \cdot 2HCl$) which is nonvolatile and readily soluble in water. With minor modifications in the concentrations of the components, this mixture is the same as the Griess-Saltzman reagent[451] previously used extensively in automatic colorimetric analyzers and designated as a Tentative Method by the Intersociety Committee on Methods for Ambient Air Sampling and Analysis,[251] and a Standard Method by the American Society for Testing and Materials.[18] A later modification of the same reagent proved more economical and faster in response.[452] To eliminate the corrosive acetic acid, several formulations appeared with less corrosive organic acids[296,297,375] or with isopropyl

alcohol [$(CH_3)_2CHOH$].[611] However, the presence of acetic acid seems to inhibit secondary reactions, leading to higher sensitivity.

Studies initiated in 1965 by the Air and Industrial Hygiene Laboratories, California Department of Public Health, showed that possibilities for formulating similar reagents are seemingly endless, provided the fundamental components are present: diazotizer, coupler, buffer, and a surfactant.[297,298,299,374,375] The reagent inducing the shortest response time in a microcell is formulated as follows:[296] 0.12% 2-amino-p-benzenedisulfonic acid [$C_6H_3NH_2(SO_3H)_2$] or sulfanilamide ($H_2NC_6H_4SO_2NH_2$); 0.18% sulfuric acid (H_2SO_4) 0.025% 1-(N-1-naphthyl)-4-(acetyl) ethylenediamine-p-toluene sulfonate [$C_{10}H_7NHCH_2CH_2NH_2(OCCH_3)O_3SC_6H_4CH_3$]. This formulation reduces the response time 10 to 20 times faster than earlier formulations. A similar speed could be obtained with 2,5-dichlorosulfanilic acid ($C_6H_2Cl_2NH_2SO_3H$) and 1-amino-7-naphthalenesulfonic acid ($C_{10}H_6NH_2SO_3H$). During sampling, some of the incoming nitrogen dioxide (NO_2) is converted to nitric oxide (NO). Nitric oxide in the mixture affects the overall recovery, otherwise it is not an interferent. Aging effects limited earlier attempts to sample dilute ambient levels of nitrogen dioxide for an extended time and to store the exposed reagent. Some interfering gases, such as sulfur dioxide (SO_2), produce noticeable bleaching effects during storage, which make the direct absorbing azo dye reagents useless. To prevent this, the addition of acetone (CH_3COCH_3)[451] has been suggested. Controversial effects[62,519] of this addition have not been reevaluated.

Several simple methods are available to measure higher concentrations of nitrogen oxides. With the syringe technique,[392] a gas sample is drawn into a syringe containing oxygen and Griess-Saltzman reagent. Shaking

promotes oxidation of nitric oxide to nitrogen dioxide, absorption, and color development. The phenoldisulfonic acid [$C_6H_3OH(SO_3H)_2$] method[254] is also suitable for these higher concentrations. Possibly most of the other methods described for nitrate on page 19 of this chapter can also be used after adequate aliquoting of the absorbing solutions.

Stoichiometric Factor. In the determination of gaseous nitrogen dioxide with azo-dye reagents, the nitrogen in nitrogen dioxide reacts to produce a colored specimen. This conversion is not quantitative. The factor introduced to express the conversion efficiency of the reaction is called the stoichiometric factor.

Under strict sets of chemical and physical conditions, the stoichiometric factor for each method considered remains constant and can be established by a previous calibration with known concentrations of nitrogen dioxide.

Methods using azo-dye reagents can also be calibrated with secondary calibration standards, such as with nitrite solutions, once the stoichiometric factor is known. Secondary calibration standards are preferred in routine work over the more cumbersome gas calibration techniques.

The stoichiometry depends on the geometry of the sampling device, the chemical characteristics of the components in the reagent mixture, the presence of impurities, the temperature during sampling, light exposure during or after sampling, and aging.[298,451,452]

Authors who suggested that the stoichiometric factor differs greatly from the established value of 0.72 either used doubtful diluting devices,[62,205,208,395,519] differed markedly in their experimental designs to prove

new theories,[247] or sampled higher concentrations.[155,392] A carefully designed experiment using preconditioned permeation tubes[461] produced a factor of 0.764, which is close to values found working under standardized procedures[451,452,455,478] at atmospheric concentrations of nitrogen dioxide. The Air and Industrial Hygiene Laboratory, of the California Department of Health, evaluated the factor in 1969 by generating a 28 $\mu g/m^3$ (0.15 ppm) nitrogen dioxide stream using the asbestos plug dilutor.[455] The nitrogen dioxide was then absorbed into alkaline-potassium permanganate ($KMnO_4$)[294] and the absolute amount of nitrate generated was determined by UV-spectrophotometry[29,34,81] after separation of the manganese dioxide (MnO_2). The extensive study produced the factor 0.735 which nearly coincided with the values reported earlier. Experimentally weak designs lead to equivocal results, such as the variable factor[291] which was analyzed and explained later.[478]

Methodology for Establishing Nitrogen Dioxide Concentrations. The principal function of a chemical method is to determine concentrations. To establish values with precision and accuracy, the method must withstand certain tests. It must be sufficiently rugged to give valid results under differing ambient conditions. Most azo-dye reagents stand up well under tests of temperature, variations in chemical concentrations, and long-term storage, but they are sensitive to strong lights, especially at low nitrogen dioxide concentrations. For calibration purposes, light shielding can be arranged so that azo-dye formulations can effectively standardize pure gas streams containing nitrogen dioxide generated by diluting techniques.[252,405,444,455] This standardization process is facilitated with aqueous solutions of nitrite.[251,451] Thus, a dilute gas

stream can be fed into a manifold from which different sampling devices and instruments can simultaneously draw samples of gas. One port of the manifold is used for analyzing the gas with a reference method for nitric oxide or nitrogen dioxide.[250,251]

Indirect Methods

Absorbing Solutions. The use of alkaline absorbing solutions has been described by Jacobs and Hochheiser:[259]

To avoid the bleaching effect of sulfur dioxide also present in atmospheric samples when using azo-dye reagents directly, the pollutant gases are first sampled into 0.1 N sodium hydroxide (NaOH) solution containing a surfactant such as butanol which improves the gas transfer efficiency. Dispersion is facilitated by use of a fritted sparger. Sulfite formed during the absorption of sulfur dioxide into the sodium hydroxide solution is oxidized to sulfate by the addition of hydrogen peroxide (H_2O_2) which does not affect the nitrite formed by reaction of nitrogen dioxide with the alkaline solution. The nitrite is stable for 48 hr, which allows sufficient time for transporting the aqueous sample to a centrally located laboratory. The nitrite in solution can be analyzed by addition of azo-dye forming chemicals and acidifying the solution with phosphoric acid (H_3PO_4).

Stoichiometric Factor. The term "stoichiometric factor" used in conjunction with alkaline absorbers has the same significance as in direct single-solution azo-dye forming reagents. However, to avoid confusion, the product of sampling efficiency and stoichiometric factor was used in

some of the reported investigations and called empirical conversion factor.[427,566] This is clearly not a precise use of terms.

The sampling efficiency can be determined by sampling a pure stream of a dilute mixture of a known nitrogen dioxide concentration or with a train composed of several identical collecting devices. With the first scheme, only an empirical conversion factor can be calculated by simple azo-dye colorimetry. To establish percentage recovery of sampling, the nitrate formed in solution must also be determined. With the second alternative, carryover of nitrogen dioxide to the subsequent bubblers can be established, and sampling efficiency can be calculated by assuming that no secondary reactions (e.g., reduction to nitric oxide) has taken place during collection in the first bubbler. Since losses through secondary reactions are unlikely when alkaline absorbing solutions are used, a close approximation can be expected.

The sampling efficiency obtained in controlled, clean laboratory conditions may be greater than can be achieved in the field.[229] Sampling efficiency is also affected by such variables as: flowrate,[566] porosity of frits,[364,365,566] liquid level,[364,365] container material,[294] incoming pollutant concentration,[164,412,485,486,527,569] and contaminants present at sampling location.[364,365] Because of these variables, the term "alkaline absorber" will denote a specific combination of absorber and absorbing solution.

The stoichiometric factor in alkaline absorbers is variable. One of the first methods, which was developed in 1958, was originally designed for intermittent 40 min sampling.[259] In 1965 this was modified[238] to a batch-type 24-hr operation. This was adopted with further modifications

by the National Air Sampling Network and in 1971 was promulgated as a reference method in the Federal Register.[566] Originally, the absorption efficiency was found to be better than 90% and a stoichiometric factor of 52% to 65% was assumed.[410] During operation of the Network the factor varied from 0.45 to 0.85. In 1966 the average for 11 locations was 0.62 ± 0.07.[364,365]

A detailed study by Huygen and Steerman[246] revealed that the factor varies with incoming nitrogen dioxide concentration, decreasing in the presence of sulfur dioxide and increasing in the presence of nitric oxide.[229,300] In 1972, Purdue et al.[427] postulated that the stoichiometric factor may not deviate greatly from unity and that the product with the sampling efficiency is equal to the so-called empirical conversion factor used in the reference method described above.[566] This postulation does not stand up well when compared to other investigations using similar reagents.[87,246,385] Variations encountered during sampling with alkaline solutions were affected by the presence of even minute amounts of hydrogen-donors which greatly increased the solubility of nitrogen dioxide.[87,246, 256,318,385] Many air contaminants may be hydrogen-donors. This provides one explanation for the variability of the stoichiometric factor with location.[364,365]

Data collected on continuous recording instruments for ambient levels of nitrogen dioxide were compared with data collected with alkaline absorbers. Results were quite different for field as compared to laboratory conditions.[349,526] (Kinosian 1971, personal communication.) This difference may result from the delay in response to nitrogen dioxide variations found in continuous analyzers which prevents the recording of the entire area integrated by calculation. The calculation may be affected by a negative error of up to

20%. The integration of all the errors resulting from different variables which affect conversion factors and absorption efficiencies in unmodified alkaline absorbers are unpredictable and deviate by a factor of 2 or greater. To reduce the sampling error caused by a single alkaline absorber, a method using two absorbers in series was adopted in 1970[486] for an epidemiological study. Results from different methods using alkaline absorbers are compared in Table 4-1.

Nitrite Reagents. After collection of the nitrogen dioxide in the alkaline absorber, the solution is acidified and the nitrite formed during collection is measured after reaction with an azo-dye forming mixture. Methods to determine nitrites in aqueous solutions are countless. In the classicial book Organic Analytical Reagents, written in 1948, Welcher[594] lists over 100 possibilities which, when added to those published in the last 25 years, show that methods to determine nitrite are not restricted to those few proposed in those papers (loc. cit.) for use with alkaline absorbers. The amount of nitrite in the absorbing solution is established by reference to a calibration curve prepared from nitrite standards.

Jacobs-Hochheiser Modifications. A method used to establish an air quality standard should be scientifically and practically tested before adoption. Unfortunately, many established methods have not been subjected to such exhaustive tests. Pressures of time have resulted in the use of methods which have not even been tested against all possible naturally occurring variables. Because some methods satisfactorily produced a great mass of information during an initial test phase, their optimal performance has been often taken for granted.

TABLE 4-1

Alkaline Absorber Methods

Absorber	Reagent	Variable	Sampling Efficiency	Conversion Factor	Interference
Fritted glass	0.1 N NaOH	Flowrate	±5%[566*] variation	100%	NO, SO_2
	0.2% Butanol	Porosity	30 to 70%[364,365*]	100%	
			up to 90%[259*]	57%[410]	
Fritted plastic	0.1 N NaOH	Liq. level 7.6 cm	40 to 60%[364,365*]	100%[427]	NO, SO_2
		Liq. level 5.1 cm	30 to 53% (id)	100% (id)	
Fritted plastic	0.1 N NaOH	Concentration			
		NO_2 0.02 ppm (37 μg/m^3)	50 to 75%[569*]	100%	NO, SO_2
		NO_2 0.05 ppm (94 μg/m^3)	40% (id)	100%	NO, SO_2
		NO_2 0.1 ppm (188 μg/m^3)	22 to 30% (id)	100%	NO, SO_2
		NO_2 0.2 ppm (376 μg/m^3)	15 to 25% (id)	100%	NO, SO_2

TABLE 4-1 - continued

Absorber	Reagent	Variable	Sampling Efficiency	Conversion Factor	Interference
Fritted plastic (2 tubes)	0.1 N NaOH	NO_2 0.06/0.1 ppm (112/188 μg/m^3)	70 to 78%[485*]	62±7%[364]	NO, SO_2
		NO_2 0.5/1.0 ppm (940/1880 μg/m^3)	27 to 37% (id)		
Fritted glass	0.1 N NaOH	NO_2 0.05 ppm (94 μg/m^3)	65%[47*]	100%[47*]	NO, SO_2
		NO_2 0.10 ppm (188 μg/m^3)	56% (id)	100% (id)	NO, SO_2
		NO_2 0.20 ppm (376 μg/m^3)	38% (id)	100% (id)	NO, SO_2
Fritted glass (2 tubes)	0.1 N NaOH	NO_2 0.5/0.2 ppm (940/376 μg/m^3)	74%[47*]	100%[47*]	NO, SO_2
Fritted plastic	0.1 N tri-ethanolamine [$(CH_2CH_2OH)_3$] + butanol (C_4H_9OH)	NO_2 0.03/0.5 (56/940 μg/m^3)	95 to 99%[256,318*]	90% (mean 76 to 100%)[256,318*]	No interference

TABLE 4-1 - continued

Absorber	Reagent	Variable	Sampling Efficiency	Conversion Factor	Interference
Impinger (glass)	1% NaOH	NO_2 2-5 ppm (3,760-9,400 $\mu g/m^3$)	95%[87*]	100%[87*]	NO, SO_2
	0.1% Na_3AsO_3				
	0.75% sulfanilic acid				
Impinger (plastic)	4% NaOH	NO_2 0.003/0.4 (5.6/754 $\mu g/m^3$)	85%[571*]	100%[571*]	NO, SO_2
	0.1% Na_3AsO_3				
Arnold tubes	0.1 N NaOH	?	90 to 100%[385*]	73%[385*]	NO, SO_2
	0.05% guaiacol ($CH_3OC_6H_4OH$)				
Fritted glass	0.1 N NaOH	NO_2 0.026 ppm (49 $\mu g/m^3$)	97%[246*]	93%[246*]	NO, SO_2
	0.02% R-salt [$C_{10}H_5OH(SO_3H)_2$]	NO_2 0.19 ppm (357 $\mu g/m^3$)	(id)	96% (id)	NO, SO_2

TABLE 4-1 - continued

Absorber	Reagent	Variable	Sampling Efficiency	Conversion Factor	Interference
Fritted glass	0.1% triethanolamine $[(HOCH_2CH_2)_3N]$	NO_2 0.6 ppm (1,130 $\mu g/m^3$)	(id)	87% (id)	NO, SO_2
		NO_2 1.17 ppm (2,200 $\mu g/m^3$)	(id)	80% (id)	NO, SO_2

*These superior numbers are references.

NOTE: A 100% factor is an assumed value. In such cases the recorded sampling efficiency value is the product of the true sampling efficiency multiplied by the true conversion factor.

One such case is the Jacobs-Hochheiser Method[259] for collecting 40 min samples in a fritted glass collector using a 0.1 N sodium hydroxide solution with some butanol added to improve the gas-liquid transfer. The sampling efficiency of the first bubbler was calculated by analyzing the total nitrogen dioxide collected in two absorbers in series. The stoichiometric factor was taken from a previous similar work.[410] Later, Hochheiser and Ludmann modified the method to a 24-hr batch sampler.[238] Confusion arose because the findings of one method were applied to another. Although there were only minor differences, equivalency was assumed without thorough experimental confirmation.

During development of the Federal National Air Sampling Network (NASN), the Hochheiser[238] Method was adapted to the entirely different NASN gas samplers.[364,365] Absorbing efficiency (60-70%) was checked by four orifice bubblers in series and stoichiometry was arbitrarily assumed to be 50%. Meadows and Stalker[349] omitted the butanol in the absorbing solution and placed six orifice bubblers in series. They determined the sampling efficiency to be poor and highly variable with flow rate. Fritted tips were not used because they tend to clog and are difficult to clean.

Nonuniformity of pore sizes also increased the variability coefficient. The method described in the Federal Register[566] (glass frits and plastic container) and the method used in the Chattanooga study[212,486] (two bubblers in series) are entirely different methods that have departed from Jacobs-Hochheiser in their design and performance.[47,212,427] The only similarity retained is the final measurement of the nitrite content in the absorbing solution.

As a result of the Air Quality Act of 1967 (Public Law 88-206), and a request by the City of Chattanooga, Tennessee, an air quality survey was initiated on October 1, 1967, covering an interstate area extending from Chattanooga to Rossville, Georgia, and over parts of Hamilton, Walker, and Catoosa counties in Tennessee.[219] This area was selected because its manufacturing plants produce a large amount of NO_x. Data collected by the continuous colorimetric Saltzman monitoring method from October 1967 to November 1968 were used to establish a frequency distribution of NO_x concentration.[219]

From November 1968 to April 1969, the National Air Pollution Control Administration (NAPCA) and the U.S. Army made an epidemiological study covering the same geographical area.[485,486,563] They adopted a modification of the alkaline absorber method first used by the National Air Sampling Network,[364,365,412,427] and later used in the first Chattanooga study. Since the method used in the first study was subject to question at extreme concentrations, comparison of the modification with its prototype[47] was not practicable.[229] Approximate comparisons can be attempted, however, by combining statistical values of recovery information from two alkaline bubblers in series using permeation tubes[47,485,486] with the frequency distribution of the atmospheric concentration obtained with monitoring instruments during the first two studies[561] and from April 1972 to December 1973[100] in the same area of Tennessee. Data resulting from this comparison could provide a basis on which the collected concentration values for nitrogen dioxide can be saved, confirmed, and adjusted. The true values will probably be not more than 20% lower than the concentration reported in the original study.

Although these corrections are not extreme, they suggest the need to replace data obtained with the modified Jacobs-Hochheiser two-tube collecting method.

MANUAL METHODS FOR CHEMICAL ANALYSIS OF NITRIC OXIDE

Direct Methods

There are only a few manual methods for the direct measurement of nitric oxide in gaseous mixtures. Ferrous sulfate ($FeSO_4$) can be used to determine nitric oxide at concentrations >30 ppm (56.4 mg/m^3).[295,400,401] Direct instrumental measurement in the UV[112,113,525] and IR[30,31,112,113,413] can be made only at high concentrations or by using long optical paths such as those used when monitoring from aircraft. Mass spectrometry[390] and gas chromatography are batch instrumental methods.

Indirect Methods

Indirect methods to determine nitric oxide concentrations involve selective oxidation of nitric oxide to nitrogen dioxide. Separation of coexisting nitrogen dioxide in the mixture before oxidation of the nitric oxide is necessary if both gases are to be determined separately. If not separated, the result would be an unresolvable mixture (NO_x).

Selective Absorbers for Nitrogen Dioxide. The first attempt to separate nitrogen dioxide at low levels involved the passing of effluent gas through an azo-dye forming reagent contained in a fritted glass bubbler.[373] In this method, however, some of the nitrogen dioxide converts to nitric oxide, thereby increasing the nitric oxide concentration.

The conversion percentage of nitrogen dioxide to nitric oxide varies with the chemicals used and their concentration in the particular azo-dye reagent formulation.[247] Generation of 7 to 16% nitric oxide can occur with the Griess-Saltzman reagent.

Widely used absorbers for nitrogen dioxide in the late 1960's were ascarite (asbestos-supported sodium hydroxide) and soda lime. Although this worked well with dilute air, or nitrogen streams of either nitric oxide or nitrogen dioxide alone, their absorption efficiency varied with mixtures of the two gases.[319] During the absorbing process of nitrogen dioxide, some nitric oxide is lost as with absorbers using dilute alkali.

Most recently, a tube containing granules impregnated with triethanolamine[250,319] has been used for nitrogen dioxide absorption. In this process, only 2 to 4% of the incoming nitrogen dioxide is converted to nitric oxide.

Oxidizers for Converting Nitric Oxide to Nitrogen Dioxide. The oldest oxidizers for the conversion of nitric oxide to nitrogen dioxide used either manganese[539] or chromium compounds[270,437,601] at their highest valence state. Nitric oxide can be oxidized in the gas phase by slowly introducing a small excess over the stoichiometric amount of ozone (O_3)[454,564] or chlorine dioxide (ClO_2).[535] Other oxidizers include manganese dioxide[209] and iodine pentoxide (I_2O_5).[241] A chromic oxide (CrO_3) oxidizer, supported on inert granules made by soaking firebrick in a chromic oxide solution and drying in an oven [in absence of any other inorganic acid (H_3PO_4; H_2SO_4; etc.)], has been shown to be insensitive to small humidity changes and gives practically quantitative (>99%) conversion of nitric oxide to nitrogen dioxide between 20 and 80% RH.[250,316,317]

ANALYSIS OF NITRATE

Oxidizers for Total Oxidation of Nitrogen Oxides to Nitrate

Methods which measure nitrate after conversion of higher than ambient concentrations of nitrogen oxides, such as in source analysis, are both cumbersome and time-consuming. They require large batch sampling and the difficult procedure by which gas phase nitrogen oxides are quantitatively transferred to an aqueous solution; however, the analysis of the formed nitrate poses no particular problems.

To ensure complete conversion of nitrogen oxides to nitrate, the method using phenoldisulfonic acid[254] needs a long reaction time which permits slow thermal oxidation of nitric oxide to nitrogen dioxide. A strong oxidizing absorbing solution, such as hydrogen peroxide in dilute sulfuric acid,[17,254] aids in the absorption of nitrogen dioxide. However, dilute sulfuric acid is not the ideal absorbent since nitrogen dioxide has a lower solubility in dilute acid than it does in distilled water.[492] Nitrogen dioxide dissolves most easily in alkaline solutions containing any one of many hydrogen donors,[87,246,256,318,385] alkalies, and strong oxidizers such as hydrogen peroxide,[293,302] or alkalies and permanganate.[283,294] By this latter methodology, the nitric oxide in a gas mixture can be oxidized as well as absorbed. A potassium carbonate-(K_2CO_3) impregnated paper can be used for dry absorption of nitrogen dioxide.[166]

Analysis of Nitrate from Particulate Matter

Although most of the nitration methods described below were originally designed to analyze nitrate in natural waters, they have also been used to analyze nitrate in aqueous extracts of particulate matter[226] and in solutions obtained through absorption of streams of nitrogen oxides in air.[283,294]

The oldest nitration procedures use brucine ($C_{23}H_{26}N_2O_4$)[255,263,327,443] and phenoldisulfonic acid.[17,36,124,254,528] Newer procedures extensively used to analyze nitrate in atmospheric particulate matter extracts involve the nitration of xylenols [$(CH_3)_2C_6H_3OH$] and separation of the nitro-derivative by extraction or distillation.[20,63,210,242,249,524,609] Nitration of chromotropic acid [$C_{10}H_4(OH)_2(SO_3H)_2$][597] and coumarin ($C_9H_6O_2$) analogs[306,493] have also been reported. The advantage of coumarin analogs over other phenolic compounds is their easier nitration rate at lower concentrations of sulfuric acid (Kothny, unpublished). Small amounts of nitrate can be determined by the quenching of the flourescence after nitration of fluorescein ($C_{20}H_{12}O_5$).[26] Finally, nitrate analysis can be accomplished through reduction with Devarda alloy to ammonia (NH_3),[282,435] or reduction of nitrate to nitrite with the help of zinc (Zn),[86] cadmium (Cd),[367,521,606] or hydrazine (NH_2NH_2).[380] Automation instituted by the NASN has improved the hydrazine reduction process by curtailing the unwanted effects resulting from its sensitivity to motion.[366] The addition of antimony sulfate [$Sb_2(SO_4)_3$] eliminates the chloride interference found in most nitration methods.[597] The brucine procedure circumvents the effect of chlorides by adding an excess of sodium chloride before nitration.[255]

Nitrate analysis by ion-selective electrodes has several disadvantages: potential drifts caused by agitation speed, necessity of frequent restandardization, interferences caused by nonspecificity of the electrodes which respond to other ions in the aqueous extracts, and nonstoichiometric absorption of the gases in the collecting reagent.[111,121,182,188] In atmospheric analysis, the electrode has no advantage over direct UV determination of either nitrite formed in an alkaline absorbent[15] (with the inherent weakness of all alkaline absorbers) or nitrate[81] obtained after

oxidation and absorption of nitrogen oxides in alkaline permanganate[283] and separation of the manganese with hydrogen peroxide.[294] Microscopic techniques also allow analysis of individual nitrate particles.[44]

PRIMARY REFERENCES FOR NITROGEN DIOXIDE AND NITRIC OXIDE

In 1973 Hauser *et al.*[211] defined the only purpose of a reference method as the provision of the best measurement of a pollutant in an environment. Questions of cost, field worthiness, and convenience should be of secondary importance.

There are two classifications of reference methods--primary and secondary. In primary reference methods, accurate data can be produced through repetition of chemical or physical operations and calibration can be done by weighing a standard substance. A secondary reference method cannot give reliable data without a concurrent standardization or comparison with a primary reference method.

Since a primary reference method may only be used to calibrate other methods or instruments by monitoring a single diluted pollutant gas, it need not be absolutely free of interferences, as long as it gives highly accurate information. Conversely, a secondary reference method used for intermittent or continuous sampling of real gas mixtures should be free of interferences in order to provide specific information with reasonable accuracy and precision over an extended time.

The advent of permeation tubes has elevated many secondary methods to the primary reference category.

Reference Methods

The Griess-Saltzman Method for determining nitrogen dioxide is a primary method which has been thoroughly tested and used for many decades.[18,38,251] In 1973 Stevens *et al.*[515] criticized the variability of its response to mixtures containing simultaneously-introduced ozone, as compared to results obtained from sequential introduction of ozone used in the initial testing of the method. Despite criticisms of the instability of its conversion factor,[291] the manual Griess-Saltzman Method is one of the most specific and reproducible primary methods available. If it is accurately followed[251,451] the factor is constant and interferences do not affect its use in standardizing calibrating gases. At present, this is the only method collaboratively tested and endorsed by the American Society for Testing and Materials.[152]

Standard Gas Sources

Primary standard gas sources were developed to obviate the need for a primary reference method to test approximate or unknown gas mixtures or the purity of standard gases in cylinders.[193] The first attempt to develop such a source involved the careful dilution of pure or concentrated mixtures of nitrogen dioxide and dinitrogen tetroxide (N_2O_4) by inert gases or air.[62,205,208,252,291,395,444,451,455,519]

A diluting device with turning stopcocks with a precisely known volume of the bore enabled calculation of precise mixing ratios.[62,519] With this device, several European scientists discovered independently that the ratio of gaseous nitrogen dioxide to azo-dye formed after absorption was unity. Shortly after, a departure from unity to lower values was reported for higher levels of nitrogen dioxide.[205,395]

Through volatilization of liquid nitrogen dioxide into a high velocity air stream, Thomas and Amtower[534] generated known concentrations of nitrogen dioxide by adjusting the weight loss rate and measuring the air volume.

In 1965 Saltzman and Wartburg[455] developed a procedure consisting of a one step dilution of a 0.4% nitrogen dioxide mixture with purified air by an asbestos plug with a precisely known leakage rate. This concentrated mixture could be analyzed with a gravimetric procedure by absorption into ascarite (asbestos + NaOH) and anhydrone [anhydrous magnesium perchlorate, $Mg(ClO_4)_2$].

The development and study of permeation tubes, correctly used and carefully calibrated, enabled creation of dilute mixtures.[405,461,485,486] The prior usage of the tubes (e.g., exposure to atmospheric humidity) may, however, affect rate and stability of permeation.

Dilute nitric oxide streams can be generated by permeation of compressed nitric oxide through membranes. Calibration is still made with carefully prepared gas mixtures,[444] and dilution techniques.[250] In 1963, Hersch and Deuringer[227] proposed that electrolytic generation of nitric oxide is a precise source of this gas; but this has not yet been exploited.

Precise concentrations of nitric oxide for primary calibrating purposes may also be generated by nitrogen dioxide permeation tubes followed by a highly efficient catalyst (i.e., pyrolyzed sugar in glass wool) that converts nitrogen dioxide to nitric oxide.[58] Photolysis of known concentrations of nitrogen dioxide in pure nitrogen gas and rapid dilution by purified air is another source of low concentrations of nitric oxide.[197]

SECONDARY REFERENCES FOR NITROGEN DIOXIDE AND NITRIC OXIDE

National Bureau of Standards Publication 351[12] stresses that the criteria for air monitoring instrumentation should be simplicity, reproducibility, and low cost.

It also emphasizes that a new instrument or method may require 2 to 4 years to progress from the drawing board to actual implementation. We cannot, therefore, be overly optimistic about adopting any new development in a short time because of the necessary exhaustive testing, cost analyses (of calibration, maintenance, data reduction, salaries, etc.), and comparison with other techniques such as manual methods.

A constantly upgraded list of commercially available instruments is published by the University of California.[134] It records manufacturers' parameters, a glossary, and a summary of available calibration procedures, methods, and policies. An evaluation of procedures for determining performance of air monitors, routine calibration, and their limitations was published in 1974 by Mueller and his associates in A Guide for the Evaluation of Atmospheric Analyzers.[378]

Traditional Monitoring Instrumentation

Continuous instrumentation based on the Griess-Ilosvay Reaction for nitric oxide after oxidation by permanganate,[243] and instruments using the similar Griess-Saltzman reagent are still in operation. These instruments require constant calibration and replenishment of reagent. Some minor changes have included refinements in reagent formulation,[296,297,298,299,374,375,451,452,611] operational modes,[539,563] and oxidizer design.[209,250,270,316,317,437,535,539,601]

When permanganate is used as an oxidizer, it tends to dry out and accumulate crystals. A dilute sulfuric acid solution[492] can minimize these problems. Substitution of permanganate solutions by solid chromic oxide supported on firebrick, creates problems in regulation of incoming relative humidity of the sampled atmosphere. Sodium acetate ($NaC_2H_3O_2 \cdot 3H_2O + NaC_2H_3O_2$) humidifier used for regulating the RH in the manual procedure[250] cannot be applied in continuous instrumentation; therefore, use of an aqueous humidifier, followed by heating of the chromic oxide oxidizer to 10 to 15°C above ambient temperature, controls the range of optimal performance.[316,317] Instruments using this principle of measurement differ in design of flow systems and contact columns and range from large stationary installations to smaller semiportable units.[418,533] Not all contact columns absorb gases efficiently, and the conversion factor in liquid azo-dye continuous colorimetric instruments varies from 0.5 to 1.0 depending on the design and construction material of these columns.[484,533,535] Error-free static calibration of these instruments is impossible; therefore, dynamic calibration with known gas mixtures is preferable.

An Intersociety Committee report details procedures for the calibration of continuous colorimetric monitoring instruments for nitrogen oxides using liquid azo-dye forming reagents.[252] A summary of the required performance criteria with definitions was published in the Federal Register.[565] Mueller et al.[377] considered interferences in the colorimetric method to be negligible at atmospheric concentrations,[377] although many parameters and operational conditions of real atmospheric sampling have not been thoroughly evaluated.

Chemiluminescent Instrumentation

A reproducible reaction for nitric oxide is the chemiluminescent oxidation with ozone.[150] This reaction is almost specific for nitric oxide and can be applied to air pollution measurement.[515,516] Its principal drawbacks, like those of the other continuous analyzers, are the needs for frequent calibration with complex instrumentation[99] and electromechanical maintenance. To adapt this reaction for nitrogen dioxide analysis, a highly effective thermostat-equipped converter for nitrogen dioxide to nitric oxide must be incorporated into the sampling line.[58,240] This converter must be checked frequently to ensure its continued efficiency.[453,515,516]

Different materials have been evaluated as working catalysts. Ammonia and other nitrogen-containing compounds may be oxidized in certain converters,[572] but other converters discriminate against such compounds. Many substances produce chemiluminescent reactions with ozone, such as ethylene (CH_2CH_2), triethylamine [$(C_2H_5)_3N$], carbonyl compounds (R_2CO), mercaptans (RSH), and other sulfur compounds.[303,389,421,506] For separation of these interferences, optical filters can be used.[515,516] Nitrogen dioxide is electronically estimated from the difference between the signals obtained with and without passing air through the converter. Error is possible through the batch sampling and the consequent uneven intake. Breakdown of valves and pumps is possible.[515,516] Other deficiencies of this method are chronicled in the Federal Register.[570]

The chemiluminescent titrator described in the Federal Register[564] is an outgrowth of a technique described by Fontijn *et al.* in 1970.[150] In this technique the fast gas phase reaction between nitric oxide and ozone produces stoichiometric conversion of the nitric oxide to nitrogen dioxide.[267, 268,454]

The method has been used to measure ozone indirectly through reaction with nitric oxide[454] and may be used to calibrate chemiluminescent instruments.[99] However, it is complicated and its instrumentation is delicate.

To assume that calibration of nitric oxide in cylinders serves as a secondary calibration standard is highly idealistic. Oxygen traces in the nitrogen used for dilutions generate nitrogen dioxide over a span of several days. Once stabilized, a slow reaction occurs over several weeks or months, especially when using common steel cylinders, causing disappearance of nitrogen dioxide (Air and Industrial Hygiene Laboratory, Department of Health, State of California, unpublished). Newly prepared nitric oxide cylinders may therefore contain both oxides of nitrogen.

In a further evaluation of this problem, Decker *et al.*[99] suggest bi-monthly controls. Chemiluminiscent instruments need one or two primary methods for calibration such as neutral buffered potassium iodide (KI) for ozone[253] or the manual Griess-Saltzman[251] for nitrogen dioxide. Since many calibrating steps result in a deterioration in overall precision, a more direct approach is desirable; for example, calibration of chemiluminescent instruments with dilute nitric oxide as indicated under Standard Gas Sources. Since 1975, the Bureau of Standards has offered stable, certified mixtures of nitric oxide in nitrogen for this purpose.

A chemiluminescent method for both nitrogen dioxide and nitric oxide analysis at atmospheric levels is the reaction with atomic oxygen obtained by thermal decomposition of ozone.[45,351] The resulting signal has to be corrected for nitric oxide in order to obtain nitrogen dioxide concentration. A more specific, but indirect method, is photolysis of nitrogen dioxide with UV light after removal of atmospheric ozone. During this photolysis an equivalent amount of ozone is produced which is measured by a chemiluminescent reaction.[197,340]

Batch Methods

Batch methods are used for grab, or manual, sampling on a predetermined time basis, e.g., a few minutes, 2 hr, 24 hr, etc. Instrumental methods in which gas is analyzed intermittently, can also be considered as batch methods. In principle, all instrumental methods can be adapted to intermittent data by electronically-operated integrators. Most of these methods require constant standardization.

Volumetric wet chemical analysis of nitric oxide is performed in nitric acid plants.[123] Other wet chemical methods were explained above.

High concentrations of nitrogen oxides are suitable to several instrumental procedures after thermal oxidation of nitric oxide to nitrogen dioxide. The sampling and preparation of the gas before measurement has been audited for the following methods, e.g., Nondispersive Ultraviolet (NDUV),[112,113] Nondispersive Infrared (NDIR),[112,113,413] phenoldisulfonic acid (PDS),[112,113] and spectrophotometry.[112,113,393]

Gas chromatographic methods can be used at higher than atmospheric concentrations only. Nitrogen dioxide and nitric oxide are collected separately on cold traps containing the polyethylene glycol, "Carbowax 1500," and porous polystyrene, "Poropak Q," respectively. A thermal conductivity cell acts as a detector[106] after separating the gases on Poropak Q[49,106] contained in a stainless steel column.

Heated iodine pentoxide oxidizes nitric and nitrous oxide (N_2O) to nitrogen dioxide.[106,241] For low nitrous oxide concentrations, gas chromatography is the best, if not only, available method,[59,106,576] but, as previously stated, this oxide is not an important air pollutant. Nitric oxide can be separated on molecular sieve 5A[284] or silica gel[59] in the

absence of oxygen, and detected at very low levels (0.01 μl) with the aid of an argon ionization cell.[284] For low levels of nitrogen dioxide, no satisfactory gas chromatographic method has been found;[59] the lower limit is 5 ± 2 ppm (9,400 ± 3,800 $\mu g/m^3$) using an electron-capture detector.[371]

Miscellaneous Methods

To determine health hazards in industrial situations, indicator tubes containing substances sensitive to nitrogen dioxide are commonly used.[312,313,362,363] They are accurate within 20 to 30%. Continuous batch type analyses have also been made with the Auto Analyzer.[205] The determination of nitrogen oxides with surface-specific electrochemical sensors[120,125,515] is limited since it does not reach the lower atmospheric levels.

A field method for analysis of atmospheric concentrations of nitrogen dioxide employs a visual color comparator.[239] This is useful for levels of nitrogen dioxide above 0.05 ppm (94 $\mu g/m^3$) whereas common indicator tubes are sensitive for levels of 5 ppm (9,400 $\mu g/m^3$) or more. This method is accurate within 15 to 20%.

Electrochemical methods are based on galvanic cells[10] such as the Mast Nitrogen Dioxide Analyzer and the Hersch Cell. Water interference in nitrogen dioxide analysis could be eliminated if the gases could be dried without loss of nitrogen oxides. These two methods are in good agreement with other methods (loc. cit.) when tested with combustion gases,[203] but are unsuitable for ambient levels. Instrumental methods using fluorescent excitation of nitrogen oxides are under development.[549] Other miscellaneous instruments and principles of lesser importance have been described by the Lawrence Instrumentation Group in Berkeley.[134]

SOURCE MONITORS FOR NITROGEN OXIDES

The two main sources of manmade oxides of nitrogen are combustion of fossil fuels and the internal combustion engine. Instrument systems designed to monitor stack gas emissions of nitrogen oxides have been studied intensively by the Environmental Protection Agency and others.[348,497] The handling of the hot, wet, corrosive, and often dirty sample, and its introduction to the analyzer in a condition permitting correlation between the analytical result and the original sample composition are most difficult. Additionally, the sample must be representative of all the gases passing out of the stack, and the total flow of gases must be measured, or otherwise determined to calculate the total quantity of nitrogen oxides being discharged to the atmosphere. This applies to both instrumental and manual wet chemical methods.

Two analytical approaches are currently in use. Conventionally, a gas sample is withdrawn from the source via a high temperature probe. It is then filtered, and possibly chilled, or dried by some other means, depending on the requirement of the analyzer. The other approach uses an *in situ* monitoring instrument, measuring absorption of infrared,[413] visible,[393] or ultraviolet[112,113] radiation traversing through the gas. Since the *in situ* instruments require that optical components not be too heavily loaded with particulates, means to compensate for fouling must be implemented.

Nitrogen dioxide absorbs radiation in the visible and UV spectral regions, and can be quantitatively determined using these wavelengths. Nitric oxide absorbs weakly in the ultraviolet region; in the infrared, its absorption ability increases. Since this absorption is overlapped by the

spectrum of water vapor, correction or compensation for large percentages of water, typically present in combustion gases, is necessary.

Nitric oxide may be determined indirectly by oxidation to nitrogen dioxide, followed by UV determination. Since the concentration of nitrogen dioxide in combustion gases is typically less than 5% of total NO_x, infrared determination of nitric oxide is frequently acceptable. The chemiluminescent analyzer can determine both nitric oxide and nitrogen dioxide, operating on either a wet or dried sample. An electrochemical cell, appropriately sensitized, can give quantitative measurement of NO_x.[120,125] Galvanic cells such as the Hersch cell, have been used for monitoring nitrogen dioxide.[203]

Nitric oxide and nitrogen dioxide in internal combustion engine emissions are measured almost exclusively by the chemiluminescent analyzer.[112,113] Several hundreds of these instruments are in operation. The chemiluminescent analyzer is preferred because of its specificity, speed of response, wide dynamic range, and relative simplicity. Analysis may be made on raw exhaust, where concentrations of nitrogen dioxide, carbon monoxide (CO), molecular oxygen (O_2), and unburned hydrocarbons are correlated directly with the mode of engine operation. In some tests, diluent air is added to the engine emission from which a sample is collected over a certain period in a plastic bag for later analysis. This dilution technique, which reduces the concentration of nitrogen oxides to a few ppm, retards the thermal oxidation of nitric oxide to nitrogen dioxide to a few percent before analysis.

Optical instruments based on correlation spectroscopy have been mounted on aircraft for surveying emissions on a regional scale.[30,31,600]

Miscellaneous Considerations in the Choice of Methods

In choosing the method to be used, one must consider the cost and bulkiness of each method on a sliding scale. Regarding field methods, for instance, distinction must be made between a mobile laboratory and a portable, suitcase-sized unit.

Equivalency testing of methods would be best done in a well-equipped laboratory having a programmable manifold from which mixtures of varied concentration and composition of gases could be tapped.

Altshuller[12] and Stevens and Hodgeson[516] noted that target criteria for air monitoring instrumentation should be simplicity, reproducibility, and low cost. Other goals are accuracy within certain limits in the presence of interferences, ease of calibration, and simple maintenance. Few methods in use meet all these criteria.

The most ambiguous goal is "low cost" since the relativity of that term defies definition. Regional surveys with the alkaline plate method would provide the lowest cost empirical method available;[166,587] however, this method is of unknown accuracy, reproducibility, and sensitivity to interferents. There is no calibration necessary. The plates are simple to make, analyze, install, and maintain. The estimated datum cost per unit per month, including personnel and overhead, is about $15.00, compared with $20.00 for a single determination or calibration of a gas mixture in a laboratory using the Griess-Saltzman manual method. On the other hand, a 24-hr method would cost $400 a month and a continuous monitor about $600 per channel. The information provided from these three methods is quite different. The higher expense is justifiable in an area with a progressive worsening of the air quality discovered in surveys of large regions by empirical methods such as the alkaline plate.

The need would then be defined for each area of use. Regionally, a tested method giving a monthly average is still needed, or an existing method such as the alkaline plate should be calibrated side by side with a monitoring station, to provide a better understanding of the mechanism and insight into data interpretation.

Secondly, several 24-hr average nitrogen dioxide concentration methods are collaboratively tested. Four methods, tested side by side, all indicated good precision (<±4%). The two methods using sodium hydroxide and arsenite demonstrated lower sampling efficiency and more interference from nitric oxide than the two methods using triethanolamine [$(HOCH_2CH_2)_3N$] as an alkalyzer.[211] Methods selected from this and future evaluations should always be used with strict adherence to details to avoid cases similar to those in which the term "Jacobs-Hochheiser" was erroneously applied.

A precise method for establishing primary nitrogen dioxide concentrations is the Griess-Saltzman Method,[251] to which Griess gave the principle and Saltzman optimized the conditions. This method must be used without departure from the original design. The most important components of this method are the bubbler (with a specified porosity glass frit) and the reagent formulation. There is no need to improve this calibrating method if a standard factor can be universally agreed upon. For short-term field use, however, an improvement in the light stability of the reagent formulation is desirable.

Quantitation of nitrate in particulate matter can be achieved by several methods. The results of each method are uncertain since the efficiency of all the different steps, such as sampling, aqueous extraction,

nitration, extraction or distillation of the products, and colorimetric determination, is unknown. Filter preparation to reduce blanks is very important. Better and more uniform filter material is needed. A good study could determine the efficiency of the first two steps, whereas an intercomparison of methods for extracts from real samples would determine which is the most precise, the most accurate, or both. Automation of such analyses would eliminate the high cost of handling and processing; miniaturization would permit the study of short-term ambient variation on a continuous basis.

Instrumental methods appeared rapidly after the development of chemiluminescent reactions. Many initial inconveniences and instrumental variations were eliminated or reduced as a result of a strong advance in electronics. The instruments are less subject to flow rate variations and do not need reagents to operate. However, in chemiluminescent methods applied to nitrogen dioxide, reliability is in demand. Inconveniences are the batch sampling procedure for nitric oxide and NO_x with a further subtraction of the signals for obtaining the nitrogen dioxide concentration. The reducing catalyst for nitrogen dioxide has an unknown lifetime in real situations and the instruments need frequent calibration with calibrating gases or permeation tubes, which are delicate and need working thermostats with supporting gases. A better approach might be photofragmentation by UV irradiation to ozone and chemiluminescent measurement of that ozone.[197,340] It is not known if this principle worked trouble-free in other situations besides those studied initially. This method can be used as a continuous monitoring method for nitrogen dioxide up to 1,880 $\mu g/m^3$ (1,000 ppb).

Inconveniences cited for continuous colorimetric azo-dye forming instruments can be summarized as follows: variability of the flowrates of intake air and reagent, long response time, negative ozone interference at high ratios of ozone to nitrogen dioxide, and reagent replenishment. Ozone interference on the continuous colorimetric instruments occurs only occasionally in areas of high pollution.

The azo-dye method has given us the first accurate information about nitrogen oxides and should not be disregarded despite its weaknesses. Many instruments having weaknesses continue to be used and have provided the majority of valuable information. These older instruments could be improved by replacing the inefficient liquid permanganate oxidizer for nitric oxide with the efficient chromic acid (CrO_3) solid oxidizer and a humidifier.[316,317] This would prevent the appearance of negative nitric oxide values on the recorder. More compact instruments using the same principle have been marketed, but further size reduction is still desirable to facilitate transportation and lessen monitoring space requirements.

SUMMARY

This review of available methodology for oxides of nitrogen summarizes the development of procedures designed to establish an air quality standard. Only traditionally accepted terms have been used. "Reference" denotes only a calibrating method. Primary reference methods serve as calibrating methods; secondary reference methods are reliable, accurate, and mostly interference-free measuring methods. Weighable primary standards, or secondary standards measurable by a primary reference method, can both be used for calibration. Permeation tubes are primary standards, whereas a

cylinder with a dilute gas mixture, previously calibrated by measurement with a primary reference method, is a secondary standard.

The 100-year-old chemical methodology for nitrogen oxides measurement involves the transfer of the nitrogen from nitrogen oxides directly into a measurable compound. This methodology, and many of its variations, has generated most of the data over the last 40 years and is still being used locally and internationally. The manual Griess-Saltzman Method (not to be confused with the Griess-Saltzman reagent or principle) has been used to calibrate continuous analyzers since the 1950's. It is based on the direct reaction of nitrogen dioxide with a single reagent mixture to form a colored azo-dye. The Griess-Saltzman reagent and several modifications thereof form the basis for many continuous analyzers. Nitric oxide cannot be measured directly with this technique and must be oxidized to nitrogen dioxide with catalyst.

A recent development is the technique based on chemiluminescence which measures the light emitted by reaction of two gases. Improvements to this technique have come from both the official and the private sector. Nitric oxide is measured through reaction with ozone produced within the instrument by irradiation of air with a short-wave UV lamp. Measurement of nitrogen dioxide by chemiluminescence requires a previous reduction step to nitric oxide over a heated catalyst. Photofragmentation of nitrogen dioxide by ultraviolet light and measurement of the ozone produced does not involve contact with a catalyst but does need nitric oxide as a supporting gas.

Precisely known concentrations of dilute gases are required. These concentrations can be obtained by dynamic dilution of more concentrated

gas mixtures or with permeation tubes having precisely known leakage rates. In most wet chemical methods an alkaline nitrite or a nitrate is used as a primary standard.

Alkaline methodology has also been used to sample and measure nitrogen dioxide. The departure from original sampling conditions, absorbing reagent formulations, and assumption of constant performance generated equivocal results. A variety of new methods called Jacobs-Hochheiser were created but have not been completely evaluated. In these absorbing solutions nitrite is analyzed applying the Griess principle of azo-dye formation. More recent methods without these shortcomings have been developed and evaluated.

Most methods transforming nitrogen oxides into nitrate are most satisfactory at higher than atmospheric levels of nitrogen oxides. Higher levels of nitrogen oxides can also be analyzed with the same methods used in atmospheric analysis after proper aliquoting or dilution. Additionally, some optical methods, currently used for higher concentrations, cannot be applied to atmospheric analysis except when measuring from aircraft.

CONCLUSIONS AND RECOMMENDATIONS

Methods should be recommended only after careful evaluation of performance through collaborative testing by a balanced team of scientific groups and representatives of official and private agencies. These recommendations should consider the short- and long-term budgetary outlays of those involved. The user must be satisfied with their accuracy (equivalency) and precision (reproducibility) under real sampling conditions. Simple means of calibration should be available. Very few of the methods in use would meet all these parameters.

Some wet chemical methods may be reproducible if the methods are strictly followed. As a primary reference method, the manual Griess-Saltzman Method (not to be confused with the Griess-Saltzman reagent or principle) is recommended. The primary standard for calibration of this method is sodium nitrite.

Physical methods of analysis need frequent, regular calibrations, preferably in well-equipped laboratories. The most direct chemiluminescent method for nitrogen dioxide involves photofragmentation to ozone, then chemiluminescent measurement of this ozone. Less direct is the differential measurement of nitrogen oxides (less nitric oxide) by chemiluminescence after reduction over a heated catalyst. This second method requires close control because the lifetime and memory effects of the reducing catalyst under real situations are not known. More experience with this method is required before it can be recommended for nitrogen dioxide. Calibration of chemiluminescent methods with permeation tubes instead of secondary standards changes their category to primary reference methods. Despite recent developments of chemiluminescent instrumentation, improvements in reliability and simpler quality control checks are desirable.

To calibrate analytical methods using nitric oxide as standard gas, primary reference methods are preferred.

During environmental monitoring, the position of either the make-up atmosphere inlet or the sampling probe (or both) is of great importance and should be established by a team effort. A real time measurement of nitric oxides can be provided by chemiluminescent instrumentation; however, due to the uncertainties of their performance, the manual Griess-Saltzman Method should be used concurrently at the same manifold as a back-up control.

Future development should produce size and cost reduction of large, stationary instrumentation without impairing performance. A new regional survey method for background level control over large areas is also desirable and could reduce the monetary burden of local agencies. Further needs include continuous particulate nitrate and gas phase nitric acid monitors.

Finally, simplicity and a more precise terminology in the editing of documents would preclude misinterpretations.

CHAPTER 5

ATMOSPHERIC LEVELS OF NITROGEN OXIDES

The distribution of nitrogen oxides is by no means uniform. Localized concentrations often exceed the "average" concentration by a factor as high as 100.[563] These concentrations are located in urban areas having heavy automotive traffic, and in those major industrial areas whose nitric acid plants and uncontrolled stationary combustion sources produce oxides of nitrogen. The effect of these sources on actual pollutant levels is largely determined by the movement of the air mass into which the pollutants are emitted.

The most complete and authentic source of data on nitrogen oxide concentrations is the National Aerometric Data Bank of the Environmental Protection Agency (EPA). This data bank receives inputs from the National Air Surveillance Network (NASN) as well as from other state and local sources. NASN is comprised of approximately 100 sites which monitor nitrogen dioxide (NO_2) and sulfur dioxide (SO_2). Until recently, it also included six Continuous Air Monitoring Project (CAMP) stations. Additional data have been provided by more localized studies, such as the Chattanooga Study,[219] and the California Air Resources Board, which recently issued a 10-year summary of data[70] gathered through their large network of monitoring stations.

Current techniques for measuring oxides of nitrogen are not entirely satisfactory (See Chapter 4). As continuous instrumental methods are refined, and advances in the technology of preparing stable calibration mixtures are made, a more adequate data base will be developed. Until

such a data base is available, however, each analytical method used should be carefully evaluated in order to draw accurate conclusions from the data.

The EPA formerly designated the modified Jacobs-Hochheiser method as the Reference Method for nitrogen dioxide. This method has subsequently been proven inaccurate under certain conditions (See Chapter 4). Since data from this method must be used only with extreme care, they have not been included in this chapter.

The balance of the existing data was generated primarily by the continuous colorimetric technique, a modification of the Saltzman method.[554] This technique, which uses either the Griess-Saltzman reagent or the Lyshkow modified Griess-Saltzman reagent,[145] is inaccurate at low concentrations, and is subject to negative interferences from large concentrations of ozone.[569] Nevertheless, results from this method compare favorably with those of the chemiluminescent method, which does not suffer ozone interference, and with the so-called "Arsenite" method of Christie *et al.*[87] The EPA now regards these three methods as the most promising, and is subjecting them to rigorous evaluation.

GLOBAL DISTRIBUTION OF NITROGEN OXIDES

Ninety percent of nitrogen oxides in the earth's atmosphere is produced by natural bacterial action (See Chapter 3).

Many studies have been made to determine global background levels of nitrogen oxides. This task is difficult, and the results become less precise, in areas having many manmade sources. For 1968, Hamilton[204] reported average concentrations of nitric oxide (NO) at 3.4 $\mu g/m^3$ (2.7 ppb) and nitrogen dioxide at 7.7 $\mu g/m^3$ (4.1 ppb) at Pike's Peak, Colorado. Earlier

observations by Ripperton *et al.* at an altitude of 1,573 meters in the Appalachian area of North Carolina[438] had indicated similar concentrations--nitric oxide at 3.4 $\mu g/m^3$ (2.6 ppb) and nitrogen dioxide at 8.6 $\mu g/m^3$ (4.6 ppb). At the same time, these authors observed mixing effects of small scale turbulence, and of larger scale mixing caused by large vertical movements of air masses, such as those accompanying a cold front. Observations in Hawaii,[273] showing average nitrogen dioxide concentrations of 2.4 $\mu g/m^3$ (1.3 ppb), and in Panama,[323] showing total NO_x values from 0.9 to 3.6 ppb, suggest that background concentrations in continental states are higher than those in less industrialized areas.

On the basis of observed background levels and the emission rates of nitrogen oxides, Robinson and Robbins[440] estimated the average residence times for nitric oxide and nitrogen dioxide at 4 and 3 days, respectively. Scavenging processes that limit residence times and, hence, the build up of nitrogen oxides, include photochemical reaction, oxidation of nitric oxide to nitrogen dioxide, nitrate formation, and probably other mechanisms.

The stratosphere, roughly between 11 and 35 km in altitude, is currently drawing considerable interest. Using airborne and balloonborne spectroscopy, Ackerman *et al.*[3] observed nitric oxide and nitrogen dioxide levels of 1 ppb at 16 km altitude, increasing to 5 ppb each additional 30 km altitude. The supersonic Concorde 001 was used to collect the airborne data. The observations of nitric oxide and nitrogen dioxide were not made concurrently. Using the same aircraft and a fast Fourier interferometer spectrometer, Farmer *et al.*[140] computer concurrent values of 0.2 to 0.4 ppb nitric oxide and 1.8 ppb nitrogen dioxide at approximately 16 km, giving a nitrogen

dioxide to nitric oxide ratio of approximately 4.5. These observations were made at sunset in June 1973. For additional data on the contribution of high-flying aircraft to nitrogen oxide levels in the stratosphere, see the report of the Advisory Group for Aerospace and Development.[6]

Concentration of nitrous oxide (N_2O) has also been determined in the experiments of Farmer *et al.*[140] A relatively constant value of 0.2 ppm nitrous oxide was observed between 12 and 20 km.

Nitrous oxide concentrations are significant as the probable source of nitric oxide in the lower stratosphere, and the effect which supersonic aircraft might have on nitrogen dioxide concentration.[394]

The two primary sources of oxides of nitrogen generated by man are the internal combustion engine and fossil fuel burning power plants (See Chapter 3).

Five CAMP stations[568] recorded a gradual increase in average NO_x concentration (Figure 5-1). The same trend is indicated in limited data available from European cities[399] (Table 5-1). Although the trends are the same, the trend lines vary slightly from location to location.

TIME RELATED VARIATIONS IN NITROGEN OXIDE CONCENTRATIONS

Diurnal Patterns

Geographic and meteorological factors can combine to amplify the effect of manmade emissions. In the calm air mass of the Los Angeles basin, both horizontal and vertical movement of the air mass are minimal. Under these conditions, nitrogen oxides build up, and nitric oxide converts to the more harmful nitrogen dioxide (Figure 5-2). Figure 5-3 contrasts the patterns of nitrogen dioxide concentrations found on days of low and high ventilation and solar radiation in Vienna, Austria.[399]

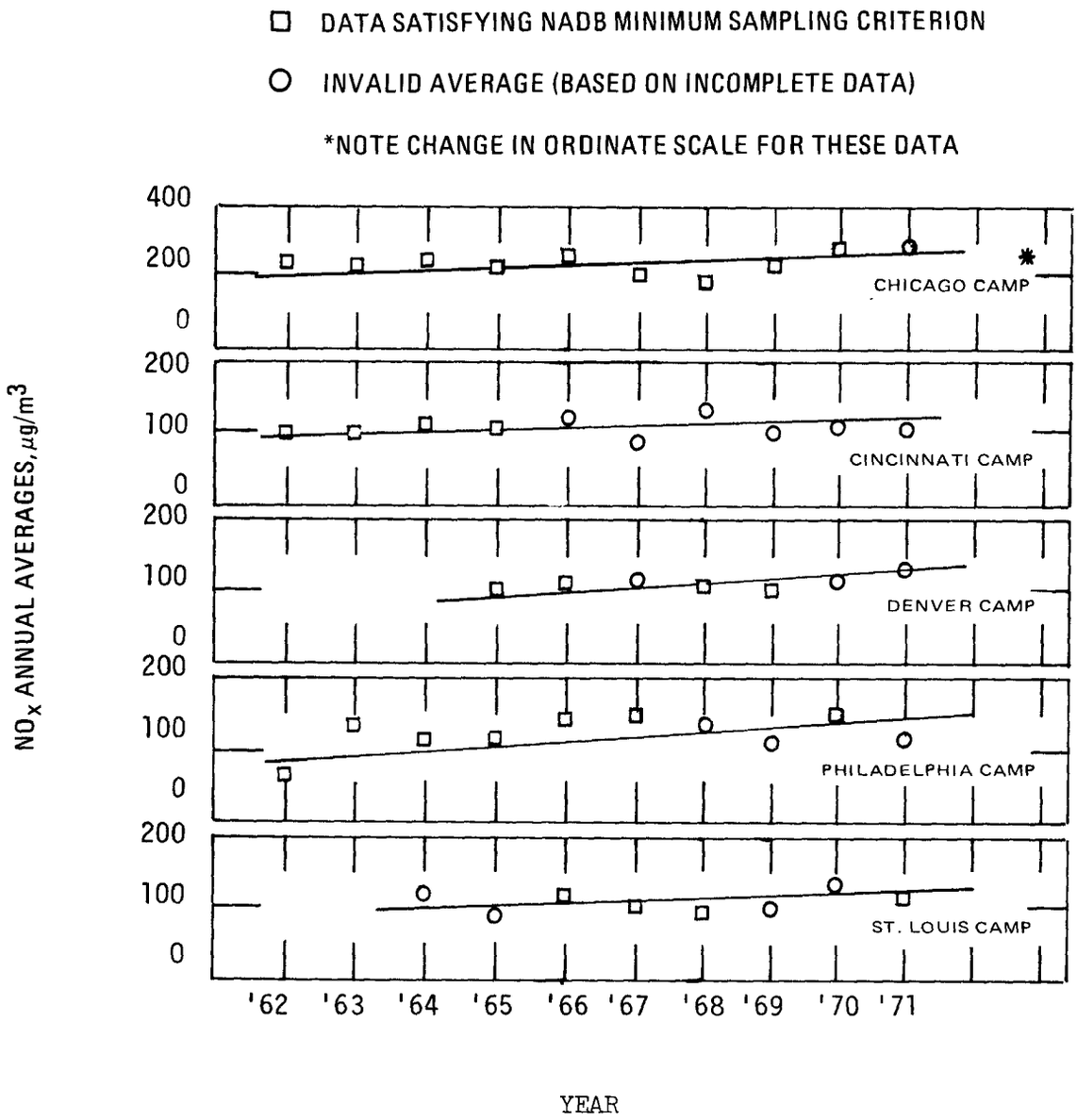


FIGURE 5-1. Trend lines for NO_x annual averages in five CAMP cities.
From U.S. Environmental Protection Agency, 1973.

TABLE 5-1

Annual Average Concentrations of Nitrogen Dioxide in Selected European Cities[a]

	Concentration, $\mu g/m^3$ (ppb)				
	Rome[b]	Rotterdam[c]	Vlaardingen[c]	Frankfurt[c]	Munich[b]
1962	--	--	--	19 (10)	--
1963	--	--	--	23 (12)	--
1964	--	--	--	28 (15)	--
1965	--	--	--	30 (16)	--
1966	--	35 (19)	--	47 (25)	--
1967	--	43 (23)	--	34 (18)	56 (30)
1968	--	43 (23)	40 (21)	41 (22)	132 (70)
1969	--	43 (23)	38 (20)	100 (53)	489 (260)
1970	30 (16)	45 (24)	39 (21)	--	827 (440)
1971	36 (19)	--	--	--	--

[a] From North Atlantic Treaty Organization, Committee on the Challenges of Modern Society. 1973.[399]

[b] Method of measurement not indicated.

[c] Concentrations measured by Griess-Saltzman Method.

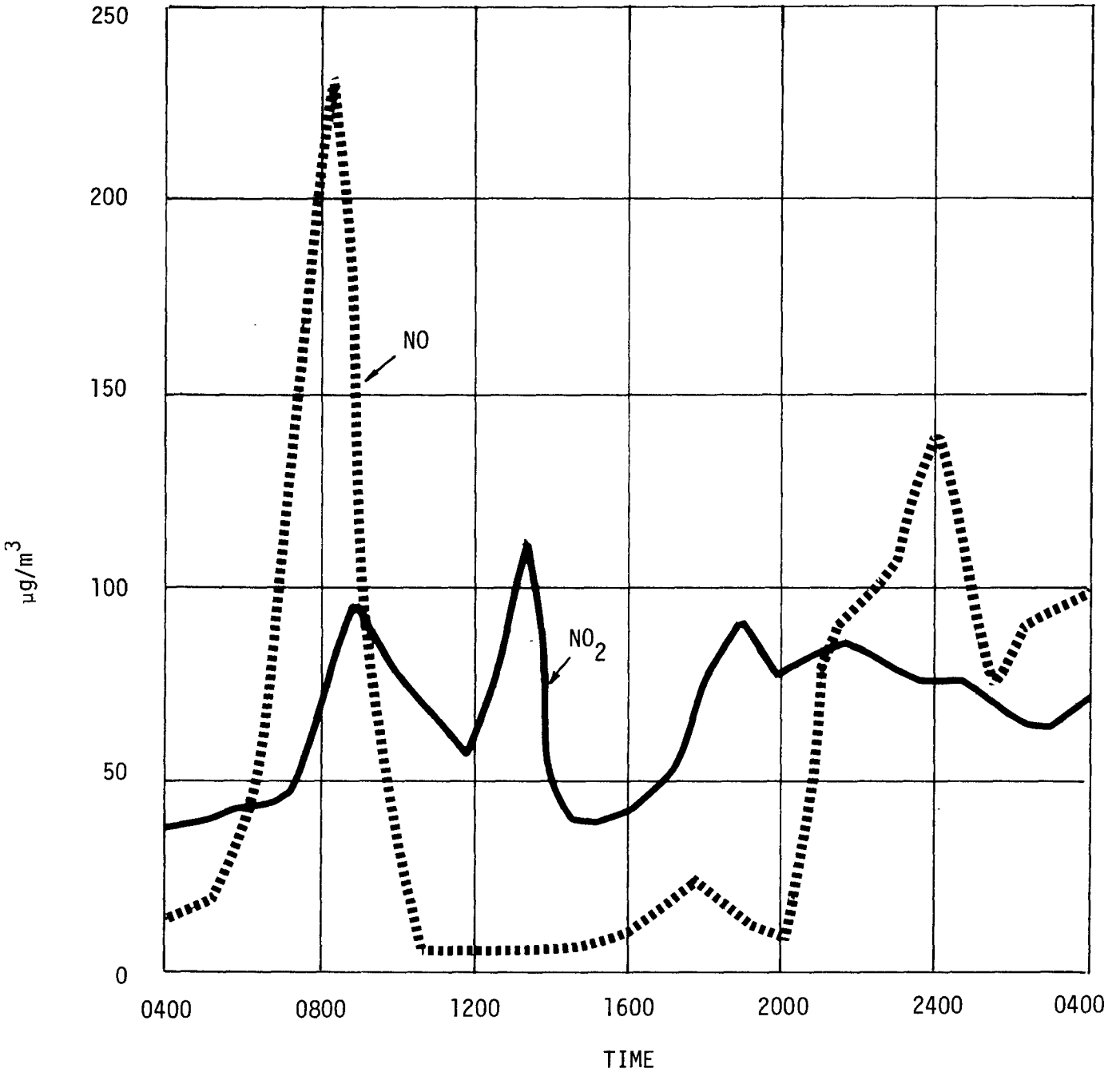


FIGURE 5-2. Variation in nitric oxide and nitrogen dioxide concentration, Orange County, California, October 16-17, 1974.

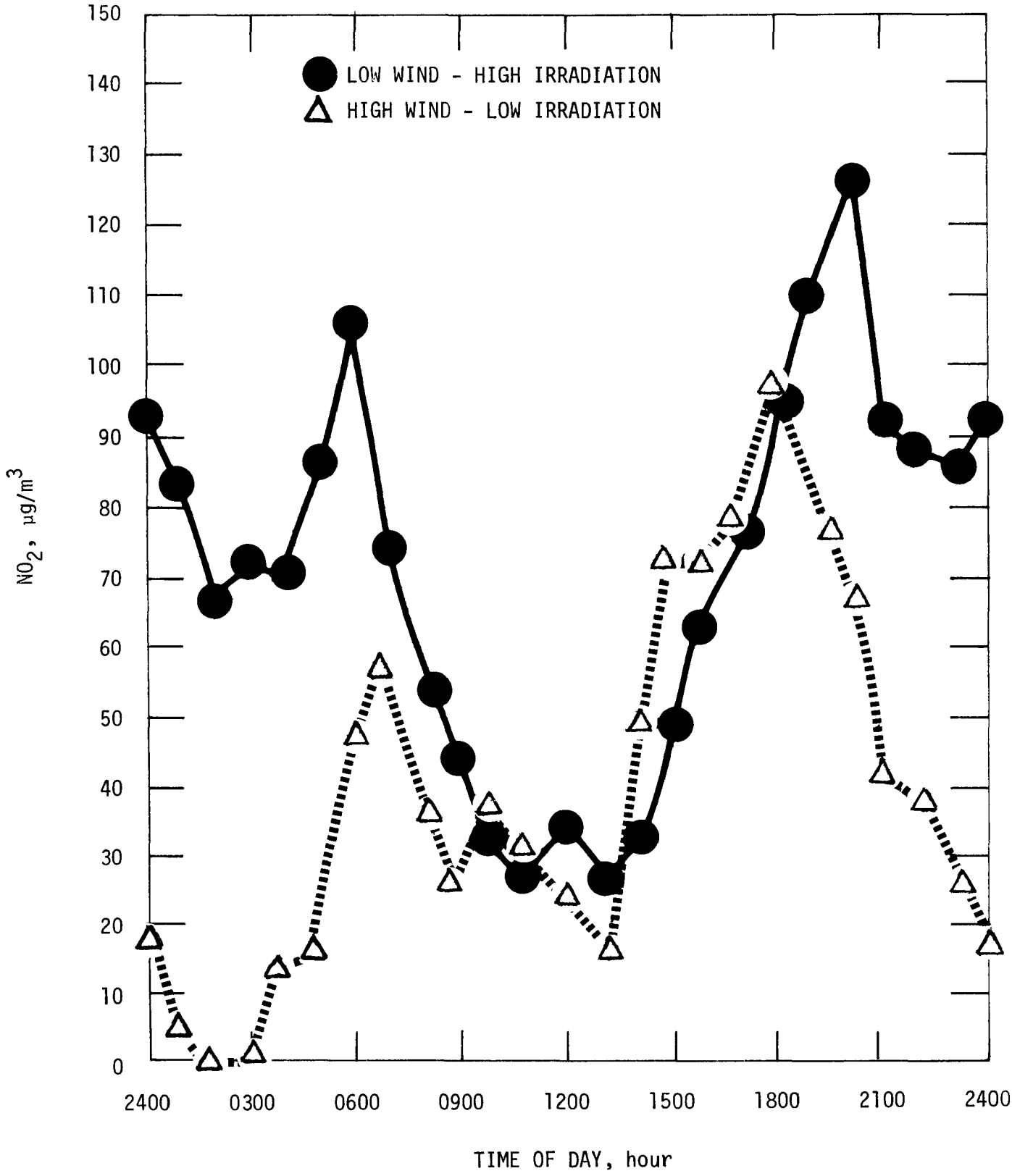


FIGURE 5-3. Diurnal variation of nitrogen dioxide in Vienna, Austria, 1966. From North Atlantic Treaty Organization, Committee on the Challenges of Modern Society, 1973.[399]

Because a large portion of urban nitrogen oxides is generated by human activity, variations in oxide concentrations correlate directly with such activities. The major variable is vehicular traffic. Periods of heavy traffic, such as morning and evening rush hours, produce correspondingly high concentrations of nitric oxide. During slack periods, such agents as breezes and sunlight disperse, convert, or otherwise reduce these high concentrations. These competing factors create typical diurnal patterns (Figure 5-2).

Figure 5-2 data were collected from a relatively stable air mass in Orange County, California, 48 km southeast of Los Angeles, on October 16, 1974. The day's intense sunshine was followed by low clouds in the evening and throughout the night. The high temperature was approximately 32°C.

Vehicular traffic in Orange County increases sharply beginning at 6 a.m., accompanied by a rapid nitric oxide buildup. This nitric oxide converts quickly to nitrogen dioxide in bright sunlight. A local influence--perhaps a short-lived breeze or a brief period of cloudiness--can cause a minimum nitrogen dioxide concentration at noon. The late afternoon levels of 75.2-94 $\mu g/m^3$ (40-50 ppb) nitrogen dioxide, with nitric oxide levels of approximately 12.5 $\mu g/m^3$ (10 ppb), are typical of a sunny day. In the early evening, concentrations of both pollutants build up. Nitric oxide concentrations increase appreciably immediately after sunset.

Certain elements of this diurnal pattern are shared by other urban population centers. Nitrogen dioxide data from Rome, Italy; Vienna, Austria; and Rotterdam, The Netherlands[398] all show the morning and evening maxima, with tendencies toward lower concentrations between these periods.

Seasonal Patterns

Patterns associated with the seasonal variation of temperature and prevailing winds may also be traced. Increased use of heating fuels during the winter months increases the contribution from this source. Since the rate at which nitric oxide converts to nitrogen dioxide is related to the intensity of solar radiation, conversion is most rapid during warm summer days. Figure 5-4 shows the seasonal variation of nitric oxide in the Los Angeles area, and Figure 5-5 shows the variations of nitrogen dioxide.

Each "typical" pattern can be disturbed by factors localized in space or time. A cloudy day will prolong the duration of high nitric oxide concentrations. A strong wind will quickly disperse all pollutants and provide a clean atmosphere. Geographical features, particularily hills and valleys, affect both temperature profiles and air movement, and can have a major effect on pollutant levels.

Annual Trends

The most complete continuous data base used to determine long-term trends of oxides of nitrogen on a national scale has been generated by the Federal Government's Continuous Air Monitoring Program (CAMP). One CAMP station is currently located in the main business district of each of the following cities: Chicago, Cincinnati, Denver, Philadelphia, St. Louis, and Washington, D.C. Since the Washington, D.C. station was relocated in 1969, its data are not continuous. For the other five cities, the CAMP program has produced continuous data for 12 years.[567]

All sampling methods and operating modes were chosen to facilitate comparison of data between cities. Nitric oxide and nitrogen dioxide are

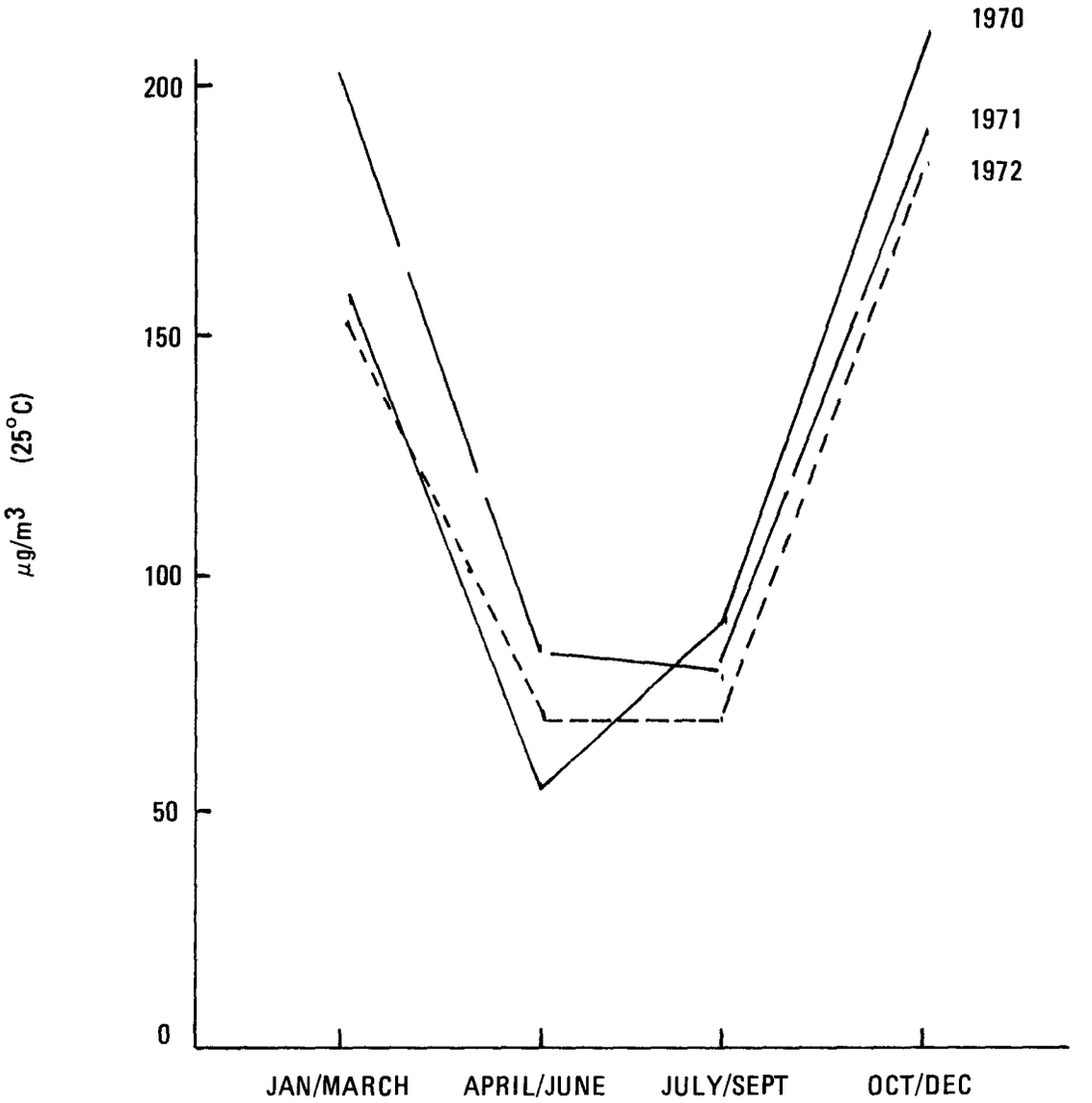


FIGURE 5-4. Quarterly mean of hourly average concentration of nitric oxide at downtown Los Angeles, California. From State of California, 1974.[70]

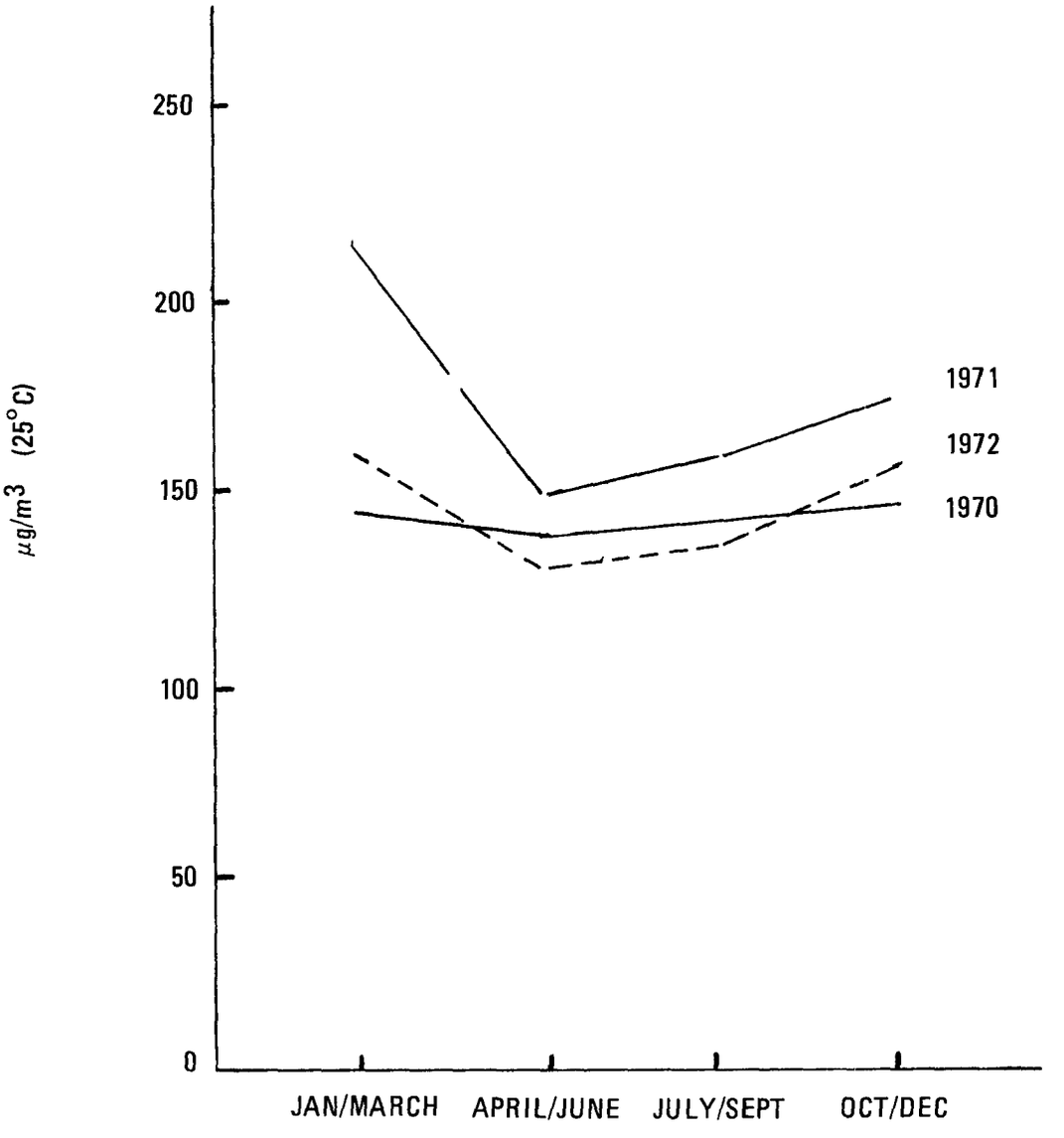


FIGURE 5-5. Quarterly mean of hourly average concentration of nitrogen dioxide at downtown Los Angeles, California From State of California, 1974.[70]

determined individually by the continuous Saltzman colorimetric method. In addition to oxides of nitrogen, the CAMP stations determine carbon monoxide, sulfur dioxide, total hydrocarbons, and methane.

