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Air



**REVIEW OF THE NATIONAL AMBIENT AIR QUALITY STANDARDS
FOR NITROGEN DIOXIDE
ASSESSMENT OF SCIENTIFIC AND TECHNICAL INFORMATION**

OAQPS STAFF PAPER

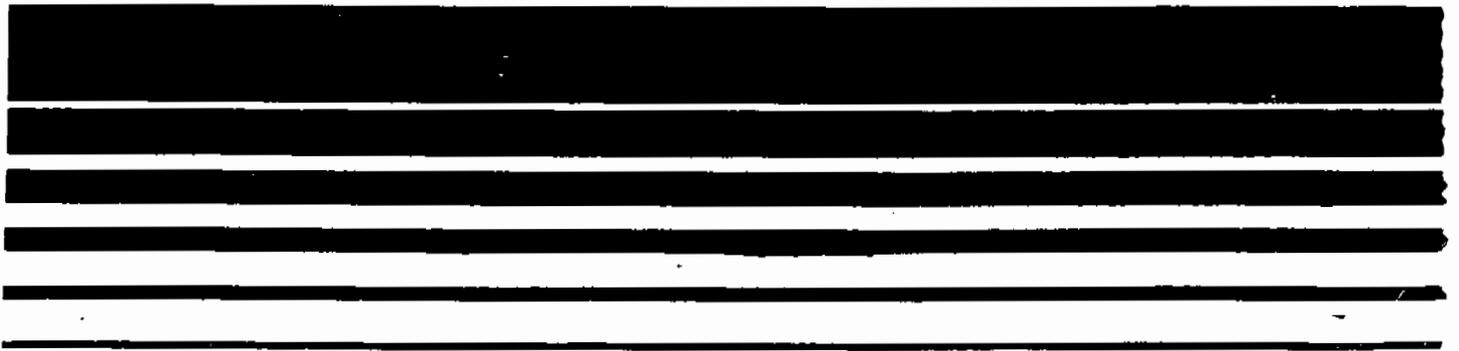


TABLE OF CONTENTS

Acknowledgements	i
List of Tables	iv
List of Figures	v
Executive Summary	vi
I. Purpose	1
II. Background	1
A. Legislative Requirements	1
B. Establishment of Nitrogen Dioxide NAAQS	3
C. First Review of Nitrogen Dioxide NAAQS	4
D. Current Review of Nitrogen Dioxide NAAQS	5
III. Approach	5
IV. Air Quality Trends	6
A. Long-Term Trends	7
B. Recent Trends in Urban Areas of the U.S.	9
C. Nitrogen Deposition Trends in the U.S.	10
V. Health Effects of Nitrogen Dioxide	13
A. Introduction	13
B. Mechanisms of Toxicity, Transport, and Fate	13
C. Health Effects Evidence	15
1. Susceptibility to Respiratory Illness	15
a. Epidemiological Studies	16
1. Indoor Studies	17
2. Outdoor Studies	22
b. Meta-analysis of Gas Stove Studies	24
c. Biological Plausibility	28
2. Lung Function, Symptoms and Airway Resistance	33
3. Increased Airway Responsiveness	36
4. Emphysema	38
D. Populations Potentially at Risk	39
E. Effects of Concern	43
VI. Averaging Time and Form of the Primary NAAQS	46
A. Averaging Time of the Primary NAAQS	46
B. Form of the Primary NAAQS	48
VII. Summary of Staff Conclusions and Recommendations for the Nitrogen Dioxide Primary NAAQS	49

VIII. Environmental Effects of Nitrogen Deposition	52
A. Introduction	52
1. Context of Current Standard Review	52
2. Atmospheric Nitrogen Inputs	53
3. Nitrogen Cycle	55
B. Ecosystem Response	57
1. Terrestrial Systems	58
a. Vegetation	58
b. Plant/Soil Interactions	60
c. Soils	62
2. Wetlands	65
a. Vegetation	66
b. Soils	68
3. Aquatic Systems	68
a. Acidification	69
1. Chronic Acidification	69
2. Episodic Acidification	71
b. Eutrophication	74
1. Freshwater Eutrophication	74
2. Estuaries and Coastal Waters	76
c. Direct Toxicity	77
C. Critical Loads and Other Potential Regulatory Actions	78
1. Critical Loads Concept	78
2. Target Loads	85
3. Applicability to U.S. Situation	85
D. Visibility	86
1. Major Categories	86
2. Contributors to Visibility Reduction	87
E. Non-biological Materials	90
IX. Summary of Staff Conclusions and Recommendations for Secondary Nitrogen Dioxide NAAQS	91
Appendix A: CASAC Correspondence	
Appendix B: Analysis of Current NO ₂ NAAQS Attainment	
Appendix C: Definitions	
Appendix D: References	

List of Tables

V-1	Health outcome and nitrogen dioxide exposure measures used in selected indoor nitrogen dioxide epidemiology studies	20
V-2	Summary of odds ratios from indoor studies of the effects of nitrogen dioxide	25
V-3	Key epidemiological studies of exposure to nitrogen dioxide - Summary of meta-analysis	27
V-4	Key animal toxicological effects of exposure to nitrogen dioxide	32
V-5	Summary of potentially sensitive groups	42
VIII-1	Measurements of various forms of annual nitrogen deposition to North American and European ecosystems	81
VIII-2	Nitrogen input/output relationships for several ecosystems	83

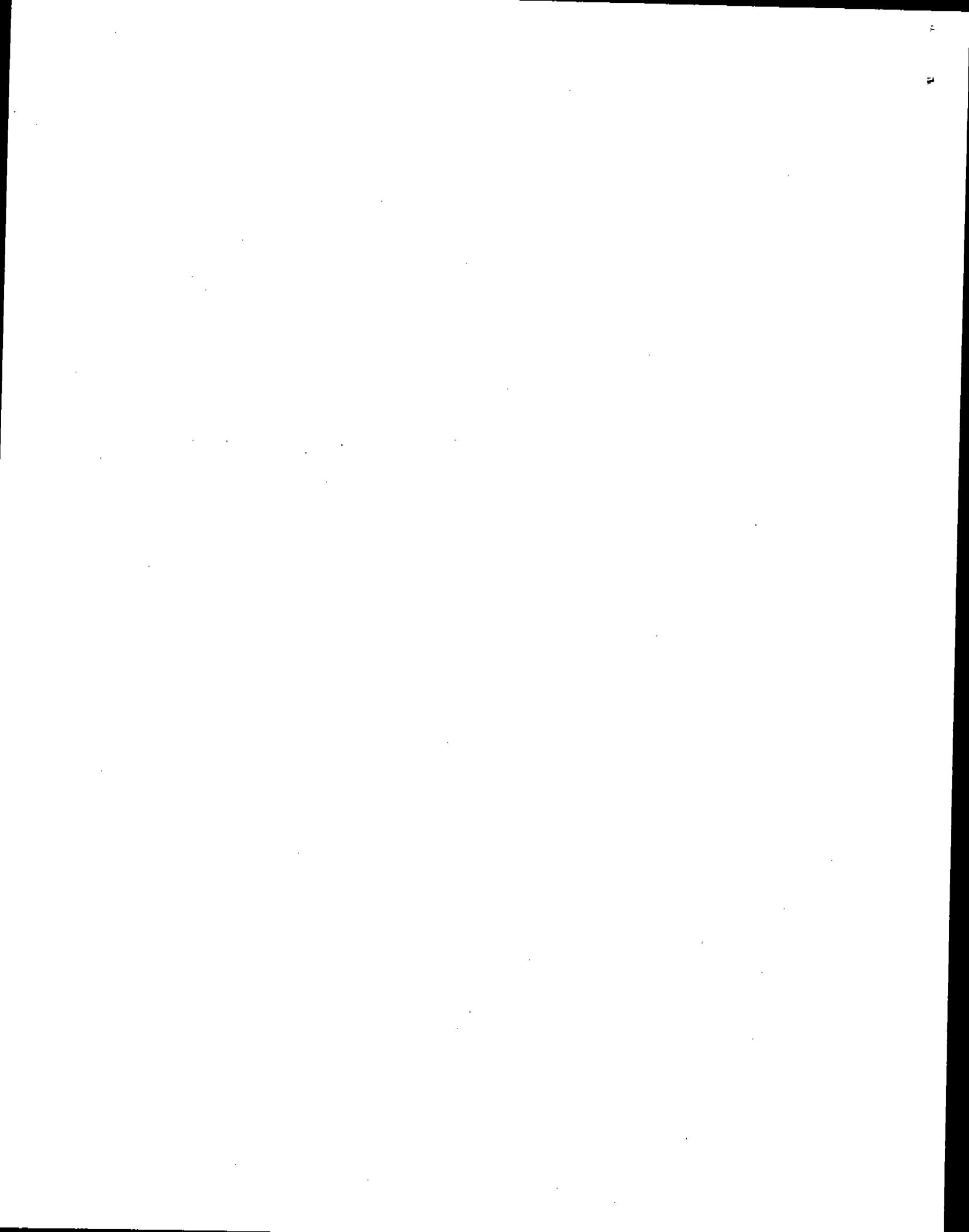
Acknowledgments

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The Clean Air Scientific Advisory Committee (CASAC) formally reviewed this document in December 1994. CASAC comments and recommendations have been carefully considered in revising this draft. A copy of the CASAC closure letter for this Staff Paper is included in Appendix A of this document.

List of Figures

IV-1	National trend in the composite annual average nitrogen dioxide concentration at both NAMS and all sites with 95 percent confidence intervals, 1983-1992.	8
IV-2	Regional comparisons of 1990, 1991, 1992 composite averages of the annual mean nitrogen dioxide concentrations	9
IV-3	Mean annual wet nitrate and ammonium deposition to various states located throughout the United States.	11
VIII-1	Schematic representation of the nitrogen cycle, emphasizing human activities that affect fluxes of nitrogen.	56
VIII-2	Schematic diagram of cation exchange for base cations, aluminum ions, and hydrogen ions in circumneutral and acid soils.	63



Executive Summary

In 1971, the Environmental Protection Agency (EPA) promulgated identical primary and secondary national ambient air quality standards (NAAQS) for nitrogen dioxide (NO_2) at 0.053 ppm annual average (36 FR 8186). Section 109 of the Clean Air Act requires EPA to periodically review the NAAQS to ensure the scientific adequacy of these air quality standards. The last review of the NO_2 criteria and NAAQS was completed in 1985. At that time, EPA published a rule retaining the NO_2 NAAQS at 0.053 ppm annual average (50 FR 25532).

As part of the current review, the Office of Air Quality Planning and Standards (OAQPS) developed this Staff Paper to summarize and integrate the key studies and scientific evidence contained in the revised document, "Air Quality Criteria for Oxides of Nitrogen" (EPA, 1993), and to identify the critical elements the staff believes should be considered in the review of the NAAQS. Summarized below are the staff's key findings and recommendations from the current review of the NO_2 NAAQS.

Air Quality Trends

Nitrogen dioxide (NO_2) is a brownish, highly reactive gas which is formed in the ambient air through the oxidation of nitric oxide (NO). Nitrogen oxides (NO_x), the term used to describe the sum of NO and NO_2 , play a major role in the formation of ozone in the atmosphere through a complex series of reactions with volatile organic compounds. Anthropogenic (i.e., man-made) sources of NO_x emissions account for a large majority of all nitrogen inputs to the environment. The major sources of anthropogenic NO_x emissions are mobile sources and electric utilities. Ammonia and other nitrogen compounds produced naturally do play a role in the cycling of nitrogen through the ecosystem.

Typical peak annual average ambient concentrations of NO₂ range from 0.007 to 0.061 ppm (CD, pg. 7-10). The highest hourly NO₂ average concentrations range from 0.04 to 0.54 ppm (CD, pg. 7-10). Currently, all areas of the United States, including Los Angeles (which is the only area to record violations in the last decade), are in attainment of the annual NO₂ NAAQS of 0.053 ppm. Analysis of air quality data indicates that if the annual standard continues to be attained, risk of exposure to high short-term peaks is low (McCurdy, 1994).

Health Effects

The staff concludes that exposure to NO₂ is associated with a variety of acute and chronic health effects. Clearly adverse health effects (e.g., pulmonary edema, death) have been reported following accidental short-term (e.g., 6 to 7 hours) exposures of humans to 150 to 200 ppm of NO₂ or greater (CD, pg. 14-55). Furthermore, animal studies have provided evidence of emphysema caused by long-term exposures to greater than 8 ppm NO₂. It is clear that these health endpoints are caused by exposures which are much higher than those found in the ambient air and only serve as indicators of the most extreme effects of a potentially very noxious oxidant.

Other acute health endpoints that have been associated with much lower NO₂ concentrations include (1) changes in pulmonary function, (2) increases in airway responsiveness, and (3) increased risk for developing respiratory diseases and illnesses. Based on the assessment of available clinical and epidemiological information regarding the health effects associated with short-term exposure to NO₂, the staff concludes that the two potentially sensitive population groups at risk are (1) individuals with pre-existing respiratory diseases and (2) children five to twelve years old.

The available clinical data indicate that short-term exposure (e.g., less than 3 hours) to NO₂ may cause an increase in airway responsiveness in asthmatic individuals at rest.

This response has been reported only at relatively low concentrations (mostly within the range of 0.2 to 0.3 ppm NO₂) which are of concern in the ambient environment. Similarly, NO₂ induced pulmonary function changes in asthmatic individuals have been reported at low, but not high, NO₂ concentrations. For the most part, the small changes in pulmonary function that have been observed in asthmatic individuals have occurred at concentrations between 0.2 and 0.5 ppm, but not at much higher concentrations (i.e., up to 4 ppm) (CD, pg. 16-3). These findings contrast with the findings for healthy individuals exposed to low NO₂ concentrations. In healthy individuals, there is no evidence of lung function decrements or changes in airway responsiveness at concentrations below 1.0 ppm NO₂.

The epidemiological evidence includes a meta-analysis of nine epidemiological studies of children (5-12 years old) living in homes with gas stoves. The results of the meta-analysis show that children (5-12 years old) living in homes with gas stoves are at increased risk for developing respiratory diseases and illnesses compared to children living in homes without gas stoves. Typical mean weekly NO₂ concentrations in bedrooms in studies reporting NO₂ levels were predominately between 0.008 and 0.065 ppm NO₂. Most of the NO₂ measurements reported in the studies are for indoor 1- to 2-week averages, but very little information is provided regarding either peak hourly exposures or measurements providing estimates of annual exposures. Because of this, the studies and, therefore, the meta-analysis cannot distinguish between relative contributions of peak and longer term exposure and their relationship with the observed health effects. Given the uncertainty associated with determining actual exposure patterns in these homes and the difficulty in extrapolating the data to ambient exposures, results of the meta-analysis provide insufficient data to support specific limits for either short-term or long-term standards for nitrogen dioxide. Based on this current assessment of the

available scientific and technical information (which remains largely unchanged since the 1985 review), the staff again recommends consideration be given to setting the level of the annual primary standard within the range of 0.05 to 0.08 ppm NO₂. In the staff's judgement, selecting a standard within this range would provide adequate protection against the health effects associated with chronic or long-term NO₂ exposure. In reaching a determination on the standard, the staff also recommends that consideration be given to the degree of protection that would be provided against repeated short-term peak exposures. Based on air quality analyses, a standard selected from the lower portion of this range would effectively limit the frequency and magnitude of 1-hour NO₂ concentrations. McCurdy (1994) estimated that if the existing standard of 0.053 ppm NO₂ is attained, the occurrence of 1-hour NO₂ values greater than 0.2 ppm would be unlikely in most areas of the country. At the upper end of the range, the frequency of 1-hour NO₂ peaks of 0.2 or higher could increase significantly. However, because all areas of the country reporting NO₂ air quality data are attaining the existing standard and because of the nonlinear relationship of 1-hour peaks and annual averages, it is not possible to estimate with any degree of confidence what the frequency and magnitude of 1-hour peaks would be if the standard was selected from the upper end of the suggested range. Given this uncertainty, the staff recommends consideration be given to selecting the standard from the lower portion of the range in order to provide a reasonable measure of protection against repeated 1-hour peaks of potential health concern.

Environmental Effects

Nitrogen oxides have been associated with a wide range of effects on vegetation, natural ecosystems, visibility, and materials. Studies show that nitrogen oxides are contributing to the observed effects either through direct deposition or by indirectly altering processes within the system.

After reviewing the key criteria concerning the effects of exposure to nitrogen oxides on the environment, the staff concludes that the impact on terrestrial vegetation from short-term exposures to NO_2 under existing ambient levels is insignificant and does not warrant a change in the secondary NAAQS at this time. Studies of short-term, acute effects of nitrogen additions to vegetation have demonstrated that NO_2 in mixtures with other pollutants or at higher than ambient concentrations can produce adverse effects on plant growth or reproduction. Though the sensitivity of vegetation to NO_2 varies both within and between species and can be modified by season, developmental stage, or other climate or environmental factors, taking all of these variables into account, the ambient levels of NO_2 in the United States are considered below those that evoke a short-term, acute response. The staff also concludes that there is little potential for concentrations of nitrogen deposition to be sufficient to acidify soils to the extent that it would cause direct phytotoxic effects in plants.

Based on existing European studies and information in the United States, it seems that addition of nitrogen to ecosystems in the U.S. (specifically in mature forests and wetlands which host a number of endangered species adapted to nitrogen-poor habitats) may lead to shifts in species competition and composition and may represent a significant change in the environment. However, there is not sufficient evidence to conclude that changes in plant communities have occurred in the past or are now occurring in the United States due to nitrogen inputs. Furthermore, there are only limited long-term data available on plant community composition. Additional research is needed to more accurately characterize any potential threat to mature forests and wetland species from atmospheric nitrogen. Once this information is available, the need for additional ecosystem protection should be reassessed by the Administrator.

Based on the review of the available scientific information in the CD, the staff concludes that nitrogen deposition is a

contributor to the episodic acidification of some streams and lakes in the United States. Thus, it can also be concluded that some freshwater ecosystems will benefit from reductions in nitrogen inputs from man-made sources. However, at this time, the staff finds insufficient evidence to quantify how much of a contribution nitrogen deposition is making to the acidification problem and what levels of reduction are necessary to remedy the situation. Therefore, staff recommends that further research be conducted to provide the information needed to evaluate this issue in its next standard review. When additional scientific information is available that adequately quantifies the relationship between nitrogen deposition and acidification, the staff recommends that the Administrator consider appropriate regulatory approaches to address the problem. Key considerations will be the major differences evident in the occurrence, nature, location, timing of episodes at different sites, and the subsequent effects produced. Such differences will make setting a single national standard which would adequately protect all areas of the country difficult and complex. Therefore, staff believes regional approaches that take into account such variations should be considered for providing the level of control needed to address the problem of acidification.

It is very unlikely that eutrophication of freshwater systems is due to atmospheric nitrogen additions since phosphorus, not nitrogen, is generally the limiting factor for algae growth. However, both upland and direct atmospheric nitrogen deposition can significantly affect the trophic status of estuarine and coastal waters. As mentioned before, once a relationship can be reasonably quantified, staff recommends that the Administrator consider appropriate regulatory approaches, including a uniform national standard or regional standards, to protect estuarine and coastal waters.

The concept of critical loads is addressed in the Staff Paper as a possible means for assessing and controlling NO₂ emissions in the future. Critical loads are defined as

"quantitative estimates of an exposure to one or more pollutants below which significant harmful effects on specified sensitive elements of the environment do not occur according to present knowledge." Staff concluded that it would be premature to consider establishment of a long-term national critical loads secondary ambient air quality standard for NO₂ given the state of the science of critical loads estimation that exists at the current time. However, this methodology has the potential to provide a useful tool for making regulatory decisions at the regional level in the future.

With regard to visibility impairment, the scientific evidence indicates that light scattering by particles is generally the primary cause of degraded visual air quality. The evidence also suggests that aerosol optic effects alone can impart a reddish-brown color to a haze layer. While it is clear that particles and NO₂ contribute to brown haze, in the staff's judgement reducing ground level NO₂ concentrations would result in little if any improvement in visual air quality. Therefore, the staff concludes that an ambient secondary standard for NO₂ to protect visibility is not warranted at this time.

Based on the available data, the staff concludes that it is unlikely that NO₂ is playing a significant role in the damage to non-biological materials and therefore does not recommend consideration be given to setting a secondary standard to protect against material damage.

If the Administrator determines that a separate secondary standard is appropriate, the staff recommends that it be set identical to the primary standard for NO₂. A secondary standard set within the range recommended for the primary standard would, in the staff's judgement, provide adequate protection against the direct effects of NO₂ on the environment.

It is clear that additional studies need to be conducted to reduce the uncertainty in the relationship between nitrogen deposition and forest health and in determining the adverse

effects on surface waters and estuaries arising from the long-term accumulation of nitrogen. The staff recommends substantial efforts be undertaken to illuminate the quantitative relationships between nitrogen deposition and changes in forest nitrogen cycling, episodic and chronic acidification of surface waters, and estuarine eutrophication, either through ongoing research or new initiatives. Such efforts will provide an improved scientific basis for the next standard review.

REVIEW OF THE NATIONAL AMBIENT AIR QUALITY STANDARDS
FOR NITROGEN DIOXIDE

ASSESSMENT OF SCIENTIFIC AND TECHNICAL INFORMATION

I. PURPOSE

The purpose of this Office of Air Quality Planning and Standards (OAQPS) Staff Paper is to summarize and integrate the key studies and scientific information contained in the revised EPA document, "Air Quality Criteria for Oxides of Nitrogen" (U.S. EPA, 1993a; henceforth referred to as CD), and to identify the critical elements EPA staff believes should be considered in the review of the national ambient air quality standards (NAAQS) for nitrogen dioxide (NO₂). Factors relevant to the evaluation of current primary (health) and secondary (welfare) NAAQS, as well as staff conclusions and recommendations, are provided in this Staff Paper.

II. BACKGROUND

A. Legislative Requirements

Two sections of the Act govern the establishment and revision of NAAQS. Section 108 (42 U.S.C. 7408) directs the Administrator to identify pollutants which "may reasonably be anticipated to endanger public health and welfare" and to issue air quality criteria for them. These air quality criteria are to "accurately reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the presence of [a] pollutant in the ambient air . . ."

Section 109 (42 U.S.C. 7409) directs the Administrator to propose and promulgate "primary" and "secondary" NAAQS for pollutants identified under Section 108. Section 109(b)(1) defines a primary standard as one "the attainment and maintenance of which, in the judgment of the Administrator, based on the criteria and allowing an adequate margin of safety, [is]

requisite to protect the public health."¹ A secondary standard, as defined in Section 109(b)(2), must "specify a level of air quality the attainment and maintenance of which, in the judgment of the Administrator, based on [the] criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of [the] pollutant in the ambient air." Welfare effects as defined in Section 302(h) [42 U.S.C. 7602(h)] include, but are not limited to, "effects on soils, water, crops, vegetation, manmade materials, animals, wildlife, weather, visibility and climate, damage to and deterioration of property, and hazards to transportation, as well as effects on economic values and on personal comfort and well-being."

The U.S. Court of Appeals for the District of Columbia Circuit has held that the requirement for an adequate margin of safety for primary standards was intended to address uncertainties associated with inconclusive scientific and technical information available at the time of standard setting. It was also intended to provide a reasonable degree of protection against hazards that research has not yet identified. Lead Industries Association v. EPA, 647 F.2d 1130, 1154 (D.C. Cir. 1980), cert. denied, 101 S. Ct. 621 (1980); American Petroleum Institute v. Costle, 665 F.2d 1176, 1177 (D.C. Cir. 1981), cert. denied, 102 S. Ct. 1737 (1982). Both kinds of uncertainties are components of the risk associated with pollution at levels below those at which human health effects can be said to occur with reasonable scientific certainty. Thus, by selecting primary

¹The legislative history of Section 109 indicates that a primary standard is to be set at "the maximum permissible ambient air level . . . which will protect the health of any [sensitive] group of the population," and that for this purpose "reference should be made to a representative sample of persons comprising the sensitive group rather than to a single person in such a group." S. Rep. No. 91-1196, 91st Cong., 2d Sess. 10 (1970). The legislative history specifically identifies bronchial asthmatics as a sensitive group to be protected. Id.

standards that provide an adequate margin of safety, the Administrator is seeking not only to prevent pollution levels that have been demonstrated to be harmful but also to prevent lower pollutant levels that may pose an unacceptable risk of harm, even if the risk is not precisely identified as to nature or degree.

In selecting a margin of safety, the EPA considers such factors as the nature and severity of the health effects involved, the size of the sensitive population(s) at risk, and the kind and degree of the uncertainties that must be addressed. Given that the "margin of safety" requirement by definition only comes into play where no conclusive showing of adverse effects exists, such factors, which involve unknown or only partially quantified risks, have their inherent limits as guides to action. The selection of any numerical value to provide an adequate margin of safety is a policy choice left specifically to the Administrator's judgment. Lead Industries Association v. EPA, supra, 647 F.2d at 1161-62.

Section 109(d)(1) of the Act requires that "not later than December 31, 1980, and at 5-year intervals thereafter, the Administrator shall complete a thorough review of the criteria published under Section 108 and the national ambient air quality standards . . . and shall make such revisions in such criteria and standards . . . as may be appropriate" Section 109(d)(2)(A) and (B) require that a scientific review committee be appointed and provide that the committee "shall complete a review of the criteria . . . and the national primary and secondary ambient air quality standards . . . and shall recommend to the Administrator any . . . revisions of existing criteria and standards as may be appropriate"

B. Establishment of Nitrogen Dioxide NAAQS

On April 30, 1971, the EPA promulgated NAAQS for NO₂ under Section 109 of the Act (36 FR 8186). Identical primary and secondary NAAQS were set at 100 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$) [0.053 parts per million (ppm)] as an annual arithmetic

mean. Scientific and technical bases for these NAAQS are provided in the document, "Air Quality Criteria for Nitrogen Oxides" (U.S. EPA, 1971). The primary standard was based largely on several epidemiology studies (Shy et al., 1970a; 1970b; Pearlman et al., 1971) conducted in Chattanooga. These studies reported respiratory effects in children exposed to low-level ambient NO₂ concentrations over a prolonged period. Reevaluation of these studies, particularly the method used to monitor NO₂, indicates that the studies provide only qualitative evidence for an association between health effects and exposure to NO₂.

C. First Review of Nitrogen Dioxide NAAQS

The first review of NO₂ criteria and NAAQS was initiated in 1977. The Clean Air Scientific Advisory Committee (CASAC) held meetings in 1979 and 1980 before providing written closure on the revised criteria document (U.S. EPA, 1982) in June 1981. A staff paper, which identified critical issues and summarized staff's interpretation of key studies received verbal closure at a CASAC meeting in November 1981 and formal written closure in July 1982. In the paper, staff recommended that the Administrator select an annual standard "at some level between 0.05 ppm and 0.08 ppm." Based on the analysis of the criteria, staff concluded that choosing an annual standard within this range would "provide a reasonable level of protection against potential short-term peaks."

On February 23, 1984, the EPA proposed to retain the existing annual primary and secondary standards and defer action on the possible need for a separate short-term primary standard until further research on health effects of acute exposures to NO₂ could be conducted. CASAC met to consider the Agency's proposal July 19-20, 1984. In an October 18, 1984 closure letter, based on weight of evidence, CASAC concurred with the Agency's recommendation to retain the annual average primary and secondary standards at 0.053 ppm. The Committee further concluded that, "while short-term effects from nitrogen dioxide are documented in the scientific literature, the available

information was insufficient to provide an adequate scientific basis for establishing any specific short-term standard...."

Following a period of public review and comment, the final rule to retain the NAAQS for NO₂ was published in the Federal Register on June 19, 1985 (50 FR 25532).

D. Current Review of Nitrogen Dioxide NAAQS

EPA's Environmental Criteria and Assessment Office (ECAO) initiated action on a revised CD, which was released for public review in August 1991. The revised CD includes a meta-analysis of several studies, conducted in homes with gas stoves, which reported increased rates of respiratory symptoms and illness for children living in those homes. The CD also discussed information relevant to nitrogen deposition and potential impacts on natural ecosystems. The CASAC reviewed the CD at a meeting held on July 1, 1993 and concluded in a letter to the Administrator that the CD ". . . provides a scientifically balanced and defensible summary of current knowledge of the effects of this pollutant and provides an adequate basis for EPA to make a decision as to the appropriate NAAQS for NO₂" (see Appendix A).

The current review of the primary NAAQS will focus primarily on the health effects associated with exposure to nitrogen dioxide since it is the NO_x compound measured in most epidemiological studies and is currently of greatest concern from a public health perspective. On the other hand, staff will consider the total class of nitrogen compounds and their effects on the environment when evaluating the secondary standard.

III. APPROACH

The approach taken in this Staff Paper is to assess and integrate the scientific and technical information contained in the revised CD in order to evaluate the adequacy of the current NAAQS for NO₂. Consideration of a qualitative exposure analysis is used in assessing the primary NAAQS, and information on nitrogen deposition is used in determining the possible need for revising the secondary NAAQS for NO₂. Critical elements have

been identified which staff believes should be considered in review of the NAAQS for NO₂. Attention is drawn to those judgments that must be based on careful interpretation of incomplete or uncertain evidence. In such instances, the Staff Paper provides staff's evaluation, sets forth alternatives that staff believes should be considered, and recommends a course of action.

Following a short presentation of air quality information in Section IV, the Staff Paper provides in Section V a discussion of mechanisms of NO₂ toxicity, an evaluation of effects of concern and effect levels, and a description of most sensitive population groups and size estimates. Staff judgments are made concerning which effects are important for the Administrator to consider in selecting appropriate primary standard(s). Section VI identifies and discusses factors important in selecting a primary standard including possible averaging times and forms of the standard. Drawing on these factors and information contained in Section V, staff conclusions/recommendations are presented in Section VII for the Administrator to consider in selecting primary NO₂ NAAQS.

In a similar approach for selecting a secondary standard or standards for NO₂, Section VIII provides information on ecosystem response (i.e., impact on terrestrial systems, wetlands and bogs, and aquatic systems), critical loads and other policy options, visibility effects, human welfare effects, and materials damage. Using this information, Section VIII offers an analysis of the key information the Administrator needs to consider in selecting an appropriate secondary standard for NO₂. Staff conclusions and recommendations concerning the secondary standard for NO₂ are summarized in Section IX.

IV. AIR QUALITY TRENDS

Nitrogen dioxide (NO₂) is a brownish, highly reactive gas which generally exists at higher concentrations in urban than in rural atmospheres. It is formed in the ambient air through the oxidation of nitric oxide (NO), a primary pollutant emitted directly from stationary (e.g., electric utility and industrial

boilers) and mobile (e.g., automobiles) sources. Nitrogen oxides (NO_x), the term used to describe the sum of NO and NO_2 , play a major role in the formation of ozone in the atmosphere through a complex series of reactions with volatile organic compounds. About 5 to 10 percent by volume of the total emissions of NO_x from combustion sources is in the form of NO_2 , although substantial variations from one source to another have been observed (CD, pg. 3-10).

A. Long-Term Trends

Long-term trends for NO_2 are based on monitoring data collected over a ten-year period using instruments capable of recording as many as 8760 hourly observations per year. For the period 1983 to 1992, the long-term trends were based on data from 183 sites, which were selected because each had annual mean NO_2 concentrations based on a minimum of 4380 hourly observations. Although the sites had to meet only the minimum data requirements to be included in the trends database, 90 percent of the sites had greater than 75 percent data completeness. 43 National Air Monitoring Station (NAMS) sites had data for at least 8 of the last 10 years and thus met the selection criteria for the 10-year database.

Figure IV-1 represents the composite long-term trend for the NO_2 mean concentrations at 183 trend sites and 43 NAMS sites. The 95 percent confidence intervals shown represent the confidence range of the annual averages for the years reviewed. For the period 1983 to 1989, the 95 percent confidence intervals about the composite means indicates that NO_2 levels are statistically indistinguishable. However, the 1992 composite average NO_2 level is 8 percent lower than the 1983 level, and the difference in the two levels is statistically significant.

At the NO_2 NAMS sites, which are located only in large urban areas with populations of one million or greater, a similar trend is apparent, although the composite averages of the NAMS data are higher than those of all 183 sites. Compared to the composite

average in 1983, the composite average of the annual NO_2 mean concentration of data collected at the 43 NAMS sites was 11

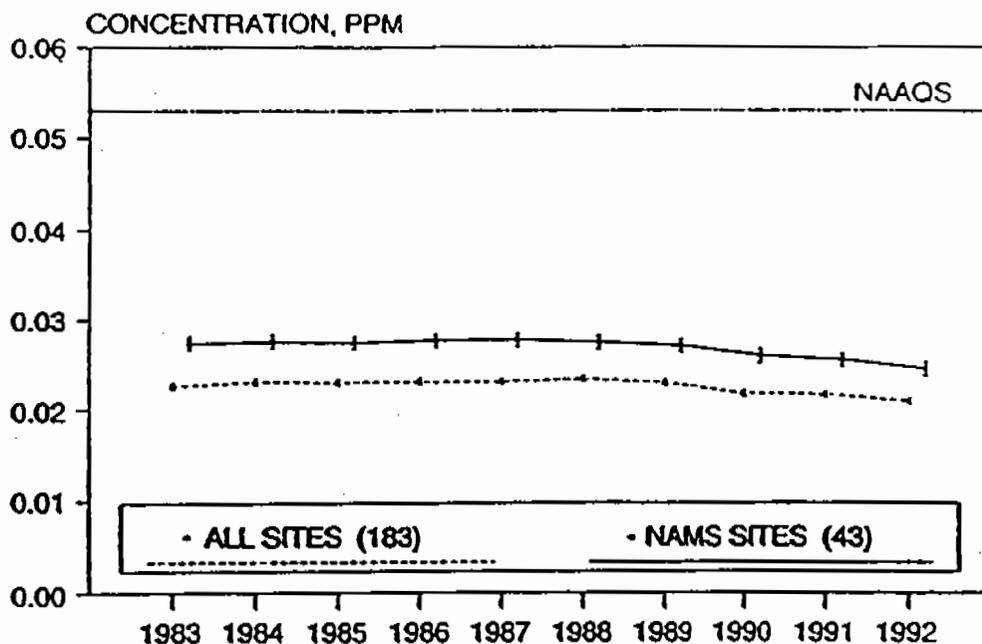


Figure IV-1. National trend in the composite annual average nitrogen dioxide concentration at both NAMS and all sites with 95 percent confidence intervals, 1983-1992.

Source: Figure 3-13, p. 3-17 of the National Air Quality and Emissions Trends Report (U.S. EPA, 1993b).

percent lower. This is also a statistically significant difference.

It is important to note here the relationship between short-term exceedances of NO_2 concentrations and the annual NO_2 mean. In 1994, McCurdy analyzed air quality data from the period 1988-1992 to determine the estimated number of exceedances of various NO_2 short-term air quality indicators would occur given attainment of a range of annual averages. The annual averages McCurdy analyzed ranged from 0.02 to 0.06 ppm and included the current NO_2 NAAQS of 0.053 ppm. The 1-hour and daily concentration levels chosen for analyses were 0.15, 0.20, 0.25, and 0.30 ppm. The results of this analysis are reported in "Analysis of High 1 Hr NO_2 Values and Associated Annual Averages Using 1988-1992 Data" (McCurdy, 1994). In his report, McCurdy

concluded that areas attaining the current annual NO₂ NAAQS reported few, if any, one hour or daily exceedances above 0.15 ppm. Excerpts from the conclusion section of this report can be found in Appendix B of this Staff Paper.

B. Recent Trends in Urban Areas of the United States

Los Angeles (LA) is the only city in the U.S. to record violations of the annual average NO₂ NAAQS during the past decade. However, in 1992, Los Angeles reported air quality measurements which meet the NO₂ NAAQS for the first time. Thus, currently, the entire U.S. is in attainment of the current NO₂ NAAQS.

Regional trends in the composite average NO₂ concentrations for the years 1990 to 1992 are presented as bar graphs in Figure IV-2 with the exception of Region X, which had inadequate data.

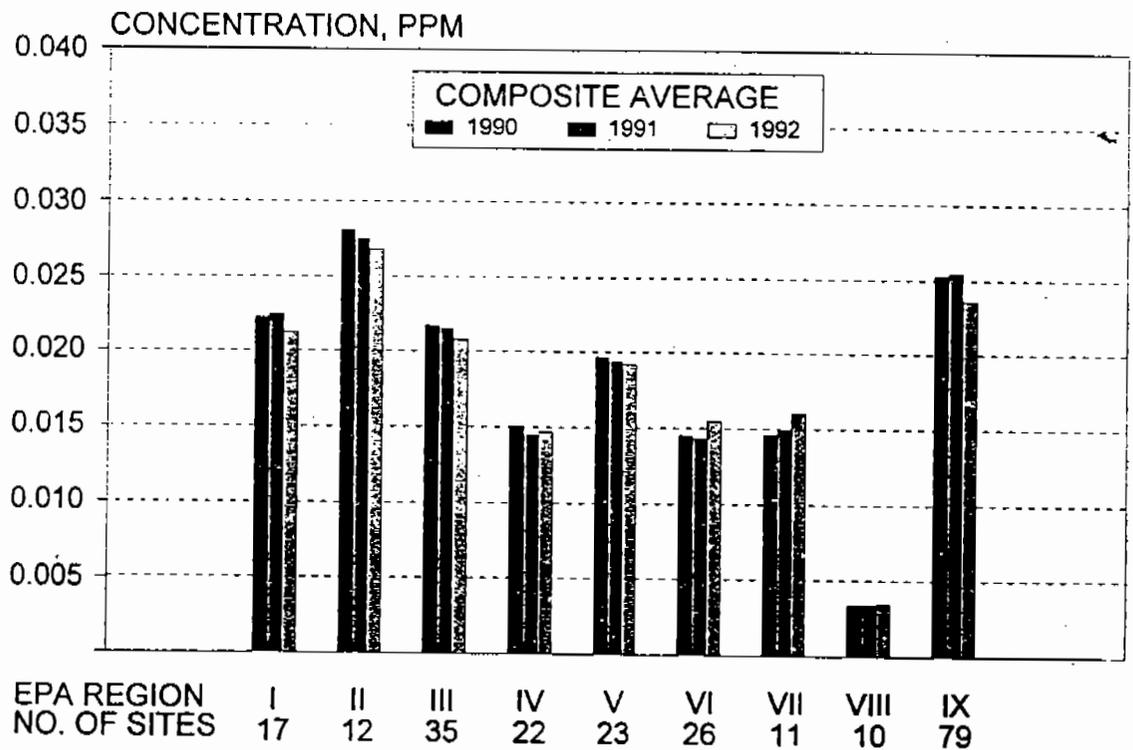


Figure IV-2 Regional comparisons of 1990, 1991, 1992 composite averages of the annual mean nitrogen dioxide concentrations.

Source: Figure 3-15, p. 3-20 of the National Air Quality Emissions Trends Report (U.S. EPA, 1993b).

Although there was no change between 1990 and 1991 in the composite annual mean NO₂ levels at 235 sites, there was a 3 percent decrease between 1991 and 1992.

Different selection criteria are used to determine which sites will be included in the long-term national trends database described in the preceding section than is used to determine which sites will be used in the short-term regional trends analysis. 50 NAMS sites had data in each of the last three years and thus met the criteria to be included in the 3-year database. For these 50 NAMS sites, the composite mean concentration decreased 4 percent between 1991 and 1992.

Bar graphs for five of the nine regions (I, II, III, V, and IX) depicted in Figure IV-2 show 1992 composite average NO₂ annual mean concentrations that are lower than the composite average NO₂ annual mean concentrations for 1990 and 1991. The other four regions (IV, VI, VII, and VIII) recorded increases between 1991 and 1992, although the 1992 composite average NO₂ annual mean concentrations for Region IV are less than the 1990 composite mean. (These graphs are intended to depict relative changes in regional concentrations only and should not be used to indicate differences in absolute concentrations). For a more thorough discussion and description of national and international air quality trends, the reader is referred to the National Air Quality and Emissions Trends Report, 1992 (U.S. EPA, 1993b).

C. Nitrogen Deposition Trends in the United States

Atmospheric nitrogen inputs occur as both wet and dry deposition. Most of the data on nitrogen deposition are limited to information on nitrate deposition in rainfall (i.e., wet deposition). Figure IV-3 summarizes mean annual wet deposition rates for nitrate for states that were part of the National Acid Deposition Program (NADP). The figure shows that the highest deposition rates are concentrated in the northeastern United States. The highest rates noted were in Pennsylvania and New York which had nitrate deposition rates of 12 and 11 kg/ha/yr,

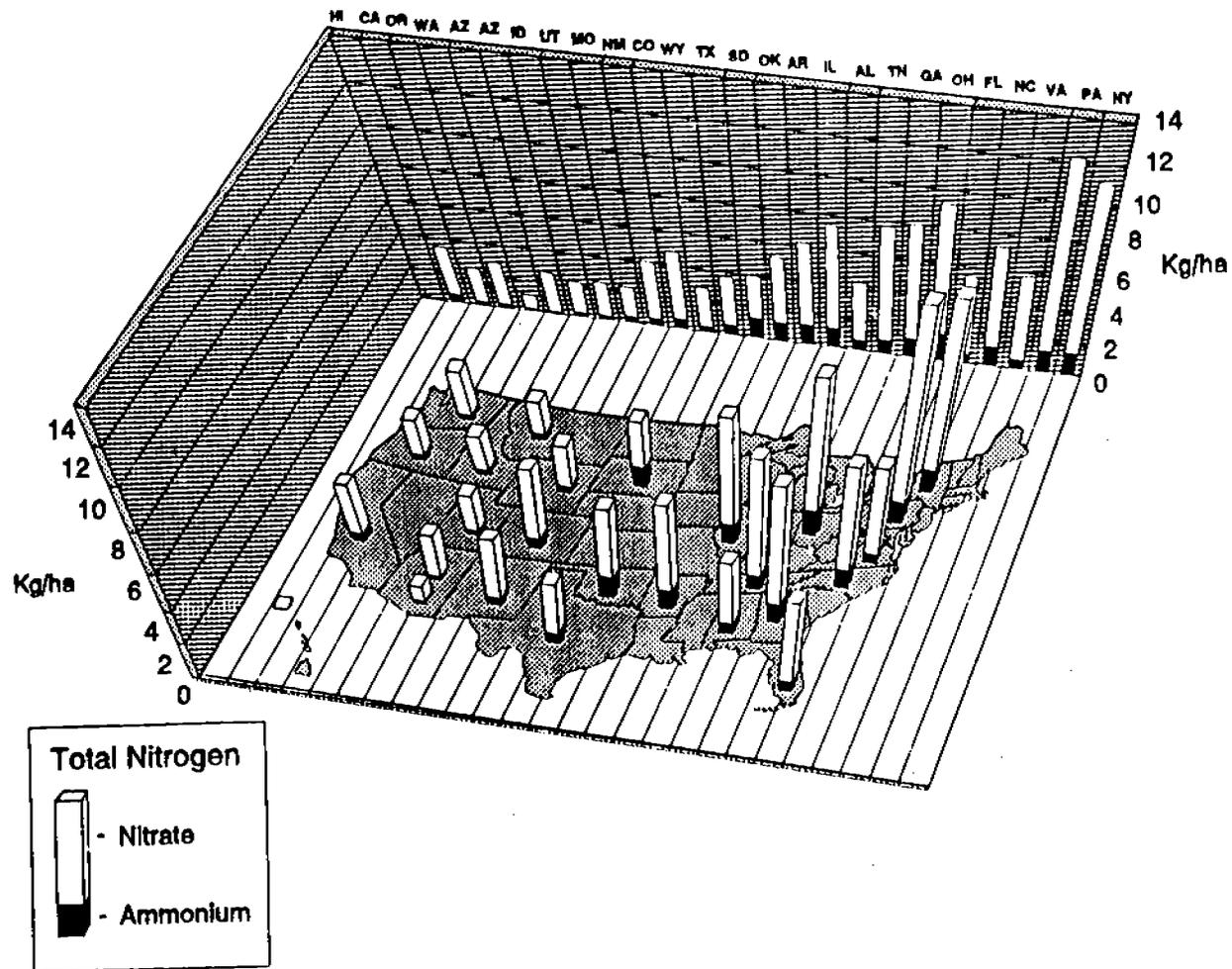


Figure IV-3 Mean annual wet nitrate and ammonium deposition to various states located throughout the United States.