Tables 5-2, 5-3, and 5-4[568] compare the average concentrations of nitric oxide, nitrogen dioxide, and NO_x for two 5-year periods, 1962-1966 and 1967-1971. The data indicate a general trend toward higher concentrations. Nitric oxide increased by an average of 13%, nitrogen dioxide by 6%, and the combined NO_x by 9% over the two 5-year periods. The tables also list the average annual second highest observed values to indicate changes in extreme concentrations. In Chicago and Cincinnati nitric oxide concentrations increased considerably (32 and 36%, respectively), while in Denver, Philadelphia, and St. Louis, only minor changes were recorded. The second highest values for nitrogen dioxide do not parallel those for nitric oxide, and show only a 1% increase in the two averages. This lack of correlation between nitric oxide and nitrogen dioxide values could result from differing solar radiation or variation in reactive hydrocarbons, both of which affect the nitric oxide to nitrogen dioxide conversion rate.

Tables 5-5 and 5-6 give the year to year arithmetic mean and maximum values of nitric oxide and nitrogen dioxide for six CAMP stations, including Washington, D.C. These data are from the National Aerometic Data Bank. The general upward trend of most of these data is evident in Figures 5-6 and 5-7, where computed regression lines have been included.[568] Exceptions to the general trend are the nitrogen dioxide levels for Denver and St. Louis which are fairly stable.

Mathematical Models for Predicting Pollutant Concentrations

Marked progress is being made in the mathematical simulation of environmental pollution phenomena. Simulations range from the relatively simple

models developed by Larsen on the basis of empirical observations[307] to sophisticated systems such as the Implementation Planning Program model developed for EPA by TRW Systems.[545] The TRW model uses individual sources of pollutants as the basic input to a diffusion model. Concentration of pollutants expected at several selected receptor points are calculated. The program is designed to facilitate calculation of the effect of various control strategies, differing pollutant standards, and the cost of the different approaches.

Larsen's model, which predicts pollutant concentrations on the basis of previously observed average values and an assumed lognormal distribution (found to be true for data from eight cities, for seven pollutants, over a period of several years) continues to produce good forecasts of pollutant levels. This model has determined pollutant reductions required at the source in order to meet air quality standards.[32] A recent paper[419] confirms the validity of Larsen's approach, while at the same time questioning the stringency of the vehicle emission standards considered necessary by EPA to meet the air quality standards.

Several mathematical models which have been developed for specific regions are capable of broader application. The Fortak model[151] has been used in city planning, and for determining the potential effect of proposed major commercial or industrial installations on an existing pollutant profile. It has been applied successfully in Duesseldorf and Stockholm.[290] Kohn has studied the St. Louis, Missouri area and developed a model of air quality, and Haagenson *et al.*[200] have modeled pollutant plume behavior in the same area to determine locations of mobile monitoring stations. MacCracken *et al.*[328] used a multi-box approach and a mass consistent wind

TABLE 5-2

5-yr Averages of Nitric Oxide Concentrations at CAMP Stations, Measured by Continuous Saltzman Colorimetric Method[a]

Station	Average concentration, $\mu g/m^3$ (ppb) 25°C			Average of annual 2nd highest value, $\mu g/m^3$ (ppb) 25°C		
	1962-1966	1967-1971	Change, %	1962-1966	1967-1971	Change, %
Chicago	122.6 (98.1)	125.4 (100.3)	+ 2	731 (584.8)	969 (775.2)	+32
Cincinnati	43.8 (35.0)	53.6 (42.9)	+22	782 (625.6)	1,067 (853.6)	+36
Denver	44.9 (35.9)	54.4 (43.52)	+21	633 (506.4)	620 (496.0)	- 2
Philadelphia	55.2 (44.2)	65.4 (52.3)	+18	1,331 (1,064.8)	1,395 (1,116.0)	+ 5
St. Louis	39.8 (31.8)	47.6 (38.1)	+19	541 (287.8)	578 (462.4)	+ 7
CAMP average	61.2 (49.0)	69.3 (55.4)	+13	804 (643.2)	926 (740.8)	+15

[a]From U.S. Environmental Protection Agency, 1973.

TABLE 5-3

5-yr Averages of Nitrogen Dioxide Concentrations at CAMP Stations, Measured by the Continuous Saltzman Colorimetric Method[a]

Station	Average concentration, $\mu g/m^3$ (ppb) 25°C			Average of annual 2nd highest value, $\mu g/m^3$ (ppb) 25°C		
	1962-1966	1967-1971	Change, %	1962-1966	1967-1971	Change, %
Chicago	86.1 (45.8)	101.2 (53.8)	+18	444 (236.2)	499 (265.4)	+12
Cincinnati	62.0 (33.0)	60.0 (31.9)	- 3	391 (208.0)	367 (195.2)	- 6
Denver	66.0 (35.1)	67.9 (36.1)	+ 3	498 (264.9)	493 (262.2)	- 1
Philadelphia	67.7 (36.0)	77.6 (41.3)	+15	361 (192.0)	414 (220.2)	+15
St. Louis	58.5 (31.1)	54.2 (28.8)	- 7	320 (170.2)	267 (142.0)	-16
CAMP average	68.1 (36.2)	72.2 (38.4)	+ 6	403 (214.4)	408 (217.0)	+ 1

[a]From U.S. Environmental Protection Agency, 1973.

TABLE 5-4

5-yr Average NO_x Concentrations at CAMP Stations, Measured by the Continuous Saltzman Colorimetric Method[a]

Station	Average concentration, $\mu g/m^3$ 25°C		
	1962-1966	1967-1971	Change, %
Chicago	208.7	226.6	+ 8
Cincinnati	105.8	113.6	+ 7
Denver	110.9	122.3	+10
Philadelphia	122.9	143.0	+16
St. Louis	98.3	101.8	+ 3
CAMP average	129.3	141.5	+ 9

[a] From U.S. Environmental Protection Agency, 1973.

TABLE 5-5

Yearly Average and Maximum Concentrations of Nitric Oxide at CAMP Stations, $\mu g/m^3$ (ppb) 25°C, Measured by the Continuous Saltzman Colorimetric Method[a]

	Denver		Washington		Chicago		St. Louis		Cincinnati		Philadelphia	
	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max
1962	--	--	37	788	123	739	--	--	37	702	25	431
	--	--	(30)	(630)	(98)	(704)	--	--	(30)	(562)	(20)	(345)
1963	--	--	49	1,060	123	615	--	--	37	615	62	1,845
	--	--	(39)	(848)	(98)	(492)	--	--	(30)	(492)	(50)	(1,476)
1964	--	--	49	1,070	123	1,105	49	923	49	787	62	1,400
	--	--	(39)	(856)	(98)	(884)	(39)	(738)	(39)	(630)	(50)	(1,120)
1965	37	652	37	751	123	750	37	443	37	750	62	1,083
	(30)	(522)	(30)	(600)	(98)	(600)	(30)	(354)	(30)	(600)	(50)	(866)
1966	49	627	49	1,240	123	775	37	688	49	1,230	74	2,290
	(39)	(502)	(39)	(1,000)	(98)	(620)	(30)	(550)	(39)	(984)	(60)	(1,832)
1967	49	590	62	1,390	98	763	49	393	37	1,685	74	1,820
	(39)	(472)	(50)	(1,112)	(78)	(610)	(39)	(314)	(30)	(1,348)	(60)	(1,456)
1968	49	738	49	837	86	739	37	492	74	1,242	62	1,735
	(39)	(590)	(39)	(670)	(77)	(591)	(30)	(394)	(60)	(994)	(50)	(1,388)
1969	49	677	49	959	135	1,920	37	873	49	861	49	1,083
	(39)	(542)	(39)	(767)	(108)	(1,536)	(30)	(698)	(39)	(689)	(39)	(866)
1970	62	750	62	1,430	172	2,240	62	689	49	960	74	1,672
	(50)	(600)	(50)	(1,144)	(137)	(1,792)	(50)	(551)	(39)	(768)	(60)	(1,338)

TABLE 5-5 - continued

	Denver		Washington		Chicago		St. Louis		Cincinnati		Philadelphia	
	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max
1971	62	677	49	775	135	824	62	615	62	750	49	935
	(50)	(542)	(39)	(620)	(108)	(659)	(50)	(492)	(50)	(600)	(39)	(748)
1972	74	788	86	825	160	787	62	714	49	763	62	800
	(60)	(630)	(69)	(660)	(128)	(630)	(50)	(571)	(39)	(610)	(50)	(640)
1973	74	652	123	640	221	775	74	750	49	689	--	--
	(60)	(522)	(98)	(512)	(177)	(620)	(60)	(600)	(39)	(551)	--	--

[a] From National Aerometric Data Bank, 1975.

TABLE 5-6

Yearly Average and Maximum Concentrations of Nitrogen Dioxide at CAMP Stations, $\mu g/m^3$, 25°C Measured by the Continuous Saltzman Colorimetric Method[a]

	Denver		Washington		Chicago		St. Louis		Cincinnati		Philadelphia	
	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max
1962	--	--	56	545	75	394	--	--	56	470	38	226
1963	--	--	56	394	75	376	--	--	56	377	75	584
1964	--	--	75	413	94	865	56	394	56	620	75	471
1965	75	507	56	770	75	319	56	226	56	301	75	358
1966	56	602	75	319	113	564	56	357	75	432	75	413
1967	75	583	75	376	94	470	38	282	56	432	75	413
1968	75	488	94	432	94	319	38	319	56	1,036	75	358
1969	56	620	75	432	94	358	56	714	56	394	75	245
1970	75	639	94	545	113	395	56	244	75	433	94	377
1971	75	676	75	413	113	1,090	56	244	56	301	75	770
1972	75	583	113	1,202	94	413	94	376	75	319	75	545
1973	94	846	--	--	132	676	56	508	75	207	--	--

[a] From National Aerometric Data Bank, 1975.

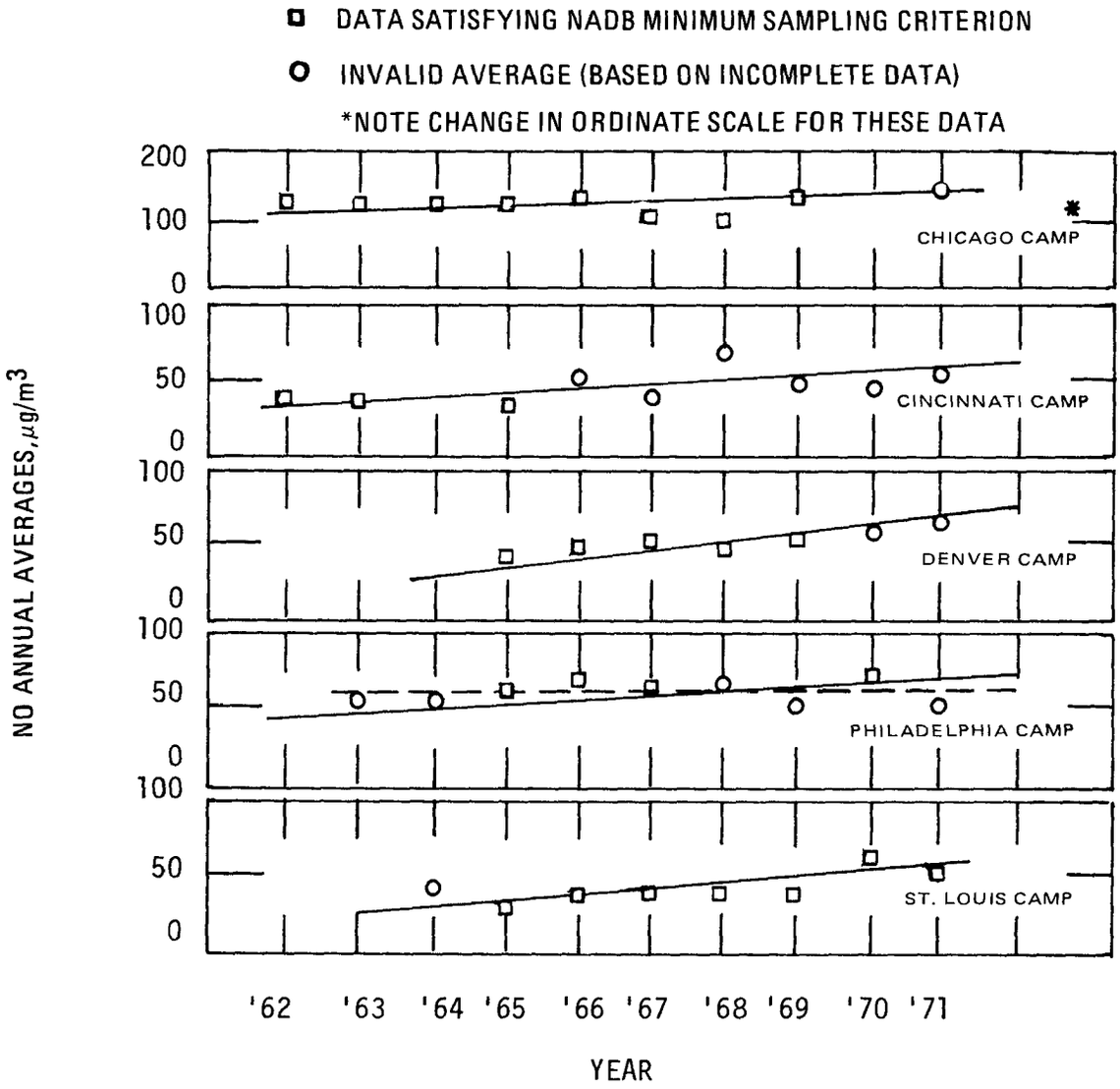


FIGURE 5-6. Trend lines for nitric oxide annual averages in five CAMP cities. From U.S. Environmental Protection Agency, 1973.[568]

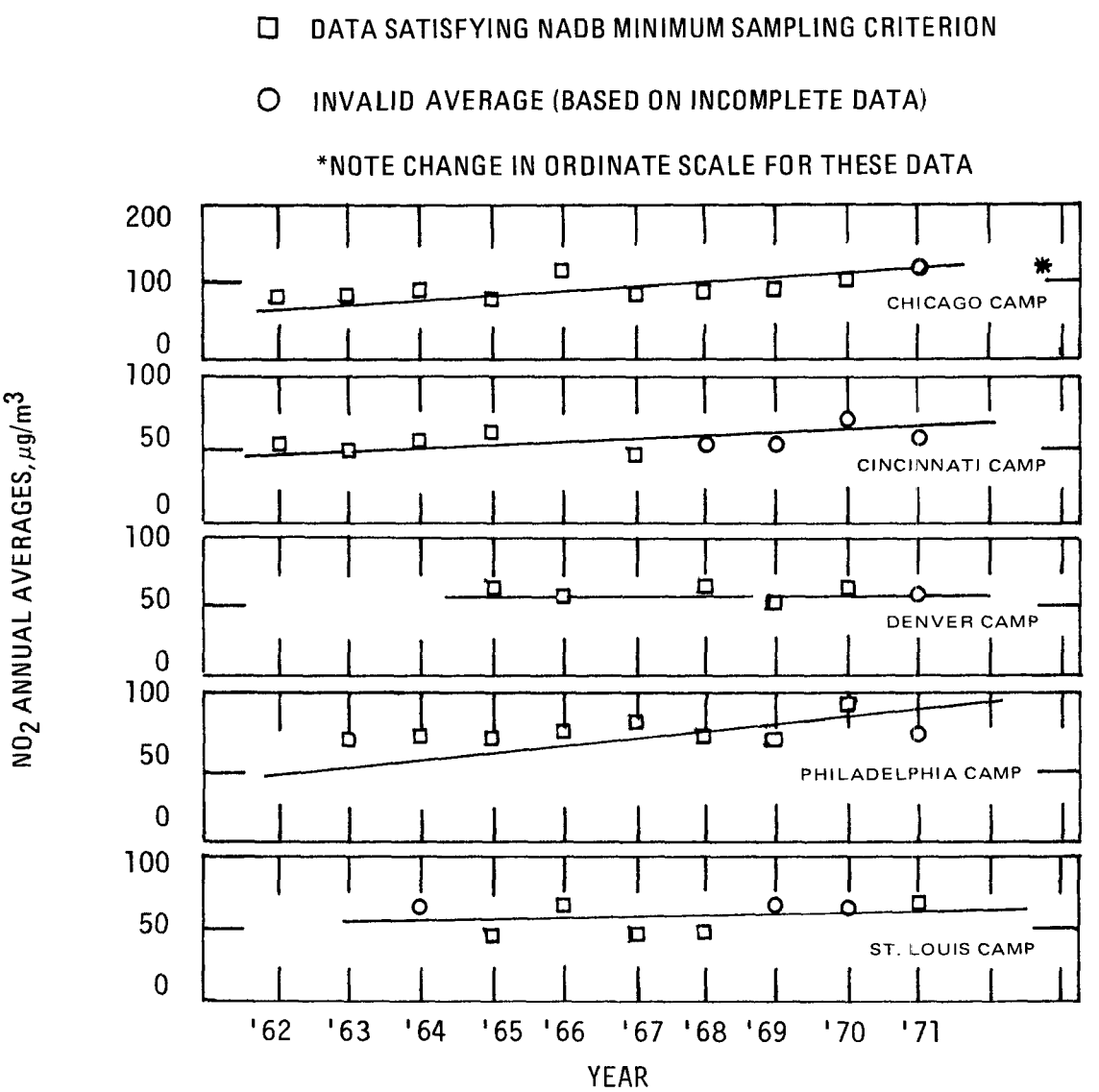


FIGURE 5-7. Trend lines for nitrogen dioxide annual averages in five CAMP cities. From U.S. Environmental Protection Agency, 1973.[568]

field submodel to simulate the San Francisco Bay region. Their model furnished frequency distribution data compatible with observed data.

EFFECTS OF METEOROLOGICAL FACTORS

The study of air pollution involves the generation of pollutants and their transport to receptors, usually at or close to ground level. This simplistic relationship immediately brings to mind the importance of the transport mechanism in determining the final effect of a given pollutant loading. Increasingly taller industrial smoke stacks are visible reminders that wide range dispersal is one means of "solving" pollution problems. When one considers the vast quantities of nitrogen oxides generated and disposed of by natural processess (see Chapter 3), the relative ineffectiveness of man's efforts becomes quite apparent.

Movements of the air mass into which pollutants are emitted are governed largely by meteorological factors, with some influence from local topographic features. The velocity and direction of surface winds determine the fraction of a given emission mass to be received at each receptor point. Vertical movement of the air mass is also important. Episodes of severe pollution are usually characterized by stagnant air masses, with inversion of the normal profile of temperature decrease with altitude. Such inversion tends to inhibit vertical movement of air.

Temperature and the amount of solar radiation reaching the earth's surface have particular influence on the oxidation of nitric oxide to nitrogen dioxide, and subsequent photochemical reactions. Figures 5-4 and 5-5 show seasonal nitric oxide and nitrogen dioxide variation for the Los Angeles area. Although fall and winter levels of nitric oxide are typically much

higher than summer levels, nitrogen dioxide concentrations are only marginally higher. An added indication of the effect of solar radiation is the incidence of photochemical smog in this region, which is most pronounced in the summer and early fall (the seasons of highest radiation).

Fortak's paper on mathematical modeling[151] includes a diagram which indicates the interrelationship of many meteorological factors in their effect on pollutant dispersion (Figure 5-8). Meteorological factors add greatly to the complexity of air pollutant problems. Fortak discusses atmospheric motions in some detail, pointing out the great difficulty of forecasting any movements of air masses other than large scale, synoptic movements.

OBSERVED URBAN NO_x CONCENTRATIONS

Table 5-7 gives the mean hourly average concentration and the maximum observed hourly concentration of nitrogen dioxide for representative U.S. cities. These data were obtained from the National Aerometric Data Bank, with some augmentation of the California data from the California Air Resources Board (ARB) Ten-year Summary.[70] In all cases the indicated method of analysis is the instrumental colorimetric method, using either the Griess-Saltzman reagent or the Lishkow reagent. As indicated earlier, data from this method of analysis are the best comprehensive data currently available. Major revision will probably not be required.

The California ARB data include determination of nitric oxide and total NO_x (see Tables 5-8 and 5-9). Most of the nitric oxide was determined with the parallel mode of operation, in which nitrogen dioxide is measured in one system and NO_x in a second. In the NO_x system, nitric oxide is oxidized to nitrogen dioxide prior to the determination. A few district

TABLE 5-7

Nitrogen Dioxide Hourly Average Concentrations in Selected U.S. Cities, $\mu g/m^3$, 25^oC[a]

Location	1963		1964		1965		1966		1967		1968		1969		1970		1971		1972		1973	
	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max
ARIZONA																						
Phoenix	--	--	--	--	--	--	--	--	75	1,504	68	1,504	--	188	--	--	--	--	--	83	--	--
CALIFORNIA																						
Anaheim	62	376	68	414	60	432	75	508	81	508	104	639	96	658	93	639	98	1,015	86	620	--	--
Azusa	76	677	87	395	79	564	88	583	91	602	88	639	95	658	118	808	117	696	116	827	--	--
Burbank	112	733	107	564	129	884	130	752	167	1,053	202	1,018	180	940	172	902	173	996	149	922	--	--
Fresno	38	188	42	320	40	470	47	263	51	320	54	320	60	301	--	--	--	--	--	--	--	--
La Habra	--	--	--	--	--	--	--	--	72	470	92	884	79	771	54	414	100	639	101	808	--	--
Lennox	--	--	--	--	120	808	111	752	147	1,260	135	1,072	123	752	132	752	130	808	120	733	--	--
Long Beach	118	1,072	113	1,109	104	865	118	790	142	865	156	1,316	141	996	148	978	126	865	132	1,034	--	--
Los Angeles																						
Downtown	122	1,034	107	1,316	134	1,316	146	1,504	126	1,015	132	884	122	1,504	140	1,335	173	1,391	146	733	--	--
Westwood Blvd.	89	771	93	752	121	996	110	884	115	940	136	884	120	902	127	978	141	1,128	119	790	--	--
County	--	--	--	--	--	620	99	658	113	827	118	865	110	658	130	733	133	714	129	790	--	--
Oakland	62	526	62	771	60	432	64	545	74	620	66	470	--	--	--	--	--	--	--	--	--	--
Oakland-Jackson	--	--	--	--	--	--	--	--	--	--	--	--	64	470	76	508	75	470	67	376	--	--
Pasadena																						
Villa St.	94	620	110	677	106	639	107	620	121	696	109	602	--	--	--	--	--	--	--	--	--	--
Pasadena	--	--	--	--	--	--	--	--	--	--	150	997	148	997	175	1,560	145	1,030	165	810	--	--
Pomona	--	--	--	--	122	545	119	545	131	696	148	790	145	808	158	846	146	752	145	808	--	--
Redlands	--	--	--	--	--	--	--	--	--	--	41	376	32	188	60	508	80	602	75	432	--	--
Redwood City	--	--	--	--	--	--	--	--	70	620	53	395	64	526	66	489	56	395	58	263	--	--
Richmond	--	--	--	--	--	--	49	263	63	395	72	376	66	547	64	376	40	357	51	150	--	--
Riverside	85	508	77	1,053	87	921	72	470	83	583	--	414	--	--	--	--	72	714	58	301	--	--
Sacramento	69	545	78	602	74	564	52	508	67	564	--	--	--	--	--	--	--	--	--	--	--	--
Sacramento Cty.	--	--	--	--	--	--	--	--	--	--	--	338	57	376	54	282	48	470	--	--	--	--
San Bernardino	23	263	--	113	51	207	77	470	90	414	66	470	79	396	94	451	94	508	87	546	--	--
San Diego	52	620	37	658	43	978	62	752	34	639	53	1,130	82	376	37	376	75	828	58	376	--	--
San Diego Cty.	--	--	--	--	--	--	--	696	45	395	66	639	--	--	47	338	75	752	--	470	--	--

Continued--

TABLE 5-7 - continued

Location	1963 Mean	1963 Max	1964 Mean	1964 Max	1965 Mean	1965 Max	1966 Mean	1966 Max	1967 Mean	1967 Max	1968 Mean	1968 Max	1969 Mean	1969 Max	1970 Mean	1970 Max	1971 Mean	1971 Max	1972 Mean	1972 Max	1973 Mean	1973 Max
CALIFORNIA cont'd																						
San Francisco																						
Ellis St.	--	--	--	--	--	--	--	--	--	--	85	640	81	639	92	791	53	508	65	620	--	--
Union Square	86	602	100	771	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
Mission St.	--	--	--	188	61	376	57	451	65	658	70	489	--	--	--	--	--	--	--	--	--	--
Santa Cruz Cty.	--	--	--	--	--	--	36	226	33	169	32	226	32	282	26	113	29	169	36	263	--	--
Stockton	--	244	49	414	39	263	38	301	41	338	60	414	73	414	39	320	43	282	45	207	--	--
FLORIDA																						
Jacksonville	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	206	--	1,562
ILLINOIS																						
Chicago																						
Taft H.S.	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	5,640	--	5,884	--	--	--	--
Lakeview H.S.	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	4,926	--	--	--	--
GSA Bldg.	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	884	--	2,970	--	--	--	--
Austin H.S.	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	4,268	--	--	--	--
Lindblom H.S.	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	5,584	--	--	--	--
Hyde Park H.S.	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	4,380	--	--	--	--
MARYLAND																						
Baltimore	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	320	--	921
MASSACHUSETTS																						
Boston	--	--	--	--	--	--	--	--	--	--	--	--	--	--	1,888	9,964	--	9,588	--	9,400	--	--
MINNESOTA																						
Minneapolis	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	117	--	--
MISSOURI																						
St. Louis Cty.																						
Residential	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	316	--	600	--	--
Commercial	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	301	--	936	--	--

TABLE 5-7 - continued

Location	1963		1964		1965		1966		1967		1968		1969		1970		1971		1972		1973	
	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max
NEW JERSEY																						
Bayonne	--	--	--	--	--	--	--	596	94	532	99	545	103	487	81	692	--	--	85	568	--	353
Camden	--	--	--	--	--	--	--	--	--	--	87	506	86	1,002	90	543	--	--	84	438	--	487
Newark	--	--	--	--	--	--	--	564	115	681	--	474	118	453	95	637	--	--	108	530	--	436
NEW YORK																						
Buffalo	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	500	--	252	--	417
Hempstead	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	1,028	--	438	--	286
New York City	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	744	--	703	--	521
Syracuse																						
S.T.P.	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	38	372	--	397	--	308
State Street	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	791	--	899	--	250
OHIO																						
Akron	--	--	--	--	--	--	--	--	--	--	--	--	--	282	--	752	--	376	--	301	--	226
OKLAHOMA																						
Oklahoma City	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	154	--	154	--	--
OREGON																						
Portland	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	357	--	227
PENNSYLVANIA																						
Philadelphia	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	85	9,494

[a]From National Aerometric Data Bank, 1975, and State of California Air Resources Board, 1974.

TABLE 5-8

Nitric Oxide Hourly Average Concentrations in California, $\mu g/m^3$, 25°C [a]

Location	1963		1964		1965		1966		1967		1968		1969		1970		1971		1972	
	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max
Anaheim	26	360	31	370	31	860	26	490	50	810	31	1,600	74	1,190	59	1,090	80	1,000	91	1,050
Azusa	25	520	23	260	20	290	21	320	25	490	37	360	26	430	42	500	43	500	42	790
Burbank	124	1,400	105	890	123	1,260	115	1,110	127	1,110	131	1,620	156	1,780	167	1,350	160	1,370	150	1,130
Fresno	13	1,070	23	580	18	640	16	660	21	730	21	780	23	1,050	--	--	--	--	--	--
La Habra	--	--	--	--	--	--	--	--	--	--	62	1,130	41	570	42	620	58	583	68	980
Lennox	--	--	--	--	127	1,250	148	2,160	181	2,220	166	1,660	157	2,050	176	2,270	181	2,340	172	1,870
Long Beach	134	1,140	140	1,540	117	920	123	970	140	1,280	143	1,170	130	1,160	148	1,960	118	1,070	128	1,520
Los Angeles																				
Downtown	121	1,050	98	1,600	101	1,410	112	1,020	119	1,410	120	1,360	119	1,490	130	1,600	139	1,410	122	1,130
U.S.C.	69	700	71	1,000	70	740	73	850	74	750	--	--	--	--	--	--	--	--	--	--
West LA	101	1,050	89	1,170	92	1,290	83	1,300	88	1,330	92	1,050	89	1,840	108	1,360	111	1,720	91	1,120
Oakland	58	1,140	90	1,140	58	810	60	840	60	1,120	55	910	--	--	--	--	--	--	--	--
Oakland-Jackson	--	--	--	--	--	--	--	--	--	--	--	--	43	850	40	710	44	1,050	45	660
Pasadena	--	--	--	--	--	--	--	--	--	--	81	820	71	820	84	980	69	690	89	860
Villa Street	70	850	63	760	50	520	60	610	66	650	57	550	--	--	--	--	--	--	--	--
Pomona	--	--	--	--	68	580	75	550	95	760	105	910	103	900	118	810	121	780	116	820
Redlands	--	--	--	--	--	--	--	--	--	--	22	460	20	210	22	1,170	32	1,760	43	650
Redwood City	--	--	--	--	--	--	--	--	42	530	54	590	47	630	42	580	41	780	48	590
Richmond	--	--	--	--	--	--	62	910	46	710	60	500	52	870	32	820	31	730	65	490
Riverside	--	--	57	1,350	46	700	46	530	53	640	--	--	--	--	--	--	--	--	41	680
Sacramento	48	1,330	49	1,330	41	1,190	44	920	42	1,110	33	1,140	33	1,140	35	750	33	780	--	--
San Bernardino	11	310	11	310	27	420	39	620	41	440	42	360	26	260	39	280	52	490	41	630
San Diego	44	910	30	1,350	38	1,110	62	1,480	43	980	57	1,350	38	1,110	36	860	81	2,150	66	1,300
San Francisco																				
Ellis Street	--	--	--	--	--	--	--	--	--	--	70	1,600	38	740	58	630	48	690	68	620
Union Square	102	1,590	103	950	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
Mission Street	--	--	--	--	52	760	--	--	49	1,090	63	1,670	--	--	--	--	--	--	--	--
Santa Cruz County	--	--	--	--	--	--	9	160	6	160	7	200	7	250	--	--	--	--	--	--
Stockton	--	--	39	620	32	590	41	1,070	16	440	25	620	26	640	22	700	25	700	30	570

[a] From State of California Air Resources Board, 1974.[70]

TABLE 5-9

NO_x Hourly Average Concentration in California, ppm[a]

Location	1963 Mean	1963 Max	1964 Mean	1964 Max	1965 Mean	1965 Max	1966 Mean	1966 Max	1967 Mean	1967 Max	1968 Mean	1968 Max	1969 Mean	1969 Max	1970 Mean	1970 Max	1971 Mean	1971 Max	1972 Mean	1972 Max
Anaheim	0.06	0.42	0.06	0.35	0.06	0.80	0.06	0.53	0.08	0.76	0.12	1.45	0.11	1.10	0.10	1.05	0.12	1.18	0.12	1.02
Azusa	0.06	0.48	0.07	0.28	0.06	0.35	0.06	0.48	0.07	0.52	0.08	0.45	0.07	0.53	0.10	0.56	0.10	0.54	0.10	0.77
Burbank	0.16	1.20	0.14	0.83	0.17	1.18	0.16	1.02	0.19	1.08	0.25	1.72	0.22	1.73	0.23	1.30	0.22	1.22	0.20	1.16
Fresno	0.03	0.93	0.04	0.52	0.04	0.55	0.04	0.60	0.04	0.66	0.05	0.70	0.05	0.94	--	--	--	--	--	--
La Habra	--	--	--	--	--	--	--	--	--	--	0.10	0.96	0.07	0.52	0.06	0.60	0.10	0.65	0.11	1.05
Lennox	--	--	--	--	0.17	1.21	0.18	1.95	0.23	2.10	0.21	1.66	0.19	1.86	0.21	2.15	0.22	2.21	0.21	1.71
Long Beach	0.17	1.14	0.18	1.36	0.15	0.85	0.16	0.85	0.19	1.24	0.20	1.20	0.18	1.15	0.20	1.94	0.16	1.05	0.17	1.50
Los Angeles																				
Downtown	0.16	1.11	0.14	1.46	0.16	1.35	0.17	0.98	0.17	1.34	0.17	1.27	0.16	1.41	0.18	1.50	0.20	1.42	0.18	1.16
U.S.C.	0.12	0.88	0.12	0.89	0.13	0.82	0.12	0.86	0.13	0.84	--	--	--	--	--	--	--	--	--	--
Oakland	0.10	0.95	0.11	1.26	0.08	0.74	0.08	0.79	0.09	1.06	0.08	0.85	--	--	--	--	--	--	--	--
Oakland-Jackson	--	--	--	--	--	--	--	--	--	--	--	--	0.07	0.70	0.07	0.66	0.08	0.96	0.07	0.67
Pasadena	--	--	--	--	--	--	--	--	--	--	0.15	1.03	0.14	1.19	0.16	1.20	0.13	0.86	0.16	0.94
Pasadena																				
Villa St.	0.11	0.79	0.11	0.70	0.10	0.60	0.11	0.78	0.12	0.71	0.10	0.58	--	--	--	--	--	--	--	--
Pomona	--	--	--	--	0.12	0.59	0.12	0.59	0.15	0.67	0.16	0.84	0.16	0.89	0.18	0.81	0.18	0.80	0.17	0.94
Redlands	--	--	--	--	--	--	--	--	--	--	0.04	0.43	0.03	0.22	0.05	1.00	0.07	1.51	0.07	0.60
Redwood City	--	--	--	--	--	--	--	--	0.07	0.49	0.07	0.52	0.07	0.70	0.07	0.58	0.06	0.74	0.07	0.55
Richmond	--	--	--	--	--	--	0.08	0.80	0.07	0.90	0.09	0.55	0.08	0.80	0.06	0.72	0.05	0.63	0.08	0.44
Riverside	--	--	0.09	1.16	0.09	0.76	0.08	0.55	0.09	0.67	--	--	--	--	--	--	0.04	0.20	0.06	0.60
Sacramento	0.08	1.19	0.81	1.22	0.07	1.05	0.07	1.00	0.07	1.14	0.05	1.06	0.06	1.03	0.06	0.72	0.05	0.74	--	--
San Bernardino	0.02	0.25	0.01	0.25	0.05	0.39	0.07	0.57	0.08	0.48	0.07	0.36	0.06	0.33	0.08	0.30	0.08	0.48	0.08	0.58
San Diego	0.06	0.96	0.04	1.18	0.05	0.98	0.08	1.42	0.05	0.84	0.07	1.66	0.05	0.94	0.05	0.73	0.10	1.82	0.09	1.14
San Francisco																				
Ellis St.	--	--	--	--	--	--	--	--	--	--	0.10	1.42	0.07	0.64	0.09	0.62	0.07	0.70	0.09	0.58
Union Sq.	0.13	1.34	0.14	0.93	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
Mission St.	--	--	--	--	0.08	0.71	--	--	0.07	0.97	0.09	1.50	--	--	--	--	--	--	--	--
Santa Cruz Co.	--	--	--	--	--	--	0.03	0.17	0.02	0.20	0.02	0.22	0.02	0.23	--	--	--	--	--	--
Stockton	--	--	0.06	0.68	0.05	0.53	0.05	0.90	0.04	0.42	0.05	0.60	0.06	0.62	0.04	0.62	0.04	0.68	0.05	0.55

[a] From State of California Air Resources Board, 1974.[70]

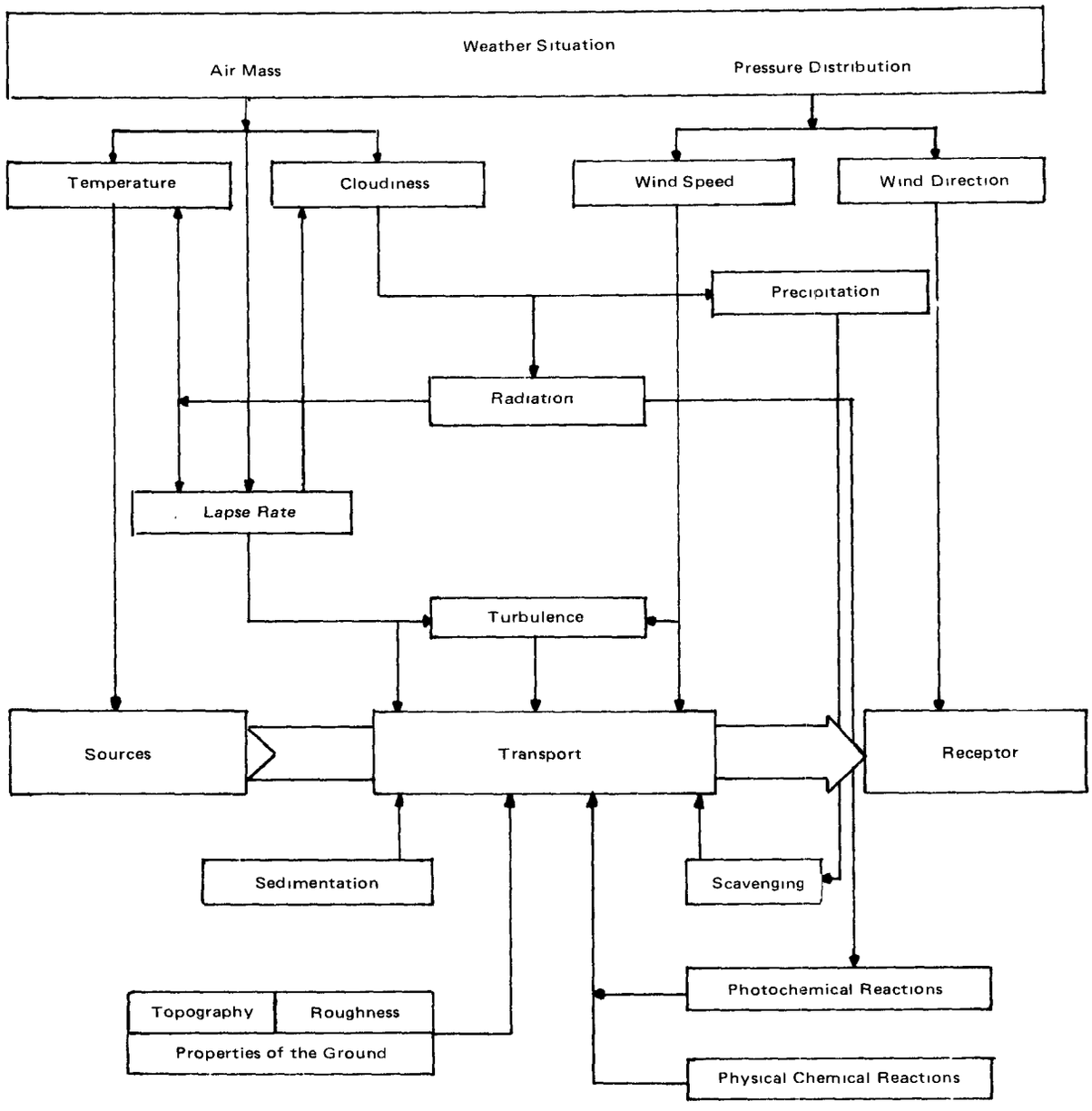


FIGURE 5-8. Meterological influences on transport and turbulent diffusion of air pollution components in the atmosphere. From Fortak, 1973.[151]

stations use the series mode of operation in which nitric oxide is oxidized and measured after the initial measurement of nitrogen dioxide. Table 5-10 shows the frequency distribution of daily maximum hourly concentrations of nitrogen dioxide for a number of California monitoring stations. These data are also from the ARB Ten-year Summary.

Annual average concentrations of nitrogen dioxide for five European cities indicate a trend toward higher concentrations (Table 5-1). Munich data were collected near streets with high traffic density, and are not representative of typical ambient levels for the city.

These data are supplemented by those for CAMP stations (Tables 5-5 and 5-6), whose general upward trend in total oxides of nitrogen was discussed above.

In a 1972 program to recheck nitrogen dioxide concentrations in the 47 "Priority I" Air Quality Control Regions, the EPA used the arsenite chemical and chemiluminescent instrumental methods of analyses. Simultaneous measurments were made by the original reference method for comparison purposes, with the understanding that uncertain results should be expected. Regions were classified "Priority I" if their average annual concentration of nitrogen dioxide exceeded 0.0585 ppm. Results for the 47 regions (Table 5-11)[569] cannot be used to compare the two analytical methods, because sampling locations and analysis periods were different.

The effects of a localized source of oxides of nitrogen, and other pollutants, are illustrated by the Chattanooga study.[219] This study was undertaken to determine pollutant levels and their possible effects on materials and vegetation in the area. The major pollutant source in this area is the Volunteer Army Ammunition Plant at Tyner, Tennessee, where

TABLE 5-10

Frequency Distribution of Daily Maximum Hourly Average Concentrations of Nitrogen Dioxide, pphm[a]

	Concentrations Equalled or Exceeded by Stated Percent of Observations								
	1%	2%	3%	4%	5%	10%	25%	50%	75%
Anaheim									
1963	19	16	15	13	12	10	7	5	4
1964	13	13	11	10	10	9	7	6	5
1965	19	18	18	17	17	12	8	5	3
1966	20	20	19	18	18	14	10	7	5
1967	24	24	22	21	20	17	11	7	5
1968	32	30	25	25	23	20	13	9	6
1969	27	23	22	20	19	15	11	8	6
1970	25	21	19	19	18	15	11	8	6
1971	42	31	25	24	23	18	11	8	5
1972	29	21	20	19	19	15	10	7	5
Azusa									
1963	21	20	17	16	16	14	11	8	5
1964	20	19	18	17	16	14	11	8	6
1965	24	20	19	19	17	14	10	7	5
1966	24	22	19	19	18	15	12	8	6
1967	29	25	21	19	19	16	13	8	6
1968	29	26	26	25	23	18	12	8	4
1969	30	21	19	18	18	15	12	8	6
1970	32	29	27	26	25	19	15	10	6
1971	30	27	26	24	23	19	14	10	8
1972	34	29	27	25	24	19	14	10	7
Bakersfield ARB-Golden									
1963	12	11	11	10	10	8	7	5	4
1966	14	12	11	11	10	9	8	6	5
1967	16	15	14	13	13	11	8	6	5
1968	16	15	14	13	13	11	8	6	5

TABLE 5-10 - Continued

	Concentrations Equalled or Exceeded by Stated Percent of Observations								
	1%	2%	3%	4%	5%	10%	25%	50%	75%
Bakersfield ARB-Golden cont'd.									
1969	16	14	14	14	13	12	10	8	6
1970	17	16	15	14	14	12	10	8	6
1971	11	11	10	10	10	9	8	7	5
Bakersfield-Chester St.									
1971	15	13	12	12	12	11	9	8	6
1972	14	13	12	12	11	10	8	7	5
Burbank									
1963	36	30	29	26	24	20	14	9	6
1964	26	24	22	22	20	18	14	10	6
1965	38	32	30	28	27	23	17	11	7
1966	35	32	29	28	27	24	17	11	8
1967	44	42	37	35	33	29	21	14	9
1968	47	43	42	40	38	33	26	17	11
1969	47	42	36	35	34	30	22	15	10
1970	43	38	36	34	34	28	22	14	9
1971	40	38	37	36	34	30	22	15	10
1972	35	31	31	27	26	22	19	13	9
Fresno-Cedar St.									
1963	9	8	8	7	7	6	5	4	3
1964	11	10	9	9	9	8	6	4	3
1965	19	13	12	11	10	8	5	4	2
1966	12	12	11	10	10	8	6	4	3
1967	14	12	12	12	11	9	6	5	3
1968	14	13	12	11	10	9	7	5	4
1969	13	12	11	11	10	9	8	6	4

TABLE 5-10 - Continued

	Concentrations Equalled or Exceeded by Stated Percent of Observations								
	1%	2%	3%	4%	5%	10%	25%	50%	75%
Fresno-Courthouse 8th Floor									
1970	17	15	14	13	13	11	9	6	5
1971	14	13	11	11	10	9	7	5	4
1972	11	10	10	9	9	8	6	5	3
La Habra									
1965	25	22	20	19	17	14	10	7	4
1966	25	21	20	19	18	15	11	8	6
1967	26	25	23	21	20	14	10	6	4
1968	30	27	23	23	21	17	13	9	6
1969	31	28	20	20	19	14	10	7	4
1970	17	16	15	14	12	11	8	5	3
1971	28	24	23	22	21	17	11	8	6
1972	28	23	21	20	19	17	11	8	6
Lennox									
1965	36	34	30	27	25	21	16	10	7
1966	32	28	28	26	25	22	15	10	7
1967	63	55	50	48	41	30	20	13	8
1968	46	39	36	34	33	28	18	12	8
1969	30	27	26	25	24	20	15	11	7
1970	35	31	30	28	27	24	16	12	8
1971	35	33	32	30	28	24	16	11	8
1972	35	32	28	26	24	21	15	10	7
Long Beach									
1963	38	34	30	29	27	23	15	9	6
1964	31	29	26	25	25	20	15	10	6
1965	39	32	30	28	26	20	14	9	6
1966	30	29	27	26	24	20	15	10	7
1967	40	36	35	31	30	26	18	13	7
1968	45	42	41	39	38	32	20	18	9
1969	41	35	34	33	32	26	18	12	9

TABLE 5-10 - Continued

	Concentrations Equalled or Exceeded by Stated Percent of Observations								
	1%	2%	3%	4%	5%	10%	25%	50%	75%
Long Beach - cont.d									
1970	40	38	36	32	30	25	19	13	9
1971	41	36	34	31	27	24	15	10	7
1972	39	32	31	30	28	24	16	11	8
Los Angeles-Downtown									
1963	43	37	32	31	30	24	17	11	7
1964	44	34	33	29	26	22	15	10	6
1965	47	38	36	34	33	27	20	12	7
1966	44	40	35	32	31	28	20	13	10
1967	44	40	38	34	33	27	16	11	7
1968	40	37	35	32	30	25	15	11	8
1969	33	27	27	25	24	20	13	9	7
1970	53	40	40	36	32	26	19	12	8
1971	56	46	45	40	37	31	22	14	10
1972	37	35	34	33	32	26	18	13	9
Mission Valley									
1966	34	30	26	24	21	14	9	6	3
1967	15	13	13	12	12	9	6	4	3
1968	22	17	15	15	14	11	8	6	4
1969	20	18	15	15	15	11	8	6	4
1970	15	13	13	11	11	9	6	4	3
1971	32	26	23	20	20	15	10	7	5
1972	24	22	21	19	19	16	11	6	4
Monterey APCD									
1968	13	9	9	8	8	6	4	2	2
1969	10	9	9	8	8	6	5	4	2
1970	11	10	9	8	7	6	4	3	2
1971	10	9	8	7	7	6	4	3	2
1972	8	7	6	6	6	5	4	3	2

TABLE 5-10 - Continued

	Concentrations Equalled or Exceeded by Stated Percent of Observations								
	1%	2%	3%	4%	5%	10%	25%	50%	75%
Oakland									
1963	26	17	15	14	14	12	8	5	4
1964	31	24	22	19	18	14	8	5	4
1965	17	16	15	15	14	11	7	5	4
1966	25	19	18	17	16	11	8	5	4
1967	25	21	20	18	17	14	10	6	4
1968	20	17	16	15	14	12	8	6	4
Oakland-Jackson									
1969	21	18	16	15	14	12	8	5	4
1970	22	19	18	18	16	13	8	6	4
1971	20	19	16	14	14	12	8	6	4
1972	18	16	15	14	13	10	7	5	4
Pasadena									
1968	42	39	37	37	36	32	22	14	8
1969	40	36	32	29	27	22	17	13	9
1970	50	47	45	40	37	28	21	15	11
1971	42	38	33	32	30	25	17	12	9
1972	41	40	35	34	30	28	20	14	10
Pasadena-Villa St.									
1963	30	27	24	22	21	17	13	8	6
1964	26	24	22	21	20	18	14	10	7
1965	27	25	24	22	22	19	14	9	6
1966	28	24	22	22	22	17	13	9	7
1967	30	27	26	25	24	20	15	11	7
1968	26	24	20	20	19	15	13	10	7
Pomona									
1965	27	25	23	22	21	20	14	10	7
1966	26	22	22	21	21	17	14	10	7
1967	28	26	26	23	22	19	15	11	8
1968	38	35	30	29	27	22	16	12	9

TABLE 5-10 - Continued

	Concentrations Equalled or Exceeded by Stated Percent of Observations								
	1%	2%	3%	4%	5%	10%	25%	50%	75%
Pomona - cont'd.									
1969	35	27	26	25	23	21	16	12	9
1970	31	30	29	27	26	22	18	13	10
1971	35	32	28	27	24	21	16	11	8
1972	30	27	26	24	22	20	15	11	8
Redlands									
1968	19	16	11	11	10	8	6	4	3
1969	9	9	8	8	8	7	4	3	2
1970	19	17	16	15	14	11	8	6	4
1971	22	20	18	17	16	13	11	8	5
1972	18	16	16	15	14	13	10	7	4
Redwood City									
1967	23	21	18	18	16	12	8	5	4
1968	18	14	13	12	12	10	6	4	3
1969	22	21	20	17	16	13	8	5	4
1970	17	16	14	14	13	11	8	5	4
1971	17	13	13	12	11	9	6	4	3
1972	12	12	11	11	10	9	6	5	4
Reseda									
1965	28	25	23	21	21	17	12	8	5
1966	29	26	24	22	22	17	13	10	6
1967	40	33	30	28	27	21	15	10	6
1968	33	30	26	26	24	20	15	10	7
1969	26	23	22	21	20	19	14	10	7
1970	33	31	28	28	27	22	17	12	7
1971	34	30	29	27	26	22	16	12	8
1972	35	29	28	26	25	21	17	12	8
Richmond									
1966	14	12	11	11	10	9	6	4	3
1967	15	14	13	12	11	9	7	6	4

TABLE 5-10 - Continued

	Concentrations Equalled or Exceeded by Stated Percent of Observations								
	1%	2%	3%	4%	5%	10%	25%	50%	75%
Richmond - cont'd.									
1968	16	15	14	12	12	10	8	6	4
1969	19	17	14	14	14	11	8	5	4
1970	17	15	14	13	12	9	7	6	4
1971	12	9	9	8	7	6	5	4	3
1972	8	8	8	7	7	6	5	4	4
Riverside-11th St. Mall									
1963	26	24	23	23	22	19	14	9	6
1964	32	29	28	22	20	16	11	7	5
1965	37	34	31	28	25	19	12	8	5
1966	22	20	19	18	17	13	10	7	4
1967	23	20	19	19	17	15	12	8	5
1969	31	23	22	22	21	18	13	8	6
1970	23	22	20	20	19	15	10	7	4
1971	25	24	21	20	19	15	12	8	5
1972	14	13	11	11	11	10	7	5	4
Sacto-1025 P St.									
1972	12	11	11	10	9	8	6	4	3
Sacto-13th and J St.									
1963	20	19	17	16	15	13	10	6	3
1964	25	20	17	16	16	14	8	6	4
1965	22	20	17	16	14	11	8	6	4
1966	17	15	14	12	10	8	6	4	3
1967	23	16	16	15	14	12	8	5	4
1968	14	11	10	10	9	8	6	4	3
1969	17	14	13	12	12	10	7	5	4
1970	14	12	12	11	11	8	6	4	3
1971	13	12	11	10	10	8	6	4	3
Salinas-Alisal									
1966	11	10	10	9	8	7	5	4	3
1967	8	7	6	6	6	5	4	3	2

TABLE 5-10 - Continued

	Concentrations Equalled or Exceeded by Stated Percent of Observations								
	1%	2%	3%	4%	5%	10%	25%	50%	75%
Salinas-Alisal - cont'd.									
1968	8	8	7	7	7	6	4	3	2
1969	9	7	7	7	7	6	5	4	3
1970	9	8	7	7	7	6	5	4	3
1971	9	9	8	8	8	6	5	4	3
1972	10	9	9	7	7	6	5	4	3
San Bernardino									
1963	12	11	10	10	9	7	5	2	1
1964	4	4	4	3	3	2	1	1	0
1965	11	10	9	9	9	8	7	5	4
1966	21	18	18	16	16	12	9	7	5
1967	22	21	20	19	18	16	12	8	5
1968	19	16	15	13	13	10	8	6	4
1969	19	16	16	14	13	12	9	7	5
1970	22	20	17	16	16	14	11	9	6
1971	23	22	21	20	18	15	12	8	6
1972	21	19	17	16	15	14	10	8	6
San Diego-8th and E St.									
1963	25	21	16	15	14	11	7	5	3
1964	22	16	15	14	14	9	6	3	2
1965	26	23	18	15	13	9	6	3	2
1966	26	21	19	19	18	12	8	6	4
1967	20	17	15	14	12	8	6	3	2
1968	23	20	18	15	14	11	7	5	3
1969	17	16	12	12	11	8	5	3	2
1970	15	13	11	11	11	8	6	3	2
1971	35	29	23	20	18	14	8	6	4
1972	20	18	16	15	14	10	7	5	3
San Francisco-Ellis St.									
1968	31	27	27	26	23	16	9	7	6
1969	23	22	20	17	17	13	8	6	4

TABLE 5-10 - Continued

	Concentrations Equalled or Exceeded by Stated Percent of Observations								
	1%	2%	3%	4%	5%	10%	25%	50%	75%
San Francisco-Ellis St. - cont'd.									
1970	29	25	21	19	17	13	9	6	5
1971	16	15	14	11	10	8	6	4	3
1972	18	14	13	12	12	8	6	5	4
San Jose-5th St.									
1964	34	29	25	23	21	18	11	7	5
1965	23	23	20	20	18	15	12	10	7
1966	23	22	20	20	18	14	9	6	5
1967	23	22	20	20	19	16	11	7	5
1968	23	22	20	17	16	13	8	6	4
1969	20	18	16	16	15	13	8	5	4
1970	14	13	13	13	13	12	8	5	4
San Jose-West Alma Ave.									
1970	19	18	14	13	13	10	7	5	3
1971	20	16	16	15	14	11	8	5	4
1972	18	16	13	13	13	12	7	5	4
Stockton-Hazelton St.									
1964	18	17	16	15	12	10	6	4	3
1965	12	10	9	8	7	6	5	3	2
1966	12	10	9	8	7	6	5	3	2
1967	13	12	11	10	8	7	6	4	3
1968	14	14	13	12	11	9	8	6	4
1969	18	18	18	17	16	14	10	5	4
1970	12	10	10	9	8	6	4	3	2
1971	13	11	9	8	7	6	5	4	3
1972	10	9	8	7	7	6	5	4	3
West Los Angeles									
1963	37	34	31	28	27	21	13	8	5
1964	33	30	28	27	24	19	13	9	6
1965	45	40	36	34	32	26	18	10	7
1966	40	34	29	28	27	22	15	10	7

TABLE 5-10 - Continued

	Concentrations Equalled or Exceeded by Stated Percent of Observations								
	1%	2%	3%	4%	5%	10%	25%	50%	75%
West Los Angeles - cont'd.									
1967	38	34	31	30	28	23	15	10	7
1968	43	39	36	33	32	27	18	12	9
1969	33	29	27	26	25	22	15	10	7
1970	42	35	32	30	30	23	17	11	8
1971	46	38	36	35	31	27	18	12	8
1972	38	33	31	29	29	23	16	11	7

[a] From State of California Air Resources Board, 1974.

TABLE 5-11

Nitrogen Dioxide Concentrations by Various Methods, 1972, for Air Quality Control Regions (AQCR) Originally Classified Priority 1[a]

Region	Nitrogen Dioxide Average Concentration for Period of Operation, $\mu g/m^3$, 25°C		
	FRM[b]	Arsenite[c]	Chemiluminescent
Atlanta	183	80	62
Baltimore	159	96	64
Boston	132	74	--
Chattanooga	125	53	38
Chicago[d]	238	117	121
Cincinnati	156	73	61
Cleveland	126	57	53
Columbus	149	68	52
Corpus Christi-Victoria	85	43	43
Dallas-Fort Worth	145	76	47
Dayton	158	64	53
Denver[d,e]	106	42	110
Detroit-Port Huron	180	80	60
Dubuque	70	30	23
Florida, Southeast (Miami)[f]	120	55	53
Florida, West Central (Tampa)	156	56	52
Four Corners	47	30	--
Genesee-Finger Lakes (Rochester)	98	48	26
Hampton Roads (Norfolk)	123	52	39
Hartford-New Haven-Springfield	125	82	73
Houston-Galveston	137	64	66
Indianapolis	107	61	56
Los Angeles	252	182	118
Louisville	184	87	68
Massachusetts, Central (Worcester)	120	71	--
Memphis	148	64	31
Michigan, Central (Grand Rapids)	127	59	44
Minneapolis-St. Paul	57	31	47
National Capital[d]	146	88	64
New York-New Jersey-Connecticut	182	100	65
Niagara Frontier (Buffalo)	76	32	49
Omaha-Council Bluffs	113	60	30
Pennsylvania, Central (Johnston)	--	25	64
Pennsylvania, South Central (Lancaster)	132	60	36
Pennsylvania, Southwest (Pittsburgh)	177	78	64
Pennsylvania-Upper Delaware Valley, Northeast (Reading)	158	52	60

TABLE 5-11 - continued

Region	Nitrogen Dioxide Average Concentration for Period of Operation, $\mu g/m^3$, 25°C		
	FRM[b]	Arsenite[c]	Chemiluminescent
Philadelphia	197	83	84
Phoenix-Tucson	159	80	69
Providence	98	45	--
Puget Sound (Seattle)	134	47	51
San Diego	136	63	76
San Francisco Bay Area[a]	193	85	84
St. Louis	123	79	58
State Capital (Richmond)	171	58	37
Toledo	139	54	38
Wasatch Front (Salt Lake City)	159	62	114
Wisconsin, Southeast (Milwaukee)	124	76	--

[a] On basis of earlier Federal Reference Method (FRM) determinations. From U.S. Environmental Protection Agency, 1973.[569]

[b] Federal Reference Method.

[c] Arsenite data are corrected to reflect 85% collection efficiency. Available data indicate that there is 95% confidence that the corrected measurements are within ±10% of actual NO_2 concentrations.

[d] All measurements at same site. In other AQCR's, all measurements were not made at the same site.

[e] Originally classified priority III.

[f] City names in parentheses are for identification only.

high levels of nitrogen oxides emissions result from the production of trinitrotoluene.

Observation sites included areas where high levels of pollutants were expected and control areas where near normal levels were anticipated. Nitric oxide and nitrogen dioxide were determined with the Saltzman method. Tables 5-12 and 5-13 show average hourly concentrations and maxima of nitric oxide and nitrogen dioxide for the various sites. Stations 5 and 27 were in downtown Chattanooga; Station 7 was in Rossville, Georgia. Stations 15, 17, 19, 20, 21, 161, and 201 were near the ammunition plant.

SALTS OF NITROGEN IN THE ATMOSPHERE

NASN has collected data on suspended particulate nitrate levels since 1958. Selected data from this source are presented in Table 5-14. Some earlier data were obtained using nitration of 2,4-xylenol [$(CH_3)_2C_6H_3OH$] as the analytical method, after collecting particulates on a high volume sampler. These data are underlined in the table. The remaining data were obtained using high volume sampling, diazotization, coupling with N-(1-naphthyl)ethylenediamine dihydrochloride ($C_{10}H_7NHCH_2CH_2NH_2 \cdot 2HCl$), and colorimetric determination.

Nitrates in Chattanooga Study

The Chattanooga study was designed to gather air quality information in the Chattanooga, Tennessee and Rossville, Georgia interstate areas, particularly as affected by the Volunteer Army Ammunition Plant at Tyner, Tennessee. The study included determinations of suspended nitrates, sulfates, and ammonium particulates (Table 5-15).

Stations 17, 19, 20, and 21 were closest to the ammunition plant. Nitrate concentrations at each station exceeded the NASN maximum station average for 1965 which was 13.5 $\mu g/m^3$. The reading at Station 19 was 48.9 $\mu g/m^3$, more than three times higher than that average.

Sampling means at five stations were selected for their ability to measure respirable particles (those under 5 μm diam) separately from the total sample collected by the high volume sampler (Table 5-16).

The data represent seasonal averages. Time periods during which observations were made were different for the various stations.

Individual analyses indicated the smaller, respirable portion contained higher concentrations of nitrate, sulfate, and ammonia than did the total sample of suspended particulates (Table 5-17).

Nitrate was analyzed by hydrazine reduction and diazotization; sulfate by the methylthymol blue method; and ammonia by the indophenol method.

Nitrates in California South Coast Air Basin Study

A recent study by the Air and Industrial Hygiene Laboratory of the California State Department of Health has determined nitrate and sulfate concentrations found in aerosols throughout California. A paper by Appel *et al.*[21] summarizes the data. The study attempts to define mechanisms which form the compounds which account for the concentrations, cation moieties, and particle sizes observed. Table 5-18 gives 24-hr average values for eight locations. Nitrate determination was made with a micro version of the 2,4-xylenol procedure.

The study indicated that most of the particulate nitrate and sulfate was in the form of ammonium salts. Nitrate concentrations tend to increase

TABLE 5-12

Hourly Average and Maximum Hourly Concentration of Nitric Oxide--Chattanooga Study[a]

Location	Operating Period	No. of Observations	Arithmetic Mean, $\mu g/m^3$, 25°C	Maximum Value, $\mu g/m^3$, 25°C
VAAP-Administration	5/68-11/68	4,611	74	1,350
VAAP-Pond	7/68-11/68	3,174	140	1,880
VAAP-Rod and Gun Club	9/67-11/68	10,678	99	2,640
VAAP-Harrison School	9/67-11/68	10,720	74	3,380
Golden Gateway	6/68-11/68	2,292	37	440
Hickory Valley	6/68-11/68	3,756	62	870

[a]From Helms *et al.*, 1970.

TABLE 5-13

Hourly Average and Maximum Hourly Concentration of Nitrogen Dioxide--Chattanooga Study[a]

Location	Operating Period	No. of Observations	Arithmetic Mean, $\mu g/m^3$, 25°C	Maximum Value, $\mu g/m^3$, 25°C
Post Office	12/67- 3/68	1,823	75	300
Rossville	4/68-11/68	5,044	75	490
B.T. Washington	12/67-11/68	7,465	150	340
E. Brainerd	12/67- 3/68	2,126	56	880
VAAP-Administration	2/68-11/68	6,970	170	4,600
VAAP-Pond	5/68-11/68	4,717	400	7,150
VAAP-Rod and Gun Club	9/67-11/68	10,607	260	7,150
VAAP-Harrison School	9/67-11/68	10,724	170	5,000
Golden Gateway	4/68-11/68	3,741	75	1,670
Hickory Valley	4/68-11/68	5,136	150	2,920
PHS Rod and Gun Club	12/67- 5/68	2,615	300	5,650

[a]From Helms *et al.*, 1970.[219]

TABLE 5-14

Nitrate Ion. Annual Mean and Maximum Concentrations, $\mu g/m^3$, 25°C, Sampling Interval; 24 hr.[a]

(Underlined numbers indicate data collected by nitration of 2,4-xylenol. The other data were obtained by high volume sampling, diazotization, coupling with N-(1-naphthylethylenediamine dihydrochloride, and colormetric determination.)

Location	1958		1959		1960		1961		1962		1963		1964		1965		1966		1967		1968		1969		1970	
	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max
Alabama																										
Birmingham	--	--	2.47	5.50	2.23	5.90	2.13	4.80	2.11	3.90	2.06	4.00	2.35	5.30	2.79	5.10	--	--	--	--	--	--	--	--	--	--
Gadsden	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	1.48	2.40	--	--	2.72	7.70	1.23	3.30	2.38	6.60
Alaska																										
Anchorage	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	0.77	3.20	0.60	1.10	0.71	1.70	0.73	6.50
Arizona																										
Phoenix	--	--	--	--	--	--	2.36	6.50	2.40	7.40	2.90	5.00	2.99	5.60	2.88	6.30	2.59	6.00	2.89	8.40	3.82	9.50	2.70	6.10	--	--
Tucson	--	--	--	--	--	--	1.55	3.40	1.28	2.40	--	--	1.72	2.90	1.21	2.40	1.32	2.60	1.68	3.20	1.65	3.10	2.19	4.40	2.46	5.10
Arkansas																										
Little Rock	--	--	1.20	2.60	0.91	2.40	1.01	2.70	1.25	4.90	--	--	--	--	--	--	1.83	3.00	1.81	3.00	2.23	4.60	1.34	4.20	2.27	4.30
California																										
Los Angeles	--	--	6.81	16.40	8.48	15.20	7.45	16.00	10.19	24.70	9.72	21.60	--	--	13.51	25.40	5.20	10.30	4.26	8.00	9.22	20.50	6.64	23.90	12.56	24.60
Oakland	3.58	13.70	--	--	--	--	--	--	3.12	8.00	3.41	7.90	--	--	--	--	2.28	6.70	4.74	11.30	3.03	9.70	3.37	7.80	3.93	10.20
San Diego	4.13	8.30	5.09	19.10	4.10	10.00	5.77	13.30	5.34	14.60	4.87	13.00	4.75	10.30	5.30	10.50	2.72	6.30	3.10	8.30	4.02	10.10	3.93	9.60	5.36	17.10
San Francisco	--	--	2.69	11.60	2.25	7.70	--	--	--	--	--	--	2.39	8.20	--	--	1.97	8.00	3.47	10.50	3.91	12.30	2.22	6.20	2.76	11.20
Colorado																										
Denver	3.92	13.00	2.26	7.70	2.04	7.80	1.80	8.00	1.72	7.80	3.24	22.00	2.41	10.00	4.16	12.70	1.44	5.80	1.91	4.50	2.30	6.00	3.42	12.10	3.09	14.90
Connecticut																										
Bridgeport	--	--	--	--	1.34	4.70	--	--	1.31	4.70	--	--	--	--	--	--	--	--	--	--	--	--	1.19	6.20	1.70	5.80
Hartford	2.12	5.20	1.00	3.40	1.87	5.50	1.12	2.50	1.70	4.90	2.30	9.40	2.72	6.70	3.32	9.40	1.21	2.80	2.72	17.50	1.11	3.20	0.68	2.10	1.23	3.70
New Haven	1.81	5.60	0.91	2.80	1.32	4.70	1.29	3.40	1.64	3.60	2.13	8.40	2.77	7.20	2.80	8.20	1.07	2.80	1.01	3.30	1.29	3.60	0.58	1.80	1.38	6.10
Delaware																										
Wilmington	--	--	--	--	2.27	3.80	2.23	6.70	2.05	4.10	2.38	8.20	3.42	6.70	3.15	6.60	1.54	3.80	2.06	6.10	2.21	6.30	1.27	3.60	2.46	6.70
DC																										
Washington	3.10	5.40	--	--	2.37	7.10	1.82	4.40	1.36	2.40	2.38	5.60	1.96	5.50	3.47	7.20	1.82	3.90	1.90	3.80	2.01	4.50	2.04	3.70	--	--
Florida																										
Tampa	--	--	1.71	3.20	1.94	4.50	2.05	4.40	2.11	6.80	--	--	--	--	--	--	--	--	--	--	2.77	10.90	1.61	6.50	3.01	7.50
Georgia																										
Atlanta	--	--	2.18	5.40	2.12	5.10	--	--	--	--	--	--	--	--	--	--	1.74	4.40	2.28	5.20	2.09	4.60	1.59	4.10	2.62	4.80
Hawaii																										
Honolulu	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	0.97	8.50	0.71	2.10	0.63	2.80	0.70	2.20	0.51	1.60
Idaho																										
Boise City	1.51	6.50	1.34	4.40	1.07	2.20	1.36	3.10	1.30	3.80	--	--	1.72	4.90	2.01	5.30	0.99	2.80	0.98	1.80	1.05	3.50	1.20	5.20	1.34	4.30
Illinois																										
Chicago	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	1.69	4.50	1.51	6.30	2.68	8.40	1.99	5.40	2.65	6.60
Indiana																										
East Chicago	--	--	--	--	--	--	--	--	--	--	1.53	4.10	3.21	9.30	3.20	9.20	2.93	8.80	2.21	5.20	3.21	10.60	2.56	14.00	4.40	11.60
Hammond	--	--	--	--	--	--	1.89	6.00	--	--	2.59	8.10	--	--	--	--	--	--	2.76	8.40	2.82	8.60	2.38	11.30	3.62	10.10
Indianapolis	2.68	5.50	--	--	3.27	7.80	1.89	5.20	1.84	3.30	2.44	4.20	4.12	11.30	3.10	7.10	2.14	5.20	2.54	5.30	2.52	5.20	1.68	4.80	3.86	9.40
South Bend	--	--	--	--	--	--	--	--	--	--	2.25	5.40	--	--	2.87	6.30	1.45	3.20	1.45	5.10	3.17	10.70	1.05	3.40	3.53	10.00
Iowa																										
Des Moines	2.00	4.90	2.17	8.00	2.48	8.80	2.16	6.60	1.98	6.20	1.44	3.10	2.43	7.40	2.71	5.90	1.73	3.80	2.00	4.60	1.78	3.80	1.84	5.90	3.15	7.30
Kansas																										
Kansas City	--	--	--	--	1.87	5.00	--	--	1.81	6.20	--	--	--	--	--	--	--	--	--	--	2.32	3.90	2.09	10.10	2.83	7.10
Wichita	--	--	--	--	1.96	6.60	--	--	--	--	--	--	--	--	--	--	1.63	3.50	1.38	2.80	1.76	3.80	1.21	2.50	3.40	11.30
Kentucky																										
Louisville	--	--	--	--	--	--	--	--	--	--	--	--	--	--	3.82	6.70	2.19	6.90	2.12	6.10	3.80	8.60	1.98	4.00	--	--
Covington	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	1.40	3.70	1.29	3.80	1.60	4.70	1.41	3.10	3.02	7.70
Louisiana																										
New Orleans	2.20	4.50	1.45	4.90	2.42	6.10	3.16	8.10	2.13	4.60	2.60	4.40	3.45	5.60	3.39	6.80	2.20	7.00	3.08	7.10	4.09	7.00	2.69	5.90	3.85	13.80

Maine																										
Portland	—	—	0.64	1.40	0.56	1.70	0.52	2.00	1.02	5.80	0.94	3.20	1.21	4.50	—	—	—	—	—	—	0.79	2.60	1.17	3.10	—	—
Maryland																										
Baltimore	2.24	4.90	1.42	4.20	2.04	5.10	1.62	4.00	1.78	4.10	1.78	4.12	2.62	9.20	2.87	6.40	2.41	5.50	2.14	6.20	2.83	6.00	1.82	3.80	3.71	13.60
Massachusetts																										
Boston	2.05	6.20	0.88	2.40	—	—	0.94	2.20	0.61	1.80	—	—	2.29	6.50	—	—	—	—	—	—	1.40	5.10	0.99	4.60	—	—
Fall River	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1.00	3.20	1.99	6.30
Springfield	—	—	—	—	—	—	—	—	1.30	3.30	—	—	—	—	—	—	—	—	—	—	—	—	0.46	1.70	1.18	3.30
Worchester	—	—	1.29	3.30	—	—	—	—	—	—	1.41	4.30	—	—	—	—	—	—	—	—	1.10	4.00	0.62	2.30	1.81	4.00
Michigan																										
Detroit	—	—	2.20	5.20	2.48	6.00	2.30	9.30	1.83	9.50	2.09	7.40	5.00	10.00	3.43	8.00	3.72	9.30	2.75	6.30	4.70	13.80	3.32	10.90	4.45	9.60
Flint	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2.92	7.60	—	—	2.00	9.80	2.67	5.20	3.73	12.80	2.67	6.30
Grand Rapids	—	—	2.05	6.60	—	—	—	—	1.39	3.50	2.92	6.00	—	—	2.61	8.20	—	—	2.04	5.40	3.34	10.00	3.83	11.30	3.23	10.60
Minnesota																										
Minneapolis	1.79	8.30	1.59	4.40	1.28	3.10	—	—	1.61	4.40	1.20	3.70	1.55	3.90	1.83	5.70	1.01	2.20	1.69	5.20	1.41	2.80	1.50	6.50	2.77	7.70
St. Paul	—	—	—	—	—	—	—	—	—	—	1.42	5.10	—	—	—	—	1.49	6.30	1.91	6.60	1.96	5.30	1.11	3.90	2.57	6.40
Mississippi																										
Jackson	—	—	0.96	2.30	1.23	2.20	—	—	—	—	1.74	3.70	—	—	—	—	1.32	3.00	—	—	1.90	3.90	—	—	—	—
Missouri																										
Kansas City	—	—	1.62	3.30	1.61	4.20	1.58	3.00	2.47	9.40	1.58	3.80	2.03	5.40	2.45	4.40	1.88	3.40	2.10	4.40	2.69	6.30	1.89	5.30	3.04	8.40
St. Louis	2.47	4.80	1.89	4.40	2.15	6.50	1.86	5.10	1.71	4.00	1.72	3.60	2.44	7.70	2.58	5.20	1.69	3.40	1.54	3.70	—	—	3.24	9.20	—	—
Montana																										
Helena	—	—	0.32	0.70	0.41	1.00	0.59	1.50	0.49	1.00	0.61	2.80	—	—	—	—	0.47	2.00	0.93	2.90	1.20	6.30	0.62	1.60	—	—
Nebraska																										
Omaha	2.24	4.90	1.38	3.70	2.22	5.80	2.01	11.40	1.59	5.50	1.49	4.00	2.33	8.20	2.38	6.20	1.84	6.00	1.70	3.30	2.16	5.20	1.96	7.20	—	—
Nevada																										
Las Vegas	—	—	—	—	—	—	—	—	—	—	—	—	2.34	5.40	2.77	7.70	1.50	4.30	1.80	3.50	2.00	3.90	1.91	3.60	—	—
Reno	—	—	—	—	—	—	—	—	—	—	1.27	4.90	—	—	2.61	6.10	—	—	1.69	12.80	1.58	6.20	1.57	6.40	—	—
New Hampshire																										
Concord	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1.47	3.20	0.26	1.00	0.46	1.80	0.74	2.30	0.37	2.10	1.05	4.40
New Jersey																										
Camden Co.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1.37	10.10	0.92	2.00	1.28	3.00	2.08	5.50	2.48	6.40
Glassboro	—	—	—	—	—	—	—	—	—	—	—	—	2.92	6.40	3.02	6.00	1.13	2.60	0.97	2.10	1.58	4.70	1.13	2.90	2.17	7.30
Jersey City	1.52	5.60	—	—	—	—	2.15	15.50	—	—	—	—	—	—	—	—	1.27	3.70	1.45	3.90	2.01	5.90	1.31	2.80	2.13	5.10
Newark	—	—	1.19	2.60	1.62	3.00	1.10	3.00	1.70	4.30	2.15	7.20	3.25	7.30	2.50	6.10	1.40	2.40	1.87	6.40	1.66	3.00	1.27	2.50	2.77	11.10
Paterson	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1.90		1.23	2.70	1.83	5.19	0.96	2.50	2.21	4.30
New Mexico																										
Albuquerque	1.31	2.90	1.69	4.50	1.85	3.40	—	—	—	—	—	—	—	—	—	—	1.30	3.30	1.82	5.30	1.53	4.30	1.70	5.70	1.44	2.90
New York																										
Binghamton	—	—	—	—	1.17	2.90	1.28	2.90	1.21	2.50	1.15	2.60	1.76	6.10	1.37	3.30	—	—	—	—	—	—	—	—	—	—
New York City	2.18	7.40	—	—	1.51	3.00	1.78	6.00	1.44	4.30	2.95	6.80	2.17	5.40	2.39	6.00	1.84	6.00	1.37	3.10	—	—	1.28	2.60	2.74	7.10
Rochester	—	—	—	—	1.91	5.20	1.68	5.10	0.89	2.40	1.77	5.00	—	—	—	—	—	—	—	—	—	—	1.76	5.10	3.74	9.10
North Dakota																										
Bismarck	—	—	0.57	1.70	0.79	2.90	0.81	1.50	0.66	2.10	0.52	1.20	0.54	1.10	—	—	—	—	—	—	0.71	2.20	1.20	5.30	1.00	3.00
Ohio																										
Akron	—	—	—	—	2.42	6.60	1.98	4.00	2.31	6.00	2.45	6.50	3.05	7.60	3.32	6.90	—	—	—	—	—	—	—	—	—	—
Cincinnati	—	—	—	—	2.11	6.60	1.82	3.00	1.51	3.90	2.22	7.00	3.65	7.30	3.40	6.20	2.13	6.10	1.75	4.30	2.52	6.20	3.55	6.60	3.48	6.60
Cleveland	3.28	9.20	1.67	4.30	1.92	5.10	1.91	4.60	1.03	3.20	2.84	5.30	2.85	5.10	2.59	7.00	1.35	4.00	1.61	4.40	2.64	7.20	2.69	6.80	2.94	9.00
Columbus	3.48	7.00	2.26	5.30	2.27	5.00	2.50	5.30	2.36	6.40	2.65	7.40	4.65	23.20	3.77	8.80	2.07	5.30	1.88	4.30	2.55	7.20	3.77	8.50	3.58	8.70
Dayton	—	—	2.00	9.00	—	—	—	—	—	—	—	—	—	—	—	—	2.18	9.10	1.67	4.40	2.39	5.50	3.61	7.80	3.07	10.40
Toledo	—	—	2.15	5.30	—	—	—	—	2.54	7.50	2.48	7.20	4.32	9.20	—	—	1.75	6.70	2.28	6.40	2.86	7.90	1.71	4.40	3.56	8.10
Youngstown	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2.35	6.30	2.10	5.70	2.80	5.00	2.15	7.20	3.69	8.00
Oklahoma																										
Oklahoma City	—	—	—	—	—	—	1.42	4.00	—	—	—	—	—	—	2.51	4.80	1.83	4.20	1.65	3.10	1.61	3.60	1.62	3.30	2.70	8.90
Tulsa	—	—	—	—	—	—	—	—	1.66	5.80	1.43	2.40	—	—	2.17	4.10	1.49	3.00	1.36	3.30	1.73	5.20	2.88	10.80	2.39	6.40
Oregon																										
Portland	3.97	21.80	2.81	13.80	3.17	16.80	4.09	14.70	—	—	8.78	39.70	6.78	21.10	7.42	27.50	3.17	11.80	3.56	6.30	4.67	11.40	3.24	8.10	4.81	13.00
Pennsylvania																										
Allentown	—	—	3.43	9.20	—	—	—	—	—	—	3.07	7.60	—	—	—	—	—	—	3.25	11.90	3.45	9.00	5.21	10.80	5.89	19.20
Bethlehem	—	—	—	—	—	—	—	—	—	—	3.10	10.20	—	—	—	—	—	—	3.90	12.90	3.97	8.90	2.32	7.20	3.56	10.00
Philadelphia	2.78	6.70	—	—	1.74	4.00	2.83	9.30	2.72	10.00	2.62	6.50	2.67	6.50	3.04	7.60	2.05	3.60	1.83	7.50	2.28	4.20	—	—	3.63	6.40
Pittsburgh	2.65	5.80	2.80	7.80	2.63	5.80	2.38	6.60	2.74	6.10	2.94	8.50	3.04	5.10	2.88	5.40	2.13	4.60	1.96	4.80	3.03	7.10	—	—	3.00	7.00
Reading	1.95	7.10	—	—	1.86	5.10	—	—	1.59	8.20	—	—	—	—	—	—	2.12	9.40	2.76	21.60	3.00	6.30	—	—	3.56	16.70
York	—	—	3.34	10.60	—	—	3.42	12.80	—	—	4.13	10.40	—	—	—	—	—	—	2.54	8.80	3.26	5.60	—	—	3.59	7.40

TABLE 5-14 - continued

Location	1958 Mean	1958 Max	1959 Mean	1959 Max	1960 Mean	1960 Max	1961 Mean	1961 Max	1962 Mean	1962 Max	1963 Mean	1963 Max	1964 Mean	1964 Max	1965 Mean	1965 Max	1966 Mean	1966 Max	1967 Mean	1967 Max	1968 Mean	1968 Max	1969 Mean	1969 Max	1970 Mean	1970 Max
Puerto Rico																										
Bayamon	--	--	--	--	--	--	--	--	--	--	--	--	--	--	0.81	1.90	0.58	1.80	0.93	2.00	1.58	2.70	--	--	1.47	2.40
Guaynilla	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	0.28	0.90	0.56	0.90	0.83	2.40	0.72	1.50	1.19	2.60
Ponce	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	0.39	1.60	0.78	1.70	1.68	3.60	0.82	1.50	1.01	2.30
Rhode Island																										
Providence	2.55	5.40	1.10	2.50	1.83	4.60	0.93	2.80	1.25	2.70	--	--	2.52	5.30	2.42	5.00	1.28	4.80	1.13	4.40	2.26	5.00	1.02	2.90	1.21	3.30
Tennessee																										
Memphis	1.81	4.10	--	--	--	--	2.28	4.60	2.78	5.00	--	--	--	--	--	--	1.94	6.00	1.89	6.30	2.84	5.10	1.17	4.50	2.78	5.90
Nashville	3.30	10.10	2.50	5.50	2.59	6.00	2.94	10.00	2.22	3.90	1.97	3.80	2.61	5.10	3.13	5.50	2.15	4.40	1.96	4.70	2.94	5.50	1.41	3.40	2.20	4.50
Texas																										
Houston	2.85	6.30	--	--	2.61	6.90	2.17	4.40	2.13	3.90	2.49	6.00	3.00	6.50	3.06	11.10	2.65	7.20	3.20	9.10	3.62	9.70	3.34	7.20	3.55	7.30
San Antonio	--	--	--	--	2.23	6.00	--	--	--	--	2.28	3.60	2.23	5.60	--	--	1.88	3.50	2.72	7.20	2.95	5.40	1.79	3.60	2.02	4.60
Utah																										
Salt Lake City	2.22	7.70	2.93	11.40	2.73	9.10	--	--	1.70	5.30	2.88	8.00	2.69	9.40	2.30	8.70	1.37	3.50	1.04	2.70	2.54	7.70	2.39	15.20	2.31	4.30
Vermont																										
Burlington	--	--	1.31	4.40	1.53	6.40	1.15	3.30	1.45	6.90	--	--	3.58	16.40	--	--	1.09	2.60	--	--	1.09	3.50	1.50	4.00	1.30	4.70
Virginia																										
Norfolk	2.38	4.80	1.26	2.70	1.18	2.70	1.25	3.50	1.99	4.70	2.12	4.50	2.27	4.50	2.98	6.70	1.66	4.40	1.94	5.40	2.25	5.10	1.92	7.00	2.35	5.70
Richmond	--	--	1.27	2.40	--	--	1.41	2.90	--	--	2.34	4.90	--	--	--	--	--	--	1.18	2.80	1.47	3.50	1.28	7.90	1.74	3.40
Charleston	2.10	4.10	--	--	1.14	3.70	--	--	0.73	2.10	0.91	2.00	1.54	3.80	1.48	3.40	1.10	2.80	1.48	4.80	1.32	4.00	1.32	7.30	1.81	4.90
Washington																										
Seattle	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	1.41	2.50	1.73	3.60	1.70	5.60	1.29	2.80	1.88	5.60
Wisconsin																										
Milwaukee	2.25	6.10	--	--	2.05	9.20	--	--	--	--	1.91	6.90	3.00	9.80	2.61	7.10	2.18	6.50	2.43	8.20	4.65	17.90	2.76	12.00	3.32	8.70
Wyoming																										
Cheyenne	0.90	1.80	0.53	1.00	--	--	0.83	7.70	0.60	1.80	--	--	0.65	1.90	0.74	2.20	0.67	1.10	0.74	1.60	0.78	2.40	1.04	4.50	0.76	3.30

[a] From National Aerometric Data Bank, 1975.

TABLE 5-15

Suspended Sulfate, Nitrate, and Ammonium Particulates--Chattanooga Study[a]

Location	No. of Observations	Nitrates, $\mu g/m^3$		Sulfates, $\mu g/m^3$		Ammonium, $\mu g/m^3$	
		Max	Mean	Max	Mean	Max	Mean
WDEF-TV	92	5.6	2.5	21.3	9.2	5.5	1.1
Post Office	82	7.5	2.4	24.6	8.7	4.6	1.2
Rossville	85	11.9	2.7	22.2	9.1	2.9	0.7
Lovell Field	81	21.4	3.8	54.8	10.6	14.4	1.1
VAAP-Administration	37	57.8	19.9	60.6	18.7	16.6	4.5
VAAP-Pond	38	377.6	48.9	36.4	18.2	110.3	17.6
VAAP-Rod and Gun Club	39	232.2	37.4	35.1	17.3	64.6	12.1
VAAP-Harrison School	39	94.1	13.2	61.7	21.7	35.1	6.8

[a]From Helms *et al.*, 1970.

TABLE 5-16

Concentrations of Suspended Particulates Estimated as Respirable--Chattanooga Study[a]

Station Location	No. of Observations	Suspended Particulate, $\mu g/m^3$ Total	Suspended Particulate, $\mu g/m^3$ Respirable	Respirable, %
WDEF-TV	100	108	49	45
Post Office	47	104	44	42
Rossville	58	118	42	36
Lovell Field	20	73	29	40
PHS Rod and Gun Club	47	75	56	75

[a]From Helms *et al.*, 1970.[219]

TABLE 5-17

Analysis of Selected Dust Samples--
Chattanooga Study[a]

Station	Total, $\mu g/m^3$ Respirable Dust	Total, $\mu g/m^3$ Suspended Particulate	Total, % Nitrate	Total, % Sulfate	Total, % Ammonia
WDEF-TV		106.2	2.4	8.7	1.0
	48.8		3.3	18.6	2.9
Post Office		110.0	2.2	8.7	1.1
	44.7		4.2	20.4	4.0
Rossville		117.9	2.3	7.7	0.6
	42.7		2.8	19.7	2.3

[a]From Helms *et al.*, 1970.[219]

TABLE 5-18

Aerosol Characterization Study (ACHEX) Data for Nitrate and Sulfate in the South Coast Basin, California, 1972-1973, 24-hr Average Values[a,b]

Sampling Location	Episodes, No.	NO_3^-		SO_4^-	
		$\mu g/m^3$	Mass, %	$\mu g/m^3$	Mass, %
Harbor Freeway	2	6.3	5.6	5.7	5.1
Dominguez Hills	2	4.1	3.1	17.8	13.6
Pasadena	6	6.5	9.0	7.0	9.7
West Covina	5	8.2	4.5	21.8	12.0
Pomona (1972)	5	19.8	14.6	9.6	7.1
Pomona (1973)	2	7.9	4.8	11.5	7.0
Rubidoux	3	31.5	12.5	10.7	4.2
Riverside	9	15.2	13.4	7.7	6.8

[a] Analyses on hi-vol samples collected on Whatman-41 filters during July-October periods.

[b] From Appel *et al.*, 1974.[21]

during west to east moves across the basin while average particle size decreased with this movement. Maximum concentrations of nitrate tend to correspond with maximum NO_x values.

SUMMARY

Despite the loss of data resulting from disqualification of the modified Jacobs-Hochheiser method, there is a sizeable body of reliable information on nitric oxide and nitrogen dioxide concentrations. Most of these data were generated by the continuous colorimetric Saltzman method. This method has correlated quite well with the instrumental chemiluminescence method, which is not subject to major interference, and with the 24-hr arsenite method of Christie *et al*.[87]

Data on nitrogen dioxide and nitric oxide levels have been collected by the NASN, CAMP stations, and the California Air Resources Board network. In general, these data provide mean hourly average and maximum hourly average concentrations. EPA data on nitrogen dioxide led to reclassification of 43 regions from Priority I to Priority III [below 110 $\mu g/m^3$ (58.5ppm) annual arithmetic mean].

In contrast to pollutants showing a decreasing or stable trend, total emissions of oxides of nitrogen continue to increase. This is reflected in concentrations observed at the CAMP stations, where a general upward trend is evident. At five CAMP stations, the average NO_x concentration for 1967 through 1971 was 9% higher than for 1962 through 1966.

Nitrate concentrations have been observed by the NASN and the 1967-1968 Chattanooga study. The Chattanooga study recorded nitrate levels up to 10 times the national average.

Current concern with pollution of the stratosphere has resulted in recent determinations of its nitric oxide and nitrogen dioxide concentrations. Nitric oxide in the stratosphere probably results from oxidation of nitrous oxide, which is present in the stratosphere at 0.20 ppm.

Several mathematical models are of value in predicting maximum pollutant levels on a short-term basis, in deploying mobile monitors, and in estimating the impact to be expected from major new pollutant sources.

CONCLUSIONS

An appreciable amount of data must be discarded because of questionable accuracy of the methods used to measure nitric oxide and NO_x. However, sufficient accredited data exist to support the following conclusions:

1. Highest average concentrations of oxides of nitrogen are found in heavily populated, industrialized urban areas. (Localized sources such as munitions plants are excepted.)
2. These concentrations show a generally increasing trend between 1962 and 1971.
3. The current national primary air quality standard of 100 $\mu g/m^3$ (0.05 ppm) nitrogen dioxide is consistently exceeded in the Los Angeles area and, if present trends continue, will soon be exceeded in Chicago and Philadelphia areas.
4. Physico-chemical models of pollutant distribution, based on analytical data, are of value in predicting maximum concentration values of NO_x which will be reached under a given set of conditions for localized areas. Data from these models can be used to give warning of impending "alert" conditions.

5. Relationships between nitrogen dioxide and nitrate concentrations, and the types of nitrate formed in different areas, are currently under study.

RECOMMENDATIONS

1. Continuing increase in NO_x levels in regions of high population density necessitates the speedy and precise determination of the short- and long-term effects of this pollutant. This information is basic in determining whether more, or fewer, monitoring stations are required.
2. Marked variations in local concentrations over short periods create the necessity of continuous monitoring in regions of high NO_x concentrations.
3. Stratospheric data on nitric oxide, nitrogen dioxide, and nitrous oxide concentrations are incomplete, and chemical interactions occurring in this region are not precisely known. Therefore, additional study is required. Effects at the surface of the earth of stratospheric pollutants also require further investigation.
4. The relationship between nitrogen dioxide and nitrate concentrations under various meterological and geographical conditions requires further study. The health hazard of nitrogen dioxide and nitrates, and synergistic effects between these pollutants and sulfates in the atmosphere, need further evaluation.

CHAPTER 6

CHEMICAL INTERACTIONS OF NITROGEN OXIDES IN THE ATMOSPHERE

Solar radiation triggers a series of reactions in the atmosphere between gaseous organic molecules and nitrogen oxides. This produces a wide variety of secondary pollutants. The totality of primary and secondary pollutants involved in these photochemical reactions is known as photochemical smog. The major chemical characteristic of this mixture is its oxidative nature in contradistinction to sulfur oxide pollutants which are of a reductive nature.

Secondary pollutants, photochemically produced in the atmosphere from primary pollutants, create unique analytical problems that cannot be solved by conventional techniques: there are no point sources to sample; many significant intermediate products are present in such small concentrations that their presence can only be hypothesized; and many intermediate products are very transient. Despite these problems these intermediates must be qualitatively and quantitatively identified so hazards can be evaluated, limits established, and controls developed.

This chapter identifies the intermediates primarily by chemical and kinetic modeling, relating laboratory studies to atmospheric observations. Starting with the relatively innocuous materials, nitric oxide and simple hydrocarbons, information is advanced on the complex photochemical interactions eventually explaining the diurnal genesis of smog and the potential sinks and end products. The oxidizing intermediates, i.e., nitrogen dioxide

(NO_2) and peroxyacylnitrates (PAN's), responsible for eye irritation, adverse respiratory effects, plant damage, and browning of the atmosphere, presumably end as nitric acid incorporated into aqueous aerosol agglomerations as nitrate salts. Many of the organic molecules present as primary pollutants or their oxidation products polymerize as they mature forming organic aerosols which reduce visibility and also brown the atmosphere. These are also eventually incorporated into aqueous aerosol agglomerations and scrubbed from the atmosphere. Tables in this chapter indicate the intermediates, their calculated concentrations, and their theoretical first half-lives to facilitate future evaluations of possible health effects.

Demerjian *et al.*[105] developed a chemical reaction mechanism after a thorough study and evaluation of all available, related kinetic and mechanism studies. This mechanism was tested and improved through the use of a great variety of smog chamber data from many research groups. This was not the only effort in this area,[74,75,102-104,135,136,162,217,396,472,589,598] but represents the most complete chemical system developed to date. Any such complex chemical scheme, no matter how complete and accurate its reaction details, must be cautiously used. No model today can legitimately predict quantitatively the chemical and physical changes occuring in the atmosphere, since certain key information related to many reactants in the system is still lacking. However, the main features of this model should be qualitatively correct and should provide a reasonable basis for the consideration of detailed atmospheric interactions.

CHEMICAL INTERACTIONS IN THE SUNLIGHT-IRRADIATED, NO_x-HYDROCARBON-POLLUTED ENVIRONMENT

Data obtained on a smoggy day in Los Angeles,[556] indicate that the level of nitrogen oxides rises during peak traffic hours (Figure 6-1). Nitric oxide concentration declines as that for nitrogen dioxide rises to its maximum, suggesting a conversion of nitric oxide to nitrogen dioxide. After most nitric oxide has disappeared, the ozone (O_3) concentration rises to a maximum near the middle of the day, then falls off during the afternoon. The dependence of these product rates on the intensity of the solar radiation, and the extent and nature of the contamination present, suggested to early workers that the observed changes resulted largely from sunlight's action on the components of the polluted atmospheres. Thus, the unpleasant character of these atmospheres was designated as "photochemical smog."

The major portion of the total oxides of nitrogen emitted by combustion sources is nitric oxide. The rate nitric oxide is converted to nitrogen dioxide through thermal oxidation by the oxygen in air:

$$2NO + O_2 \rightarrow 2NO_2 \qquad (1)$$

is proportional to the square of the nitric oxide concentration; it is therefore very sensitive to changes in nitric oxide concentration. Reaction (1) can be important (conversion rate: 8% per min) in generating a small level of nitrogen dioxide (up to 25% of total NO_x) during the initial stage of dilution with air. Nitric oxide concentration is then commonly at a level of 625 mg/m^3 (500 ppm). Reaction (1) is much too slow, however, to account for any significant fraction of the nitric oxide to nitrogen dioxide

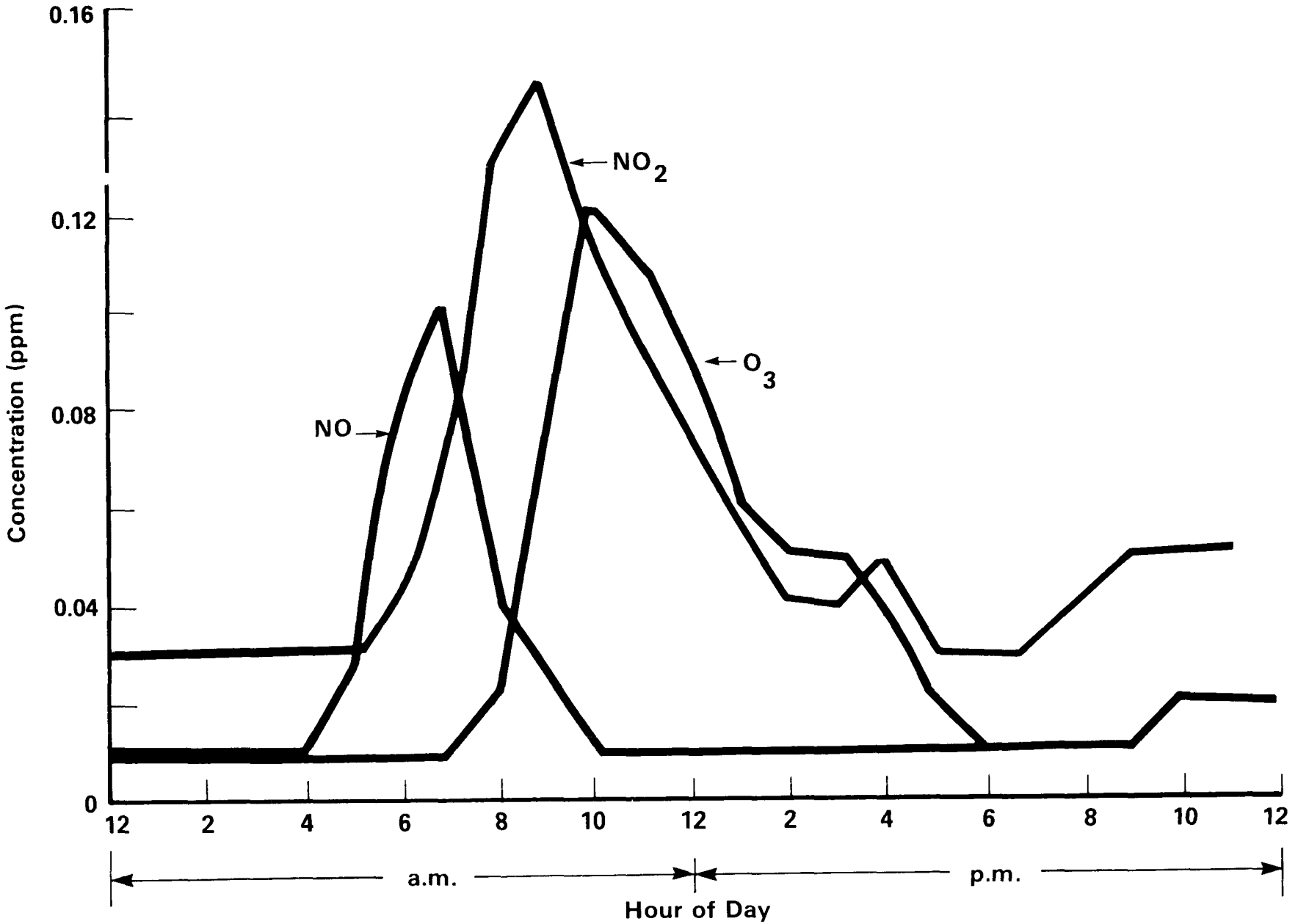


FIGURE 6-1. Example of photochemical smog build up in Los Angeles, California; the diurnal variation of nitric oxide, nitrogen dioxide, and ozone concentrations on July 19, 1965.[556]

conversion observed in the real atmosphere for typical ambient levels of nitric oxide 0.06-0.63 mg/m^3 (0.05-0.5 ppm). A much more imaginative reaction scheme must be developed to account for the data in Figure 6-1.

Most of the chemistry that occurs in a sunlight-irradiated urban atmosphere involves the interaction of a variety of unstable, excited molecules and molecular fragments (free radicals) which have only a transitory existence. These species include: the electronically excited forms of molecular oxygen, singlet-delta oxygen [$O_2(^1\Delta_g)$], and singlet-sigma oxygen [$O_2(^1\Sigma_g^+)$]; the unexcited and first excited electronic states of the oxygen atom, triplet-P oxygen atoms [$O(^3P)$] and singlet-D oxygen atoms [$O(^1D)$], respectively; ozone; symmetrical nitrogen trioxide (NO_3); dinitrogen pentoxide (N_2O_5); hydroxyl radicals (HO); hydroperoxyl radicals (HO_2); formyl radicals (HCO); alkyloxyl radicals (RO); alkylperoxyl radicals (RO_2); acylperoxyl radicals ($R\overset{O}{\overset{\|}{C}}O_2$); and other less important species. R in the formulas represents a methyl (CH_3), ethyl (C_2H_5), or another, more complex hydrocarbon radical. The paths by which these intermediates are formed and destroyed are important keys in explaining the chemical changes that occur in the polluted atmosphere.

Since sunlight triggers the phenomenon of photochemical smog formation, it is important to recognize those impurities that will absorb light energy. In some cases, these impurities decompose or become activated for reaction. A dominant sunlight absorber in the urban atmosphere is the brown gas, nitrogen dioxide. Light absorption at wavelengths <4,300 Å can cause the rupture of one of the nitrogen-oxygen (N-O) bonds in the nitrogen dioxide (O-N-O) molecule and generate the reactive ground state oxygen atom, the

triplet-P oxygen atom, and a nitric oxide molecule. The efficiency of this process is wavelength-dependent:

$$NO_2 + \text{sunlight } (2{,}900\text{-}4{,}300\ \text{Å}) \rightarrow O(^3P) + NO \qquad (2)$$

The highly reactive triplet-P oxygen atom formed in air collides frequently with oxygen molecules. During such encounters ozone may be formed:

$$O(^3P) + O_2 + M \rightarrow O_3 + M \qquad (3)$$

M in this equation represents a nitrogen, oxygen, or other third molecule that absorbs the excess vibrational energy released thereby stablizing the ozone produced. For most concentration conditions common in polluted atmospheres, the very reactive ozone molecules regenerate nitrogen dioxide by reaction with nitric oxide:

$$O_3 + NO \rightarrow NO_2 + O_2 \qquad (4)$$

Alternatively, ozone may react with nitrogen dioxide to create a new transient species, symmetrical nitrogen trioxide:

$$O_3 + NO_2 \rightarrow NO_3 + O_2 \qquad (5)$$

The nitrate species forms dinitrogen pentoxide, the reactive anhydride of nitric acid, by reaction with nitrogen dioxide:

$$NO_3 + NO_2 \rightarrow N_2O_5 \qquad (6)$$

Dinitrogen pentoxide may redissociate to form symmetrical nitrogen trioxide and nitrogen dioxide or possibly react with water to form nitric acid ($HONO_2$):

$$N_2O_5 \rightarrow NO_3 + NO_2 \tag{7}$$

$$N_2O_5 + H_2O \rightarrow 2HONO_2 \tag{8}$$

The two electronically excited and chemically reactive singlet molecular oxygen species, singlet-delta oxygen and singlet-sigma oxygen, are produced in the atmosphere by at least three different mechanisms--

(1) by direct absorption of sunlight:

$$O_2 + \text{sunlight} \left(\begin{array}{l} \sim 12{,}700\ \text{Å} \\ \sim 7{,}600\ \text{Å} \end{array} \right) \begin{array}{l} \rightarrow O_2(^1\Delta_g) \quad (9a) \\ \rightarrow O_2(^1\Sigma_g^+) \quad (9b) \end{array}$$

(2) by electronic energy transfer from electronically excited nitrogen dioxide molecules (NO_2*) formed when nitrogen dioxide absorbs sunlight at wavelengths >4,000 Å. (This does not provide sufficient energy to dissociate the nitrogen dioxide):

$$NO_2^* + O_2 \rightarrow NO_2 + O_2(^1\Delta_g) \tag{10}$$

(3) and by ozone phytolysis in sunlight:

$$O_3 + \text{sunlight} \left(\begin{array}{l} 2{,}900\text{-}3{,}060\ \text{Å} \\ 2{,}900\text{-}3{,}500\ \text{Å} \\ 4{,}500\text{-}7{,}000\ \text{Å} \end{array} \right) \begin{array}{l} \rightarrow O_2(^1\Delta_g) + O(^1\underline{D}) \quad (11a) \\ \rightarrow O_2 + O(^1\underline{D}) \text{ or } O(^3\underline{P}) \quad (11b) \\ \rightarrow O_2 + O(^3\underline{P}) \quad (11c) \end{array}$$

The singlet-$\underline{D}$ oxygen atom is much more reactive than the ground state triplet-$\underline{P}$ oxygen atom. For example, it reacts efficiently during collision with a water molecule to form an important transient species in smog, the hydroxyl radical:

$$O(^1\underline{D}) + H_2O \rightarrow 2HO \tag{12}$$

This radical is also formed through the sunlight photodecomposition of nitrous acid (HONO):

$$HONO + \text{sunlight } (2{,}900\text{-}4{,}000\ \mathring{A}) \rightarrow HO + NO \tag{13}$$

The hydroxyl radical can reassociate with nitrogen dioxide to produce nitric acid:

$$HO + NO_2 + M \rightarrow HONO_2 + M \tag{14}$$

or form nitrous acid by reacting with nitric oxide:

$$HO + NO + M \rightarrow HONO + M \tag{15}$$

A careful review of the net results of Reactions (1) through (15) reveals that these reactions alone cannot explain the rapid conversion of nitric oxide to nitrogen dioxide observed in the real atmosphere (Figure 6-1). In fact, if these reactions alone occurred the original supply of nitrogen dioxide in our atmosphere would be slightly depleted as irradiation with sunlight occurred, and a small and near constant level of ozone would be created in a few minutes. The key to the observed nitric oxide to nitrogen dioxide conversion lies in a sequence of reactions between the transient species which have been generated and other reactive molecules such as carbon monoxide (CO), the hydrocarbons, and the aldehydes present in the polluted atmosphere.

One such rational sequence of reactions was first delineated independently by two groups of scientists, Heicklen and coworkers[218] of Aerospace

Laboratories and Pennsylvania State University, and Stedman and coworkers at the Ford Motor Company. They noted that a reaction "chain" involving the hydroxyl radical and carbon monoxide may be important in driving nitric oxide to nitrogen dioxide in the atmosphere:

$$HO + CO \rightarrow H + CO_2 \qquad (16)$$

$$H + O_2 + M \rightarrow HO_2 + M \qquad (17)$$

$$HO_2 + NO \rightarrow NO_2 + HO \qquad (18)$$

$$HO + CO \rightarrow H + CO_2, \text{ etc.} \qquad (16)$$

The hydrogen atom (H) produced in Reaction (16) forms a new transient species, the hydroperoxyl radical [Reaction (17)] which may oxidize nitric oxide to nitrogen dioxide [Reaction (18)]. But since a hydroxyl radical is regenerated in Reaction (18), many cycles of sequence (16), (17), and (18) may occur and many molecules of nitric oxide may be oxidized to nitrogen dioxide for each hydroxyl radical formed. In principle, therefore, the nitric oxide to nitrogen dioxide conversion can be accelerated through such a series of reactions.

Several other molecules present in the polluted atmosphere can play a similar potentially important role for carbon monoxide. The aldehydes and hydrocarbons may participate in the sequence of reactions reforming hydroperoxyl radicals from hydroxyl radicals. Following is the reaction path involving formaldehyde (CH_2O):

$$HO + CH_2O \rightarrow H_2O + HCO \qquad (19)$$

$$HCO + O_2 \rightarrow HO_2 + CO \qquad (20)$$

$$HO_2 + NO \rightarrow HO + NO_2 \qquad (18)$$

The aldehydes in the polluted atmosphere can also function to generate the hydroperoxyl radical. For example, formaldehyde is decomposed by sunlight:[76]

$$CH_2O + \text{sunlight } (3{,}700\text{-}2{,}900\ \mathring{A}) \begin{cases} \rightarrow H + HCO & (21a) \\ \rightarrow H_2 + CO & (21b) \end{cases}$$

Both the formyl radical and the hydrogen atom produced in Reaction (21a) will react in air to form the hydroperoxyl radical:

$$H + O_2 + M \rightarrow HO_2 + M \qquad (17)$$

$$HCO + O_2 \rightarrow HO_2 + CO \qquad (20)$$

Direct clues to additional steps in the mechanism of smog formation are provided by both smog chamber studies of hydrocarbon-nitric oxide-nitrogen dioxide mixtures and atmospheric studies of changes in the relative composition of hydrocarbon pollutants as the day progresses. For example, Stephens and Burleson[511] found that the complex mixture of hydrocarbon pollutants sampled in the early morning atmosphere contained a much greater fraction of olefinic hydrocarbons than a similar sample taken in the late afternoon. Similar direct observations were made on the composition of products of trapped auto exhaust before and after irradiation with ultraviolet lamps in the laboratory.[510,511] These results clearly imply that the chemical reactions initiated in the complex nitric oxide-nitrogen

dioxide-hydrocarbon mixtures by the action of sunlight remove the olefinic hydrocarbons at a much higher rate than the paraffinic hydrocarbons.

A great variety of excellent research in both industrial and governmental laboratories has helped establish various reactivity scales for hydrocarbons, based on the relative ability of the hydrocarbons to generate nitrogen dioxide from nitric oxide in chamber experiments.[14,66,114,466,550,602]

From these experiments and many others, scientists have concluded that one or more intermediate species present in smog must react with the olefins and ultimately create hydroperoxyl radicals and organic peroxy radicals which stimulate the observed conversion of nitric oxide to nitrogen dioxide. In the lower atmosphere, the olefins are transparent to sunlight; therefore, they are not acted upon by the sunlight. However, when an olefin is added to an irradiated mix of nitrogen oxides in air, its removal can be relatively rapid when attacked by various reactive intermediates. The olefin propene (C_3H_6) may undergo the following primary reactions:

$$O + CH_3CH = CH_2 \rightarrow \dot{C}_3H_6\dot{O} \tag{22a}$$

$$\rightarrow HO + CH_2 = CH\dot{C}H_2 \tag{22b}$$

$$O_3 + CH_3CH = CH_2 \rightarrow [CH_3\overbrace{CHCH_2OOO}] \tag{23}$$

$$HO + CH_3CH = CH_2 \rightarrow H_2O + CH_2 = CH\dot{C}H_2 \tag{24a}$$

$$\rightarrow CH_3\dot{C}HCH_2OH \tag{24b}$$

$$\rightarrow CH_3CHOH\dot{C}H_2 \tag{24c}$$

$$HO_2 + CH_3CH = CH_2 \rightarrow CH_3\dot{C}HCH_2O_2H \tag{25a}$$

$$HO_2 + CH_3CH = CH_2 \rightarrow H_2O_2 + CH_2 = CH\dot{C}H_2 \tag{25b}$$

$$NO_3 + CH_2CH = CH_2 \rightarrow HONO_2 + CH_2 = CH\dot{C}H_2 \tag{26}$$

$$O_2(^1\Delta_g) + CH_3CH = CH_2 \rightarrow CH_2 = CHCH_2O_2H \tag{27}$$

A great variety of chemical reactions follow the formation of the initial reaction products (10) through (15). For simplicity, consider only the net effect of the reaction sequences. These reactions ultimately generate some number (α) of hydroperoxyl radicals, some (β) of alkylperoxy or acylperoxy radicals, and a variety of different molecules which are largely aldehydes, ketones, and acids.

$$HO \left(\begin{array}{l} \text{or other intermediate} \\ \text{in the mixture} \end{array} \right) + RH \left(\begin{array}{l} \text{olefinic, paraffinic,} \\ \text{or aromatic hydro-} \\ \text{carbon or aldehyde} \end{array} \right) \rightarrow$$

$$\alpha HO_2 + \beta RO_2 \text{ or } (R\overset{O}{\overset{\|}{C}}O_2) + \text{other products} \tag{28}$$

The hydroperoxyl radicals resulting from hydroxyl-olefin interactions [Reaction (28)] may react in Reaction (18) to cause nitric oxide conversion to nitrogen dioxide. Presumably the alkylperoxy and acylperoxy radicals formed in Reaction (28) may oxidize nitric oxide as well in Reactions (29) and (30). These reactions are analogous to the Reaction (18) involving hydroperoxyl radicals:

$$RO_2 + NO \rightarrow RO + NO_2 \tag{29}$$

$$R\overset{O}{\overset{\|}{C}}O_2 + NO \rightarrow R\overset{O}{\overset{\|}{C}}O + NO_2 \tag{30}$$

The peroxyacylnitrates, of which peroxyacetylnitrate ($CH_3\overset{O}{\overset{\|}{C}}O_2NO_2$), or PAN, is the most common, will form in the atmosphere when acylperoxy radicals associate with nitrogen dioxide ($\underline{R}-\overset{O}{\overset{\|}{C}}-O_2NO_2$):[198]

$$R\overset{O}{\overset{\|}{C}}O_2 + NO_2 \rightarrow R\overset{O}{\overset{\|}{C}}O_2NO_2 \qquad (31)$$

Speculation and uncertainty exist concerning the importance of the various atmospheric intermediates in the attack on the olefin hydrocarbons in smog. Leighton[314] discussed the probable importance in photochemical smog of oxygen atom and ozone molecule reactions with the olefinic hydrocarbons. The limited data available in 1961 precluded his evaluation of these reactions compared to those for transients such as hydroxyl, hydroperoxyl, nitrate, alkyloxy, and alkylperoxy radicals, and singlet oxygen [$O_2(^1\Delta_g)$ and $O_2(^1\Sigma_g)$]. Since 1961 several investigators have provided further insight into the potential significance of these possible reactions. It is now generally accepted that the theoretically calculated combined rate of attack of triplet-$\underline{P}$ oxygen atoms and ozone molecules on olefinic hydrocarbon molecules in the photooxidation of nitric oxide-olefin systems in air may be significantly less than the experimentally observed rate of olefin loss in such systems.[13,115,465,509] Recently Heicklen *et al.*[218] and Stedman *et al.*[507] have presented evidence that the hydroxyl radical plays a major role in both the nitric oxide-carbon monoxide- and the nitric oxide-olefin-photooxidation chains in photochemical smog simulation studies. Also Bayes,[35,154,271,604] Khan,[280] Kummler,[304] and their research groups have stimulated interest in reactions of singlet-delta oxygen in the atmosphere. They suggested that this species participates in the olefinic hydrocarbon

removal reactions in photochemical smog. The extent of this involvement remains untested. Stephens and Price,[514] Wilson *et al.*,[603] and Louw *et al.*,[325] have speculated on the role of nitrate and dinitrogen pentoxide in the chemistry of polluted air.

A detailed chemical model of the polluted atmosphere, in conjunction with available computer techniques, can simulate smog formation. This process has the potential to answer key questions concerning the detailed mechanism of smog formation and, in particular, to evaluate the various intermediates involved in smog formation.

SIMULATION OF CHEMICAL CHANGES IN A TYPICAL NITRIC OXIDE-NITROGEN DIOXIDE-HYDROCARBON-CARBON MONOXIDE-ALDEHYDE-POLLUTED ATMOSPHERE

In the simulations of the polluted atmosphere-like systems described, it is assumed that there is no atmospheric dilution of products and reactants and that the sunlight is of fixed intensity. All photochemical rates have been estimated for a solar zenith angle of 40°, a value near the average encountered during a typical day in the United States. Estimates of the actinic irradiance were taken from Leighton,[314] and are presumed to be applicable to representative atmospheric conditions near sea level on a clear day. Modifications incorporating diffusion and regular changes of solar zenith angle can be made readily, but offer a degree of sophistication that current chemical knowledge does not warrant.

This model will not provide product rates for positions near point or line sources such as smoke stacks or freeways where large fluctuations in the input pollutant levels occur.[236] Nevertheless, the "box" models should give reasonably good answers to chemical questions related both to those

urban atmospheres somewhat removed from major pollutant sources as well as for conditions of strong inversion. Such chemical schemes must be coupled to atmospheric diffusion and local emission models to achieve the full predictive potential necessary for planning local air pollution control.[136,162,472]

Sophisticated kinetic detail might be employed with our present mechanism to attempt simulating chemical events that take place in simulated NO_x-hydrocarbon polluted atmospheres. A simple mixture of the important classes of reactants has been chosen to illustrate the chemistry of these systems. To the mixture of nitric oxide at 0.075 ppm and nitrogen dioxide at 0.025 ppm, is added a background level of methane (CH_4), 1.5 ppm; a typical level of carbon monoxide, 10 ppm; typical levels of total olefinic hydrocarbon, represented by trans-2-butene (C_4H_8) at 0.10 ppm; and aldehydes, formaldehyde at 0.10 ppm, and acetaldehyde (CH_3CHO), at 0.06 ppm (representative of all of the higher aldehydes). The effects of adding 0.10 ppm of butane (C_4H_{10}), representing the saturated paraffinic hydrocarbons, will be considered later. The relative humidity is assumed to be at 50% (25° C). Computer simulation enables calculation of the theoretical time-dependence of products expected when this mixture is irradiated in sunlight (zenith angle = 40°) (Figure 6-2). Calculations have been made using two different assumptions concerning the rates of Reactions (8), (32), and (33).

$$N_2O_5 + H_2O \rightarrow 2HONO_2 \tag{8}$$

$$NO + NO_2 + H_2O \rightarrow 2HONO \tag{32}$$

$$2HONO \rightarrow NO + NO_2 + H_2O \tag{33}$$

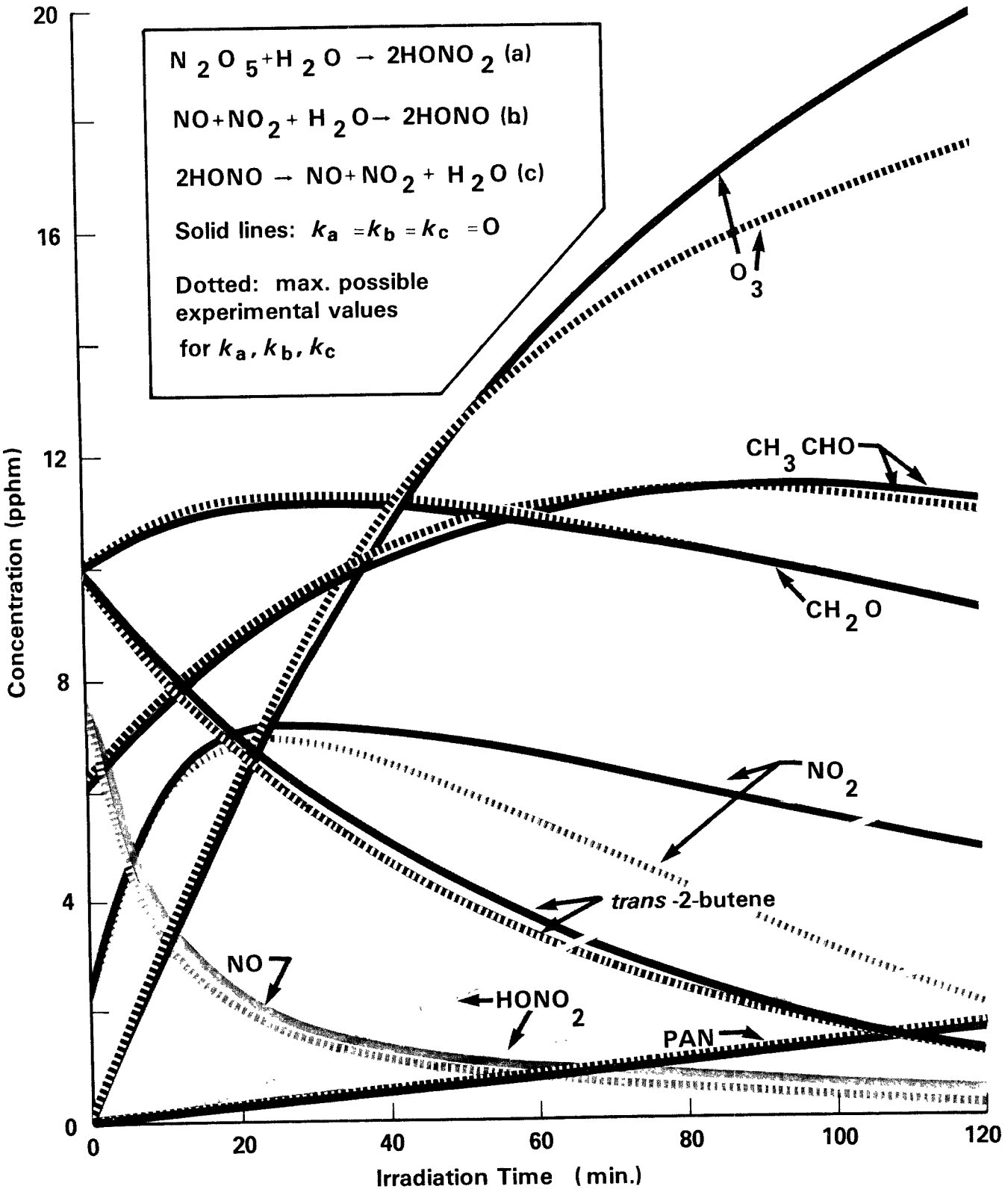


FIGURE 6-2. Theoretical rates of product formation in a sunlight irradiated (z = 40°), simulated nitric oxide-hydrocarbon-aldehyde-polluted atmosphere; initial concentrations (ppm): [nitric oxide]° = 0.075; [nitrogen dioxide]° = 0.025; [carbon monoxide]° = 10.0; [methane]° = 1.5; [trans-2-butene]° = 0.10; [formaldehyde]° = 0.10; [acetaldehyde]° = 0.06; relative humidity, 50% (25°C).[105]

The degree to which these reactions are involved in the real atmosphere is not yet known. Certainly the magnitude of the homogeneous component of these rates remains unclear. Thus the simulations have been carried out using the two possible extreme views concerning the rates of these reactions. In both extremes (Figure 6-2) it is assumed that there is no prior establishment of the equilibrium level of nitrous acid before sunlight irradiation. The data represented by the dashed curves in Figure 6-2 were calculated using the "high" values for the rate constants, k_{32} and k_{33}, estimated by Wayne and Yost,[590] who employed conditions of high surface-to-volume ratio. The solid curves were calculated using the value, $k_8 = 1.0 \times 10^{-4}$ ppm^{-1} min^{-1}, which was derived from "best fits" in the simulation of several smog chamber studies; this value is about 25 times lower than a previously published estimate of this constant.[260] On the other hand, in calculating the product concentrations in Figure 6-2, it has been assumed that Reactions (8), (32), and (33) do not occur at all; that is, the rate constants k_8, k_{32}, and k_{33} are all equal to 0.

Surprisingly, comparisons of the two sets of data in Figure 6-2 indicate that the choice of rate constants for the Reactions (8), (32), and (33) produces results with only minor differences. For example, solid curves show a slightly higher concentration of nitrogen dioxide and ozone for longer periods. This results from neglecting, in the calculation, the rate dinitrogen pentoxide is removed by water. It is evident that the rapid occurrence of Reactions (32) and (33) is not a critical factor in photooxidations of hydrocarbons at the levels encountered in real NO_x-hydrocarbon polluted atmospheres.

The relative significance of the different intermediates involved in the attack on the hydrocarbons and aldehydes in this simulated polluted atmosphere can be examined in Figure 6-2. Since the reactant concentrations in these simulations are comparable with those in real atmospheres, the results are of direct interest to the atmospheric scientist. The computer-estimated concentrations of the various intermediates and products at several time intervals during the sunlight exposure are shown in Table 6-1. These concentrations are very small compared to the stable product molecules, but their reactivity is so great that they can participate in many chemical reactions at significant rates. The rates at which given intermediates react with certain molecules in the atmosphere (olefinic hydrocarbons in this case) create the conditions governing the nature and extent of atmospheric reactions. Thus, the product of the concentration of a given intermediate at a specific time, times the concentration of the olefin at that same instant, times the rate constant for the given reaction [analogous to those shown in Reactions (22) to (27)], gives the instantaneous rate of reaction of the given intermediate with the olefin. (See data summaries in Table 6-2.)

The data predict the great importance of the hydroxyl radical species in the reaction with the olefin. Only over long periods do the ozone and hydroperoxyl radicals contribute rates comparable to those for the hydroxyl radical. The attack of triplet-P oxygen atoms is significant only at short times, but even here its rate is much less than that of the hydroxyl radical. The forced conversion of the methoxy radical (CH_3O) to formaldehyde occurs for these conditions in which the ratio of oxygen concentration to olefin concentration is high. There is a marked decrease in methoxy radical attack

TABLE 6-1

Estimated Concentrations of Intermediates in Simulated Smog Mixture as Function of Time[a]

Species	Estimated Concentrations, ppm		
	2 min	30 min	60 min
HO	1.7×10^{-7}	0.88×10^{-7}	0.72×10^{-7}
HO_2	2.1×10^{-4}	3.2×10^{-4}	3.7×10^{-4}
NO_3	0.03×10^{-6}	2.2×10^{-6}	7.4×10^{-6}
$O(^3\underline{P})$	3.8×10^{-9}	8.9×10^{-9}	9.5×10^{-9}
$O(^1\underline{D})$	0.30×10^{-15}	3.0×10^{-15}	5.0×10^{-15}
$O_2(^1\Delta_g)$	3.9×10^{-6}	5.1×10^{-6}	5.2×10^{-6}
O_3	0.0085	0.0838	0.1390
NO	0.0669	0.0163	0.0092
NO_2	0.0324	0.0720	0.0668
C_4H_8	0.0960	0.0595	0.0358
$\frac{[O_3][NO]}{[NO_2]}$	0.0175	0.0190	0.0192

[a]The simulated auto exhaust polluted atmosphere has the following initial concentrations (ppm) of trace contaminants: $[NO]^\circ = 0.075$; $[NO_2]^\circ = 0.025$; $[\underline{trans}\text{-}2\text{-}C_4H_8]^\circ = 0.10$; $[CO]^\circ = 10$; $[CH_2O]^\circ = 0.10$; $[CH_3CHO]^\circ = 0.060$; $[CH_4]^\circ = 1.5$; relative humidity, 50% (25° C); z = 40°; the rate constants for the Reactions (8), (32), and (33) are assumed to be equal to zero.

TABLE 6-2

Rate of Attack of Various Reactive Intermediate Species on trans-2-butene in a Sunlight-Irradiated, Simulated Auto-Exhaust-Polluted Atmosphere[a,b]

Time, min	Rate of Attack, ppm $min^{-1} \times 10^4$						
	$O(^3P)$	O_3	HO_2	HO	CH_3O	NO_3	$O_2(^1\Delta_g)$
2	0.13	0.26	1.69	18.1	0.026	0.00005	0.000029
	(0.13)	(0.26)	(1.62)	(17.2)	(0.025)	(0.00005)	(0.000029)
10	0.20	0.83	1.84	12.5	0.018	0.00051	0.000027
	(0.20)	(0.79)	(1.69)	(11.2)	(0.016)	(0.00050)	(0.000027)
30	0.17	1.58	1.49	5.9	0.008	0.0022	0.000020
	(0.18)	(1.60)	(1.48)	(5.5)	(0.009)	(0.0028)	(0.000021)
60	0.08	1.43	0.88	2.6	0.004	0.0026	0.000012
	(0.11)	(1.59)	(0.93)	(2.7)	(0.004)	(0.0045)	(0.000013)
90	0.03	1.00	0.49	1.2	0.002	0.0090	0.000007
	(0.05)	(1.15)	(0.52)	(1.5)	(0.002)	(0.0041)	(0.000008)

TABLE 6-2 - continued

Time, min	Rate of Attack, ppm $min^{-1} \times 10^4$						
	$O(^3P)$	O_3	HO_2	HO	CH_3O	NO_3	$O_2(^1\Delta_g)$
120	0.01	0.65	0.28	0.7	0.001	0.0018	0.000004
	(0.02)	(0.74)	(0.23)	(0.6)	(0.001)	(0.0028)	(0.000004)

[a] Initial concentrations (ppm); $[NO]^\circ = 0.075$; $[NO_2]^\circ = 0.025$; $[\underline{trans}\text{-}2\text{-}C_4H_8]^\circ = 0.10$; $[CO]^\circ = 10$; $[CH_2O]^\circ = 0.10$; $[CH_3CHO]^\circ = 0.060$; $[CH_4]^\circ = 1.5$; relative humidity = 50% (25° C); z = 40°.

[b] Results from two simulations are shown; one was made assuming finite literature values for the rate constants k_8, k_{32}, and k_{33}; the other (results shown in parentheses) was made assuming k_8, k_{32}, and k_{33} equal 0.

when olefin concentration is very high in comparison to its relatively important position observed in previous studies.[104] The theoretical fraction of olefin attack resulting from nitrate radical and singlet-delta oxygen is negligible for these conditions. In Table 6-2 the numbers in parentheses were calculated assuming the rate constants k_8, k_{32}, and k_{33} equal to 0 as in the simulation shown by the solid lines in Figure 6-2. The other numbers were calculated using the finite values for these rate constants as in the simulation shown by the dashed lines in Figure 6-2. There is no significant change in the rates obtained for the attack on the olefin by the various species when these alternative assumptions are used.

The paths of destruction of the aldehydes are also interesting to consider for this case. Table 6-3 summarizes the rates of attack of the various intermediates on formaldehyde. There is even more selectivity shown between species here than seen in Table 6-2 for the olefin. The hydroxyl radical attack represents about 99.94% of the total rate of aldehyde reaction which occurs in short times, and this order of preference continues through the 2-hr irradiation period. The attack on acetaldehyde, or the higher aldehydes if present, is similar to that on formaldehyde and need not be considered in detail here. The estimated rate of formaldehyde loss through photodecomposition (7.3×10^{-4} ppm min^{-1} at 2 min) is about twice the rate due to radical attack alone. The lifetime of the aldehydes at a concentration of 0.1 ppm is relatively long (about 62 min) in these systems.

Questions have been raised concerning which reactions trigger the process of nitrogen dioxide conversion in sunlight-irradiated polluted

TABLE 6-3

Rate of Attack of Various Reactive Intermediate Species on Formaldehyde in a Sunlight-Irradiated, Simulated Auto-Exhaust-Polluted Atmosphere[a,b]

Time, min	Rate of Attack (ppm min^{-1} x 10^4)				
	$O(^3P)$	HO	HO_2	CH_3	NO_3
2	0.0010	3.86	0.0046	0.0005	0.0000
	(0.0010)	(3.66)	(0.0045)	(0.0005)	(0.0000)
10	0.0019	3.31	0.0063	0.0004	0.0004
	(0.0019)	(2.93)	(0.0057)	(0.0004)	(0.0004)
30	0.0023	2.34	0.0077	0.0003	0.0029
	(0.0024)	(2.05)	(0.0071)	(0.0003)	(0.0034)
60	0.0019	1.69	0.0075	0.0002	0.0058
	(0.0023)	(1.66)	(0.0073)	(0.0002)	(0.0092)
90	0.0013	1.27	0.0064	0.0002	0.0307
	(0.0019)	(1.45)	(0.0066)	(0.0002)	(0.0133)
120	0.0008	1.05	0.0056	0.0001	0.0078
	(0.0014)	(0.96)	(0.0046)	(0.0001)	(0.0151)

[a]Initial concentrations (ppm): $[NO]^\circ = 0.075$; $[NO_2]^\circ = 0.025$; $[\underline{trans}\text{-}C_4H_8] = 0.10$; $[CO]^\circ = 10$; $[CH_2O]^\circ = 0.10$; $[CH_3CHO]^\circ = 0.06$; $[CH_4]^\circ = 1.5$; relative humidity = 50% (25° C); z = 40°.

[b]Results from two simulations are shown; one was made assuming finite literature values for the k_8, k_{32}, and k_{33} rate constants; the other (results shown in parentheses) was made assuming k_8, k_{32}, and k_{33} equal 0.

atmospheres.[314] The simulation under discussion shows the hydroxyl radical to be the most important transient species from the standpoint of initiating the olefin oxidation; however, any reactant which creates either the hydroxyl or hydroperoxyl radical will effectively generate hydroxyl radicals, since, for these conditions, the reaction $HO_2 + NO \rightarrow HO + NO_2$ is the major fate of hydroperoxyl radicals. To understand this system, it is important to investigate the relative importance of the several possible sources of both these radicals for the present system.

The cases representing the two possible extremes for the real atmosphere are again encountered. At one extreme, there is no initial nitrous acid formation; that is, the rate constants k_{32} and k_{33} equal 0. For assumed impurity levels there will be only the following initial sources of stimulus to chemical change:

$$NO_2 + \underline{h}\nu \rightarrow O(^3\underline{P}) + NO; \text{ Initial rate} = 120 \times 10^{-4} \text{ ppm min}^{-1} \qquad (2)$$

$$H_2CO + \underline{h}\nu \rightarrow HCO + H; \text{ Initial rate} = 2.2 \times 10^{-4} \text{ ppm min}^{-1} \qquad (21a)$$

$$CH_3CHO + \underline{h}\nu \rightarrow CH_3 + HCO; \text{ Initial rate} = 1.5 \times 10^{-4} \text{ ppm min}^{-1} \qquad (34)$$

Although the rate of oxygen atom formation is by far the largest for the intermediates formed here, very little impetus to the rate of photooxidation of the olefin or the conversion of nitric oxide to nitrogen dioxide is given by Reaction (2). The occurrence of this reaction establishes an appreciable initial rate of ozone formation. However this rate is not seen since both ozone destruction and the very fast regeneration of nitrogen dioxide occur through Reaction (4):

$$O_3 + NO \rightarrow O_2 + NO_2 \qquad (4)$$

The extent of chemical change in nitrogen dioxide and ozone is limited very quickly. If only nitric oxide and nitrogen dioxide were present as impurities in this system, it would be expected that the concentration of nitrogen dioxide would fall somewhat as Reaction (2) occurs, and that of ozone would rise to a relatively small steady-state value of about 5.1 x 10^{-3} ppm in about 2 min. Reaction (2) results in too small a rate of oxygen atom attack on the olefin (0.13 x 10^{-4} ppm min^{-1}), to explain the rates of chemical change observed in Figure 6-2.

Thus, the major part of the initial impetus to oxidize nitric oxide and olefin in this system must come from the aldehyde photolyses. Since most formyl radicals react to form hydroperoxyl radicals under these conditions, the total initial rate of hydroperoxyl radical generation from the aldehyde photolyses is expected to be about 5.5 x 10^{-4} ppm min^{-1}. From the initial reactant concentrations, about 94.8% of the hydroperoxyl radicals oxidize nitric oxide and about 5% react with olefin:

$$HO_2 + NO \rightarrow NO_2 + HO \qquad (18)$$

$$HO_2 + CH_3CH = CHCH_3 \rightarrow CH_3CH(O_2H)\dot{C}HCH_3 \qquad (35)$$

In Reaction (34), an additional reactant, the methyl radical, is formed, most of which will then form the methylperoxyl radical (CH_3O_2):($CH_3 + O_2 \rightarrow CH_3O_2$). Thus, the rate of formation of the methylperoxyl radical is: $Rate_{CH_3O_2} \cong 1.5 \times 10^4$ ppm min^{-1}. For these conditions nearly all of the methylperoxyl radicals will react to oxidize nitric oxide:

$$CH_3O_2 + NO \rightarrow CH_3O + NO_2 \qquad (36)$$

Thus the initial rate of nitrogen dioxide formation by way of Reactions (18) and (36) will be about 7×10^{-4} ppm min^{-1}. This rate of formation is much less than the initial rate of destruction of nitrogen dioxide by photolysis (120×10^{-4} ppm min^{-1}). Nitrogen dioxide increase in the smog system is therefore caused primarily by hydroxyl-olefin reactions which generate the chain processes that indirectly allow each hydroxyl radical to oxidize several nitric oxide molecules.

The rates at which hydroxyl and hydroperoxyl free radicals are generated from the several major sources in the system at various times throughout photooxidation are shown in Figure 6-2. Most hydroxyl radicals are formed by the following reactions:

(A) $HONO + \underline{h}\nu \rightarrow HO + NO$ (13)

(B) $O(^1\underline{D}) + H_2O \rightarrow 2HO$ (12)

(C) $HO_2 + NO \rightarrow HO + NO_2$ (18)

(D) $H_2O_2 + \underline{h}\nu \rightarrow 2HO$ (37)

(E) $CH_3\dot{C}HO_2H \rightarrow CH_3CHO + HO$ (38)

Compare the rates of hydroxyl radical generation from each of these steps as functions of the irradiation time in Table 6-4.

At very short periods, the photolysis of nitrous acid is not significant because by setting the rate constant $k_{32} = 0$, its presence is excluded from consideration. However, even in this case nitrous acid is formed in Reactions (14) and (39) as the run progresses,

TABLE 6-4

Comparison of the Theoretical Rates of the Hydroxyl Radical Forming Reactions in a Simulated, Sunlight-Irradiated, Auto-Exhaust-Polluted Atmosphere[a]

Time, min	Rates of HO Formation (ppm $min^{-1} \times 10^4$) Reaction (A)[b]	Reaction (B)[b]	Reaction (C)[b]	Reaction (D)[b]	Reaction (E)[b]
0.05	0.0088	0.0015	28.3	0.0003	1.5
0.50	0.0907	0.0108	29.3	0.0029	1.6
2.0	0.32	0.023	27.8	0.013	1.6
10.0	0.64	0.082	18.5	0.080	1.7
30.0	0.29	0.23	10.0	0.33	1.5
60.0	0.12	0.38	5.9	0.79	0.9
90.0	0.07	0.48	4.0	1.2	0.5
120.0	0.05	0.55	1.2	1.6	0.2

[a]The simulated auto-exhaust-polluted atmosphere has the following initial concentrations (ppm) of trace contaminants: $[NO]^\circ = 0.075$; $[NO_2]^\circ = 0.025$; $[\underline{trans}\text{-}2\text{-}C_4H_8]^\circ = 0.10$; $[CO]^\circ = 10$; $[CH_2O]^\circ = 0.10$; $[CH_3CHO]^\circ = 0.060$; $[CH_4]^\circ = 1.5$; relative humidity, 50% (25° C); z = 40°. The rate constants for the Reactions (8), (32), and (33) are assumed to be equal to 0 as in the data for the solid line shown in Figure 6-2 for this same system.

[b]See page 26 for Reactions (A) through (E).

$$HO_2 + NO_2 \rightarrow HONO + O_2, \tag{39}$$

and its photolysis contributes a maximum of about 3% to the total rate of hydroxyl radical generation at 10 min. Ozone is absent at the start of the run; hence, the singlet-D oxygen atom, its photolysis product, is also absent, and Reaction (12) is not important. However, late in the photo-oxidation, as ozone builds up, the singlet-D oxygen atom contributes significantly to the hydroxyl radical formation rate. Hydrogen peroxide photolysis, also unimportant initially, provides steadily increasing amounts of the hydroxyl radical as hydrogen peroxide builds up; in fact, after most of the butene has been oxidized at 120 min, Reaction (37) becomes the major source of hydroxyl radicals in the mixture with Reaction (38) providing a steady but rather minor source of hydroxyl radicals. The radical CH_3CHO_2H is one of the products of the reaction sequence which follows hydroperoxyl addition to the butene.

The highest rate of hydroxyl radical generation occurs through reaction of the hydroperoxyl radical with nitric oxide [Reaction (18); see Table 6-4]. In fact, the magnitude of this rate (29.3×10^{-4} ppm min^{-1} at 0.5 min) is much greater than the rate at which radicals are made in sunlight-initiated steps (7.0×10^{-4} ppm min^{-1}); from hydrogen and formyl radicals formed by aldehyde photolyses (5.5×10^{-4} ppm); and by way of methyl formation from acetaldehyde photolysis (1.5×10^{-4} ppm min^{-1}) [Reaction (34)]:

$$CH_3 \xrightarrow{(O_2)} CH_3O_2 \xrightarrow{(NO)} CH_3O \xrightarrow{(O_2)} HO_2 + CH_2O$$

Hydroperoxyl radicals must be regenerated in a chain reaction to provide the observed rate of Reaction (18). This chain process can be understood by comparing the rates of hydroperoxyl formation from the various sources of this radical:

(A) $CH_2O + h\nu \rightarrow HCO + H;\ H + O_2 + M \rightarrow HO_2 + M$ (21a)

$HCO + O_2 \rightarrow HO_2 + CO$

$$\rightarrow HCOO_2 \xrightarrow{(NO)} NO_2 + HCO_2 \xrightarrow{(O_2)} HO_2 + CO_2$$

$$\xrightarrow{(NO_2)} (HCOO_2NO_2) \rightarrow HONO_2 + CO_2$$

(B) $CH_3CHO + \underline{h}\nu \rightarrow CH_3 + HCO$ (34)

$HCO + O_2$ [as in (A)] $\rightarrow \alpha HO_2$ + products

(C) $HO + CO \rightarrow CO_2 + H;\ H + O_2 + M \rightarrow HO_2 + M$ (16)

(D) $CH_3CH(\dot{O})CHO \rightarrow CH_3CHO + HCO$ (40)

$HCO + O_2$ [as in (A)] $\rightarrow \alpha HO_2$ + products

(E) $CH_3O + O_2 \rightarrow HO_2 + CH_2O$ (41)

(F) $CH_3CH(\dot{O})\dot{O} + O_2 \rightarrow CH_3CO_2 + HO_2$ (42)

The rates of hydroperoxyl radical formation from each source are compared in Table 6-5. This comparison indicates that the photolyses of both formaldehyde and acetaldehyde continue as significant sources of the hydroperoxyl radical through hydrogen and formyl radical formation (see columns A and B in Table 6-5). Acetaldehyde photolysis also forms hydroperoxyl radicals through these methyl radical reactions:

$$CH_3 \xrightarrow{(O_2)} CH_3O_2 \xrightarrow{(NO)} CH_3O$$

The methoxy radical generates the hydroperoxyl radical in Reaction (41); however, this reaction provides only a small part, 1.5×10^{-4} ppm min^{-1}, of the total rate after a short period (Table 6-5, Column E).

The reaction of the hydroxyl radical with carbon monoxide is a third source of the hydroperoxyl radical [Reactions (16) and (17)]. This may be the major hydroperoxyl regeneration step in the chain oxidation of nitric oxide to nitrogen dioxide:[218,507]

$$HO + CO \rightarrow H + CO_2 \tag{16}$$

$$H + O_2 + M \rightarrow HO_2 + M \tag{17}$$

$$HO_2 + NO \rightarrow HO + NO_2 \tag{18}$$

Although important in this regard, it is not the main source of the hydroxyl radical to hydroperoxyl radical conversion in this simulated polluted atmosphere.

In sequence (40), the hydroperoxyl radical is produced in the decomposition of the theoretical intermediate radical, $CH_3CH(\dot{O})CHO$, formed in the reaction sequence which follows the hydrogen atom abstraction from butene

TABLE 6-5

Comparison of the Theoretical Rates of the Hydroxyl-Forming Reactions in a Simulated, Sunlight-Irradiated, Auto-Exhaust-Polluted Atmosphere[a]

Time, min	Rate of HO_2 Formation, ppm min^{-1} x 10^4					
	Reaction (A)[b]	Reaction (B)[b]	Reaction (C)[b]	Reaction (D)[b]	Reaction (E)[b]	Reaction (F)[b]
0.05	4.0	1.5	4.2	5.1	15.8	0.05
0.5	4.0	1.5	4.4	5.2	16.5	0.1
2.0	4.1	1.6	4.3	4.9	15.9	0.2
10.0	4.3	1.8	3.2	3.0	11.8	0.6
30.0	4.3	2.2	2.2	1.5	8.8	1.1
60.0	4.2	2.5	1.8	0.7	6.9	1.1
90.0	3.8	2.5	1.7	0.4	5.4	0.8
120.0	3.4	2.4	1.3	0.2	3.9	0.5

[a]The simulated auto-exhaust-polluted atmosphere has the following initial concentrations (ppm) of trace contaminants: $[NO]^\circ = 0.075$; $[NO_2]^\circ = 0.025$; $[\text{trans-}2\text{-}C_4H_8]^\circ = 0.10$; $[CO]^\circ = 10$; $[CH_2O]^\circ = 0.10$; $[CH_3CHO]^\circ = 0.060$; $[CH_4]^\circ = 1.5$; relative humidity, 50% (25° C); z = 40°. The rate constants for the Reactions (8), (32), and (33) are assumed to 0 as in the data shown in the solid curves in Figure 6-2 for this same system.

[b]See page 29 for Reactions (A) through (E).

by the hydroxyl radical (or other radicals that abstract hydrogen atoms to a much smaller extent).

The rates in Column E, Table 6-5, show the largest source of hydroperoxyl radicals in this system to be the methoxy radical [Reaction (41)]. Shortly after photooxidation of the butene-containing mixture begins, the methoxy radical is formed from methyl radicals derived from several reaction paths. Acetaldehyde photolysis provides a relatively minor source. The methyl radical is also formed in the reaction sequence which follows hydroxyl radical addition to the butene. Specifically, its formation is postulated in the following step:

$$CH_3CH(OH)\dot{O} \rightarrow CH_3 + HCO_2H \qquad (43)$$

Another source of methyl radicals is the attack by hydroxyl radicals on acetaldehyde:

$$CH_3CHO \xrightarrow{(HO)} CH_3CO \xrightarrow{(O_2)} CH_3COO_2 \xrightarrow{(NO)} CH_3CO_2 \rightarrow CH_3 + CO_2$$

When the ratio of nitric oxide to nitrogen dioxide concentration is relatively high, the nitric oxide oxidized by acetylperoxyl radicals (CH_3COO_2) yields primarily the acetate (CH_3CO_2) radical; peroxyacetyl nitrate is the favored product only at low ratios of nitric oxide to nitrogen dioxide concentrations over the long run.

In theory, the reaction sequence which follows ozone attack on the olefin leads to the acetylhydroperoxyl radical [$CH_3CH(\dot{O})\dot{O}$]. Its reaction with the oxygen molecule is the fifth source of the hydroperoxyl radical (Reaction (42)]; this contribution to the rate, shown in Column F, Table

6-5, is negligible at short times, but as ozone builds up over long exposures, it makes a finite contribution.

These considerations illustrate the complex interplay among the various reactants in this simulated smog-forming atmosphere which stimulates the generation of the important hydroxyl and hydroperoxyl radicals. In the selected reactant conditions, the olefin is the major source of conversion of hydroxyl to hydroperoxyl radicals. Both the abstraction of hydrogen atoms from the olefin by the hydroxyl radical, and the addition of the hydroxyl radical to olefin ultimately generate a hydroperoxyl radical with moderate efficiency. In this reaction sequence at least one nitric oxide molecule is also oxidized to nitrogen dioxide by various alkylperoxy radicals. Thus, during short exposures (Figure 6-2), when each hydroxyl radical abstracts hydrogen atoms from butene, at least one molecule of nitric oxide is converted to nitrogen dioxide and one hydroperoxyl radical is formed. For each addition of a hydroxyl radical to butene, nitrogen dioxide and another hydroperoxyl radical are formed from an average of three nitric oxide molecules. Most of the hydroperoxyl radicals are rapidly reconverted to hydroxyl radicals through the reaction: $HO_2 + NO \rightarrow HO + NO_2$. Therefore, the chain sequence of hydroxyl radical attack on butene (C_4H_8) continually degrades the olefin until the chain is terminated. The rate of hydroperoxyl formation in the primary photolytic processes (7.0×10^{-4} ppm min^{-1} at 2 min) and the total rate of attack by both hydroxyl and hydroperoxyl radicals on this (19.8×10^{-4} ppm min^{-1} at 2 min) suggests a short chain reaction (about 2.8 cycles in length at 2 min) involving hydroxyl and hydroperoxyl radical oxidation of the olefin. Of course, the chains stop whenever a hydroperoxyl or hydroxyl radical is removed through

reaction with another radical or an odd electron molecule such as nitric oxide or nitrogen dioxide. The dominant chain-ending steps for the concentration conditions selected for this system are:

$$2HO_2 \rightarrow H_2O_2 + O_2 \tag{44}$$

$$HO + NO + M \rightarrow HONO + M \tag{15}$$

$$HO + NO_2 + M \rightarrow HONO_2 + M \tag{14}$$

$$CH_3O_2 + HO_2 \rightarrow CH_3O_2H + O_2 \tag{45}$$

The rates radicals are removed by reactions in the Figure 6-2 simulation at 2 min, are as follows (ppm min^{-1}): $Rate_{43} = 4.6 \times 10^{-4}$; $Rate_{15} = 1.2 \times 10^{-4}$; $Rate_{14} = 0.8 \times 10^{-4}$; $Rate_{44} = 0.07 \times 10^{-4}$. These account for hydroperoxyl and hydroxyl radical removal rates of 6.6×10^{-4} ppm min^{-1}. This should match the rate of primary radical production (7.0×10^{-4} ppm min^{-1}) if all radical sources and termination reactions are included.

The origin of the rapid rise in the concentration of nitrogen dioxide in the smog simulation is shown in Figure 6-2. The nitrogen dioxide oxidation rate in these experiments also reflects the occurrence of these chain processes. In the simulations the observed rate of nitrogen dioxide formation at 2 min is about 52×10^{-4} ppm min^{-1}. The true rate of formation of nitrogen dioxide at 2 min is much greater (208×10^{-4} ppm min^{-1}) since nitrogen dioxide is destroyed in Reaction (2) at 156×10^{-4} ppm min^{-1} by the action of sunlight. The major processes forming nitrogen dioxide at the 2 min exposure are:

$$O_3 + NO \rightarrow NO_2 + O_2 \quad (4)$$

$$HO_2 + NO \rightarrow HO + NO_2 \quad (18)$$

$$RO_2 + NO \rightarrow RO + NO_2 \quad (29)$$

The simulation predicts that the rate nitrogen dioxide forms in Reaction (4) at 2 min is about 131 x 10^{-4} ppm, while that of Reaction (18) is 28 x 10^{-4} ppm min^{-1}. Therefore, the several reactions of the various acyl and alkylperoxy radicals [RO_2 in Reaction (29)] must account for about (208-131-28) x 10^{-4} = 49 x 10^{-4} ppm min^{-1}. This expectation is consistent with the rates of alkyl and acylperoxyl radical generation at 2 min. The hydroxyl radical attack on olefin and acetaldehyde, and the hydroperoxyl radical attack on olefin are the only important sources of alkyl and acylperoxyl radicals for these conditions. The simulations show that at 2 min irradiation the hydroxyl radical reacts with olefin to add at a rate of 12.3 x 10^{-4} ppm, and to abstract hydrogen atoms at a rate of 4.9 x 10^{-4} ppm min^{-1}; the hydroxyl radical abstracts hydrogen atoms from acetaldehyde at 2.4 x 10^{-4} ppm min^{-1}; and the hydroperoxyl radical adds to olefin at 1.6 x 10^{-4} ppm min^{-1}. Each hydroxyl radical addition to olefin forms about three alkylperoxyl radicals; each hydroxyl radical that attacks acetaldehyde creates about two acylperoxyl radicals; each hydroxyl radical that abstracts hydrogen atoms from olefin and each hydroperoxyl radical which adds to olefin usually form one alkylperoxyl radical. Therefore, the total rate of alkyl and acylperoxyl radical formation can be given by:

$$Rate_{RO_2} \simeq (3 \times 12.3 + 2 \times 2.4 \times 4.9 + 1.6) \times 10^{-4} \simeq 48 \times 10^{-4} \text{ ppm min}^{-1}.$$

This agrees with the rate of nitrogen dioxide formation attributed to nitric oxide oxidation by alkyl and acylperoxyl species in Reaction (29) ($\sim 49 \times 10^{-4}$ ppm min^{-1}).

Nitrogen dioxide destruction by photolysis and its subsequent reformation by reaction with nitric oxide and ozone [Reaction (4)] can by itself only lead to a net decrease in the nitrogen dioxide equal to the ozone level developed.

$$NO_2 + \underline{h}\nu \rightarrow O + NO \tag{2}$$

$$O + O_2 + M \rightarrow O_3 + M \tag{3}$$

$$O_3 + NO \rightarrow O_2 + NO_2 \tag{4}$$

Therefore, the nitrogen dioxide increase observed in irradiated smog results from both hydroperoxyl and alkyl and acylperoxyl radical Reactions (18) and (29), and under the conditions at the 2 min point of the photochemical smog simulation (Figure 6-2), the alkyl or acylperoxyl radical contributes to the observed rate of nitrogen dioxide formation about 1.8 times more than the hydroperoxyl radical. Both types of radicals, however, are very important in this conversion.

Since the ozone-olefin and oxygen-atom-olefin reactions contribute little to the rate at which hydroperoxyl and hydroxyl radicals form early in the run, it is incorrect to assume, as many investigators have, that the original driving force for the nitric oxide photooxidation must result from radicals produced in the ozone and oxygen atom reactions with olefin; obviously this is not the case when aldehydes are initially present.

In the alternate hypothesis of nitrous acid preequilibration in the simulated polluted atmosphere, nitrous acid is assumed to have been formed at its equilibrium value, [HONO] = 6.1×10^{-3} ppm, before sunlight irradiation of the mixture. All other compounds remain at their previous concentrations; Reactions (32) and (33), in which nitrous acid is generated and destroyed, respectively, are assumed to have their literature values. The initial rate of radical generation in this system includes the same rates of hydroperoxyl and methylperoxyl formation from formaldehyde and acetaldehyde photolyses as before, but there is an additional source of hydroxyl radical formation from the nitrous acid photolysis:

$$HONO + \underline{h}\nu \rightarrow HO + NO \qquad (13)$$

This rate is about 7.3×10^{-4} ppm min^{-1} for zenith angle = 40°. Compare this with the rate of hydroperoxyl formation from all of the aldehyde photolysis processes; $Rate_{HO_2} = 7.0 \times 10^{-4}$ ppm min^{-1}. The nitrous acid in the atmosphere can significantly boost the initial rate of olefin photooxidation and nitric oxide to nitrogen dioxide conversion, approximately doubling these initial rates for the simulations if nitrous acid preequilibrium were allowed before irradiation. Assuming preequilibration of nitrous acid and allowing the finite values for the rate constants k_8, k_{32}, and k_{33} as before, it can be predicted from simulations not shown here that the maximum in the nitrogen dioxide concentration would occur at 22 min, compared with 24 min to reach the maximum concentration for the same mixture but without nitrous acid initially. (See the dashed curves, Figure 6-2.) Assuming the rate constants $k_8 = k_{32} = 0$, it takes 31 min to reach this maximum when there is no nitrous acid initially present. (See the solid curves, Figure 6-2.)

Nitrous acid is not necessary for smog formation; but its presence can enhance the initial rate of the smog-forming reactions. Its presence is invoked to rationalize nitric oxide photooxidation in moist atmospheres containing carbon monoxide as the only other oxidizable component. The levels of nitrous acid present in the real atmospheres must be experimentally determined along with which of the two extremes considered above best represents the real situation.

The large ozone concentration formed in these simulated polluted atmospheres is significant in view of the low concentration limits specified for ozone by the U.S. Environmental Protection Agency: a maximum concentration of 0.08 ppm/1-hr period, not to be exceeded more than one time/yr.[566] In theory, the ozone concentration ultimately formed in these systems is largely controlled by the magnitude of the ratio of nitrogen dioxide to nitric oxide concentration and the sunlight intensity. The product of the concentrations of nitric oxide and ozone to nitrogen dioxide ratio is virtually a constant for the zenith angle = 40° simulations at the three times shown (see Table 6-1). The concentrations of hydrocarbons (olefins, paraffins, and aromatics), aldehydes, and carbon monoxide, however, all influence the final nitrogen dioxide to nitric oxide concentration ratio, determining ozone concentrations in a complex and interrelated way.