Source: Data from the National Atmospheric Deposition Program (1988) are for a single year, and data summarized by Bohm (1991) are for the period 1985 through 1988.

respectively. Illinois, Tennessee, and Georgia showed rates of approximately 8 kg/ha/year.

Accurate measurements of wet deposition are carried out by analyzing nitrogen in precipitation immediately following a precipitation event. Frequently, however, the rainfall is accumulated over some period of time before it is analyzed. The resulting measurement of deposition rate is usually referred to as bulk deposition because it combines wet deposition with some component of dry deposition.

Because of the difficulty associated with accurately measuring dry deposition, researchers have assumed that dry deposition rates are some fraction of wet deposition rates. Researchers have attempted to model the fate of NO_x emissions to the atmosphere. The models have estimated that dry deposition accounts for one-half to two-thirds of the total NO_x deposition in North America (Levy and Moxim, 1987; Hicks et al., 1991). The correlation between wet and dry deposition is discussed further in Section VIII.A.2 of this Staff Paper and in Chapters 5 and 10 of the Criteria Document.

V. HEALTH EFFECTS OF NITROGEN DIOXIDE

A. Introduction

This section presents those elements which are critical to the review of the primary NAAQS for NO_2 . In addition to NO_2 , there is a variety of NO_x and related nitrogen compounds which occur in the ambient air both naturally and as a result of human activities, including NO , nitrous oxide (N_2O), gaseous nitric (HNO_3) and nitrous (HNO_2) acids, dinitrogen pentoxide (N_2O_5), dinitrogen trioxide (N_2O_3), dinitrogen tetroxide (N_2O_4), and ammonia (NH_3). However, based on currently available information, only NO_2 is sufficiently widespread and commonly found in ambient air at high enough concentrations to be a matter of public health concern. Therefore, this section will focus on mechanisms of toxicity, health effects of concern, and susceptible subpopulation groups for NO_2 , the control of which serves as a surrogate for the control of other nitrogen compounds in the troposphere.

B. Mechanisms of Toxicity, Transport, and Fate

Nitrogen dioxide has been shown to produce lung cell injury and/or death and other respiratory effects in humans and animals exposed both acutely and chronically. The principle mechanisms of toxicity associated with NO_2 inhalation involve oxidation of unsaturated fatty acids in cell membranes and of functional groups in soluble (e.g., enzymes) and structural (e.g., cell membranes) proteins (Menzel, 1976; Freeman and Mudd, 1981). Support for this mechanism of toxicity is provided by studies which have shown an increase in both lipid peroxidation products and in lung antioxidant enzymes immediately following exposure to NO_2 (Sagai et al., 1984). Further supporting this oxidative mechanism are reports that animals deficient in vitamins C and E (i.e., antioxidant vitamins) tend to be more susceptible to NO_2 exposures (Selgrade et al., 1981; Sevanian et al., 1982). The fundamental mechanism of pulmonary edema (i.e., excess fluids in the lungs) resulting from exposure to NO_2 may be the cytotoxic effects of NO_2 directly on epithelial cell membranes, while the

mechanism responsible for increased susceptibility to viral and bacterial infection may be the cytotoxicity of NO_2 on membranes of alveolar macrophages (AM's) (CD, p. 13-197). The above mechanisms of toxicity are generally considered to be related to those health effects of NO_2 which are of greatest public health concern and will be discussed further in Section V.C below.

Because NO_2 is not very soluble in the fluids lining the respiratory tract, it is capable of penetrating to the distal airways, particularly during heavy exercise. The reactive nature of NO_2 , however, tends to result in scrubbing of a large fraction in the upper respiratory tract under normal conditions. Total respiratory tract uptake in humans has been reported between 72 and 92%, depending on investigator and breathing state (Wagner, 1970; Bauer et al., 1986). Similarly, total respiratory tract uptake in dogs at rest is 78% versus 94% in exercising dogs (Kleinman and Mautz, 1991). Generally, as ventilation rate increases, the percentage uptake of NO_2 in the lower respiratory tract, as well as total uptake, increases (CD, p. 13-196), therefore resulting in an increase in effects associated with NO_2 exposure.

Mathematical modeling of the lower respiratory tracts of humans, rats, guinea pigs, and rabbits by Miller et al. (1982) and Overton et al. (1984) has indicated that the greatest dose of NO_2 is delivered to the centriacinar region (CAR) (i.e., the junction between conducting airways and the gas-exchange region). This is consistent with findings in animal studies reporting that the CAR is the site where NO_2 -induced lesions are observed morphologically.

The ultimate fate of NO_2 following reaction with respiratory fluids and tissues appears to be formation of other chemicals, such as nitrous and nitric acids, which then can be systemically transported (Goldstein et al., 1973). Nitrite produced in the lungs may enter the bloodstream and react with hemoglobin to increase methemoglobin levels (Postlethwait and Mustafa, 1981, 1989; Saul and Archer, 1983). For a more thorough discussion of

mechanisms of toxicity, transport, and fate of inhaled NO_2 , the reader is referred to Chapter 13 of the CD.

C. Health Effects Evidence

Health effects information which is pertinent to review of the NAAQS for NO_2 has been thoroughly reviewed in Chapters 13 through 16 of the CD. Many of the health studies, particularly animal toxicology studies, reviewed in the CD were conducted at levels of NO_2 much higher than those typically found in the ambient air. Although reference is made in this section to some studies conducted at very high NO_2 levels, the focus of this Staff Paper is primarily on those key studies conducted at levels of NO_2 which are relevant to regulatory decision making.

Key health effects which have been associated with exposure to NO_2 include: (1) increased susceptibility to respiratory symptoms and disease in children; (2) pulmonary function decrements [e.g., forced expiratory volume in 1 second (FEV_1), forced vital capacity (FVC)], symptoms (e.g., cough, odor detection, nasopharyngeal irritation), and increased airway resistance (R_{aw}) in asthmatic subjects and in patients with chronic obstructive pulmonary disease (COPD); (3) increased airway responsiveness in asthmatics; and (4) emphysema observed only in animals after exposure to very high NO_2 levels for extended periods. Although all of these effects have been reported in numerous studies, there continue to be great uncertainties regarding the concentration-response relationships between each category of health effect and exposure to NO_2 .

1. Susceptibility to Respiratory Illness

Respiratory illness and those factors which affect either susceptibility or severity are important public health concerns. Any increase in susceptibility to respiratory illness in children which might be caused by NO_2 is of particular concern due to the potential for human exposure to NO_2 , the common occurrence of respiratory illness in children, and the fact that recurrent childhood respiratory illness may be a risk factor for later

increased susceptibility to lung disease (Samet et al., 1983; Samet and Utell, 1990; Glezen, 1989).

Support for the hypothesis that exposure of children to NO₂ increases susceptibility to childhood respiratory illness comes primarily from epidemiological and animal toxicological studies and to a lesser extent from clinical studies. Epidemiological evidence is largely from studies which investigated respiratory effects on children living in homes with gas stoves. Although there continues to be great uncertainty in quantitatively extrapolating results of animal studies to human health effect levels, the animal studies do provide a biologically plausible hypotheses for relating NO₂ exposure to the types of respiratory morbidity observed in humans. Although the studies are few, controlled human exposures suggest a link between NO₂ inhalation and alterations in host defenses.

a. Epidemiological Studies

A thorough discussion of relevant epidemiological studies reporting evidence of an association between NO₂ exposure and respiratory illness can be found in Chapter 14 of the CD. The most pertinent of these studies reported effects of indoor NO₂ on children (ages 5 to 12) and infants (ages < 2) living in homes with gas stoves as the major source of NO₂. Although far fewer, there also have been several studies published which investigated the impact of ambient NO₂ on prevalence of respiratory disease in children and young adults.

One important consideration in analyzing these studies for the purpose of standard setting is that human exposure patterns may differ significantly for the indoor environment in a home with a gas stove versus a typical outdoor environment. Typically, indoor exposures can be characterized by higher peak exposures. Because of the marked differences in exposure patterns, extrapolation of the results of the meta analysis described below to ambient conditions is difficult. Further discussion concerning the uncertainties associated with exposure patterns can be found in section V.C.1.b below.

1) Indoor Studies

During the past 15 years, most of the key epidemiological studies of NO₂ have been conducted in homes with gas stoves. Among the earliest of the gas-stove studies were those conducted by Melia et al. (1977, 1979, 1980), Goldstein et al. (1979, 1981), and Florey et al. (1979, 1982), who reported that children from randomly selected areas of Scotland and England had an increased risk of respiratory disease if they lived in homes with gas stoves. In a later study conducted by Melia et al. (1982), results of a questionnaire completed by parents showed that respiratory symptoms of girls living in urban areas were positively related to bedroom levels of NO₂. Subsequent reanalysis by Hasselblad et al. (1992) suggested that an increase of 0.015 ppm in bedroom NO₂ levels yields an 11% increase in the odds of respiratory illness, where mean weekly concentrations in bedrooms in studies reporting NO₂ levels were predominantly between 0.0008 and 0.065 ppm NO₂.

Odds ratios offer a useful measure for health assessment and have been used to report results for many of the gas-stove studies. For example, when Hasselblad et al. (1992) reanalyzed the Melia et al. (1977) data using a multiple logistic model, they found the combined odds ratio for boys and girls to be 1.31 (95% confidence limits of 1.16 and 1.48). This suggests that gas stove use in this study is associated with an estimated 31% increase in the odds for children having respiratory illness symptoms. Similar reanalysis of Melia et al. (1979) data by Hasselblad et al. (1992) produced a combined odds ratio for both genders of 1.24 (95% confidence limits of 1.09 and 1.42), suggesting that gas-stove use is associated with an estimated 24% increase in the odds of having respiratory symptoms in children.

In another series of studies conducted in six U.S. cities (often referred to as the Six City Study), respiratory illness of children was investigated in a large number of homes with gas stoves. In an early analysis of Six City Study data, Speizer et al. (1980) calculated an odds ratio of 1.12, suggesting that NO₂

emissions from gas stoves increase the rate of serious respiratory illness before age 2 by 12%. Later analysis of 1974-1979 data from the Six City Study by Ware et al. (1984) yielded an unadjusted odds ratio of 1.08 (95% confidence limits of 0.97 and 1.19) for a lower respiratory illness index associated with gas stove use. Other indicators such as bronchitis, cough, and wheeze did not show any increased incidence.

Dockery et al. (1989a) analyzed 1983-1986 data on 5,338 white children (ages 7 to 11); they reported only a marginally significant association between gas stove usage and doctor-diagnosed respiratory illness but found no association with chronic cough, bronchitis, persistent wheeze, and restriction of activity due to chest illness.

A different Six City Study cohort was investigated by Neas et al. (1990, 1991), who studied a stratified one-third random sample of the children that were part of the Dockery et al. (1989a) analysis. The sample was restricted to 1286 white children (ages 7 to 11) living in homes with at least one valid indoor measurement of NO_2 and respirable particles. Parents completed a questionnaire regarding symptoms during the previous year, which included attacks of shortness of breath with wheeze, persistent wheeze, chronic cough, chronic phlegm, and bronchitis. Neas et al. (1990, 1991) defined a combined symptom measure as the presence of any of the above symptoms, and in a multiple regression of this combined lower respiratory symptom measure estimated the odds ratio to be 1.40 (95% confidence limits of 1.14 and 1.72) for an additional exposure to NO_2 of 0.015 ppm. This estimated effect was consistent across seasons and sampling locations.

Several other studies provided somewhat more equivocal evidence. In a survey of 1,355 children (ages 6 to 12) conducted in Iowa City, Ekwo et al. (1983) reported a statistically significant association between gas stove use in the home and hospitalization for chest illness before age 2, but the association between gas-stove use and chest congestion/phlegm

with colds was not significant. A study of 775 children (ages 6 to 12) conducted in the Netherlands by Brunekreef et al. (1989) and Dijkstra et al. (1990) showed no evidence of an increase in respiratory disease with increasing NO₂ exposure, but the range of uncertainty is large and rates were not adjusted for covariates such as parental smoking and child's age. Keller et al. (1979a,b) reported no statistically significant changes in respiratory disease associated with living in homes with gas stove use (odds ratio of 0.72, 95% confidence limits of 0.30 and 0.74). Many of the studies of children (ages 5 to 12) discussed above are summarized in Table V-1 and form the basis for a meta-analysis to be discussed in Section V.1.b.

Individual studies of the effects of NO₂ exposure on infants (ages \leq 2 years) provide no consistent relationship between estimates of NO₂ exposure and the prevalence of respiratory symptoms and disease. In Albuquerque, NM, Samet et al. (1993) conducted a prospective cohort study of infants, during their first 18 months of life, using 2-week average NO₂ concentrations in bedrooms as estimates of exposure to NO₂. In this carefully conducted study, Samet et al. (1993) defined illness events of at least two consecutive days of either runny nose, wet cough, wheezing, or trouble with breathing. The analysis was limited to 1205 subjects completing at least one month of observation. Findings indicate that in a population of healthy infants there was no significant association between NO₂ exposure estimates (in the range 0 to 0.04 ppm NO₂) and respiratory illness when precaution was taken to make an accurate assessment of exposures, to validate measurements of respiratory illness, to eliminate potentially confounding variables, and to adjust for key variables (CD, p. 14-33).

Several other epidemiology studies attempting to associate NO₂ exposure with increased risk of respiratory effects in infants offer mixed results. Presence of a gas stove was

TABLE V-1. HEALTH OUTCOME AND NITROGEN DIOXIDE EXPOSURE MEASURES USED IN SELECTED INDOOR NITROGEN DIOXIDE EPIDEMIOLOGY STUDIES

Reference	Health Outcome Used in Meta-Analysis	Method ^a	NO ₂ Exposure Measure Used in Analysis ^b	Age (years)	Sample Size	Where/When
Melia et al., (1977)	Colds going to chest showed a prevalence of 26.8-19.8%.	Symptoms during past 12 mo recalled by child's parent in completing respiratory symptoms questionnaire.	Gas stove vs. electric stove.	6-11	5,658	28 Areas of England and Scotland (1973)
Melia et al., (1979)	Responses to respiratory questions grouped into (a) none or (b) one or more symptoms or disease types. Colds going to chest (26.4-19.6%) showed the highest prevalence, followed by wheeze (10.1-6.2%), cough, and episodes of asthma or bronchitis in last year.	As above.	Gas stove vs. electric stove.	5-10	4,827	27 areas of England and Scotland (1977)
Melia et al., (1980) Florey et al., (1979) Goldstein et al., (1979)	Group response to respiratory questions as above.	As above.	NO ₂ measured with Palmes tubes. Gas stove homes only.	6-7	103	Middlesborough, England (1978)
Melia et al., (1982a)	As above.	As above.	NO ₂ measured with Palmes tubes. Gas stove homes only.	5-6	188	Middlesborough, England (1978)

Reference	Health Outcome Used in Meta-Analysis	Method ^a	NO ₂ Exposure Measure Used in Analysis ^b	Age (years)	Sample Size	Where/When
Ware et al., (1984)	Lower respiratory illness index (index of respiratory health) indicating during past year the presence of (a) bronchitis, (b) respiratory illness that kept the child home 3 days or more, or (c) persistent cough for 3 mo of the year.	Questionnaire (Ferris, 1978) completed by parent for symptoms during previous 12 mo.	Gas vs. electric.	6-10	8,240	Six U.S. cities (1974-1979)
Neas et al., (1990, 1991)	Combined indicator of one or more lower respiratory symptoms as defined. The highest prevalences were for chronic phlegm and wheeze. The other symptoms in the index are shortness of breath, chronic cough, and bronchitis. Chest illness reflects a restriction of the child's activities for 3 or more days.	Symptom questionnaire completed by parent for the year during which measurements of NO ₂ were taken.	NO ₂ measured with Palmes tubes. Gas and electric stoves.	7-11	1,286	Six U.S. cities (1983-1986)
Ekwo et al., (1983)	Chest congestion and phlegm with colds.	Questionnaire (ATS) completed by parent.	Gas stove vs. electric stove.	6-12	1,138	Iowa City, Iowa
Dijkstra et al., (1990) Brunekreef et al., (1987)	Respiratory illness combination variable of presence of one or more of cough, wheeze, or asthma.	Questionnaire (WHO) completed by parent.	NO ₂ measured with Palmes tubes. Gas and electric appliances.	6-12	775	Netherlands (1986)
Keller et al., (1979a,b)	Respiratory illness.	Telephone interview by nurse epidemiologist.	Gas stove vs. electric stove.	<12	176	Columbus, Ohio (1978)

^aATS = American Thoracic Society
^bWHO = World Health Organization

Source: Table 14-18, pp. 14-57 to 14-58 of the CD (U.S. EPA, 1993a).

associated with increased upper respiratory illness in 1-year olds living in northern Scotland (Ogston, et al. 1985), while Melia et al. (1983) found no relationship between use of gas stoves and prevalence of respiratory symptoms in infants under one year old. Margolis et al. (1992) studied the prevalence of persistent respiratory symptoms in 393 infants of different socioeconomic status (SES) living in North Carolina during their first year of life and reported that infants in the low-SES were more likely to have symptoms than high-SES infants, though the difference was statistically significant only for those not in day care. Of the infants studied by Margolis et al. (1992), only 41 lived in homes with gas stoves, and the relative risk of persistent respiratory symptoms among those infants exposed to NO_2 from gas stoves, unadjusted for any covariates, was 1.12 (95% confidence interval of 0.63 to 2.04). The studies of infants discussed above formed the basis for a meta-analysis discussed in Section V.C.1.b below.

2) Outdoor Studies

Several epidemiological studies have been conducted in the U.S. and other countries to examine the relationship between ambient NO_2 levels and respiratory health effects. In one part of the Six Cities Study, Dockery et al. (1989b) used data from centrally located ambient monitors to investigate associations between respiratory symptoms and air pollution and found the strongest associations were between respiratory symptoms and particulate matter. Even though the odds ratios for respiratory symptoms (e.g., bronchitis, chronic cough, chest illness) with ambient NO_2 were not statistically significant, the direction was consistent with results reported in the indoor studies.

Several studies conducted in various locations around the U.S. have reported little or no relationship between ambient NO_2 levels and respiratory illness. In a study conducted in Chestnut Ridge, PA, Vedal et al. (1987) reported no quantitative relationship between daily respiratory symptoms (e.g., wheeze, pain on breathing, or phlegm) and ambient NO_2 levels; however,

the number of subjects included in the analysis was only 55. A California Seventh Day Adventist study (Euler et al., 1988) concluded that NO₂ exposure levels were not linked to chronic respiratory disease symptoms. Although the Chattanooga studies conducted by Shy et al. (1970a,b; 1973) offer qualitative evidence of higher respiratory illness rates for families living in higher-NO₂ compared to lower-NO₂ neighborhoods, Pearlman et al. (1971) reported results in Chattanooga that were not completely consistent with the exposure gradient because rates of bronchitis were just as high in the intermediate-pollution area as in the high-pollution area.

In contrast, several European studies have investigated and found a relationship between respiratory disease and ambient NO₂ levels, albeit with the contribution of other pollutants. In a study conducted in Switzerland, Braun-Fahrlander et al., (1989, 1992) found that outdoor levels of NO₂ were predictive of duration of respiratory disease episodes. In two studies conducted in Germany, Schwartz et al. (1991) showed a relationship between short-term fluctuations in ambient NO₂/particulate matter levels and medical visits for croup symptoms, while Rebmann et al. (1991) reported a relationship between croup with positive virologic testing and ambient NO₂ levels. Finally, in Finland Jaakkola et al. (1991) report a significant association between the occurrence of upper respiratory infections and living in an air-polluted area for both infants and 6-year old children. The authors did conclude that other pollutants (e.g., particulate matter, SO₂, H₂S, etc.) may have contributed to the effects.

Outdoor epidemiological studies do appear to provide limited evidence of an association between ambient exposures to NO₂ and increases in respiratory symptoms and illness. However, uncertainties regarding actual exposures to NO₂ and the extent to which other factors (e.g., other pollutants, allergens, weather) may have contributed tend to limit development of a quantitative

relationship. Thus, staff concludes that this information should be factored into developing an adequate margin of safety.

b. Meta-Analysis of Gas Stove Studies

Conclusions of the individual epidemiological studies cited above and others discussed in Chapter 14 of the CD are somewhat mixed with regard to the effect of NO₂ on lower respiratory symptoms and disease. However, most of the indoor studies used in a synthesis of evidence, also referred to as a meta-analysis, conducted by Hasselblad et al. (1992) showed significantly increased respiratory disease rates associated with increased NO₂ exposure in children (ages 5 to 12) but not in infants. As previously mentioned, human exposure patterns differ significantly for the indoor environment in a home with a gas stove versus a typical outdoor environment. Therefore, extrapolation of the results of the meta-analysis to ambient conditions is difficult. A detailed discussion of the meta-analysis can be found in the CD (pp. 14-55 to 14-78), and only a summary will be provided here.

The standard health effect endpoint chosen for the analysis was the presence of lower respiratory symptoms and illness. Requirements for inclusion of a study in the analysis were: (1) the health endpoint of the study must be reasonably close to the standard endpoint; (2) significant exposure differences between subjects must exist and some estimate of exposure must be available; and (3) an odds ratio for a specified exposure estimate must have been calculated or data must be presented so that an odds ratio can be calculated. Table V-2 is a summary of studies selected for the meta-analysis and of estimated odds ratios and confidence intervals for each.

Meta-analysis of data from the selected gas-stove studies gives an estimated odds ratio of 1.2 (95% confidence limits of 1.1 and 1.3) for increased lower respiratory symptoms and illness in children ages 5 to 12 (Hasselblad et al., 1992). This odds ratio corresponds to each increase of 0.015 ppm in estimated 2-week average NO₂ exposure, where mean weekly concentrations in

TABLE V-2. SUMMARY OF ODDS RATIOS FROM INDOOR STUDIES OF THE EFFECTS OF NITROGEN DIOXIDE

Author	Estimated Odds Ratio	2.5 and 97.5 Percentiles (Confidence Interval)
Melia et al. (1977)	1.28	1.14 to 1.43
Melia et al. (1979)	1.22	1.08 to 1.37
Melia et al. (1980)	1.49	1.04 to 2.14
Melia et al. (1982a)	1.11	0.84 to 1.46
Ware et al. (1984)	1.07	0.98 to 1.17
Neas et al. (1991)	1.40	1.14 to 1.72
Ekwo et al. (1983)	1.09	0.82 to 1.45
Dijkstra et al. (1990)	0.94	0.70 to 1.27
Keller et al. (1979b)	0.75	0.35 to 1.62

Source: Table 14-19, p. 14-71 of the CD (U.S. EPA, 1993a).

bedrooms in studies reporting NO_2 levels were predominately between 0.008 and 0.065 ppm NO_2 .

For studies of infants 2 years old and younger, a meta-analysis yielded no consistent relationship between estimates of NO_2 exposure and prevalence of respiratory symptoms and illness. The combined odds ratio for increased respiratory disease per increase of 0.015 ppm NO_2 (2-week average) was 1.09 (confidence interval of 0.95 to 1.26). Based on these results which clearly contain the no-effect value of 1.0 (i.e., not statistically significant), the CD (p. 17-78) concluded that the evidence did not suggest an effect in infants comparable to that seen in children 5 to 12 years of age. Results of the both the 5-12 year old and the infant meta-analysis are summarized in Table V-3.

In summary, two major conclusions which can be drawn from the indoor- NO_2 epidemiology studies and the associated meta-analysis are: (1) for studies of children (ages 5 to 12 years), an increased risk of about 20% for developing respiratory symptoms and disease corresponds to each increase of 0.015 ppm NO_2 in estimated 2-week average NO_2 exposure, where mean weekly concentrations in bedrooms reporting NO_2 levels were predominately between 0.008 and 0.065 ppm NO_2 (CD, p. 14-73); and (2) for studies of infants (< 2 years old), although the combined odds ratio estimate is positive (1.09, CI 0.95 to 1.26) for the increase in respiratory disease per increase of 0.015 ppm NO_2 , it clearly contains the no-effect value of 1.0 (i.e., is not statistically significant), and so the evidence does not suggest an effect in infants comparable to that seen in older children (CD, p. 14-75).

Assessing the potential value of the meta-analyses to developing the basis for a NAAQS for NO_2 is limited by several considerations. There remains substantial uncertainty about the actual exposures of subjects in the above studies. The NO_2 levels which were monitored in the gas-stove studies are only estimates of exposure and do not represent actual exposures. The

TABLE V-3. KEY EPIDEMIOLOGICAL STUDIES OF EXPOSURE TO NITROGEN DIOXIDE – SUMMARY OF META-ANALYSIS

NO ₂ (ppm) (Exposure Duration)	Observed Effects	References
0.015-ppm increase, where mean weekly concentrations in bedrooms in studies reporting levels were predominately between 0.008 and 0.065 ppm NO ₂ (in 1- and 2-week integrated average NO ₂ concentration estimating an unspecified long-term average)	A meta-analysis shows increased risk of lower respiratory symptoms/disease in children 5 to 12 years old associated with exposure estimates of NO ₂ levels. The 95% confidence interval of the odds ratio estimated by Hasselblad et al. (1992) was 1.1 to 1.3. Predominant source of exposure contrast is homes with gas stoves vs. homes with electric stoves.	Melia et al. (1977, 1979, 1980, 1982) Ware et al. (1984) Neas et al. (1991) Ekwo et al. (1983) Dijkstra et al. (1990) Keller et al. (1979)
0.015-ppm increase in annual average of 2-week NO ₂ levels, where mean weekly concentrations in bedrooms were predominately between 0.005 and 0.050 ppm NO ₂	In individual indoor studies of infants 2 years of age and younger, no consistent relationship was found between estimates of NO ₂ exposure and the prevalence of respiratory symptoms and disease. Based on a meta-analysis of these infant studies, the combined odds ratio for the increase in respiratory disease per increase of 0.015 ppm NO ₂ was 1.09 with a 95% confidence interval of 0.95 to 1.26. Thus, although the overall combined estimate is positive, it clearly contains the no-effect value of 1.0, (i.e., is not statistically significant); and so we cannot conclude that the evidence suggests an effect in infants comparable to that seen in older children (see Chapter 14).	Samet et al. (1993) Margolis et al. (1992) Dockery et al. (1989) Ogston et al. (1985) Ware et al. (1984) Ekwo et al. (1983) Melia et al. (1983)

Source: Table 16-2, p. 16-14 of the CD (U.S. EPA, 1993a).

studies collected 2-week average NO₂ measurements and, therefore, cannot distinguish between relative contributions to respiratory symptoms and illness of peak and average exposure to NO₂. To the extent that the endpoints investigated are affected by a peak exposure, repeated peak exposures, or a combination of peak and longer-term average exposures, uncertainty is increased in the results and conclusions of the meta-analysis. Another factor to consider is the presence of indoor nitrous acid, which is a byproduct of gas combustion; although nitrous acid might affect the health endpoints observed, very little is known about either health effects or indoor concentrations associated with this substance. Finally, and perhaps most significant to this NAAQS review, is the fact that indoor exposure patterns to NO₂ are quite different compared to outdoor exposure patterns. With much higher peaks and average indoor exposures than would be found outdoors, it is extremely difficult to extrapolate the results of the meta-analysis in a manner which would provide quantitative estimates of health impacts for outdoor exposures to NO₂.

c. Biological Plausibility

Animal toxicology studies, and to a lesser extent controlled human exposure studies, provide evidence for possible underlying mechanisms of NO₂-induced respiratory illness. These studies have shown that exposure to NO₂ can impair components of the respiratory host defense system and increase susceptibility to respiratory infection. The increased respiratory symptoms and illness in children reported in the epidemiology studies cited above may be a reflection of the increased susceptibility to respiratory infection caused by the impact of NO₂ on pulmonary defenses. The studies discussed below, and in greater detail in Chapters 13, 15, and 16 of the CD, provide a plausible biological basis for making such a hypothesis.

Although the lungs are commonly invaded by microorganisms (e.g., viruses, bacteria) which have the potential to initiate respiratory infection, respiratory defense mechanisms play a key role in eliminating the infectious agent, thereby reducing the

risk of illness. Animal studies support the contention that inhalation of NO₂ damages respiratory defense systems, including important components of the host defenses such as the humoral and cell-mediated immune system and the alveolar macrophages (AMs). Animal infectivity studies also have provided evidence that NO₂ exposure increases susceptibility to infection by affecting overall function of the host defense mechanisms.

Humoral and cell-mediated immune systems are essential for both antibody production and secretion of cellular products, which regulate normal defense responses and/or destroy certain invading organisms. Although the pulmonary immune system has not been adequately studied to assess the impact of NO₂ exposure, there is some indication that NO₂ suppresses some systemic immune responses and that these responses may be both concentration and time dependent. Although the cause of suppression was not clear in either study, 4 weeks of continuous exposure to 0.4 ppm NO₂ resulted in significant suppression of antibody production by spleen cells (Fujimaki et al., 1982), and 7 weeks of 7 hour/day exposure to \geq 0.25 ppm NO₂ had a significant systemic effect on cell-mediated immunity in mice (Richters and Damji, 1988, 1990).

When particles deposit below the mucociliary region in the gaseous exchange region of the lungs, the AMs kill and remove viable particles, remove nonviable particles, and process/present antigens to lymphocytes for antibody production. In this area of investigation, animal studies of NO₂ exposure have shown: (1) a decrease in phagocytic ability of AMs at 0.3 ppm for 13 days for 2 hours per day (Schlesinger et al., 1987) but increased phagocytosis at 1.0 ppm for 2 days (Schlesinger, 1987a,b); (2) decreased pulmonary bactericidal activity, altered metabolism, increased numbers of macrophages, and morphological changes (Rombout et al., 1986; Aranyi et al., 1976; Goldstein et al., 1973; Suzuki et al., 1986; Chang et al., 1986; Mochitate et al., 1986; Robison et al., 1990); and (3) morphological changes in the ciliated epithelial cells involved in mucociliary transport following exposures of 0.5 ppm for 7 months (Yamamoto and

Takahashi, 1984), however, mucociliary clearance was not affected for exposures up to 5.0 ppm (Schlesinger et al., 1987).

A series of animal infectivity studies has shown that exposure to NO₂ can increase susceptibility to respiratory infection and result in microbial-induced mortality. These studies involve exposure of animals to varying concentrations and durations of NO₂ followed by exposure to an aerosol laced with an infectious agent (e.g., bacteria or virus). Although the lowest acute (2-hour) exposure to affect bacteria-induced mortality was 2.0 ppm (Ehrlich et al., 1977), subchronic exposures to NO₂ concentrations as low as 0.5 to 1.0 ppm have increased both bacteria-induced and influenza-induced mortality (Ehrlich and Henry, 1968; Ito, 1971; Ehrlich et al., 1977). These as well as numerous other studies (Parker et al., 1989; Gardner et al., 1977a,b, 1979, 1980, 1982; Graham et al., 1987; Jakab, 1987a,b; Motomiya et al., 1973; Miller et al., 1987; Coffin et al., 1977) have provided support for the contention that NO₂ increases microbial-induced mortality by impairing the host's ability to defend the respiratory tract from infectious agents, thereby increasing susceptibility to viral, mycoplasma, and bacterial infections. Using susceptibility to respiratory infection as an index, Gardner et al. (1977a,b) and Coffin et al. (1977) concluded that incidence of mortality was significantly more influenced by concentration of NO₂ than by duration of exposure. These studies however, used a large range of exposure concentrations (0.5 - 28 ppm), beginning above typical ambient concentrations. In the ambient range of exposures, time may be a more important influence than concentration. However, there was no data showing clearly the effect of time on effects of long-term, low-level exposures representing ambient exposure levels. In the urban air, the typical pattern of NO₂ is a low-level baseline exposure on which peaks are superimposed. When the relationship of the peak to baseline exposure and of enhanced susceptibility to bacterial infection was investigated, the results indicated that no simplistic concentration times time

relationship was present, and that peaks had a major influence on the outcome (Gardner, 1980; Gardner et al., 1982; Graham et al., 1987).

Several other animal infectivity studies (Miller et al. 1987; Gardner et al., 1982; Graham et al., 1987) offered evidence which indicated that mice exposed to baseline plus short-term peaks were more susceptible to respiratory infection than either those exposed to control or background levels of NO₂. This research also indicated that the pattern of NO₂ exposure had a major influence on the response. Several of the above-cited animal studies, which have been identified in the CD (p. 16-16) as representing key toxicological effects of exposure to NO₂, are summarized in Table V-4.

Relatively few controlled-exposure studies have been conducted using human subjects exposed to NO₂ and infectious agents. One such study (Goings et al., 1989) examined the effects of NO₂ on pulmonary host defense systems using live attenuated influenza virus and reported a non-statistically significant trend toward elevated rate of infection. Similarly, Frampton et al. (1989a) reported a trend for less effective inactivation of virus by AMs taken from subjects exposed continuously to 0.6 ppm NO₂ for 3 hours, although no effects were reported in those exposed continuously to 0.05 ppm with three 15 minute 2.0 ppm spikes with exercise. In a related investigation, Frampton et al. (1989b) found that 3 hours of exposure to 0.60 ppm NO₂ may transiently increase levels of antiprotease alpha-2-macroglobulin in lung lavage fluid and thereby may alter AM defenses against infection. Although these studies are suggestive, they do not provide clear evidence that NO₂ increases susceptibility of humans to respiratory infection.

The weight of evidence provided by animal toxicology and human exposure studies supports the contention that NO₂ impairs the ability of host defense mechanisms to protect against respiratory infection. Although some of the health endpoints may not be valid for humans (e.g., increased mortality), there are

TABLE V-4. KEY ANIMAL TOXICOLOGICAL EFFECTS OF EXPOSURE TO NITROGEN DIOXIDE^a

NO₂ (ppm) (Exposure Duration)	Species	Observed Effects	References
0.2 ppm (continuous base for 1 year) plus 0.8 ppm (1-h peak, 2x/day, 5 days/week)	Mouse	Increased susceptibility to respiratory infection and decreased vital capacity and respiratory system compliance, compared to control or baseline only	Miller et al. (1987)
0.25 ppm (7 h/day), 5 days/week, 7 weeks)	Mouse	Systemic effect on cell-mediated immunity	Richters and Damji (1988, 1990)
0.3 ppm (2 h/day, 2 days)	Rabbit	Decreased phagocytosis of alveolar macrophages	Schlesinger (1987a,b)
0.4 ppm (continuous, 4 weeks)	Mouse	Decreased systemic humoral immunity	Fujimaki et al., (1982)
0.5 ppm (continuous, 3 mo)	Mouse	Increased susceptibility to respiratory infection	Ehrlich and Henry (1968)
0.5-28 ppm (6 min to 1 year)	Mouse	Linear increase in susceptibility to respiratory infection with time; increased slope of curve with increased concentration; C more important than T	Gardner et al. (1977a,b) Coffin et al. (1997)

^aNO₂ = Nitrogen dioxide.

C = Concentration of exposure.

T = Duration (time) of exposure.

Source: Table-16-3, p. 16-16 of the CD (U.S. EPA, 1993a).

many shared mechanisms between animals and humans which support the hypothesis of association between NO₂ exposure and increases in respiratory symptoms and illness reported in the epidemiological studies. According to the CD (p. 16-9), however, testing this hypothesis would require performing additional animal toxicology studies, conducting more diagnostic tests for infection in future epidemiological studies, and applying additional approaches in controlled human exposure studies.

2. Lung Function, Symptoms and Airway Resistance

At sufficiently high exposures (e.g., 2 to 8 ppm), acute exposure to NO₂ can induce statistically significant pulmonary function decrements, symptomatic effects, and increased airway resistance (R_{aw}) in both healthy and sensitive subjects. Airway resistance is defined as the (frictional) resistance to airflow afforded by the airways between the airway opening at the mouth and the alveoli. Although many controlled human exposure studies of healthy individuals conducted at NO₂ concentrations even above 1.0 ppm report negative results, some studies of asthmatics and patients with chronic obstructive pulmonary disease (COPD) observe effects following exposures to NO₂ at concentrations less than 1.0 ppm.

Human exposure studies summarized in Section 15.2 of the CD (pp. 15-10 to 15-36) provide very limited evidence of functional alterations, symptoms, or R_{aw} changes in healthy subjects exposed for short periods (5 minutes to 2 hours) to NO₂ concentrations ranging from 2 to 8 ppm. Most of the studies cited report no changes in lung function or symptoms; however, increased R_{aw} was observed after short-term exposures of healthy subjects to 5-8 ppm NO₂ (Von Nieding et al., 1979; Von Nieding and Wagner, 1977; Von Nieding et al., 1980; Islam and Ulmer, 1979 a,b). When healthy subjects were exposed to somewhat lower NO₂ levels (2 to 4 ppm), no changes were reported in either spirometry or R_{aw} (Linn et al., 1985b; Mohsenin, 1987b, 1988). None of the studies investigating healthy subjects exposed to less than 1.0 ppm NO₂ demonstrated any clear responses to NO₂ (CD, p. 15-36).

In contrast to the lack of effects reported with healthy subjects at lower NO_2 levels, there is some evidence that asthmatics experience symptoms, functional changes, and increased R_{aw} when exposed to NO_2 levels below 1.0 ppm (CD, p. 15-36 to 15-65). In one early study of asthmatics, symptoms of respiratory discomfort were experienced by 4 of 13 asthmatics exposed to 0.5 ppm for 2 hours; however, Kerr et al. (1979) concluded that the symptoms were minimal and did not correlate well with functional changes. In several other studies of asthmatics, very small changes in spirometry or plethysmography were reported following acute exposures in the range of 0.1 (Hazucha et al., 1982, 1983) to 0.6 ppm NO_2 (Avol et al., 1988). Hazucha et al. (1982, 1983) found an eight percent increase in specific airway resistance (SR_{aw}) after mild asthmatics were exposed to 0.1 ppm NO_2 at rest. However, this finding is not considered statistically significant. Bauer et al., (1986) reported statistically significant changes in spirometric response in mild asthmatics exposed for 20 minutes (with mouthpiece) to 0.3 ppm NO_2 and cold air. Avol et al. (1988) found significant changes in SR_{aw} and FEV_1 as a function of exposure concentration and duration for all exposure conditions (i.e. exposure of moderately exercising asthmatics for 2 hours to 0.3 ppm and 0.6 ppm NO_2); however, it was concluded that there was no significant effect of NO_2 exposure on these measures of pulmonary function (CD, p. 15-47). Exercising adolescent asthmatics exposed (with mouthpiece) to air, 0.12 ppm and 0.18 ppm NO_2 exhibited small changes in FEV_1 , but there were no differences in symptoms between air and either of the NO_2 exposures (Koenig et al., 1987a,b). The absence of spirometry or plethysmography changes in studies (Avol et al., 1986; Bylin et al., 1985; Linn et al., 1985b; Linn et al., 1986) conducted at higher NO_2 concentrations makes developing a concentration-response relationship problematic (CD, p. 15-62).

Patients with COPD also have been used as subjects in NO_2 exposure studies. Due to the hyperresponsiveness of their airways to physical and chemical stimuli, their already

compromised lung function, and the poor distribution of ventilation leading to greater NO₂ delivery to the segment of the lung that is well ventilated (thus resulting in a greater local dose), patients with COPD might be expected to experience a heightened response to NO₂ exposures compared to healthy individuals. Early studies (Von Nieding et al., 1970, 1971, 1973; Von Nieding and Wagner, 1979) found increased R_{aw} with exposure of COPD patients to 1.6 ppm NO₂ or greater. However, in a more recent comparative study of healthy and bronchitic subjects, Von Nieding et al. (1980) reported that responses of subjects with bronchitis were similar to those seen in healthy subjects. Linn et al. (1985a) investigated effects of a 1-hour exposure to 0.5, 1.0, and 2.0 ppm NO₂ on intermittently exercising patients with emphysema and chronic bronchitis; they found no statistically significant changes in arterial oxygenation, lung function, or symptoms. Finally, Morrow and Utell (1989) exposed COPD patients for 3.75 hours to 0.3 ppm NO₂ during intermittent mild exercise and found progressive and statistically significant decrements in FVC and FEV₁ during and after exposure.