THE EFFECTS OF THE SATURATED HYDROCARBONS ON THE CHEMISTRY OF THE SIMULATED, POLLUTED ATMOSPHERE

The "reactive" paraffinic hydrocarbons have been excluded previously in an attempt to simplify the simulated system. If butane or similar paraffins were present, hydroxyl radicals would attack them at a moderate rate to abstract hydrogen atoms; the secondary butyl radical ($CH_3-\dot{C}H-CH_2-CH_3$) is the major organic product of this interaction with normal butane ($CH_3-CH_2-CH_2-CH_3$).

$$\underline{n}\text{-}C_4H_{10} + HO \rightarrow H_2O + \underline{sec}\text{-}C_4H_9 \qquad (46)$$

The main sequence of reactions for the higher alkyl radicals in oxygen leads to the generation of carbonyl compounds and hydroperoxyl radicals; thus, for secondary butyl radicals, the major sequence at early stages of this reaction, for the concentrations of mixture components chosen in the simulation, would be the following:

$$\underline{sec}\text{-}C_4H_9 \xrightarrow{(O_2)} \underline{sec}\text{-}C_4H_9O_2 \xrightarrow{(NO)} \underline{sec}\text{-}C_4H_9O \begin{cases} \xrightarrow{(O_2)} CH_3COC_2H_5 + HO_2 \\ \rightarrow CH_3CHO + C_2H_5 \end{cases}$$

$$C_2H_5 \xrightarrow{(O_2)} C_2H_5O_2 \xrightarrow{(NO)} C_2H_5O \begin{cases} \xrightarrow{(O_2)} CH_3CHO + HO_2 \\ \rightarrow CH_3 + CH_2O \end{cases}$$

$$CH_3 \rightarrow CH_3O_2 \xrightarrow{(O_2)} CH_3O \xrightarrow{(NO)} CH_2O + HO_2$$

Using the rate constant estimates and the initial concentration conditions, about one hydroperoxyl radical is formed for every hydroxyl radical which attacks butane and about 1.5 nitrogen dioxide molecules are generated from nitric oxide. Butane, in the simulated atmosphere at the level of the olefin, 0.10 ppm, would be attacked by the hydroxyl radical at about 1.2×10^{-4} ppm min^{-1} at 2 min. This increases the rate of nitrogen dioxide formation in the system by only 2%. Paraffin hydrocarbons are added to the real atmosphere at levels near those of olefins, during the early morning auto-exhaust-pollution.[510] This causes only minor disturbances in the reaction scheme outlined for the simpler, simulated polluted atmospheres. (See page 51 for further considerations of the olefin-free system containing NO_x and paraffin hydrocarbons.)

THE EFFECTS OF VARIATION IN THE INITIAL ALDEHYDE CONCENTRATIONS IN THE SIMULATED POLLUTED ATMOSPHERE

Calvert and McQuigg[77] have investigated the influence of the initial aldehyde concentrations on the smog chemistry of the simulated polluted atmosphere. Figure 6-3 plots product-time data for simulated smog mixtures containing aldehydes initially (solid curves) and without initial aldehydes (dashed curves); each of the other components is initially at the same concentration in both cases. As the aldehydes are removed completely for the initial conditions, a small induction period appears in the rates of olefin removal, and nitrogen dioxide, ozone, PAN, and nitric acid formation. The final levels of the dashed and solid curves indicate higher concentrations of ozone, PAN, and nitric acid when aldehydes are present initially.

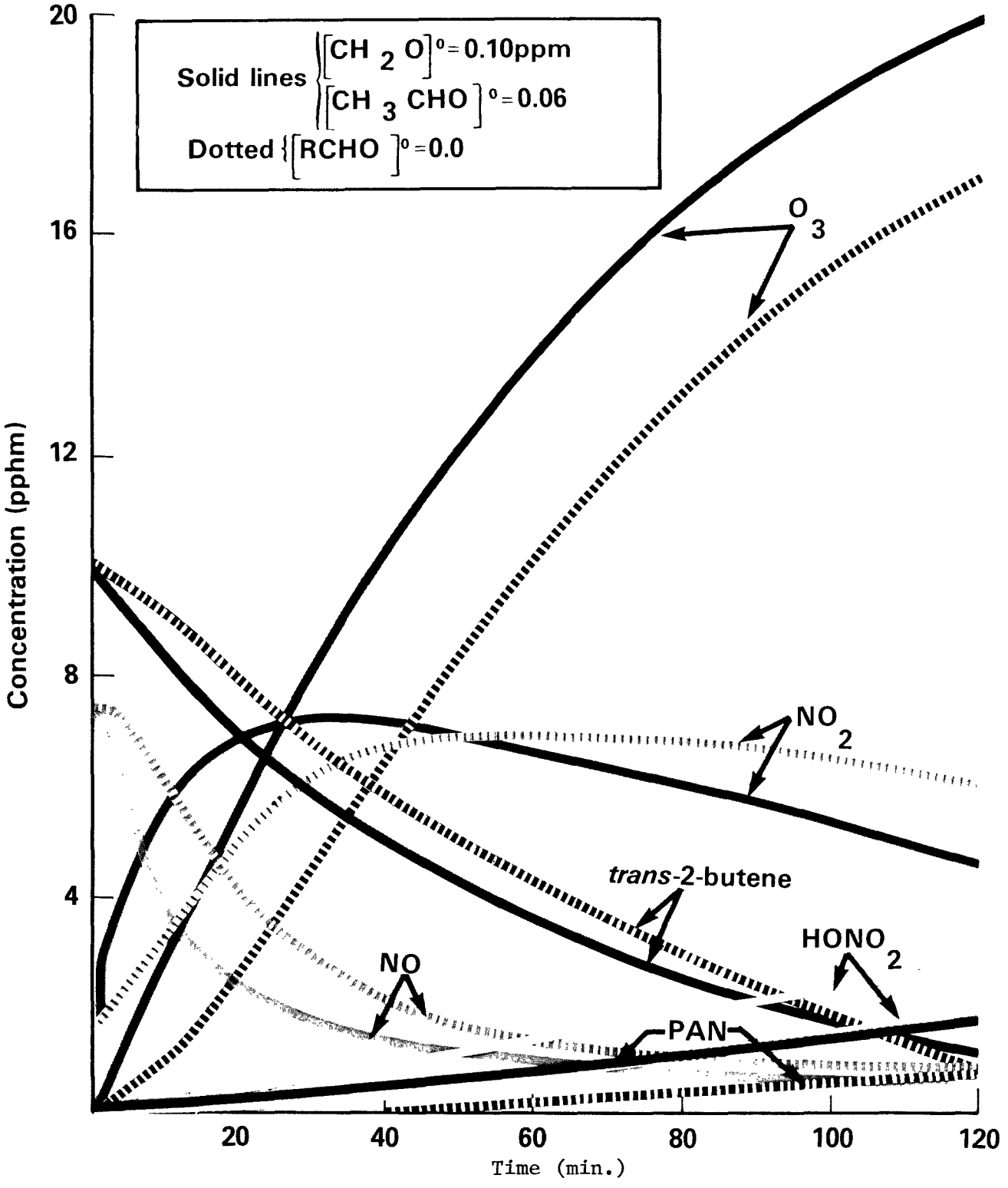


FIGURE 6-3. The effect of initial aldehyde concentration on the theoretical rates of product formation in a sunlight irradiated (z = 40°) simulated auto exhaust polluted atmosphere; composition same as that in Figure 6-2 with the exception of the absence of initial aldehydes in the dashed curves.

THE EFFECTS OF VARIATION OF THE OLEFINIC HYDROCARBON CONCENTRATIONS ON THE PRODUCT-TIME DATA IN A SIMULATED POLLUTED ATMOSPHERE

Observing the relationship of certain smog manifestations with the concentrations of hydrocarbons and the oxides of nitrogen, Faith *et al.*[138] derived the relation of eye irritation to NO_x and hydrocarbon levels in smog chamber studies (Figure 6-4). Figure 6-5 shows the now famous relationship between the maximum daily 1-hr average oxidant levels and the 6-9 a.m. average concentration of nonmethane hydrocarbons for several cities (Schuck *et al.*[464]). The maximum observed oxidant values produce a curve that falls with decrease in hydrocarbon level; but the shape of the curve at low hydrocarbon values is obscure. A search for similar predicted correlations with the simulated atmosphere should be conducted cautiously because of the serious limitations imposed by our present state of knowledge.

The predicted time dependence of ozone as a function of olefin (*trans*-2-butene) level (Calvert and McQuigg[77]) is summarized in Figure 6-6. In this series of experiments the initial concentrations of the oxides of nitrogen are fixed: [nitric oxide]° = 0.075 ppm and [nitrogen dioxide]° = 0.025 ppm; and aldehydes absent initially. The relationship between ozone and olefin is obviously complex; but, in general, some decrease in ozone concentration can be expected for lowered initial olefin concentration. The final ozone concentration reached in these systems can be well above the initial total NO_x present. Figure 6-7 effectively illustrates the 8-hr integral of the ozone concentration versus time, and the concentration of peroxyacetylnitrates versus time data as a function of the olefin concentration.[77] Data are given for with aldehydes present initially (dashed curves) and with no aldehydes present

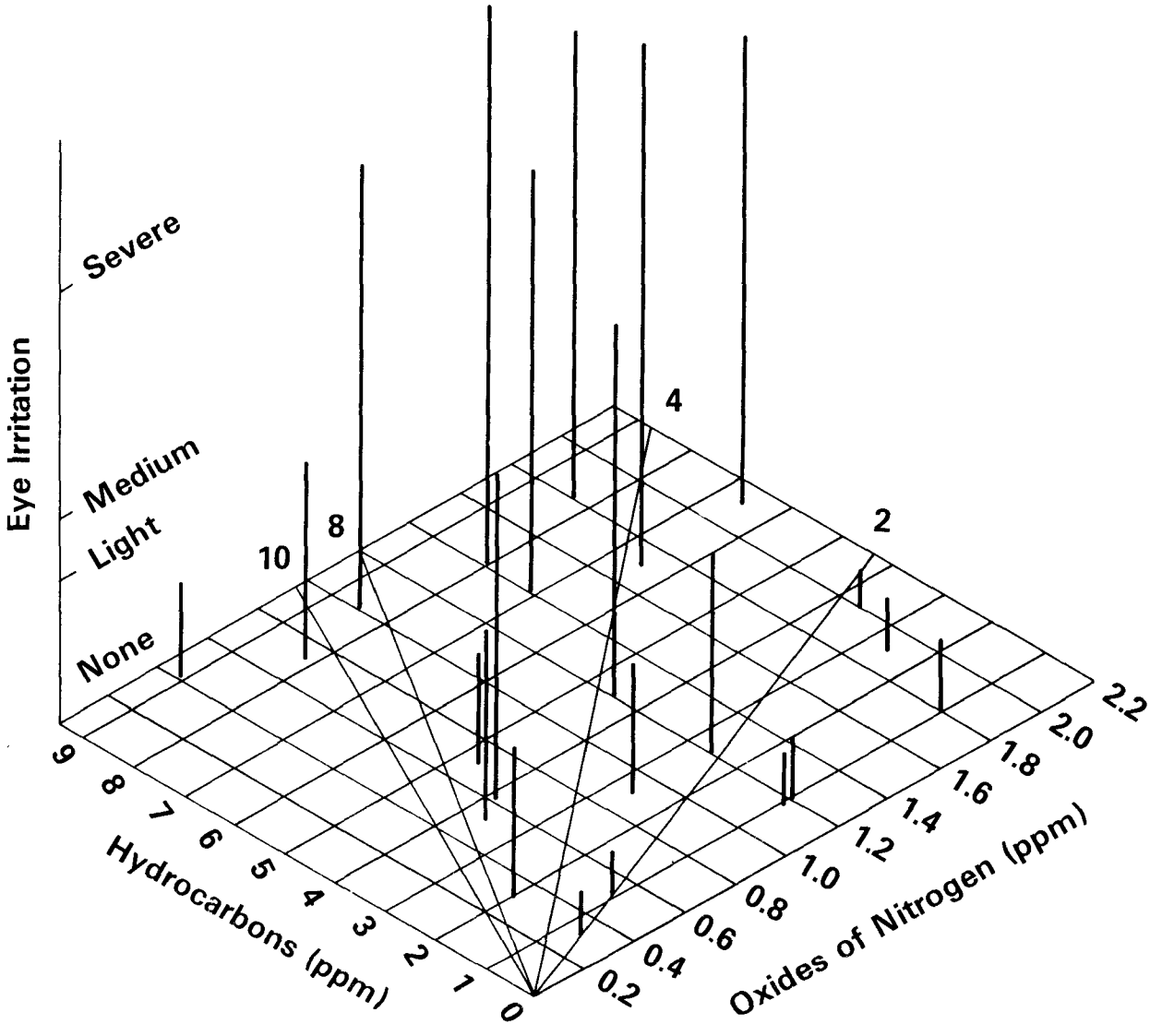


FIGURE 6-4. Relation of eye irritation to hydrocarbon and oxides of nitrogen levels in smog chamber experiments from Faith et al.[138]

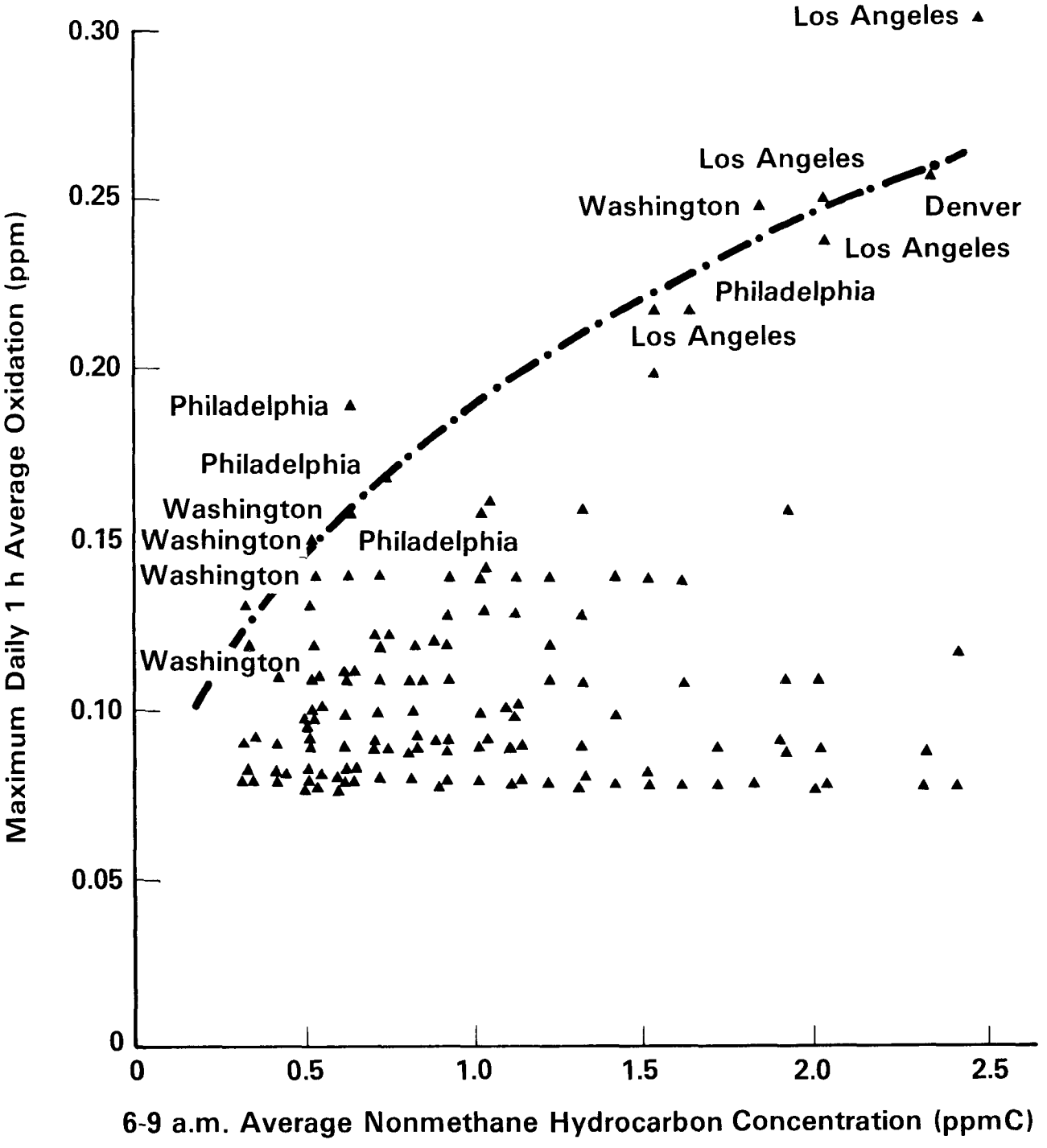


FIGURE 6-5. Relationship between the maximum daily 1-hr average oxidant levels and the 6-9 a.m. average concentration of nonmethane hydrocarbons derived from the data from several cities; from Schuck *et al.*[464]

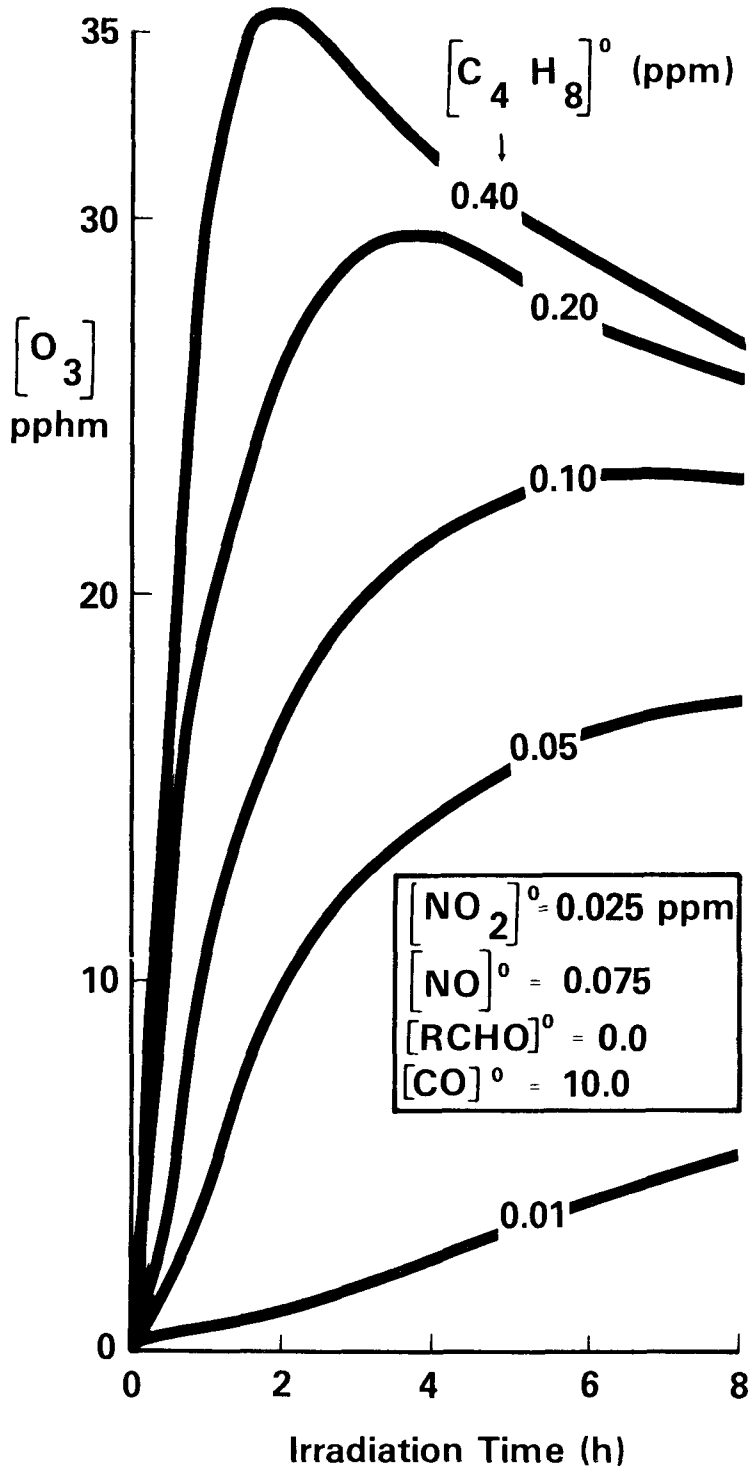


FIGURE 6-6. The theoretical time dependence of O_3 as a function of olefinic hydrocarbon (trans-2-butene) level in a sunlight irradiated (z = 40°), simulated auto exhaust polluted atmosphere; aldehydes are assumed absent initially in this case; from Calvert and McQuigg.[73]

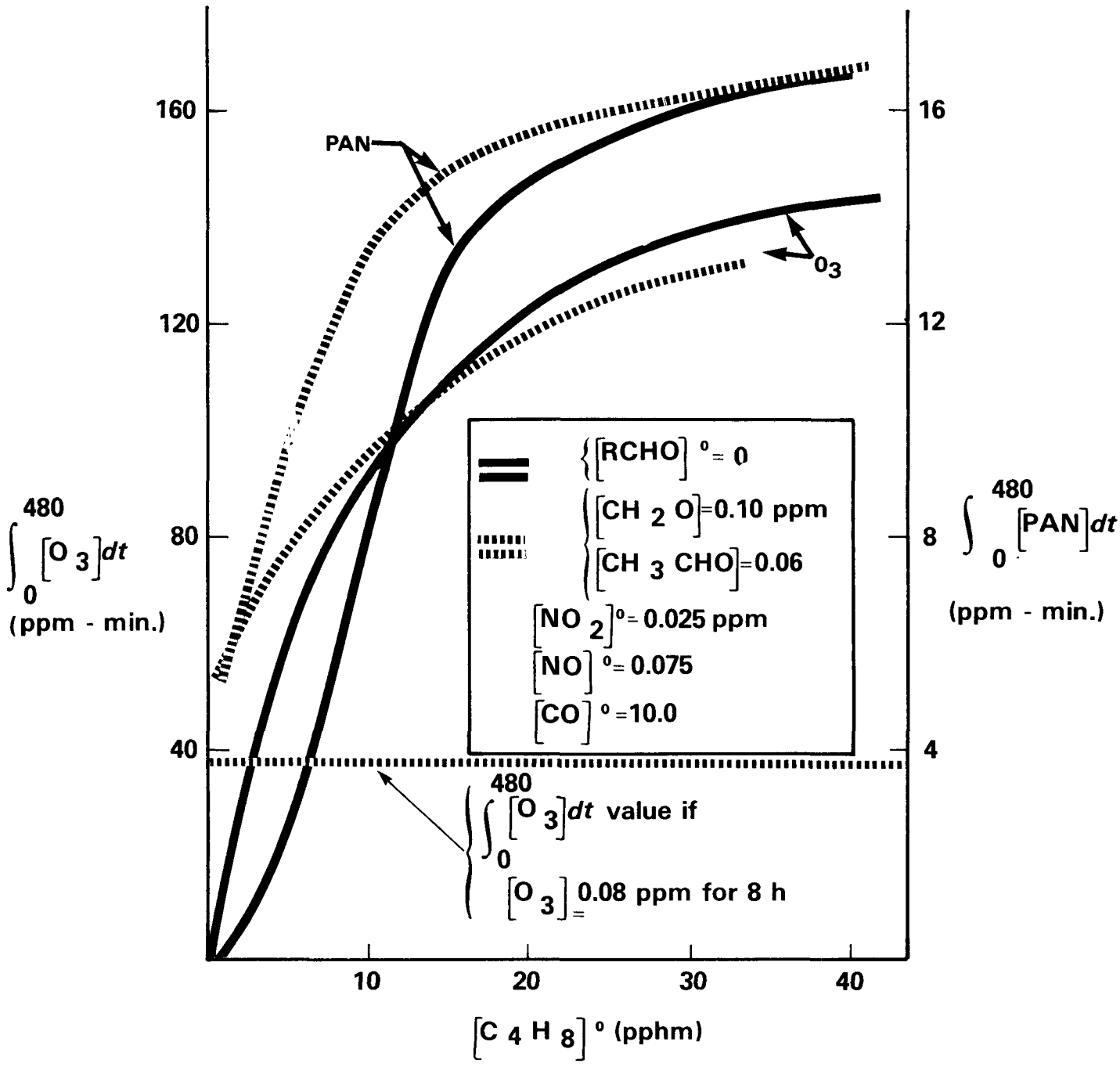


FIGURE 6-7. The theoretical effect of the reactive hydrocarbon concentration (trans-2-butene) on the 8-hr integral of the [ozone] and [peroxyacylnitrate] versus time data derived from a simulated sunlight irradiated (z = 40°), auto exhaust polluted atmosphere; data from Calvert and McQuigg.[73]

initially (solid curves). The variation of integrated oxidant and peroxyacetylnitrate levels with the initial olefinic hydrocarbon concentration, so important in the determination of standards, are altered dramatically by the initial presence of aldehydes. A reference value of the $\int_0^{480}[O_3]dt$ data is represented by the horizontal dashed line drawn at 38.6 ppm-min, which is the value of this integral if the maximum allowable 1-hr ozone concentration average in the Environmental Protection Agency (EPA) ambient air quality standards were maintained for 8 hr. These data indicate that this standard could not be met if the aldehydes remained high (formaldehyde concentration = 0.10 ppm, acetaldehyde concentration = 0.06 ppm) even if nearly all of the olefinic hydrocarbon were removed. The increase in the integrated [PAN concentration]-time levels results from the presence of aldehydes, particularly acetaldehyde. Both ozone and PAN data for runs with and without aldehydes (Figure 6-7) converge at high olefin levels and the integrals approach plateaus. The curves representing the "true" relationship between nonmethane hydrocarbons and maximum 1-hr oxidant at low hydrocarbon levels could be influenced by the aldehydes, a variable not commonly measured.

THE EFFECTS OF VARIATION OF THE CONCENTRATIONS OF THE NITROGEN OXIDES ON THE PRODUCT-TIME CURVES IN A SIMULATED POLLUTED ATMOSPHERE

For years smog chamber experiments have indicated that an inhibiting effect on product rates and certain smog manifestations can be expected at very high nitric oxide concentrations. For example, in runs at a fixed hydrocarbon concentration of about 4.5 ppm, the expected eye irritation passes through a maximum level, then decreases again as NO_x is

increased (Figure 6-4). The same inhibiting effect of high nitric oxide concentrations has been observed in product rate data determined by Tuesday and coworkers in smog chamber experiments using olefin-NO_x mixtures.[550] Predictions can be made from the simple model.

Figure 6-8 shows the time dependence of expected ozone concentration for runs at fixed hydrocarbon levels: [butene]° = 0.10 ppm; [aldehydes]° = 0; [carbon monoxide] = 10 ppm; [methane]° = 1.5 ppm; 50% relative humidity; the ratio of the initial concentrations of nitric oxide to nitrogen dioxide was held constant at 3.0. Concurrent with the progression from the very low level of an initial nitric oxide concentration of 0.15 to 15 pphm, is a gradual increase in oxidant level at 2 hr of irradiation; but further increases of the initial nitric oxide concentration to 30, 60, and 120 pphm cause a suppression in the ozone concentration-time curves. In Figure 6-9, the 2-hr integrals of the concentrations of ozone- and PAN-time data are given as a function of the initial nitric oxide concentration. The maximum in the integrals occurs near the stoichiometric mixture of one olefin to one NO_x molecule.

These data do not mean that unrestricted emissions of NO_x would solve the smog problem; however, they do imply that smog formation would be delayed. At some point downwind, the turbulent mixing, diffusion, and dilution of the NO_x-containing mixture will cause a reduction in the NO_x level which will be loaded for smog formation. The recent experience in the Los Angeles area probably relates to this phenomenon. Ozone levels are lower in downtown Los Angeles compared to previous years, probably as

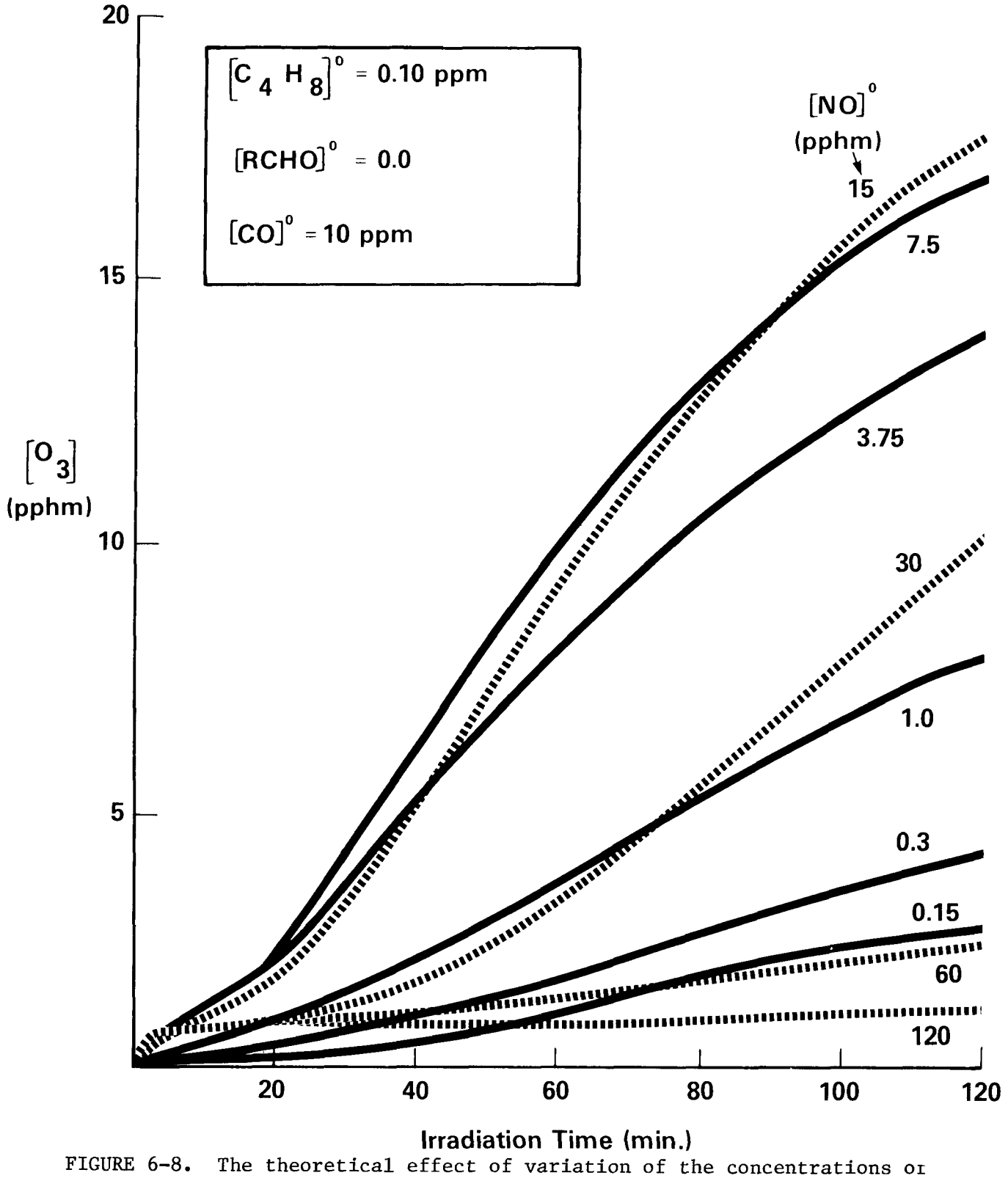


FIGURE 6-8. The theoretical effect of variation of the concentrations of the nitrogen oxides on the ozone product-time curves in a simulated polluted atmosphere; the initial concentrations of the reactants are as shown, and [nitric oxide]°/[nitrogen dioxide]° is constant at 3.0. From Calvert and McQuigg.[73]

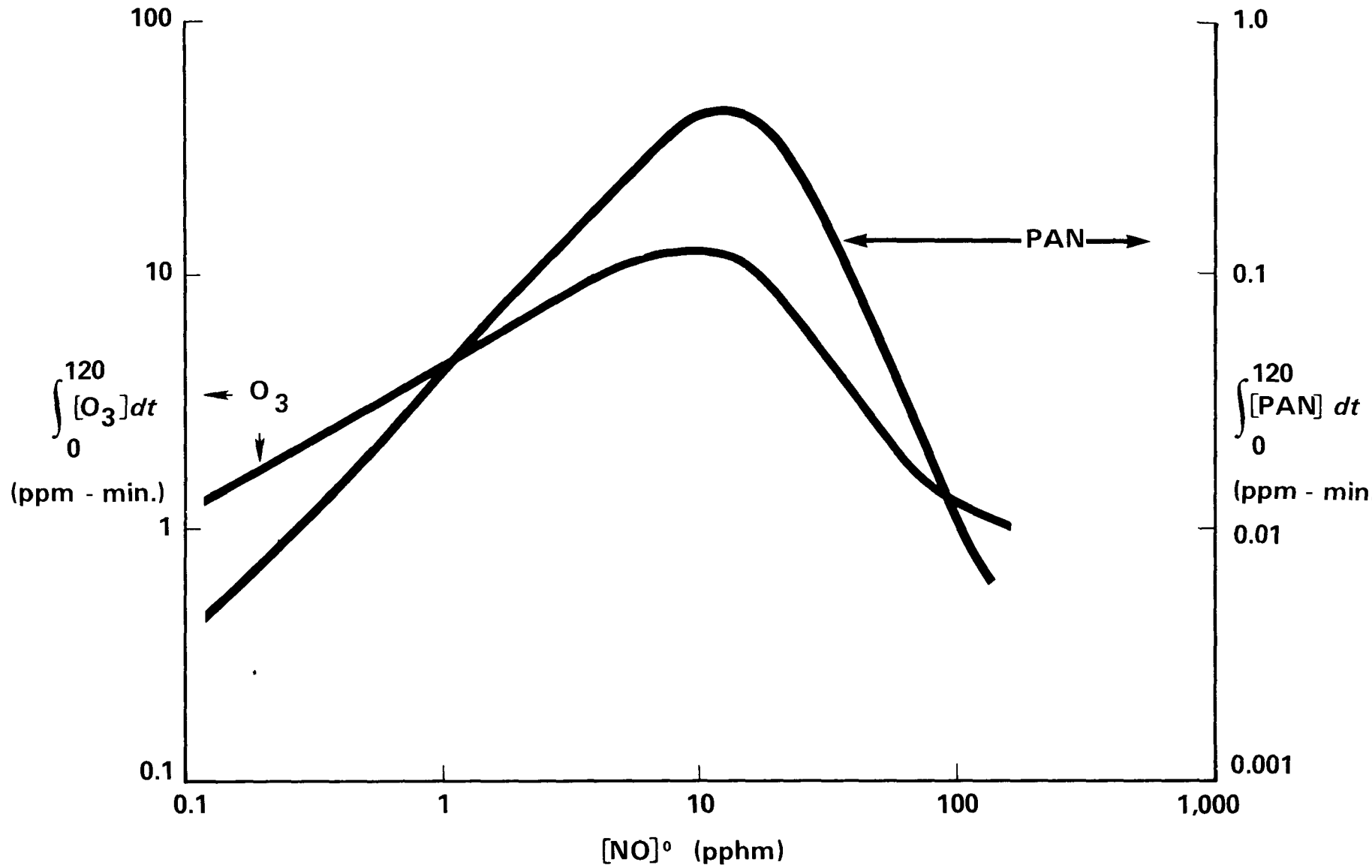


FIGURE 6-9. The theoretical effects of variation of the concentrations of the nitrogen oxides on the 2-hr integrals of the [ozone] and [peroxyacylnitrate] versus time curves obtained in the sunlight irradiated (z = 40°), simulated NO_x-hydrocarbon-polluted atmosphere; initial concentrations (ppm): [trans-2-butene]° = 0.10; [carbon monoxide]° = 10; [methane]° = 1.5; [Aldehydes]° = 0; [nitric oxide]°/[nitrogen dioxide]° = 3.0; relative humidity = 50% (25° C); from Calvert and McQuigg.[73]

a result of an increase in nitric oxide levels; yet, very high levels of ozone and other smog products are seen in the Riverside area after the Los Angeles mixture has been modified and diluted during transport to the Riverside area by prevailing winds.

THE EFFECTS OF VARIATION IN THE INITIAL CARBON MONOXIDE CONCENTRATION ON THE PRODUCT-TIME CURVES IN A SIMULATED POLLUTED ATMOSPHERE

Relatively low levels of carbon monoxide in simulated NO_x-hydrocarbon-atmospheres produce no dramatic effects in the rates of product formation. Figure 6-10 shows that increases in initial carbon monoxide concentration from 10 to 50 ppm decreases the ozone and PAN concentrations for a given sunlight exposure time. Hydrocarbons and aldehydes are attacked primarily by the hydroxyl radicals which are generated in the smog system. The reaction of hydroxyl radicals with carbon monoxide competes with the reactions of the olefins and aldehydes at higher carbon monoxide concentrations, and an increasing share of the chain regeneration of the hydroperoxyl radicals from the hydroxyl radicals is then born by the following sequence:

$$HO + CO \rightarrow H + CO_2 \tag{16}$$

$$H + O_2 + M \rightarrow HO_2 + M \tag{17}$$

$$HO_2 + NO \rightarrow HO + NO_2 \tag{18}$$

Reduced attack by hydroxyl radicals on aldehyde and olefinic hydrocarbon results in lower alkylperoxy and acetylperoxy radical formation. Thus,at higher concentrations of carbon monoxide,there is a reduction in the nitric

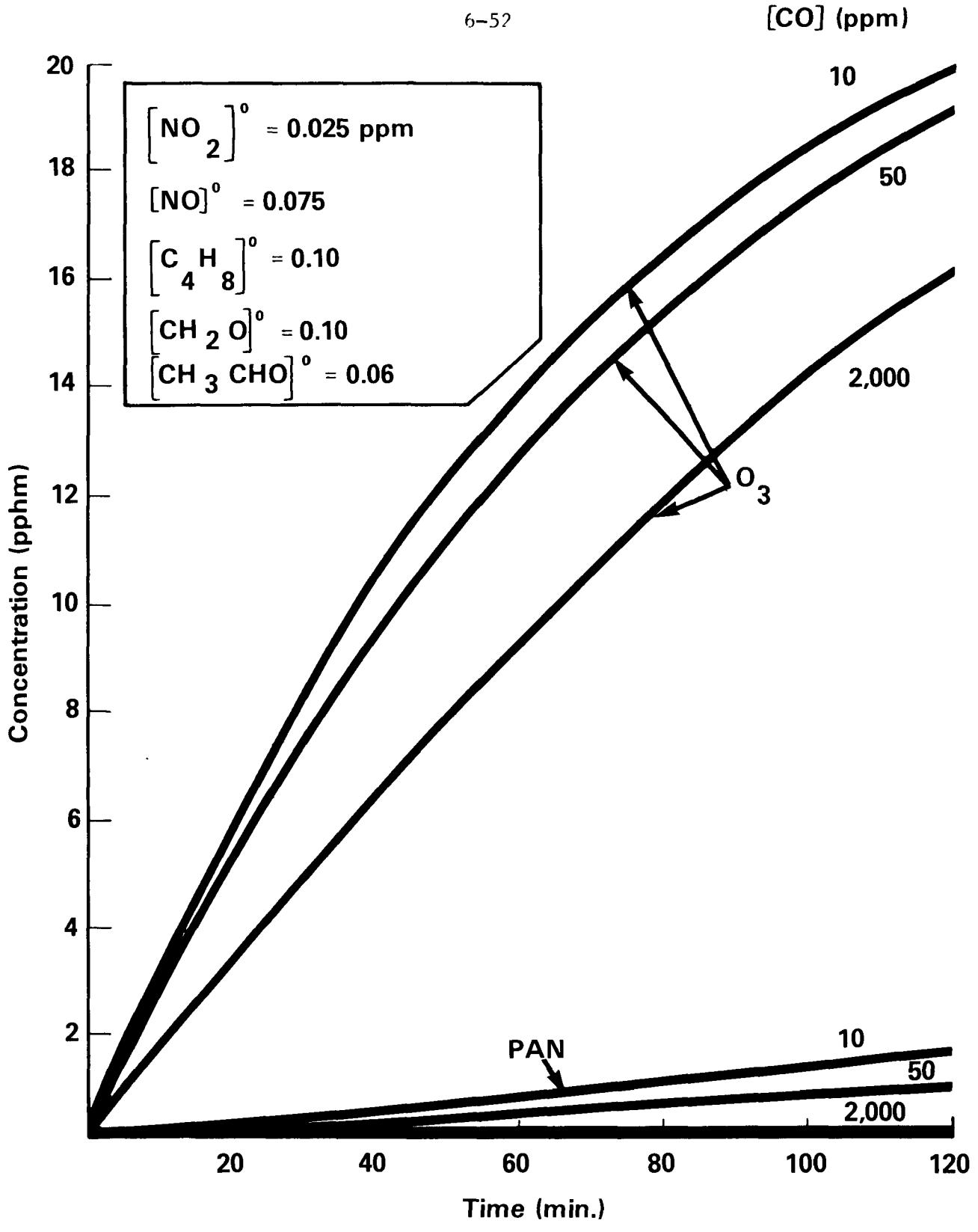


FIGURE 6-10. The theoretical effects of variation of the initial concentration of the carbon monoxide on the ozone and peroxyacylnitrate formation in a sunlight irradiated (z = 40°), simulated NO_x-hydrocarbon polluted atmosphere. From Calvert and McQuigg.[73]

oxide conversion to nitrogen dioxide associated with the alternative chain processes involving the hydrocarbons and aldehydes. As a result the ozone concentrations are slightly lowered. The lowered rate of acetylperoxyl radical ($CH_3\overset{O}{\overset{\|}{C}}O_2$) generation is principally caused by the less important attack by hydroxyl radicals on acetaldehyde and olefin. This is reflected in a lowered PAN concentration since PAN comes largely from the following sequence:

$$CH_3CHO + HO \rightarrow CH_3CO + H_2O \tag{47}$$

$$CH_3CO + O_2 \rightarrow CH_3\overset{O}{\overset{\|}{C}}O_2 \tag{48}$$

$$CH_3\overset{O}{\overset{\|}{C}}O_2 + NO_2 \rightarrow CH_3\overset{O}{\overset{\|}{C}}O_2NO_2 \tag{49}$$

At extremely high levels of carbon monoxide (2,000 ppm), PAN formation is practically eliminated since attack of hydroxyl radicals on acetaldehyde and olefin no longer competes with that of carbon monoxide [Reaction (16)]. It is not suggested that PAN formation be reduced in our polluted atmospheres by removing controls on carbon monoxide emissions since the toxic properties of carbon monoxide outweigh the usefulness gained by PAN reduction.

There is one other unexpected aspect of the chemistry of carbon monoxide-containing atmospheres. If the hydrocarbon and aldehyde impurities were entirely removed from the atmosphere and carbon monoxide allowed to rise along with the oxides of nitrogen, then carbon monoxide could act as an effective reactant, pumping nitric oxide to nitrogen dioxide, and contributing to ozone levels.[75]

Figure 6-11A plots the experimentally-determined concentration/time from a smog chamber study of W.E. Wilson, Jr. and D.F. Miller (personal communication). The concentrations in the chamber at the start of the irradiation were: [nitric oxide]° = 51 pphm; [nitrogen dioxide]° = 10 pphm; and [carbon monoxide]° = 100 ppm. The ozone concentration climbs to a level of about 13 pphm after 5 hr of irradiation. Figure 6-11B shows the computer simulation of these product rates based on the mechanism of photochemical smog formation described by Demerjian *et al.*[105] Figure 6-11C shows other expected products for which analyses were not made. The driving force for this reaction is the generation of hydroxyl radicals from nitrous acid photolysis:

$$HONO + \text{sunlight } (2{,}900\text{-}4{,}000\ \mathring{A}) \rightarrow HO + NO \qquad (13)$$

If the reactions of nitrous acid formation and destruction,

$$H_2O + NO + NO_2 \rightarrow 2HONO \qquad (32)$$

$$2HONO \rightarrow H_2O + NO + NO_2, \qquad (33)$$

occur in the atmosphere at rates comparable to those observed in chambers, then the carbon monoxide-effect can be significant for relatively low ambient levels of NO_x and carbon monoxide (see Table 6-6).[75] Ozone concentrations of about 7 pphm, approaching the 1-hr maximum level of 0.08 ppm, are expected after about 2.5 hr of irradiation. Further study is necessary to define the rates of nitrous acid formation in the real atmospheres before a conclusion can be reached concerning this potentially important carbon monoxide effect.

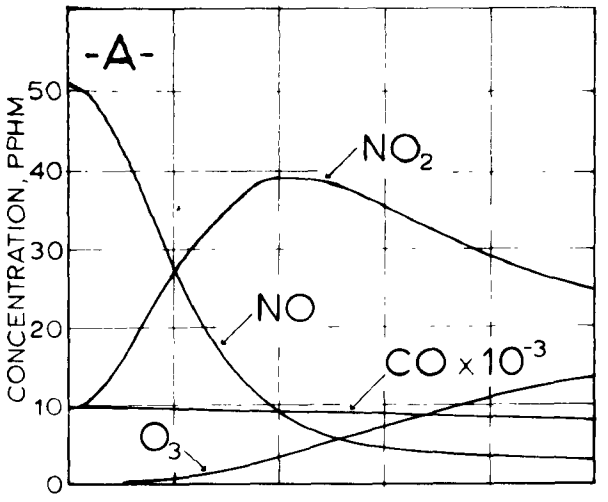


FIGURE 11A. Initial concentrations, [nitric oxide]° = 51 pphm; [nitrogen dioxide]° = 10 pphm; [carbon monoxide]° = 100 ppm; relative humidity, about 13% at 32.78° C.

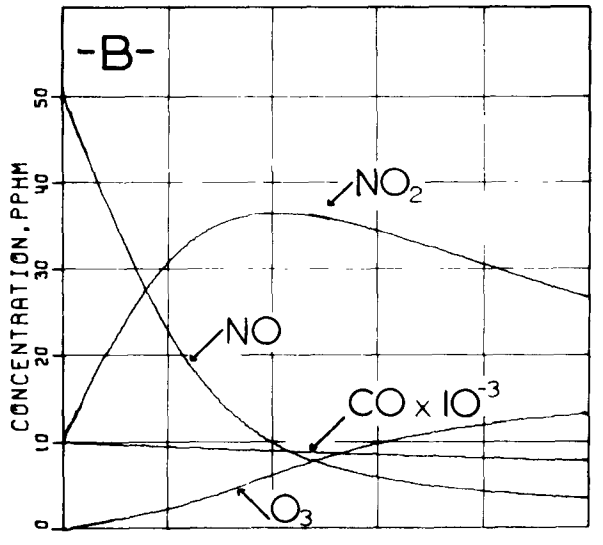


FIGURE 11B. Computer simulation for the experimental conditions employed in Figure 6-11A.

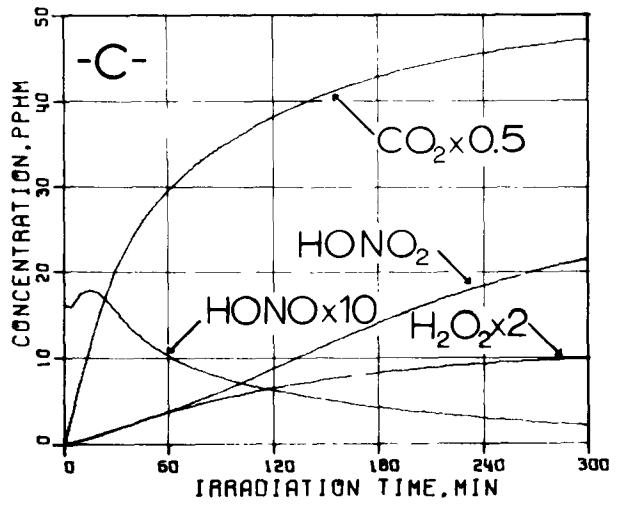


FIGURE 11C. Computer simulation of the expected time dependence of the minor products for the conditions employed in Figure 6-11A.

FIGURE 6-11. The photooxidation of nitric oxide in carbon monoxide-containing mixtures; comparison of experimental and computer simulated chemical changes in nitric oxide-nitrogen dioxide-carbon monoxide mixtures irradiated in moist air; experimental analysis for these products was not made. From W.E. Wilson, Jr., and D.F. Miller, personal communication. Data are unpublished results from studies conducted in a 17.2 m^3 smog chamber at Battelle-Columbus Laboratories, 1970.

TABLE 6-6

The Effect of Carbon Monoxide Level on the Concentrations of Products Formed in the Sunlight-Irradiated Moist Atmospheres[a]

Relative Humidity, % (25° C)	Concentrations, pphm, at t = 151.6 min										
	$[CO]^{\circ}$ (ppm)	$[NO_2]$max (ppm)	t_{max}, min	$[NO_2]$	$[NO]$	$[O_3]$	$[N_2O_5]$ x 10^4	$[HNO_2]$	$[HNO_3]$	$[H_2O_2]$ x 10^2	$[CO_2]$
100	0	2.50	0	0.64	7.98	0.17	0.00	0.34	1.0	0.03	0
	1	2.50	0	1.30	6.77	0.40	0.04	0.41	1.5	0.46	1.7
	5	3.37	148	3.37	3.39	2.05	1.7	0.33	2.9	19.1	8.2
	10	4.41	117	4.32	2.32	3.82	6.9	0.25	3.1	61.7	12.6
	25	5.50	102	5.20	1.81	5.88	17.9	0.22	2.8	145	18.0
	50	6.07	94	5.65	1.68	6.89	25.6	0.21	2.5	203	20.9
	75	6.30	92	5.83	1.64	7.28	29.2	0.21	2.3	229	22.3
	100	6.42	91	5.94	1.62	7.49	31.2	0.21	2.2	244	22.9
50	0	2.50	0	0.90	8.00	0.24	0.01	0.26	0.84	0.01	0
	1	2.50	0	1.44	7.20	0.42	0.04	0.29	1.1	0.19	1.1
	5	3.46	232	3.32	4.61	1.49	1.0	0.26	1.8	6.5	5.4
	10	4.56	176	4.53	3.22	2.90	5.0	0.21	2.0	24.7	9.0

TABLE 6-6 - continued

Relative Humidity, % (25° C)	Concentrations, pphm, at t = 151.6 min										
	$[CO]^\circ$ (ppm)	$[NO_2]max$ (ppm)	t_{max}, min	$[NO_2]$	$[NO]$	$[O_3]$	$[N_2O_5]$ x 10^4	$[HNO_2]$	$[HNO_3]$	$[H_2O_2]$ x 10^2	$[CO_2]$
50	25	5.70	140	5.69	2.32	5.03	18.0	0.16	1.8	72.6	13.7
	50	6.28	128	6.24	2.07	6.17	28.8	0.15	1.5	110	16.3
	75	6.52	125	6.46	2.00	6.62	33.9	0.15	1.4	128	17.4
	100	6.65	123	6.58	1.96	6.88	36.9	0.14	1.3	138	18.0

[a] This atmosphere has the following initial concentrations: $[NO_2]^\circ$ = 2.50 ppm; $[NO]^\circ$ = 7.50 pphm; and $[CO]^\circ$ as indicated. z = 40°. Temperature: 25° C.

In theory, the same influence noted for carbon monoxide can be expected in mixtures of the reactive paraffin hydrocarbon with NO_x that is free from olefinic hydrocarbons, provided that the rates of Reactions (32) and (33) in the real atmosphere are comparable to those in chambers. Because the values of the rate constants for the homogeneous and heterogeneous experimental olefin-free reactions are uncertain, the key questions for these systems cannot be definitively answered. However, these unexpected effects should be considered in the development of more detailed air quality standards.

Because of the technological difficulties in removing NO_x and carbon monoxide from auto exhaust and NO_x from stack gases, ozone levels may continue to plague many urban areas, although a near total removal of the reactive hydrocarbons might be effected.

THE THEORETICAL MASS BALANCE OF THE NITROGEN-CONTAINING COMPOUNDS FORMED IN THE SIMULATED POLLUTED ATMOSPHERE

The nature of the nitrogen-containing products formed in photochemical smog has been a matter of considerable interest among scientists. Note the distribution of the products predicted by the present model. Figure 6-12 plots the percentage of total nitrogen present in selected compounds at various sunlight irradiation times in the synthetic NO_x-hydrocarbon-aldehyde polluted atmosphere. Nitric oxide conversion to nitrogen dioxide is followed by a continuing transformation of the nitric oxide and nitrogen dioxide into two major products--nitric acid and PAN. A much smaller quantity of methyl nitrate builds up as the reactions continue. The percentage of the total nitrogen contained in the reactive transients, symmetrical nitrogen trioxide and dinitrogen pentoxide, is negligible. Nitrous acid and methyl

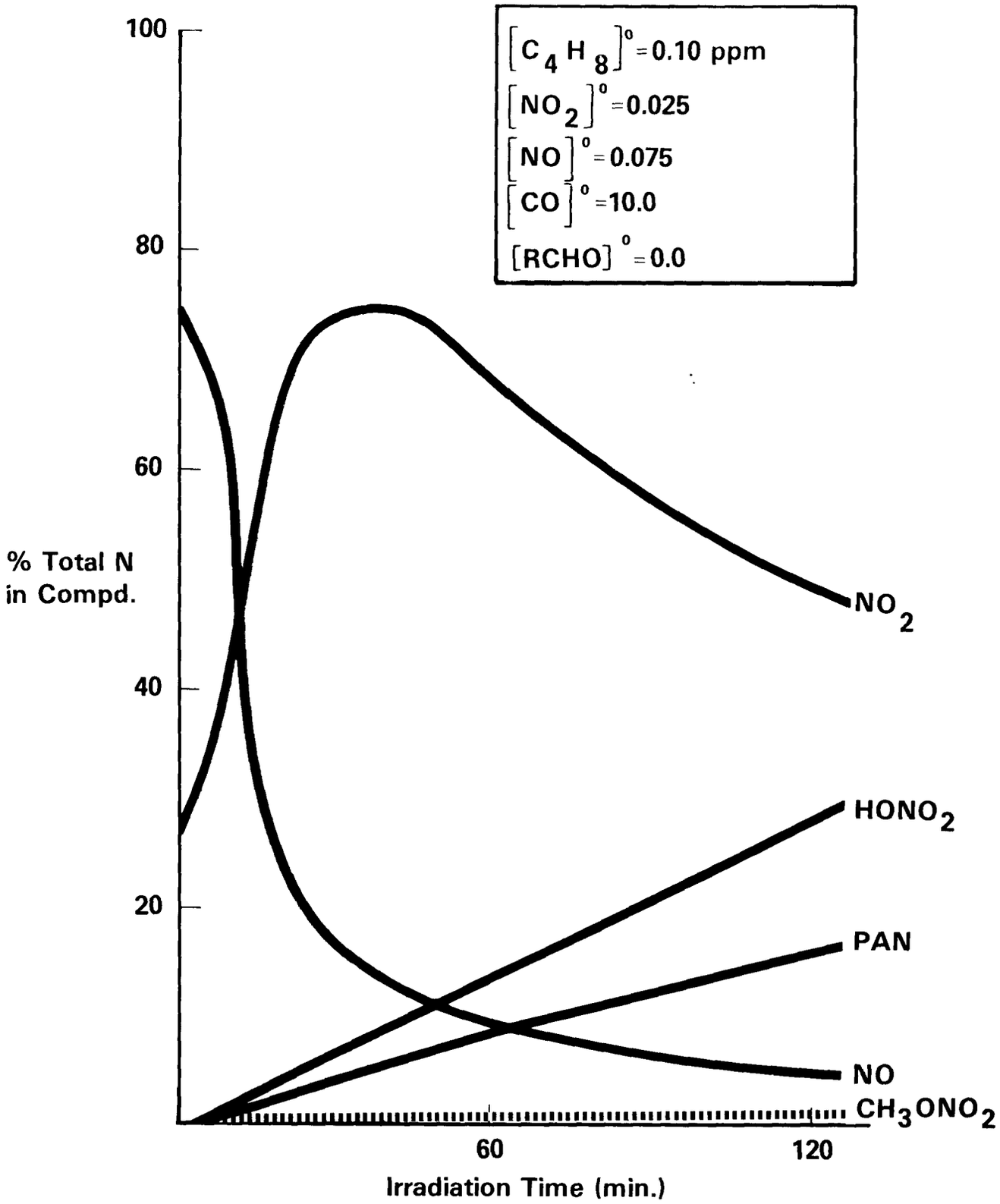


FIGURE 6-12. Theoretical distribution of the nitrogen-containing products formed in the simulated, sunlight-irradiated (z = 40°), NO_x-hydrocarbon-aldehyde polluted atmosphere; the initial composition of the mixture is the same as in Figure 6-2.[73]

nitrite are formed but their concentrations do not climb appreciably since both compounds are rapidly dissociated by sunlight.

$$HONO + \text{sunlight } (2{,}900\text{-}4{,}000\ \mathring{A}) \rightarrow HO + NO \tag{13}$$

$$CH_3ONO + \text{sunlight } (2{,}900\text{-}4{,}000\ \mathring{A}) \rightarrow CH_3O + NO \tag{50}$$

The nitric acid, PAN, and methyl nitrate absorb sunlight only weakly. Since other reactions which remove them are not fast, they accumulate. In the real urban atmosphere nitric acid does not build up as expected from the present reaction scheme; but presumably, the nitric acid that is formed reacts with certain basic impurities in the atmosphere: ammonia, the oxides of the metals, etc.

Because quantitative treatment of such heterogeneous reactions is not possible at this time, such potentially important steps have not been included in the present simulation. However, they must occur in the real atmosphere, and nitric acid may be converted efficiently to ammonium nitrate if the concentration of ammonia impurity is sufficiently high. Ammonium nitrate comprises approximately 10-15% of the total airborne particles in composite samples collected in the Los Angeles area from 1971-1972.[183]

The presence of nitrates in food and water and the possible direct deposition of the soluble ammonium nitrate salt in the respiratory system have been of concern because of possible biological effects.[23,183] The formation of the large amounts of nitrate predicted by the model has never been observed in studies attempting nitrogen balances in the urban atmosphere. Perhaps this indicates the incompleteness of the mechanism, or,

possibly, that the rate constant for nitric acid formation, derived from the limited published data, is slightly larger than the true value:

$$HO + NO_2 + M \rightarrow HONO_2 + M \qquad (15)$$

It is also possible that there are some as yet unrecognized analytical problems associated with nitric acid and nitrate determinations in the urban atmospheres.

FORMATION OF NITRATE SALTS IN THE ATMOSPHERE

Nitrate salts may be formed in the atmosphere through a variety of reaction paths. One path, similar to the mechanism forming sulfuric acid (H_2SO_4) and sulfate salts,[73,77,557] is not an important source of nitric acid and nitrate salt. The low vapor pressure of pure sulfuric acid results in a rapid homogeneous nucleation at partial pressures of sulfuric acid in the range 10^{-8} to 10^{-10} torr, according to Doyle[119] and Kiang *et al.*[281] The hydration of sulfuric acid droplets is thermodynamically favorable over a wide range of relative humidities, and the aerosol particles of the sulfuric acid solution grow as they take on water. This process continues until the vapor pressure of the water over the sulfuric acid solution equals the partial pressure of water in the atmosphere. In comparison, the vapor pressure of nitric acid is very high, and homogeneous nucleation of nitric acid aerosol formation in a moist atmosphere is not expected to occur under normal atmospheric conditions.[281] However, nitric acid does form some mixed compounds or complexes of considerable stability in sulfuric acid solutions[174] and may become incorporated within sulfuric acid droplets at the lower temperatures of the upper atmosphere.[79,433] To assess the importance of this possible removal pathway for nitric acid requires the measurement of the nitric acid vapor pressures over solutions of sulfuric acid-water-nitric acid at various compositions and temperatures.[79]

Presumably such a chemical entrapment of nitric acid would be followed by neutralization of the aerosol with atmospheric ammonia (NH_3), ultimately producing ammonium nitrate (NH_4NO_3) and ammonium sulfate [$(NH_4)_2SO_4$].

The direct homogeneous capture of gaseous nitric acid by gaseous ammonia ($NH_3 + HONO_2 \rightarrow NH_4NO_3$) may be a significant source of ammonium nitrate salt in the atmosphere if the ammonia levels in the polluted atmosphere are sufficiently high. Countess and Heicklen[92] have found that the analogous homogeneous reaction between ammonia and hydrogen chloride (HCl) gases occurs with a rate constant of 2.8×10^{-2} ppm^{-1} min^{-1}. Assuming the ammonia-nitric acid reaction to have a rate constant of this magnitude and concentrations of 1 pphm, the homogeneous rate of ammonium nitrate formation is estimated to be 2.8×10^{-6} ppm min^{-1}. This rate is a significant fraction of the theoretical rate of nitric acid formation in Reactions (14) and (8). This nitric acid removal path is worthy of further quantitative consideration and study. The rate constant of the nitric acid-ammonia reaction must be measured, and the ambient levels and the diurnal pattern of gaseous ammonia present in the urban atmosphere must be established before any realistic modeling of this reaction can be made.

Most of the reaction paths for nitrate salt formation considered thus far involve the reactions of gaseous nitric acid. It is, therefore, important to consider the probable sources of this compound in a smoggy atmosphere. The analysis of the systems points to two major homogeneous sources of nitric acid in photochemical smog mixtures:

$$HO + NO_2 + M \rightarrow HONO_2 + M \qquad (14)$$

$$N_2O_5 + H_2O \rightarrow 2HONO_2 \qquad (8)$$

The rate nitric acid forms through abstraction of hydrogen from molecules and radicals by the intermediate, symmetrical nitrogen trioxide present in photochemical smog is much less important than Reactions (14) and (8) for normal atmospheric conditions.

Rate data for Reactions (14) and (8) are not quantitatively established, but they are quantitatively useful in estimating the approximate rates and the relative importance of the two reactions in a simulated sunlight-irradiated, NO_x-hydrocarbon-polluted atmosphere. The best current estimates of the homogeneous rate constant for Reaction (14) vary within the range 1.5×10^4 to 0.46×10^4 ppm^{-1} min^{-1}, expresed as apparent second order reactions with M=1 atm of nitrogen gas. These values have been estimated by Demerjian *et al.*[105] and Tsang.[546] To date, the best kinetic data for the homogeneous Reaction (8) are those of Morris and Niki[368] which give the rate constant $k_8 \leq 1.9 \times 10^{-5}$ ppm^{-1} min^{-1}. These rate data and concentrations of the intermediates and reactants for a simulated, typical NO_x-hydrocarbon-polluted atmosphere yield the following rates of nitric acid formation:

$$Rate_{14} \cong 2.0 \times 10^{-5} \text{ ppm min}^{-1} \text{ (using rate data of ref. 508)}$$

$$Rate_{14} \cong 6.6 \times 10^{-5} \text{ ppm min}^{-1} \text{ (using rate data of ref. 105)}$$

$$Rate_{8} \leq 1.6 \times 10^{-5} \text{ ppm min}^{-1}$$

The estimated rate of Reaction (8) is probably much higher than the actual homogeneous rate since it is very difficult to extract the rate constant k_8 from experiments in which a large heterogeneous component of

the reaction dominates the rate. Thus, a lower value ($k_8 = 5 \times 10^{-6}$ ppm^{-1} min^{-1}) was required to match chamber data in some of the simulation studies of Demerjian *et al.*[105] The nitric acid formed homogeneously in the smoggy atmosphere probably results largely from Reaction (14). The rate of nitrogen dioxide conversion to nitric acid in this reaction amounts to about 2.4-7.9% per hr at the 30 min irradiation point picked for the simulation. This range is slightly higher,[183] but not out of line with the formation rates of the aerosol containing nitrate ions observed in Los Angeles smog (Whitby, personal communication). Nitric acid conversion to aerosol containing nitrate ions may not be complete on the time scale selected since capture mechanisms ultimately require ammonia (or another basic compound) as a neutralizing agent, which may be in short supply. The typical homogeneous rate of nitric acid formation given above should, therefore, represent a maximum rate of nitrate salt formation from these particular reactions.

The nature of the nitrogen-containing products expected in smog simulations, which neglect the heterogeneous removal processes, reveal that nitric acid, peroxyacetylnitrate, and methyl nitrate are the major sumps for the nitrogen oxides in smog.[73] As described previously, the conversion of nitric oxide to nitrogen dioxide is followed by a continuing transformation of nitric oxide and nitrogen dioxide into two major products--nitric acid and peroxyacetylnitrate.

The Demerjian *et al.*[105] estimate for k_{14} has been used in these simulations. Thus, the rate of nitric acid formation may be lower somewhat if the equally reliable estimate of Tsang is employed for

the rate constant k_{14}.[508] It follows that the amount of peroxyacetylnitrate expected may exceed the nitric acid formed.

The simulation predicts that a large fraction of the NO_x ultimately ends up as peroxyacetylnitrate. It is known that peroxyacetylnitrate hydrolyzes in solution to form nitrite ions.[119] Therefore, the heterogeneous removal of peroxyacetylnitrate may follow encounters with an absorption by the aerosol droplet, and nitrite salts may be formed from the peroxyacetylnitrate if the solution's pH remains sufficiently high through ammonia molecule capture by the aerosol.

The extent to which nitrate and nitrite salts result from gaseous nitrogen dioxide and nitric oxide absorption into aerosol droplets is not yet known. But the following reactions can lead to nitrate in principle, if sufficient ammonia, or other basic compounds, neutralize the acids formed upon formation of the solution:

$$2NO_2 + H_2O \rightleftarrows H^+ + NO_3^- + HONO \quad \text{(Aqueous Solution)}$$

$$NO + NO_2 + H_2O \rightleftarrows 2HONO \quad \text{(Aqueous Solution)}$$

$$HONO + OH^- \rightarrow H_2O + NO_2^- \quad \text{(Aqueous Solution)}$$

$$2NO_2^- + O_2 \text{ (in aerosol solution)} \rightarrow 2NO_3^-$$

$$NO_2^- + O_3 \text{ (in aerosol solution)} \rightarrow NO_3^- + O_2$$

These potential heterogeneous reactions forming nitrate may be sufficiently rapid to account for all of the observed nitrate formation according to Hidy.[232,233] However, this conclusion remains open to question at this

incomplete stage of knowledge concerning these mechanisms. A major problem with the former "standard" method for nitrogen dioxide analysis in the atmosphere (Jacobs-Hochheiser method) is the inefficient collection of nitrogen dioxide by the basic solution used in the bubblers in the collection train. Although the nitrogen dioxide should dissolve readily in the highly basic bubbler solution, the rate of solution is also a function of the interfacial area. In the acidic or near neutral aerosol droplets finely dispersed in the atmosphere, however, solution is slow and insignificant. Further study of this system is necessary to evaluate its significance in nitrate salt formation in smog and health-related problems.

De Pena *et al.*[107] have observed in the laboratory that ammonium nitrate is generated by a complex reaction between ammonia and ozone. There is evidence of heterogeneity associated with the rate-determining step in the overall reaction. It is not clear if any fraction of the rate is truly homogeneous in character.

A variety of additional homogeneous and heterogeneous reactions have been considered in simulations of reactions in atmospheres containing sulfur dioxide (SO_2).[73,183] Space limitations prevent their detailed considerations. However, such reactions are expected to lead to sulfuric acid, sulfate salts, and other sulfur containing compounds which presumably contribute to aerosol formation.[557]

There are many uncertainties concerning the mechanism generating sulfate and nitrate salts in the sunlight-irradiated, NO_x-sulfur dioxide-hydrocarbon polluted atmosphere. However, theoretical homogeneous rates of transformation of NO_x to nitric acid and sulfur dioxide to

sulfur trioxide and sulfuric acid are significant. Free radicals generated in the smog reactions probably are the important reactants that promote these changes in the real atmosphere.

Several heterogeneous paths may independently effect these transformations, although their importance is also uncertain. The heterogeneous reactions leading to the accumulation of acids in the aerosol particles and the subsequent neutralization reactions must be significant in the overall conversion of these acids to the salts.

Extensive fundamental research related to these processes is necessary to establish the relative importance of the different possible mechanisms for the atmospheric conversion of sulfur dioxide and NO_x to sulfate and nitrate salts and to develop realistic control procedures. An aerosol characterization study by Appel *et al.*[21] relates analytical data to possible formation mechanisms.

HEALTH EFFECTS RELATED TO REACTIVE INTERMEDIATES FORMED IN THE SUNLIGHT-IRRADIATED, URBAN ATMOSPHERE

Since the early days of research in photochemical smog mechanisms, there has been interest and speculation concerning the influence on biological systems of the transients in smog (such as triplet-*P* oxygen atoms, etc.). For example, many years ago Johnston,[265] and Leighton and Perkins[315] discussed this possibility in relation to eye irritation. The probability that these species dominate the chemistry within the atmosphere suggests that they might react equally efficiently with various biological systems.

These intermediates probably do not act directly on lung tissue since most of the driving force for radical formation in the atmosphere

comes from the sunlight. Therefore, when a portion of the atmosphere is inhaled into the darkness of the lung, the intermediates should die too quickly to act on the lung tissue. But, more detailed investigation indicates that the lifetime of some intermediates is not so short after all. The present knowledge of the reactions of the transients and their rate constants encourages reconsideration of the possibility of this direct influence of the intermediates.

Inhalation of urban air removes this air from the sunlight turning off most of the radical forming steps.

The transients then decay by the various paths characteristic of these species and the other contaminant molecules present. The time required for each transient species in the simulated atmosphere to decay to one-half of its initial concentration when removed from the sunlight into a dark region,such as the lung,is compared in Table 6-7. (No dilution effects were considered in deriving the data of Table 6-8.) Calculations have been made for the concentration conditions which are present at 2, 30, and 60 min sunlight irradiation of our polluted atmosphere employed previously (Figure 6-2, solid curves). Compare these times with the period for the inhalation cycle, about 3.5×10^{-2} min. If the half-life of the species is very short with respect to this time, then there should be few species left as the air reaches the lung.

As expected, the ozone has a half-life as long as 4 min when nitric oxide and olefinic hydrocarbon concentrations decrease after 60 min of irradiation. It lives a slightly shorter time ($t_{1/2} \simeq 0.4$ min) when a sample is taken earlier during sunlight exposure. These times are sufficient to allow levels of ozone in the lung to approach those of the

atmosphere itself. Furthermore, the ozone-olefin reaction will continue to occur in the dark, generating within the lung the reactive ozonide, the peroxy diradical (the so-called zwitterion), and various other free radical intermediates unique to this pollutant.

The life of the hydroperoxyl radical may be comparable to the breathing cycle (Table 6-7). This species should be considered as a possible source of health effects. The conclusions concerning the possible roles of singlet-delta oxygen and symmetrical nitrogen trioxide are not as clear cut, since the half-times are a factor of 10 to 100 times shorter than that of the inhalation cycle. These species might seem unimportant; however, look at the singlet-delta oxygen system in more detail. Firestone and Calvert[146] have estimated the time dependence of singlet-delta oxygen in a model lung by computer simulation of a breathing cycle and the kinetics of transport and decay of singlet-delta oxygen. The model involves the initial impurity concentrations of the simulated atmosphere employed in Figure 6-2. The level of singlet-delta oxygen at the steady state in this irradiated atmosphere is about 6.2×10^{-6} ppm. Two different mathematically simplified models of the air-flow time pattern have been employed in these calculations: a linear lung expansion model and a sinusoidal variation in the lung volume with time. During the inhalation cycle of about 0.035 min, 500 cm^3 of air is brought into a 500 cm^3 volume of retained gases in the lung. Perfect mixing of this "new" air occurs during the turbulent inhalation and it is saturated with water vapor. Then 500 cm^3 of the 1,000 cm^3 total lung gases is exhaled in a cycle of equal duration (Figure 6-13).

TABLE 6-7

Theoretical First Halflives for Some Reactive Chemical Species in Photochemical Smog When Moved from the Irradiated, Simulated Polluted Atmosphere to a Dark Volume[a]

Species	Time into Simulated Irradiation for which concentration conditions were chosen, ppm		
	2 min	30 min	60 min
$O(^1D)$	8.9×10^{-12}	8.9×10^{-12}	8.9×10^{-12}
$O(^3P)$	1.7×10^{-7}	1.7×10^{-7}	1.7×10^{-7}
HO	3.9×10^{-5}	5.0×10^{-5}	6.0×10^{-5}
NO_3	7.1×10^{-4}	1.1×10^{-3}	1.5×10^{-3}
$O_2(^1\Delta_g)$	9.0×10^{-4}	9.0×10^{-4}	9.0×10^{-4}
HO_2	4.2×10^{-2}	9.5×10^{-2}	1.1×10^{-1}
O_3	4.3×10^{-1}	2.1	4.2

[a]Composition of simulated polluted atmosphere same as that in Figure 6-2.

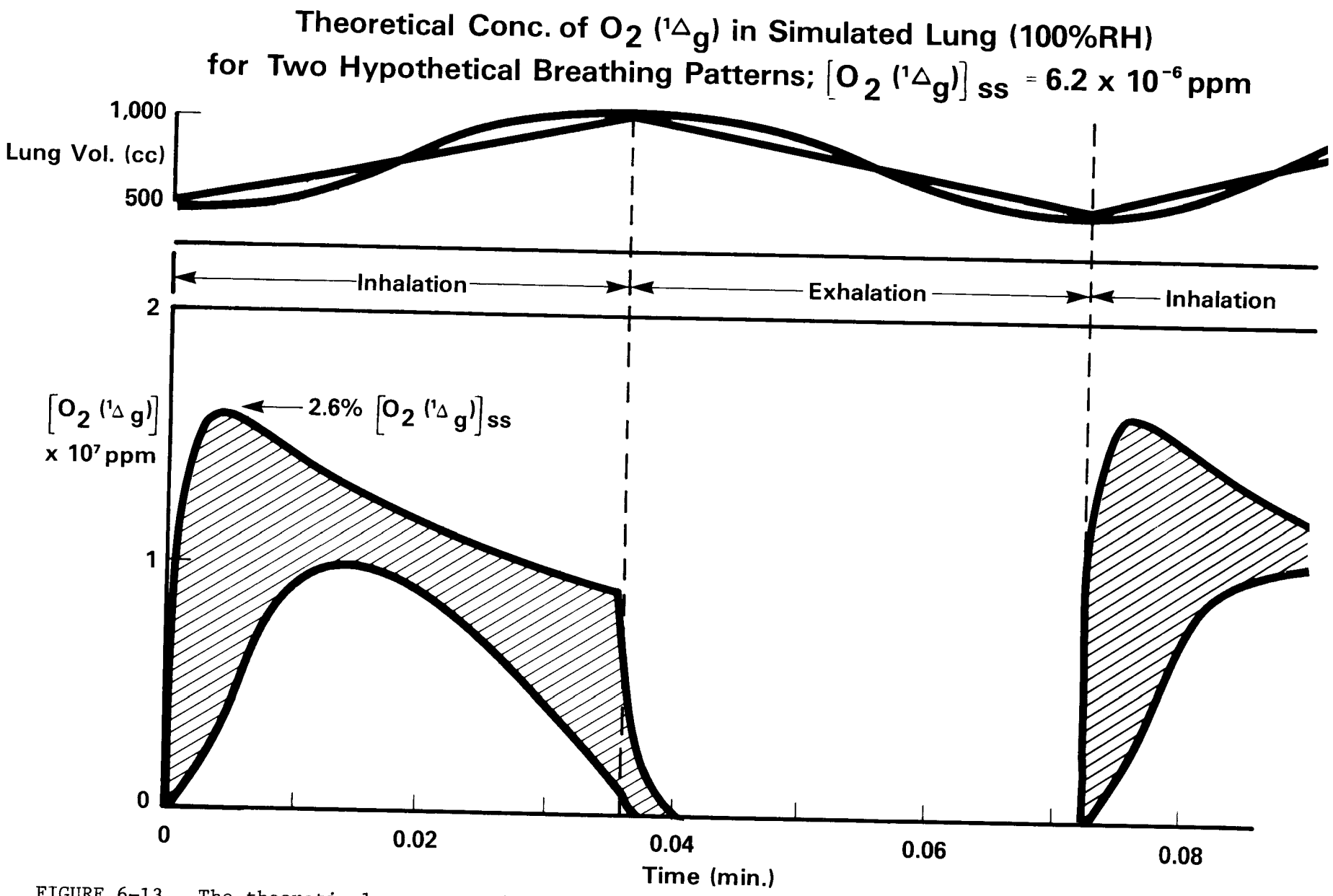


FIGURE 6-13. The theoretical concentration of singlet-delta oxygen in a simulated lung for two hypothetical breathing patterns; the steady state concentration of the singlet-delta oxygen in the sunlight irradiated atmosphere from which the air originates is at 6.2×10^{-6} ppm. From Firestone and Calvert.[146]

In Figure 6-13, the cross-hatched area is bounded by the concentration curves obtained using the two different breathing models. The linear model, which more closely simulates the deep breathing pattern, shows the highest maximum that one would expect. The concentration of singlet-delta oxygen which is expected to be reached during the inhalation is significant; this amounts to 2.6% of the original level of singlet-delta oxygen in the irradiated air. The gradual decline in the concentration of singlet-delta oxygen after the maximum is reached comes from the increasing extent of dilution of the incoming air as the lung expands. As exhalation starts, there is no further supply of singlet-delta oxygen available and the decay rate alone determines the rapid fall observed for this period. Symmetrical nitrogen trioxide will exhibit a similar pattern since its lifetime for these conditions is about the same as that for singlet-delta oxygen.

The expected maximum concentrations for the hydroxyl radical are significantly lower than the conditions chosen here. The lifetime data for triplet-P oxygen atoms and singlet-D oxygen atoms suggest that few of these species will live long enough to populate our lungs.

We should remain suspicious that some of the transients in photochemical smog may themselves introduce health related problems. Until more direct evidence is available to evaluate this possibility, no firm decision seems possible.

SUMMARY STATEMENTS

Rational hypotheses have been developed from present knowledge of chemical kinetics allowing mathematical descriptions of the relationships between photochemical intermediates such as ozone, PAN's, and hydroxyl

radicals. Such relationships will be useful in the development of air quality control planning.

Typical polluted atmospheres used in chemical and kinetic models have indicated concentrations, measured in ppb, of photochemical intermediates such as hydroperoxyl radicals and symmetrical nitrogen trioxide which have a long enough life to allow transport to the respiratory system. The significance must still be determined.

Because of the many interdependent factors involved in photochemical intermediates and the high uncertainty surrounding some aspects of the reaction mechanism and rates, it is not possible to set numerical standards for individual pollutants without further definition of the important interrelationships between pollutant molecules.

CONCLUSIONS

Solar radiation induces a number of reactions in the atmosphere between gaseous organic molecules and nitrogen oxides producing a variety of so-called secondary pollutants. These secondary pollutants are present in extremely small concentrations and many are very transient. Because of these two factors it is very difficult, if not impossible, to identify these pollutants by conventional analytical techniques.

Starting with simple hydrocarbons and nitric oxide, attempts have been made to identify intermediate and end products of the photochemical reactions by relating laboratory studies involving chemical and kinetic modeling to atmospheric observation. Since certain key information is still lacking, it is not yet legitimately possible to make quantitative predictions.

Enough information is available to indicate that the main features of the model are correct, placing considerable reliability on the qualitative predictions.

Based on calculated concentrations and theoretical first half-lives, it is possible to predict photochemical intermediates that might persist long enough to be transported into the respiratory system. The health significance must still be determined.

It is not possible to set numerical standards on secondary pollutants; however, sufficient information is available concerning the different relationships to assist in the development of air quality controls.

With respect to heterogeneous reactions, the extent to which nitric and nitrous acids and nitrate and nitrite salts form as a result of gaseous nitrogen dioxide and nitric oxide absorption into aerosol droplets is not now known. It is known that the rate of absorption of acidic gases is a function of the partial pressure of the gases, rate of diffusion of the gases into solution, the pH of the solution and the interfacial area. It is still necessary to evaluate these parameters relative to nitrate salt formation in smog and in related potential health problems.

RECOMMENDATIONS FOR FUTURE STUDY

Efforts should be made to determine the effect on health of photochemical intermediates such as hydroxyl, hydroperoxyl, and symmetrical nitrogen trioxide which can be predicted in typical polluted atmospheres and which have a life long enough to allow transport to the respiratory system.

These intermediates must always be produced in a reaction vessel as one of an array of intermediates. They cannot be studied as independent entities.

Further smog chamber studies of hydrocarbon-nitric oxide-nitrogen dioxide mixtures should be made in which intermediate and final products are related to the composition of hydrocarbon pollutants found in atmospheric samples.

It is necessary to continue kinetic studies of all possible photochemical reactions that can be induced by sunlight and of transients that are encountered within the troposphere.

More detailed information, such as turbulent diffusion, local emissions, and solar zenith angle changes, must be included as well as chemical reactions in the development of useful simulations of actual urban atmospheric reactions.

Future studies should continue to investigate the relative importance of the various reactive species in the removal of olefin hydrocarbons from the atmosphere.

More meaningful reactivity scales should be developed for hydrocarbons based on their ability to generate nitrogen dioxide from nitric oxide in chamber studies.

Further elucidation is required concerning the modes of formation of nitrate salts in the atmosphere in both homogeneous and heterogeneous reactions (primarily in gas-gaseous liquid phases) and their relation to potential adverse health effects.

Ultramicroanalytical techniques should be developed to analyze suspect trace intermediate products and short-lived products formed in chamber studies and in the real atmosphere.

The inhibiting effect of very high nitric oxide concentrations on product rates and certain smog manifestations such as eye irritation requires further study.

CHAPTER 7

EFFECTS OF NITROGEN OXIDES ON NATURAL ECOSYSTEMS

It is difficult to assess the complex cause and effect relationships of any pollutant with a single organism. When attempting to assess such relationships with populations, communities, and ecosystems, the problems increase still further. The determination of the effects of pollutants on natural communities is additionally complicated by the presence of multiple contaminants that might promote synergistic or antagonistic effects.

The constant fluctuation in the number of organisms in natural systems results from both the normal interactions of the physicial and biotic environment, and either purposeful or inadvertant interference by man. Since man-produced pollutants are now distributed globally, they should be considered in the study of any natural community, regardless of the distance from man's influence.

ANTICIPATED EFFECTS OF NO_x ON NATURAL PLANT COMMUNITIES AND ECOSYSTEMS

Ecosystems are integrated units of organisms and the abiotic physicochemical factors with which the organisms interact. After centuries of stable annual climatic and geochemical conditions, ecosystems become self-perpetuating, or climax, units.[403]

Climatic, physicochemical, or biological changes, regardless of their source or nature, will affect the nature of the ecosystem. Some ecosystems are durable and relatively stable when subjected to a given environmental change; others become unstable given the same change. W.H. Smith[496] examined these differences and divided these air pollutant relationships into three classes: low, intermediate, and high dosage.

EFFECTS OF NO_x ON ANIMAL COMMUNITIES

It is difficult to disassociate the effects of NO_x on animals from the effects of other air pollutants. However, the meager information that does exist fails to indicate that current levels of nitrogen oxides have much influence on functioning animal communities. Another difficulty encountered in the assessment of NO_x effects on natural animal communities is the unreliability of the cause and effect relationships predicted in laboratory studies. Extrapolation from one species to another is also untrustworthy since susceptibility can vary widely among species.

McArn *et al.*[339] demonstrated that granule-laden microphages developed in the lung tissues of English sparrows (*Passer domesticus*) from high pollution urban centers, but not in the lungs of those from windswept, unpolluted environments. Because the sparrow is a relatively short-lived species, potential chronic effects could not be observed. The longer-lived domestic pigeon or rock dove (*Columba livia*) can also be used as a model in similar studies. But because pigeons are intimately associated with urban pollution problems, pest control programs might eliminate a study population at a crucial time.

EFFECTS OF NO_x ON PLANT COMMUNITIES

In most terrestrial ecosystems, the majority of both long- and short-term effects of pollutants on ecosystems are caused by the sensitivity of various species to environmental change. Additional long-term effects are created by pollutant action on reproductive capacity and genetics.[491, 607]

Dowdy[117] and Elton[133] provided evidence that the basic biotic responses of an ecosystem to disturbance may be simply the replacement of unsuited species by those newly favored in a changing environment.

Odum[404] listed the general effects of environmental perturbation on ecosystems as: reduction in standing crop; reduction in productivity; differential kill; food chain disruption; succession setback; and changes in nutrient cycling rates.

Woodwell[608] added that any chronic pollutant affecting ecosystem structure reduces the recovery potential of the site. Changes in the plant community, such as size, rate of energy fixation, or species complement, affect the structure of the animal and microbial communities and, therefore, the ecosystems. These changes alter behavior patterns and disrupt the competitive relationships among species.[608] Structural changes that reduce environmental or biological variability in plant communities have been shown to cause new species to assume dominance.[52, 97, 272, 277, 342, 343, 355, 356, 496, 544, 608]

Very little is known about the specific effects of NO_x on native plant species. As pointed out in Chapter 9, however, NO_x shares the following characteristics of other pollutants on crop species: differential susceptibility of species; differential effects due to diurnal and age conditions; and generally unpredictable synergistic effects when considered simultaneously with other environmental factors.

Although NO_x compounds have seldom been demonstrated to cause ecosystem, or even species, damage, visible damage may constitute only a small part of the actual damage to plant communities.[543] It is difficult to single out the effect of any particular environmental factor on plant communities because plants are influenced by many factors simultaneously. Hepting[225] stated, "Since the rate of growth of forest stands and the vigor and appearance of individual trees are influenced by so many

site factors, including soil type, moisture, temperature, drainage, competition, etc., alien impacts not identifiable with known diseases or insects can go unrecognized unless very severe damage is done."

Reductions in plant vigor and survival rates have recently been attributed to air pollution.[225] A well-publicized example is the ozone (O_3) damage to ponderosa pine forests in Southern California which had been previously ascribed to pathogenic action.[355]

Secondary or synergistic effects of air pollutants on plant species and communities are known.[275] In Chapter 9, the synergistic action of NO_x with sulfur dioxide (SO_2) and ozone is reviewed. Studies on crop species showed gas mixtures to be damaging at concentrations well below the threshold causing injury when gases were applied alone. In natural ecosystems environmental factors as diverse as insects, bacteria, fungi, soil, water stress, etc., are candidates for synergistic impacts and should be considered when determining the effects of pollutants.[501, 502, 544]

Woodwell[608] established that the effects of all environmental perturbants are similar. Odum[403] reviewed those effects, but they were perhaps most succinctly summarized by Platt.[422] Environmental changes affect single species and populations in different ways depending on genotypic variation, life cycle stage, and microhabitat condition. Among the ecological effects of a given pollutant are the physiological tolerances to other environmental stresses such as heat, moisture, and light; competitive capacity; and susceptibility to parasites and other disease organisms. Physiological phenomena such as growth, flowering, photosynthesis, and respiration also result. In plant communities or ecosystems, environmental changes, including chemical changes, affect

energy flow, productivity, succession, community structure and composition, and such interspecific characteristics as competition and various aspects of symbiosis.

Although the responses of communities and ecosystems to pollutants are very difficult to examine, some published studies indicate progress in this area. The proceedings of the first two U.S. National Symposia on Radioecology, published in 1963 and 1969,[391,468] are major contributions to the literature on this subject. In 1973, the excellent work of Miller and his colleagues[355] provided a model demonstrating the expected effects of pollutants on plant communities.

The impact of air pollutants on plant communities and ecosystems has been studied extensively.[409,496,596] Each investigator restated the 1963 conclusions of Platt[422] and called for immediate and intensive research on the response of ecosystems to acute and chronic environmental pollution. They all included NO_x among the pollutants definitely requiring further research.

There are several possible techniques for studying ecosystems.[52,53,170] Specific models for studies on radioactivity appear in the aforementioned proceedings of the U.S. National Symposia on Radioecology.[391,468] Models for ozone have been described by Miller and Yoshiyama.[356]

EFFECTS OF NITROGEN OXIDES ON MICROBIAL PROCESSES IN SOILS AND WATERS

Microorganisms are essential to the biosphere and to the function of diverse natural ecosystems. They are chiefly responsible for destruction by decomposition of dead plant and animal tissues. Their metabolism regenerates the carbon dioxide (CO_2) essential to plant life. Microfloras are

the major agents for destruction of synthetic chemicals introduced into soils and waters. Marine algae are essential for the generation of the oxygen required to sustain life in all higher animals. In soil, the bacteria, fungi, and actinomycetes convert compounds of nitrogen, sulfur, and phosphorus to the inorganic state, thereby providing plants with the required inorganic nutrients. Biological nitrogen fixation and nitrification are affected solely by these microscopic organisms, which also maintain soil structure and form the humus important to abundant plant growth. In addition, many of the pathogens that are constantly discharged into soils and waterways are eliminated by microbial actions.

Since microorganisms are critical to the balance of ecosystems, any disturbance in their activities could have serious consequences on a local, regional, or global scale. The potential impact on microorganisms by substances as widespread and pervasive as the nitrogen oxides must therefore be assessed. Surprisingly, this subject has been neglected to date. The few data are based on NO_x concentrations in excess of those found in the atmosphere.

In addition to nitric oxide (NO) and nitrogen dioxide (NO_2), the effects of nitrous oxide (N_2O) must also be considered in any effective assessment of the responses of natural ecosystems to the nitrogen oxides.

Nothing is known about the influence of NO_x on microbial activities in soils, waters, or other ecosystems in which microorganisms multiply, and the published reports deal only with individual species <u>in vitro</u>. Furthermore, generalizations are impossible because some reports show effects where other studies show no effect at low NO_x concentrations. For example, 0.002% nitric oxide, the lowest concentration tested by Krasna and Rittenberg,[301] inhibited hydrogenase activity of the bacterium

Proteus vulgaris by 87%, whereas high nitric oxide concentrations failed to kill significant numbers of bacteria when tested in the absence of oxygen but in highly artificial test conditions.[477] Conversely, a 70 ppm concentration of nitrogen dioxide stimulates luminescence by an unnamed bacterium, although at a concentration of 100 ppm, it was toxic.[495] Nitrogen dioxide also affects the survival of airborne microorganisms. Thus, a 1.5 ppm concentration of nitrogen dioxide was lethal for the bacterium *Rhizobium meliloti* in aerosols.[605] Airborne Venezuelan equine encephalomyelitis virus was inactivated at a 5 ppm nitrogen dioxide concentration, but not at 0.5 ppm. At 10 ppm, the highest concentration tested, the viability of airborne spores of the bacterium *Bacillus subtilis*, was not reduced.[131]

The antibacterial effects of radiation are modified by nitrogen oxides. For example, a 0.5% concentration of nitric oxide sensitized wet spores of *Bacillus megaterium* to radiation, but the sensitivity of the spores decreased at higher nitric oxide concentrations.[449] Nitrogen fixation by *Clostridium pasteurianum* ceases at a 0.1% concentration of nitric oxide.[56] Nitrogen-fixing enzyme preparations are also sensitive to nitric oxide; it has been reported that concentrations of 0.039% and 0.0025%[322a,438a] have depressed the reduction of molecular nitrogen (N_2) to ammonia (NH_3).

The nitrite formed from the oxides in natural ecosystems has long been known to be an antimicrobial agent. As an illustration, *Clostridium perfringens* may fail to grow in laboratory media containing an 80 ppm concentration of sodium nitrite ($NaNO_2$).[436] Considerable information is also available on the inhibition of toxin production by the bacterial agent of botulism, *Clostridium botulinum*, but the levels needed for toxicity are

considerably higher than those found in soils and waters. The inhibition is affected appreciably by pH and salt concentration.[439] Nitrite that occasionally accumulates in soils is toxic to fungi in that environment.[473]

Recent evidence indicates that blue-green algae are inhibited markedly by nitrite (NO_2^-), one of the products of NO_x with water. Thus, the rate of photosynthesis at pH 6.0 by blue-green algae was reduced from 75 to 100% by 10^{-3} M nitrite. By contrast, photosynthesis by green, yellow, and red algae and respiration of several bacteria were inhibited from 0 to 20% by the same nitrite concentration. Thus, blue-green algae are apparently uniquely sensitive to the nitrite formed from nitrogen oxides, an inhibition which might be of considerable importance in those natural ecosystems where these algae are abundant (R.S. Wodzinski, D.P. Labeda, and M. Alexander, unpublished observations).

Nitrous oxide has been the subject of some attention, but again the interest has been on individual microorganisms *in vitro*. For example, 0.25 atm of nitrous oxide, a very high level, inhibits nitrogen fixation but not the assimilation of ammonium-nitrogen by *Azotobacter vinelandii*.[434] Nitrous oxide is also a specific inhibitor of molecular nitrogen utilization by two other types of nitrogen-fixing bacteria, *Clostridium* and *Bacillus polymyxa*;[56] but the concentrations required for effect are probably greater than those in the atmosphere.

Some microorganisms are more sensitive than others to nitrous oxide. Only one of three strains of *Escherichia coli* survived after exposure to a solution through which nitrous oxide had been bubbled.[163] The growth of two fungi and only one of two species of *Clostridium*, but none of the other bacteria, was inhibited in an atmosphere with 90 psig of nitrous oxide.[216] Nitrous oxide also supresses gaseous hydrogen

accumulation in anaerobic soils.[308] The applicability of these findings to problems of air pollution is minimal because of the high gas levels used.

Therefore, the knowledge concerning the potential impact of nitrogen oxides on microbial processes in soils and waters is sparse. Although ambient concentrations probably do not significantly affect biological processes in natural ecosystems, it is not possible to support this view with experimental data.

REACTIONS OF NITROGEN OXIDES WITH SOILS

In studies considering the possible use of nitrogen dioxide as a fertilizer,[9] liquid nitrogen dioxide was injected into 1 kg soil samples in amounts equivalent to 100 to 1,000 kg of nitrogen/ha. Losses as gaseous nitrogen dioxide or nitric oxide were less than 1%. In acid soils, the nitrogen dioxide was rapidly oxidized to nitric acid (HNO_3). In highly buffered calcareous soils, however, some nitrite was observed temporarily.

Soils adsorb large amounts of nitrogen dioxide (and probably nitric oxide) when the gas is injected into the soil. However, the ability of soils to scavenge small amounts of NO_x from the air is probably more relevant to the study of air pollution.

Abeles *et al.*[2] measured the uptake of nitrogen dioxide by 250 g of soil in petri dishes contained in 10 liter desiccators. Resulting data indicated that the concentration of nitrogen dioxide was reduced from 100 to 3 ppm in 24 hr. Since autoclaving the soil altered the final concentration only slightly, the authors concluded that the nitrogen dioxide was probably removed by some chemical reaction. From their data, they calculated that soil in the United States could remove 5.4×10^{10} kg of nitrogen dioxide

annually--about 20 times the estimated annual U.S. production of nitrogen dioxide. At present, emissions are estimated to be 207×10^8 kg/year (Table 3-2). This figure suggests that the production estimate of Abeles and his colleagues is high. Extrapolation of data from petri dish experiments to the entire U.S. land mass is risky. Their report does not state that soil actually can serve as a sink for the present NO_x production.

Prather and his associates[425,426] measured the sorption of nitrogen dioxide from dry (humidity <5%) and moist air (humidity > 95%) by calcareous and noncalcareous soils. Under dry conditions, sorption of nitrogen dioxide from air streams containing 0.1 to 0.5% nitrogen dioxide by volume reached an equilibrium value within 2.5 min. The amount sorbed was related to the surface area of the soil and amounted to as much as 1% by weight of the soil. Moist air increased the sorption capacity by as much as 10-fold and resulted in a measurable decrease in titratable basicity of the soils. The authors assumed that sorbed nitrogen dioxide probably oxidized to nitrate as reported in 1955 by Aldrich and Buchanan.[9] W.C. Chiorse and M. Alexander (unpublished data) showed that the sorbed nitrogen dioxide was converted chemically to nitrite and nitrate, but the nitrite was readily oxidized to the nitrate by soil microorganisms.

Soils serve as both a sink and a source of nitric oxide. However, when nitric oxide enters the atmosphere, it is ultimately converted to nitrogen dioxide which can react with soils. We are therefore concerned here only with soil as a sink for nitric oxide.

The clay mineral montmorillonite adsorbed nitric oxide by chemical reactions when the cation-exchange complex was saturated with such transition metal cations as iron, cobalt, or nickel. Only physical adsorption was observed when the clay was saturated with alkali metal or alkaline

earth metals.[372] In the latter case, exposure to air resulted in rapid oxidation to nitrite.

Prather *et al.*[425,426] and Miyamoto *et al.*[359] examined the reactions of nitric oxide with both dry and moist calcareous soils. As with nitrogen dioxide, sorption under dry conditions was related to the specific surface area of the soil, but the nitric oxide sorption capacity was about half that for nitrogen dioxide. Sorption increased with increasing moisture in the soil or in the air + nitric oxide stream, up to the titratable basicity of the soil. Under these extreme conditions, where the soil sorbed as much as 7% by weight nitric oxide, the soil pH was reduced to the 2.0 to 3.5 range. Most of the nitric oxide sorbed by the soil was converted to nitrate.

Aside from the above, much remains to be learned about the reactions of nitric oxide and nitrogen dioxide with soil.

EFFECTS ON AQUATIC ECOSYSTEMS

Although there are no data, nitrogen oxides may have important environmental consequences on individual species and food chains in aquatic ecosystems. The data cited above on the sensitivity of blue-green algae to the nitrite formed from NO_x may be of some significance.

CONCLUSIONS

The influence of NO_x on natural plant and animal communities is an area in which data are limited. Although no specific information is available for ecosystem responses to NO_x, research on crop plants indicates that NO_x compounds have the characteristic effects of other air pollutants on some species. Consequently we anticipate: differential species sensitivity to NO_x; complications due to synergistic or antagonistic interactions

between NO_x, other air pollutants, and natural environmental stresses; and secondary ecosystem responses caused by changing symbiotic and competitive interactions as species respond differentially.

Nitric oxide, nitrogen dioxide, and nitrous oxide all affect the growth or survival of individual microorganisms when tested in artificial media; but the effect of nitrogen oxides on microorganisms or microbial processes at common atmospheric concentrations are unknown. No attention has been given to the effect of ambient NO_x concentrations on populations or activities in both natural habitats and in vitro. Although it is likely that little suppression of heterotrophs arises from the presence of these gases, a definitive conclusion is impossible without direct experimentation.

The algae that are especially important to primary production in fresh and marine waters, the algae and lichens that are significant in the weathering of rocks and in certain soil processes, and the activity of microorganisms colonizing leaves and causing plant disease are affected by low concentrations of some air pollutants, but their sensitivity to ambient NO_x concentrations has yet to be tested.

Both nitric oxide and nitrogen dioxide react readily with soils, and generally are converted to nitrate. Sorption of large amounts of NO_x lowers soil pH. The addition of lime to the soil can correct this acidity. Since the sorption of NO_x from airstreams containing the low concentrations typically found in polluted air has not been reported, the scavenging role of soil in removing NO_x from the atmosphere cannot be evaluated.

RECOMMENDATIONS

Research on several major types of animal/plant communities and ecosystems is necessary to determine the effects of NO_x at ambient concentrations on species and systems.

Data should be collected in order to determine the effects of NO_x, at ambient concentrations on microorganisms and microbial processes in soils and water. Particular attention should be given to the effects of NO_x on microbial breakdown of organic matter and plant remains, biological nitrogen fixation, nitrification, and processes effected by algae.

In view of the indications that the nitrite that would be formed from NO_x has a deleterious effect on blue-green algae, additional evaluations are necessary to determine whether this effect is of ecological significance and what might be the consequences of such an inhibition in aquatic and terrestrial ecosystems.

More studies are also needed to provide the basis for determining the reactions of soil with NO_x at low concentrations and to determine whether soil serves as a sink at these concentrations.

Further, it is important that special attention be given to the degree of contamination of natural ecosystems by atmospheric nitrogen oxides. The effects of air to water transfer of these oxides should also be studied.

CHAPTER 8

EFFECTS OF NITROGEN OXIDES ON MATERIALS

Field studies and laboratory research have demonstrated that nitrogen oxides can have deleterious effects on textile dyes, natural and synthetic fibers, metals, and various rubber products. The cost of this pollution damage can be considerable, especially at the consumer level.

Of the various oxides of nitrogen in the atmosphere, the most damaging to materials are nitrogen dioxide (NO_2) and airborne nitrates. Nitrogen oxides also play an indirect role in material damage by atmospheric pollutants. Participation of nitric oxide (NO) and nitrogen dioxide in the atmospheric photolytic cycle results in the formation of ozone (O_3) and in the photooxidation of sulfur dioxide (SO_2) in the presence of reactive hydrocarbons to produce sulfuric acid (H_2SO_4) aerosols. These photochemical reaction products significantly damage a wide range of materials.[457,556,557]

EFFECTS OF NITROGEN OXIDES ON TEXTILES

Awareness of Problem - Communication

The atmospheric contaminants known to affect fabrics and their dyes are nitrogen oxides, sulfur dioxide, and ozone. These effects are known to fabric manufacturers and usually are considered in the manufacturing process. Manufacturers of such end-use products as carpets and apparel may be aware of the problem; however, they generally react only to complaints. Unfortunately, the number of consumer complaints made at the retail level is small.

The following damage to textiles from air pollutants is of concern to both consumer and producer:

- Soiling of fabrics due to particulates
- Premature degradation of cotton and nylon due to sulfur oxides and nitrogen oxides
- Fading of dyes on cellulose acetate due to nitrogen oxides
- Yellowing of white fabrics due to nitrogen oxides
- Fading of dyes on cotton and viscose due to nitrogen oxides
- Fading of dyes on wool due to sulfur oxides
- Fading of dyes on acetate due to ozone
- Fading of dyes on nylon due to ozone and nitrogen oxides
- Color destruction on permanent press garments due to ozone and nitrogen oxides

Since manifestation of fabric deterioration and color failure can require up to one year, retailers resist assuming responsibility for such damage when charged by the consumer. This is especially true in cases of fading due to light. Consequently, such deterioration of lightfastness is considered a shared responsibility.

Of major concern to manufacturers is the fading of fabrics or garments in warehouses or on retail shelves. This deterioration generates complaints to the fiber producer, the fabric manufacturer, or the dyers and finishers. Such damage can be of major economic importance when garments affected number in the thousands.

Adequate technical information and testing procedures for the manufacture of lightfast fabrics are available. Therefore, the cause of

fading, whether it be discoloration of whites or fading of dyes, can be determined. It is possible to avoid the manufacture of such unsaleable products which are a source of expensive complaints.

As the textile industry has become consolidated into fewer and larger companies, better quality control has become possible. Fiber producers have set quality standards which they have used in the promotion of their brand names. Competitors have consequently become aware of deficiencies of one fiber as compared with another. In setting up quality control procedures, manufacturers have recognized atmospheric contaminants as important factors to be considered.

Test procedures developed by the American Association of Textile Chemists and Colorists (AATCC)[16a] and the American Society of Textile Manufacturers,[18a] and bulletins issued by manufacturers of dyes all emphasize the effects of air pollution on colorfastness of dyes. The L-22 standards are voluntary standards set by apparel manufacturers for fabric shrinkage and abrasion resistance as well as colorfastness to light, washing, and atmospheric pollutants as measured by the AATCC tests for degree of change. Tags on garments and home furnishings do not consistently refer to damage due to atmospheric contaminants, although the fabric's resistance to light exposures and washing are specified.

Many home economists and other consumer-oriented individuals have examined consumer attitudes toward textile damage due to air pollution.

Fading caused by atmospheric pollutants ranked as a major consumer complaint at a large Pittsburgh department store.[305] Statistical analysis showed such fading as a long-standing source of damage claims received by dry cleaners.[264] Studies have specified oxides of nitrogen, ozone, and acids derived from sulfur dioxide as the pollutants responsible for this color loss.[345]

From the retailers point of view, colorfastness is a principal consumer requirement.[446,494] Standards and test methods for effects of atmospheric pollutants on colorfastness are consequently of considerable importance to fabric production.[505] Testing laboratories on the retail level enable better merchandising resulting in consumer satisfaction.[167]

Several department stores, such as Macy's, and major chains, including Sears Roebuck & Company and J. C. Penney, have their own laboratories. Among the standard test methods adopted by these laboratories are those determining colorfastness of fabrics exposed to atmospheric contaminants.

The Detroit Dry Cleaning and Laundry Institute maintains extensive files on textile performance. A large portion of their complaints stem from atmospheric fume fading.[350]

In the complaints mentioned above, blue was the color most affected. Data compiled in 1962[350] indicated the majority of complaints were received during October, November, and December--the months garments were removed from storage.

Cost of Air Pollution Effects on Textiles

The following data must be obtained in order to make sound economic estimates:

- The incidence of air pollutant damage to specific fibers or end uses.
- The magnitude of the damage compared to total fiber used in the specific example.
- The acceleration of depreciation resulting in decreased wear life (due to air pollution).

- The expected wear life of the article in areas of negligible pollution effect and the calculation of reduction in wear life from the standard.
- The dollar value of the damaged textile product.
- The percentage of the fabric used in air pollution areas: urban versus rural.

Consideration of all these factors should provide a logical basis on which costs can be calculated for textile products used in their unmodified forms, i.e., without the fiber modifications or protective additives.

A separate series of estimates must be made for textiles intentionally modified to reduce the effects of air pollution. The textile industry is aware of the vulnerability of dyes and fibers to such specific air pollutants as sulfur oxides and nitrogen oxides, and has sought to eliminate claims for damage due to air pollution by (a) instituting research programs to seek remedial measures, (b) modifying processes or substituting dyes and inhibiting chemicals, and (c) setting up test methods and other quality controls in an attempt to meet consumer demands. The extra costs incurred in the implementation of these measures are offset by the decreased cost of handling consumer complaints.

The current trend involves modification of the yarn or the manufacturing process to minimize a deficiency or to gain a new useful or marketable property. Manmade fibers can be physically altered, chemically modified, or cospun with additives to meet desired criteria. Natural fibers such as cotton and wool cannot be physically changed with the exception of the addition of resins during the finishing process to control shrinkage and shape retention.

The manufacturers of cellulose acetate fibers, both secondary acetate and triacetate, have been faced with fading of the blue disperse dyes. This fading has been attributed to nitrogen dioxide. In the early 1940's, this problem restricted the marketing of acetate fibers for such end uses as linings, draperies, rugs, and apparel. By instituting research on dyes and inhibitors, the manufacturers found ways to control this fading problem. The costs of these controls may be legitimately passed on to the consumer since such controls are part of the manufacturing process and their cost is included in the retail price.

Such costs to the consumer include:

- Research costs to improve product.
- Added costs caused by more expensive chemicals or dyes, or extra processing.
- Costs of quality control tests.

The remedial measures may not be completely effective, and wear life may not be optimized. Distinction should be made between gradual degradation and "catastrophic" losses in which textiles lose color within three to six weeks or become weakened to one-tenth of their expected wear life. In the cases of sudden and unexpected behavior, the consumer suffers an immediate loss necessitating replacement at his own or the supplier's expense.

There is no excuse for offering merchandise known to suffer catastrophic changes, and such products are usually withdrawn from the market. An expensive alternative may then be necessary. For example, draperies and bedspreads made of cellulose acetate require high resistance to light and to atmospheric pollutants. Since this cannot be guaranteed with conventionally dyed acetate, acetate fiber cospun with colorfast pigment dyes

has been substituted. These yarns, Celaperm by Celanese Fibers Company and Chromspun by Eastman Kodak, have saved this market for acetate fibers, but at added cost of manufacture. In some cases, the effects of air pollution can be prevented by replacing a vulnerable fiber by a resistant one. Drapery fabric that is vulnerable to degradation by sulfur oxides can be replaced by glass or polyester fabrics that retain their strength in a sulfur oxide rich environment.

Such alternatives are not always used since the consumer may wish to risk air pollution effects in order to obtain a particular color or texture available only in cotton, or other nonresistant fabric. In industry, on the other hand, where cost considerations outweigh appearance, fibers are commonly substituted to avoid damage from air pollution.

The incidence and magnitude of damage reported in this chapter were derived from the published literature and from direct contacts with fiber manufacturers, fabric mills, dye and textile chemical companies, and from manufacturers of apparel, home furnishings, and industrial textile articles. Another very useful source of information was the Research Committee of the American Association of Textile Chemists and Colorists. These sources also provided information on remedial measures used to counteract the known cases of air pollution effects on textiles.

A realistic approach to the economics of air pollutant damage to textiles must apportion costs as follows:

1. Modification of fiber or process. Since textiles are manufactured throughout the United States, these costs should cover the entire production rather than be limited to metropolitan air pollution areas.

2. Variable destructive agents in addition to air pollution. The effect of light in degradation of fibers or abrasion during washing are additional factors which are active at the same time as air pollutants. Damage caused solely by air pollutants has been ascertained by comparing high pollution with standard nonpollution areas.

3. Effects of concentration of pollutants. The costs to the nation of the effects of air pollutants were estimated by V.S. Salvin in a study conducted for the Environmental Protection Agency.[551a] He selected and analyzed nine cases of fabric damage as the basis for determining costs. In all cases, the effects of small quantities of atmospheric pollutants were recognizable indicating that nitrogen oxides, sulfur oxides, and ozone will affect dyes and fibers at concentrations as low as 5 ppb, which are typical in nonpollution areas. When studying the effects of air pollutants, the effects in normal environments should be compared with those effects from high pollutant concentrations. Tests designed by the American Association of Textile Chemists and Colorists indicate the relationships between results obtained in a laboratory simulator and results obtained through equivalent exposures during actual use at various locations. Values obtained give averages. However, service tests, or tests of actual use, also show that changes can occur within three months in areas of high pollution, whereas 12 to 15 months may be required in areas of low air pollutant concentrations.

Costs of Nitrogen Oxide Fading of Dyed Acetate Fabrics

Disperse dyes are used for dyeing cellulose acetate. Blue dyes are especially vulnerable to fading by nitrogen oxides; certain widely used reds and oranges are also susceptible. Some disperse dyes, known to be resistant, are not always used because of high cost and difficulty in application. The quality dyes are termed Class A; conventional dyes are designated as Class B.

Blue dyes are used in approximately 70% of all dyeings not only to obtain shades of blue but also green, brown, grey, beige, and purple. Small amounts of blue are even used to obtain certain oranges and yellows.

The Textile Organon estimates that approximately 228 million kg of acetate are dyed annually in the United States. Of this total, approximately 25%, or 57 million kg, are Class A dyed, giving reasonable resistance to air pollution damage but at higher processing costs. The remaining 171 million kg are dyed with Class B dyes, which are susceptible to air pollution-induced fading.

The estimated annual expenditures associated with the prevention of nitrogen oxide-induced fading of dyes on acetate include:

- The increased cost of Class A dyeings whose complicated structures resist nitrogen oxide-induced fading.

  Of the 57 million kg of acetate used in Class A dyeings or pigment spun, 70% or 40 million kg use blue dyes. According to the Bureau of the Census data quoted in Textile Organon, approximately 4.6 m per 2.2 kg of fiber may be made into fabric, resulting in 402 million meters of fabric for Class A dyeings. Considering an increased cost of 4.4¢ per meter by using more expensive dyes

and refined dyeing procedures, the increased cost for Class A dyeings using blues which are resistant to nitrogen dioxide is $17.6 million.

- The cost of inhibitors applied to reduce nitrogen oxide-fading.

  Of the 171 million kg of acetate used in Class B dyeings, 70% or 120 million kg use blue dyes. Converted to meters, 1.21 billion meters of fabric that use blue dyes are produced. At a cost of 1.09¢ per meter, the use of inhibitors increases total costs by $13.2 million.

- Costs of research.

  Costs of research conducted for improving the resistance of fibers and dyes to nitrogen dioxide are estimated to be $.5 million.

- Costs of product quality testing and quality control.

  Testing and quality control costs are estimated to be $.5 million based on 250,000 batches processed at a $2 testing cost per batch.

- Loss due to fading at the manufacture and retail level.

  Losses due to fading at the manufacture and retail level are estimated at $1 million. Current technology can now prevent losses before consumer sale.

- Costs to consumers resulting from reduction in wear life of apparel or home furnishings due to limited protection by inhibitors.

  Of the 120 million kg containing blue dyes of Class B, 60 million kg are in air pollution environments. Of the 60 million kg, a 22% deduction is made for blends and fashion shades where slight loss of the color will not lead to complaints. This results

in 46 million kg that are subject to fading in relation to inhibitor protection. This protection is only partial since inhibitors are consumed by sulfur dioxide in the air and the nitrogen dioxide absorbing property of the diphenylethylenediamine [$C_6H_5NHCH_2CH_2NHC_6H_5$] inhibitor structure. Some shades lose their protection from air pollutants at the end of one year; objectionable change despite the use of inhibitor is noted at 18 months. Since the consumer expects a wear life of 2 years for apparel and 5 years for home furnishings, this fading represents a value loss. The color change may not be noticed by less critical consumers who will continue to use the garment or the furnishing without complaining or discarding. However, a premature loss of wear life of 25% is not uncommon for many acetate fabrics. Garments or furnishings may also fail from light exposure, excessive wear, etc. Forty-six million kg represents a value of $400 million in end products retail. Assuming 10% loss in fabric wear life due to color failure, only half of which is severe enough to cause discarding, a value of about 5%, or $20 million, represents consumer loss.

The total cost in prevention of nitrogen oxide damage to acetate dyed fabrics and in estimated damage is $52.8 million.

Costs of Nitrogen Oxide Fading of Cotton Dyes

Total annual consumer consumption of cotton is approximately 1.4 billion kg. Consumer products made of cotton or cotton blends are estimated to include 50% of the approximately $40 billion annual apparel market,

resulting in a $20 billion market of products containing cotton. Many dyes used on cotton products are susceptible to nitrogen oxide-induced color fading. Below are estimates of dollar losses from nitrogen oxide fading of three common cotton dyes. The following standard formula was used:

Total losses (TL) = Size of market (constant at $20 billion) x percentage of goods used in air pollution areas (constant at an estimated 50%) x estimated percentage of the dyes in the specific class which is sensitive to nitrogen oxide fading x percentage yearly loss in wear life.

Sulfur Dyes. Sulfur dyes are used in approximately 5% of all cotton products. Twenty percent of these dyes are sensitive to nitrogen oxide. A yearly wear life loss of 5% is assumed.

Substituting into the formula:

TL = ($20 billion) x (.5) x (.05) x (.20) x (.05)

TL = $5 million, the annual cost of nitrogen oxide fading of sulfur dyes.

The analysis is conducted similarly for the following dye classes:

Direct Dyes

TL = ($20 billion) x (.5) x (.3) x (.25) x (.02)

TL = $15 million

Reactive Dyes

TL = ($20 billion) x (.5) x (.05) x (.08) x (.05)

TL = $2 million

Vat dyes on cotton are not reported here because they are not vulnerable to fading from nitrogen oxides.

The costs of research, quality control, and more elaborate processing techniques are not appreciable factors for the above dyes. Inhibitors are not used. The total loss caused by nitrogen oxide fading of cotton dyes is $22.05 million.

Costs of Nitrogen Oxide Fading of Dyes on Viscose Rayon

Nitrogen oxide acts primarily on direct dyes used on rayon. The viscose rayon used in apparel and home furnishings totals approximately 228 million kg annually. This figure includes fabrics made of 100% rayon as well as blends with nylon, polyester, or acetate. Direct dyes are most commonly used; reactive dyes and sulfur dyes are not used. The end uses, the percentage of vulnerable shades used, and the percentage of fabrics used in air pollution areas are essentially the same as for nitrogen oxide fading on cotton. Losses from direct dyes are reduced somewhat since certain widely used blue dyes are not susceptible to oxides of nitrogen although possibly 60% of the shades using blue are susceptible. The premature loss of wear life end use rayon products, such as apparel, can be 20-25%. Since other wear factors, such as light and abrasion, contribute to this loss, the percentage due to air pollution is probably around 10%. This can be compared to a yearly wear life loss of 5% for the nitrogen oxide fading of dyes on viscose. Following are estimates of the losses caused by the effects of nitrogen oxides on rayon dyed with direct dyestuffs in end uses:

Value of rayon goods - $2.4 billion

50% of fabric exposed in air pollution areas - $1.2 billion

60% of blue shades that are vulnerable - $720 million

60% of $720 million is dyed with sensitive dyes - $432 million

5% loss in wear life due to objectionable color fading - $21.6 million

Similar calculations can be made for other fabrics for which nitrogen oxides fading effects are clearly established; for example, yellowing of white cotton fabrics, fading on nylon carpets.

The losses incurred by oxides of nitrogen deterioration of nylon and cotton are not as clearly established since acids derived from sulfur dioxide are a major co-contributor.

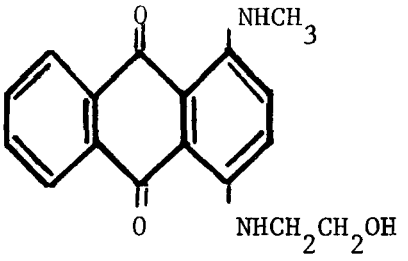
Essentials of Fading Phenomena

To ascribe fading of dyes to a particular atmospheric contaminant, the following three criteria must be observed:

1. The pollutant responsible for change must be isolated.
2. It must be demonstrated that the pollutant can react with the dye resulting in destruction of the dye.
3. The means by which the pollutant comes in contact with the dye in sufficient quatity to cause fading under exposure conditions must be proved.

Effect of Oxides of Nitrogen on Dyes - Contribution of Fiber

The classic case of dye fading due to atmospheric pollution is that of blue dyes on cellulose acetate as initially described by Rowe and Chamberlain.[448] Cellulose acetate is dyed with dyes of low water solubility that are absorbed by this fiber through a mechanism of solid solution. The blue dyes which were in widespread use are of anthraquinone structure as illustrated in Disperse Blue 3, a 1-alkylamino-4-alkylaminoanthraquinone:



Dyes of this structure are moderate in cost, dye rapidly, and have easy leveling properties.

Fading of acetate dyed blue, or shades in which blue is a component, results in pronounced reddening. Since this change occurred in rooms heated by gas heaters, it was termed "gas fading." Rowe and Chamberlain[448] demonstrated that the causative factor in the combustion gases was nitrogen dioxide. A concentration in air of 2 ppm was found sufficient to cause fading.

The fading caused by the action of nitrogen oxides on anthraquinone blue dyes of the Disperse Blue 3 structure results from both nitrosation and oxidation of the vulnerable amino group.[93]

Although cellulose acetate is an excellent absorber of gaseous nitrogen dioxide in a closed system containing fiber and injected oxides of nitrogen,[459] polyester and polyacrylic fibers have a low absorption rate; and nylon, cellulosics, and wool have an intermediate rate. Acetate and triacetate release their absorbed nitrogen oxides on heating. Nylon and wool hold the nitrogen oxides by chemical combination but release them by hydrolysis in water. The nitrogen oxides are retained within the cellulose.

The reaction of nitrogen oxides with cellulose acetate is most pronounced in the anthraquinone blues, although fading also occurs in anthraquinone reds such as Disperse Red 11 and 55. Fading also occurs with certain azo dyes, in particular, Disperse Orange 3, and with the yellows of diphenylamine structure.[25] These fading tendencies are noted in the shade books published by dye suppliers.

Although polyester, which is dyed with disperse dyes, absorbs nitrogen oxides at a low rate, slight fading changes are recorded in the DuPont shade books on disperse dyes. The rate of fading accelerates when temperature and humidity are increased as demonstrated in the noticeable fading that occurs with Disperse Blue 81 in the presence of combustion gases during the curing of resins in finishing processes.

The fading of disperse dyes on nylon due to nitrogen oxides has resulted in consumer complaints. Nylon contains amino groups that act as acceptors for the nitrogen oxides partially neutralizing their action as nitrosating or oxidation agents. Excess nitrogen oxides act on nylon causing a yellowing indicative of nitrosation and diazotization. However, susceptibility of specific dyes to nylon gas fading is noted in the dye bulletin: Disperse Dyes on Nylon Piece Goods - General Aniline.[170a] This tendency is great in nylon carpets in which the nylon is textured by a process that increases fiber absorption of nitrogen oxides.

Fading of Dyes on Cellulose Acetate

The fading of dyes on cellulose acetate fabrics presents serious marketing problems especially in those end uses where color retention is critical, e.g., suit linings and draperies. Test procedures have been established to predict performance of dyed fabrics. These test procedures showed excellent correlation with service trials. Seibert[471] examined the behavior of dyed acetate material under different conditions and recommended the use of more resistant structures where available. He also suggested the use of inhibitors to get better end use performance.

Greenspan and Spoerri[190] studied the factors influencing the action of nitrogen oxides on the vulnerable amino anthraquinone dyes. The work of

these early investigators was instrumental in developing the standard test method later recommended by the American Association of Textile Chemists and Colorists (AATCC) for evaluating the resistance of dyed textiles to color change caused by oxides of nitrogen. This test method is described in the AATCC Manual as "Colorfastness to Oxides of Nitrogen in the Atmosphere."[16a] Basically, in this method a test specimen is exposed to combustion gases containing a 1 to 2 ppm concentration of nitrogen oxides. These combustion gases are derived from a gas burner using natural gas or butane as fuel. Specimens remain in the chamber until the control sample shows a change corresponding to a fading standard. This exposure is considered equivalent to a 6-month exposure in populated southern New Jersey.

The results of various test methods can vary with temperature and humidity. Ray[432] tested for dye-fastness to burnt gas fumes under controlled conditions of temperature and humidity as well as with chemically generated nitrogen oxides with and without the sulfur dioxide. Concentration and time of exposure are the most critical factors requiring control in test methods. The effects of temperature and humidity on acetate fabrics are of less importance.

Rabe and Dietrich[429] conducted comparative tests to determine effects of gas fading using equipment suggested by the International Standards Organization and the German Fastness Commission. In the German test method, the nitrogen oxides are generated by the addition of phosphoric acid to a dilute sodium nitrite solution. This procedure is carried out in a closed system in which the dyed fabric is exposed to the generated nitrogen oxides under high humidity conditions. In the AATCC test procedure for nitrogen oxide fading, nitrogen oxides are generated by combustion of natural or propane gas under conditions of low humidity. Dyed cotton fabrics that

have received service complaints of fading duplicate the fading change under the German test method whereas the AATCC method does not show fading change. This deficiency of the AATCC test method in predicting changes on cotton and also on nylon has led the AATCC to develop a test method for nitrogen oxide fading at high humidity.

Options for Protection Against Damage by Nitrogen Oxides

When nitrogen oxides were established as the cause of cellulose acetate fading and the high absorption rate of nitrogen dioxide by cellulose acetate was determined, the textile industry initiated an extensive research effort to prevent this color loss.

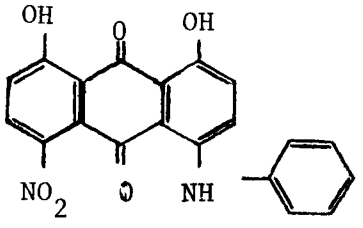
Inhibitors are effective controls which are used in two ways.[459] Most commonly, fabrics are co-dyed with an inhibitor of diphenylethylenediamine structure. This inhibitor reacts rapidly with nitrogen dioxide thereby producing nitroso compounds. Inhibitor reaction with nitrogen oxides blocks the competitive reaction with the dye. This method has two disadvantages. The light yellow coloring of the nitroso compound is often as objectionable as the reddening of the unprotected dyeing. Careful control is therefore necessary to avoid excessive inhibitor action. The inhibitor must be purified to prevent excessive discoloration. This method does not give permanent results since the dye becomes more vulnerable to absorbed gases as the inhibitor is used up. The use of nonyellowing inhibitors of the diphenylacetamidine structure has not proved effective due to their lack of affinity.

The second method for inhibition is the maintenance of an alkaline condition in the fabric since nitrogen oxides are not effective when neutralized with alkali. Diphenylacetamidine is a weak organic base whose effectiveness is its alkalinity. Fabric is padded in the finishing step with solutions of water-soluble alkaline salts such as sodium formate

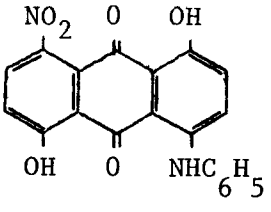
(HCOONa), sodium acetate (CH_3COONa), or organic amines such as triethanolamine [$(HOCH_2CH_2)_3N$]. This protects dyed fabrics against change, and is especially useful in fabrics requiring dry cleaning, since the padded water-soluble inhibitor is removed on washing.

Although inhibitors protect textile products in warehouses and on retail shelves, the protection is not effective enough for such end uses as upholstery, draperies, linings, etc. Blue dyes of high resistance to gas fading have been known almost from the first complaints of atmospheric fading. Since these dyes of azo structure (-N=N-) have very poor light-fastness, they are used primarily on suit linings, which are not exposed to light.

Anthraquinone blue dyes



which combine both excellent lightfastness and resistance to nitrogen oxides, were synthesized by Salvin.[474] Whereas the conventional blue dyes contain alkyl amino groups (e.g., Disperse Blue 3) which react readily with nitrogen oxides, the resistant dyes do not contain the reactive alkyl amino group. The structure contains an aryl amino group which is much less reactive because of the lower basicity of the anilino or substituted anilino group. This work has led to the use of Disperse Blue 27 and Disperse Blue 70 as dyes resistant to nitrogen oxides.



A reduction in reactivity to nitrogen oxides may also be effected by introduction of slightly negatively-charged (polar) groups which are adjacent on the benzene ring (ortho) to the amino groups. These abstract electrons from the amino group reduce basicity and reactivity to nitrogen oxides. This dye is marketed as Disperse Blue 60.

These anthraquinone blue dyes require costly intermediates and several manufacturing steps. They have slower dyeing rates, poorer leveling properties, and their dyeing procedures are lengthy, complex, and require great care in application.

Dyes with high resistance to nitrogen oxides are recommended by the producers of acetate and triacetate. Their quality standards require that fabric resistance to gas fading be approved before either the trademark Arnel for triacetate or Kodel for polyester is granted.

The desire for fastness to atmospheric fading and high light exposure resulted in the development of drapery fabrics in which pigment dyes are cospun in the filament.

The fading of disperse dyes, especially blues and violets on cellulose acetate, is well known. However, a survey of reported fading of acetate fabrics revealed the number of complaints to be quite low from all areas--fiber producers, dye suppliers, fabric mills, retail organizations, chain or major department stores, and consumer organizations. This improvement stems from increased awareness of the problem by both the fabric producer and the retailer. Resistant dyes, sufficient quantities of inhibitors, pigment-dyed fabrics, and fibers other than acetate in vulnerable end uses combine to minimize material damage. The fading of dyes on acetate fabrics is an example of a problem whose causes are known and for which effective controls have been implemented.

Fading on Cellulosics - Cotton and Viscose

Although the effects of nitrogen oxides on acetate dyes is now well-known and preventative measures have been taken, effects on cotton and other fibers have only recently be publicized.

A survey of the effects of air pollutants on fabrics as reported by Upham and Salvin[551a] has produced documentation of fading of direct dyes on cellulose and viscose, of vat dyes on cotton, and of sulfur and reactive dyes on cellulosic fiber. The effect of nitrogen oxides on fiber reactive dyes has been reported by Imperial Chemical Industries in its shadecard of Procion dyes and by J. Hertig in his critical study of the International Standards Organization test for fastness to nitrogen oxides.[228]

Salvin[457] has reported the fading of various dye classes on a range of fabrics including cotton when exposed to ambient air in the high pollution areas of Los Angeles and Chicago for periods of 30 to 120 days. He documented the fading of dyes on cotton due to nitrogen oxides, ozone, or both, and also the fading of direct dyes, vat dyes, sulfur dyes, and fiber reactive dyes on cellulose. The AATCC test for fading from oxides of nitrogen shows no change; the AATCC tests for ozone and sulfur dioxide fading show only insignificant changes. However, the nitrogen oxides test procedure involving oxides of nitrogen generated under high humidity does produce changes which correspond to those observed in service. Salvin reported fading of direct dyes using the AATCC nitrogen oxides test under conditions of high humidity.

Ajax *et al.*[8] conducted tests as long as 24 months in light-sheltered cabinets exposed to various urban and rural environments. A high susceptibility to fading resulted for many fabrics exposed to urban environments with monitored nitrogen oxides and ozone content.

Further effects of nitrogen oxides were documented by McLendon and Richardson[346] in their study of color changes in dyed cotton placed in gas clothes dryers. In the heated air of the gas dryer nitrogen oxides were

present in sufficient amounts to change dyes on cotton fabrics as well as to yellow cationic finishes. In a study of the destructive action of home gas-fired dryers on dyes as carried out by the AATCC Midwest section,[16] the dyes sensitive to generated nitrogen oxides were also those which were affected by combustion products on moist dyed fabrics.

Fading on Nylon and Polyester

The amount of color fading for a given combination of pollutant depends on the humidity, temperature, and migration of dyes to the more gas absorbent surface or fabric finishes.

Such conditions are important in determining the causes of air pollutant fading on polyester/cotton permanent press fabrics and on nylon carpeting dyes. For example, high humidity is necessary to duplicate fading of nylon carpets where ozone is the air pollutant. Results of the nitrogen oxides test reveal that nylon carpets do not fade under low humidity conditions, but do fade when subjected to high humidity.

Fading of dyed polyester/cotton and textured polyester knits has been a problem. This fading does not occur with the disperse dyes on polyester as first dyed. It occurs only after the curing process for polyester/cotton permanent press fabrics and after the heat setting process for polyester knit fabrics in which temperatures of 182-204°C are used. The dyestuff within the polyester migrates to the finish. The finish contains a surfactant or softener that acts as both a solvent for the dye and an absorbing medium for both ozone and oxides of nitrogen. The criteria for fading are met--dyes that are susceptible to oxides of nitrogen change and the air pollutant is absorbed.

Fading of Dyes by Ozone and Other Air Contaminants

The fading of dyes resulting from exposure to the atmosphere is not due to oxides of nitrogen alone. Exposure to light causes a photochemical destruction of the dye molecules. In fact, many cases of color fading have been falsely attributed to action of light rather than air pollutants. In addition to oxides of nitrogen, color change on certain dyes is known to be caused by sulfur dioxide, ozone, and, to a minor extent, the acid base change of acidic air pollutants.

Ozone concentrations of 20-50 ppb cause destructive action on disperse dyes on cellulose acetate. This was discovered when cellulose acetate dyed with blue dyes, experimentally determined to be fast to oxides of nitrogen, faded in service trials. In 1955 Salvin and Walker[460] reported that drapery fabrics dyed with Disperse Blue 27, a dye fast to oxides of nitrogen, were simultaneously exposed with drapery fabrics dyed with Disperse Blue 7 and Disperse Blue 3 (dyes which are vulnerable to oxides of nitrogen fading). Inhibitors were used to protect the blues sensitive to oxides of nitrogen. To indicate the quantity of oxides of nitrogen, the AATCC ribbon, used as a control in the test procedures, was exposed with all draperies.

Service tests were made in homes located in Pittsburgh, Pennsylvania during periods of high concentrations of air pollutant gas, and in Ames, Iowa, a nonindustrial town with minimum air pollution. Tests were made on swatches of fabric from inside hems where light fading was not a factor.

The AATCC ribbon in Pittsburgh reddened, indicating an appreciable concentration of nitrogen oxides; in Ames, very little reddening occurred.

The color changes noted after 6 months became more pronounced after a 12-month exposure. The changes in nitrogen oxide-resistant blues were most noticeable in Ames where nitrogen oxides were of lower concentrations. In Pittsburgh the nitrogen oxide resistant blues did not fade significantly. The unexpected fading of the Disperse Blue 27 was attributed to ozone. In Pittsburgh, the sulfur dioxide interacted with the ozone to reduce its concentration. Moreover, the inhibitor used against fading is the anti-oxidant used to protect rubber against degradation by ozone. The discovery that dye fading may be due to ozone as well as nitrogen oxides is important since the photochemical effect achieved when hydrocarbons from auto exhaust are combined with oxides of nitrogen produces increased concentrations of ozone and oxidants (Los Angeles smog).

Yellowing of Whites

The susceptibility of dyes to fading from chemical action of air pollutants is well known. But fiber interaction and action of specific air pollutants, such as sulfur dioxide, ozone, and oxides of nitrogen that cause yellowing of white fabrics has not been well established. The survey of the effects of atmospheric pollutants on fabrics[551a] documented a considerable number of color changes on white fabrics during storage of garments in warehouses or retail shelves as well as in homes.

Salvin[458] examined 18 white fabrics for yellowing. The air pollutant responsible for yellowing was determined through tests for exposure to sulfur dioxide, nitrogen dioxide, ozone, and hydrogen sulfide. Tests of exposure to heat and humidity in the absence of air pollutants were also made.

The results are summarized as follows:

1. Nitrogen dioxide is the air pollutant responsible for yellowing of white fabrics in the complaint fabrics tested. Yellowing is not caused by ozone, sulfur dioxide, or hydrogen sulfide.
2. The standard AATCC test procedure for effects of oxides of nitrogen does not always show the yellowing effect observed on service exposure in warehouses or retail shelves away from light.
3. The tests include the use of high humidity and temperatures to determine the effects of oxides of nitrogen. The migration of chemicals from the interior of the fabric to the surface or to the finish on the surface under the conditions of high humidity and temperature contributes to the yellowing observed.

Fibers yellow only in Spandex, a polyurethane segmented fiber which reacts directly with nitric dioxide to form the yellow nitroso compound.

In rubberized fabrics, the anti-ozonant used to protect the rubber migrates or sublimes to the cotton where it yellows on contact with nitrogen dioxide forming the yellow nitroso derivative. Optical brighteners of coumarin structure react with nitrogen dioxide to form yellow compounds. This is shown by standard AATCC test procedure for cellulose acetate. No color change is noted on nylon until a high humidity test procedure is used. Coumarin brighteners are used on acetate and nylon but other brighteners which do not yellow are available for use. They are difficult to apply and are more expensive. Several cationic softeners containing amines will yellow on exposure to nitrogen dioxide when used on natural and manmade fiber. Anti-stats used on nylon and polyester have also shown yellowing.

Use of Dyed Fabrics as Area Monitors for Nitrogen Oxides

The EPA has produced an "exposure package" which includes, among other things, a strip of acetate ribbon dyed to a medium shade with Disperse Blue 3. This is the AATCC control ribbon mentioned above which is used in the oxides of nitrogen test method. It changes to violet-red as it reacts with nitrogen oxide from combustion gas, cylinder gas, or generated nitrogen oxides. Its color change is easily recognized. The degree of reddening indicates the severity of the nitrogen oxide pollution. Because this change is cumulative and sensitive to low nitrogen dioxide concentrations, the ribbon should be exposed over a long period. The ribbon will not produce results in the short periods used in instrumental methods. Monitoring factors can be used to characterize a specific area for integrated effects over a period of 30 to 90 days.

The use of the AATCC ribbon containing Disperse Blue 3 is not free from interference. Sulfur dioxide and sulfuric acid derived from sulfur trioxide (SO_3) do not cause a color change, but ozone does. Ozone produces a bleaching effect resulting in color lightening rather than the reddening conferred by oxides of nitrogen. Thus, the color change recognition is open to question.

It is possible to formulate a blue dyeing on cellulose acetate which resists ozone but reacts with nitrogen oxides. This is accomplished by co-application with the Disperse Blue 3 of an anti-ozonant which is an inhibitor against ozone fading. Such a fabric would be useful for producers of dyed or white fabrics who, in their concern about effects of nitrogen oxides, wish to learn the degree of exposure to which their fabrics or garments will be subjected.

It is also possible to formulate a ribbon that can be used to monitor ozone similar to the AATCC ribbon used as control for the ozone fading test.

Time - Concentration Exposures - Field Studies - AATCC Field Trial

The AATCC conducted service exposure trials to determine the extent of fading of representative types of dyes on a range of fiber types.[457] These trials consisted of 90-day exposures during October, November, and December of 1961 in Los Angeles, Phoenix, Sarasota, and Chicago, where the concentrations of atmospheric pollutants were reasonably well-known. The contaminants studied in these trials were nitrogen oxides, ozone, sulfur dioxide, and the products of photochemical action on hydrocarbons. The physical effects of humidity in both the swelling of the fiber and in increasing absorption were considered.

A range of fibers were dyed--wool, cotton, rayon, nylon, Orlon, acetate, and polyester. Although not all-inclusive, the dyes chosen were those commonly used for each fiber. For example, the dyes used for cotton included directs, fiber-reactives, vats, and sulfurs. The colors chosen were medium depths of yellow, red, and blue with no after finish.

Color Changes in Service Tests

The quantity of atmospheric contaminants, e.g., sulfur dioxide, oxides of nitrogen, and ozone, present in the area of exposure was taken from air-pollution data recorded for that period in Chicago and in Los Angeles. These data approximated the values in Table 8-1.

Table 8-2 shows fading on a range of fibers (cotton, rayon, nylon, wool, and acetate) of dyes of various structures (vats, directs, fiber reactives, acid, and disperse).

TABLE 8-1

Typical Concentration of Atmospheric Contaminants, ppm[a]

	Rural	Los Angeles	Chicago
Oxides of Nitrogen	0.01	0.26	0.22
Sulfur Dioxide	0.03	0.05	0.25
Carbon Monoxide	--	23.00	16.00
Ozone	0.06-0.11	0.21	0.005
Aldehydes	--	0.3	--

[a]From V.S. Salvin.[457]

TABLE 8-2

Color Changes on Dyed Fabric--Exposed Without Sunlight in Pollution and Rural Areas[a]

International Grey Scale;[b] 5 = no change. Y = yellow; W = weaker; G = greener; R = redder; and B = bluer

Code Index No.	Phoenix	Los Angeles	Chicago	Sarasota
ACETATE				
Disperse Red 35	4.5Y	4.0Y	4.5Y	4.5Y
Disperse Blue 27	3.0W	2.0W	2.5W	2.0W
Oxides of nitrogen fading control				
Disperse Blue 3	3.5	1.5R	2.0R	3.5
Ozone control--grey dyed with:	3.0	1.5	3.5	2.5
Disperse Blue 27				
Disperse Red 35				
Disperse Yellow 37				
POLYESTER				
Disperse Yellow 37	4.5	5.0	4.0	4.0
Disperse Blue 27	4.5	4.0	3.5	4.5
Disperse Red 60	5.0	5.0	4.0	4.5
WOOL				
Acid Black 26A	5.0	4.5	3.5	4.5
Acid Red 89	5.0	3.5	3.5	2.5Y
Acid Violet 1	4.5	4.0	3.0	4.0
Acid Blue 92	4.5	4.0	2.5	4.0
Acid Red 18	5.0	4.0	3.5	4.5
COTTON				
Direct Dyes				
Direct Red 1	4.0	1.5	1.5	3.0
Congo Red B	4.0	2.5	2.5	2Y
Direct Red 10	4.5	3.5	3B	4.0
Direct Blue 76	4.0	2 grey	1R	3.0
Direct Blue 71	4.0	2.5R	2R	3R
Direct Blue 86	4.0	1G	1G	2.5G
Vats				
Vat Yellow 2	5.0	4.0	3G	5.0
Vat Blue 29	3G	3G	2.5G	1.5G
Vat Blue 6	4.0	3.5R	3.0	4R
Vat Red 10	5.0	4.5	3.5	5.0
Fiber Reactives				
Reactive Yellow 4	5.0	5.0	4.5	4.5
Reactive Red 11	5.0	5.0	4.5	5.0
Reactive Blue 9	4.5	4R	3.0R	4.5
Reactive Yellow 16	5.0	5.0	4.0	5.0
Reactive Yellow 13	5.0	5.0	4.0	5.0

TABLE 8-2 - continued

Code Index No.	Phoenix	Los Angeles	Chicago	Sarasota
Reactive Red 23	5.0	5.0	4.5	5.0
Reactive Red 21	5.0	5.0	4.0	5.0
Reactive Blue 19	4.5	3.0	1R	4.0
Reactive Blue 21	4G	1.5G	1.5G	3G
Reactive Yellow 12	5.0	4.5	3.5	4.5
Reactive Red 19	5.0	5.0	3.5B	4.5
Reactive Red 20	5.0	4.0	4.0	4.5
Reactive Blue 17	4.5	3.0	1 grey	4.0
COTTON				
Sulfur Dyes				
Sulfur Yellow 2	3.5R	3R	2.0R	2.5R
Sulfur Brown 37	5.0	4.5	4.5	4.0
Sulfur Green 2	3B	2.0B	2.0B	2B
Sulfur Blue 8	4.0	3.5G	3G	4.0
Sulfur Black 1	4.5	4.5	4.5	4.5
NYLON				
Acid Red 85	5.0	4.5	3.0	5.0
Acid Orange 49	5.0	4.5	3.0	4.5
Disperse Blue 3	5.0	4.0	3.5	3.0W
Disperse Red 55	5.0	4.5	4.0	3.5
Disperse Red 1	5.0	4.5	3.0	4.5
Alizarine Light Blue C	5.0	4.5	3.5	4.5
ORLON				
Basic Yellow 11	5.0	4.0	4.0	4.5
Basic Red 14	5.0	5.0	4.5	4.5
Basic Blue 21	4.5	4.5	4.0	5.0
Disperse Yellow 3	4.0	5.0	4.5	4.5
Disperse Red 59	5.0	5.0	4.5	4.5
Disperse Blue 3	5.0	5.0	4.0	4.5

[a] From V. S. Salvin.[457]

[b] The International Grey Scale is a numerical method of showing the degree of shade change. It is geometric rather than arithmetic. Essentially, a shade change of 4 shows a change which is slight and is not too easily recognized. A shade change of 3 is appreciable and is easily recognized. A shade change of 2 is severe. A shade change of 1 is disastrous. These numbers are indicative of the shade change with 4 being passable and 3.5 a matter of judgment.

The fading observed in air pollution areas can be attributed to the effect of oxides of nitrogen and ozone. However, such changes were also found in Phoenix and Sarasota where oxides of nitrogen are absent but ozone is present.

In Chicago's sulfur dioxide-rich atmosphere color destruction occurs which is not observed in Los Angeles. The direct cotton dyes suffer more pronounced hue change in Chicago.

In Sarasota, exposures created greater changes than in Phoenix. Both areas are free of oxides of nitrogen and contain ozone in the same concentrations. The major difference is the greater humidity found in coastal Florida. This discovery led to accelerated testing of the entire series at low and high humidities.

EPA Field Trial

The Environmental Protection Agency (EPA) initiated a field study of the fading of the dye-fiber combinations exposed by the AATCC Committee on Colorfastness to Atmospheric Contaminants.[40] The fading characteristics of the 67 dye-fabric combinations were obtained by exposure in lightfast cabinets for consecutive 3-month periods in 11 nationwide urban and rural sites. The objectives of the study were:

- to determine how the colorfastness of dye-fabric combinations are affected by existing levels of gaseous atmospheric pollutants,
- to find a possible relationship between dye-fading and exposure location, and
- to identify pollutants or environmental factors that influence dye fading.

The National Bureau of Standards (NBS) has examined quantitatively the degree of color change in which the NBS unit represents a value based on spectrophotometric readings. The International Grey Scale shows color differences by visual comparison to color standards that are directly compared to the original shade. The NBS units are used for comparison of color depths as well as fading changes.

In the Beloin article,[40] shade changes instrumentally shown to be less than 3 NBS units are not easily recognizable by visual comparison. They would correspond to the International Grey Scale reading of 4 or in certain cases 3.5.

Fading Category	NBS Units	Fading Category	NBS Units
Trace	0- 0.5	Appreciable	3.1- 6.0
Slight	0.6- 1.5	Much	6.1-12.0
Just noticeable	1.6- 3.0	Very much	>12.1

Exposure Sites

The 11 exposure sites (Table 8-3) for this study were Los Angeles, California; Chicago, Illinois; Tacoma, Washington; Washington, D.C.: Cincinnati, Ohio; and Phoenix, Arizona; plus corresponding rural sites for each city. These cities were selected because they represent the various types of pollution and climates throughout the country. The rural sites provided controls with the same climatic conditions, but with low levels of pollution.

Phoenix, Arizona and Sarasota, Florida were chosen because AATCC has used these sites in the past, and because they have extremes in relative humidity at high temperatures with low levels of pollution. Cincinnati was chosen for its proximity to EPA laboratories which facilitated sampling.

The Los Angeles, Chicago, Cincinnati, and Washington sites were located a few blocks from the Continuous Air Monitoring Program (CAMP) stations of the National Air Sampling Network (NASN). The Los Angeles site was near a locally-operated continuous air monitoring station. The data from these stations give a continuous record of selected pollutant concentrations.

TABLE 8-3

Exposure Sites[a]

City	Location	Type	Average Fade, NBS Units
Washington, DC	Municipal Building	Urban	5.0
Poolesville, MD	Poolesville High School	Rural	4.3
Tacoma, WA	Franklin Gault School	Urban	4.3
Purdy, WA	PHS Shellfish Laboratory	Rural	2.9
Los Angeles, CA	LA County Air Pollution Control District Building	Urban	5.7
Santa Paula, CA	Federal Post Office	Rural	4.0
Chicago, IL	Central Office Building	Urban	7.2
Argonne, IL	Argonne National Laboratory	Rural	4.0
Phoenix, AZ	Desert Sunshine Exposure Tests	Suburban	2.7
Sarasota, FL	Sun Test Unlimited	Rural	3.1
Cincinnati, OH	Taft High School	Urban	4.8

[a] From N. J. Beloin.[40]

This 2-year study consisted of eight consecutive 3-month seasonal exposures beginning in spring (March-April-May, 1966).

Conclusions of the EPA Study

1. Appreciable fading occurs in the absence of light. Sixty-four percent of the fabrics tested faded appreciably.
2. The amount of fading varies among urban areas and seasons. A significant difference in fading for the four urban areas of Los Angeles, Chicago, Washington, and Tacoma occurred for 63% of the fabrics having more than "trace" fading, and 86% of them showed a seasonal variation.
3. Urban sites produce significantly higher fading than corresponding rural sites. Eighty percent of the fabrics showing an appreciable color change had significant urban-rural fading.
4. By themselves, temperature and humidity appear to have little effect on dye fading, but they may increase fading when pollution is present.
5. Sulfur dioxide, ozone, and nitrogen dioxide appear to be the pollutants that cause dye fading.

EPA Laboratory Exposures

The EPA study confirmed that high percentage of dye-fabric combinations changed appreciably as first noted in the AATCC study. However, the EPA was particularly interested in the minimum level of pollution at which fading will occur. A controlled environment laboratory study was conducted in which dyed fabrics were exposed for 12 weeks to two levels of sulfur dioxide, nitric oxide, nitrogen dioxide, and ozone under four combinations

of temperature and humidity.[41] The combinations that showed greatest tendency to fade in the field study reported above were selected. With the exception of Fabric Number 20, the AATCC Ozone Ribbon, all combinations listed in Table 8-4 are used commercially.

Experimental Variables

Exposing 20 fabrics to atmospheric pollutants (sulfur dioxide, nitrogen dioxide, nitric oxide, and ozone) at two temperatures and two relative humidity levels, resulted in a very large number of exposures. Since humidity and temperature are variables in the environment, the use of more than one temperature and humidity would determine whether these variables are of importance in establishing conditions for change. To obtain realistic results, the high pollutant concentration that corresponded to the maximum hourly average in a polluted urban atmosphere and the low concentration that corresponded to the yearly average of a polluted urban atmosphere were selected.

The concentrations of sulfur dioxide, 260 $\mu g/m^3$ ± 15% (0.1 ppm) and 2,620 $\mu g/m^3$ ± 15% (1.0 ppm), were continuously monitored during the 12-week exposure period with a Beckman K-76 Acralyzer by using a hydrogen peroxide (H_2O_2) method.[459]

The ozone concentrations, 100 $\mu g/m^3$ ± 20% (0.05 ppm) and 980 $\mu g/m^3$ ± 20% (0.5 ppm), were generated by passing dry air over an ultraviolet lamp; concentration was monitored with a Mast Model 924-2 ozone meter.[25]

The nitric oxide concentrations used were 120 $\mu g/m^3$ ± 15% (0.1 ppm) and 1,230 $\mu g/m^3$ ± 15% (1.0 ppm); the nitrogen dioxide concentrations were 90 $\mu g/m^3$ ± 15% (0.05 ppm) and 940 $\mu g/m^3$ ± 15% (0.5 ppm). Both gases were monitored continuously with a Beckman K-76 Acralyzer by a modified Saltzman method.[471]

TABLE 8-4

Numbering Code for Dyed Fabric Study[a]

Fabric Number	Color Index No.	Dye	Material	Amount of Dye
1	Red 1	Direct	Cotton	1.0%
2	Red 1	Direct	Rayon	0.5%
3	Red 151	Acid	Wool	--
4	Red 2	Reactive	Cotton	2.0%
5	Red 14	Basic	Acrylic	0.5%
6	Red	Azoic[b]	Cotton	474 g/1
7	Orange 45	Acid	Nylon	0.5%
8	Yellow 65	Acid	Wool	--
9	Yellow 11	Basic	Acrylic	0.05%
10	Green 2	Sulfur	Cotton	40 g/1
11	Violet 1	Acid	Wool	--
12	Blue 86	Direct	Cotton	1.0%
13	Blue 3	Disperse	Cellulose Acetate	1.0%
14	Blue 3	Disperse	Nylon	1.0%
15	Blue 27	Disperse	Cellulose Acetate	1.0%
16	Blue 27	Disperse	Polyester	20 g/1
17	Blue 1	Reactive	Cotton	2.0%
18	Blue 2	Reactive	Cotton	2.0%
19	Blue 14	Vat	Cotton	45 g/1
20	AATCC Ozone Ribbon	[c]	Acetate	--

[a]From N. J. Beloin.[41]

[b]Coupling Component 2, Azoic Diazo Component 32.

[c]C.I. Disperse Blue 27, C.I. Disperse Red 35, C.I. Disperse Yellow 37.

The chamber average temperature was kept at either 32.22°C or 12.78°C, and the relative humidity was maintained at either 50 ± 5% or 90 ± 5%. Both variables were automatically controlled and continuously monitored. Pollutant concentrations were maintained within the stated range at least 90% of the time.

Results from these experiments are presented in Hunter Color Units which are based on spectrophometric readings of color on original fabrics and on exposed fabrics with values less than three units designated as trace. (See Tables 8-5, 8-6, and 8-7.)

To summarize, the study showed that nitrogen dioxide, ozone, and, to a lesser extent, sulfur dioxide can cause appreciable dye fading; that nitric oxide has little or no effect; that higher temperatures and relative humidities increase dye fading; and that the rate of fading as a function of exposure time appears to be nonlinear.

EFFECT OF AIR CONTAMINANTS ON FIBERS

The degradation of fibers during atmospheric exposure is affected not only by natural agents such as air, sunlight, and atmospheric moisture, but also by air contaminants such as ozone, nitrogen oxides, sulfur dioxide, airborne dirt, etc. Cotton and nylon are vulnerable to chemical action of acids which reduce the strength of the polymer.[456]

Fiber degradation assumes additional economic importance in uses other than garments and home furnishings. From 18 to 20% of all fiber uses are industrial. In some of these products, such as tarpaulins and cords, premature losses in strength are costly and create safety hazards. Brysson *et al.*[60,61] show that cotton fabrics deteriorate in less than two months. This explains manufacturers search for fibers best suited to resist airborne acids and protective fabric coatings which reduce acid absorption.

Degradation of Cotton by Airborne Acids

Race[430] studied the degradation of cotton in industrial regions in England. In these regions the degradation of cotton is greater during the winter than during the summer. Further, he observed that the smallest loss in strength occurs in spring and autumn. Race determined that the longer,

TABLE 8-5

Average Fading After 12 Weeks Exposure to Nitrogen Dioxide[a]

Fabric	Hunter Color Units[b] 90 $\mu g/m^3$ NO_2 Low Temp. Avg., 12.78°C	High Temp. Avg., 32.22°C	Low Humidity Avg. (50% RH)	High Humidity Avg. (90% RH)	940 $\mu g/m^3$ NO_2 Low Temp. Avg., 12.78°C	High Temp. Avg., 32.22°C	Low Humidity Avg. (50% RH)	High Humidity Avg. (90% RH)
1	7.2	8.0	7.4	7.8	18.0	20.4	16.1	22.3
2	3.4	T	T	T	13.4	16.3	12.6	17.0
3	T	T	T	T	T	T	T	T
4	T	T	T	T	10.4	6.9	9.7	7.6
5	T	T	T	T	T	T	T	T
6	T	T	T	T	T	T	T	T
7	5.6	17.0	10.1	9.5	21.5	27.9	24.3	25.1
8	T	T	T	T	T	T	T	T
9	T	T	T	T	T	T	T	T
10	T	3.3	T	T	6.5	6.6	6.1	7.1
11	T	T	T	T	T	T	T	4.1
12	5.9	9.5	9.4	6.0	14.1	17.2	14.2	17.1
13	29.0	42.3	37.7	33.6	86.9	75.6	88.0	74.4
14	5.5	14.7	5.9	14.2	39.6	45.5	34.0	51.1
15	6.4	4.9	3.8	7.5	20.5	26.8	17.0	29.6
16	T	T	T	T	T	T	T	T
17	3.9	13.6	9.6	7.9	31.8	41.7	35.4	38.1
18	6.4	10.6	8.2	8.9	30.5	41.6	33.8	38.4
19	6.3	6.7	3.3	9.7	34.3	30.4	23.4	41.3
20	T	T	T	T	5.7	11.7	5.7	11.7

[a]From N. J. Beloin.[41]

[b]T=trace (less than 3 units of fading).

TABLE 8-6

Average Fading After 12 Weeks Exposure to Ozone[a]

Fabric	Hunter Color Units[b] 90 μg/m³ NO_2 Low Temp. Avg., 12.78°C	High Temp. Avg., 32.22°C	Low Humidity Avg. (50% RH)	High Humidity Avg. (90% RH)	940 μg/m³ NO_2 Low Temp. Avg., 12.78°C	High Temp. Avg., 32.22°C	Low Humidity Avg. (50% RH)	High Humidity Avg. (90% RH)
1	4.7	4.3	T	6.4	6.4	8.2	4.0	10.6
2	T	T	T	T	T	4.6	T	6.6
3	T	T	T	T	T	T	T	T
4	T	T	T	T	4.0	4.9	T	7.5
5	T	T	T	T	T	T	T	T
6	T	T	T	T	T	T	T	T
7	T	3.3	T	3.3	3.8	10.4	6.6	7.6
8	T	T	T	T	T	T	T	T
9	T	T	T	T	T	T	T	T
10	T	T	T	T	T	4.4	T	6.3
11	T	T	T	T	T	T	T	T
12	3.4	5.0	3.1	5.3	6.7	9.5	5.6	10.5
13	15.7	22.8	16.3	22.2	44.6	59.2	42.8	61.0
14	3.8	18.4	T	20.6	3.4	19.2	T	20.3
15	21.4	26.2	18.4	29.1	38.1	38.8	36.8	40.0
16	T	T	T	T	T	T	T	T
17	T	3.7	T	4.5	5.5	9.7	3.6	11.6
18	3.4	T	T	4.1	5.2	5.7	3.1	7.8
19	4.1	5.0	T	7.3	10.7	13.0	5.2	18.6
20	15.5	21.4	14.5	22.4	51.0	54.5	48.2	57.3

[a]From N. J. Beloin.[41]

[b]T=trace (less than 3 units of fading).

TABLE 8-7

Average Fading After 12 Weeks Exposure to Sulfur Dioxide[a]

Fabric	Hunter Color Units[b]							
	90 $\mu g/m^3$ NO_2				940 $\mu g/m^3$ NO_2			
	Low Temp. Avg., 12.78°C	High Temp. Avg., 32.22°C	Low Humidity Avg. (50% RH)	High Humidity Avg. (90% RH)	Low Temp. Avg., 12.78°C	High Temp. Avg., 32.22°C	Low Humidity Avg. (50% RH)	High Humidity Avg. (90% RH)
1	5.9	4.2	T	7.7	7.0	6.8	4.2	9.5
2	T	T	T	3.4	T	T	T	5.2
3	T	6.8	T	8.6	8.6	12.9	T	19.9
4	T	T	T	T	T	T	T	T
5	T	T	T	T	T	T	T	T
6	T	T	T	T	T	T	T	T
7	5.2	5.4	3.1	7.5	7.2	6.4	3.7	9.9
8	T	T	T	T	T	T	T	T
9	T	T	T	T	T	T	T	T
10	T	T	T	T	T	T	T	T
11	T	T	T	T	7.1	10.2	T	15.6
12	6.2	9.2	4.6	10.7	8.2	12.3	6.9	13.6
13	8.9	12.4	5.9	15.5	13.6	13.6	7.1	20.1
14	T	4.7	T	6.4	3.0	3.7	T	5.4
15	T	T	T	T	3.7	T	T	3.3
16	T	T	T	T	T	T	T	T
17	5.1	5.7	2.1	8.7	7.3	12.0	3.2	16.2
18	5.2	7.2	2.0	10.5	6.4	16.4	T	20.3
19	T	T	T	3.3	T	T	T	4.4
20	T	T	T	T	T	T	T	T

[a] From N. J. Beloin.[41]

[b] T=trace (less than 3 units of fading).

more direct sunlight in summer and the increased acidity of atmospheric moisture in winter were causes of the increased damage in those seasons.

The specific action of nitrogen oxides as an important cause of degradation of fibers is not evidenced in the studies published to date. The concentration of sulfur acids resulting from high sulfur fuel combustion is higher than that of oxides of nitrogen in industrial areas. The nitrogen oxides present increase the total concentration of airborne acids. These factors preclude the isolation of the effects of nitrogen oxides from that of sulfur oxides in field exposures.