In summary, in healthy individuals, even very high acute NO₂ exposures do not appear to cause pulmonary function effects, symptoms, or increases in R_{aw}. The current database does, however, show that small pulmonary function changes have occurred in asthmatics at low, but not high (i.e., up to 4 ppm), NO₂ concentrations. Although the observed effects were noted in different studies, no plausible explanation is offered to account for this lack of a concentration-response relationship. The CD, in assessing the available data on pulmonary function responses to NO₂ in asthmatic individuals, concludes that the most significant responses to NO₂ that have been observed in asthmatics have occurred at concentrations between 0.2 and 0.5 ppm (CD, pg. 16-3). Patients with COPD experience pulmonary function changes with brief exposure to high concentrations (5 to

8 ppm for 5 minutes) or with more prolonged exposure to lower concentrations (0.3 ppm for 3.75 hours).

3. Increased Airway Responsiveness

There is little, if any, convincing evidence that healthy individuals experience increases in airway responsiveness when exposed to NO₂ levels below 1.0 ppm. However, studies of asthmatics have reported some evidence of increased airway responsiveness caused by acute exposure to NO₂ in the range of 0.2 to 0.3 ppm. A detailed discussion of these responses is provided in Chapter 15 of the CD.

Responsiveness of an individual's airways is typically measured by evaluating changes in airway resistance or spirometry following challenge with a pharmacologically active chemical (e.g., histamine, methacholine, carbachol), which causes constriction of the airways. Airway hyperresponsiveness is reflected by an abnormal degree of airway narrowing caused primarily by airway smooth muscle shortening in response to nonspecific stimuli. Asthmatics experience airway hyperresponsiveness to certain chemical and physical stimuli and have been identified as one of the population subgroups which is most sensitive to acute NO₂ exposure (CD, p. 16-1).

Evidence of increased airway responsiveness in normal adults has been reported in very few studies. Mohsenin (1988) found increased airway responsiveness to methacholine following exposure to 2 ppm NO₂ for 1 hour at rest. Frampton et al. (1991) reported statistically significant airway responsiveness following a 3-hour exposure of healthy subjects to 1.5 ppm NO₂ and carbachol challenge, but no increase in airway responsiveness was reported with exposure to 0.6 ppm NO₂. In one study (Hazucha et al., 1992), airway responsiveness, which was subsequently induced by a 2-hour exposure to 0.3 ppm O₃, was augmented by a 2-hour preexposure to 0.6 ppm NO₂. None of these studies, however, provide clear evidence of increased airway responsiveness in normal individuals when exposed only to NO₂ at concentrations of 1 ppm or lower.

There is evidence that exposure of mildly allergic asthmatic patients to NO_2 levels below 1 ppm may cause increased airway responsiveness. Several controlled exposure studies (Ahmed et al., 1983a,b; Bylin et al., 1985; Hazucha et al., 1982, 1983; Koenig et al., 1985; Orehek et al., 1981) of asthmatics showed no significant effect on responsiveness at very low NO_2 concentrations of 0.1 to 0.12 ppm. Using a mouthpiece and somewhat higher exposures of 0.3 ppm NO_2 , Bauer et al. (1986) reported a statistically significant response to NO_2 after 20 minutes rest followed by 20 minutes of exercise (30 L/min); all had elevated response to cold air bronchoprovocation. A subsequent study (Morrow and Utell, 1989), using some of the same subjects in the same laboratory, showed no change in lung function, symptoms, or carbachol reactivity following exposure to 0.3 ppm NO_2 . It is important to note, however, that the Morrow and Utell (1989) study was a chamber study; thus, the difference in exposure mode (mouthpiece vs. chamber) could account for the significant difference in the study results. Studies which investigated concentration-response relationships for exposures of ≤ 0.6 ppm NO_2 (Roger et al., 1990; Bylin et al., 1985, 1988; Avol et al., 1988) found no significant changes in spirometry or airway reactivity as a result of NO_2 exposure. Even for exposures to 3.0 ppm (Linn et al., 1986) and 4.0 ppm NO_2 (Linn and Hackney, 1984), no effects of NO_2 on SR_{aw} , symptoms, heart rate, or skin conductance were reported in exercising asthmatics.

Folinsbee (1992) analyzed data on asthmatics experimentally exposed to NO_2 in various studies which used challenges producing increased airway responsiveness in 96 subjects and decreased airway responsiveness in 73 subjects. For exposures in the range of 0.2 to 0.3 ppm NO_2 , he found that the excess increase in airway responsiveness was attributable to subjects exposed to NO_2 at rest. Because NO_2 at these levels does not appear to cause airway inflammation and the increased airway responsiveness appears fully reversible, implications of the observed increases in responsiveness remain unclear. It has been hypothesized that

increased nonspecific airway responsiveness caused by NO₂ could lead to increased responses to a specific antigen; however, there is no plausible evidence to support this.

In summary, there is some evidence that acute exposure to NO₂ may cause an increase in airway responsiveness in asthmatic individuals. This response has been observed only at relatively low NO₂ concentrations, mostly in the range of 0.2 to 0.3 ppm NO₂. However, the above findings, taken as a whole, do not provide any clear quantitative conclusions about the health effects of short-term exposures to NO₂.

4. Emphysema

Although no attempt has been made to quantitatively assess the potential risk of NO₂-induced emphysema to humans, the CD (p. 16-9) clearly states that because emphysema is an irreversible disease representing an important public health concern, the potential for risk warrants discussion. Investigations of chronic exposures of animals to NO₂ levels much higher than those found in the ambient air have demonstrated that NO₂ can cause morphologic lung lesions that meet the criteria for a human model of emphysema (which requires the presence of alveolar wall destruction in addition to enlargement of the airspace distal to the terminal bronchiole). However, only a few studies cited in Chapter 13 of the CD document emphysema (according to the human definition) resulting from chronic NO₂ exposure. Three studies (Haydon et al., 1967; Freeman et al., 1972; Port et al., 1977), in which rats and rabbits were exposed for periods ranging from 1 month to more than 30 months to NO₂ concentrations greater than 8 ppm, showed morphologic lung lesions that meet the criteria for a human model of emphysema. Another study (Hyde et al., 1978) provided evidence of emphysema (including alveolar destruction) in dogs exposed to a mixture of 0.64 ppm NO₂ and 0.25 ppm NO for a period of 68 months. The dogs were examined 2.5 to 3 years after exposure to the mixture. The finding that pulmonary function decrements progressed post-exposure suggests that morphological effects may have been progressive also. In the

same study (Hyde et al., 1978), a low NO₂ (0.14 ppm) and high NO (1.1 ppm) mixture produced no evidence of emphysema, thus implying that NO₂ was a significant etiologic factor. Numerous other studies cited in Chapter 13 of the CD reported emphysema, but they either lacked sufficient detail for independent conclusions to be drawn or did not meet all of the criteria for human emphysema.

In conclusion, it is clear that at sufficiently high concentrations of NO₂ (i.e., > 8 ppm) for long periods of exposure, NO₂ can cause emphysema (meeting the human definition criteria) in animals. Although current information does not permit identification of the lowest NO₂ levels and exposure periods which might cause emphysema, it is apparent that levels required to induce emphysematous lung lesions in animals are far higher than any NO₂ levels which have been measured in the ambient air.

D. Populations Potentially at Risk

Two general groups in the population may be more susceptible to the effects of NO₂ exposure than other individuals. These groups include persons with pre-existing respiratory disease and children (5-12 years old). Individuals in these groups appear to be affected by lower levels of NO₂ than individuals in the rest of the population.

With regard to decreased respiratory function caused by NO₂, a reasonable hypothesis of enhanced susceptibility to NO₂ has been offered for those with preexisting respiratory disease. Since these individuals live with reduced ventilatory reserves, any reductions in pulmonary function caused by exposure to NO₂ have the potential to further compromise their possibly marginal health status. Compared to healthy individuals with normal ventilatory reserves who may not notice small reductions in lung function, those with preexisting respiratory disease may be prevented from continuing normal activity following exposure to NO₂.

Numerous epidemiological studies conducted in homes with gas stoves provide evidence that children (5-12 years old) are at increased risk of respiratory symptoms/illness from exposure to elevated NO_2 levels (Melia et al., 1977, 1979, 1983; Ekwo et al., 1983; Ware et al., 1984; Ogston et al., 1985; Dockery et al., 1989a; Neas et al., 1990, 1991, 1992; Dijkstra et al., 1990; Brunekreef et al., 1987; Samet et al., 1993). Because childhood respiratory illness is very common (Samet et al., 1983; Samet and Utell, 1990), any impact which NO_2 might have of increasing the probability of respiratory illness in children is a matter of public health concern. This is particularly true in light of evidence that recurrent childhood respiratory disease may be a risk factor for later susceptibility to lung damage (Glezen, 1989; Samet et al., 1983; Gold et al., 1989) (CD, p. 16-4). In the United States, there are approximately 35 million children in the age group 5 to 14 years (Centers for Disease Control, 1990).

Airway inhalation challenge tests have been used to evaluate the responsiveness of asthmatics' airways and have demonstrated that asthmatics are hyperresponsive to a variety of inhaled substances (e.g., pollens, cold/dry air, dust, and air pollutants). With regard to NO_2 , asthmatics are considered to be one of the groups in the population most responsive to NO_2 exposure (CD, p. 16-1). There is evidence that asthmatics exposed to low levels of NO_2 (0.2 to 0.3 ppm) will experience an increase in airway responsiveness (Folinsbee, 1992). The National Institutes of Health (1991) estimates that approximately 10 million asthmatics live in the U.S. Because asthmatics tend to be much more sensitive to inhaled bronchoconstrictors than nonasthmatics, there is the added concern that NO_2 -induced increase in airway response may exacerbate already existing hyperresponsiveness caused by preexposure to other inhaled materials.

Patients with chronic obstructive pulmonary disease (COPD) constitute another subpopulation which is potentially susceptible to NO_2 exposure. This group, which is estimated to be 14 million

in the U.S. (U.S. Department of Health and Human Services, 1990), includes persons with emphysema and chronic bronchitis. One of the major concerns for COPD patients is that they do not have an adequate ventilatory reserve and, therefore, would tend to be more affected by any additional loss of ventilatory function as may result from exposure to NO_2 . It is also possible that NO_2 might further damage already impaired host defense mechanisms, thus putting COPD patients at increased risk to lung infection.

A final subpopulation group which the CD (p. 1-24) has identified as potentially susceptible to NO_2 is immunocompromised individuals. Persons in this group would have an increased susceptibility for infectious pulmonary disease as well as other health effects. Examples of such individuals would be those suffering from acquired immune deficiency syndrome (AIDS) and cancer patients being treated with chemotherapy. Approximately 1 million persons were estimated by the Centers for Disease Control (CDC, 1990) to be infected with the human immunodeficiency virus, which precedes development of AIDS symptoms (Karon et al., 1990). There are about 1 million patients diagnosed with cancer each year (U.S. Bureau of the Census, 1991), about 25% of whom are prescribed chemotherapy as a first course of treatment (Steele et al., 1991). Although the above immuno-compromised groups represent populations potentially at risk for NO_2 effects, no human research has examined the effects associated with NO_2 exposure in these groups. Additionally, no such studies have been conducted in similarly immuno-compromised animals exposed to NO_2 . Although it is clear that NO_2 can affect alveolar macrophages, humoral immunity, and cell-mediated immunity in otherwise normal animals (Chapter 13), the animal-to-human extrapolation cannot yet be made quantitatively. Thus, there only now exists a hypothesized association with increased susceptibility to NO_2 . Nevertheless, it may be prudent to consider including such reduced immune function groups as susceptible subpopulations at potentially increased risk for NO_2 -induced health effects" (CD, p. 1-25). Table V-5 summarizes

TABLE V-5

SUMMARY OF POTENTIALLY SENSITIVE GROUPS

SENSITIVE GROUP	SUPPORTING EVIDENCE	REFERENCES FOR SUPPORTING EVIDENCE	POPULATION ESTIMATES
CHILDREN	Exposure to NO ₂ in homes with gas stoves increases risk of children developing respiratory symptoms and disease.	Melia et al., 1977, 1979, 1980, 1982 Ware et al., 1984 Neas et al., 1991 Ekwo et al., 1983 Dijkstra et al., 1990 Keller et al., 1979	Ages 5-14 ¹ 35 million
ASTHMATICS	Asthmatics exhibit greater functional decrements and airway responsiveness at lower levels of NO ₂ than nonasthmatics.	Kerr et al., 1979 Folinsbee, 1992 Bauer et al., 1986 Koenig et al., 1987a,b	10 million ²
Patients with Chronic Obstructive Pulmonary Disease (COPD)	Patients with COPD experience pulmonary function changes with acute exposures to high NO ₂ levels or prolonged exposures to lower levels	Morrow and Utell, 1989 Von Nieding et al., 1980	14 million ³
Immunocompromised Individuals	Immunocompromised individuals tend to be more susceptible to lung infection and other health effects	CD, p. 1-141	1 million infected with HIV, ⁴ 1/4 million using chemotherapy ⁵

¹ Centers for Disease Control, 1990.

² National Institutes of Health, 1991

³ U.S. Department of Health and Human Services, 1990

⁴ Karon et al., 1990

⁵ U.S. Bureau of the Census, 1991, Steele et al., 1991

evidence and population estimates for groups identified above.

E. Effects of Concern

As discussed in detail in the CD (U.S. EPA, 1993a) and in earlier sections of this Staff Paper, there is substantial scientific literature providing evidence of health effects associated with exposure to NO₂. These effects vary widely depending on the level of exposure, pattern of exposure, and conditions of subjects during exposure (e.g., ventilation rate, sensitivity). Although scientific research provides an essential basis upon which conclusions can be drawn relating various health effects to exposure patterns and levels of exposure to NO₂, the specific level, averaging time, and form of any primary NAAQS are largely a public health policy judgment of the Administrator.

In Chapter 16 of the CD (U.S. EPA, 1993a), key information and conclusions of the health assessment chapters are summarized and integrated to provide a framework for decisions regarding health risks of NO₂. That discussion provides the basis for the following conclusions:

1. Evidence is insufficient at this time to establish clear quantitative conclusions regarding airway responsiveness and pulmonary function changes resulting from short-term exposures of healthy and asthmatic individuals to near-ambient NO₂ levels. (CD, pp. 16-3 and 16-4)

Evidence of health effects occurring below 1.0 ppm NO₂ is equivocal at best in healthy individuals. There is limited evidence of increased airway responsiveness in asthmatics following short-term exposures as low as 0.2 to 0.3 ppm NO₂ (Folinsbee et al., 1992), even though changes in airway responsiveness have not been observed at much higher concentrations of up to 4 ppm (CD, p. 16-3). The implications of this increased responsiveness in asthmatics, however, remain unclear due to the apparent total reversibility of acute effects and the apparent lack of airway inflammation at low-level NO₂ exposures.

Although no consistent pattern of pulmonary function response in normal subjects has been reported for short-term exposure studies conducted at < 1.0 ppm NO_2 (CD, p. 15-36), there is limited evidence of small changes in lung function in asthmatics following short-term exposures to NO_2 . Most of the responses to NO_2 in asthmatic individuals have been observed at concentrations between 0.2 and 0.5 ppm (CD, pg. 16-3). The significance of these findings, however, is somewhat diminished by the absence of changes reported at higher (3 to 4 ppm) NO_2 concentrations (Bylin et al., 1985; Linn et al., 1984, 1985 a, b, 1986) and the apparent lack of a concentration-response relationship for pulmonary function changes.

2. Although there is substantial evidence which suggests that increased exposure to NO_2 is associated with increased respiratory illness in 5- to 12-year old children, exposure estimates appear to be inadequate to establish a quantitative relationship between estimated exposures and symptoms. (CD, 16-5)

As described in detail in Chapter 14 of the CD, the meta-analysis of nine "gas stove" epidemiological studies (Melia et al., 1977, 1979, 1980, 1982; Ware et al., 1984; Neas et al., 1991; Ekwo et al., 1983; Dijkstra et al., 1990; Keller et al., 1979) is supportive of a relationship between estimates of exposure to NO_2 and respiratory symptoms and disease in children aged 5 to 12, but not in infants (< 2 years old). Furthermore, underlying mechanisms which support NO_2 exposure as a cause of increased susceptibility to respiratory infection in animals provide a plausible biological hypothesis for such effects occurring in humans (CD, 16-5). However, the "gas stove" studies do not provide sufficient exposure information, including human activity patterns, to establish whether the health effects are related primarily to peak, repeated peak, or long-term exposures to NO_2 . Also, extrapolation of indoor exposure to outdoor exposure is difficult according to the CD (p. 16-5) and CASAC (1993).

3. Animal toxicology studies provide qualitative evidence of NO₂-induced morphologic lung lesions indicative of emphysema; however, NO₂ exposures which have been reported to cause emphysema in animals are far greater than levels currently measured in ambient air. (CD, p. 16-11)

Morphologic lung lesions that meet the NIH (1985) workshop criteria for a human model of emphysema (i.e., alveolar wall destruction occurs in addition to other characteristic changes) have been reported in rats and rabbits following exposures of 1 to 30 months to NO₂ concentrations of > 8 ppm (Haydon et al., 1967; Freeman et al., 1972; Port et al., 1977). Another study (Hyde et al., 1978) in which dogs were exposed to a mixture of 0.64 ppm NO₂ and 0.25 ppm NO for up to 68 months suggested that NO₂ was the cause of morphologic lesions that meet the criteria for human emphysema 32 to 36 months after a 68-month exposure.

Although there are numerous other NO₂ investigations in which emphysema has been reported and several which have found no emphysema, it is not possible at this time to establish a "no-observed effect" level. Reasons for this cited by the CD (p. 16-11) include: complexity of changes occurring with NO₂ exposure, the lack of published papers utilizing appropriate morphometric techniques, interspecies differences in response, and inadequate description of methods and findings in some published reports. Although it is clear that at sufficiently high concentrations NO₂ can induce emphysematous lesions, the levels of NO₂ which are known to cause emphysema are far higher than those which have been reported in the ambient air.

4. There are other factors which staff believes should be considered in evaluating the adequacy of current NAAQS for NO₂.

First, the Administrator should take into consideration the range of health effects associated with NO₂ exposure and that these effects vary widely depending on the level of exposure, pattern of exposure, and condition of subjects during exposure (e.g., ventilation rate, sensitivity). Second, ethical considerations in selecting subjects for studies suggest that the

most sensitive individuals have not been studied. Finally, the staff must reemphasize that most effects of NO₂ which have been reported in animal studies have not yet been demonstrated in human studies (e.g., immunological effects).

VI. AVERAGING TIME AND FORM OF THE PRIMARY NAAQS

A. Averaging Time of the Primary NAAQS

When the EPA promulgated the NO₂ primary NAAQS in 1971 (36 FR 8186, April 30, 1971), an annual (arithmetic mean) averaging time was selected. The annual standard was selected in part because epidemiology studies (Shy et al., 1970 a,b) conducted in Chattanooga reported small, but statistically significant, decreases in FEV for children (ages 7 to 8) living in areas with relatively high (> 0.06 ppm) annual average NO₂ levels. Follow-up studies (Shy et al., 1973, 1978; Pearlman et al., 1971) failed to support the earlier findings, and the measurement method used in the studies was called into question.

When the decision was made on June 19, 1985 to retain then-existing primary NAAQS for NO₂ (50 FR 25532), emphasis was placed on animal studies which provide evidence of health effects associated with chronic high-level NO₂ exposures (e.g., emphysematous-like lesions in the lungs and damage to host defense mechanisms resulting in increased susceptibility to infection). Other evidence suggesting health effects associated with long-term, low-level NO₂ exposures, such as the Chattanooga community epidemiology studies and the studies of children living in homes with gas stoves, were viewed as providing limited qualitative support for a long-term standard. Although there was substantial debate regarding the need for a short-term (1- to 3-hour) primary NAAQS, it was concluded that there was insufficient evidence to set a short-term standard until further research could be conducted. Based on the data available in 1985, retaining the annual NAAQS of 0.053 ppm was seen as a means of providing protection from long-term health effects and some measure of protection against possible short-term health effects (50 FR 25541, June 19, 1985).

Subsequent research has provided little, if any, evidence of significant health effects of short-term exposures to NO₂. No consistent pattern of response in normal subjects has been reported for short-term exposure studies conducted at < 1.0 ppm NO₂ (CD, p. 15-36). There is only limited evidence of small changes in spirometry or plethysmography in asthmatics following short-term exposures to 0.2 to 0.5 ppm NO₂. The significance of these findings, however, is diminished by the absence of changes reported at higher (up to 4 ppm) NO₂ concentrations (CD, pg. 16-3). Patients with chronic obstructive pulmonary disease experience pulmonary function changes with brief exposures to high concentrations (i.e., 5 to 8 ppm for 5 minutes) or with more prolonged exposure to lower concentrations (0.3 ppm for 3.75 hours). As the criteria document discussed at some length, several questions remain unanswered regarding the correlation of disease state with exposure variables for both persons with asthma and COPD patients (CD, pg. 15-62).