Airborne acids such as sulfuric acid and sulfur dioxide on cotton appear mainly in industrial and thickly populated areas. The effects of these acids on cotton have been studied by Howard and McCord.[244] These acid constituents behave as aerosols and are deposited on exposed fabric in several ways. Precipitation (rain, snow, fog, or dew) forms around such particles and transports them to the fabric. Gravity, electrostatic attraction, and direct interception also play a part on the deposition of particles on the fabric.

Pomroy and Stevens[424] conducted studies in two different locations: Tallahassee, Florida (intense sunlight) and Charleston, West Virginia (industrial air pollution). Six drapery lining fabrics (cotton and acetate) were subjected to outdoor and indoor seasonal exposures.

The results, reported as breaking strength retained, showed statistically significant differences between fabrics weathered in the two geographic regions. The cotton sateens exposed to the contaminated air of Charleston lost about 50% more strength than those exposed in Tallahassee. Charleston's climate and topography, together with its industries, provide conditions

for outdoor exposure similar to those reported by Race. Accordingly, the cellulose degradation and deterioration is attributed to the action of atmospheric moisture, airborne acids, and sunlight. The authors observed that most deterioration occurs during the summer and least during the winter in both Charleston and Tallahassee. This is in contrast to Race's observations that deterioration in winter was due to the sulfur content of domestic fuel (coal). Sunlight was an important factor in Charleston; it became the determining one in fiber deterioration. But when the sunlight was minimal, air pollution appeared as the major factor.

Brysson *et al.*[60,61] established a definite relationship between air pollution and accelerated degradation of cotton fabrics. These authors conducted studies over 12-month periods at sites in the St. Louis, Missouri and Chicago, Illinois areas. The strength retention and degree of soiling of untreated heavy, and untreated and treated lightweight cotton fabrics were related to the air pollution data determined by periodic measurement of dustfall, suspended particulate matter, sulfation, and sulfur dioxide. Sulfation and sulfur dioxide measurements correlate best with fabric degradation and soiling. Measurements of other air pollutants, including nitrogen oxides, dust, and suspended particulate matter, also correlate but to a lesser degree.

Degradation of Nylon

The destruction of nylon stockings by acid droplets, generated in polluted atmosphere under certain weather conditions, has been reported in many newspaper articles over several years.

Trávníček[541] investigated the effect of air pollution on nylon. He studied the conditions leading to fiber damage by analyzing the different

contaminated atmospheres and types of damage they caused. He also developed a smog simulator which produced exhaust gases in an aerosol chamber.

Corrosive gases cause most damage to high polymers when absorbed on soot, smut, and other carrier substances. Using the aerosol chamber, Trávníček simulated the effect of various corrosive gases on textiles when absorbed on solid carrier aerosols. So far, there is no protection against sulfur dioxide-laden soot attaching itself to fibers, if outside conditions favor a rapid oxidation of sulfur dioxide to sulfur trioxide and its transfer to the fiber surface (high humidity, higher temperature). The formed sulfuric acid will degrade or dissolve the polymer. There is some similarity, or common action, between the degradation by light and that by exhaust gases. Some light stablizers and dyestuffs protect fibers from degradation by exhaust gases, expecially if these gases are transformed into oxidizing agents by nitrogen oxides and light. Polyamide fibers that are not light stabilized are easy prey for atmospheric pollutants originating from exhaust gases. Polyamides are susceptible to hydrolysis by mineral acid and also suffer degradation by oxidants.

Little and Parsons[322] recorded data on cotton, nylon, and polyester at eight exposure sites in England during 1961 and 1962. The loss in strength that occurred when textiles were subjected to unprotected outdoor exposure varied considerably with the location. In rural areas cotton fabric was found to be more resistant than polyester or nylon. In urban or industrial areas, cotton is inferior to polyester and, to a lesser extent, to nylon. The effect of contaminated air on nylon as compared with clean air is demonstrated by figures resulting when strength is plotted against relative viscosity. After a loss of about 40% in strength, the

samples fall into two groups. The curve for samples taken from urban industrial exposure is different than the curves from samples exposed to cleaner air. The figures for the exposed nylon samples plotted against amino end-groups show strength decreasing rapidly with reduction in end groups as the retained strength falls below 70%. The recorded values for the intrinsic viscosity of polyester confirm its insensitivity to photochemical attack. The pH of the aqueous extract is also an insensitive factor, possibly from the leaching effect of rain. The figures obtained indicate, however, that aqueous extracts of samples exposed to urban areas are more acidic than those in rural areas.

Laboratory Studies of Nitrogen Oxides on Fibers

The direct effects of nitrogen oxides on cellulose fibers have not been studied specifically. Morris[370] conducted a service exposure in Berkeley, California in which observed damage was attributed to ambient levels of nitrogen oxides. Cotton yarn samples were exposed in two cabinets open to sunlight. In one cabinet, air was filtered through carbon to remove nitrogen dioxide. In the other, ambient air was circulated. The samples were exposed for three separate 28-day periods. During this exposure, air pollution and weather measurements were made. Essentially, the experiment determined the effect of sunlight with and without air pollutants--oxidants and NO_x--on fiber deterioration. Because the Berkeley area is low in sulfur dioxide, this pollutant was not measured in this study. Resulting data show that unfiltered air deteriorated the cotton yarn to a greater extent than filtered air. It is known that sunlight will cause fiber degradation. The results of this study

show that sunlight also accelerates the reaction between the air pollutants and fibers. Carbon filter is not a good absorber of nitric oxide which could have been present and which could convert to nitrogen dioxide. If this is the case, air free of nitric oxide would be expected to show less degradation.

In laboratory trials, Zeronian[620] exposed manmade fibers to simultaneous action of light energy (Xenon Arc) and air containing 0.2 pph nitrogen dioxide at 48°C and 39% RH. The fabric samples used were modacrylic (Dynel), acrylic (orlon), nylon 66, and polyester (dacron). The fibers were exposed to light in a Weatherometer and were intermittently sprayed with water. Identical fabrics were subjected to the same tests under the same weathering conditions but without introduction of nitrogen dioxide.

After exposure for 168 hrs, modacrylic fabrics showed no difference with and without nitrogen dioxide. Orlon and polyester showed only slight differences. The results for nylon were not clear cut, although temperatures did cause significantly greater degradation in the presence of NO_x.

During examinations of the reaction of linear polymers, including nylon and polypropylene, Jellinik[262] found that nylon 66 suffers chain sission in the presence of dry nitrogen dioxide, whereas polypropylene tends to cross link.

Dimaio and Manganelli[110] investigated the effects on Lycra, nylon, and polyester of nitrogen dioxide at 20 pphm, 25°C, in the absence of light. The degradation of the fibers was measured by increases in viscosity and by changes in chemical structure of the fibers as shown by infrared spectra.

EFFECTS OF NITROGEN OXIDES ON METALS AND ALLOYS

Oxidation - General

The rate of corrosion is significant to the life of a metallic component. Almost all structural alloys in use are thermodynamically unstable with a tendency to revert to their more stable state, generally the oxide. Fortunately, the product of the reaction between the substrate and the environment is usually an adherent scale which separates the reacting species. Anything that changes the characteristics of this scale could increase or decrease the reaction rate and, hence, alter the life of the substrate.

The rate of scaling or consumption of substrates that do not form protective films is usually linear in relation to time of exposure. In general, such materials are not of great concern to designers since they can account for the material loss in their design. The unexpected failure of a system,which should form a protective scale but does not, is of more concern. Reasons for potential failures are outlined below.

In order for the films formed to be protective, they must completely cover the surface of the substrate from which they are formed and they must be able to accommodate the stress within the film caused by differences in specific volumes between the scale and the substrate, which is called the Pilling-Bedworth ratio.[420] The nature of the scale could depend upon the environment, and in areas of high humidity the scale formed could be a hydrated oxide or hydroxide instead of a simple oxide. If the scale does not adhere, the rate of corrosion is increased.

The growth of protective layers is controlled by diffusion of ionic defects through the scale. The rate at which the metallic substrate recedes

can be increased or decreased by altering the concentration of ionic defects in the scale. The incorporation of lower valent materials in "p" type scales generally decreases the oxidation rate, whereas for "n" type scales, the reverse is true.

The rates of reactions generally increase with increasing temperature although the driving force, the change in free energy for the reaction, is decreasing. The rate of reaction (K) can be described by an Arrhenius-type equation,

$$K = Ae^{-E_a/RT} \tag{1}$$

where E_a = the activation energy, R = gas constant, T = absolute temperature, and A is the preexponential factor, a constant for the system.

The rate of corrosion of structural steels in air, Y, has been described by the empirical equation[215]

$$\log Y = A_o + A_1[SO_2] - A_2[\text{Oxidant}] + b_o \ln(t) - b_1[\text{Oxidant}]\ln(t) + b_2[NO_2]\ln(t),$$

where the A's and b's are constants, [ ] is concentration, and t is exposure time. It was reported that sulfur dioxide increases corrosion and oxidants decrease corrosion by oxidizing ferrous oxide (FeO) and ferrosoferric oxide (Fe_3O_4) to the more protective ferric oxide (Fe_2O_3). This explanation is incorrect since sulfur dioxide itself is a strong oxidant. Furthermore, as long as the metallic substrate (in this case, iron) exists and the metal exhibits more than one valency state, the oxide in contact with the metal will be that representative of the "reduced" or lowest valency and the oxide in contact with the oxidant will be the highest valency state. Therefore, the equilibrium product next to the steel will always tend to be ferrous oxide.

Increasing or decreasing the rate of reaction requires changing the defect structure of the oxide(s) formed, since the relative thickness of the scale of a multicomponents system depends on the relative diffusion rates. If no protective scale forms, then oxidation ceases only when the chemical activity of the oxidant is reduced to a level below that necessary for scale formation.

Wet Corrosion

Fontana and Greene[149] define eight forms of corrosion: uniform and general attack, galvanic, crevice, pitting, intergranular, selective leaching, erosion, and stress corrosion. Although this list is arbitrary, it covers a large variety of problems.

A liquid film or the presence of a hydrated salt plays an important role in most of these types of corrosion. Each mechanism of corrosion must be understood to attenuate the problem and to predict the effect of a variable change.

Galvanic, pitting, crevice, and selective leaching require the presence of an electrolyte or solvent. Hydrated salt crystals can serve as the electrolyte in galvanic corrosion. The more conductive condensate found near the sea is more corrosive than the condensates found inland, even under equal humidity and temperature conditions. Moreover, the position of a metal or alloy in the galvanic table varies with geographic location.[149]

Pits or pitting corrosion, whose incubation periods can range from days to years, usually grow in the direction of gravity. This corrosion is generally associated with the presence of the chloride ion (Cl^-), which may be related to the formation of the strong, highly ionized hydrochloric acid (HCl). Hence, the salts of fluorine and iodine are not regarded as

strong pit formers, but nitrates may be strong pit formers. Since solutions containing ferric chloride ($FeCl_3$) and cupric chloride ($CuCl_2$) or nitrate [$Cu(NO_3)_2 \cdot 3H_2O$] are aggressive because of both the presence of the chloride ion and the reducibility of the cation, oxygen is not necessary.

In both crevice corrosion and pitting corrosion, the rate at which metal dissolves when in contact with the solution is accelerated by the presence of the chloride ion. For example, in the vicinity of the pit or crevice, the oxidation reaction

$$M \rightarrow M^+ + e \tag{2}$$

is increased while the reduction reaction

$$O_2 + 2H_2O + 4e \rightarrow OH^- \tag{3}$$

can continue on all surfaces. The greater mobility of the halide ion is postulated to be significant since the chloride content of the liquid within a crevice can be more concentrated than that in the bulk solution.

Stress corrosion and corrosion fatigue require the presence of a corrosive media and a mechanical force. The corrosive media initiates the stress riser from which the crack progagates. The applied stress cracks any protective scale that separates the corrodent from the substrate. The application of stress initiates corrosion. The pit thus formed then increases the stress.

Material failure caused by the selective leaching of a component of the alloy is exemplified by dezincification (loss of zinc), or dealuminification (loss of aluminum), etc. Dezincification is a well-established form of corrosion common to brasses. A material which has undergone dezincification is weak, brittle, and possesses little strength.

Past Experiences

The composition of a corrodent, gaseous or liquid, influences the rate of corrosion of substrates. The materials engineer must understand the various processes that occur in order to negate the harmful effects prior to their occurrence.

Nickel/brass wire springs in telephone equipment failed prematurely, primarily in the Los Angeles area.[226] Although most failures occurred on the West Coast, springs have also failed sporadically in other parts of the country. Investigators found that the springs had a fogged appearance and were covered with a hygroscopic dust rich in nitrates. Failure was attributed to stress corrosion. The solution to the problem was a change in materials. A zinc free alloy did not fail in service. To minimize failure of equipment which still contained the nickel/brass springs, air conditioning and air filtration units were installed.

Although the failure symptoms of the nickel/brass springs were attributed to stress corrosion, these failures could be due to selective leaching, especially since the cure to the problem was the removal of zinc from the alloy. Zinc-containing alloys such as brass are most susceptible to selective leaching corrosion. To minimize this corrosion, the composition of the alloy is altered, usually by changing the zinc content.

Stress corrosion is alleviated by reducing the stress on the component, eliminating the critical environmental species, changing the alloy, or cathodic protection.

The telephone company changed the alloy and altered the environment by installing air filters and air conditioning as mentioned above. They reduced the quantity of "dust" and controlled the humidity. These steps were effective for both stress corrosion and selective leaching.

Air filtration to control total accumulation is, under many conditions, questionable due to traffic patterns of people entering and leaving the area.[503] Humidity control is important to all wet corrosion problems since hydroscopic salts pull the moisture out of the air. Although nitrogen-containing compounds have not been shown to cause accelerated corrosion, evidence indicates that they may contribute to the overall common corrosion problems. Specifically, the nitrate salts are more hydroscopic than the chloride and sulfate salts.[226] As such, they may lower the "threshold" humidity requirements for the formation of an aqueous media which can serve as the electrolyte or solvent for wet corrosion. The "creeping green corrosion" associated with nickel-palladium-capped contacts probably falls into this category. Nitrogen can change the defect structure of many oxides thereby increasing or decreasing the rate of oxidation of alloys.[310] If the magnitude of the problem associated with the presence of NO_x, etc., is to be evaluated, specific studies should be performed and statements such as: " . . . did not show noticeable relation to dialing trouble, so it was thought that nitrogen oxides were responsible" be disregarded until a cause and effect is shown.

In summary, evidence exists that (a) nitrogen can affect the defect structure of oxides thereby increasing or decreasing the rates of oxidation of metals and alloys, and (b) nitrates adsorption of water moisture aids the formation of a solvent or electrolyte for all the aforementioned forms of wet corrosion; however, a direct relationship has not been shown to exist between a given NO_x content and change in corrosion behavior of structural materials. Since it is not conclusive that no such relationships exist or that synergistic relationships are not possible, more careful examinations must be made.

POTENTIAL EFFECTS OF NITROGEN OXIDES ON RUBBER COMPOUNDS

Ozone is the only major pollutant whose ability to shorten the life of rubber products is well-documented. However, there is some experimental evidence suggesting that limited damage to foamed rubber products is caused by nitrogen dioxide.[376] This observed damage takes the form of discoloration and deterioration of the strength of the foam. Because information on nitrogen dioxide damage to rubber products is limited, additional data must be accumulated to definitively establish the effect of nitrogen dioxide on rubber compounds.

SUMMARY

Field studies and laboratory research have demonstrated that atmospheric pollutants can cause certain textile dyes to fade and certain white fabrics to yellow. Because consumer awareness of pollutant fading is minimal, color fading has often been erroneously attributed to sunlight. Nitrogen dioxide, ozone, and sulphur dioxide can cause appreciable dye fading, while nitric oxide has little or no effect. The chemical mechanisms for nitrogen oxide fading of dyes are fairly well-established. Higher temperatures and relative humidities can increase the degree of dye fading. Various methods for color protection are available, including inhibitors, nitrogen oxide resistant dyes, cospun pigment dyes, and substitution of alternate fabrics. The cost to the consumer of color fading of dyes has been estimated to be more than $100 million annually. The effect of pollutants on yellowing of white fabrics has not been well-established. Recent studies show that nitrogen dioxide is the principal pollutant responsible for this problem.

Evidence supporting specific action of nitrogen oxides as an important cause of degradation of textile fibers has not yet been published. However,

the presence of nitrogen oxides could increase the concentration of airborne acids which degrade cotton fabrics and nylon.

Experimental evidence suggests that nitrogen oxides can affect the defect structure of metal oxides thereby increasing or decreasing the oxidation rates of metals and alloys. Furthermore, airborne nitrates can adsorb water and aid in the formation of a solvent or electrolyte for wet corrosion. However, a direct relationship has not been established between a given nitrogen oxide concentration and a change in the corrosion behavior of structural materials. Additional data are required if the role of nitrogen oxides in damage to metals and alloys is to be defined.

Information on direct nitrogen oxide damage to other materials, such as foam rubber, is limited, and further study is required to establish the effect of nitrogen dioxide on rubber compounds.

CONCLUSIONS

Field studies and laboratory research have demonstrated that nitrogen dioxide, as well as sulfur dioxide and ozone, can cause certain textile dyes to fade and certain white fabrics to yellow. The chemical mechanisms for nitrogen oxide fading of dyes are fairly well-established, and various methods for color protection are available. The cost to the consumer of color fading of dyes by nitrogen oxides has been estimated to be more than $100 million annually. The effect of pollutants on yellowing of white fabrics has not been well-established; however, recent studies suggest that nitrogen dioxide is the principal pollutant responsible for this problem. No estimates of the cost to consumer of yellowing of white fabrics are available.

Available data do not indicate a direct role for nitrogen oxides in the degradation of textile fibers. However, the presence of nitrogen oxides are likely to increase the concentration of airborne acids which degrade cotton fabrics and nylon.

Experimental evidence exists which suggests that nitrogen oxides can affect the defect structure of metal oxides thereby increasing or decreasing the rates of oxidation of metals and alloys. Furthermore, airborne nitrates can adsorb water, thereby aiding in the formation of a solvent or electrolyte for wet corrosion. However, a direct relationship has not been established between a given nitrogen oxide concentration and a change in the corrosion behavior of structural materials.

RECOMMENDATIONS FOR FUTURE RESEARCH

1. Additional data are required to define the role of nitrogen oxides in the degradation of textile fibers and rubber compounds.
2. Further studies should be made of the effects of nitrogen oxides and airborne nitrates on metals and alloys.
3. Estimates should be made of the cost of yellowing of white fabrics, degradation of textile fibers, and damage to metals and alloys by nitrogen oxides and airborne nitrates.

CHAPTER 9

EFFECTS OF NITROGEN OXIDES ON VEGETATION

Nitrogen oxides are less phytotoxic than other constituents of the photochemical oxidant complex. Most of the vegetation injury they cause is indirect through their participation in photochemical reactions that produce atmospheric oxidants, including both ozone (O_3) and peroxyacylnitrates (PANs) (see Chapter 6).

The direct effects of atmospheric oxides of nitrogen on vegetation are usually associated with and confined to areas near specific industrial sources. For example, injury to vegetation from exposures to nitrogen dioxide (NO_2) has been observed near nitric acid (HNO_3) plants, but there are no published reports of vegetation injury in the field due to nitric oxide (NO) or other oxides of nitrogen.

The direct effects of NO_x on vegetation are reviewed in this chapter. Greatest emphasis is placed on reports relating NO_x effects to known concentrations and durations of exposure. Since the majority of available data pertain to nitrogen dioxide, this pollutant receives the most attention in the following pages. The effects of nitric oxide on vegetation are discussed less because existing data is sparse.

NATURE OF EFFECTS

General

To evaluate the effects of NO_x on vegetation, the ultimate use of a plant must first be considered. These uses include three broad categories: commercial, aesthetic, and ecologic. Commercial plants are those grown for

food, forage, fiber, or fuel. Plants grown in private gardens and public parks have aesthetic value. Examples of complex ecologic functions are the participation of plants in soil and water conservation, the oxygen-carbon dioxide (O_2-CO_2) balance in nature, the accumulation and recycling of elements, and the provision of food and habitats for wildlife.

Although a plant or plant community can serve any or all of these functions simultaneously, the effect of NO_x on vegetation becomes significant when it is related to the function of the plant. In this regard Guderian *et al.*[196] have differentiated between air pollution *injury* and *damage*:

> The term "injury" should be considered to include all plant responses resulting from the action of air pollutants. Temporary reductions of assimilation rate or alterations in content of plant constituents thus are included, along with leaf necrosis, leaf abscission, or retarded growth. Of the many functional disturbances and dramatic visible effects that can result from air pollutants, only those of significance to the desired use of the plant should be considered damage.[196]

For example, in root crops (e.g., beet, carrot, radish, and turnip) or in tree fruits, foliar injury is not necessarily "damage" because it does not affect the usable crop. Until injury is sufficient to affect yield, it cannot be considered as damage. Conversely, injury to leaves of ornamental plants or leafy vegetables (e.g., spinach and lettuce), however slight, is classified as damage since the appearance of these leaves determines the market value of the plant. The NO_x effects on vegetation described in this chapter have been observed in experiments designed to answer specific questions. Interpretation is therefore required in order to determine the effects on the functional role of plants.

Acute vs. Chronic

Exposures to most pollutants, including NO_x, are usually classified arbitrarily as acute or chronic. In experimental fumigations, acute exposures are those of short duration at high pollutant concentrations; chronic exposures are for longer periods (occasionally intermittent, but usually continuous) at low pollutant concentrations. The ranges of concentrations and durations of exposure (doses) for acute and chronic exposures have not been defined. Most botanical investigators would designate nitrogen dioxide exposures of 1.6 to 2.66 ppm (3 to 5 mg/m^3) or greater for periods up to 48 hr as acute, and those for longer periods at lower concentrations as chronic. However, these definitions do not apply in the field near sources of NO_x emissions. There, an acute exposure would be any single exposure causing plant injury; the term "chronic" would be applied to a series of exposures that result in injury, where no single exposure has an effect by itself. These two types of exposures often elicit completely different responses. For example, leaf injury is usually characterized by necrosis in acute exposures, but by chlorosis in chronic exposures.

Metabolic Responses

Leaf injury is the most obvious effect of NO_x on plants, but it is only the end result of a series of events that have occurred at lower levels of biological organization. The responses of plants at these various levels have been described by Vogl *et al.*[581] for sulfur dioxide (SO_2) and by Weinstein and McCune[593] for fluoride. These authors have related effects at the cellular level to effects on leaves, the entire plant, and even communities of plants. A similar scheme may be applied to NO_x effects.

Atmospheric NO_x is received by plants primarily through the stomata, after which the pollutant changes from a gaseous to an aqueous form. In its aqueous form, the pollutant may alter the pH of the aqueous solution in or around cells, or react with one or more constituents of the plant in such a manner that both reactants are changed to forms affecting plant metabolism.

In Vitro Responses. NO_x effects on plants at the cellular level are not well understood. The reaction of nitrogen dioxide with water, which occurs in plant tissues, results in a mixture of nitrous acid (HNO_2) and nitric acid. Enzymic experiments *in vitro* have shown that 1.0 *M* nitrous acid inactivated β-amylase isolated from barley and malted barley, possibly through the oxidation of essential sulfhydryl groups.[592] Later studies with α-amylase extracts from *Bacillus subtilis*[108] showed that nitrous acid interacted with amino and tyrosine groups as well as sulfhydryl groups. The oxidizing action of nitrous acid was reversible with sulfhydryl groups, but irreversible with amino and tyrosine groups. Nitrous acid causes oxidative deamination of amino groups of viral nucleic acids which reduces infectivity of some viruses,[469] or mutagenic effects in tobacco mosaic virus,[381,489,548] and polio virus.[50] However, the concentrations of nitrous acid used in all of these *in vitro* experiments were much greater than those that would result from gaseous NO_x exposures.

In Vivo Responses. Wellburn *et al.*[595] studied the effects of nitrogen dioxide exposures on the ultrastructure of chloroplasts *in vivo*. Broad bean (*Vicia faba*) plants were exposed for 1 hr to 1.0, 2.0, or 3.0 ppm (1.9, 3.8, or 5.6 mg/m^3). The leaves were harvested immediately after exposure and prepared for electron microscopy. Examination of the chloroplasts showed

that nitrogen dioxide caused a swelling of the thylakoids associated with the stroma. These swellings appeared to be reversible because thylakoid swelling was not observed in chloroplasts of leaves exposed to unpolluted air immediately following nitrogen dioxide fumigation.

In vivo experiments performed by Hill and Bennett[234] have shown that both nitric oxide and nitrogen dioxide inhibit apparent photosynthesis of oat (*Avena sativa*) and alfalfa (*Medicago sativa*) plants at concentrations below those that cause foliar lesions. The threshold dose for this inhibition was about the same for both pollutants [0.6 ppm (1.1 mg/m^3) for nitric oxide and 0.4 ppm (0.8 mg/m^3) for nitrogen dioxide in 90 min fumigations], but the inhibition occurs faster for nitric oxide than nitrogen dioxide. The NO_x-induced inhibition of photosynthesis is not a permanent effect. Plants exposed to nitric oxide for 2 hr recover and assimilate carbon dioxide at prefumigation rates within 1 hr after exposure, whereas plants exposed to nitrogen dioxide require more than 4 hr to recover. In fumigations introducing nitric oxide and nitrogen dioxide (1:1) simultaneously, the degree of inhibition of apparent photosynthesis was the same as the sum of that induced by each pollutant when introduced simultaneously. Unlike ozone-induced inhibition of photosynthesis,[235] which was determined to be a consequence of stomatal closure, Hill and Bennett[234] suggested that nitric oxide and nitrogen dioxide affect photosynthesis directly. They found no evidence that NO_x affects stomata.

To summarize, pollutant-altered pH of the cellular milieu and pollutant reactions with cellular constituents lead to altered metabolism, ultrastructural changes, reduced photosynthesis, and probably many other effects that have not been observed or measured. These, in turn, lead to effects at progressively higher levels of biological organization. Pre-

mature senescence, chlorosis, necrosis, or abscission of leaves affect the entire plant causing reduced growth or reproduction, and even death of individual plants or entire plant communities.

Visible Symptoms

Although foliar symptoms are the most obvious effects of NO_x exposure, less obvious effects have also been reported. Continuous exposure of Pinto bean (*Phaseolus vulgaris*) seedlings for 10 and 19 days to 0.33 ppm (0.62 mg/m^3) of nitrogen dioxide resulted in a gradual change in the appearance of leaves without causing necrosis.[529] The leaves developed a downward curvature and a darker green color. The chlorophyll content (per unit weight) of unifoliate leaves was greater for nitrogen dioxide-fumigated plants than for control plants, but there was no difference in the amount of chlorophyll per leaf. Deeper green color and downward curvature of leaves were also noted for tomato (*Lycopersicon esculentum*) seedlings exposed to slightly higher nitrogen dioxide concentrations.[529] But the loss of chlorophyll is a more common response to NO_x exposure. Extraction and chromatographic separation of pigments from nitrogen dioxide-injured leaves by Kändler and Ullrich[274] revealed a loss of carotene, a reduction in chlorophyll, and the appearance of a decomposition product of chlorophyll that was specific for nitrogen dioxide. They proposed that these pigment changes could be used as a diagnostic tool to differentiate between acute sulfur dioxide and nitrogen dioxide injury.

In continuous long-term exposures of tomato plants to 0.25 ppm (0.47 mg/m^3) of nitrogen dioxide, Spierings[500] noted premature senescence of older leaves after 37 days which, with time, gradually increased in intensity, affected younger leaves, and resulted in premature abscission of leaves. Thompson *et al.*[536] observed chlorosis and severe defoliation of navel orange

trees (Citrus sinensis) after exposure for 35 days to 0.50 ppm (0.9 mg/m^3) of nitrogen dioxide. Acute nitrogen dioxide exposures of Citrus sp. [200 ppm (376 mg/m^3) for 4 to 8 hr or 250 ppm (470 mg/m^3) for 1 hr] were observed by MacLean, et al.[330] to cause rapid wilting and abscission of nearly all spring leaves. Abscission frequently occurred before necrosis developed, and some succulent young shoots were killed. Abscission of potato (Solanum tuberosum) flowers and lodging of oats were observed by Czech and Nothdurft[95] after 1 hr exposures to 100 ppm (188 mg/m^3) nitrogen dioxide.

There is no "typical" leaf symptom for diagnosing nitrogen dioxide injury to plants. The extent and type of leaf injury are determined by many factors. As mentioned above, acute and chronic exposures produce different symptoms. In addition, susceptibility of plants to nitrogen dioxide varies among species and even among varieties and cultivars within a species. Climatic and other external factors also influence the form of leaf injury.

Descriptions of nitrogen dioxide injury to leaves of various plant species and color plates showing nitrogen dioxide symptoms have been published by van Haut and Stratmann[577] and by Taylor and MacLean.[530] Their studies indicate that the primary leaf symptom of chronic nitrogen dioxide exposure is chlorosis, whereas acute injury to leaves of dicotyledonous plants is usually characterized by the rapid appearance of irregularly-shaped intercostal necrotic lesions. On most conifers and monocotyledonous plants, nitrogen dioxide-induced foliar necrosis usually occurs at or near the tips of leaves. The length or area of affected tissues varies with the severity of the nitrogen dioxide exposure.

The best available descriptions of nitrogen dioxide-induced symptoms on various plant species were published by van Haut and Stratmann:[577]

Acute leaf injuries (necrosis)

Diffuse gray-green or lightly brownish discolored spots appear on the leaves as early symptoms of acute nitrogen dioxide injury. They dry rapidly and bleach, through various intermediate colors, becoming ivory to white; or, as with many deciduous trees, they take on a reddish-brown, rarely black-brown final color. The time course of acute injury depends on light and temperature. On warm, sunny days, usually only 24 hr pass between the less well recognizable early symptoms and the more distinct later stages. In deciduous trees, the final coloration begins about one week after the injury.

With some types of plants, such as clover, lucerne, or rape, the characteristic ivory-bleached spots can even appear on the day of exposure.... Necrosis is often surrounded by cork-like dark brown tissue....

Dicotyledonous plants: Intercostal necrosis prevails in broad-leafed dicotyledonous plants. After severe exposure, the damage pattern extends over onto the leaf veins, which then also die and discolor. Necrotic portions of tissue can tear apart and break out of the leaf width, leaving a colored edge behind. This "window formation" is observed mainly in broad leaves with tender and succulent tissue, as well as in the leaves of various kinds of deciduous trees. In deciduous trees, intercostal spots often combine to form intercostal stripes. Point-like necrosis distributed irregularly over the lamina can give the leaf a sprinkled or rusted appearance. In other dicotyledonous plants the edges of the leaves are the preferred points of attack. Leaf edge necrosis is particularly characteristic for representatives of the family of the papilionaceous flowers.... Injuries [in clover] begin at the edges of the pinnate leaves and from there extend inwards. Not uncommonly, necrosis first develops at a small distance from the edge of the leaflets and only after that does it affect the outer marginal zone, as in peas.... Occasionally small fleck-like necrosis also appears near the margin. The younger pinnate leaflets of lucerne and lupine often show point necrosis, while marginal necrosis at the leaf base is most common in older leaflets.... Marginal necrosis can appear in Robinia.... Oak and maple leaves develop light red-brown spots and marginal necrosis, while those of apple are dark red-brown, and those of pear are black-brown. With more severe exposure, necrosis penetrates in tongue shapes from the edges into the intercostal areas. [These necrotic areas] often coalesce so that a green zone remains only along the central vein. Furthermore, compound leaves that are strongly segmented (carrot and parsley plants) show injuries predominantly at the edges and tips. Simple leaves with deep incisions (gooseberry bush) also react to the action of nitrogen dioxide more with edge and tip damage than on the leaf width. Finally, discolorations proceeding from the leaf tip are also observed in species with lanceolate leaves (flax, sweet William) as well as on small bracts and sepals.

Monocotyledonous plants: Acute injuries are expressed in most monocotyledonous plants in the form of yellow-white necrosis which spreads mainly from the tips of blades or just below them. In addition to this injury syndrome, necrosis also occurs at the leaf margins, and as necrotic stripes on the leaf.

...tip necrosis, which can extend along the leaf edges, is found in cereals, corn, and meadow grasses. After severe exposures, as much as half of each of the leaves dies, and they may even die back to the base. Gray-green necrosis is observed just below the leaf tips in garden hyacinths and narcissus. Here, though, the injury soon spreads to the tips. Marginal necrosis is typical for oval-leafed monocotyledonous plants. Not uncommonly, the leaves are damaged at opposite sides of the lamina, as in the tulip, for example. In young leaves these damage zones often form just below the tips, while in old leaves they appear more toward the base. Marginal necrosis spreads over the leaf more or less widely, depending on the intensity of the action.... Longitudinal necrotic strips should be mentioned as another damage pattern. They occur oftener and more distinctly in cereals than in corn and gladiolus. Later, adjacent strips can merge so that large longitudinally extended damage zones cover the lamina. Tip necrosis occasionally spreads as streaks also.

The sensitivity of the beards on the inflorescences of rye and barley is noticeable. They usually bleach down from the tips. After severe exposure the discoloration spreads over the hulls. With oats, the necrotic stripes can join the yellow-white discolored tips of the hulls.

In monocotyledonous plants, too, diffusely green colored groups of cells, which soon assume a greenish-brown tone, occur as early symptoms of leaf damage. In cereals, the gray-white final color usually appears one day after the damage. The bleaching spreads for several days. One week after the exposure the necrosis may be surrounded by a thin red-brown border.

Apparently, there is a relationship between the location of necrosis and the age of the leaf. While the injury proceeds preferentially from the tips in young leaves--particularly narrow-leaved types (Graminea)--the damage zones in older leaves are more often in the middle or basal part of the leaf. This can be explained as due to basal leaf growth. Those groups of cells that were last to complete their development are always the most sensitive....

Conifers: Acute damage in conifers is expressed as a red-brown or fuchsia discoloration of the needles, usually beginning at the tips, which can extend to the base. There are also limited local damage zones at the tips, in the centers, or at the bases of the needles. Distinct boundaries between the necrotic and uninjured parts of the needles can be observed in pines and firs. Here the dead red-brown part of the needle is delineated from the green tissue by a sharp boundary line. Later, some one to two months after the exposure, a ring-shaped, dark red-brown zone appears at this transition. At this time, however, the initial

red-brown discoloration of the needles has bleached somewhat. [The retention time for necrotic needles varies with the species. Spruce needles fall] soon after the red coloration [develops]. In contrast, injured larch needles can remain on the twigs for several months, and pine needles for more than a year. The fir needles also remain for months, or even until the following year if the injury is limited to the tips. But if the damage extends over half of the needles, most of them fall within the first month after exposure.

In the initial stage of injury the needle tips or the upper sides of the needles discolor to a pale or gray-green, and the damaged part of the needle loses its gloss. With intense sunlight a light brown intermediate color appears after only a few hours. In sunny weather the red-brown final color appears the first week after exposure. However, this may take 14 days in cloudy weather. This explains why the needles exposed to sunshine often discolor sooner and more intensely than those in the shade on the same tree. Later, the needles can bleach and take on light red-brown tones. The spring twigs show less severe necrosis than the needles from the previous year. The color tone of the necrosis is noticeably light on very young needles, particularly in pine. Shortly after leaving the sheathing leaves, the damaged needles are differentiated by their yellow-brown color from the red-brown of the older needles. In the pines, it is possible to detect relations between the appearance of necrosis in certain portions of the needles and the ages of the needles. In young needles which have just left the sheathing leaves the damage progresses from the tips, while older needles often show damage zones shortly below the tips. This banding is usually only transient because the discoloration soon spreads to the tips. With advancing age, necrosis occurs more frequently in the center or basal part of the needle also....

Leaf Chlorosis

Along with acute damage, other phenomena are observed, for which the term "chronic damage" has become accepted. We can mention leaf chlorosis as a striking characteristic of this type of injury. This is expressed as greenish yellow spots or as yellowing of the entire leaf surface. Chlorosis occurs due to the long-continued effects of low nitrogen dioxide concentrations, and less often from peak concentrations which can also cause necrosis at the same time.

Spotty chlorosis is characterized by small yellow zones. They are distributed across the lamina in large numbers, giving the leaf a marbleized appearance. Later, adjacent spots can merge. In other cases, chlorosis is limited to the leaf margins, or it may spread from there across the surface. In cereals, chlorotic discolorations often begin at the tips and progress towards the base if the exposure continues. At the beginning of damage, conifers often show chlorotic zones on the upper side of the needles or at the tips. Later the light green or yellow-green discoloration spreads over the entire needle. Finally, the yellow-green tone in the upper part of the needle can change to a pale yellow and the tip itself may die....

> A general chlorosis of the older leaves is often the only symptom of injury after long-continuous exposures to low concentrations. It resembles early senescence and can be linked with premature falling of leaves. With young leaves, on the other hand, regeneration of the chloroplast pigments frequently begins after the exposure stops, so that the leaves are a normal green again after some weeks. The green-brown discolorations that occasionally appear can also be reversible. It is true for all chlorotic injury patterns that they are nonspecific and indicate gas exposures only [when they occur] in combination with symptoms of acute injury (necrosis). Both types of injury can occur on the same plants, as well as adjacent to each other on one leaf. In general, the older leaves tend to become chlorotic while the younger leaves show severe necrosis. In tobacco, for instance, leaves in the lower segment of the plant become yellow over their entire surface; in the center section chlorotic discolorations appear along with necrosis; while the younger leaves in the upper part of the plant show only necrosis. In deciduous trees, necrosis and yellowing appear preferentially at the same time in older leaves.... In the narrow-leafed monocotyledonous plants diffuse chlorotic zones often form a transition from the necrotic leaf tip to the green basal part of the leaf. Cereals also occasionally show fine chlorotic longitudinal strips which sometimes transform into longitudinally extended necrosis. Corn often exhibits distinct longitudinal chlorotic stripes before the appearance of necrosis. In conifers there can be a chlorotic zone adjacent to the necrotic needle tip, which advances toward the base or, as in the narrow-leafed monocotyledonous plants, forms a transition zone between the dead leaf tip and the green basal part.

Growth and Yield

Data concerning the effects of NO_x on plant growth and yield are limited. Nevertheless, it is reasonable to assume that NO_x-induced reductions in the assimilatory capacity of plants through altered metabolism, leaf injury, or abscission also affect the growth of plants. One of the most comprehensive reports on the effects of nitrogen dioxide on growth and yield is that of Spierings.[500] Continuous exposures of tomato plants (*Lycopersicon esculentum*) to 0.25 ppm (0.47 mg/m^3) during the entire growth period (128 days), caused growth reduction of leaves, petioles, and stems. The crop matured slightly earlier with substantial decreases in fresh weight yield (22%), average fruit weight (12%), and the number of fruits (11%). After exposures to 0.5 ppm (0.94 mg/m^3) for 10 days or

0.25 ppm (0.47 mg/m^3) for 49 days, tomato plants were taller than control plants, but stems were smaller in diameter, leaves were not as large, and the fresh weights of entire plants were less. Thompson *et al.*[536] exposed navel orange trees to nitrogen dioxide continuously for 290 days. Compared to trees exposed to filtered air, those exposed to nitrogen dioxide concentrations between 0.06 and 0.25 ppm (0.12 and 0.47 mg/m^3) showed an increase in fruit drop throughout the exposure period and a significant reduction in both the number and weight of fruit at harvest. Czech and Nothdurft,[95] using 1-hr exposures to 1,000 ppm (1,880 mg/m^3) nitrogen dioxide, reported that the fresh weight of sugar beet (*Beta* sp.) roots was one-third less than that of control plants.

Both stimulation and reduction of plant growth have been reported for nitrogen dioxide. As noted above, Spierings[500] found growth reduction in tomato plants, and Taylor and Eaton[529] reported growth suppression based on leaf weights. The fresh and dry weights of unifoliate leaves of Pinto bean plants exposed to nitrogen dioxide at 0.33 ppm (0.62 mg/m^3) for 10 and 19 days, or tomato leaves from plants exposed for 10 to 22 days to 0.11 to 0.62 ppm (0.21 to 1.17 mg/m^3) were usually significantly less than corresponding leaves of nonfumigated plants.[529]

Conversely, Faller[139] found that nitrogen dioxide stimulated growth. Fumigations providing nitrogen dioxide at 0.8, 1.6, 2.39, and 3.19 ppm (1.5, 3.0, 4.5, and 6.0 mg/m^3) during daylight hours for three weeks resulted in increases in plant height and in the dry weight of leaves, stems, and roots of sunflower (*Helianthus annuus*) plants.

FACTORS AFFECTING PLANT RESPONSES

The extent, severity, and type of NO_x effects on plants can be altered by both external and internal factors. Environmental conditions, the

presence of additional pollutants in the atmosphere, and the condition or status of the plant itself can all influence the response of the plant to NO_x.

Biological Factors

Grouping plants according to their susceptibility to NO_x[530,577] reveals the influence of genetic factors. As with most other pollutants, susceptibility to NO_x varies greatly among plant species and even among varieties, cultivars, or clones of the same species.

Another important biological factor is the stage of development or age of the plant. Van Haut and Stratmann[577] found that the stage of development at which nitrogen dioxide exposures occur affects the degree of yield reduction. For example, fumigation of oats during flowering has the greatest effect on the yield of grain. Exposures during the earlier, vegetative state of development, or later, when the grain is yellow-ripe, have no effect on yield. The critical stage for radish (*Raphanus sativus*) is the period of root development.

The age of leaves can affect their susceptibility to NO_x. In tobacco (*Nicotiana* sp.) the oldest leaves become chlorotic, middle-aged leaves become chlorotic with necrotic lesions, and injury to the younger leaves is limited to necrosis.[577] Benedict and Breen[42] reported that age-dependent foliar susceptibility is not the same for all species. Middle-aged leaves are most susceptible in dandelion (*Taraxacum officinale*), cheeseweed (*Malva parviflora*), lamb's-quarter (*Chenopodium album*), pigweed (*Amaranthus retroflexus*), and Kentucky bluegrass (*Poa pratensis*), whereas the susceptibility of middle-aged and old leaves is about the same in sunflower, annual bluegrass (*Poa annua*), and nettle-leaved goosefoot (*Chenopodium* sp.). Emerging or elongating needles of conifers are more susceptible than mature needles.[577]

Environmental Factors

Theoretically, any external factor than can influence plant growth or development may also affect the response of plants to a pollutant. For sulfur dioxide, fluoride, and ozone, some of the influences of climatic (temperature, relative humidity, light intensity, light quality, and photoperiod) and edaphic (soil temperature, soil moisture, and mineral nutrition) factors on plant responses have been reported. But little is known of these influences on the susceptibility of plants to NO_x.

Taylor and Eaton[529] suggested that the rate of nitrogen dioxide absorption by plants is greater in darkness than in light, and Zahn[618] reported that exposures of alfalfa at night were more injurious than daytime exposures. These findings are supported by Czech and Nothdurft[95] who discovered the toxic dose for 1-hr exposures of sugar beet plants to be 10 times greater in daytime fumigations [100 ppm (188 mg/m^3)] than at night [10 ppm (18.8 mg/m^3)]. Van Haut and Stratmann[577] found leaf injury in oats to be greater at night than during the day. But they also reported that once injury was initiated, the development of necrotic lesions was most rapid on warm sunny days.

Susceptibility varies at different times of the day. In a series of 2-hr exposures, beginning at 0800-1000 hr and ending at 2000-2200 hr, injury to rye (Secale cereale) plants was greatest at midday.[577] Soil moisture stress reduces the susceptibility of various weeds to nitrogen dioxide. In 4-hr exposures to 20 or 50 ppm (37.6 or 94 mg/m^3), plants grown in moist soil developed injury covering up to 50% of the surface area of their leaves; but little foliar injury occurred under conditions of moisture stress.[42] With respect to mineral nutrition, Zahn[618] reported that an increase in the amount of available nitrogen resulted in a reduction in nitrogen dioxide-induced foliar injury.

Other Pollutants

The presence of other pollutants in the atmosphere with nitrogen dioxide is another important consideration. When sulfur dioxide and nitrogen dioxide occur together in the atmosphere, each at a concentration that is not harmful to plants, their combined effect can result in plant injury. For example, Dunning *et al.*[122] evaluated six different crop species and found the threshold for nitrogen dioxide and sulfur dioxide injury to be 2.0 ppm (3.8 mg/m^3) and 0.5 ppm (1.3 mg/m^3), respectively, in 4-hr exposures. When the gases were provided together [at concentrations of 0.05 to 0.25 ppm (0.09 to 0.47 mg/m^3) of nitrogen dioxide and 0.05 to 0.25 ppm (0.13 to 0.66 mg/m^3) of sulfur dioxide] leaf injury was induced. Tingey *et al.*[538] noted similar responses. Pinto bean, oat, radish, soybean (*Glycine max*), tobacco (*Nicotiana tabacum*), and tomato plants exposed for 4 hr to a mixture containing 0.15 ppm (0.28 mg/m^3) of nitrogen dioxide and 0.10 ppm (0.26 mg/m^3) of sulfur dioxide developed injury. These concentrations were well below the threshold concentrations for injury due to these pollutants when provided alone. After exposing several garden vegetables to mixtures of nitrogen dioxide and sulfur dioxide, Matsushima[336] observed greater leaf injury than that caused by each pollutant alone. He also provided different sequences of exposure. When nitrogen dioxide exposure preceded sulfur dioxide exposure the degree of injury was similar to that resulting from individual exposures to either of the gases. But when sulfur dioxide exposure was followed by nitrogen dioxide exposure, the degree of leaf injury increased as in simultaneous exposures to both pollutants together. Mixtures of nitrogen dioxide and ozone caused less injury to tomato and pepper (*Capsicum frutescens*) plants than when either was supplied alone.[336]

SUSCEPTIBILITY OF PLANTS TO NITROGEN DIOXIDE

The relative susceptibilities of some plant species to nitrogen dioxide are listed in Table 9-1. The three classes--susceptible, intermediate, and resistant--are approximate because they are based on subjective criteria from several sources.[42,95,330,331,353,530,577] Most of the classifications are derived from experimental fumigations at various locations at different times of the year using different nitrogen dioxide exposures. Methods for assessing such injury as percentage of leaves injured, amount of leaf surface affected, defoliation, etc. also varied. Because of these variables, a plant species considered resistant by one investigator may be considered susceptible by another.

PLANT RESPONSES IN RELATION TO AIR QUALITY

The concentration of the pollutant and the duration of exposure are collectively referred to as dose. The lowest dose that produces an effect is termed a threshold dose. Because of the interrelationship between concentration and time, there is no single threshold dose for an effect to occur. For example, leaf injury on a given plant or set of plants exposed to 20 ppm (38 mg/m^3) of nitrogen dioxide might occur after only 1 hr, but an exposure of 1 ppm (1.9 mg/m^3) might require up to 100 hr to produce leaf injury. Threshold doses are therefore often described as functions of the pollutant concentration and exposure time.[344]

MacLean[329] recently reviewed the responses of plants to NO_x. His threshold curves for complete defoliation of citrus (*Citrus sinensis*), azalea (*Rhododendron canescens*), and hibiscus (*Hibiscus rosa-sinensis*) illustrate the relation between nitrogen dioxide concentration and duration of exposure (Figure 9-1). The discussion presented by MacLean[329] stated that:

> Although these threshold curves [Figure 9-1] are based on limited data, they suggest that a function relating exposure time and

TABLE 9-1

Susceptibilities of Selected Plants to Nitrogen Dioxide[a]

Plant	Susceptible	Intermediate	Resistant
Conifers			
Abies alba Mill. - White fir		+	
Abies homolepis Sieb. & Zucc. - Nikko fir		+	
Larix decidua Mill. - European larch	+		
Larix leptolepis Gord. - Japanese larch	+		
Picea glauca (Moench) Voss - White spruce		+	
Picea pungens cv. *glauca*, Regel - Colorado blue spruce		+	
Pinus nigra Arnold - Austrian pine			+
Taxus baccata L. - English yew			
Deciduous trees and shrubs			
Acer palmatum Thunb. - Japanese maple		+	
Acer platanoides L. - Norway maple		+	
Betula pendula Roth. - European white birch	+		
Carpinus betulus L. - European hornbeam			+
Fagus sylvatica L. - Beech			+
Gingko biloba L. - Gingko			+
Quercus robur L. - English oak			+
Robinia pseudoacacia L. - Black locust			+
Sambucus nigra L. - Elder			+
Tilia cordata Mill. - Little-leaf linden		+	
Ulmus glabra Huds. - Scotch elm			+
Field crops and grasses			
Avena sativa L. - Oats	+		
Hordeum distichon L. - Barley	+		
Medicago sativa L. - Alfalfa (lucerne)	+		
Nicotiana glutinosa L. - Tobacco	+		
Poa annua L. - Annual blue grass		+	
Poa pratensis L. - Kentucky blue grass			+
Secale cereale L. - Rye		+	
Solanum tuberosum L. - Potato		+	
Trifolium incarnatum L. - Spring clover	+		
Trifolium pratense L. - Red clover	+		
Triticum sativum Lam. - Wheat		+	
Vicia sativa L. - Spring vetch	+		
Zea mays L. - Sweet corn		+	

TABLE 9-1 - continued

Plant	Susceptible	Intermediate	Resistant
Fruit trees			
Citrus sp. - Orange, grapefruit, tangelo		+	
Malus sp. - Apple (wild)	+		
Pyrus sp. - Pear (wild)	+		
Garden crops			
Allium cepa L. - Onion			+
Allium porrum L. - Leek	+		
Apium graveolens L. - Celery[b]	+	+	
Asparagus officinalis L. - Asparagus			+
Brassica oleracea cv. *capitata* L. - Cabbage			+
Brassica caulorapa Pasq. - Kohlrabi			+
Daucus carota L. - Carrot[b]	+		+
Lactuca sativa L. - Lettuce	+		
Lycopersicon esculentum Mill. - Tomato		+	
Petroselinum hortense Nym. - Parsley	+		
Phaseolus vulgaris cv. *Pinto* L. - Pinto bean	+		
Phaseolus vulgaris cv. *humilis* Alef. L. - Bush bean		+	
Pisum sativum L. - Pea	+		
Rheum rhaponticum L. - Rhubarb	+		
Ornamental shrubs and flowers			
Antirrhinum majus L. - Snapdragon	+		
Begonia sp. - Tuberous begonia	+		
Bougainvillea Spectabilis Willd. - Bougainvillea	+		
Carissa carandas L. - Carissa			+
Chrysanthemum leucanthemum L. - Daisy			+
Codiaeum variegatum Blume - Croton			+
Convallaria majalis L. - Lilly-of-the-valley			+
Dahlia variabilis Willd. - Dahlia		+	
Erica carnea L. - Spring heath			+
Fuchsia hybrida Voss - Fuchsia		+	
Gardenia jasminoides Ellis - Cape jasmine		+	
Gardenia radicans Thunb. - Gardenia		+	
Gladiolus sp. - Gladiolus			+
Hibiscus rosa-sinensis L. - Chinese hibiscus	+		
Hosta sp. - Plantain lily			+

TABLE 9-1 - continued

Plant	Susceptible	Intermediate	Resistant
Ornamental shrubs and flowers cont'd.			
Ixora coccinea L. - Ixora		+	
Juniperus conferta Parl. - Shore juniper			+
Lathyrus odoratus L. - Sweet pea	+		
Ligustrum licidum Ait. - Ligustrum		+	
Melaleuca leucadendra L. - Bottle brush		+	
Nerium oleander L. - Oleander	+		
Petunia multiflora - Petunia		+	
Pittosporum tobira Ait. - Japanese pittosporum		+	
Pyracantha coccinea Roem. - Pyracantha	+		
Rhododendron canescens Sweet - Azalea	+		
Rhododendron catawbiense Michx. - Catawba rhododendron		+	
Rosa sp. - Rose[b]	+		+
Weeds			
Amaranthus sp. - Pigweed			+
Brassica sp. - Mustard	+		
Chenopodium album L. - Lamb's-quarters			+
Chenopodium sp. - Nettle-leaved goosefoot			+
Helianthus annuus L. - Sunflower	+		
Malva parviflora L. - Cheeseweed		+	
Stellaria media Cyrill. - Chickweed		+	
Taraxacum officinale Weber - Dandelion		+	

[a] Compiled from references: 42,95,330,331,353,530, and 577

[b] Different investigators reported different susceptibilities.

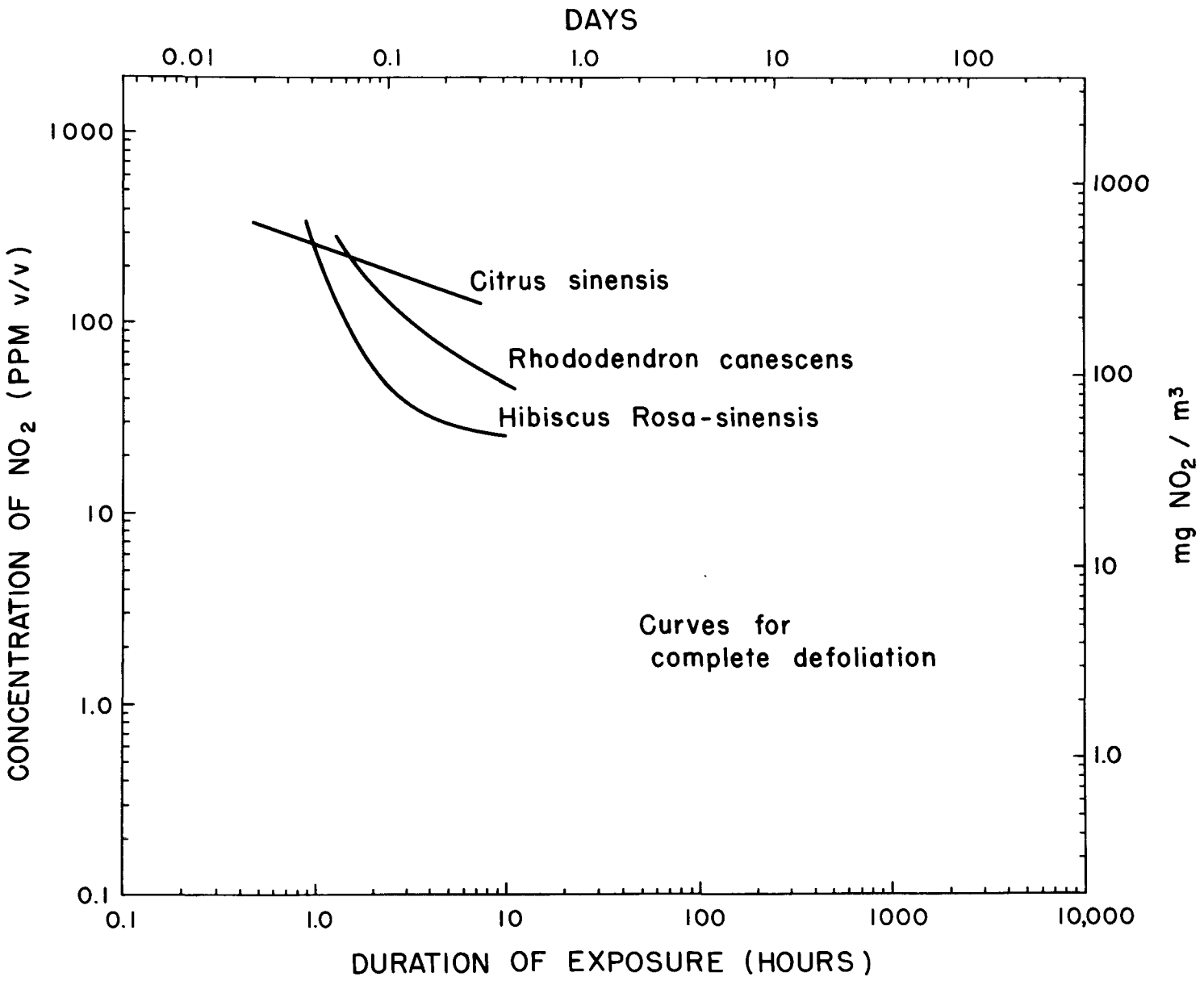


FIGURE 9-1. Threshold curves for complete defoliation of Citrus sinensis, Rhododendron canescens, and Hibiscus rosa-sinensis as related to the nitrogen dioxide concentration and the duration of exposure. (Reprinted with permission from MacLean.[329])

concentration can be computed for every effect of NO_2 if enough information is known about that effect over a relatively wide range of concentrations and exposure periods.

Three threshold curves are shown in Figure [9-2]. These are approximate estimates based on the various responses of many plant species determined in all of the acute and chronic NO_2 doses reported in references [42,43,95,122,234,330,331,336,353, 500,529,536,538,599]. The threshold curve for NO_2 doses that result in the death of plants is short because it is based on limited information.[95] NO_2 doses approaching this threshold result in complete defoliation of some plant species, but are not lethal.[330] The threshold curve for leaf injury is based on observations at many NO_2 doses. The shift in leaf injury from necrosis to chlorosis for NO_2 doses along this curve generally occurs between 10 and 100 hr. Because no measurable effects have been reported for NO_2 doses below the lower curve, it can be considered as the threshold for metabolic and growth effects. NO_2 doses in the area between this curve and the threshold curve for leaf injury are those that do not injure leaves, but often result in growth suppression or effects on photosynthesis or other plant processes.

These thresholds [Figure 9-2], assuming that they are reasonable estimates for vegetation in general, can serve as points of reference to evaluate air quality standards for NO_2, and they can be viewed with respect to the NO_2 concentrations that occur in the atmosphere.

The U.S. air quality standard for nitrogen dioxide [0.05 ppm (0.10 mg/m^3) for an annual average] is below the threshold curve for metabolic and growth effects in vegetation, and it should afford reasonable protection from nitrogen dioxide-induced plant damage (economic losses).

MacLean[329] also pointed out that:

Localized episodes resulting from accidental releases or spills of NO_x account for most NO_2-induced plant injury and damage. But for agricultural activities near urban areas where the emissions of NO_x to the atmosphere are primarily from stationary sources that burn coal, oil, natural gas, and other fuels, and from the combustion of motor vehicle fuels, the probability of direct effects of NO_2 on vegetation is very low. Average NO_2 concentrations in most major cities of the United States are well below the threshold curve for metabolic and growth effects. In fact, the maximum NO_2 concentrations recorded in Los Angeles, California[563] for an entire year (1966) are only slightly higher than the threshold curve for metabolic and growth effects for averaging times of one day or less and below this threshold curve for longer averaging periods [Figure 9-2].

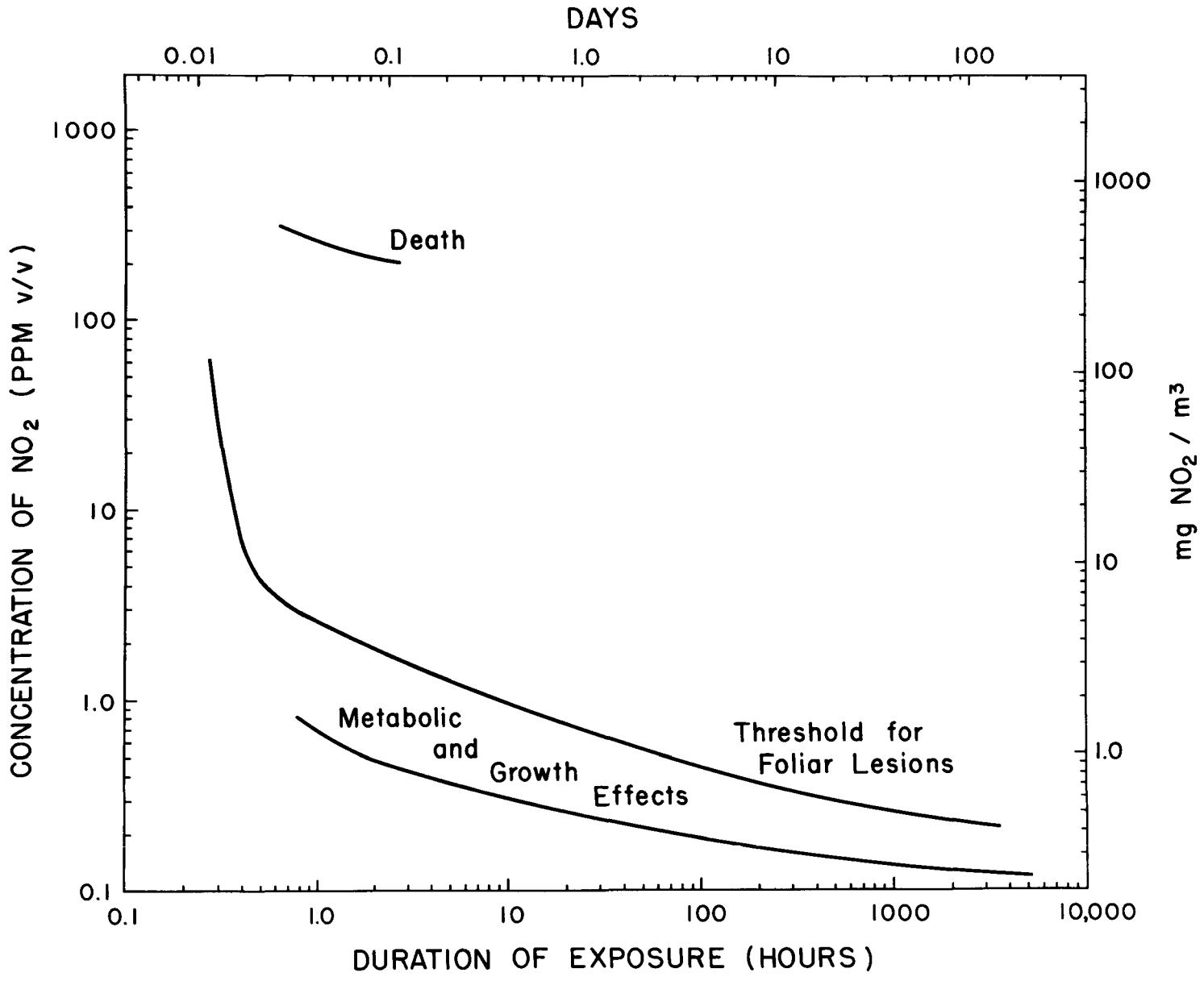


FIGURE 9-2. Threshold curves for the death of plants, foliar lesions, and metabolic or growth effects as related to the nitrogen dioxide concentration and the duration of exposure. (Reprinted with permission from MacLean.[329])

Indirect effects of NO_2 on vegetation, however, probably do occur. The participation of NO_2 in atmospheric reactions leading to the production of ozone and peroxyacyl nitrates, and the synergistic effects on plant injury of relatively low concentrations of mixtures of NO_2 and SO_2 in the atmosphere pose a real threat to vegetation growing in or near metropolitan areas.

FUTURE RESEARCH

Since plants are relatively insensitive to nitric oxide as compared with nitrogen dioxide, the emphasis in NO_x research should be directed toward the latter. The gross effects of nitrogen dioxide have been fairly well documented, and the utility of additional research would be limited primarily to the evaluation of injury due to localized accidental nitrogen dioxide releases. Future research, therefore, should emphasize the effects of chronic and intermittent nitrogen dioxide exposures, at realistic concentrations and exposure times, on symptomatology, and on plant growth and yield. Furthermore, little is known about the primary effects of nitrogen dioxide at the cellular or metabolic levels of organization. Information in this area is needed to provide an understanding of nitrogen dioxide effects on vegetation.

The combined effects of NO_x and other substances in the atmosphere on plants is another important area in which information is limited. In a few plant species, atmospheres containing both nitrogen dioxide and sulfur dioxide cause greater injury than when either pollutant occurs alone,[122,336,538] whereas the combination of nitrogen dioxide and ozone results in reduced leaf injury.[336] Experimental exposures of nitrogen dioxide in combination with other atmospheric constituents should be carried out on a wide variety of plant species to reveal the interacting effects of nitrogen dioxide and other pollutants on phytotoxicity.

SUMMARY AND CONCLUSIONS

Nitric oxide is less injurious to vegetation than nitrogen dioxide, and both are less phytotoxic than sulfur dioxide, gaseous fluoride, or photochemical oxidants. Although injuries to plants by both nitric oxide and nitrogen dioxide have been experimentally induced, there have been no confirmed reports for nitric oxide injury to vegetation in the field. Nitrogen dioxide-induced injury in the field has been associated with accidental acute exposures from certain industrial processes.

Nitrogen dioxide doses in or near metropolitan areas rarely exceed the threshold dose for effects on plant metabolism or growth; those that occur in suburban or rural areas are even lower. The existing U.S. air quality standard for nitrogen dioxide [0.05 ppm (0.10 mg/m^3) for an annual average] is below the threshold for detectable effects on vegetation. Therefore, secondary standards to protect vegetation from the direct effects of nitrogen dioxide are not necessary.

Indirect effects of nitrogen dioxide on vegetation, however, are more likely. Nitrogen dioxide participation in atmospheric reactions leading to ozone and peroxyacylnitrate production, and the synergistic effects on plant injury of low concentrations of nitrogen dioxide and sulfur dioxide mixtures in the atmosphere may pose a real threat to vegetation growing in or near metropolitan areas. As knowledge of these phenomena increases, air quality standards for nitrogen dioxide to protect vegetation may have to be reevaluated.

CHAPTER 10

HEALTH EFFECTS OF OXIDES OF NITROGEN

Considerable evidence has recently accumulated indicating that oxides of nitrogen are deleterious components of air pollution.[91, 177, 199, 486, 487, 518] Most of this evidence is based on the relationship of human or animal exposure to oxides of nitrogen and resultant measurements of pulmonary dysfunction. It is implicit in these experiments that the measured abnormality either results in chronic pulmonary disease (emphysema or bronchitis) or increases the deficit from an already present chronic respiratory disease. Inasmuch as these diseases affect approximately 4% of the American population,[176] this postulated sequence of events is of paramount importance. More recently, human exposure to oxides of nitrogen has been associated with an increased susceptibility to acute respiratory infections.[411, 486, 487] The morbidity from these frequently occurring illnesses can have considerable economic and social significance. Exposure to nitrogen dioxide (NO_2) also causes visual and olfactory disturbances of uncertain medical significance.[51, 224]

Nitrous oxide (N_2O), nitric oxide (NO), nitrogen dioxide, dinitrogen trioxide (N_2O_3), dinitrogen pentoxide (N_2O_5), and nitrate ions (NO_3^-) are the oxides of nitrogen that are present in the atmosphere. Of these oxides, nitrogen dioxide is by far the most significant biologically.

Interest in the toxicity of nitrogen dioxide has led to many investigations with laboratory animals in which pollutant exposure has been related to mortality, respiratory pathology and physiology, pulmonary resistance to microbial infection, and, in a few instances, to extrapulmonary dysfunction.

Before evaluating these studies, some aspects of the experiments are worth emphasizing. Under the true environmental conditions the deleterious

effects of air pollution caused by exposures to a mixture of interacting pollutants, one of which is nitrogen dioxide. Realistic assessment of the significance of health effects of nitrogen dioxide must be considered within this context. However, before toxic effects of mixtures of air pollutants can be determined, the effects of individual pollutants must be known. Studies conducted in laboratory animals provide information on the maximum tolerated dose in various animal species, define the target organs, and provide clues as to the mechanisms of damage. Considerable information is now available on the toxic effects of nitrogen dioxide in animals and man and engineering technology has advanced to a point where exposure to mixtures of airpollutants can be carefully monitored. Consequently, effects of nitrogen dioxide on humans can now be studied under conditions more realistically resembling true environmental exposures.

A second important factor concerns the relation of the animal model to the human. Animal models are often similar but not completely analogous to humans. As an example, the respiratory anatomy of rats (the animal most frequently used in studies of nitrogen dioxide-induced emphysema) differs from that of humans in that they do not have interlobular septa, they have fewer generations of airways, and their respiration occurs through distal bronchioles rather than alveoli and pulmonary vasculature.[551] The epithelium of the tracheobronchial tree of rats also differs from that of man. It has more large mucus-secreting glands lining the trachea and fewer along the bronchi.[445] Important anatomic differences between humans and rabbits, mice, guinea pigs, monkeys, and dogs[551] explain why identical concentrations of nitrogen dioxide can cause diverse pathologic disturbances which make it difficult to apply such results to disease in man.

The few studies of inhaled nitrogen dioxide indicate that this pollutant is probably distributed throughout the lung and that a high percentage of it is absorbed.[248, 584] Von Nieding and his coworkers exposed volunteers to nitrogen dioxide concentrations of 0.94-9.4 mg/m^3 (0.5-5.0 ppm) and analyzed the gas concentrations in exhaled air to determine the extent of absorption.[584] These investigators reported that 81-87% of inhaled nitrogen dioxide is absorbed during normal ventilation and that more than 90% is absorbed during periods of maximal ventilation. Ichioka designed a model airway to simulate the dynamic behavior of nitrogen dioxide within the respiratory system. When nitrogen dioxide at 9.4 mg/m^3 (5.0 ppm) flowed through the model, most of the pollutant penetrated the more distal regions.[248] Although more exacting studies of intrapulmonary distribution and absorption, subh as those available for sulfur dioxide,[153] have not been performed, much of the inhaled pollutant seems to react throughout the lung.

TOXICOLOGIC EFFECTS OF NITROGEN DIOXIDE IN LABORATORY ANIMALS

Mortality

Figure 10-1 compares mortality for rats, mice, guinea pigs, rabbits, and dogs after a 1-hr exposure to increasing concentrations of nitrogen dioxide. These data, which are abstracted from a study by Hine *et al.*,[237] indicate a mortality threshold concentration of 75-94 mg/m^3 (40-50 ppm) after 1-hr exposures of these laboratory animals. Rats, mice, and guinea pigs appear to be more susceptible to the pollutant than rabbits or dogs. Experiments in which nonhuman primates were exposed to nitrogen dioxide for 8 hr at 122 mg/m^3 (65 ppm) have shown that monkeys are slightly more susceptible to nitrogen dioxide than similarly exposed rodents.[504] Once the

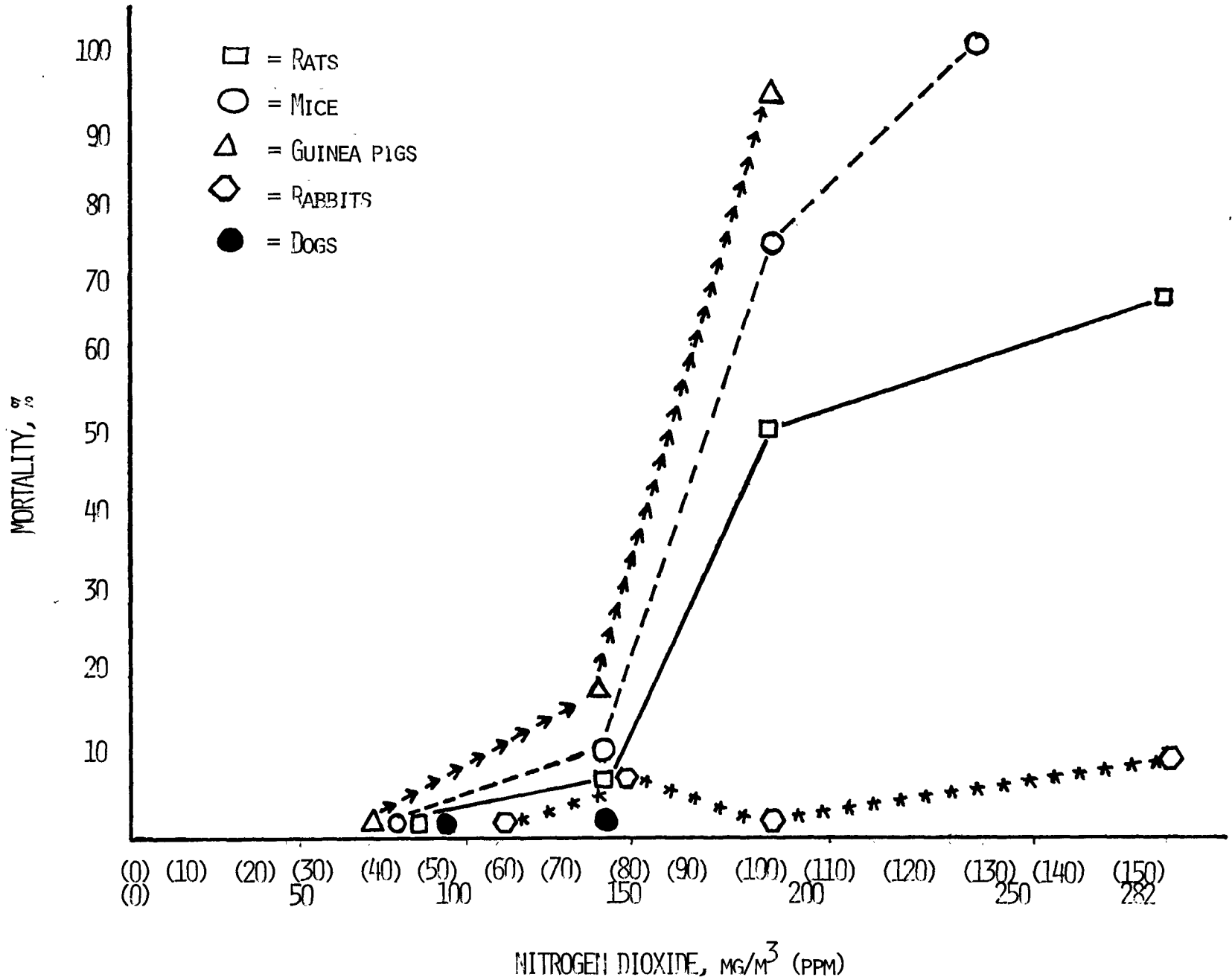


FIGURE 10-1. MORTALITY OF DIFFERENT ANIMAL SPECIES AFTER A 1-HR EXPOSURE TO NITROGEN DIOXIDE
DATA FROM HINE, MEYERS, AND WRIGHT. TOXICOLOGY AND APPLIED PHARM. 16:201-213, 1970.[237]

the threshold concentration is exceeded, the death rate for each species increases as the exposure period is lengthened. When compared, brief exposures to high concentrations of nitrogen dioxide are much more toxic than exposures to low concentrations for long periods.

All animal species studied survive continuous exposures of a year or more to peak ambient concentrations of nitrogen dioxide.[27,129,144,158,159,160,161,586] (See Table 10-1.) Because these studies did not include higher concentrations of nitrogen dioxide, it is not known whether any of these species would tolerate even more severe exposures.

Pulmonary Effects

Pulmonary Function. The effect of short-term exposure to nitrogen dioxide on pulmonary function has been studied in the guinea pig,[382] monkey,[221] and rabbit.[583] Guinea pigs manifest an increase in respiratory rate and a decrease in tidal volume after a 2-hr exposure to nitrogen dioxide at 122 mg/m^3 (65 ppm).[382] Similar results occur after monkeys are exposed for 2 hr to 28 mg/m^3 (15 ppm).[221] These abnormalities are reversible. Lesser exposures have not caused dysfunction. Pulmonary diffusing capacity is reduced in rabbits after a 15-min exposure to 56 mg/m^3 (30 ppm).[583]

Table 10-2 summarizes the respiratory effects that follow chronic exposure to nitrogen dioxide. Data in that table indicate that prolonged exposure to peak ambient concentrations of nitrogen dioxide causes abnormalities in respiratory function, which appear to revert to normal after the exposure ends.

Pathologic Changes. Pathology in the lungs occurs primarily in animals exposed for less than 24 hr to concentrations of nitrogen dioxide exceeding 13.16 mg/m^3 (7 ppm).[397] Various degrees of vascular congestion, edema, bronchiolitis, consolidation, exudative plugging of the bronchi, tissue destruction,

TABLE 10-1

Survival of Animals Exposed Chronically to High Concentrations of Nitrogen Dioxide

Species	Concentration mg/m^3	Concentration ppm	Duration of Exposure	Fatalities Attributed to Exposure	Ref
Mice	0.94	0.5	12 months	None reported pneumonitis	129
Rats	1.52	0.8	Lifetime	None	159
	3.76	2.0	Lifetime	None	161
	23.50	12.5	213 days	11% fatality	160
Guinea pigs	7.52	4.0	4 hr/day, 5 days/week for 6 months	None	27
	28.20	15.0	6 months	None	27
Squirrel Monkeys (*Saimiri sciureus*)	1.88	1.0	16 months	None	144
Stump-tailed macaque (*Macaca speciosa*)	3.76	2.0	2 years	None	158
Dogs	9.40	5.0	15 months	None	586
Rabbits	2.44	1.3	17 weeks	None	358

TABLE 10-2

Respiratory Effects of Chronic Exposure to Nitrogen Dioxide

Species	Concentration mg/m^3	Concentration ppm	Duration	Effect	Ref
Rats	1.504	0.8	Lifetime	Tachypnea	159
	3.760	2.0	Lifetime	Tachypnea, normal resistance, and dynamic compliance	161
Guinea pigs	9.4	5.0[a]	5.5 months	No change expiratory flow resistance	27
Rabbits	1.88	1.0	18 months	Normal oxygen consumption	397
	9.4	5.0	18 months		
	15.04-22.56	8.0-12.0	3-4 months	Decreased arterial blood oxygen and static lung compliance, increase in nonelastic resistance and functional residual capacity. These effects disappeared when rabbits were permitted to breathe room air.	98
Dogs	0.94-1.88	0.5-1.0[b]	16 hr/day for 18 months	Normal $D_L CO$	579
Squirrel Monkeys (*Saimiri sciureus*)	9.4	5.0	2 months	Tachypnea, decreased tidal volume, normal minute volume	222
	9.4	5.0	2 weeks	Decreased tidal volume	222
Stump-tailed macaque (*Macaca speciosa*)	3.76	2.0	2 years	Tachypnea	158

[a]For 4 or 7.5 hr/day, 5 days/week
[b]Plus 0.250 mg/m^3 (0.2 ppm) nitric oxide

abscess formation, and pneumonitis occurred in all species that have been studied.[78, 118, 221, 237, 286, 287, 309, 504]

The extent of damage corresponds to exposure dose. Acute exposures to nitrogen dioxide concentrations of 3.760-5.640 mg/m^3 (2-3 ppm), which are close to ambient levels, have not resulted in canine or murine morphologic abnormalities,[118, 513] nor have those exposures affected cellular structure.[513] At higher concentrations, ultrastructural and scanning electron micoscopic studies have revealed loss of cilia, swelling, and disruption of type I alveolar cells, fibrin deposition along basement membranes, and an influx of macrophages.[408, 513] These pathologic lesions appear to be transient. When the insult is discontinued, the damaged tissue returns to normal.

Rodents have most commonly been used to study the pathology resulting from prolonged exposure to nitrogen dioxide (Table 10-3).[48, 65, 85, 156-161, 194, 213, 214, 286, 360, 383, 504, 512, 617] In rats exposed to nitrogen dioxide at 18.8, 23.5, or 47 mg/m^3 (10, 12.5, or 25 ppm) for 3 or more months, the thoracic cavities become larger, dorsal kyphosis develops, and the animals acquire an inflated appearance. There is distention of alveolar ducts, dilatation of alveoli, and hyperplasia of bronchiolar epithelium. Alveolar septa are occasionally missing, but destruction of parenchyma is unusual.[157, 214] These pathologic features are similar but not identical to those of human emphysema. A critical difference is the absence of alveolar necrosis.

Destructive bullous lesions are the sine qua non of emphysema,[19] but these bullae do not develop in rodent models, even in rats exposed for a lifetime to nitrogen dioxide concentrations of 1.5 and 3.8 mg/m^3 (0.8 and 2.0 ppm). The lungs from these animals are grossly normal; microscopic examination shows only minor ciliary loss, epithelial hypertrophy, and "cytoplasmic blebbing."[159, 214] These animals have a normal lifespan and die of diseases unrelated to nitrogen dioxide exposures.[156]

TABLE 10-3

Pathology in Animals Exposed Chronically to High Concentrations of Nitrogen Dioxide

Species	Concentration mg/m^3	Concentration ppm	Duration of Exposure	Pathology Attributed to Exposure	Ref
Mice	0.9	0.5	3 months	Ciliary loss, alveolar cell disruption	156
	0.9	0.5	6, 18, and 24 hr/day for 3-4 months	Expanded alveoli, reduction of distal airway size, progressive parenchymal damage	48
	0.6- 0.9	0.3- 0.5	6 months	Destruction bronchial epithelium, lymphocytic infiltration	85
	75.2	40.0	6-8 weeks	Epithelial abnormalities of terminal bronchioles	65
Rats	1.5- 3.8	0.8- 2.0	Lifetime	Ciliary loss, epithelial hyperplasia	159, 214
	3.8	2.0	2 or more years	Thickened basement membrane	512
	31.9	17.0	20 months	Massive increase in collagen fibrils	512
	18.8- 47.0	10.0-25.0	3 or more months	Enlarged thoracic cavities, dorsal kyphosis, distended alveoli and alveolar ducts	157, 214
Guinea pigs	9.4	5.0	7.5 hr/day, 5 days/week, 5.5 months	Perivascular and tracheal inflammation, desquamative pneumonitis	27
	18.8	10.0	6 weeks	Hyperplasia of type 2 pneumatocytes	617
	28.2- 37.6	15.0-20.0	2 hr/day, 5 days/week, 21 months	Inflammation bronchiolar epithelium	287
	41.4	22.0	2 hr/day, 3 weeks	Multifocal emphysematous changes	194
Rabbits	15.0- 22.6	8.0-12.0	3-4 months	Necrosis of alveolar walls with enlargement of air spaces	213
	28.2- 47.0	15.0-25.0	2 hr/day up to 2 years	No emphysematous lesions	286
Hamsters	84.6-103.4	45.0-55.0	21-23 hr/day	Dilated alveolar spaces, inflammatory cells, epithelial hyperplasia, increased lung volume	286
Dogs	9.4	5.0	15-18 months	No abnormalities	586
	48.9	26.0	6 months	Bullous emphysema	320
Squirrel Monkeys (*Saimiri sciureus*)	9.4	5.0	169 days	Focal alveolar edema	143

Electron microscopic studies of the lungs of rats exposed to nitrogen dioxide at 3.8 mg/m^3 (2.0 ppm) revealed hypertrophy and focal hyperplasia in the epithelium of the terminal bronchiole and a loss of cilia. These minor abnormalities, which appear after the third day of exposure, apparently heal in the presence of nitrogen dioxide and disappear at 21 days.[513] After 2 or more years exposure to the same concentration, the only abnormality in animals was thickening of the basement membrane under the epithelium of the terminal bronchiole due to enlarged collagen fibrils.[512]

Exposure to 32 mg/m^3 (17 ppm) causes much more severe ciliary loss, injury to the epithelium lining the alveoli adjacent to the terminal bronchioles, sloughing of type I alveolar epithelial cells, thickening of the air-blood barrier, and fibrin deposition along the basement membrane. But even in animals exposed to this high concentration, the destructive phase was followed by a period of adaptation and repair.[513] The principal abnormality in 2-year-old rats after exposure to 32 mg/m^3 (17 ppm) for 610 days was a massive increase in the size of the collagen fibrils.[512]

Mice are more susceptible to the toxic effects of nitrogen dioxide than rats. Continuous exposure to nitrogen dioxide at 0.94 mg/m^3 (0.5 ppm) for 3 months causes loss of cilia, alveolar cell disruption, and obstruction of respiratory bronchioles.[156] Longer exposures cause more severe bronchiolar inflammation, pneumonitis, and increases in alveolar area, secondary to alveolar expansion rather than septal breakage.[48]

Epithelial cell proliferation of the peripheral bronchus occurred in mice that were exposed to 0.94-1.5 mg/m^3 (0.5-0.8 ppm) for 30-45 days.[383] Electron microscopic studies of these lungs revealed mild

ciliary abnormalities, degeneration of the mitochondria of Clara and alveolar cells, and edematous changes within the cytoplasm.[383] These abnormalities have been confirmed histopathologically,[85] and in later scanning and electron microscopic studies.[360] Some investigators classified these changes as representative of chronic bronchitis and chronic tracheitis.[85] Germ-free mice that were exposed continuously to 75 mg/m^3 (40 ppm) for 6-8 weeks developed epithelial abnormalities throughout the bronchial tree. These were most pronounced at the terminal bronchioles and in the alveoli immediately surrounding the terminal bronchioles.[65] Inflammation, characterized by edema and exudation of white blood cells, was not observed in the nitrogen dioxide-provoked lesions.[65]

Haydon _et al_. exposed rabbits continuously to atmospheres containing nitrogen dioxide at 15-23 mg/m^3 (8-12 ppm) for 3-4 months and reported destructive changes in alveolar walls and abnormal enlargement of the distal air spaces.[213] These findings approximate the emphysematous lesions observed in humans. Unfortunately, there are no reports of data obtained from rabbits exposed to lower concentrations of nitrogen dioxide. Other investigators have failed to find emphysematous changes in rabbits exposed for 2 hr/day to nitrogen dioxide at 28-47 mg/m^3 (15-25 ppm) for periods up to 2 years.[286]

A multifocal type of emphysema has been induced in guinea pigs after 3 weeks of exposure for 2 hr/day to nitrogen dioxide at 41 mg/m^3 (22 ppm).[194] An ultrastructural study of the lungs of 12 guinea pigs exposed continuously to 18.8 mg/m^3 (10 ppm) for 6 weeks revealed greatly increased proportions of type II pneumatocytes, presumably resulting from hyperplasia,[617] and minor cellular changes consisting of increased numbers of tightly packed lamellae and of small and giant lipid bodies.

Kleinerman and Cowdry did not find emphysematous changes in hamsters after exposing them to 85-103 mg/m^3 (45-55 ppm) for 21-23 hr/day for 10 weeks. Dogs are also very resistant to the toxic effects of nitrogen dioxide. Wagner et al. determined that lungs of dogs exposed to 9.4 mg/m^3 (5.0 ppm) for 15-18 months did not exhibit differences when compared with lungs of control animals.[586] These negative results were confirmed by later investigators.[504, 579] Bullous emphysema has been induced in dogs but only after prolonged exposure (6 months) to high concentrations, 48.9 mg/m^3 (26 ppm).[320]

Vascular Permeability and Edema Formation. A potentially important pathophysiologic consequence of exposure to nitrogen dioxide is injury to vascular membranes with a resulting increase in capillary permeability, transudation of protein into alveoli, and edema formation.[191, 237, 482, 518] Edema formation has been a significant abnormality in experiments in which animals were exposed acutely to high concentrations of nitrogen dioxide.

The difficulties encountered with present measuring techniques should be thoroughly understood before attempts are made to evaluate chronic exposures that do not generally produce edema. The most common method determines a wet:dry weight ratio that is a very crude estimate of edema fluid.[11, 132] This ratio remains unaltered in rats that are exposed chronically to high concentrations of nitrogen dioxide.[157] Pulmonary weights, both dry and wet, increase proportionately in these animals, indicating that pulmonary mass (cell and fibrous tissues) increases as a consequence of pollutant exposure.[156, 157, 160] Although histologic and electron microscopic examination of pulmonary tissue provides a more precise determination of the presence of edema,[195] small quantities of edema fluid may still remain undetected.[11, 195]

Focal alveolar edema was reported in squirrel monkeys that were exposed continuously to nitrogen dioxide at 9.4 mg/m^3 (5.0 ppm) for 169 days.[143] Edema was either absent[157, 504, 586] or not mentioned[48, 98, 213, 617] in most other pathologic studies. Edema was not noted in three electron microscopic investigations of the effect on the lung of prolonged exposure to nitrogen dioxide.[408, 512] The results of one ultrastructural study indicated early edematous changes within alveolar epithelial cells and alveolar interstitium in mice that were exposed continuously to nitrogen dioxide at 0.94-1.5 mg/m^3 (0.5-0.8 ppm) for 30-45 days.[383]

A new and potentially precise method for detecting small alterations in pulmonary capillary permeability has been reported by Sherwin and Richters.[482] This method, in which tritium-labeled serum is used to assess intrabronchial protein leakage, showed that exposure of mice to nitrogen dioxide at 9.4 mg/m^3 (5.0 ppm) for 14-72 hr was associated with transient increases in intrabronchial radioisotope. Because of the preliminary nature of these studies and the small number of animals that have been studied, these data cannot be used to form conclusions about edema formation. More recently, Sherwin and Carlson have used a disk-gel electrophoresis method to measure intrabronchial protein in guinea pigs that were exposed for a week to nitrogen dioxide at 0.75 mg/m^3 (0.4 ppm).[480] Their preliminary results suggest that transudation of protein results from such exposures.

Biochemical Abnormalities. The biochemical mechanisms by which nitrogen dioxide causes cellular dysfunction are in the initial stages of investigation. Although a few potentially important biochemical abnormalities have been described, their significance in relation to the observed pathophysiologic disturbances is speculative.

Nitrogen dioxide causes lipoperoxidation of murine lung lipids[531] and decreases in pulmonary lipid content and total saturated phospholipid fatty acids.[24,278] These changes, similar to those noted with ozone, have been interpreted as evidence that free radical formation is the mechanism by which injuries from nitrogen dioxide exposure occur.[475,518,578] Of potential practical significance is the additional finding by Thomas and his coworkers[531] that pretreatment with antioxidant (10 mg of vitamin E per day) was partially effective in preventing the lipid peroxidative changes induced by nitrogen dioxide. Such sulfur compounds as α-naphthylthiourea ($C_{10}H_7NHCSNH_2$) and phenylthiourea ($C_6H_5NHCSNH_2$) also protect rodents from nitrogen dioxide-induced damage, presumably by acting as antioxidants. Exposure to nitrogen dioxide adversely alters pulmonary surfactant, which may explain the observed impairments in lung compliance.[24,118]

In studies of the interaction of nitrogen dioxide and cellular enzyme systems,[27,64,354,578] it was discovered that some enzyme systems, such as catalase[578] and lactic dehydrogenase,[27] are susceptible to damage by the pollutant, whereas other enzymes such as cathepsin D, seem to be impervious.[354]

A possible relation between exposure to nitrogen dioxide and pulmonary neoplasia has been the subject of preliminary studies.[169,407] Gardner obtained some evidence that nitrogen dioxide exposures may contribute to lung tumor development in mice.[169] Because benzopyrene hydroxylase presumably inactivates the carcinogenic potential of polyaromatic hydrocarbons, the effect of an acute exposure to nitrogen dioxide on this enzyme system was evaluated in studies of rabbit tracheobronchial mucosa. No effect was found.[407]

Extrapulmonary Effects

Nitrogen dioxide has been associated with some systemic effects. A diminished rate of weight gain occurs in rats exposed chronically to nitrogen dioxide at a high concentration [23 mg/m^3 (12 ppm)][156] and in rabbits exposed to lower concentrations [2.4 and 5.6 mg/m^3 (1.3 and 3.0 ppm)].[358] Growth abnormalities do not occur uniformly. Dogs have been exposed to 1.8 and 9.4 mg/m^3 (1.0 and 5.0 ppm) for 6 hr/day for 18 months[586] and mice to 0.94 mg/m^3 (0.5 ppm) for 5 days each week for up to 12 months without alterations in weight gain.[129]

Minor changes in hematologic indexes have been reported in some species after prolonged exposure to nitrogen dioxide. Rats develop a transient polycythemia characterized by increases in hematocrit and hemoglobin and decreases in mean corpuscular volume and hemoglobin.[158] These changes appear 2-3 weeks after exposure and spontaneously regress, despite continued exposure, after approximately 3 months. Leukocytosis has been reported in rabbits that were exposed to 2.4 and 5.6 mg/m^3 (1.3 and 3.0 ppm) for 15-17 weeks.[358] Other investigators have not reported hematologic abnormalities in rats,[504] rabbits,[504] dogs,[504,586] guinea pigs,[504] and squirrel monkeys[143] resulting from continuous exposure to nitrogen dioxide.

The effect of nitrogen dioxide on organs other than the lung has received little attention.[48,481,504] Pathologic studies of the heart, thymus, spleen, liver, and kidney did not disclose alterations in mice exposed continuously to 0.94 mg/m^3 (0.5 ppm) for 12 months.[48] Studies using other animal species have also failed to reveal extrapulmonary abnormalities.[504]

Proteinuria has been reported in guinea pigs after continuous exposure to nitrogen dioxide at 0.94 mg/m^3 (0.5 ppm) for 7-14 days.[481] This urinary abnormality was not associated with renal pathology.

EFFECTS OF COMBINED EXPOSURES TO NITROGEN DIOXIDE AND OTHER AGENTS IN LABORATORY ANIMALS

The importance of nitrogen dioxide as a causative or aggravating agent in respiratory infections is well documented.[91, 126-130, 144, 177, 178, 221, 257, 428, 486, 487, 519] The pollutant appears to enhance host susceptibility to bacterial infection by injuring the defense mechanisms that ordinarily maintain pulmonary sterility. Aspirated microorganisms in the lungs are either removed via the mucociliary transport mechanism or destroyed *in situ* by alveolar macrophages.[187] The mode of microbial clearance depends on the site of deposition which in turn is affected by the particle size of the inhaled microorganism. In one method of clearance microorganisms are trapped in the mucous covering of the bronchial epithelium and transported from the lungs by ciliary activity. Expectoration is another mode. Microorganisms that reach the distal regions of the lung are phagocytised by alveolar macrophages.[127, 129, 130, 178] There is considerable evidence that nitrogen dioxide, like other air pollutants,[108, 245, 428, 450, 588] damages these bronchopulmonary defenses, thereby allowing bacterial proliferation and invasion.

Mortality and Survival

Short-Term Exposures. Ehrlich and his coworkers demonstrated in a series of studies that acute exposure to low concentrations of nitrogen dioxide reduces the resistance of mice,[126, 127, 129, 428] hamsters,[127] and squirrel monkeys[143, 144, 221, 222] to bacterial pneumonia. This increased susceptibility is evidenced by increased mortality rate, reduced lifespan, and reduced ability to clear viable bacteria from the lungs. In their experiments Ehrlich and his colleagues exposed animals to atmospheres containing measured quantities of nitrogen dioxide before or after respiratory challenge with aerosols of virulent Klebsiella *pneumoniae*.

In studies of acute exposures, mice were subjected for 2 hr to nitrogen dioxide concentrations of 2.8 to 47.0 mg/m^3 (1.5 to 25 ppm) and then challenged with an aerosol of K. pneumoniae within 1, 6, or 27 hr after the nitrogen dioxide exposure.[127,428] The minimal nitrogen dioxide concentration required to produce a statistically significant ($p < 0.05$) rise in mortality after a 2-hr exposure was 6.6 mg/m^3 (3.5 ppm) when the infectious challenge occurred within 1 hr after that exposure. When the infectious challenge was delayed, a statistically significant effect was noted after 6 hr, but not after 27 hr after exposures to nitrogen dioxide concentrations of 9.4 mg/m^3 (5 ppm) and above. Exposure to 47.0 mg/m^3 (25 ppm) 6 or 14 days before the challenge with K. pneumoniae did not increase mortality.[126] When mice were infected with K. pneumoniae then later exposed to 47.0 mg/m^3 (25 ppm) for 2 hr within 1, 6, 27, 48, or 72 hr after the infectious challenge, the mortality increase was statistically significant. This effect was not observed at 4.7 mg/m^3 (2.5 ppm).[428]

A similar increase in mortality was observed in hamsters exposed to nitrogen dioxide at 65.8 mg/m^3 (35 ppm) or more for 2 hr and then challenged within less than 1 hr with K. pneumoniae.[127]

Henry et al.[221] exposed three squirrel monkeys to nitrogen dioxide at 18.8-94.0 mg/m^3 (10-50 ppm) for 2 hr. He then challenged them with K. pneumoniae introduced intratracheally. Neither the infectious challenge nor the 2-hr exposure to 94.0 mg/m^3 (50 ppm) alone was fatal; whereas exposure to the same nitrogen dioxide concentration followed by challenge with K. pneumoniae was fatal for all three monkeys. Increased mortality rates were not observed in squirrel monkeys exposed to the lower concentrations between 9.4 and 65.8 mg/m^3 (5-35 ppm). In all three species of

animals (mice, hamsters, and squirrel monkeys), the increased mortality rates were consistently paralleled by significant decreases in mean survival times.

In Figure 10-2 the acute exposure data for the three species of laboratory animals are summarized. The data show the actual mortalities of animals in each species that were not exposed to nitrogen dioxide but were challenged with K. pneumoniae, of those that were exposed to low nitrogen dioxide concentrations and K. pneumoniae but did not incur much increase in mortality rates, and of those that were exposed to high nitrogen dioxide concentrations and K. pneumoniae that reacted synergistically to cause high mortality rates.

Mortality rates among the control animals reflect the natural resistance of each species to the infectious agent (41% for mice, 11% for hamsters, and no mortality for monkeys). Moreover, the estimated dose of inhaled K. pneumoniae for monkeys and hamsters was approximately 10^5 microorganisms, compared with 10^3 microorganisms for mice.[130] Thus, the susceptibility of a species to respiratory infection was partially responsible for the increased mortality rate after exposure to nitrogen dioxide.

The rate at which inhaled bacteria were cleared from the lungs of mice and hamsters decreased after exposure to nitrogen dioxide. In control animals that were not exposed to nitrogen dioxide, the bacterial population was markedly reduced during the 6-hr period after the challenge. Thereafter, the population increased, reaching its initial concentration after approximately 8 hr. In mice and hamsters exposed to 9.4 mg/m^3 (5 ppm), the period of initial clearance was reduced to 4.5 and 5 hr, respectively, and the original concentration was reestablished in less

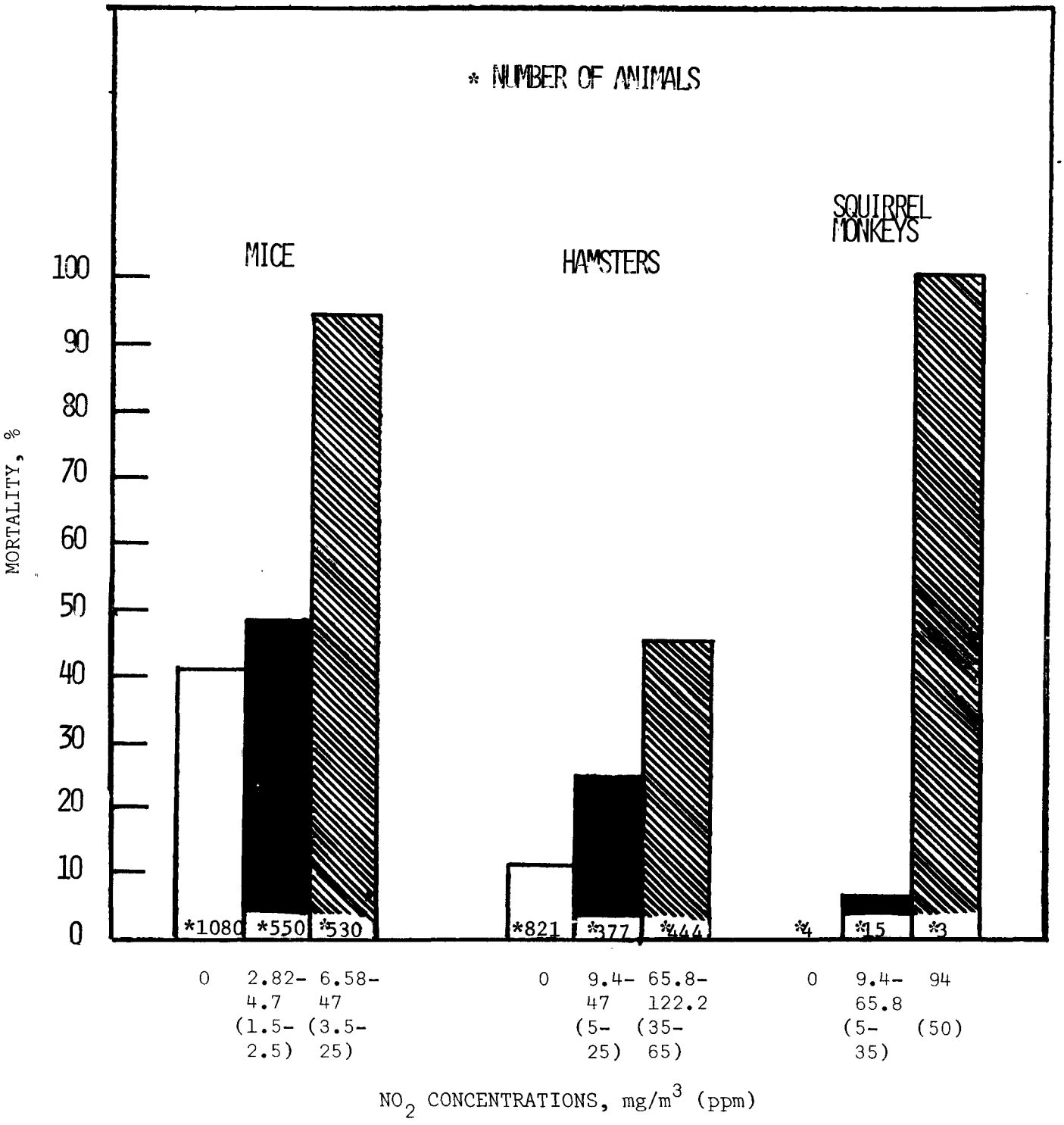


FIGURE 10-2. Mortality in mice, hamsters, and monkeys after challenge with K. pneumoniae (10^3 microorganism for mice, and 10^5 each for hamsters and monkeys) alone ☐ and when administered after 2-hr exposures to low ■ and high ▨ concentrations of nitrogen dioxide. Synergy is evident from increased fatalities after high concentration exposures. From Ehrlich, Henry, and Fenters, 1970.[130]

than 7 hr.[127] Squirrel monkeys exposed to 18.8 mg/m^3 (10 ppm) for 2 hr and then challenged with K. pneumoniae had bacteria present in their lungs 19-51 days after challenge.[221]

These findings were corroborated by Goldstein et al.[178] who studied the effect of nitrogen dioxide at concentrations from 3.6 to 28.0 mg/m^3 (1.9 to 14.8 ppm) on the rate of bacterial removal and bactericidal activity of mouse lungs. Mice were infected with an aerosol of Staphylococcus aureus labeled with radioactive phosphorus and then exposed to nitrogen dioxide for 4 hr. The removal of the bacteria from the lungs was unaffected by exposure to nitrogen dioxide. However, pulmonary bactericidal activity decreased progressively on exposure to nitrogen dioxide concentrations above 13.2 mg/m^3 (7.0 ppm). A similar effect was observed in mice exposed to 4.3 or 12.4 mg/m^3 (2.3 or 6.6 ppm) for 17 hr before challenge with the infectious agent, but the effect was slight at the lowest concentration. These observations suggest that prolonged exposures to nitrogen dioxide cause bactericidal dysfunction even at low concentrations.

Autopsies of mice that died after the bacterial challenge revealed a high incidence of purulent exudate in the pleural cavities. Lungs of mice exposed to nitrogen dioxide concentrations of 6.6 mg/m^3 (3.5 ppm) or higher incurred various degrees of congestion and dilation of veins and capillaries. Pathologic changes were not observed in lungs of mice exposed to lower concentrations of nitrogen dioxide.[127]

The lungs of squirrel monkeys that were exposed to nitrogen dioxide and then challenged with K. pneumoniae developed massive infection. The infectious microorganisms were recovered from kidney, heart, liver, adrenals, and spleen, as well as lung. Exposure to increasing

concentrations of nitrogen dioxide caused progressively severe histopathology of the alveoli. The primary defects were various degrees of alveolar expansion and a high incidence of septal breaks in the alveoli. The combination of exposures to nitrogen dioxide and infectious challenge appeared to result in superimposed effects, rather than an enhancement of the individual effect of either exposure.

Chronic Exposures

Bacterial pneumonia: In several studies mice or squirrel monkeys were exposed to nitrogen dioxide for extended periods and, at various intervals during the exposure, challenged with K. pneumoniae aerosols. Ehrlich[127] exposed Swiss albino mice for 30 days to nitrogen dioxide at 0.94 mg/m^3 (0.5 ppm) continuously, 24 hr/day for 7 days/week, and intermittently, 6 hr/day for 5 days/week. Both exposure conditions resulted in increased mortality rates, compared with controls that were not exposed to nitrogen dioxide. Intermittent exposure appeared to cause greater effects than continuous exposure.

Ehrlich and Henry[129] exposed groups of mice to nitrogen dioxide at 0.94 mg/m^3 (0.5 ppm) for 6, 18, or 24 hr/day for 7 days/week for up to 12 months. Challenge with an aerosol of K. pneumoniae occurred after 1, 3, 6, 9, or 12 months of exposure. Statistically significant increases in mortality were observed after continuous exposure to 0.94 mg/m^3 (0.5 ppm) for 3 months (Figure 10-3), and after 6- and 18-hr/day exposure for 6 months. After 12 months of exposure, increases in mortality were evident only when the nitrogen dioxide exposure was continuous.

The rate at which viable bacteria were cleared from lungs of mice was also affected by the long-term exposure to nitrogen dioxide at

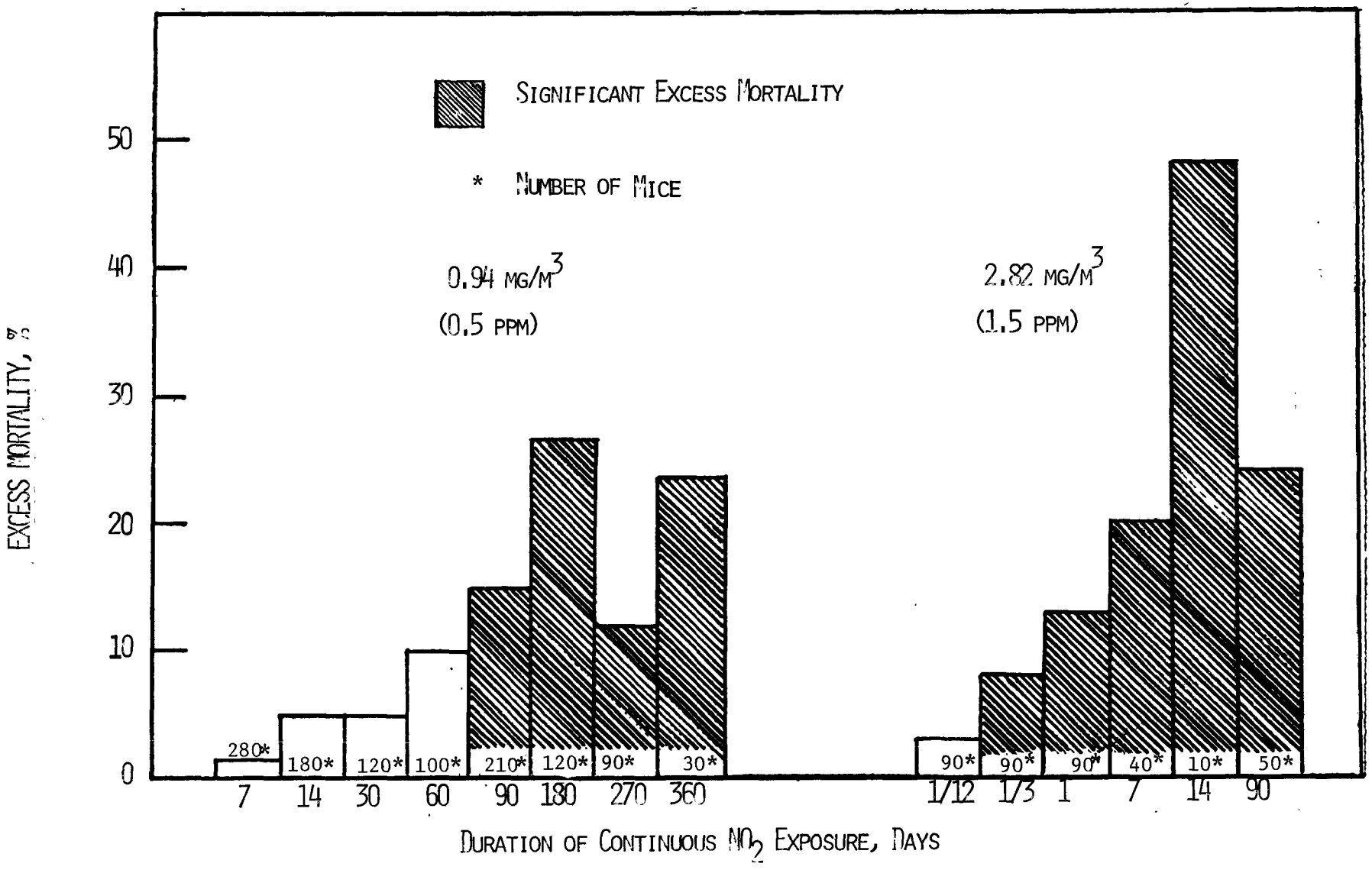


FIGURE 10-3. EXCESS MORTALITIES IN SWISS ALBINO MICE AFTER CHRONIC EXPOSURES TO 0.94 MG/M^3 (0.5 PPM) AND 2.82 MG/M^3 (1.5 PPM) NITROGEN DIOXIDE AND CHALLENGE WITH K. PNEUMONIAE. FROM EHRLICH, HENRY, AND FENTERS, 1970.[130]

0.94 mg/m^3 (0.5 ppm). When mice were exposed to nitrogen dioxide for 6 or 18 hr/day, during 9 and 12 months, capacity to clear bacteria from lungs decreased; mice that were exposed for 24 hr/day incurred reduced capacity after 6 months of exposure.

No decrease in pulmonary clearance rate in germfree and conventional mice was observed by Buckley and Loosli after exposing the mice 6 weeks to nitrogen dioxide at 71 mg/m^3 (38 ppm), then to a respiratory challenge with S. aureus aerosols.[65] Although the rates of bacterial clearance differed between the germfree and conventional mice, neither group was affected by exposure to nitrogen dioxide. The investigators interpreted their data as showing that nitrogen dioxide did not influence bacterial clearance rates. These studies may be criticized on two counts. First, only three animals were studied for each exposure period--too few for statistical analysis. Second, S. aureus is not pathogenic for mice, and 99% of the microorganisms are removed from the lungs within 24 hr. Hence, significant differences in clearance that might have occurred during the first 24 hr with other microorganisms would not have been caused by S. aureus.

Henry et al.[222] exposed male squirrel monkeys continuously to nitrogen dioxide and then challenged them with aerosols of K. pneumoniae. One of the four monkeys that were exposed to 18.8 mg/m^3 (10 ppm) for 1 month died, and in two others the infectious agent was present in the lungs at autopsy. Two of seven monkeys that were exposed to 9.4 mg/m^3 (5 ppm) for 2 months died, and in five the infectious agent was present in the lungs at autopsy.

Influenza: Squirrel monkeys were infected with mouse-adapted influenza A/PR/8 virus 24 hr before exposure to nitrogen dioxide at 18.8 or 9.4 mg/m^3 (10 and 5 ppm). The six monkeys that were exposed continuously to 18.8 mg/m^3 (10 ppm) died within 3 days, whereas only one of three that were exposed to 9.4 mg/m^3 (5 ppm) succumbed to the disease. There were no deaths in a control group of monkeys that were supplied with filtered air and then challenged with influenza virus.[222]

Mouse-adapted influenza A/PR/8 virus was instilled intratracheally into monkeys that were exposed for approximately 5 months to nitrogen dioxide at 9.4 mg/m^3 (5 ppm).[128,143] The virus was injected 24 hr before the continuous exposure began, and again, 37 and 77 days after. The production of serum neutralizing (SN) antibodies appeared to be influenced by the nitrogen dioxide exposure. After a 3-month exposure, two of the three monkeys that were exposed to filtered air had SN titers of 1:256 or higher, whereas the titer in three of the four monkeys that were exposed to the nitrogen dioxide did not exceed 1:21. However, after 5 months of exposure, when the study ended, the SN antibody contents of the exposed monkeys had returned to the levels of the controls.

In another group of experiments, monkeys were exposed to nitrogen dioxide at 1.9 mg/m^3 (1 ppm) for 16 months. They were challenged by intratracheal instillations of a monkey-adapted influenza A/PR/8 virus 24 hr before the exposure began, and again 41, 83, and 146 days after.[128,144] An additional challenge was introduced on the 266th day of a 20-min exposure to influenzal virus aerosol. Monkeys exposed to nitrogen dioxide produced SN antibodies within 21 days after the first virus infection, whereas only one of the three control monkeys, who were given the same challenge but

exposed to filtered air, showed a comparable response. At 41 days after the first challenge, three of five monkeys exposed to nitrogen dioxide had SN titers of 1:128 or greater, reflecting a sixfold increase over the titer in the one control monkey that showed any response. After 12 months, monkeys exposed to nitrogen dioxide showed consistently higher SN antibody titers than did those that were exposed to air (Figure 10-4). The pathologic changes in the lungs of monkeys exposed to nitrogen dioxide and challenged with the virus consisted of slight emphysema and thickened bronchial and bronchiolar epithelium. These changes were not observed in monkeys exposed to air and challenged with the virus.

The effects of low concentrations of nitrogen dioxide on the immunologic response of squirrel monkeys appeared to be partially related to the ability of the virus to multiply in the lung tissue of the host and to the native resistance of the host to the infectious agent. With the mouse-adapted strain of influenza virus, the ability to form SN antibodies apparently decreased in monkeys exposed to nitrogen dioxide at 9.4 mg/m^3 (5 ppm). Conversely, after challenge with a monkey-adapted strain of the same virus, SN antibodies appeared sooner and in higher amounts in monkeys that had been exposed to nitrogen dioxide at 1.9 mg/m^3 (1 ppm). In this instance, exposure to nitrogen dioxide appeared to enhance the establishment and multiplication of the monkey-adapted influenza virus.

Ito *et al.*[257] exposed mice to nitrogen dioxide concentrations of 18.8 mg/m^3 (10 ppm), for 2 hr/day over 1, 3, or 5 days and then challenged them with mouse-adapted influenza A/PR/8 virus. Other mice were challenged with the same virus after a continuous exposure to nitrogen dioxide concentrations of 0.94 mg/m^3 (0.5 ppm) or 1.9 mg/m^3 (1.0 ppm)

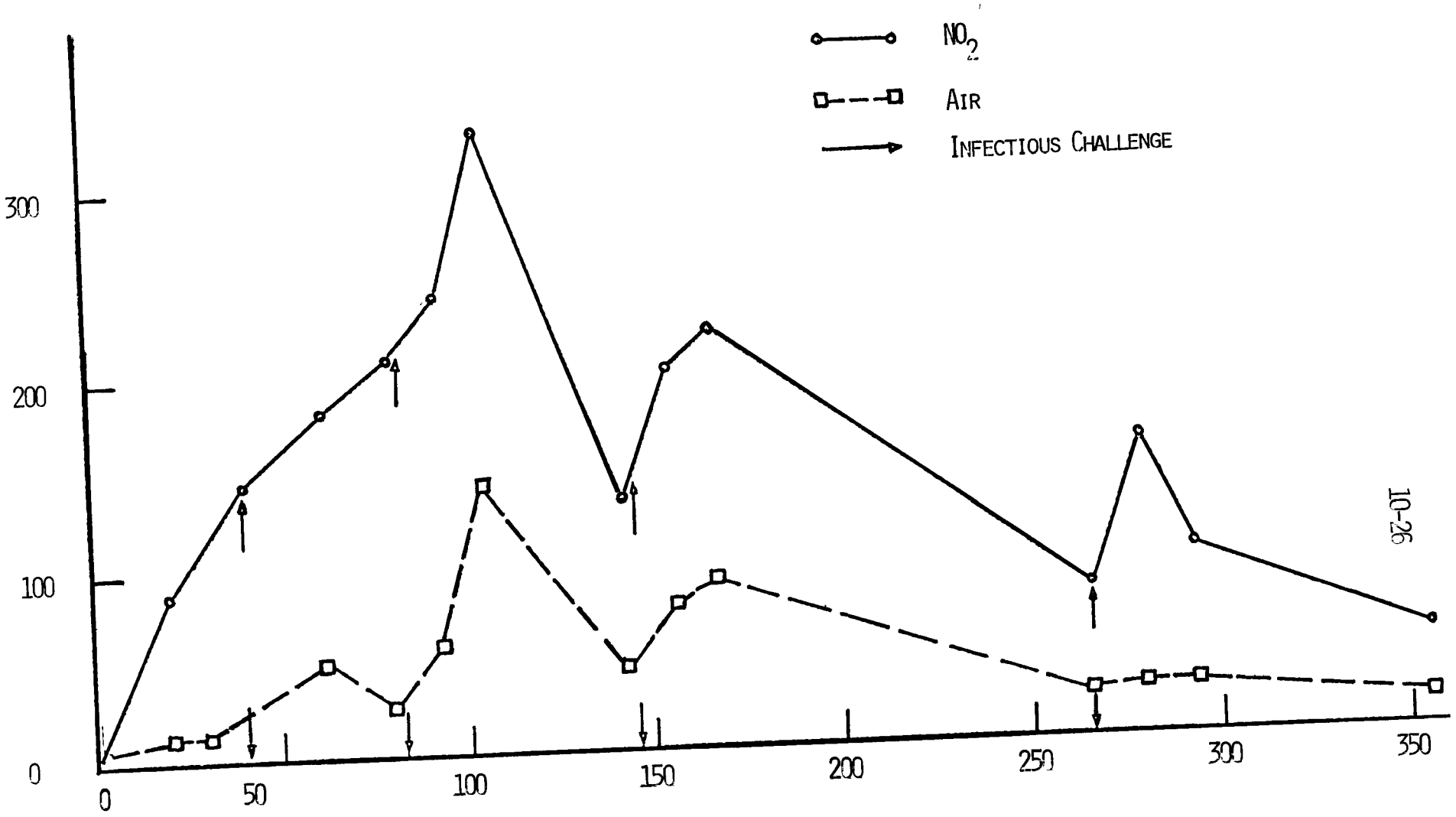


FIGURE 10-4. MEAN RECIPROCAL SN ANTIBODY TITERS OF SQUIRREL MONKEYS EXPOSED TO 1.880 MG/M^3 (1.0 PPM) OF NITROGEN DIOXIDE AND CHALLENGED WITH MONKEY ADAPTED A/PR/8 INFLUENZA VIRUS. FROM EHRLICH AND FENTERS, 1973.[128]

for 39 days. Exposure to 18.8 mg/m^3 (10 ppm) for 2 hr/day for 5 days increased the susceptibility of mice to influenza virus resulting in increased mortality. Interstitial pneumonia was more extensive in mice challenged with the virus after both acute and chronic exposures than in the controls exposed to air. Moreover, adenomatous proliferations of bronchial and bronchiolar epithelium were marked in the mice challenged with influenza virus after the continuous exposure to the low nitrogen dioxide concentrations.

Buckley and Loosli[65] exposed germfree and conventional mice to air containing nitrogen dioxide concentrations of 71 mg/m^3 (38 ppm) for 6 weeks. After this exposure, mice were challenged with aerosols of mouse-adapted influenza A/PR/8 virus. Mortality rates and average day of death after the challenge with the virus indicated that exposure to nitrogen dioxide resulted in a marked increase in resistance to influenza infection. The mice that inhaled nitrogen dioxide had higher survival rates after inoculation with an LD_{50} virus and increased survival days after an LD_{100} virus inoculation. These observations differ from those reported by other investigators.

Immunologic Effects

Matsumura[334] recently reported results of studies on guinea pigs that were sensitized to egg albumen. A 30-min exposure to nitrogen dioxide at 132 mg/m^3 (70 ppm) increased the susceptibility of those guinea pigs to systemic anaphylaxis after inhalation of egg albumen aerosols. Exposure to nitrogen dioxide at 75 mg/m^3 (40 ppm) caused increased dyspneia.[335] Circulating antibodies reactive with pulmonary tissue have also been found in guinea pigs exposed to 9.4-28.0 mg/m^3 (5-15 ppm) for up to 12 months.

The effects of extended exposures to low concentrations of nitrogen dioxide on the immunologic response were studied by vaccinating mice with 279 CCA units of chick embryo A_2 /Taiwan/1/64 influenza virus vaccine.[490] Before vaccination, one group of mice was exposed continuously for 3 months to nitrogen dioxide at 3.8 mg/m^3 (2 ppm); another group was exposed to 0.94 mg/m^3 (0.5 ppm) with daily 1-hr-long peaks of 3.8 mg/m^3 (2 ppm) for 5 days per week. After the 3-month exposure, the mice were vaccinated with the influenza vaccine and held in either of the exposure conditions for up to 7 months. At various intervals, the hemagglutination inhibition (HI) and SN antibody titers and the concentrations of immunoglobulins were measured. Two weeks after vaccination, the SN antibody decreased and seroconversion rates were markedly lower especially among mice exposed to 0.94 mg/m^3 (0.5 ppm) with the daily 1-hr peaks of 3.8 mg/m^3 (2 ppm). After 4 weeks of exposure, the SN titers and seroconversion rates did not differ significantly from those in control mice exposed to filtered air.

Serum immunoglobulin concentrations in nonvaccinated mice were altered during the 3 months of exposure to nitrogen dioxide. In general, IgA decreased while IgM, IgG_1 and IgG_2 increased. During the 28 weeks after vaccination, exposure to nitrogen dioxide did not further influence IgA concentration. However, serum IgM, IgG_1, and IgG_2 were higher in mice exposed to nitrogen dioxide than in those maintained in filtered air. More specifically, mice consistently showing higher immunoglobulin concentrations were those exposed continuously to 0.94 mg/m^3 (0.5 ppm) with daily 1-hr peaks of 3.8 mg/m^3 (2 ppm) before and after vaccination. Moreover, mice exposed to filtered air before vaccination and to nitrogen dioxide at 3.8 mg/m^3 (2 ppm) after vaccination and those exposed to

nitrogen dioxide at 3.8 mg/m^3 (2 ppm) before vaccination and to filtered air after vaccination showed similar increases in serum immunoglobulins.[490]

These results suggest that fluctuation in atmospheric nitrogen dioxide has more influence on the immune response than constant, although higher, concentrations of nitrogen dioxide. Continuous exposure of mice to 3.8 mg/m^3 (2 ppm) for approximately 10 months did not appear to influence the formation of antibodies or the immunoglobulin concentrations. Conversely, exposure to 0.94 mg/m^3 (0.5 ppm), with daily 1-hr peaks of 3.8 mg/m^3 (2 ppm), appeared to decrease the ability to form SN antibody and significantly altered the concentrations of serum IgM, IgG_1, and IgG_2.

Mucociliary Clearance Mechanisms

When a highly irritant gas reaches the mucous membranes of the trachea or bronchi, it can increase mucus secretions, paralyze the cilia, and eventually result in mucosal thickening, thereby reducing the effectiveness of the physical airway-clearing mechanisms. In severe cases, it may cause desquamation of the epithelium, exposing the deeper and more sensitive cell layers to infectious agents.

The majority of the few existing reports describing the effect of nitrogen dioxide in the mucociliary activity are based on observations of *in vitro* rabbit tracheal preparations. Cralley[94] reported that a nitrogen oxide gas mixture (nitric oxide, nitrogen dioxide, nitric acid (HNO_3), etc.) at approximately 188 mg/m^3 (100 ppm) caused irreversible cessation of ciliary beating after a 5-min exposure. Dalhamn and Sjoholm[96] reported that nitrogen dioxide between concentrations of 188 and 280 mg/m^3 (100 and 150 ppm) caused cessation of ciliary movement. These investigators did not state whether the effect was permanent or temporary. Kensler and Battista[279]

reported that an 18-sec exposure to nitrogen dioxide at 790 mg/m^3 (420 ppm) produced a 50% inhibition in the rate particles were transported by the mucociliary apparatus.

More recently, Giordano and Morrow[175] studied the mucociliary clearance in anesthesized rats after a 6-week exposure to nitrogen dioxide at 11.3 mg/m^3 (6 ppm). The two transport characteristics measured were "first edge time" and a "20% transport time." Exposure to nitrogen dioxide caused a significant increase in both, indicating a significant inhibition of the mucociliary clearance mechanism. The decrease in ciliary activity was not accompanied by any observable abnormality of the airways. Pathologic changes of edema and vascular congestion did occur, but these were confined to the alveolar regions. The authors suggest that one point of interaction between the nitrogen dioxide and the mucociliary apparatus might be the energy source of the cilia. Buckley and Balchum[64] have reported that adenosine triphosphate, ATP ($C_{10}H_{12}N_5O_3H_4P_3O_9$), is the source of energy for ciliary motion and that nitrogen dioxide can affect lung homogenate enzymes linked to ATP formation. Other possible reactions affect the mucus, i.e., through modification of the cross-linking of polymer chains which affects the viscosity of the mucus.[172]

The *in vivo* clearance of inhaled bacteria from the lungs can be hindered by the action of a number of different agents[188] presumably by inhibition of ciliary activity. However, removal of the inhaled bacteria by the ciliary mechanisms and mucus flow is probably not a major part of lung antibacterial defense. Other mechanisms must be involved, inasmuch as the loss of viability of inhaled bacteria in the lungs occurs much faster than their removal.[179, 276]

Phagocytic Activity of Alveolar Macrophages

When inhaled bacteria first enter the lungs, they are free in the air spaces; later, they are almost entirely contained within phagocytic alveolar cells.[179, 188, 276] The removal of inhaled bacteria by these cells is consequently of utmost importance as a defense mechanism against respiratory infections.

The action of nitrogen dioxide on the alveolar macrophage system was reported by several investigators. Gardner *et al.*[168] stated that exposure to nitrogen dioxide reduced the number of alveolar macrophages obtained by pulmonary lavage. Moreover, the phagocytic activity of the surviving fraction of the macrophages was also reduced. Exposure of rabbits to nitrogen dioxide at 15 mg/m^3 (8 ppm) resulted in a significant increase in the percentage of intra-alveolar heterophiles obtainable by pulmonary lavage. A 3-hr exposure to 18.8 mg/m^3 (10 ppm) resulted in a marked decrease in phagocytic activity, as reflected by an approximately 50% reduction in phagocytized *Streptococcus pyogenes* cells. In addition, although 83% of the macrophages from control rabbits contained at least one bacterium, only 66% of the macrophages from exposed rabbits showed phagocytic activity.

Valand *et al.*[575] reported that alveolar macrophages harvested from rabbits that received intratracheal injections of para-influenza-3 virus were resistant to later *in vitro* challenge with rabbit pox virus. However, when the rabbits were exposed to nitrogen dioxide at 47 mg/m^3 (25 ppm) for 3 hr immediately after challenge with the para-influenza-3 virus, during the challenge, or 3, 5, 12, or 24 hr before, this resistance did not develop. This refractory state lasted at least 96 hr; the alveolar

macrophages isolated from rabbits exposed to nitrogen dioxide were unable to produce interferon when inoculated *in vitro* with the parainfluenza-3 virus. Exposure to nitrogen dioxide also appeared to increase the adsorption rate of the virus in the lungs of rabbits, but did not enhance the infectivity of the virus.

The inhibition of alveolar macrophage function was further demonstrated by Acton and Myrvik. They exposed rabbits to nitrogen dioxide concentrations of 28.2, 47, or 94 mg/m^3 (15, 25, or 50 ppm) for 3 hr[4] and inoculated them with killed BCG vaccine. Macrophages lavaged from the lungs of these rabbits had a markedly impaired ability to phagocytize the vaccine, compared with normal controls. This effect was not observed in cells from animals exposed to nitrogen dioxide at 9.4 mg/m^3 (5 ppm).

Vassallo *et al.*[578] measured the *in vitro* effect of nitrogen dioxide on the rates of entry and killing of *Staphylococcus epidermidis* and *Pseudomonas aeruginosa* in rabbit alveolar macrophages adherent to a glass surface. Exposure to nitrogen dioxide diminished the bacterial entry and intracellular killing of the alveolar macrophages.

Aranyi and Port[22] observed a significant decrease in the phagocytic function of alveolar macrophages obtained from mice exposed to low concentrations of nitrogen dioxide. The impairment of phagocytosis of heat-killed *Escherichia coli* was seen in mice exposed continuously to nitrogen dioxide at 0.94 mg/m^3 (0.5 ppm) with daily 1-hr peaks of 3.8 mg/m^3 (2 ppm) 5 days per week for 3.5 and 7 months. However, decreases in this phagocytic function did not occur in mice exposed continuously to 3.8 mg/m^3 (2 ppm) for the same periods. Moreover, macrophages isolated from mice vaccinated with influenza virus vaccine after 5 months of

exposure to either of the nitrogen dioxide concentrations showed distinct alteration in surface structure when examined with a scanning electron microscope.

Combined Exposures

Coffin and Blommer[89] studied effects on resistance to infection caused by exposure to nitrogen dioxide in combination with other pollutants. They exposed mice to light-irradiated automobile exhaust for 4 hr and then infected them with airborne streptococci. An increased mortality rate was observed in mice exposed to exhaust containing concentrations of carbon monoxide (CO) at 47 mg/m^3 (25 ppm) and oxidants at 0.28 mg/m^3 (0.15 ppm). The nitrogen dioxide concentration in the exhaust gas was 0.6 mg/m^3 (0.3 ppm), approximately 10% reportedly required to increase susceptibility to respiratory infection.[127] Because oxidant concentrations were within the effective range for ozone [threshold values of 1.88 mg/m^3 (0.1 ppm) have been reported[88]], the reduction in resistance to bacterial infection was attributed to the presence of oxidants in the automobile exhaust.

Hamsters were exposed to combinations of nitrogen dioxide, tobacco smoke, and *K. pneumoniae*.[223] A 2-hr exposure to nitrogen dioxide at 28.2 mg/m^3 (15 ppm) followed by a 1-hr exposure to 3% (v/v) tobacco smoke decreased their resistance to bacterial pneumonia, as evidenced by increased mortality and reduced survival time. Furthermore, the combined exposures reduced the rate at which viable bacteria were cleared from their lungs to a greater extent than individual pollutant exposures.

Scanning electron microscopic examination of lung tissues indicated that *K. pneumoniae* had no demonstrable effect and exposure to nitrogen dioxide had only a slight effect on the surface structure of the respiratory

airways.[220] Exposure to tobacco smoke and bacteria produced reversible alterations in the surface structure of the bronchi. The changes were present 2, but not 7, days after the exposure terminated. Exposure to nitrogen dioxide and tobacco smoke followed by inhalation of K. pneumoniae produced marked loss of cilia and alteration of surface morphology of nonciliated cells 2 days after exposure. These changes were progressive, rather than reversible, and the surface structure of the affected bronchi showed an even greater disruption 7 days after the exposures.

A similar loss of cilia from the terminal bronchiolar surface and leveling of the terminal bronchiolar epithelium were observed in rats after a 24-hr exposure to nitrogen dioxide at 28.2 mg/m^3 (15 ppm).[408] When examined with a scanning electron microscope, the terminal bronchiole presented an even surface and most areas were free of cilia. Moreover, tiny microvilli covered most of the nonciliated areas, and the population of brush cells increased markedly. These changes in terminal bronchioles were more pronounced after 2 and 7 days of exposure to this nitrogen dioxide concentration.

Goldstein et al.[181] studied the pulmonary defense mechanisms as manifested by bactericidal dysfunction in mice exposed to various combinations of concentrations of nitrogen dioxide and ozone, and infected with aerosols of S. aureus. Their results suggested that the pollutants did not act synergistically, since the reduction in bactericidal function was equivalent to that expected if each pollutant acted independently. These experiments also indicated that measurement of oxidant-induced disturbance in airway resistance may be a more sensitive indicator of pollutant damage than measurement of bactericidal function.

TOXICOLOGIC EFFECTS OF OTHER OXIDES OF NITROGEN IN LABORATORY ANIMALS

Nitrous Oxide

Nitrous oxide is an inert gas with anesthetic characteristics. Its balanced system of biologic production and biologic and stratospheric degradation is independent of the cycles of other oxides of nitrogen.[441] The normal ambient concentration of nitrous oxide is 0.9 mg/m^3 (0.50 ppm),[91,199] which is considerably below the threshold concentration for a biologic effect. Consequently, the potential toxicity of this gas as a pollutant is nil.

Nitric Oxide

In addition to its biologic formation, large quantities of nitric oxide form as a result of industrial processes, especially those which burn petroleum. Its ambient concentrations are almost always less than 0.6 mg/m^3 (0.5 ppm).[69] Although this pollutant is a principal component of photochemical smog and accounts for a major portion of the oxides of nitrogen, its biologic toxicity is much less than that of nitrogen dioxide. After rats have been exposed for 2.5 to 24 months to automotive exhaust gases containing carbon monoxide at 67.4 mg/m^3 (57.8 ppm); nitric oxide at 27.8 mg/m^3 (22.6 ppm); and nitrogen dioxide at 1.7 mg/m^3 (0.92 ppm), they incur decreases in body weight, diminution of the sound-avoiding reflexes, and an increase in the number of spontaneous tumors.[522] Similarly, although nitric oxide has undoubtedly been present in cases of human poisoning due to oxides of nitrogen, poisoning from nitric oxide alone has never been reported.[518]

The rapid conversion of nitric oxide to nitrogen dioxide, a much more toxic gas, makes nitric oxide very difficult to investigate. The few reported studies suggest that exposure to high concentrations of nitric oxide will result in tissue injury caused by methemoglobin formation.[184,189,540]

Experiments in dogs have demonstrated that these animals are capable of breathing air containing nitric oxide at 1,230 mg/m^3 (1,000 ppm) for 136 min before cyanosis appears.[189] The arterial oxygen pressure in these animals was 309 mm of mercury and the methemoglobin concentration was 5%.

Dogs that were exposed to nitric oxide at 6,200 mg/m^3 (5,000 ppm) for 24 min or more succumbed to pulmonary edema after exposure. Shorter exposures resulted in respiratory distress but not deaths.

Physiologic abnormalities in addition to the decrease in oxygenation were respiratory acidosis, diminished lung compliance, increased airway resistance, diminished cardiac output, and a marked reduction in heart rate.[189] The principal biochemical abnormality was the time-related formation of methemoglobin.[189,540] The major findings from autopsies of both the dogs that survived and the ones that died were pulmonary edema, hyperinflation, hemorrhage, desquamation of mucosa, and bronchopneumonia.[483] With the exception of congestion, the liver, spleen, and kidneys appeared normal. The brain manifested changes that were consistent with hypoxia. The similarity of these pathologic findings to those attributed to nitrogen dioxide, and the known contamination of nitric oxide with nitrogen dioxide support the probability that some, if not all, pathologic abnormalities were caused by nitrogen dioxide, rather than nitric oxide.

Experiments with mice have yielded similar responses. For example, lethality was not observed after 8-hr exposures to nitric oxide at 380 mg/m^3 (310 ppm).[416,417]

Dinitrogen Trioxide and Dinitrogen Pentoxide

The chemical instability of these oxides of nitrogen reduces the likelihood of their causing significant hazard in other than acute circumstances.

Dinitrogen trioxide is extremely unstable and decomposes almost immediately to nitric oxide and nitrogen dioxide.[184]

Dinitrogen pentoxide is produced from the interaction of ozone and nitrogen dioxide.[109, 441, 518] This gas is an anhydride of nitric acid, and in moist air it rapidly converts to nitric acid. Although dinitrogen pentoxide is slightly toxic, the following arguments suggest that this toxicity is not of practical importance. At ozone and nitrogen dioxide concentrations of 0.2 mg/m^3 (0.1 ppm) and a nitric oxide concentration of 0.01 mg/m^3 (0.01 ppm), the upper-limit equilibrium concentration of dinitrogen pentoxide is 0.001 mg/m^3 (0.0002 ppm).[518] The formation of dinitrogen pentoxide requires an equivalent reduction in the concentration of ozone,[109, 518] which is probably the more toxic gas.[517] Although results of different tests vary,[109, 517] it is certain that dinitrogen pentoxide can be only slightly more toxic than ozone.[109, 181] Consequently, of all the oxides of nitrogen, only nitrogen dioxide can be classified as a significant hazard to health.

SUMMARY OF EXPERIMENTAL ANIMAL STUDIES

Mice exposed for less than 24 hr to nitrogen dioxide concentrations of 3.8 mg/m^3 (2 ppm) or greater develop defects in pulmonary microbial defense mechanisms. Inhibition in intrapulmonary killing of inhaled *Staphylococcus aureus* occurs in mice exposed to 3.8 mg/m^3 (2.0 ppm) of nitrogen dioxide for 17 hr and pneumonia and death occurs in mice infected with *Klebsiella pneumoniae* following 2-hr exposures to 6.6 mg/m^3 (3.5 ppm). In other rodents, physiologic and pathologic abnormalities

appear at concentrations above 9.4 mg/m^3 (5 ppm). Mortality occurs at nitrogen dioxide concentrations of 75 mg/m^3 (40 ppm). Even the lowest concentrations at which abnormalities are occasionally observed are higher than concentrations found in the ambient atmosphere.

Prolonged exposure to nitrogen dioxide has affected normal animals only at concentrations of 0.94 mg/m^3 (0.5 ppm) and above. No deaths resulted in any animals exposed for a year or more to 0.94 mg/m^3 (0.5 ppm) of nitrogen dioxide. Such animals as dogs and guinea pigs survive exposures of a year or more to much higher concentrations of nitrogen dioxide.

Pathologic abnormalities (ciliary loss, alveolar cell disruption, and obstruction of respiratory bronchioles) occur in mice and rats after continuous exposure to nitrogen dioxide concentrations of 0.94 mg/m^3 (0.5 ppm). Exposure to higher nitrogen dioxide concentrations causes more severe cellular and structural damage, which in the rat and rabbit resembles emphysema.

Physiologic alterations (tachypnea, increases in airway resistance, decreases in tidal volume and in static compliance) occur in rodents and in nonhuman primates after exposures of 2 months or longer to nitrogen dioxide at concentrations of 9.4 mg/m^3 (5.0 ppm) or higher. More serious abnormalities in pulmonary function (decreases in blood oxygenation) occur in rabbits after continuous exposure to 15 mg/m^3 (8.0 ppm). These data are not comprehensive because neither the measurements of small airway function nor the effect of exercise have been reported.

Continuous exposures for 3 months and intermittent daily exposures for 6 months or longer to nitrogen dioxide at concentrations of 0.94 mg/m^3 (0.5 ppm) and higher diminishes murine resistance to pulmonary bacterial

infection. Exposures of 9.4 mg/m^3 (5.0 ppm) have produced similar results in nonhuman primates. These decreases in resistance to infection have caused pneumonia and death. Immunologic deficits, not associated with overt infection, occur during exposures of 16 months to 1.8 mg/m^3 (1.0 ppm). These investigations of the combined effect of nitrogen dioxide and infection have provided the most sensitive indices of pollutant-induced damage.

In a few reported studies animals were exposed to mixtures of pollutants including nitrogen dioxide. Combinations of nitrogen dioxide with carbon monoxide, ozone, or sulfur dioxide (SO_2) have usually resulted in additive or indifferent effects. Synergy has, with one exception of uncertain significance, not been reported, and to our knowledge antagonism has never been reported.

EFFECTS IN HUMANS FROM SHORT-TERM NITROGEN DIOXIDE EXPOSURES

Sensory Effects

Sensory perception, in the form of either odor or dark adaptation, is the most sensitive indicator in humans of the presence of nitrogen dioxide. Henscheler *et al.*[224] and Shalamberidze[476] have studied odor perception in volunteers. Henscheler and his colleagues exposed several groups of two to four volunteers to concentrations of nitrogen dioxide fixed at various levels between 0.23-56.8 mg/m^3 (0.12-30.2 ppm), for either 30 or 120 min. The nitrogen dioxide was prepared by adding sulfuric acid to sodium nitrite to form nitric oxide and then reacting the nitric oxide with oxygen in a 1-m reaction tube. The pollutant was introduced into exposure chambers at high flow rates, and its concentration was measured by the Saltzman method.

Olfactory responses were recorded for groups of two to four volunteers immediately after their entrance into the nitrogen dioxide-containing chambers. After 30 min of exposure, the volunteers left the chamber. They were then reexposed to several inhalations of test concentrations of nitrogen dioxide at 30-sec intervals, and their responses were again recorded. The odor of nitrogen dioxide was perceived by 3 of 9 volunteers at concentrations of 0.23 mg/m^3 (0.12 ppm) and by 8 of 13 subjects at concentrations of 0.41 mg/m^3 (0.22 ppm). (See Table 10-4.) The volunteers perceived the odor for 1-10 min after entry into the chamber at concentrations of 7.52 mg/m^3 (4.0 ppm) or less. The duration of odor perception was unrelated to the concentration of nitrogen dioxide within the chamber. The obliterated olfactory response returned within 1-1.5 min after the subject left the chamber. Some subjects reported a metallic taste and pharyngeal dryness, roughness, and constriction from exposure to 0.23 mg/m^3 (0.12 ppm) or more. These symptoms lessened with repeated exposures and eventually disappeared even at concentrations as high as 37.0 mg/m^3 (19.7 ppm). Exposure to 56.4 mg/m^3 (30 ppm) resulted in overt discomfort-burning sensation in the nose and chest, cough, dyspnea, and sputum production. These symptioms persisted for several hours after termination of the exposure. When the volunteers entered the exposure chamber before the addition of nitrogen dioxide, and the pollutant was added in gradually increasing concentrations up to 47 mg/m^3 (25 ppm), olfactory perception did not occur.

The role of humidity was investigated by exposing the volunteers to 2.26 mg/m^3 (1.2 ppm) at 55% humidity which was then increased to 78%.

TABLE 10-4

Olfactory Perception in Volunteers Exposed to Low Concentrations of Nitrogen Dioxide

NO_2 Exposure		No. Tested	No. Perceiving an Odor	Duration of Exposure, min	Average Duration (and Range) Odor Perception, min
mg/m^3	(ppm)				
0.23	(0.12)	9	3	30	5 (0.5-10)
0.41	(0.22)	13	8	30	3 (1-10)
0.79	(0.42)	8	8	30	2.5 (1-5)
7.52	(4.0)	12	12	30	5.5 (3-10)
19.9	(10.6)	4	4	120	8 (6-13)
37.0	(19.7)	8	8	120	12 (5-24)
56.8	(30.2)	3	3	120	32 (24-40)

This resulted in a sharp increase in odor perception and in irritation of the mucous membranes of the respiratory tract.

In summary, these studies demonstrate that sensitive subjects can detect the odor of nitrogen dioxide at a concentration of 0.23 mg/m^3 (0.12 ppm), but that this perception can be prevented by gradual exposure to the gas. In addition, tests have shown that increases in humidity increase odor perception and respiratory irritation resulting from nitrogen dioxide exposure. Data establishing the threshold concentration of odor perception at 0.23 mg/m^3 (0.12 ppm) have been reported.[476]

Exposure to low concentrations of nitrogen dioxide impairs dark adaptation.[51,476] According to Shalamberidze, the threshold for this sensory defect may be as low as 0.14 mg/m^3 (0.075 ppm), which is about 40% lower than that for odor perception.[476] This result, however, was not reproduced by Bondareva[51] in his studies of the effect of nitrogen oxides on eye sensitivity to darkness. Since Bondareva did not indicate whether he used nitrogen dioxide alone or in combination with nitric oxide, the higher threshold values he found may have resulted from differences in test atmospheres.

Bondareva determined the normal adaptation curve in five volunteers and then exposed the subjects to atmospheres containing nitrogen oxides at 0.15-0.50 mg/m^3 (0.08-0.26 ppm). The nitrogen oxides were measured colorimetrically by the Griess-Ilosvay reaction. These data indicated that exposure to a nitrogen oxide concentration of 0.3 mg/m^3 (0.16 ppm) did not cause changes in dark adaptation. A sharp rise in the adaptation curve did occur at 0.5 mg/m^3 (0.26 ppm). Repeated exposures over 2.5 to 3 months resulted in a slight decrease in the adaptation curve, suggesting some physiologic adjustment to the pollutant.

Pulmonary Function

The effect of exposure to nitrogen dioxide on pulmonary function has been studied by several investigators.[1, 384, 523, 532, 582-585, 613-616] Abe[1] exposed five healthy men (four nonsmokers and one light smoker), between 21 to 40 years of age, to nitrogen dioxide concentrations of 7.5-9.4 mg/m^3 (4.0-5.0 ppm) for 10 min. Measurements of pulmonary compliance, inspiratory flow resistance, maximal midexpiratory and peak flow rates, and vital capacity were made immediately before and 10, 20, and 30 min after exposure. Compliance curves were obtained with intraesophageal balloons and flow transducers. Nitrogen dioxide concentrations were determined by the Saltzman method. The study was repeated but with 10-min exposures to sulfur dioxide at 11.4-14.3 mg/m^3 (4.0-5.0 ppm) and to combinations of nitrogen dioxide at 4.7 mg/m^3 (2.5 ppm) and sulfur dioxide at 7.5 mg/m^3 (2.5 ppm).

The effects of the different pollutant exposures on expiratory flow resistance are shown in Table 10-5. The maximal response for nitrogen dioxide occurred 30 min after the exposure. In contrast, exposure to sulfur dioxide caused an immediate increase in expiratory flow resistance, which rapidly returned to normal within 10 min after the exposure. The effects of sulfur dioxide and nitrogen dioxide combined were neither cumulative nor antagonistic; each gas affected expiratory flow resistance independently, creating a bimodal curve characterized by the early effect of sulfur dioxide and the delayed effect of nitrogen dioxide. Early in the course of exposure to combinations of sulfur dioxide at 7.5 mg/m^3 (2.5 ppm) and nitrogen dioxide at 4.7 mg/m^3 (2.5 ppm), an 18% increase in expiratory flow resistance occurred and was attributed to the effect of sulfur dioxide. Flow resistance returned towards normal in the first 10 min after exposure and then increased again by 45%

TABLE 10-5

Effect of Nitrogen Dioxide and Sulfur Dioxide on Expiratory Flow Resistance

	Expiratory Flow Resistance, %		
	NO_2 7.5-9.4 mg/m^3 (4-5 ppm)	SO_2 11.4-14.3 mg/m^3 (4-5 ppm)	$NO_2 + SO_2$ 4.7+7.5 mg/m^3 (2.5+2.5 ppm)
Control period	100	100	100
Exposure period (10 min)	96	141	118
Recovery period			
10 min	126	90	106
20 min	145	95	132
30 min	177	90	145

at 30 min after exposure, an effect similar to the response to isolated nitrogen dioxide exposure demonstrated earlier. Similar findings were reported for inspiratory flow resistance and pulmonary compliance. Measurements of the 1-sec forced expiratory volume, maximal midexpiratory and peak flow rates, and vital capacity during and after exposure to nitrogen dioxide alone did not reveal changes, suggesting that these tests were insensitive to the experimental conditions.

Abe[1] cites two Japanese studies with similar results.[384, 523] Suzuki and Ishikawa[523] measured pulmonary compliance and inspiratory and expiratory resistance in 10 healthy individuals after a 10-min exposure to nitrogen dioxide concentrations of 1.32-3.76 mg/m^3 (0.7-2.0 ppm). The test values did not change immediately; but, 10 min after exposure, increases of 50% and 15% in inspiratory and expiratory flow resistance and decreases of 10% in compliance were recorded. Prompt increases in airway resistance have been produced by other investigators by exposing healthy individuals for 5 min to nitrogen dioxide concentrations of 11.8 mg/m^3 (6 ppm) to as high as 75.2 mg/m^3 (40 ppm).[384]

Although increased airway resistance resulting from nitrogen dioxide exposure has been found consistently, its threshold concentration is uncertain. Using a plethysmograph on volunteers who had been exposed to nitrogen dioxide for 10-120 min, Yokoyama[613-616] measured increases in airway resistance at 13.2 mg/m^3 (7 ppm) and higher. He also reported wide variations in individual sensitivity. Some volunteers tolerated concentrations up to 30 mg/m^3 (16 ppm). Because atropine effectively blocked the bronchoconstrictive effect of sulfur dioxide, but not nitrogen dioxide, these investigators suggested that the mechanism for the increase in airway resistance was unrelated to vagal stimulation.

Von Nieding and his associates[582, 583, 585] exposed 63 patients with chronic bronchitis to 30 breaths of nitrogen dioxide from concentrations of less than 1.8 mg/m^3 (1 ppm) to 9.4 mg/m^3 (5 ppm). The concentrations were determined by the Saltzman method and airway resistance was measured by body plethysmography before and immediately after each exposure. Significant increases in airway resistance occurred upon exposure to nitrogen dioxide concentrations of 3.0 to 3.8 mg/m^3 (1.6 to 2.0 ppm). (See Fig. 10-5.) After exposure to 9.4 mg/m^3 (5 ppm), normal subjects also exhibited diminished single-breath diffusing capacity[583] and significant increases in the alveolar-arterial pO_2 gradient.[582] These findings suggest that nitrogen dioxide affects respiratory gas exchange.

Von Nieding[582] also observed that the effect of nitrogen dioxide on airway resistance and arterial oxygen pressure was prevented by administration of an antihistamine (Meclostine) but not by atropine or orciprenaline (a sympathomimetic drug). This suggests that nitrogen dioxide causes pulmonary tissue to release histamine, rather than inducing a parasympathetic reflex mechanism similar to that resulting from sulfur dioxide exposure.

Thomas and his associates[532] failed to observe nitrogen dioxide effects at 0.94-6.58 mg/m^3 (0.5-3.0 ppm) on sputum histamine concentrations or on total sputum weight either in five healthy subjects or in four patients with chronic respiratory disease.

Ten individuals who inhaled nitrogen dioxide while exercising were studied by Rokaw *et al.*[447] Six of their subjects were healthy; four had chronic pulmonary disease. Temperature and humidity were constant at 21.11°C, 50% RH. Nitrogen dioxide was generated from cylinders of liquid nitrogen peroxide-nitrogen dioxide and analyzed by the Saltzman method. Airway resistance was

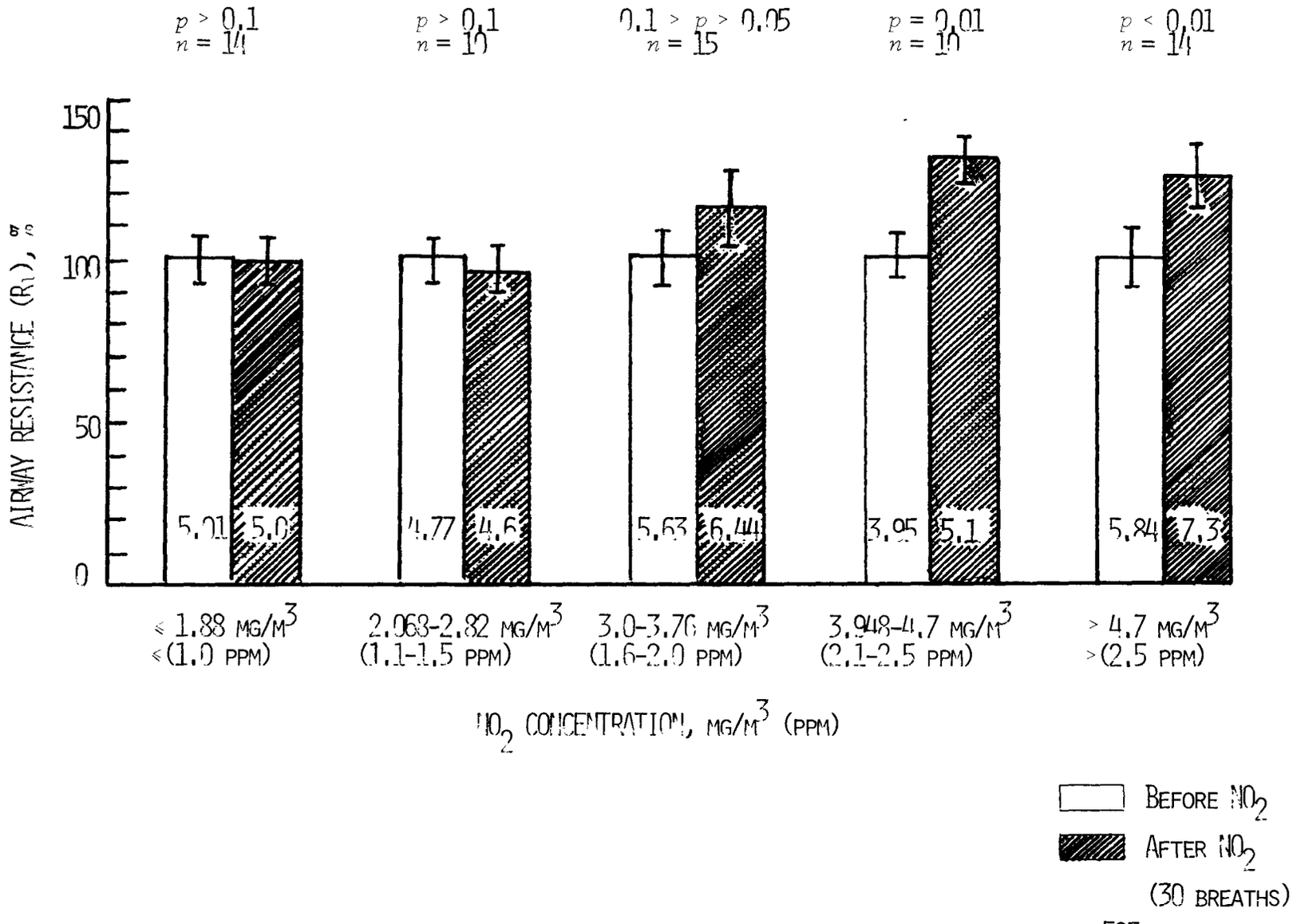


FIGURE 10-5. INFLUENCE OF NITROGEN DIOXIDE ON AIRWAY RESISTANCE. FROM VON NIEDING ET AL.[585]

measured during both rest and bicycling in chamber conditions at nitrogen dioxide concentrations of 0, 0.94, 1.88, 2.82, 3.76, 4.7, and 5.64 mg/m^3 (0, 0.5, 1.0, 1.5, 2.0, 2.5, and 3.0 ppm), and 45 min after filtering all the nitrogen dioxide from the chamber. Airway resistance increased in most normal subjects during rest when exposed to a concentration of 5.64 mg/m^3 (3.0 ppm). Resistance increased during exercise, beginning in some subjects at 2.82 mg/m^3 (1.5 ppm). Subjects with chronic respiratory disease experienced increases in airway resistance during rest beginning at 3.76 mg/m^3 (2.0 ppm), and during exercise at 2.82 mg/m^3 (1.5 ppm).

After staying in the clean, filtered chamber for 30 min at the start of each test run, most subjects showed a decline in airway resistance, compared with measurements taken immediately after entering the chamber from the ambient environment.

EFFECTS IN MAN FROM EXPOSURES TO NITROGEN DIOXIDE IN THE PRESENCE OF OTHER POLLUTANTS

Experimental Studies

- Sensory Effects. The effects of nitrogen dioxide alone and in combination with other gases on thresholds for odor and dark adaptation have been reported by several Russian scientists. Shalamberidze[476] compared the effects of nitrogen dioxide, sulfur dioxide, and combinations of the two gases on 15 human subjects. Nitrogen dioxide was prepared by heating lead nitrate with quartz sand (Maser's method). Concentrations were determined by the Griess-Ilosvay reagent. Sulfur dioxide concentrations were measured with a Nephelometer. When inhaled together, sulfur dioxide and nitrogen dioxide acted additively. That is, an odor was detected when the sum of the fractional concentrations of each gas, expressed as a ratio of the gas

concentration in the mixture to the odor threshold concentration for each gas by itself, equaled 1.0 or more.

Using the same gas mixtures, Shalamberidze studied the effects of nitrogen dioxide, sulfur dioxide, and combinations of these gases on the threshold for dark adaptation. Observations were made during three series of tests: nasal inhalation of gases separately and combined for 5 min, oral inhalation as above, and nasal inhalation of gases separately for 25 min. These tests revealed that the threshold for dark adaptation occurred at nitrogen dioxide concentrations of 0.14 mg/m^3 (0.075 ppm) and sulfur dioxide at 0.60 mg/m^3 (0.21 ppm). When tested as components of a mixture, the two gases acted additively, as they had for odor perception. The threshold for dark adaptation with each gas was lower than that for odor. The odor threshold with nitrogen dioxide was 0.23 mg/m^3 (0.12 ppm); but for dark adaptation was about 60% lower, or 0.14 mg/m^3 (0.075 ppm). With sulfur dioxide, the threshold for dark adaptation [0.6 mg/m^3 (0.32 ppm)], about 38% of the odor threshold [1.6 mg/m^3 (0.32 ppm)].