The meta-analysis presented in Chapter 14 of the CD (U.S. EPA, 1993a) provides reasonably good evidence of a quantitative relationship between indoor NO₂ exposure in homes with gas stoves and increased respiratory illness in children living in those homes. However, as noted by the CASAC at the July 1993 meeting (CASAC, 1993), it is very difficult to generalize this relationship to outdoor NO₂ exposures. The exposure patterns encountered in the homes studied with gas stoves are different from exposure patterns which would be encountered in the ambient air. Also, in spite of the large number of studies which have focused on respiratory effects experienced by children living in homes with gas stoves, there remains considerable uncertainty with regard to the averaging time(s) associated with these effects. Although it is reasonable to hypothesize that repeated exposure to peak levels of NO₂ may be responsible for increased respiratory illness, the contribution of low-level chronic exposures to NO₂ cannot be ruled out at this time. It is, therefore, not feasible for staff to recommend a NAAQS for NO₂

with a short-term averaging time due to the lack of adequate information indicating that acute health effects of concern occur at or near ambient exposures.

With regard to chronic effects, a relatively large data base, including epidemiology and animal toxicology studies, provides evidence of significant biological effects resulting from long-term (months to years) exposure to NO₂. Among the best documented of these effects is damage to host defense mechanisms which can result in increased susceptibility to respiratory illness. These effects are discussed in detail in Chapters 13, 14 and 16 of the CD (U.S. EPA, 1993a). In addition, chronic exposure to NO₂ has been shown to produce changes in lung biochemistry, pulmonary function, and a variety of extrapulmonary changes. Finally, chronic NO₂ exposure has induced emphysema-like lesions in the lungs of experimental animals but only at concentrations much higher than ambient levels.

In light of the above information, staff concludes that there is a need to protect public health against short-term and long-term exposures to NO₂. This has been accomplished previously by setting the NAAQS for NO₂ at an annual average, which if met would adequately control most peak NO₂ exposures. Staff again believes that this approach is appropriate.

B. Form of the Primary NAAQS

The current annual primary NAAQS for NO₂ is based on the arithmetic mean of all valid hourly averages in a calendar year. The arithmetic mean is more sensitive to repeated short-term peaks than the alternative, which is the geometric mean, and its use is consistent with other standards.

If the Administrator chooses to establish a short-term (e.g., 1-hour, 8-hour, 24-hour) primary standard to protect the public from exposure to repeated NO₂ peak concentrations, then the staff recommends that the standard be set in a statistical form rather than a deterministic form. (The statistical form of the standard offers a more stable target for control programs and is less sensitive to truly unusual meteorological conditions than

a deterministic form). This could be accomplished by (1) setting a NAAQS with an allowable number of exceedances of the standard level which would be expressed as an average or expected number per year, or (2) setting a NAAQS where a given percent of the daily maximum hourly values would be expected to be less than or equal to the standard level. The emissions reductions achieved in the required control implementation program would be based on a statistical analysis of the monitoring data over a multi-year period (e.g., the preceding 3-year period).

VII. SUMMARY OF STAFF CONCLUSIONS AND RECOMMENDATIONS FOR THE NITROGEN DIOXIDE PRIMARY NAAQS

The key findings presented below concerning the NO₂ primary NAAQS draw upon discussions contained in previous sections of this Staff Paper. The key findings are:

1) The staff concludes that exposure to NO₂ is associated with a variety of acute (short-term) and chronic (long-term) health effects. Clearly adverse health effects (e.g., pulmonary edema, death) have been reported following accidental short-term exposures of humans to very high concentrations (150-200 ppm or greater) of NO₂ (CD, pg. 14-55) and animal studies have provided evidence of emphysema caused by long-term exposures to greater than 8 ppm NO₂. However, these health effects are caused by exposures which are much higher than those found in the ambient air and only serve as indicators of the most extreme effects of a potentially very noxious oxidant.

2) Based on the assessment of available information regarding health effects associated with exposure to NO₂, the staff concludes that the two potentially sensitive population at risk are children 5 to 12 years old and individuals with pre-existing respiratory diseases.

3) The available data indicate that short-term exposure to NO₂ may cause an increase in airway responsiveness in asthmatic individuals at rest. This response has been reported only at relatively low concentrations (mostly within the range of 0.2 to

0.3 ppm NO₂) which are of concern in the ambient environment. Similarly, NO₂ induced pulmonary function changes in asthmatic individuals have been reported at low, but not high, NO₂ concentrations. For the most part, the small changes in pulmonary function observed in asthmatic individuals have occurred at concentrations between 0.2 and 0.5 ppm, but not at much higher concentrations (i.e., up to 4 ppm) (CD, pg. 16-3). These findings contrast with the findings for healthy individuals exposed to low NO₂ concentrations. In healthy individuals, there is no evidence of lung function decrements or changes in airway responsiveness at concentrations below 1.0 ppm NO₂.

The epidemiological evidence includes a meta-analysis of nine epidemiological studies of children (5-12 years old) living in homes with gas stoves. The results of the meta-analysis show the children (5-12 years old) living in homes with gas stoves are at increased risk for developing respiratory diseases and illnesses compared to children living in homes without gas stoves. Typical mean weekly NO₂ concentrations in bedrooms in studies reporting NO₂ levels were predominately between 0.008 and 0.065 ppm NO₂. Most of the NO₂ measurements reported in the studies are for indoor 1- to 2-week averages, but very little information is provided regarding either peak hourly exposures or measurements providing estimates of annual exposures. Because of this, the studies and, therefore, the meta-analysis cannot distinguish between relative contributions of peak and longer term exposure and their relationship with the observed health effects. Given the uncertainty associated with determining actual exposure patterns in these homes and the difficulty in extrapolating the data to ambient exposures, results of the meta-analysis provide insufficient data to support specific limits for either short-term or long-term standards for nitrogen dioxide.

4) For the reasons discussed in section VI of this Staff Paper, the staff recommends that an annual arithmetic mean averaging time be retained for the primary NO₂ NAAQS.

5) Based on the assessment of the available scientific and technical information (which remains largely unchanged since the 1985 review), the staff again recommends that consideration be given to setting the level of the annual primary standard within the range of 0.05 to 0.08 ppm NO₂. In the staff's judgement, selecting a standard within this range would provide adequate protection against the health effects associated with long-term NO₂ exposure. In reaching a determination on the standard, the staff also recommends that consideration be given to the degree of protection that would be provided against repeated short-term peak exposures. Based on air quality analyses, a standard selected from the lower portion of the range suggested would effectively limit the frequency and magnitude of 1-hour NO₂ concentrations. McCurdy (1994) estimated that if the existing standard of 0.053 ppm NO₂ is attained, the occurrence of 1-hour NO₂ values greater than 0.2 ppm would be unlikely in most areas of the country. At the upper end of the range, the frequency of 1-hour NO₂ peaks of 0.2 or higher could increase significantly. However, because all areas of the country reporting NO₂ air quality data are attaining the existing standard and because of the nonlinear relationship of 1-hour peaks and annual averages, it is not possible to estimate with any degree of confidence what the frequency and magnitude of 1-hour peaks would be if the standard was selected from the upper end of the suggested range. Given this uncertainty, the staff recommends consideration be given to selecting the standard from the lower portion of the range in order to provide a reasonable measure of protection against repeated 1-hour peaks of potential health concern.

VIII. Environmental Effects of Nitrogen Deposition

This section describes the environmental effects attributed to nitrogen deposition and, where possible, sets forth judgments as to which levels of effects may be defined as adverse for standard setting purposes. In addition to the environmental features identified for protection by the secondary standard in the definition of public welfare (see Section 302(h) [42 U.S.C. 7602(h)]), the 1990 Amendments to the Clean Air Act express a new determination on the part of Congress to investigate through research "...short-term and long-term causes, effects, and trends of ecosystems damage from air pollutants..." (see Title IX, Sec. 901(e)). In keeping with this expanded scope, the Staff Paper will address as part of the secondary standard review short- and long-term effects of nitrogen deposition on biological, physical and chemical components of ecosystems and the resulting effect of changes in these components on ecosystems structure and function, as well as the traditional issues of visibility impairment, and materials damage.

A. Introduction

1. Context of Current Standard Review

In the 1982 OAQPS Staff Paper for nitrogen oxides, the staff concluded that "there is inadequate evidence to demonstrate that exposure to NO₂ alone at low levels will lead to significant impacts on growth and yield for commercially important crops and indigenous vegetation..." Additionally, though studies on pollutant combinations showed that synergistic responses could occur, this type of response was extremely variable and was not adequately documented. Therefore, the staff concluded that there existed "insufficient data on combined effects of NO₂ and SO₂ to do a quantitative evaluation of yield reduction for various ambient exposure levels." CASAC concurred that the data did not suggest significant effects of NO₂ on vegetation at or below current ambient levels and that a standard in the form of an annual arithmetic mean at the level of 0.053 ppm would provide

sufficient protection against significant adverse effects on vegetation (50 FR 25532, June 19, 1985).

More recent research in the above areas, as discussed in the 1993 NO_x Criteria Document, continues to demonstrate that pollutant levels for NO alone and in combination with other pollutants in the United States, except in very rare cases, are well below those required to produce direct phytotoxic effects on plant yield or growth. The evidence linking nitrogen deposition with ecological impacts, however, has been growing since the mid-1980's (Skeffington and Wilson, 1988). Since these new concerns have not been evaluated in the context of a secondary NAAQS for nitrogen oxides, the main focus of this Staff Paper will be on the possible cumulative impacts of nitrogen at the ecosystem level.

This Staff Paper is an effort to identify for the Administrator any existing or potentially sensitive ecosystems or ecosystem structures or functions which may be adversely affected by ambient levels of the criteria pollutant, NO₂, its oxides, and other nitrogen inputs. The definition of ecosystems set forth in the discussion on page 10-2 of the NO_x Criteria Document is used.

2. Atmospheric Nitrogen Inputs

Little, if any, research has been initiated to determine what percentage of total nitrogen deposition can be attributed to emissions of nitrogen oxides. However, it is known that there are many natural and anthropogenic sources of nitrogen that are not nitrogen oxide based.

Of the different species of nitrogen oxides present in the atmosphere (CD, Table 9-12, p. 9-141), NO and NO₂ are generally present in highest concentrations in the lower troposphere. Thus, they are considered the most likely to have a potential impact on vegetation. Research on interactions of NO_x species, other than NO and NO₂, with vegetation is sparse. Some other nitrogen-containing species do participate in photochemical reactions and may convert to NO_x in the atmosphere. According to some researchers, this is the case with ammonia (NH₃), which

originates on a global scale through both natural and anthropogenic processes. Another source of atmospheric nitrogen inputs to the environment, and one that has been the focus of growing concern, is nitric acid (HNO_3).

Under ambient air quality conditions observed to date (see Section IV, Air Quality Trends), the current secondary standard for NO_2 is rarely exceeded. However, an analysis performed by McCurdy (1994), shows that areas attaining the current NO_2 NAAQS might still experience multiple exceedances of 1 hour values greater than or equal to 0.15 ppm, and in Los Angeles, have multiple hourly or daily exceedances greater than 0.20 ppm. As will be discussed in Section B.1.a. (Vegetation) it does not appear that the concentration times duration scenarios discussed by McCurdy would be likely to adversely affect vegetation in those areas, though given other factors, some sensitive species might experience temporary foliar injury.

To have an effect, nitrogen that has been released to the atmosphere must enter the ecosystem by either wet or dry deposition. Deposition is primarily as nitrate. One-third to one-half of the emissions of NO_x in the United States are estimated to be removed by wet deposition (Levy and Moxim, 1987; Hicks et al., 1991). The measurement of dry deposition is still very much a contentious issue. Most researchers estimate rates of dry deposition by assuming they are some fraction of wet deposition rates. The assumption that dry deposition is equal to wet deposition is probably reasonable for areas directly adjacent to emissions sources (Summers et al., 1986), but the ratio of dry deposition to the sum of wet and dry deposition may fall as low as 0.2 in locations remote from sources (CD, p. 206). A third, and rarely measured mechanism of deposition that is locally important is the interception or capture of fog or cloud droplets by vegetation. Two studies (Lovett et al., 1982; Woodin and Lee, 1987) recorded the phenomenon of higher concentrations of nitrogen within the cloud droplets than were measured by bulk

deposition gauges, suggesting that bulk precipitation samplers underestimate total deposition.

This paper will limit its focus to a discussion of the mechanisms by which concentrations of nitrogen in the air may affect deposition and how, in turn, nitrogen deposition affects sensitive ecosystems. Thus, the paper will not specifically address how the nitrogen component of acid rain influences ecosystem response.

3. Nitrogen Cycle

Nitrogen is unique among nutrients in that its retention and loss within ecosystems is regulated almost exclusively by biological processes. The processes which make up the "nitrogen cycle" and transform nitrogen as it moves through an ecosystem include: (1) assimilation (2) nitrification (3) denitrification (4) nitrogen fixation and (5) mineralization (see Figure VIII-1 and definitions in Appendix C). In general, the nitrogen cycle is identical in terrestrial, freshwater, and oceanic habitats; only the microorganisms that mediate the various transformations are different (Alexander, 1977).

Mature natural ecosystems are essentially self-sufficient and independent of external additions (CD, p. 10-6). Anthropogenic inputs, by altering amounts of nitrogen moving through the cycle, can upset the relationships that exist among the various components and thus alter ecosystem structure and function. Under natural conditions, nitrogen is added to ecosystems by fixation of atmospheric nitrogen, deposition in rain, from windblown aerosols containing both organic and inorganic nitrogen, and from the absorption of atmospheric NH_3 by plants and soil (Smith, 1980). However, modern technology is altering the cycle by changing the amounts and fluxes of nitrogen in the various portions of the cycle. The effects that these perturbations are having on the nitrogen cycle are outlined in the discussion below.

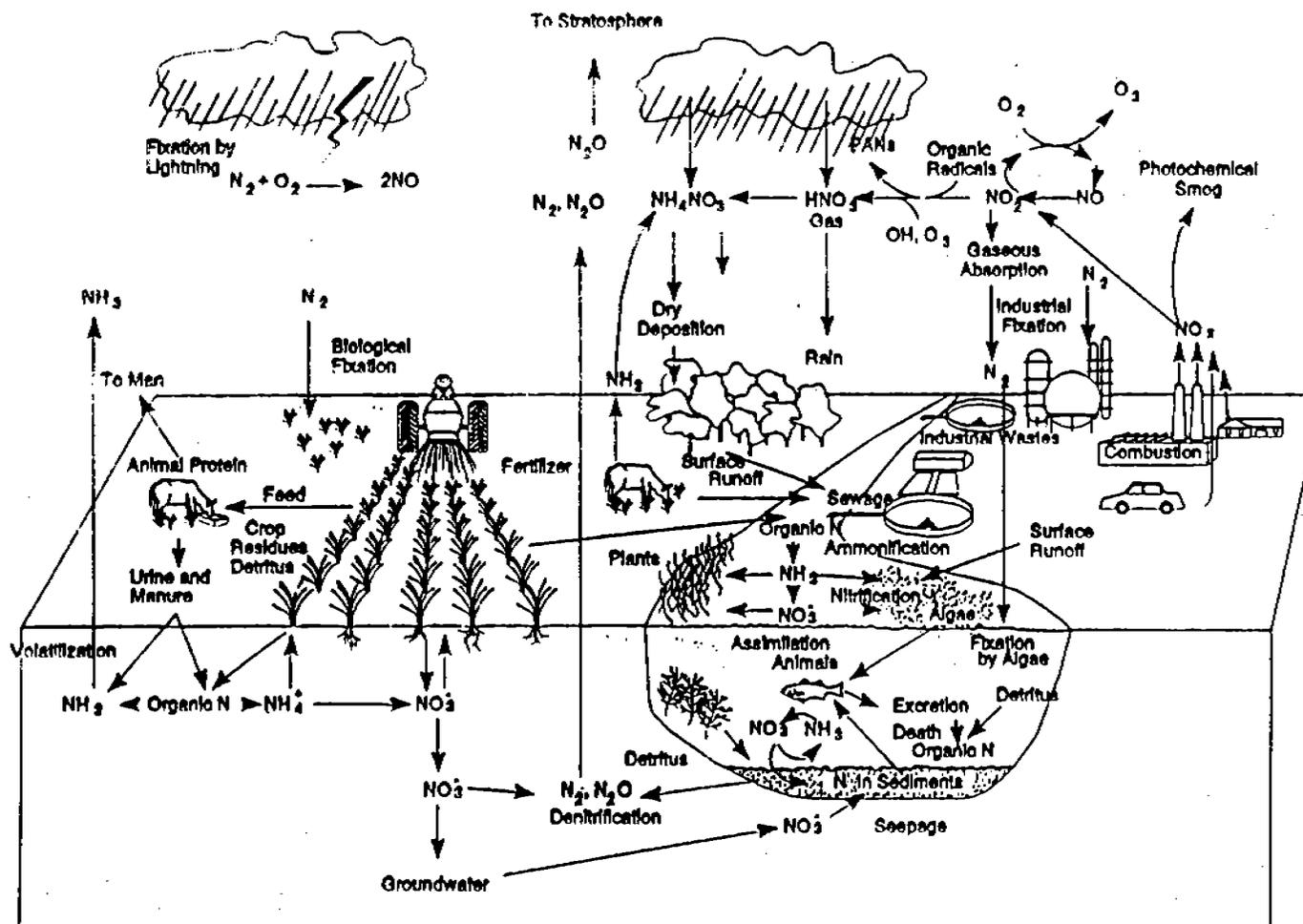


Figure VIII-1 Schematic representation of the nitrogen cycle, emphasizing human activities that affect fluxes of nitrogen. The figure depicts possible sources of nitrogen fluxes. Transformations are qualitative, not quantitative.

Source: Modified from National Research Council (1978).

B. Ecosystem Response

A great degree of diversity exists among ecosystem types as well as in the mechanisms by which each system assimilates nitrogen inputs from various sources. Because the various ecosystem components are chemically related, stresses placed on the individual components, such as those caused by nitrogen loading, can produce perturbations that are not readily reversed and will significantly alter an ecosystem. How each ecosystem responds to stress will depend on the response of the organisms that comprise that particular system. The significant effects of nitrogen inputs on terrestrial (i.e., plant communities), wetland, and aquatic ecosystems are evaluated in the following discussions.

The following discussion focuses primarily on how soil acidification and nitrogen saturation affect terrestrial ecosystems. In the terrestrial ecosystem, nitrogen may enter the plants by several different mechanisms: (1) through the roots by absorption of ammonia and ammonium; (2) by absorption of nitrate and nitrite; and, (3) through nitrogen fixation by symbiotic organisms (CD, p. 10-6). Therefore, uptake of nitrogen deposited to the soil, direct adsorption of gaseous NO_x through the plant foliage, and interactions at the soil/root interface will be addressed in the following discussion.

Exposure for the wetland ecosystem is chiefly through deposition of nitrogen to the water surface. Additional nitrogen may be added to the system from the watershed if the wetlands were formed and maintained by drainage. The discussion focuses on impacts to the waterlogged soils as well as on the affects of direct deposition on the leaves of plants within the system.

Acidification and eutrophication are issues of concern for aquatic systems. The basic concern is that deposition of nitrates alters the availability of nitrogen to the organismal constituents and thereby can cause changes in species composition within the system. The scientific evidence documenting these changes is summarized below.

1. Terrestrial Systems

a. Vegetation

Nitrogen is an essential nutrient in plant metabolism and vital to the photosynthetic process. Previous studies have demonstrated that the occurrence, type, and magnitude of NO_2 effects on terrestrial vegetation depend on the pollutant species, the concentration of pollutant, the duration of exposure, the length of time between exposures, and the various environmental and biological factors that influence the response. Foliar injury from NO_2 is rarely found in the field (CD, p. 9-1).

Though the relationship between reduced plant yield, altered biochemical processes, or foliar injury and NO_2 exposure (concentration x duration) is complex and distinctly non-linear, common features from many studies do suggest a general form for the relationship between exposure to NO_x and effect on growth or yield. These features include: (1) a threshold exposure that must be exceeded for an effect (i.e., a deviation from the unexposed state) to occur; (2) an increase in growth or yield at exposures above the threshold but below those that produce a decrease; (3) an increasingly greater reduction in growth or yield with increasing concentration of NO_x or duration or frequency of exposure (greater than those that produce an increase in growth), yielding a nonmonotonic but unimodal relationship; and (4) within the same species, the exposure-effect relationship can be different for reproductive and vegetative development and it can vary among different organs of the same plant (e.g., an effect on the growth of roots could occur at a lesser or greater exposure than what would produce the same degree of effect in the growth of stems or leaves) (CD, p. 9-89). Studies also show that at sufficiently high concentrations and durations, plants experience irreparable cell or tissue damage and ultimately, plant death.

Once a particular plant species is exposed to NO_2 concentrations above the lowest effects concentration x duration for that particular species, the observable physiological effects

of NO₂ are significant and include changes in carbon dioxide fixation (photosynthesis), alterations in specific enzymes, changes in metabolite pools, and alterations in the allocation and translocation of photosynthate. Biochemical changes within the plants may become expressed as visible foliar injury, premature senescence, increased leaf abscission, and altered plant growth and yield (CD, p. 9-1). These changes at the individual plant level may lead to altered reproduction, reduction of plant vigor, or other changes in competitive ability which could have far reaching impacts at the ecosystem level.

Gaseous concentrations of nitrogen in the United States are generally not high enough to cause the adverse effects on plants listed above. Therefore, if the current NAAQS for NO₂ continues to be attained on an annual basis, the likelihood that plants being exposed to direct atmospheric concentrations of nitrogen oxides through their leaves will cause adverse effects is minimal. Single exposures of 24 hours or less only produce acute or adverse effects at concentrations of NO₂ greater than what have been shown to occur in ambient concentrations in the United States. In experiments of 2 weeks or more, with intermittent exposures of several hours per day, adverse effects on growth or yield start to appear when the concentration of NO_x reaches the range of 0.1 to 0.5 ppm (CD, pp. 9-89,90).