Korniyenko[292] similarly evaluated odor perception as affected by a mixture of nitrogen oxides (not further specified), sulfur dioxide, sulfuric acid aerosol, and ammonia. After conducting a series of 439 tests, the author determined that the odor threshold of the gas mixture occurred at a concentration coefficient of 1.0, expressed as a sum of the fractional odor threshold concentrations of the component gases. He reported the odor threshold of the combination to be 0.02 mg/m^3 (0.011 ppm) for nitrogen oxides, 0.17 mg/m^3 (0.090 ppm) for sulfur dioxide, 0.11 mg/m^3 (0.059 ppm) for sulfuric acid aerosol, and 0.3 mg/m^3 (0.160 ppm) for ammonia.

Korniyenko also investigated the effect of the gas mixture on the electrical activity of the brain. He studied the amplitude of the alpha rhythm during a

series of 187 tests in which four subjects were exposed to combinations of the four gases at fractional concentrations of the threshold level at which changes in the amplitude of the alpha rhythms occurred. The earliest effect of combined exposure to these compounds was detected when the sum of the fractional concentrations, as determined above, was equal to one. Fractional concentrations for the mixture were 0.008 mg/m^3 (0.425 ppm) for nitrogen oxides, 0.08 mg/m^3 (0.043 ppm) for sulfur dioxide, 0.08 mg/m^3 (0.043 ppm) for sulfuric acid aerosol, and 0.2 mg/m^3 (0.106 ppm) for ammonia. The author states that these tests verified the principle that the effect of gas combinations is a simple summation of the isolated effects of individual components. This report does not describe the methods for generating and measuring gas concentrations.

V. Melekhina of the USSR studied the effect of combinations of three mineral acids [nitric ($HONO_2$), hydrochloric (HCl), and sulfuric (H_2SO_4)] on thresholds for odor and for eye adaptation.[352] Tests were performed on 16 volunteers, from 17 to 36 years of age. Acid concentrations were determined spectrophotometrically. The average odor threshold concentrations for individual acid aerosol exposures were 0.85 mg/m^3 (0.452 ppm) for nitric acid, 0.40 mg/m^3 (0.213 ppm) for hydrochloric acid, and 0.74 mg/m^3 (0.394 ppm) for sulfuric acid. When acid aerosol concentrations were expressed as mg of hydrogen ions per m^3, the odor thresholds for the different gases were nearly identical [0.0108-0.0148 mg/m^3 (0.006-0.008 ppm)].

The response to an acid aerosol mixture was equivalent to an additive response to the individual acids, if each acid concentration was expressed as a fraction of the molecular concentration necessary to produce the threshold response. When the aerosol concentration of the combined gas was expressed

as mg of hydrogen ions per m^3, the odor threshold was 0.130--essentially the same concentration as for individual acid aerosols. Thus, the effect resulting from the combination of acids in an aerosol appeared to be determined by the hydrogen ion concentration of the mixture and was nearly constant for individual or acid aerosol mixtures. Studies of dark adaptation thresholds produced similar results indicating that effects are determined by hydrogen ion concentrations of individual and of combined acid aerosols. The hydrogen ion concentration altering dark adaptation was the same as that causing an effect on odor threshold.

- Pulmonary Function. The response of the pulmonary airways to aerosol combinations of nitrogen dioxide and sodium chloride (NaCl) was investigated by Nakamura.[384] Airway resistance was measured both before and after exposure of three groups of healthy subjects from 18 to 27 years of age (Table 10-6). Nitrogen dioxide was measured by the Saltzman method. Each group was exposed to the sodium chloride aerosol alone for 5 min at a concentration of 1.4 mg/m^3 (0.75 ppm), rested for 10-15 min, exposed to to nitrogen dioxide alone at concentrations of 5.6 and 11.3 mg/m^3 (3 and 6 ppm) for 5 min, rested for 10-15 min, and then exposed to the combination for 5 min. All three groups exhibited increased airway resistance on exposure to the gas alone, but not on exposure to the isolated aerosol. Nitrogen dioxide concentrations of 5.6 and 11.3 mg/m^3 (3 and 6 ppm) produced 16 and 34% increases, respectively, in airway resistance. When the gas was combined with an aerosol, with an average particle size of 0.95 μm, airway resistance was double that caused by gas exposure alone. Aerosols with smaller particles, 0.22 μm, failed to increase the effect of nitrogen dioxide on airway resistance.

TABLE 10-6

Exposure of Humans to Sulfur Dioxide or Nitrogen Dioxide Alone and in Combination with Sodium Chloride Aerosol

Pollutant	Exposures: Gas Concentration mg/m^3 (ppm)		Exposures: Aerosol Particle Size, Median Diameter, μm	Average Effect on Airway Resistance, % (Baseline = 100%)
SO_2	25-172	(9-60)	None	127
n = 10	25-172	(9-60)	0.95	160
NO_2	5.6-43.2	(3-23)	None	119
n = 8	5.6-43.2	(3-23)	0.22	118
NO_2	11.3-75.2	(6-40)	None	124
n = 7	11.3-75.2	(6-40)	0.95	141

Schlipkoter and Brockhaus evaluated the effects of nitrogen dioxide, carbon monoxide, and sulfur dioxide on pulmonary deposition of inhaled dust in three persons.[463] Homogenized soot with particle sizes from 0.07 to 1.0 μm was suspended in Tween-80 solution. This suspension was sprayed through a heating tube, combined separately with maximal allowable concentrations of nitrogen dioxide [9 mg/m^3 (4.79 ppm)], carbon monoxide (55 mg/m^3), and sulfur dioxide (13 mg/m^3),[603a] and then administered to the three subjects by inhalation.

Differences between the concentrations of inhaled and exhaled dust were measured to indicate total pulmonary retention. Fifty percent of the inhaled dust was retained under control conditions after the sulfur dioxide and carbon monoxide exposures. Maximal allowable concentrations of nitrogen dioxide caused 76% pulmonary dust retention; the greatest difference from control conditions occurred when dust particle sizes ranged from 0.3 μm to 0.8 μm.

Epidemiological Studies

In all epidemiological studies of health effects of air pollution, multiple pollutants have been present in the ambient atmosphere. Respiratory illness or impaired pulmonary function may well have been a result of exposure to combinations of pollutants that included gaseous nitrogen dioxide as the pollutant of major interest but have not been limited to this agent. It is not possible, on the basis of epidemiological studies available, to ascribe a health hazard to nitrogen dioxide alone at a given concentration in the ambient atmosphere. However, since these studies are critical to the understanding of human health hazards from air pollution, the data reviewed below are important to the assessment of health effects of atmospheres containing nitrogen dioxide.

• Pulmonary Function. Mogi et al.[361] and Yamazaki et al.[610] examined the possible effects on pulmonary function from prolonged exposure to nitrogen dioxide in diesel exhaust from railroad engines in Japanese railroad tunnels and in inspection and repair sheds. The average nitrogen dioxide concentrations in four inspection and repair sheds, as determined by the Saltzman method, were 0.3-1.13 mg/m^3 (0.16-0.60 ppm). Maximal nitrogen dioxide concentrations varied from 0.34 to 3.0 mg/m^3 (0.18 to 1.6 ppm). The spirometric measurements of lung function of 475 railroad workers included vital capacity, 1-sec forced expiratory volume, maximal midexpiratory flow, and peak flow rates. The effects on lung function of age, height, smoking, family allergy history, pollutant concentrations at work location, and job description were factors considered in the analysis.

Spirometric test results were highest in workers from "no-pollution" job areas, as opposed to light-, medium-, and high-pollution work locations. However, values of lung function tests were not associated with a pollution gradient across the light-, medium-, and high-pollution work categories. The spirometric data, presented in the English translation, are not clearly adjusted for age and height. There is also some question whether multivariate analysis was used to adjust for possible age and height differences among work locations.

In the Chattanooga study of schoolchildren,[486] lung function in 7- and 8-year olds was assessed in relation to the nitrogen dioxide concentrations in several communities. Determinations of 0.75-sec forced expiratory volumes ($FEV_{0.75}$) were made weekly during 2 months of the school year among 306 children living in a neighborhood with relatively high concentrations of nitrogen dioxide among 264 children from an intermediate exposure area, and among 225 children from

a low exposure area. $FEV_{0.75}$ values of children in high concentration areas were significantly lower ($p < 0.05$) than those measured for children in the other areas. These differences, however, were of borderline significance and were not consistent during the 2 months of testing. The lung functions of children in the intermediate and low concentration areas were about the same. The association of diminished lung function with nitrogen dioxide concentrations as suggested in the Chattanooga study is weakly supported because results lacked consistency throughout the test period and a clear dose-response relation was not established.

In their studies of chronic respiratory disease, Speizer and Ferris,[498,499] and Burgess et al.[68] found no differences in results from pulmonary function tests administered to 128 Boston traffic police and 140 suburban patrol officers after exposure to nitrogen dioxide concentrations of 0.100 mg/m^3 (0.053 ppm) in the city and 0.080 mg/m^3 (0.040 ppm) in the suburbs; and sulfur dioxide at 0.092 mg/m^3 (0.049 ppm) in the city and 0.026 mg/m^3 in the suburbs.

Analysis by Cohen et al.[90] of several ventilatory tests, including spirometry and flow-volume curves, did not reveal any differences between nonsmoking Seventh Day Adventists living in the San Gabriel Valley of the Los Angeles Basin and nonsmokers of the same religion living in San Diego. Average and 90th percentile nitrogen dioxide concentrations in the San Gabriel Valley were respectively 0.096 and 0.188 mg/m^3 (0.051 and 0.100 ppm), and in San Diego 0.043 and 0.113 mg/m^3 (0.023 and 0.060 ppm).

The four epidemiologic surveys of lung function in relation to nitrogen dioxide exposure, cited above and summarized in Table 10-7, reveal that at the concentrations given, nitrogen dioxide in a mixed urban atmosphere has little effect on lung function.

• Acute Respiratory Disease. Petr and Schmidt found a twofold excess in acute respiratory disease among 7- to 12-year old children living near a large chemical complex in Czechoslovakia compared with children of the same age living in a low-exposure community of similar socioeconomic characteristics.[414,415] Exposures, as determined by colorimetric methods, are shown in Table 10-8. The composition of the nitrogen oxides was not specified in the report, although the concentrations appear to represent nitrogen dioxide of moderately low concentration [< 0.066 mg/m^3 (0.035 ppm)] in the "high-exposure" towns.

A greater number of hypertrophied tonsils and cervical lymph nodes were found in children from both towns with high pollution. Also, more children with retarded physical development were recorded as living in these areas. Several hematologic indexes, not clinically used in the United States, were evaluated. These included the lymphocytogram (ratio of lymphocytes with narrow cytoplasm to lymphocytes with broad cytoplasm), index of proliferation of monocytes (ratio of promonocytes to polymorphic monocytes), and an index of monocytic differentiation (ratio of monocytes to polymorphic monocytes). Children from the low-exposure area (the town of Bohdanec) had much lower lymphocytogram values and higher indexes of proliferation and differentiation of monocytes. Children from Rosice, the town with higher nitrogen oxides but lower sulfur dioxide, deviated most from the control area (Bohdanec). According to the authors, these data imply that air pollutants affect the maturation and proliferation of lymphocytes and monocytes. The clinical significance, if any, of this effect is not known. Methemoglobin content in 50% of the tested children from Rosice was above the normal physiologic range of $0.86 \pm 0.3\%$, but

TABLE 10-7

Comparison of Four Epidemiologic Surveys of Lung Function and Nitrogen Dioxide Exposure

Study Population	NO_2 Concentration, mg/m^3 (ppm)	Effect on Lung Function	Reference
Railroad tunnel and repair workers in Japan	Average 0.30-1.13 (0.16-0.60) Maximum 0.34-3.00 (0.18-1.60)	Diminished FEV_1, VC, MMF, and peak flow, compared with non-exposed workers	361,610
Schoolchildren in Chattanooga, aged 7-8 years	Average 0.15-0.28 (0.08-0.15) 90th Percentile 0.28-0.94 (0.15-0.50)	Borderline decrease in $FEV_{0.75}$	486
Central city vs. suburban Boston policemen	0.103 (0.055) vs. 0.075 (0.04)	No difference in various tests of pulmonary function	68,498,499
Nonsmoking Seventh Day Adventists in Los Angeles vs. San Diego	Average 0.096 (0.051) vs. 0.043 (0.023) 90th Percentile 0.188 (0.10) vs. 0.113 (0.06)	No difference in various tests of pulmonary function	90

TABLE 10-8

Concentrations of Nitrogen Oxides and Sulfur Dioxide in Several Czechoslovakian Communities Where Effects on Children were Studied[414,415]

Community	Distance from Source, km	Concentrations, mg/m^3 (ppm)	
		Nitrogen Oxides (undefined)	SO_2
High-exposure			
Rosice	1.5-2.0	0.02-0.07 (0.011-0.037)	0.01-0.12 (0.005-0.064)
Ohrazenice	1.5-2.0	0.005-0.05 (0.003-0.027)	0.03-0.32 (0.016-0.070)
Low-exposure			
Bohdanec	6	None detected	Traces

below the upper limit of normal in control children. These results suggest relatively high exposures of Rosice children to atmospheric nitric oxide or to nitrites in food or water. Analysis of water samples from Rosice showed that most of the wells contained high nitrite concentrations, ranging from 100 to 290 mg/l. On reexamination of the children from Rosice after an undisclosed interval, the authors report that methemoglobin values had returned to normal, after the concentration of atmospheric nitrous gases in the area had been reduced. The authors contend that these reversions support their belief that the methemoglobinemia was caused by atmospheric nitrogen oxides.

Petr and Schmidt's results are difficult to evaluate because their studies lack information on air pollutant concentrations over time. In Rosice, measured exposures to sulfur dioxide and nitrogen oxides were below US and USSR standards, and observed health effects could well have been produced by higher past exposures or by other environmental factors. Evidence of methemoglobinemia suggests much higher atmospheric nitric oxide concentrations or nitrites in food or water than are indicated in the report. The authors' conclusion that combined exposure to elevated concentrations of nitrogen oxides and sulfur oxides have a greater adverse effect on children than exposure to high levels of sulfur oxides in the presence of low nitrogen oxides, requires confirmation.

The health of preschool- and schoolchildren living 0.5 to 1.7 km south and 0.5 and 3.0 km west of a superphosphate fertilizer manufacturing plant in the USSR was compared by Lindberg[321] with children from a control area 10 km away with similar socioeconomic status. Medical examinations were conducted in the winter of 1953-1954. Pollutant concentrations near the plant were reported only in reference to the maximal permissible

concentrations for the USSR, and ranged from 0.32 to 3.4 mg/m^3 (0.17-1.8 ppm) for nitrogen dioxide, 0.25 to 1.42 mg/m^3 (0.09-0.50 ppm) for sulfur dioxide, and 0.03 to 0.16 mg/m^3 for sulfuric acid aerosol. Children living near the plant experienced a 16- to 17-fold excess in acute upper respiratory disease, a 6- to 12-fold excess in nontuberculous chest film abnormalities, and a 1.6- to 2.5-fold excess in enlarged lymph glands when compared with children in the control area. An association between length of residence in the polluted town and respiratory disease frequency was also reported. Methods of air monitoring and health examinations are not described in the report. At the high concentrations recorded for the polluted town, the excess in respiratory disease may be attributed to any one or to combinations of the measured pollutants.

Adolescents 16 to 19 years old undergoing vocational training in a factory manufacturing fertilizers in Kemerovo, USSR, and in a chemical works at Novokemerovskii, USSR, were compared by T. L. Giguz[173] with 85 controls of the same age and from the same schools but without occupational exposure to chemical pollutants. A total of 140 adolescents in training were exposed to nitrogen oxides and ammonia at concentrations that did not exceed the maximum permissible concentrations (MPC) [MPC for average daily nitrogen dioxide, 0.10 mg/m^3 (0.053 ppm) in 1964].[337] Exposures lasted 3 hr/day for 150 days in the first year of training, and 6 hr/day for 200 days in the second year. Studies of trainees, during their 2 years of training, indicated that exposed adolescents had an increased incidence of acute respiratory disease and increased serum levels of beta-lipoproteins, cholesterol, and albumin.

The investigator states that the main influence on the health of adolescents working in the fertilizer and chemical plants was exposure to low concentrations of nitrogen oxides and ammonia. Although his report lacks information on the methods used, sampling frequencies, and results of atmospheric monitoring, his data suggest an association between acute respiratory disease and nitrogen oxide exposures at ambient concentrations. Polyak reported that residents living within 1 km of the Shehelkovo, USSR, chemical works made 44% more visits to the health clinic for respiratory, visual, nervous system, and skin disorders than residents who live farther than 3 km from the chemical complex.[423] Atmospheric nitrogen dioxide exceeded the MPC by a factor of 5.8-12 [MPC, 0.10 mg/m^3 (0.053 ppm), 24-hr average]. Sulfur dioxide and sulfuric acid exceeded the MPC by factors of 1.5 and 4, respectively [MPC, 0.15 and 0.10 mg/m^3, respectively]. None of the persons studied in the two areas was employed at the chemical works; housing and living conditions were the same in the two communities. Residents of the high-exposure area also complained of odors, damage to vegetation, rusting of iron, and destruction of bee colonies as a result of emissions from the chemical works. The reported effects were probably attributable to the combination of pollutant exposures, namely, nitrogen dioxide at 0.58-1.2 mg/m^3 (0.31-0.64 ppm), sulfur dioxide at 0.225 mg/m^3 (0.08 ppm), and sulfuric acid at 0.40 mg/m^3.

Belanger[39] studied the possibility of an association between daily admissions to the Philadelphia General Hospital in 1966 and daily concentrations of nitrogen dioxide, sulfur dioxide, total suspended particles (TSP), coefficient of haze (COH), and oxidants. Although during a few months levels of COH, nitrogen dioxide, and sulfur dioxide were significantly and

positively associated with hospital admissions for respiratory disease, no consistent pattern of association between individual pollutants and hospital admissions was observed. Hospital admissions are not sensitive indicators of air pollution effects because of strong "extraneous" factors, such as availability of beds, daily and seasonal variations in hospital admission rates, and differences between inpatient and outpatient treatment for the same disorder. Hospital admission records in any US city are probably inadequate reflections of a disease burden in a community.

In the Chattanooga schoolchildren studies,[411,486-488] the effect of community exposure to nitrogen dioxide was evaluated in relation to the incidence of acute respiratory disease among schoolchildren, their siblings, and parents. Different distances of three study neighborhoods from a trinitrotoluene manufacturing plant resulted in an area gradient for exposure to nitrogen dioxide. The acute respiratory disease experience of 871 families with 4,043 individuals was assessed at biweekly intervals in these study areas between November 1968 and April 1969. As shown in Table 10-9, respiratory illness rates per 100 persons were significantly higher for each family segment in the high nitrogen dioxide exposure neighborhood. The relative excess in respiratory illness in the high nitrogen dioxide areas was 18.8% averaged over all family members compared with average rates in intermediate and low nitrogen dioxide areas. In the original reports[486,487] nitrogen dioxide concentrations were determined by the Jacobs-Hochheiser method which has subsequently been shown to be unreliable.[562] Alternate nitrogen dioxide exposure data were available from measurements obtained at an 11-station monitoring network operated by the US Public Health Service and the US Army in Chattanooga from September 1967 through November 1968.[488] The continuous Saltzman

method was used for these latter measurements. Results are given in Table 10-9.

As a follow-up to the first Chattanooga studies, the same neighborhoods were surveyed in 1970 for lower respiratory disease frequency among first- and second-grade elementary schoolchildren and among infants born between 1966 and 1968.[411] Bronchitis rates per 100 children corresponded to the area gradient in nitrogen dioxide among the population of schoolchildren who had lived in the same neighborhood for 3 or more years; rates in children from the high nitrogen dioxide area were significantly greater (32.2 per 100) than in children from the low area (23.2 per 100), and rates in the intermediate exposure area (31.2 per 100) were nearly as high as those of children from the high exposure area. For schoolchildren who had lived in the neighborhood for less than 2 years, bronchitis rates did not correspond to an area exposure gradient. Bronchitis rates among infants born between 1966 and 1968 who had lived in the same neighborhood for 1, 2, and 3 years did not always correspond to the area gradient for nitrogen dioxide, although the general pattern of highest rates in the high or intermediate exposure areas was observed.

Excess respiratory disease in the high exposure neighborhoods of Chattanooga was attributed to nitrogen dioxide concentrations exceeding annual averages of 0.113 mg/m^3 (0.06 ppm). As in all population studies, other pollutants were present in the ambient atmosphere, and these included elevated concentrations of suspended sulfates (0.010 to 0.013 mg/m^3) and suspended nitrates (0.0038 to 0.0072 mg/m^3). Reports from the US Environmental Protection Agency's Community Health and Environmental Surveillance System[574a] suggest that respiratory tract

TABLE 10-9

Average Biweekly Respiratory Illness Rates Given by Family Segment and Study Area: Chattanooga November 1968-April 1969

NO_2 Concentration mg/m^3	(ppm)	Study Area	Respiratory Illness Rate per 100 Persons by Family Segment: Second-Grade Schoolchildren	Siblings	Mothers	Fathers
0.150-0.282	(0.08-0.15)[a]	High NO_2 Exposure	22.2	18.8	14.2	12.1
0.113	(0.06)	Intermediate NO_2 Exposure	18.0	15.6	11.8	8.8
0.056	(0.03)	Low NO_2 Exposure	20.1	17.0	12.3	9.6

NOTE: Chi-square lists of area differences, High NO_2 vs. average of other areas:

Second graders, $p = 0.005$; Siblings, $p = 0.017$;

Mothers, $p = 0.004$; Fathers, $p = 0.017$

[a] Average values for each of three monitoring stations were 0.150, 0.150, and 2.82 mg/m^3.

irritation may occur at these sulfate and nitrate concentrations. Therefore, excess respiratory illness observed in the Chattanooga studies may well have been a result of exposure to combinations of pollutants that included gaseous nitrogen dioxide and particulate sulfates and nitrates, as well as intermediary compounds such as sulfuric and nitrous acids. Until more is known about the toxicities of these pollutants, both separately and in combination, it is not possible to ascribe a causal relationship to any one pollutant at a given concentration in the ambient atmosphere.

A summary of the several epidemiological studies associating nitrogen dioxide exposure, in the presence of other pollutants, with acute respiratory disease is given in Table 10-10.

- Chronic Respiratory Disease. Fujita and his associates conducted surveys of chronic bronchitis prevalence in 1962 and again in 1967, in the Tokyo, Tsurumi, and Kawasaki areas of Japan.[165] In each survey, 7,800 post office employees from the same offices in each of the three cities were evaluated and categorized by work location into downtown and industrial districts, intermediate sectors, and suburban areas. Chronic bronchitis rates were consistently higher in the 1967 survey. Increased rates were reported for all age groups, in all smoking categories, and for both indoor and outdoor employees. Average chronic bronchitis prevalence doubled in 1967. (See Table 10-11.) The authors attributed this to increases in the concentrations of sulfur dioxide, nitric oxide, and nitrogen dioxide in the atmosphere between 1962 and 1967, not to increases in cigarette smoking.

Although scant information on ambient air concentrations is given, the authors state that in all districts there has been an increase of such harmful gases as sulfur dioxide, nitric oxide, and nitrogen dioxide, in contrast to a pronounced decrease in the amount of suspended dust particles. Pollutant concentrations reported by Fujita were based on measurements taken with automatic instruments by the Environmental Pollution Section of the Tokyo metropolitan government during 1962-1966 in Tokyo, Tsurumi, and Kawasaki (See Table 10-12).

The Air Quality Bureau of the Japanese Environmental Agency published air quality data from one urban and two suburban sectors of Tokyo obtained from 1964 through 1970[7] during annual surveys conducted by the National Institute of Hygienic Sciences. (See Table 10-13.)

The air quality data in Tables 10-12 and 10-13 are insufficient documentation of increases in sulfur dioxide or nitrogen dioxide between 1962 and 1967. Although the authors carefully point out the age- and smoking-specific changes in bronchitis rates between 1962 and 1967, they do not substantiate the attribution of such changes to increasing sulfur oxide or nitrogen oxide pollution.

The report of the Expert Committee on Air Quality Criteria for Oxides of Nitrogen and Photochemical Oxidants (Japan),[82] includes a survey of bronchitis prevalence in housewives living in six Japanese communities.[82] Four hundred women, nearly all of whom were nonsmokers, 30-69 years old, were selected for study. The prevalence of chronic respiratory disease was determined through administration of the British Medical Research Council's standardized questionnaire. Table 10-14 contains the data for pollutant concentrations and symptom prevalence in each community during the winter of 1970-1971. Although simple correlation coefficients between persistent cough,

TABLE 10-10

Summary of Epidemiologic Studies of Nitrogen Dioxide Exposure and Acute Respiratory Disease

Study Population	NO_2 Concentration, mg/m^3 (ppm)	Reported Effect	Reference
Czechoslovakian children, aged 7-12 years	NO_x average = 0.02-0.07 mg/m^3[a]	Excess of hypertrophied tonsils and lymph nodes; changes in hematologic indexes	414, 415
USSR preschool and schoolchildren living near a fertilizer plant	0.32 (0.17) to 3.4 (1.8)[b]	17-fold excess of upper respiratory disease: 6- to 12-fold excess of abnormal chest films	321
USSR adolescents in vocational training at chemical and fertilizer plants	Less than 0.10 (0.053)[c]	11-27% excess of acute respiratory disease increased blood lipoproteins and cholesterol	173
Residents living within 1 km of a USSR chemical works	0.58 (0.31) to 1.2 (0.64)[d]	44% increase in clinic visits for respiratory, visual, nervous system, and skin disorders	423
Soviet children aged 8-11 years living near a ferrous metallurgic plant	Nitrogen oxides = 46.3 (87) to 93.6 (176)[e]	5-fold excess of upper respiratory disease; 3-fold excess of tonsilitis; 2.5-fold excess of atrophic rhinitis; significant lag in growth, weight, and chest circumference; decreased urinary excretion of vitamin C	612
Patients admitted to Philadelphia General Hospital for respiratory causes	----	No consistent correlation of respiratory admissions with nitrogen dioxide	39
Chattanooga schoolchildren, their siblings and parents	Average 0.15 (0.08) to 0.28 (0.15)[f] 90th Percentile 0.19 (0.10) to 0.94 (0.50)	1-17% excess of acute respiratory disease in children; 9-33% excess in adults	486-488
Chattanooga infants and children 6-9 years old	0.15 (0.08) to 0.28 (0.15)[f]	10-58% excess of acute bronchitis among infants; 39-71% excess among 6-9 year-old children	411

[a]Other pollutant exposures included sulfur dioxide at 0.01-0.12 mg/m^3.
[b]High concentrations of sulfur dioxide and sulfuric acid were also measured.
[c]Exposure to ammonia below the USSR maximal permissible concentration were reported.
[d]Concentrations of sulfur dioxide (0.225 mg/m^3) and sulfuric acid (0.40 mg/m^3) were also measured.
[e]High concentrations of sulfur dioxide and hydrogen sulfide and moderate concentrations of phenol were reported.
[f]Suspended nitrate concentrations of 3.8-7.2 $\mu g/m^3$ and suspended sulfate concentrations of 10.0 $\mu g/m^3$ were also measured.

TABLE 10-11

Bronchitis Prevalence Rates per 100 Employees

Year	Work District		
	Downtown and Industrial	Intermediate	Suburban
1962	5.0	3.7	3.7
1967	8.4	8.0	8.1

Table 10-12

Pollutant Concentrations during 1962 and 1966 by Work District, mg/m^3

Pollutant	Downtown and Industrial		Intermediate		Suburban	
	1962	1966	1962	1966	1962	1966
Nitrogen dioxide	--	0.036	--	--	--	--
Nitric oxide	--	0.100	--	--	--	--
Sulfur Dioxide	0.147	0.214	0.161	0.188	0.054	0.054

phlegm, and air pollutants were highest for nitrogen dioxide, nitric oxide, and total nitrogen oxides, the concentrations of these pollutants were usually below the US nitrogen dioxide standard of 0.10 mg/m^3 (0.05 ppm) in five of six communities. However, less significant correlation coefficients were obtained between respiratory symptoms and suspended particulates, even though the concentrations of particulates greatly exceeded the US standard of 0.075 mg/m^3 annual average. In the two communities with highest bronchitis rates, Ohmuta and Higashi-Osaka, pollutant concentrations were 4 to 5 times higher than the US air quality standard for suspended particulates and 42-79% above the standard of 0.080 mg/m^3 (0.03 ppm) for sulfur dioxide; in only one of the communities (Higashi-Osaka) was the US standard for nitrogen dioxide [0.100 mg/m^3 (0.05 ppm)] exceeded.

From these results, excess bronchitis seems to be attributable largely to high concentrations of sulfur oxides and suspended particulates although nitrogen oxides may have contributed to the observed excess bronchitis prevalence. No single pollutant can explain the observed community gradient in respiratory symptom prevalence.

In addition to the pulmonary function tests described earlier in this chapter, Speizer and Ferris[498,499] and Burgess *et al.*[68] compared the prevalence of chronic respiratory disease among 128 traffic officers working in central Boston with 140 suburban patrol car officers. The exposure of each group to nitrogen dioxide (Saltzman method) and sulfur dioxide (West-Gaeke method) was determined at several work locations for the central city officers and in the patrol cars of suburban officers. A slight but not statistically significant excess in chronic respiratory disease was found in smokers as compared with nonsmokers and exsmokers from the central city group (Table 10-15).

TABLE 10-13

Tokyo Air Quality Data, 1964-1970

Monitoring Station	Pollutant	Yearly Mean Concentration, mg/m^3 (ppm)						
		1964	1965	1966	1967	1968	1969	1970
Kasumigaseki (urban)	Nitrogen dioxide	0.036 (0.019)	0.049 (0.026)	0.056 (0.030)	0.047 (0.025)	0.058 (0.031)	0.115 (0.061)	0.120 (0.064)
	Nitric oxide	0.057 (0.046)	0.058 (0.046)	0.046 (0.037)	0.044 (0.035)	0.074 (0.059)	0.164 (0.131)	0.170 (0.136)
Itabashi (suburban)	Nitrogen dioxide	0.038 (0.020)	0.043 (0.023)	0.049 (0.026)	0.041 (0.022)	0.041 (0.022)	0.055 (0.029)	0.073 (0.039)
	Nitric Oxide	0.026 (0.021)	0.028 (0.022)	0.034 (0.027)	0.034 (0.027)	0.069 (0.055)	0.086 (0.069)	0.123 (0.098)
Okaramachi (suburban)	Nitrogen dioxide	-- --	0.077 (0.041)	0.081 (0.043)	0.073 (0.039)	0.070 (0.037)	0.100 (0.053)	0.113 (0.060)
	Nitric oxide	-- --	0.109 (0.087)	0.159 (0.127)	0.167 (0.134)	0.088 (0.070)	0.091 (0.073)	0.176 (0.141)

TABLE 10-14

Relation of Respiratory Symptoms to Atmospheric Pollutants for Various Communities in Japan

Community	Prevalence of Persistent Respiratory Symptoms, %			Atmospheric Pollutants, mg/m^3 (ppm)				
	Cough and Phlegm	Phlegm	Cough	NO_2	NO	NO_x	SO_2	Suspended Particles
Tonda-Bayashi	1.2	4.3	3.0	0.032 (0.017)	0.020 (0.016)	(0.033)	0.037 (0.013)	0.111
Sakura	1.9	4.1	3.5	0.024 (0.013)	0.018 (0.015)	(0.030)	0.069 (0.024)	0.196
Ichihara	1.9	5.3	4.8	0.028 (0.015)	0.016 (0.013)	(0.026)	0.077 (0.027)	0.352
Fukuokanishi	4.1	12.3	5.8	0.079 (0.042)	0.021 (0.017)	(0.059)	0.029 (0.010)	0.183
Ohmuta	5.3	11.0	9.4	0.038 (0.020)	0.048 (0.039)	(0.059)	0.114 (0.040)	0.350
Higashi-Osaka	5.6	11.0	7.9	0.145 (0.077)	0.046 (0.037)	(0.114)	0.143 (0.050)	0.498
Correlation coefficients for air pollutants with persistent cough and phlegm				0.71	0.88	0.85	0.68	0.65

Chronic respiratory disease among parents of high school students was studied in three exposure areas in Chattanooga.[83] Sample sizes, nitrogen dioxide exposures, and chronic bronchitis rates are given in Table 10-16. Higher nitrogen dioxide concentrations occurred in the high nitrogen dioxide area from 1966 to 1969 than during the course of the study (1970), because of decreased trinitrotoluene production at the point source. As shown in Table 10-16, chronic bronchitis prevalence rates were not associated with the area gradient in nitrogen dioxide exposure. Past exposures in the high nitrogen dioxide area had exceeded the national primary standard, and exposures at the time of testing were below the standard.

In their study of nonsmoking, 45- to 64-year-old Seventh Day Adventists (see earlier section on Pulmonary Function), Cohen and his associates[90] compared chronic respiratory disease prevalence and lung function among 136 residents of the San Gabriel Valley in the Los Angeles Basin with that among 207 residents of San Diego. Exposures to photochemical oxidants and nitrogen dioxide were determined by the potassium iodide and Saltzman methods, respectively. (See Table 10-17.) Area differences in average oxidant concentrations were small, whereas nitrogen dioxide averages differed by as much as a factor of 2. Area differences in peak oxidant and nitrogen dioxide exposures were similar for the two pollutants. The national primary standard for nitrogen dioxide, 0.1 mg/m^3 (0.053 ppm), was not exceeded, whereas the national primary standard for oxidants, 0.16 mg/m^3 (0.08 ppm), a maximal 1-hr concentration not to be exceeded more than once per year, was exceeded during 10% of the testing in San Diego, and more frequently in Los Angeles. The two study groups showed no difference in the prevalence of chronic respiratory disease; the rates of this disease were less than 4% in each age-sex-exposure combination.

TABLE 10-15

Exposures and Respiratory Disease in Traffic Officers

Exposed Groups	Concentration[a] mg/m^3 (ppm)		Prevalence of Chronic Respiratory Disease, %[b]			
	NO_2	SO_2	NS	LS	HS	ExS
Central city traffice officers (n=128)	0.103 (0.055)	0.092 (0.035)	17	50	60	28
Suburban patrol car officers (n=140)	0.075 (0.040)	0.026 (0.010)	15	43	55	31
National primary air quality standard	0.103 (0.055)	0.080 (0.030)				

[a] Average of 2 sampling days per season of the year for 1 year at each of 16 work stations.

[b] NS, nonsmoker; LS, light smoker (10-24 cigarettes/day); HS, heavy smoker (25 + cigarettes/day); ExS, Ex-smoker.

TABLE 10-16

Chronic Respiratory Disease in Chattanooga Parents of High School Students

NO_2 Exposure Group	NO_2 Concentration mg/m^3 (ppm)		Prevalence of Chronic Bronchitis, %[a]	
	June-Dec 1970	Dec 1967-Nov 1968	Early Disease	Advanced Disease
Low (n=234)	0.058 (0.031)	0.053 (0.028)	30	11
Intermediate (n=755)	0.071 (0.038)	0.117 (0.062)	33	20
High (n=652)	0.092 (0.049)	0.150-0.282 (0.080-0.150)	25	13

[a]Rates adjusted for smoking, sex, race, and age.

The study suggested that the twofold difference in nitrogen dioxide exposures that remained below the national primary standard, in the presence of oxidant exposures that exceeded the national standard in both groups, was unassociated with effects on chronic respiratory disease or lung function (previously cited) in a nonsmoking population.

- Excess Mortality. Mortality rates for various cancer categories, cardiovascular disease, and respiratory disease in 38 US Standard Metropolitan Statistical Areas (SMSA) during 1959-1961 and 1961-1964 were analyzed by Hickey *et al.* in relation to air pollutant measurements taken at the National Air Surveillance Network station in each SMSA.[230,231] Nitrogen dioxide, sulfur dioxide, suspended sulfates, total particles, calcium, chromium, copper, iron, lead, manganese, nickel, tin, titanium, vanadium, zinc, and water hardness were included in the analysis. Mortality rates were analyzed with and without regard to age, sex, and race differences. The author applied a modification of multiple regression analysis in which combinations of variables were selected from the complete set of independent variables with the objective of maximizing the square of the multiple correlation coefficient (R^2) for the selected subset. Only variables significantly predictive of each mortality rate were selected.

As shown in Table 10-18, nitrogen dioxide and sulfur dioxide were thus repeatedly positively associated with age-, race-, and sex-adjusted or unadjusted mortality rates for various cancers and for arteriosclerotic heart disease. Other pollutants were variably and often negatively associated with these mortality categories. Hickey *et al.* speculate that, if cancer and heart disease are cumulative somatic genetic diseases, then

TABLE 10-17

Concentrations of Photochemical Oxidants and Nitrogen Dioxide, 1963-1967, Used as Exposure Data in a Study of Nonsmoking Seventh Day Adventists[a]

Pollutants (hourly average)	San Gabriel Valley		San Diego	
	mg/m^3	(ppm)	mg/m^3	(ppm)
Oxidants				
Arithmetic mean	0.092	(0.046)	0.076	(0.038)
90th percentile	0.260	(0.130)	0.160	(0.080)
Nitrogen dioxide				
Arithmetic mean	0.096	(0.051)	0.043	(0.023)
90th percentile	0.188	(0.100)	0.113	(0.060)

[a] From Cohen *et al.*, 1972.

the genetic effects of exposure to nitrogen dioxide and sulfur dioxide should be demonstrable in exposed populations as excess cancer and heart disease. They present several arguments to support the possibility that atmospheric nitrogen oxides and sulfur oxides could be mutagens. The weakness of their analysis is not in the methodology or biologic plausibility of the associations, though the latter is tenuous, but in the quality of the exposure data. Neighborhood differences in pollutant exposures within the same SMSA are often larger than pollutant differences between SMSA's. Consequently, the one to three stations of the National Air Surveillance Network within an SMSA are poor estimators of the pollutant burden to the population or of differences in this burden between SMSA's. Furthermore, population mobility in the United States is so great that the place of death is often different from the longest residence location. If cumulative exposures were responsible for a postulated "cumulative somatic genetic disease," these exposures should be estimated from data from the air monitoring station where the patient lived the longest.

The consistency of Hickey's findings concerning nitrogen dioxide and sulfur dioxide prevents complete dismissal of his conclusions, which are stated more in the form of hypotheses for later verification. The biologic evidence marshaled in support of his statistical findings is not unreasonable, and the statistical analysis appears to be sound. In 27 of the 38 SMSA's studied, nitrogen dioxide concentrations ranged from 0.080 and 0.116 mg/m^3 (0.043 and 0.062 ppm). These concentrations bracket the existing US standard of 0.100 mg/m^3 (0.053 ppm) and suggest the need for analysis of mortality and pollutant concentrations in later years. The hypothesis of Hickey et al. that nitrogen oxides and sulfur oxides may be exerting genetic effects in the population should be explored in experimental studies.

TABLE 10-18

Multiple Repression Analysis of Pollutant Exposures and Cancer Mortality

Cause of Death	Year of Death	Age-Sex-Race Adjustment	Variables Associated with Death Rate	R^2
Breast Cancer	1959-1961	No	NO_2, SO_2, Cd, Cu	0.58
Breast Cancer	1959-1961	Yes	NO_2, Ni, Ti	0.56
Breast Cancer	1961-1964	No	NO_2, SO_2, Cd	0.55
Lung Cancer	1959-1961	No	NO_2, SO_4, Cu, Ti, As	0.73
Lung Cancer	1959-1961	Yes	NO_2, Mn, V, Ti, As	0.61
Lung Cancer	1961-1964	No	NO_2, SO_2, Cu	0.51
Total Cancer	1961-1964	No	NO_2, SO_2, Cd	0.55
Arterio-sclerotic heart disease	1959-1961	No	NO_2, SO_2, Cd, water hardness	0.47
Arterio-sclerotic heart disease	1959-1961	Yes	SO_2, SO_4, Cu, Zn	0.56

Lebowitz studied variations in daily mortality in relation to daily air pollution and weather variables in New York, Philadelphia, and Los Angeles during 1962-1965.[311] Nitrogen oxide measurements were available only for the New York and Los Angeles analyses. Mortality data were statistically transformed to control for extraneous variables including day of week and seasonal effects. A relation between air pollution and weather variables and daily mortality was found in each of the cities.

In New York, multiple regression analysis revealed a significant but negative influence of daily nitrogen oxide concentration on mortality in winter. Particulate matter (measured as coefficient of haze), low temperatures, and wind speed were also significant determinants of daily mortality. The R^2 for the combined variable analysis was 0.31. Nitrogen oxide concentrations were not statistically associated with mortality in summer.

In Los Angeles, winter mortality of persons 45-64 years old, 65 and older, and all ages combined was significantly and positively related to daily nitrogen oxide concentration during 1962-1965. Sulfur dioxide, temperature, and wind speed were also significant determinants of daily winter mortality variations. The R^2 for this combination of variables was 0.11. In summer, nitrogen oxides were not a significant variable, whereas sulfur dioxide, carbon monoxide, relative humidity, and wind speed were statistical determinants of daily mortality. These results fail to provide convincing evidence of a relation between nitrogen oxides and daily mortality. Findings in New York were opposite to those in Los Angeles, and the associations were not consistent for all seasons.

- Acute Toxicity. Acute exposure to high concentrations of nitrogen dioxide is an uncommon occupational hazard of welders,[5,207,341,402] silo fillers,[101,185,326,431] miners,[37,379] chemists,[338,357] firemen,[547] and workers employed in the manufacture of nitric acid.[580] These exposures are far in excess of ambient concentrations and are noteworthy only in proving that high concentrations of nitrogen dioxide are extremely toxic. Five distinct clinical responses to several such high concentrations, based on observations of occupational exposures, are summarized in Table 10-19. Acute respiratory and nasal irritation are first noted at nitrogen dioxide concentrations of 28-47 mg/m^3 (15-25 ppm). Reversible pneumonia and bronchiolitis result from exposures to 47-141 mg/m^3 (25-75 ppm), whereas concentrations of 282-564 mg/m^3 (150-300 ppm) have caused fatal bronchiolitis and bronchopneumonia. In contrast, workers in Italy employed in nitric acid manufacturing and exposed to an average of 56.4-65.8 mg/m^3 (30-35 ppm) for an unspecified number of years exhibited no signs or symptoms of injury.[580]

Accidental exposure of a large group of hospital workers occurred in 1929 as a result of a fire in the X-ray room of the Cleveland Clinic.[147] Combustion of nitrocellulose films released high concentrations of nitric oxide, carbon monoxide, and hydrocyanic acid. Ninety-seven persons died within 2 hr after exposure to carbon monoxide and hydrocyanic acid.

To investigate possible delayed effects of acute nitrogen dioxide exposure, 87% of the individuals present at the fire were studied 36 years later by Gregory and colleagues.[192] Observed survival rates among those exposed were no different than those of controls who were present at the

TABLE 10-19

Effects of Acute Exposure to High Nitrogen Dioxide Concentrations

NO_2 Concentration, mg/m^3 (ppm)	Clinical Effect	Time between Exposure and Termination of Effect
940 (500)	Acute pulmonary edema--fatal	Within 48 hr
564 (300)	Bronchopneumonia--fatal	2-10 days
282 (150)	Bronchiolitis fibrosa obliterans--fatal	3-5 weeks
94 (50)	Bronchiolitis, focal pneumonitis--recovery	6-8 weeks
47 (25)	Bronchitis, broncho-pneumonia--recovery	6-8 weeks

fire but not exposed to its fumes. Acute exposure to high concentrations of nitrogen dioxide therefore appear to have no effect on long-term survival.

SUMMARY OF HUMAN STUDIES

Acute Effects of Nitrogen Dioxide

Studies of human volunteers have provided the most precise documentation of the acute effect of nitrogen dioxide exposure (Table 10-20). The earliest response occurs in the sense organs. The odor of nitrogen dioxide is perceived at concentrations of 0.23 mg/m^3 (0.12 ppm), while changes in dark adaptation occur at 0.14 to 0.50 mg/m^3 (0.075 to 0.26 ppm). These sensory perceptors are categorized as physiologic responses; there is no evidence for sequelae in terms of human pathology, and the responses in all cases were immediately reversible.

The studies of airway resistance by Von Nieding, Rokaw, and Suzaki indicated acute effects after 15 to 45 min exposures to nitrogen dioxide concentrations of 2.8 to 3.8 mg/m^3 (1.5 to 2.0 ppm). Von Nieding and Rokaw were unable to detect any effect on airway resistance from exposures less than 2.8 mg/m^3 (1.5 ppm). Although changes in airway resistance were reversible, these manifestations of nitrogen dioxide exposure are potentially adverse, particularly for hypersensitive asthmatics or subjects with advanced chronic obstructive pulmonary disease. Measurements of potentially more sensitive indicators of pulmonary dysfunction, such as small airway resistance and perfusion indices, have not been reported. In one study, sodium chloride aerosol combined with nitrogen dioxide gas augmented the effect on airway resistance when compared with exposure to nitrogen dioxide alone.

TABLE 10-20

Summary of Human Responses to Short-Term Nitrogen Dioxide Exposures Alone

Effect	NO_2 Concentration mg/m^3	(ppm)	Time to Effect	Reference
Odor threshold	0.23	(0.12)	Immediate	224
	0.23	(0.12)	Immediate	476
Threshold for dark adaptation	0.14	(0.075)	Not reported	476
	0.50	(0.26)	Not reported	51
Increased airway resistance	1.3-3.8	(0.7-2.0)	20 min[a]	523
	3.0-3.8	(1.6-2.0)	15 min	583-585
	2.8	(1.5)	45 min[b]	447
	3.8	(2.0)	45 min[c]	447
	5.6	(3.0)	45 min[d]	447
	7.5-9.4	(4.0-5.0)	40 min[e]	1
	9.4	(5.0)	15 min	520
	11.3-75.2	(6.0-40.0)	5 min	384
	13.2-31.8	(7.0-17.0)	10 min[f]	613-616
Decreased pulmonary diffusing capacity	7.5-9.4	(4.0-5.0)	15 min	583
Increased alveolar-arterial $\underline{p}O_2$ difference	9.4	(5.0)	25 min[g]	584
No change in sputum histamine concentration	0.9-6.6	(0.5-3.0)	45 min	532

[a]Exposure lasted 10 min. Effect on flow resistance was observed 10 min after termination of exposure.

[b]Effect was produced at this concentration when normal subjects and those with chronic respiratory disease exercised during exposure.

[c]Effect occurred at rest in subjects with chronic respiratory disease.

[d]Effect occurred at rest in normal subjects.

[e]Exposure lasted 10 min. Maximal effect on flow resistance was observed 30 min later.

[f]Also failed to find increased flow resistance over the range of nitrogen dioxide exposures from 5.1 to 30.1 mg/m^3 (2.7-16.0 ppm).

[g]Effect occurred 10 min after termination of 15-min exposure.

An accidental exposure of persons to very high nitrogen dioxide concentrations established the fatality level from acute exposures at 282 mg/m^3 (150 ppm) and above. These high concentrations caused pulmonary edema or bronchiolitis fibrosa obliterans which resulted in death. Concentrations between 47 and 140 mg/m^3 (25 to 75 ppm) produced reversible pneumonia and bronchiolitis. Permanent sequelae, resulting in shortened lifespan, were not found in the only reported follow-up study of survivors of acute high level nitrogen dioxide exposure.

The literature contains little data on the health hazards of repeated nitrogen dioxide exposures of 2- to 4-hr duration, a common occurrence in the ambient environment. The few studies on exposures of human volunteers indicate an increase in airway resistance at nitrogen dioxide concentrations of 2.8 to 3.8 mg/m^3 (1.5 to 2.0 ppm) for 15 to 45 min. In order to make the results of these studies applicable to air quality standards, these studies of effects should be conducted in atmospheres that contain combinations of pollutants common to urban environments. Nitrogen dioxide is often present with sulfur dioxide, small amounts of ozone, and respirable sulfate and nitrate aerosols, at both low and high humidities. The interaction or synergy between ozone and sulfur dioxide on human airway resistance has been well demonstrated. Similar interactions between nitrogen dioxide and other atmospheric pollutants must also be investigated. Until this is done, we cannot be confident that short-term ambient exposures of less than 2.8 mg/m^3 (1.5 ppm) nitrogen dioxide have no effect on airway resistance.

Clearly, other potential manifestations of short-term exposures of humans to peak ambient nitrogen dioxide concentrations must be investigated. As a first step, we need more extensive studies of impaired

pulmonary defense mechanisms in animals caused by intermittent short-term exposures to nitrogen dioxide alone and in combination with other atmospheric pollutants. The importance of this effect of nitrogen dioxide justifies the extensive research called for in these considerations. Table 10-20 summarizes the sensory and pulmonary effects of human exposure to nitrogen dioxide.

Effect of Prolonged Exposure on Lung Function

Two epidemiological studies suggest that the combination of nitrogen dioxide at concentrations of 0.15 to 0.3 mg/m^3 (0.08 to 0.16 ppm) with other pollutants causes changes in ventilatory function. Two other studies in which lower levels of nitrogen dioxide were studied did not reveal these effects. Because of the disparity in populations and in pollutant conditions, conclusions cannot be reached regarding the effect, if any, of chronic exposure to nitrogen dioxide on ventilatory function.

Effects on Acute Respiratory Disease in Populations

Some epidemiological data support the idea that excess acute respiratory disease may occur in healthy populations following exposure to atmospheres containing nitrogen dioxide. Four studies have been reviewed in the search for an association between exposure to ambient concentrations of nitrogen dioxide from 0.10 to 0.58 mg/m^3 (0.053 to 0.309 ppm) and small excesses in respiratory illnesses. However, the variable pollutant exposures and conditions of study make it difficult to quantify the relationship of nitrogen dioxide by itself to the reported increases in respiratory disease. In each study air contaminants likely to enhance susceptibility to respiratory infection (sulfur dioxide, sulfuric acid, sulfates, nitrates, etc.) were also present.

Effects on Chronic Respiratory Disease in Populations

Evidence that nitrogen dioxide induces excess chronic respiratory disease is not convincing. Reports of excess chronic respiratory disease associated with low concentrations of ambient nitrogen dioxide [less than 0.10 mg/m^3 (0.053 ppm)] do not provide convincing evidence that other pollutants which were measured at relatively high concentrations were not the probable cause of the excess disease. In the presence of low concentrations of sulfur dioxide and particulates, three investigators failed to detect excess chronic respiratory disease in areas where nitrogen dioxide exposures were at or below 0.10 mg/m^3 (0.053 ppm).

Acute Toxicity to Humans

High level nitrogen dioxide exposures of small occupational groups establishes the fatal level of nitrogen dioxide as 282 mg/m^3 (150 ppm) and above. These deaths resulted from pulmonary edema or bronchiolitis fibrosa obliterans. Concentrations between 47 and 140 mg/m^3 (25 and 75 ppm) cause reversible pneumonia and bronchiolitis. Permanent sequelae manifested as shortening of survival time were not found in the only long-term follow-up of survivors of acute high-level exposure.

CHAPTER 11

SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS FOR FUTURE RESEARCH

PROPERTIES OF THE NITROGEN OXIDES AND THEIR PHYSICIAL EFFECTS ON ATMOSPHERIC LIGHT TRANSMISSION

Among the various oxides of nitrogen present in polluted atmospheres, nitric oxide (NO) and nitrogen dioxide (NO_2), designated by the composite formula NO_x, are the most important in relation to chemical and photochemical changes. A major source of these oxides is fuel combustion. Nitric oxide is the dominant nitrogen oxide formed in combustion. A fraction of the nitric oxide is converted to nitrogen dioxide by reaction with oxygen during the exhaust dilution process; however, the major pathway leading to formation of nitrogen dioxide from nitric oxide is the photochemical interaction between NO_x, hydrocarbons, and various other compounds and intermediate free radicals that are generated in the sunlight-irradiated polluted atmosphere.

The degree to which nitrogen dioxide causes reduction of visibility and coloration of the horizon sky is directly dependent on the concentration of the pollutant, the viewing distance, and the accompanying aerosol concentration. The presence of photochemical aerosol or other particulate matter diminishes the coloration effect of nitrogen dioxide and further decreases visibility.

SOURCES AND CONTROL OF ATMOSPHERIC NITROGEN OXIDES

Annual global emissions of nitrogen oxides from manmade sources are substantially less than from natural sources; however, manmade sources

play a very significant role in atmospheric pollution on a local level. The principal manmade source of nitrogen oxides is combustion. In this category, motor vehicle and fossil-fueled electric power generating stations are the most significant. Although industrial process losses contribute only a small amount to the total manmade nitrogen oxide emissions, they can be important sources in a local, industrialized area.

Existing methods for obtaining emission inventories have limited use in evaluating the importance of specific categories of local sources of nitrogen oxides since these methods generally involve inventories of large geographic areas over extended periods. Furthermore, differences between actual nitrogen oxide emissions from specific sources and tabulated emission factors can result in significant inaccuracies in emission inventories. Source inventories should discriminate between nitric oxide and nitrogen dioxide emissions since each of these oxides interacts with the atmospheric photolytic cycle in a different way.

The two principal sources of nitrogen oxides produced in combustion are the oxidation of atmospheric (molecular) nitrogen and oxidation of nitrogen compounds in the fuel (fuel nitrogen). Nitric oxide is produced from atmospheric nitrogen in the high temperature regions in the combustion chamber, whereas fuel nitrogen can be converted to nitric oxide at lower temperatures and can be a major source of nitrogen oxide emissions in some combustion devices.

There are two basic approaches to control NO_x emissions from combustion sources: modification of the combustion process and treatment of exhaust gases. Present understanding of the principles of nitric oxide formation in combustion is sufficient to permit development of techniques for reducing NO_x emissions from combustion sources. However, implementation of these

techniques may be limited by excessive cost, losses in combustion efficiency, and by a number of operational problems in the combustion chamber.

Recommendations:

1. Improved inventories of nitrogen oxide emissions are needed to estimate the relative importance of various sources in specific geographic locations and to assist in the evaluation of control strategies.
2. Potential techniques for reducing nitrogen oxide emissions from combustion sources should be evaluated. The implementation costs and the extent to which atmospheric levels of nitrogen oxides are reduced should be estimated. Techniques requiring evaluation include: two-stage combustion, catalytic combustion, combustion of emulsified fuels and fuel blends, combustion of prevaporized liquid fuels, combustion of gasified coal, and combustion of synthesized fuels.
3. Fuel processing methods that can reduce the nitrogen content of the fuel before combustion should be considered as a method to control nitric oxide emissions from sources burning nitrogen-containing fuels.

ANALYTICAL METHODOLOGY FOR THE DETERMINATION OF NITROGEN OXIDES IN AIR

Older chemical methods used to measure nitrogen oxides were based on the transfer of the nitrogen from the nitrogen dioxide molecule directly into a measurable compound. This method and its variations have generated most of the data over the last 40 years and are still in use. The manual Griess-Saltzman Method (not to be confused with the Griess-Saltzman reagent

or principle) for calibrating continuous analyzers is based on the direct reaction of nitrogen dioxide with a single reagent mixture to form a colored azo dye. The Griess-Saltzman reagent and several modifications thereof are the basis for many continuous analyzers. Nitric oxide cannot be measured directly with this technique; it must be oxidized to nitrogen dioxide with an appropriate catalyst.

Chemiluminescence techniques are based on optical measurements of light emitted by the reaction of two gases. In one method, nitric oxide is measured through its reaction with ozone within the chemiluminescent instrument. To measure nitrogen dioxide, a prior reduction step to nitric oxide over a heated catalyst is required. Another method involves the photofragmentation of nitrogen dioxide by ultraviolet light and chemiluminescent measurement of the ozone produced. This last approach avoids contact with a catalyst but requires nitric oxide as a reagent. For calibration of the chemiluminescent methods, precisely known standards containing appropriate concentrations of nitric oxide or nitrogen dioxide are used. These concentrations can be obtained by dynamic dilution of more concentrated gas mixtures or with permeation tubes having precisely known leaking rates. By contrast, most wet chemical methods use an alkaline nitrite or a nitrate as primary standards.

Alkaline methodology for sampling and measuring nitrogen dioxide has been criticized. The departure from original sampling conditions and absorbing reagent formulations, and the assumption of constant performance generated equivocal results. A variety of new methods (collectively called Jacobs-Hochheiser methods) were developed but not evaluated.
In these methods, nitrite in absorbing solutions is analyzed by applying

the Griess principle of azo dye formation. More recent methods without these shortcomings have been evaluated, including the 24-hr arsenite method.

Most methods in which nitrogen oxides are transformed into nitrate are suited for higher than atmospheric levels of nitrogen oxides. These higher levels can also be analyzed with the same methods used in atmospheric analysis after proper aliquoting or dilution.

Recommendations:

1. The manual Griess-Saltzman method is recommended as the primary reference method. All details of this method must be strictly followed. The recommended primary standard for calibration is sodium nitrite.
2. The continuous Saltzman method and the differential chemiluminescence method are suggested as secondary reference methods. Chemiluminescence requires careful determination of the efficiency of the nitrogen dioxide converter. Calibration of these methods with permeation tubes instead of secondary standards changes their category to primary reference methods.
3. Nitric oxide used as a secondary standard should be calibrated using one of the recommended primary reference methods rather than the gas-phase titration procedure.
4. Devices for continuous monitoring of particulate nitrates and gas-phase nitric acids should be developed.

ATMOSPHERIC LEVELS OF NITROGEN OXIDES

While there has been an appreciable loss of data due to questionable accuracy of certain methods used to measure nitric oxide and NO_x, sufficient accredited data exist to give strong support to the following conclusions:

1. The current national primary air quality standard for nitrogen dioxide is 0.1 mg/m^3 (0.05 ppm) annual average. This concentration is consistently exceeded in the Los Angeles area and, if present trends continue, will soon be exceeded in the Chicago and Philadelphia areas. Maximum 1-hr concentrations of several milligrams per cubic meter may be recorded without exceeding the existing primary air quality standard. In fact, 1-hr maximum concentrations of 5 and 9 mg/m^3 have been recorded in Chicago and Philadelphia, respectively.
2. Highest average concentrations of oxides of nitrogen and nitrates are found in heavily populated, industrialized urban areas. These concentrations show a generally increasing trend for the 10-year period between 1962 and 1971.
3. Physicochemical models of pollutant distribution, based on analytical data, are of value in predicting maximum concentration values of NO_x that will be reached under a given set of conditions in different locations. Data from these models can be used to give warning of impending "alert" conditions.
4. Relationships between nitrogen dioxide and nitrate concentrations, and the types of nitrate formed in different areas, are under study because of the potential health significance of atmospheric nitrates.
5. Because of current concern with pollution of the stratosphere, nitric oxide and nitrogen dioxide concentrations have recently been determined in that region of the atmosphere. Nitric oxide in the stratosphere is thought to result from oxidation of nitrous

oxide (N_2O). The concentration of nitrogen dioxide in the stratosphere has been estimated at 0.20 ppm. Changes in these levels are anticipated as high altitude air traffic becomes more common.

Recommendations:

1. Augment existing data on the atmospheric concentration of nitric oxide, nitrogen dioxide, and nitrates (NO_3^-). Studies should be made on the chemical interaction of these species when they are contained on discrete colloidal atmospheric particles (mineral, carbon, organic liquid, and aqueous) incorporating the effects of particle size and composition.
2. Physicochemical models used in predicting maximum concentration values of NO_x should be expanded to include information obtained from continuous monitoring in regions of high NO_x concentrations. These data should include criteria for "alert" conditions in the warning system.
3. Stratospheric data on nitric oxide, nitrogen dioxide, and nitrous oxide concentrations are incomplete. Chemical interactions occurring in this region are not precisely known and additional study is required. Effects of stratospheric pollutants at the surface of the earth require further investigation.
4. The relationship between nitrogen dioxide and nitrate concentrations under various meteorologic and geographic conditions requires further study. The relative health hazard of nitrogen dioxide and nitrates, and the synergistic effects between these pollutants and sulfates in the atmosphere, need further evaluation.

CHEMICAL INTERACTIONS OF NITROGEN OXIDES IN THE ATMOSPHERE

Solar radiation induces a number of reactions in the atmosphere between gaseous organic molecules and nitric oxide thereby producing a variety of so-called secondary pollutants. These secondary pollutants are present in extremely small concentrations and many are very transient. Because of these two factors, it is very difficult to identify or measure these pollutants by conventional analytical techniques.

Starting with simple hydrocarbons and nitric oxide, attempts have been made to identify intermediate and end products of the photochemical reactions by relating laboratory studies involving chemical and kinetic modeling to atmospheric observation. Because certain key information is still lacking, it is not yet possible to make quantitative predictions. Enough information is available, however, to indicate that the main features of the model are correct, placing considerable reliability on the qualitative predictions.

Based on calculated concentrations and theoretical first half-lives, it is possible to predict photochemical intermediates which might persist into the respiratory system. The health significance of these must still be determined.

It does not appear possible to set numerical standards on transient secondary pollutants; but, based on available information, some relationships can be used to develop air quality control planning.

The extent to which nitric and nitrous acids and nitrate and nitrite (NO_2^-) salts result from gaseous nitrogen dioxide and nitric oxide absorption into aerosol droplets is not known. It is known in general that the rate of absorption of acidic gases is a function of the partial pressure of the

gases, the rate gases diffuse into solution, the pH of the solution, and the interfacial area. However, these factors must all be evaluated regarding nitrate salt formation in smog and related potential health problems.

Recommendations:

1. The health significance of such photochemical intermediates as the hydroxyl radical (OH), the hydroperoxyl radical (HO_2), and symmetrical nitrogen trioxide (NO_3), which can exist in typical polluted atmospheres long enough to be transported to the respiratory system, should be determined. These intermediates cannot be studied independently. Rather, they should be produced in a reaction vessel as an array of intermediates. When conclusive information on these health aspects becomes available, numerical standards on these intermediates should be set.
2. Kinetic studies of possible photochemical reactions that can be induced by sunlight within the tropospheric region should be continued. The inhibiting effect of very high nitric oxide concentrations on product rates and eye irritation should also be studied further.
3. More detailed information concerning turbulent diffusion, local emissions, and solar zenith angle changes as well as chemical reactions must be included in the development of useful simulations of actual urban atmospheric reactions.
4. The methods of nitrate salt formation in the atmosphere in both homogeneous and heterogeneous reactions, primarily in gas-gaseous liquid phase reactions, should be further investigated. Meteorologic and geographic aspects should be considered.

EFFECTS OF NITROGEN OXIDES ON NATURAL ECOSYSTEMS

No specific information is available on the effects of nitrogen oxides on animals in ecosystems. Research on crop plants indicates that NO_x should have effects similar to other air pollutants on some plant communities. Consequently, we anticipate: differential species sensitivity to NO_x; complications due to synergistic or antagonistic interactions between NO_x, other air pollutants, and natural environmental stresses; and secondary ecosystem responses due to changing symbiotic and competitive interactions.

Nitric oxide, nitrogen dioxide, and nitrous oxide affect the growth or survival of individual microorganisms when tested at high concentrations in defined media, but the effect on microorganisms or microbial processes of nitrogen oxides at the levels naturally found in the atmosphere are unknown. The effects of ambient NO_x concentrations on populations or activities in both natural habitats and *in vitro* have not been studied. Although little suppression of heterotrophs is caused from the presence of these gases, a meaningful conclusion is not possible in the absence of direct experimentation.

The algae that are extremely important to primary production in fresh and marine waters, the algae and lichens that are significant in the weathering of rocks and in certain soil processes, and the activity of microorganisms colonizing leaves and causing plant disease are affected by low concentrations of such air pollutants as sulfur dioxide (SO_2), hydrogen fluoride (HF), ozone (O_3), and cement kiln dust, but their sensitivity to NO_x has yet to be tested.

Both nitric oxide and nitrogen dioxide react readily with soils, and are generally converted to nitrate. Sorption of large amounts of NO_x

decreases soil pH which presumably can be corrected by the addition of lime. The scavenging role of soil in removing typical atmospheric concentrations of NO_x is uncertain.

Recommendations:

1. It should be determined whether ambient atmospheric concentrations of nitrogen oxides affect plant and animal communities, and microorganisms and microbial processes in soils and water.
2. In addition, the efficacy of soil as a sink for atmospheric nitrogen dioxide should be ascertained.

EFFECTS OF NITROGEN OXIDES ON MATERIALS

Field studies and laboratory research have demonstrated that ambient concentrations of nitrogen dioxide, as well as sulfur dioxide and ozone, can cause fading of certain textile dyes and yellowing of some white fabrics. The chemical mechanisms for nitrogen dioxide fading of dyes are fairly well-known, and various methods for color protection are available. The cost to the consumer of color fading of dyes by nitrogen dioxide has been estimated to exceed $100 million annually. The effect of pollutants on yellowing of white fabrics has not been well-established; however, recent studies suggest that nitrogen dioxide is the pollutant principally responsible for this problem. No estimates of the cost to the consumer of yellowing of white fabrics are available.

Available data do not indicate a direct role for nitrogen oxides in the degradation of textile fibers. If nitrogen oxides increase the concentration of airborne acids, this might lead to degradation of cotton fabrics and nylon.

Evidence suggests that nitrogen oxides can affect the defect structure of metal oxides thereby increasing or decreasing the rates of oxidation of metals and alloys. Furthermore, airborne nitrates can adsorb water. This aids the formation of a solvent or electrolyte for wet corrosion. However, a direct relationship has not been established between a given nitrogen oxide concentration and a change in the corrosion behavior of structural materials.

Recommendations:

Further studies are needed to determine the mode of action of NO_x and nitrates on the degradation of textile fibers and rubber compounds, and on the corrosion of metals and alloys.

EFFECTS OF NITROGEN OXIDES ON VEGETATION

Experimental fumigations have shown that nitric oxide is less injurious to vegetation than nitrogen dioxide, and both are less phytotoxic than sulfur dioxide, gaseous fluoride, or photochemical oxidants. Nitrogen dioxide-induced injury in the field has usually been associated with accidental acute exposures near industries that manufacture or use nitric acid. There have been no confirmed reports for nitric oxide injury to vegetation in the field.

The existing U.S. air quality standard for nitrogen dioxide (0.10 mg/m^3 for an annual average) is below the threshold for detectable effects on vegetation. Therefore, secondary standards to protect vegetation from the direct effects of nitrogen dioxide are not necessary. Indirect effects of nitrogen dioxide on vegetation, however, are more important. The participation of nitrogen dioxide in atmospheric reactions leading to the

production of ozone and peroxyacylnitrates (PANs), and the synergistic effects on plant injury of low concentrations of nitrogen dioxide and sulfur dioxide mixtures in the atmosphere may pose a real threat to vegetation growing in or near metropolitan areas. As knowledge of these phenomena increases, air quality standards for nitrogen dioxide to protect vegetation may have to be reevaluated.

Recommendations:

1. Since plants are relatively insensitive to nitric oxide, future research on NO_x should concentrate on nitrogen dioxide. The gross effects of acute nitrogen dioxide exposures have been fairly well documented. Future research, therefore, should emphasize the effects of chronic and intermittent nitrogen dioxide exposures, at realistic concentrations and exposure times, on plant metabolism, growth, and yield.
2. The combined effects of NO_x and other substances in the atmosphere on plants is another important area in which information is limited. Experimental exposures to nitrogen dioxide in combination with other atmospheric constituents should be conducted on a wide variety of plant species to reveal the interacting effects of nitrogen dioxide and other pollutants on phytotoxicity.

HEALTH EFFECTS OF NITROGEN OXIDES

Effects of Short-Term Exposures (Less Than 24-hr)

Reduced resistance to respiratory infection is the most sensitive response of animals to nitrogen dioxide exposure. Significant differences in mortality from experimentally induced bacterial pneumonia were observed

in mice after a 2-hr exposure to 6.6 mg/m^3 (3.5 ppm) nitrogen dioxide. This impairment was also observed in other animal species including hamsters and nonhuman primates. The action of nitrogen dioxide appears to be mediated through alterations of specific defense mechanisms including the alveolar macrophage system, anatomical structure of lungs, and humoral immunity.

Other effects of short-term animal exposure include various physiologic and pathologic abnormalities caused by exposures to 9.4 mg/m^3 (5 ppm) nitrogen dioxide and above, and mortality which resulted when concentrations of nitrogen dioxide exceeded 75-95 mg/m^3 (40-50 ppm).

Human volunteer studies have provided precise documentation of the acute effect of nitrogen dioxide. The earliest response to nitrogen dioxide occurs in the sense organs. The odor of nitrogen dioxide can be perceived upon exposure to 0.23 mg/m^3 (0.12 ppm) nitrogen dioxide, while changes in dark adaptation were reported following exposures ranging from 0.14 to 0.50 mg/m^3 (0.075 to 0.26 ppm). These effects are immediately reversible, and there is no evidence for sequelae in terms of pathology.

Three studies have shown increases in airway resistance after 15 to 45 min exposures to 2.8 to 3.8 mg/m^3 (1.5 to 2.0 ppm) nitrogen dioxide. Although the increases in airway resistance were reversible, they may be adverse for asthmatics or subjects with advanced chronic obstructive pulmonary disease. Measurements of more sensitive parameters of pulmonary dysfunction, such as small airway resistance and perfusion indices, have not been reported.

The literature contains a paucity of data on the health effects of repeated short-term (2 to 4 hr) exposures to nitrogen dioxide, or exposures to nitrogen dioxide in mixtures of pollutants that commonly occur in the

environment. In one study, sodium chloride aerosol combined with nitrogen dioxide augmented the effect on airway resistance when compared with exposure to nitrogen dioxide alone. The synergy between ozone and sulfur dioxide on human airway resistance has also been reported. Therefore, potential interactions between nitrogen dioxide and other atmospheric pollutants must be investigated before it can be stated with confidence that short-term exposures to less than 2.8 mg/m^3 (1.5 ppm) nitrogen dioxide present in a mixture of other air pollutants have no effect on airway resistance.

Accidental exposures establish the acutely fatal concentration of nitrogen dioxide for man at 282 mg/m^3 (150 ppm) and above. Deaths were due to pulmonary edema or bronchiolitis fibrosa obliterans. Concentrations between 47 and 140 mg/m^3 (25 to 75 ppm) caused reversible pneumonia and bronchiolitis. Permanent sequelae manifested as shortening of life were not found in the only reported follow-up study of survivors of acute exposures to high nitrogen dioxide concentrations.

Chronic Effects of Long-Term Nitrogen Dioxide Exposure

As in short-term exposure studies, reduced resistance to respiratory infection appears to be the most sensitive indicator of damage produced by long-term nitrogen dioxide exposure. Continuous exposures for 3 months and intermittent daily exposures for 6 months or longer to nitrogen dioxide at concentrations of 0.94 mg/m^3 (0.5 ppm) and higher diminishes murine ability to resistant pulmonary bacterial infection. At 9.4 mg/m^3 (5.0 ppm) nitrogen dioxide, similar results have been reported for nonhuman primates after a 2 months exposure. The reduced resistance to infection has been associated with pneumonia and death. Immunologic deficits

unassociated with overt infection have been observed after prolonged exposures to 1.8 mg/m^3 (1.0 ppm). Such pathological abnormalities as ciliary loss, alveolar cell disruption, and obstruction of respiratory bronchioles, occur in lungs of mice and rats after continuous exposure to 0.94 mg/m^3 (0.5 ppm) of nitrogen dioxide.

Long-term exposures to nitrogen dioxide at concentrations of 9.4 mg/m^3 (5.0 ppm) or higher also cause rapid breathing and increases in airway resistance in rodents and in nonhuman primates. A more serious abnormality in pulmonary function, namely decreases in blood oxygenation, occurs in rabbits after continuous exposure to 15 mg/m^3 (8.0 ppm).

Limited studies were reported in which animals have been exposed to mixtures of pollutants including nitrogen dioxide. Combinations of nitrogen dioxide with carbon monoxide, ozone, or sulfur dioxide have usually resulted in additive or indifferent effects. Synergy has, with one exception of uncertain significance, not been reported, nor, to our knowledge, has antagonism.

Epidemiological studies also indicate that excess acute respiratory disease was observed in healthy populations following exposure to nitrogen dioxide. Considered broadly, four studies are consistent in suggesting an association between exposures of 0.10 to 0.58 mg/m^3 (0.053 to 0.31 ppm) nitrogen dioxide and excesses in respiratory illnesses. However, the variable pollutant exposures and conditions of study make it difficult to quantify the direct relationship between nitrogen dioxide and the increases in respiratory disease. In each of these studies other air pollutants likely to enhance susceptibility to respiratory infection (sulfur dioxide, sulfuric acid, sulfates, nitrates, etc.) were also present.

Results of two epidemiological studies in which ventilatory function was measured suggest that 0.15 to 0.3 mg/m^3 (0.08 to 0.16 ppm) nitrogen dioxide in combination with other pollutants causes changes in ventilatory function. These effects were not reported in two other studies in which nitrogen dioxide concentrations were less than 0.15 mg/m^3 (0.08 ppm) and in which a mixed pollutant atmosphere existed. The effective concentration at which ventilatory function is impaired more than likely is not a function of chronic exposure to nitrogen dioxide alone, but the combination of pollutants and other atmospheric conditions most of which were not monitored.

Reports of excess chronic respiratory disease associated with ambient concentrations of nitrogen dioxide do not provide convincing evidence that other pollutants, measured at high concentrations, did not cause the excess disease prevalence. At low concentrations of sulfur dioxide and particulates, three investigators failed to detect excess chronic respiratory disease in populations where nitrogen dioxide exposures were at or below 0.10 mg/m^3 (0.053 ppm).

While urban air pollution may be related to chronic human diseases, including cancer, a correlation with oxides of nitrogen in the atmosphere has not been established. Tumors were not observed in rats after lifetime exposure to 1.5 to 3.8 mg/m^3 (0.8 to 2.0 ppm) nitrogen dioxide although extensive lung disease occurred and epithelial hyperplasia was observed in the trachea and bronchi of these animals. Although evidence from man and animals is limited, possible participation of nitrogen oxides or their reaction products (organic and inorganic nitrates, nitrites, and possible nitroso compounds) in carcinogenesis merits attention.

Recommendations:

1. Lung function response should be studied in human volunteers to determine the potentially important health effect of repeated 2 to 3 hr nitrogen dioxide exposures. Such studies should be designed to establish the highest no-effect concentration of nitrogen dioxide. Resulting data would be directly relevant to the establishment of a short-term nitrogen dioxide standard. Several experimental conditions should be taken into account when designing these studies:

   - Lung function should be evaluated during exercise since the importance of intermittent light exercise during exposure to pollutants has been well demonstrated.
   - Lung function tests should include measurements sensitive to small airway changes, as well as the more traditional measures of total airway resistance and flow or volume. The effect of nitrogen dioxide on distal bronchioles should be studied because of the morphological evidence of damage in the terminal bronchioles and alveoli observed in laboratory animals.
   - To simulate ambient environmental conditions, the effect of repeated short-term exposures to nitrogen dioxide alone, and to nitrogen dioxide in combination with sulfur dioxide, ozone, and particulate aerosols at low and high humidity should be determined.
   - Consideration should be given to studies of human volunteers with potential high sensitivity to short-term peak nitrogen dioxide exposures. These subjects might include asthmathics and individuals with advanced chronic obstructive lung disease.

- Studies using human volunteers should be designed to establish the highest no-effect concentration of nitrogen dioxide.

2. Studies should further define the causal relationship between nitrogen dioxide and acute respiratory infections in laboratory animals. To simulate more closely true environmental air pollution situations, such studies must include mixtures of gaseous and particulate pollutants at various temperature and humidity conditions. Moreover, long-term exposures should be conducted at low concentrations of nitrogen dioxide with superimposed short-duration peaks of higher nitrogen dioxide concentrations.

   The sensitivity to nitrogen dioxide of defense mechanisms against respiratory infections, e.g., phagocytosis, cellular, and humoral immunity, should be investigated in several animal species, including nonhuman primates. Whenever possible, the biological response endpoints used in such studies should be relevant and of potential use to human epidemiological surveys.

3. The relationship between long-term nitrogen dioxide exposure and chronic respiratory disease should be carefully evaluated. Future epidemiologic surveys of chronic respiratory disease should take into account history of past exposures, indoor as well as ambient sources of nitrogen dioxide, and presence of other pollutants commonly encountered in the urban atmosphere. It may be necessary to take advantage of special occupational situations in which relatively isolated nitrogen dioxide exposures may be encountered. In addition, animal models of chronic obstructive respiratory disease should be used to determine the effects of repeated intermittent peak nitrogen dioxide exposures.

4. Since respirable solid and liquid aerosol particles carrying nitrogen oxides in condensed, dissolved, or adsorbed form are deposited in the lungs, the composition of the aerosols and the inhaled dose, deposition patterns, and pathological effects resulting therefrom require further investigation.
5. To quantify individual human exposures similar to those measured in animal dose-response studies, characteristic responses of population cohorts to air pollutant mixtures require evaluation. Pilot studies should compare actual total exposures for workers in industrial high exposure settings with groups exposed to lower concentrations. This would provide preliminary data concerning the range of individual exposures in different segments of the population. Such studies will complement the available environmental data that document large diurnal and seasonal variations in ambient nitrogen dioxide concentrations. Exposure doses calculated only from ambient air concentrations do not provide realistic estimates of actual human exposure times, particularly because of the extensive movement into and out of polluted areas by any individual on any given day.
6. The carcinogenic potential of airborne oxides of nitrogen or their reaction products should be considered. In particular, studies should be conducted on carcinogenic effects of organic nitrogen compounds in air and of nitrogen dioxide in the presence of other pollutants.
7. Wherever possible, biological studies of nitrogen dioxide effects should be made on a number of exposure levels sufficient to permit a preliminary estimate of thresholds and the slope of the biological response.

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16. ABSTRACT

This report is a review of current knowledge of the environmental health basis for control of manmade sources of nitrogen oxide emissions. The literature review covered the period through 1974. The principal subject areas considered in the report include: sources and control of atmospheric nitrogen oxides; analytical methodology; concentrations and chemical reactions in the atmosphere; and the effects of nitrogen oxides on human health, materials, vegetation, light transmission, and natural ecosystems. Emphasis is primarily on nitroc oxide (NO) and nitrogen dioxide (NO_2), designated by the composite formula NO_x for nitrogen oxides. The major manmade source is the combustion of fossil fuel. Highest atmospheric concentrations are found in heavily populated, industrialized urban areas. Both acute and chronic health effects resulting from short-term and long-term exposures, are discussed in the report. Effects range from slight increases in airway resistance to death depending upon exposure concentrations.

17. KEY WORDS AND DOCUMENT ANALYSIS

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