Many of the newer studies investigate the effects of NO₂ in concert or sequentially with other pollutants. Again, they demonstrate that though pollutants in combination can act synergistically or additively to increase plant sensitivity, effectively lowering the lowest effects concentration x exposure required to produce injury/growth effects, these effects only occur at higher than ambient concentrations of the pollutants and at frequencies of co-occurrence greater than that typically monitored in the ambient air (CD, Table 9-20, p. 9-127).

The staff concludes, therefore, based on the review of the data in the criteria document, that the existing secondary

standard provides adequate protection for vegetation from direct ambient exposure to NO_2 .

b. Plant/Soil Interactions

Plants obtain their nutrients through the soil. Nitrogen present in the soil is an essential element for healthy plant growth and development. As discussed above, biological processes which make up the nitrogen cycle regulate how much and what form nitrogen will be available to plants. Because it is the most common limiting nutrient for vegetative growth, deposition of nitrogen at non-toxic levels and durations in any biologically available form to most uncultivated areas is likely to produce some growth increase. When heterotroph and plant demand for nitrogen are substantially satisfied (i.e., when nitrogen saturation occurs) nitrification and nitrate leaching will become significant. If there is an excess of nitrogen available, the result will be alternation of the plant/soil interaction. This alteration is the most important aspect of excess nitrogen in a terrestrial ecosystem.

Vegetative nitrogen demand varies according to stand age, stand type, the availability of other nutrients, temperature, and moisture. The effects of stand age and type on vegetative nitrogen demand are illustrated in the facts that uptake rates of nitrogen for conifers are generally maximal around the time of canopy closure, whereas in deciduous forests, maximum nitrogen uptake rates occur somewhat later (and at higher rates) due to the annual replacement of canopy foliage in these ecosystems (Turner et al., 1990).

As forests mature, and nitrogen uptake rates decline, soils begin to acidify naturally. Processes, such as forest fires and harvesting, which can release nitrogen and increase ecosystem nitrogen demand can reverse this trend in the long-term. However, it should be pointed out that because of the lack of biomass to sequester the nitrogen, these events may also lead to episodic acidification. Also, depending on the intensity of the

fire, nitrogen may be redistributed from the forest floor to the surface mineral soil rather than being lost from the system.

Additional factors that influence ecosystem response to nitrogen inputs include the rate of the nitrogen additions, initial nitrogen status of the receiving system, the competitive advantage between species, and the size of the initial population of nitrifying bacteria. When nitrogen fertilizer is added in one or two large doses to sites where the initial population of nitrifiers is low, as is typical of nutrient poor areas, trees out compete soil heterotrophs and show increased growth, while a significant lag time occurs before the onset of nitrate production (CD, p. 10-49). On the other hand, slow, steady nitrogen inputs typical of atmospheric pollution to the same system would favor a buildup of nitrifying bacteria populations and thus cause a much earlier commencement of nitrate leaching. In nitrogen rich sites, researchers find much higher soil solution nitrate concentrations in general, no delay in the onset of nitrate leaching, and tree growth under both pollutant and fertilizer regimes (Tschaplinski et al., 1991). In some cases where atmospheric nitrogen deposition acts as a fertilizer and leads to increased plant production, the benefits of fertilization may be considered to outweigh the detrimental effects of soil acidification (see soil discussion below). However, in systems in which the pre-existing balance among species is mediated by competition for nitrogen, additional inputs may also bring about an alteration in species mix. For example, Van Breeman and van Dijk (1988) found a substantial displacement of heathland plant by grasses from 1980-1986 and noted increases in nitrophilous plants in forest herb layers (see wetlands discussion). In the United States, nitrogen deposition has been occurring since the 1920's. However, because we have only recently become concerned about the effects of this deposition, there are no documented accounts in the United States of ecosystems undergoing species shifts due to atmospheric nitrogen deposition.

In addition to direct effects of nitrogen on growth, several studies have examined the effects of nitrogen deposition on forest species sensitivity to drought, cold, or insect attack. Though some studies show that increased nitrogen deposition can alter tree susceptibility to insect and disease attack and plant community structure, results are mixed and often contradictory, and currently do not provide a sufficient basis on which to make a decision on a secondary standard (CD, pp. 10-92 to 10-93). Climate is thought to play a major role in the severe red spruce decline in the northeastern United States, perhaps with some additional exacerbation due to the direct effects of acid mist on foliage (Johnson et al., 1992). There is also some evidence that suggest that indirect effects of nitrogen saturation (i.e, nitrogen input in excess of total combined plant and microbial nutritional demands), namely nitrate and Al leaching, may be contributing factors to red spruce decline in the southern Appalachians (CD, p. 10-74).

While the available data in the U.S. is limited, the staff is concerned that excess nitrogen deposition to terrestrial ecosystems may be modifying interplant competitive balances leading to future changes in species composition and/or diversity (see following discussions on wetlands and aquatics). Because it is difficult to ascertain the potential for ecosystem simplification from the current concentrations of nitrogen in the atmosphere, any appropriate regulatory control option would need to take into account the current nitrogen status of the ecosystem(s) of concern and the specific dynamics of nitrogen inputs into the area.

c. Soils

Soils are the largest nitrogen pool in terrestrial ecosystems. The most obvious and immediate effect of pollutant nitrogen inputs on soils is an increase in the activity of heterotrophs and nitrifiers associated with an increase in decomposition and nitrification. The foremost concern about long-term, capacity-controlled effects of excessive nitrogen

deposition and NO_3^- leaching is soil acidification and mobilization of Al^{3+} into soil solution and surface waters.

Base saturation is the primary measure of soil acidity (CD, p. 10-66). Base saturation refers to the degree to which soil cation exchange sites (i.e., negatively charged sites to which positively charged ions (cations) are adsorbed) are occupied with base cations. Base cations are calcium ions (Ca^{2+}), magnesium ions (Mg^{2+}), and potassium ions (K^+). Aluminum ions (Al^{3+}) and hydrogen ions (H^+) are acid cations. Lower base saturation values correlate with more acidic soils. Figure VIII-2 below shows a schematic diagram of a soil with 50% base saturation versus a soil with 10% base saturation.

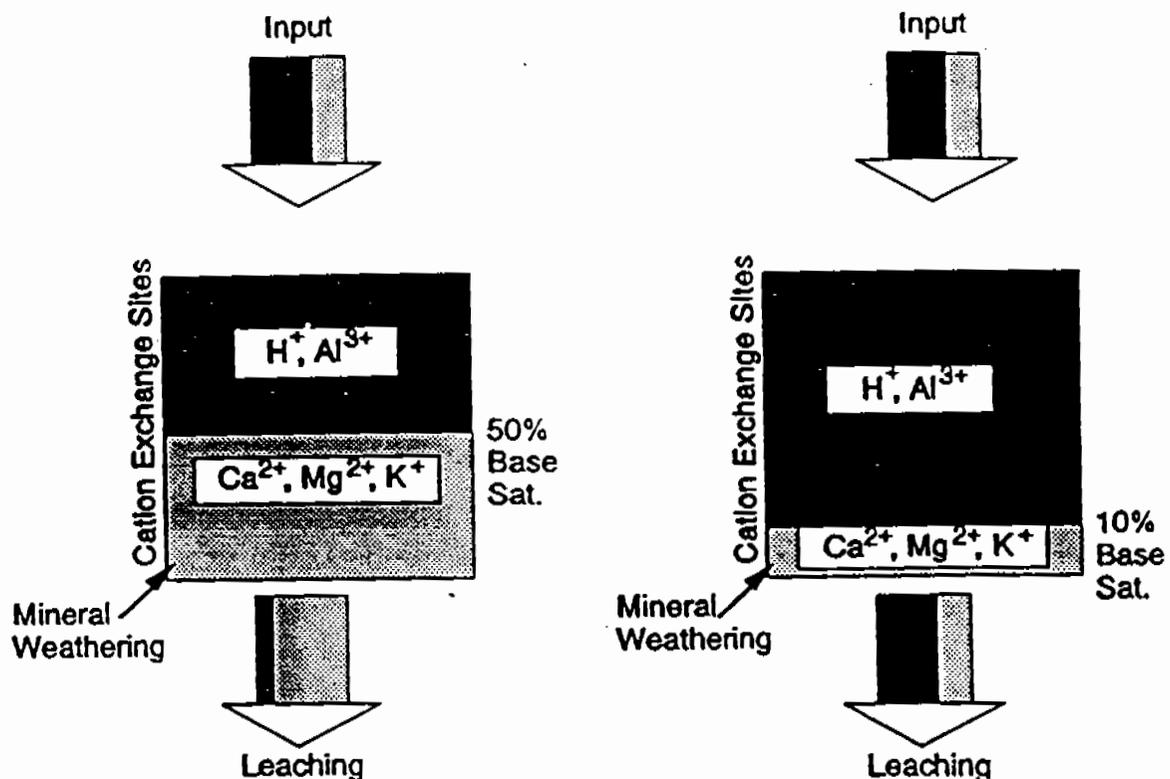


Figure VIII-2 Schematic diagram of cation exchange for base cations, aluminum ions, and hydrogen ions in circumneutral (50% base saturation, left) and acid (10% base saturation, right) soils.

Soils go through various buffering ranges as they acidify (CD, p. 10-67). First is the base cation buffering range, where incoming acid and base cations are exchanged primarily for base cations. In this buffering range, very little H^+ and Al^{3+} are leached from the soil (see Figure VIII-2, left). As soils acidify, exchangeable base cations are replaced by exchangeable Al^{3+} and H^+ , and soils are said to be in the aluminum buffering range. In this range, incoming cations (acid and base) are exchanged primarily for H^+ and Al^{3+} and acid cations become more prevalent in the soil leachate (see Figure VIII-2, right).

Atmospheric additions of nitrogen speed acidification of soils and increase aluminum mobilization if they are at or in excess of plant and microbial demand. However, the levels of nitrogen input necessary to produce measurable soil acidification are quite high. In the few studies cited (Tamm and Popovic, 1974; Van Miegroet and Cole, 1984), nitrogen inputs ranged from 50 to 3,900 kg/ha for 50 and 10 years respectively to effect a change in soil pH of 0.5 pH units. Currently, there are no documented cases in the United States in which excessive atmospheric nitrogen deposition has caused soil acidification; however, the potential exists if additions are high enough for a sufficiently long period of time. Concerns may also be greater for nitrogen deposition in certain sensitive areas of the country.

Results of the National Acid Precipitation Assessment Program (NAPAP) and other research efforts showed that the acidification of soils can alter the availability of plant nutrients (i.e., calcium and magnesium) and may expose tree roots to toxic levels of aluminum and manganese. While the impact on tree growth may be a significant cause of concern, only limited information is available on which to make quantitative assessments of damage.

In addition to anthropogenic inputs, the type and size of soil nitrogen pools are also significantly changed by living organisms (e.g., heterotrophs (decomposers such as fungi and

bacteria), autotrophs (plants), and nitrifying bacteria) in the system. These organisms compete for the biologically available portion (mineralizable or organic pools) of the total soil nitrogen. As heterotrophic bacteria decompose organic matter, they release NH_4^+ , which can subsequently be nitrified to NO_3^- by nitrifying bacteria. Because NO_3^- is poorly adsorbed to soils, it is readily leached from the system, taking nutrients essential to plant health with it. The mobilization of aluminum which follows, can be toxic to plants, and if transported to the waterways, toxic to various aquatic species.

There are a few areas in the U.S. where soils are exposed to nitrogen additions without the benefit of vegetative cover, as in some desert systems or in areas temporarily denuded by fire or flood. Because arid and semiarid soils are more alkaline than most forest and agricultural systems and have less water available than in more humid regions, they are not as susceptible to soil acidification and groundwater NO_3^- pollution and will, therefore, not be considered as a sensitive ecosystem on the basis of short-term (under 3 years) exposure to NO_2 pollution (CD, p. 10-41).

Since natural disasters such as fires or floods occur randomly, it seems impractical to consider their impacts. In intensively managed forests, fire may be used as a tool and fertilizer applied regularly. In these cases, the effects of pollutant deposition are overwhelmed by manmade manipulation of nitrogen inputs.

2. Wetlands

Wetlands function as habitats for plant and wildlife, flood control systems, stabilizers and sinks for sediments, storage reservoirs for water, and biological filters that maintain water quality. Since many different types of communities can be classified as wetlands, there is often significant disagreement over what constitutes a wetland and what terminology is used to describe them. For the purposes of this discussion, we will use four terms defined in the criteria document for wetlands: marsh;

fen; bog; and heathland or shrub bog (see definitions in CD, p. 10-110 to 10-116). The discussion that follows evaluates how nitrogen deposition from anthropogenic sources may alter the nitrogen cycle in the wetland system and analyzes the possible effects that may occur as a result of these changes.

In general, the significance of atmospheric nitrogen inputs as a percent of total nitrogen inputs from other sources to wetlands can be said to increase as rainfall increases as a fraction of the total water budget for a wetland. Salt marsh ecosystems which depend on tide and groundwater for most of their nitrogen inputs are relatively unresponsive to additional atmospheric nitrogen, while ombrotrophic bogs, which receive nutrients exclusively from precipitation, are much more responsive.

a. Vegetation

Nitrogen assimilation by wetland plant communities varies from 38 to 66 kg nitrogen/ha/year in bog ecosystems to 225 to 274 kg nitrogen/ha/year in the salt marsh and fen ecosystems, respectively. The nitrogen cycle in bog and heathland ecosystems is largely closed while in salt marshes and fens it is open, permitting a great exchange with adjacent systems. Using rates of nitrogen application ranging from 7 to 3,120 kg/ha/yr, numerous field experiments have documented standing biomass increases in a year of 6 to 413% (CD, Table 10-18, p. 10-112), showing that primary production in wetland ecosystems is most commonly limited by the availability of nitrogen. Other short-term (3 years or less) fertilization studies performed in different wetland communities demonstrate that (1) stimulation of primary production by nitrogen applications is not a linear function, with the greatest increase in standing biomass occurring in studies where the control biomass was low, and (2) the response of leaf growth is much greater than the response of roots. In addition to stimulating growth rates of wetland species, several fertilization studies demonstrated a tendency toward a change in species composition or dominance. Vermeer

(1986) found that in fen and wet grassland communities, grasses tended to increase in dominance over other species. Jefferies and Perkins (1977) also found a species-specific change in stem density at a Norfolk, England salt marsh after fertilizing monthly with 610 kg NO_3^- nitrogen/ha/year or 680 kg NH_4^+ nitrogen/ha/year over a period of 3 to 4 years.

Long-term studies (greater than three years) of increased nitrogen loadings to wetland systems further demonstrate that increases in primary production can result in changes in species composition and succession. Changes in species composition may occur from increased evapotranspiration (Howes et al., 1986; Logofet and Alexander, 1984) leading to a changed water regime that favors different species or from increased nutrient loss from the system through incorporation into or leaching from aboveground vegetation. In parts of Europe, historical data seems to implicate pollutant nitrogen in altering the competitive relationships among plants and threatening wetland species adapted to habitats of low fertility (Tallis, 1964; Ferguson et al., 1984; Lee et al., 1986).

What makes these findings important to the policymaker is that wetlands are often home to many rare and threatened plant species. Some of these plants adapt to systems low in nitrogen or with low nutrient levels. For some species, these conditions can be normal for growth. Therefore, excess nitrogen deposition can alter these conditions and thus alter species density and diversity. In what was formerly West Germany, Ellenberg (1988) surveyed the nitrogen requirements of 1,805 plant species and concluded that 50% have adapted well to habitats with low nitrogen supplies. Furthermore, of the threatened plants, 75 to 80% grow only in habitats where nitrogen availability is low. In eastern Canadian wetlands, nationally rare species are found principally on low fertility sites (Moore et al., 1989; Wisheu and Keddy, 1989). In the conterminous United States, wetlands also harbor 14% (18 species) of the total number of plant species that are formally listed as endangered. Several species on this

list, such as the insectivorous plants, are widely recognized to be adapted to nitrogen-poor environments. Based on the evidence reviewed in the criteria document, the staff believes we can anticipate similar effects from atmospheric nitrogen deposition in the United States, in areas where deposition is exceeding an area's absorptive capacity. However, there is no documentation provided in the studies reviewed that provides sufficient evidence to support the theory that species changes have occurred in the past or are occurring currently in the United States.

b. Soils

The feature of wetlands that sets them apart from terrestrial ecosystems is the anaerobic (oxygen-free) nature of their waterlogged soils. Because there is no oxygen, decomposition of organic matter in wetland soils is incomplete (CD, p. 10-110). Consequently, organic carbon is built-up in the system. This influences the various microbial transformations in nitrogen cycling. Anoxic soils favor denitrification which results in important losses of nitrogen from wetland ecosystems. Furthermore, the hydrology of wetlands favors diffusive exchanges of nitrogen compounds to and from sediments and water within the system carries nitrogen compounds between ecosystems.

Single additions of ^{15}N -labeled mineral nitrogen to vegetated soils at rates of about 100 kg nitrogen/ha/year indicate that up to 93% of applied NH_4^+ is rapidly assimilated into organic matter within a single growing season. The majority of the labeled nitrogen is lost from the system after 3 years by the combined processes of advective transport in water of particulate organic matter, advective and diffusive transport of dissolved nitrogen, and denitrification. In the absence of plants, the major fate of inorganic nitrogen applied to wetland soils is loss to the atmosphere by denitrification (CD, pp. 10-247 to 10-248).

3. Aquatic Systems

Some aquatic systems are potentially at risk from atmospheric nitrogen additions through the processes of

eutrophication and acidification. Both processes can sufficiently reduce water quality making it unfit as a habitat for most aquatic organisms and/or human consumption. Atmospheric nitrogen can enter aquatic systems either as direct deposition to water surfaces or as nitrogen deposition to the watershed. In northern climates, nitrate may be temporarily stored in snow packs and released in a more concentrated form during snow melt (see discussion on acidification). Nitrogen deposited to the watershed is then routed (e.g., through plant biomass and soil microorganisms) and transformed (e.g., into other inorganic or organic nitrogen species) by watershed processes, and may eventually run off into aquatic systems in forms that are only indirectly related to the original deposition. The contributions of direct and indirect atmospheric loadings has received increased attention. Growing evidence does indicate that the impact of nitrogen deposition on sensitive aquatic systems may be significant. However, there is still uncertainty with regard to the quantification of the relationship between atmospheric deposition of nitrogen and its appearance in receiving waters.

a. Acidification

1. Chronic Acidification. In the United States, the most comprehensive assessment of chronic acidification of lakes and streams comes from the National Surface Water Survey (NSWS) conducted as part of the National Acid Precipitation Assessment Program (NAPAP). The NSWS selected a single season of the year that exhibited low spatial and temporal variability to be the "index period", so that the general condition of surface waters could be assessed. For lakes, the index period selected was autumn overturn (a time when most lakes are mixed uniformly from top to bottom). Henriksen (1988) has proposed that the ratio of $\text{NO}_3^- : \text{NO}_3^- + \text{SO}_4^{(2-)}$ in surface waters be used as an index of the influence of NO_3^- on chronic acidification status. The results of the Eastern Lake Survey using measurements taken during the autumn overturn index period, suggest that nitrogen compounds make only a small contribution to chronic acidification in North

America. Other studies (cited in Henriksen, 1988) which did not use this index, but monitored intensively over the course of the year, however, showed that NO_3^- may be important in chronic acidification. Since annual mean values include high spring NO_3^- concentrations in runoff waters and will, therefore, be higher than concentrations measured only in the autumn, these study results cannot be compared. Regional lake surveys with representative annual or spring values for the United States were not available for review.

The index period for streams was chosen as the spring base flow (the period after spring snowmelt and before leaf-out). Unlike the lake surveys, the National Stream Survey (NSS) data have the advantage of having been collected during a spring base-flow index period. This period has been shown to be a good index of mean annual condition for streams. Though several stream regions exhibit ratios of $\text{NO}_3^-:\text{NO}_3^- + \text{SO}_4^{(2-)}$ as high as those reported for the Adirondacks, this is in part because some of the stream regions have $\text{SO}_4^{(2-)}$ concentrations that are relatively low. The stream data do suggest that the Catskills, Northern Appalachians, Valley and Ridge Province, and Southern Appalachians all show some potential for chronic acidification due to NO_3^- . Two efforts (Kaufmann et al., 1991; Driscoll et al., 1989) have also been undertaken to determine if atmospheric deposition is the source of the NO_3^- leaking out of these watersheds. Data from the NSS (Kaufmann et al., 1991) suggest a strong correlation between concentrations of stream water and levels of wet nitrogen deposition in each of the NSS regions. Secondly, Driscoll et al. (1989) collected input/output budget data for a large number of watersheds in the United States and Canada, and summarized the relationship between nitrogen export and nitrogen deposition at all the sites. Though the relationships discovered should not be over-interpreted or construed as an illustration of cause and effect, they do show that watersheds in many regions of North America are retaining less than 75% of the nitrogen that enters them, and that the

amount of nitrogen being leaked from these watersheds is higher in areas where nitrogen deposition is highest.

In Europe, many sites show chronic increases in nitrogen export from their watersheds (e.g., Henriksen and Brakke, 1988; Hauhs, 1989). Chronic acidification attributable to ammonium deposition has also been demonstrated in the Netherlands (Van Breemen and Van Dijk, 1988; Schuurkes, 1986, 1987). It has been suggested that chronic nitrogen acidification is more evident in Europe than in North America because nitrogen saturation is further progressed in Europe.

On a chronic basis in the United States, especially in the eastern part of the country, nitrogen deposition does play a role in surface water acidification. However, there are significant uncertainties with regard to the long-term role of nitrogen deposition in surface water acidity and with regard to the quantification of the magnitude and timing of the relationship between atmospheric deposition and the appearance of nitrogen in surface waters.

2. Episodic Acidification. Acidic episodes have been registered in surface waters in the Northeast, Mid-Atlantic, Mid-Atlantic Coastal Plain, Southeast, Upper Midwest, and West regions (Wigington et al., 1990). In the Mid-Atlantic Coastal Plain and Southeast regions, all of the episodes catalogued to date have been associated with rainfall. In contrast, most of the episodes in the other regions are related to snowmelt, although rain-driven episodes apparently can occur in all regions of the country. It is important to stress that even within a given area, such as the Northeast, major differences can be evident in the occurrence, nature, location (lakes or streams), and timing of episodes at different sites. A number of processes contribute to the timing and severity of acidic episodes (Driscoll and Schaefer, 1989). The most important of these are: 1) dilution of base cations (Galloway et al., 1980) by high discharge; 2) increases in organic acid concentrations (Sullivan et al., 1986) during periods of high discharge, 3) increases in

$\text{SO}_4^{(2-)}$ concentrations (Johannessen et al., 1980) during periods of high discharge, and 4) increases in NO_3^- concentrations (Galloway et al., 1980; Driscoll and Schafran, 1984; Schofield et al., 1985) during periods of high discharge. In many cases, all of these processes will contribute to episodes in a single aquatic system. In addition to these factors, the likelihood of an acidic episode is also influenced by the chemical conditions before an episode begins. For example, the magnitude of the episodes experienced by lakes depends strongly on their lake's acid neutralizing capacity (ANC) which is the same as the base cation concentration. Lakes with lower baseline ANC are affected more by nitrate pulses, and lakes with higher baseline ANC are affected more by base cation dilution from snowmelt.

Some broad geographic patterns in the frequency of episodes in the United States are now evident. Episodes driven by NO_3^- are common in the Adirondacks and Catskill Mountains of New York, especially during snowmelt, and also occur in at least some streams in other portions of the Northeast (e.g., Hubbard Brook). Nitrate contributes on a smaller scale to episodes in Ontario, and may play some role in episodic acidification in the western United States. There is little current evidence that NO_3^- episodes are important in the acid-sensitive portions of the southeastern United States outside the Great Smoky Mountains. We have no information on the relative contribution of NO_3^- to episodes in many of the subregions covered by the NSS, including those that exhibited elevated NO_3^- concentrations at spring base flow (e.g., the Valley and Ridge Province and Mid-Atlantic Coastal Plain), because temporally-intensive studies have not been published for these areas.

There is a great need to emphasize the importance of examining nitrogen episodes in a truly long-term context. Although the data reported here for the Catskills can be considered truly long-term (up to 65 years of record), data for the Adirondacks (Driscoll and Van Dreason, in press) and other

areas of the United States (Smith et al., 1987) span only 1 to 2 decades, and should be interpreted with caution.

Many of the data discussed above suggest that NO_3^- episodes are more severe now than they were in the past. These surface water nitrogen increases have occurred at a time when nitrogen deposition has been relatively unchanged in the northeastern United States (Husar, 1986; Simpson and Olsen, 1990). If we accept the idea that an increase in the occurrence of NO_3^- episodes is evidence that nitrogen saturation of watersheds is progressing, then current data suggest that current levels of nitrogen deposition are too high for the long-term health of aquatic systems in the Adirondacks, the Catskills, and possibly elsewhere in the Northeast. It is important to note that this supposition is dependent on our acceptance of NO_3^- episodes as evidence of nitrogen saturation. While some data suggest this to be the case, because of the lack of adequate air monitoring and deposition data, there are still significant uncertainties with respect to the quantification of the linkage and the timing of the relationship between the atmospheric deposition of nitrogen and its episodic or chronic appearance in surface waters.

Because the capacity to retain nitrogen differs from one watershed to another, the levels of nitrogen deposition that produce adverse effects will also vary. For example, the Northeast, because of the presence of aggrading forests and deeper soils in comparison to those of the west, may be able to absorb higher rates of deposition without serious adverse effects than areas of the mountainous West, where soils are thin in comparison and forests are often absent at the highest elevations (CD, p. 10-179). The data of Silsbee and Larson (1982) suggest strongly that forest maturation is also linked to the process of NO_3^- leakage from Great Smoky Mountain watersheds.

It should also be noted that acidification of lakes from nitrogen deposition may have some effect on the increased leaching and methylation of mercury in the aquatic ecosystem.

Current scientific evidence point to nitrogen as the principal contributor to episodic acidification of streams and lakes in the Northeast. Additionally, if further research indicates that indeed some of the watersheds of the Northeast and the mid-Appalachians are nearing nitrogen saturation, nitrogen deposition will become a more direct cause of chronic surface water acidification. However, regional and ecosystem differences will make it difficult for the Administrator to set a secondary standard which will address the problem in all areas.

b. Eutrophication

Eutrophication is the process by which aquatic systems are enriched with the nutrient(s) that are presently limiting for primary production in that system. Eutrophication may produce conditions of increased algal biomass and productivity, nuisance algal populations, and decreases in oxygen availability for heterotrophic organisms. Another effect of chronic eutrophication is increased algal biomass shading out ecologically valuable estuarine seagrass beds. During severe episodes of eutrophy, fish kills can occur and under chronic conditions, sensitive species may be permanently lost. Though this process often occurs naturally over the long-term evolution of lakes, it can be significantly accelerated by the additional input of the limiting nutrients from anthropogenic sources. A theory is that atmospheric nitrogen deposition is causing nutrient enrichment in several freshwater systems. In order to establish a link between nitrogen deposition and the eutrophication of aquatic systems, one must first demonstrate that the productivity of the system is limited by nitrogen availability, and second, that nitrogen deposition is a major source of nitrogen to the system.

1. Freshwater Eutrophication. In most lakes and streams, phosphorus, not nitrogen, is the limiting nutrient. It is thought that surplus inorganic nitrogen in lakes and streams is maintained through nitrogen fixation by various algae and aquatic plants, which must counteract the significant loss of nitrogen

that occurs in aquatic ecosystems from denitrification. Eutrophication by nitrogen inputs will only be a concern in lakes that are chronically nitrogen limited and have a substantial total phosphorous concentration. This condition is common only in lakes that have received excessive inputs of anthropogenic phosphorous, or in rare cases, have high concentrations of natural phosphorus. In the former case, the primary dysfunction of the lakes is an excess supply of phosphorous, and controlling nitrogen deposition would be an ineffective method of gaining water quality improvement. In the latter case, lakes with substantial total phosphorous concentrations would experience measurable increases in biomass from increases in nitrogen deposition.

Good predictions of nutrient limitation can now be made from ratios of total dissolved inorganic nitrogen (DIN) to total phosphorous (TP) (Morris and Lewis, 1988). Morris and Lewis found that lakes with DIN:TP values less than 9 could be limited by either phosphorous or nitrogen, whereas lakes with DIN:TP values less than 2 were always limited by nitrogen. This ratio, is a refinement from the earlier nutrient ratio of 16:1 put forth by Redfield (1934) and excludes the forms of nitrogen and phosphorus that are not biologically available. Other methods are available for measuring nutrient limitation. Bioassay experiments, for example, are more direct than nutrient ratios as various known concentrations of potentially limiting nutrients are added to an enclosed small volume of lake water and the growth response of the biomass measured. However, the results of such experiments are available for only a few selected nutrient-poor lakes and no clear pattern of nitrogen or phosphorous limitation is discernible.

Though significant percentages of lakes in the Pacific Northwest (27.7%) and the Upper Midwest (19%) indicated nitrogen limitation when the critical DIN:TP value less than 2 was applied to lakes from the Eastern Survey and Western Lake Survey, the number of lakes which meet the criteria of chronic nitrogen

limitation is likely to be small. Therefore, the potential for nitrogen deposition to contribute to the eutrophication of freshwater lakes is limited.

2. Estuaries and Coastal Waters. Estuaries come in many different shapes and sizes with many different sets of factors influencing algal production. This variety is partially captured by the classification scheme designed by Boynton et al., (1982). Because estuaries and coastal waters receive substantial amounts of weathered material from terrestrial ecosystems and from exchange with sea water, acidification is not a concern. However, this same load of weathered material and anthropogenic inputs makes these same areas prone to the effects of eutrophication. Few topics in aquatic biology have received as much attention in the past decade as the debate over whether estuarine and coastal ecosystems are limited by nitrogen, phosphorus, or some other factor. Numerous geochemical and experimental studies have suggested that nitrogen limitation is much more common in estuarine and coastal waters than in freshwater systems. Experiments to confirm widespread nitrogen limitation in estuaries have not been conducted, however, and nitrogen limitation cannot be assumed to be the rule. However, taken as a whole, the productivity of estuarine waters of the United States correlates more closely with supply rates of nitrogen than of other nutrients (Nixon and Pilson, 1983). Specific instances of phosphorus limitation (Smith, 1984) and of seasonal switching between nitrogen and phosphorus limitation (D'Elia et al., 1986; McComb et al., 1981) have been observed, however, and stand as exceptions to the general rule. Nitrogen-fixing blue-green algae are rarely abundant in estuarine waters (Howarth et al., 1988), and so nitrogen-deficient conditions may continue indefinitely in these systems.

Estimation of the contribution of nitrogen deposition to the eutrophication of estuarine and coastal waters is made difficult by the multiple direct anthropogenic sources (e.g., from agriculture and sewage) of nitrogen. In the United States, only

a few systems have been studied with enough intensity to develop predictions about the contribution of atmospheric nitrogen to total nitrogen inputs. One example is the Chesapeake Bay, where a large effort has been made to establish the relative importance of different sources of nitrogen to the total nitrogen load entering the bay (e.g., D'Elia et al., 1982; Smullen et al., 1982; Fisher et al., 1988a; Tyler, 1988). Though estimates for each individual source are very uncertain, three attempts to determine the proportion of the total NO_3^- load to the bay attributable to nitrogen deposition all produced estimates in the range of 18 to 39%. If this is the case, supplies of nitrogen from deposition exceed supplies from all other non-point sources (i.e., farm runoff) to the bay and only point-source inputs (i.e., discharges to water, emissions from industrial facilities) represent a greater input than deposition.

From the few available examples, it is clear that atmospheric nitrogen inputs to aquatic ecosystems are of concern. The significance of these inputs will vary from site to site and will depend on the availability of other growth nutrients, the flushing rate through the system, the sensitivity of resident species to added nitrogen, the types and chemical forms of nitrogen inputs from other sources, as well as other factors.

c. Direct Toxicity

In a report titled "Ambient Water Quality Criteria for Ammonia-1984", developed by the United States Environmental Protection Agency, high NH_3 concentrations are associated with lesions in gill tissue, reduced growth rates of trout fry, reduced fecundity (number of eggs), increased egg mortality, and increased susceptibility of fish to other diseases, as well as a variety of pathological effects in invertebrates and aquatic plants. Given the serious nature of these effects, EPA developed regulations requiring 4-day average concentrations of NH_3 not to exceed the limits set more often on average than once every 3 years, nor can 1 hour average concentrations exceed one-half of the limit set more often on average than once every 3 years.

Because critical concentrations of NH_3 that cause the various effects are wide ranging and are related to site specific temperature and pH values, no single toxic concentration can be established. Rather the limits (final chronic value and final acute value) must be calculated as a function of pH, temperature, and ionic strength of the water source. Because of the serious nature of the effects, it is important to determine whether nitrogen deposition could potentially contribute directly to toxic effects in surface waters. Given current maximal concentrations of NH_4^+ in deposition and reasonable maximum rates of dry deposition, even if all nitrogen species were ammonified, the maximum potential NH_4^+ concentrations attributable to deposition would be approximately 280 nmol/L and would be unlikely to be toxic except in unusual circumstances. Therefore, it appears that the information reviewed suggests that the potential for directly toxic effects attributable to nitrogen deposition in the United States is very limited.

C. Critical Loads and Other Potential Regulatory Actions

1. Critical Loads Concept

Concerns regarding nitrogen saturation have led to efforts to develop "critical loads" of nitrogen for various ecosystems. One widely applicable, general definition of a "critical load" is: "a quantitative estimate of an exposure to one or more pollutants below which significant harmful effects on specified sensitive elements of the environment do not occur according to present knowledge" (Nilsson and Grennfelt, 1988). Other more specific definitions have been developed for particular ecosystems to include relevant receptors and measurable parameters.

The critical load concept assumes a certain acceptable lifetime for a particular ecosystem resource to function at a predetermined capacity. For example, a critical load may be set so that at a given rate of nitrogen into the system, the buffering capacity of a certain soil will not be exceeded for the next 50 or 100 years. Below is a partial list of endpoints that

have been suggested as useful for defining appropriate critical nitrogen loads on ecosystems:

- * prevent nitrate levels in drinking or surface waters from rising above standard levels,
- * ensure proton production less than weathering rate,
- * maintenance of a fixed NH_3 -base cation balance,
- * maintain nitrogen inputs below nitrogen outputs (the nitrogen saturation approach), and
- * minimize accelerations in the rates of ecological succession (vegetation changes due to altered interspecific competition).

Critical load values are often expressed as weight of nitrogen (kilogram) deposited to a certain size area (hectare) over a set time period (year).

In 1986, a series of international meetings and workshops to address critical loads of nitrogen and sulfur deposition to soils, forest, vegetation, surface water, and groundwater were initiated. The goal of these meetings was to provide a technical basis for determining how extensively deposition, and hence emissions, need be reduced to preserve valuable ecological and cultural resources (i.e., develop target loads). One such workshop was conducted in Sweden by Skokloster in 1987. The Skokloster workshop report provided valuable information the United States used to start evaluating how the critical loads approach would work in this country.

Estimates of critical loads may be based upon a number of different methods and, to a great extent, the selection of a method depends upon the receptor chosen and the availability of relevant data for the calculations. Research is underway in several different countries, including the United States, to develop acceptable quantitative methods. However, after evaluating various options for setting a critical nitrogen load, Skeffington and Wilson (1988) concluded that "we do not understand ecosystems well enough to set a critical load for nitrogen deposition in a completely objective fashion."

Limited data from the National Acid Deposition Program (NADP) and other sources indicate that total deposition of nitrogen in parts of North America, particularly the eastern United States, is comparable to that found for many areas of Europe. North American sites would appear to have total nitrogen deposition rates less than 25 kg nitrogen/ha/year (Tables VIII-1 and VIII-2). Other studies indicate that wet NO_3^- deposition alone exceeds 15 kg nitrogen/ha/year over most of the midwest and 20 kg nitrogen/ha/year in portions of the northeast United States (Zemba et al., 1988).

Furthermore, it is clear that ecosystems differ in their ability to assimilate and buffer excess nitrogen inputs depending on the region they are located in. Hence, there is probably no universal critical load definition that could be applied effectively to all ecosystems. Therefore, any attempt to set a critical loads standard(s) would need to take into account regional differences in sensitivity, as well as other policy considerations.

Congress included Section 404 in Title IV of the 1990 Clean Air Act Amendments (Appendix B of the Act) which requires the Agency to provide a report to Congress on the feasibility and effectiveness of an acid deposition standard to protect sensitive and critically sensitive aquatic and terrestrial resources. The results of this Acid Deposition Standard Study should begin to answer some of the questions necessary for the Administrator to make future policy decisions related to setting critical loads. Specifically, the study seeks to accomplish the following goals: (1) to identify sensitive ecosystems in the United States and Canada; (2) to determine the Clean Air Act's (Title IV - Acid Rain) level of protection for sensitive ecosystems; (3) to determine the possible need for an acid deposition standard(s) (e.g., further emission reductions to protect these systems); and, (4) to assess the feasibility and costs of implementing an acid deposition standard.

Table VIII-1 MEASUREMENTS OF VARIOUS FORMS OF ANNUAL NITROGEN DEPOSITION TO NORTH AMERICAN AND EUROPEAN ECOSYSTEMS

Site Location/ Vegetation	Forms of Nitrogen Deposition (kg/ha) ^a				Total ^b	Reference
	Wet		Dry			
	Cloud	Rain	Particles	Gases		
<u>United States</u>						
California, Chaparral	—	8.2	—	—	23 ^c	Riggan et al. (1985)
California, Sierra Nevada	—	—	—	—	(2)	Williams and Melack (1991a)
Georgia, Loblolly pine	—	3.7	1.0	4.2	9	Lovett (1992)
North Carolina, Loblolly pine	—	8.7	2.2	4.1	15	Lovett (1992)
North Carolina, Hardwoods	—	4.8	0.5	—	5.3	Swank and Waide (1988)
North Carolina, White pine	—	3.7	0.9	2.7	7	Lovett (1992)
North Carolina, Red spruce	8.7	6.2	3.6	8.6	27	Lovett (1992)
New Hampshire, Deciduous	—	7.0	—	—	(7)	Likens et al. (1970)
New Hampshire, Deciduous	—	9.3	—	—	(9)	Likens (1985)
New York, Red spruce	7.3	6.1	0.2	2.3	16	Lovett (1992)
New York, Mixed deciduous	—	4.8	0.8	2.5	8	Lovett (1992)
Tennessee, Mixed deciduous	—	2.9	4.1	6.1	13	Kelly and Meagher (1986)
Tennessee, Oak forest #1	—	3.2	4.4	4.0	12	Kelly and Meagher (1986)
Tennessee, Oak forest #2	—	2.9	4.4	4.0	11	Kelly and Meagher (1986)
Tennessee, Oak forest #1	—	6.9	1.3	—	8	Kelly (1988)
Tennessee, Oak forest #2	—	6.0	1.2	—	7	Kelly (1988)
Tennessee, Oak forest	—	4.5	1.8	3.8	10	Lindberg et al. (1986)
Tennessee, Loblolly pine	—	4.3	0.6	1.4	9	Lovett (1992)
Washington, Douglas fir	—	2.9	1.3	0.6	5	Lovett (1992)
Washington, Douglas fir	—	1.0	—	—	(1)	Henderson and Harris (1975)
<u>U.S. Regions</u>						
Adirondacks	—	6.3	4.7	—	11	Driscoll et al. (1989a)
Midwest	—	4.2	2.9	—	7.1	Driscoll et al. (1989a)
Northeast	—	21.7	—	—	22	Munger and Eisenreich (1983)
Northwest	—	16.6	—	—	17	Munger and Eisenreich (1983)
Southeast	—	20.6	—	—	21	Munger and Eisenreich (1983)
Southeast Appalachians	—	4.2	3.1	—	7.3	Driscoll et al. (1989a)

Table VIII-1 (cont'd). MEASUREMENTS OF VARIOUS FORMS OF ANNUAL NITROGEN DEPOSITION TO NORTH AMERICAN AND EUROPEAN ECOSYSTEMS

Site Location/ Vegetation	Forms of Nitrogen Deposition (kg/ha) ^a				Total ^b	Reference
	Wet		Dry			
	Cloud	Rain	Particles	Gases		
<u>Canada</u>						
Alberta (southern)	--	7.3	12.2	--	19.5	Peake and Davidson (1990)
British Columbia	--	5.5	--	--	(5)	Feller (1987)
Ontario	--	3.7	--	--	(4)	Linsey et al. (1987)
Ontario (southern)	--	2.3	1.4	--	3.7	Ro et al. (1988)
<u>Federal Republic of Germany</u>						
Spruce (Southeast slope)	--	16.5	--	--	16.5	Hantschel et al. (1990)
Spruce (Southwest slope)	--	24.3	--	--	24.3	Hantschel et al. (1990)
<u>Netherlands</u>						
Oak-birch	--	--	--	--	24-56 ^c	Van Breemen and Van Dijk (1988)
Deciduous/spruce	--	--	--	--	21-42 ^c	Van Breemen and Van Dijk (1988)
Scots pine	--	--	--	--	17-64 ^c	Van Breemen and Van Dijk (1988)
Douglas fir	--	--	--	--	17-64 ^c	Van Breemen and Van Dijk (1988)
Douglas fir	--	19.3	95.7 ^d	--	115	Draaijers et al. (1989)
<u>Norway</u>						
Spruce	--	10.3	0.7	0.2	11.2	Lovett (1992)
	--	--	--	--	3-19 ^c	Royal Society (1983)
<u>United Kingdom</u>						
Spruce forest	1.9	8.0	--	13.5	23.4	Fowler et al. (1989a)
Cotton grass moor	0.4	8.0	--	4.0	12.4	Fowler et al. (1989a)

^a -- Symbolizes data not available or, in the case of cloud deposition, not present.

^b Measurements of total deposition data that do not include both a wet and dry estimate probably underestimate total nitrogen deposition and are enclosed in parentheses.

^c Total nitrogen deposition was based on bulk deposition and throughfall measurements and does include components of wet and dry deposition.

^d Includes deposition from gaseous forms.

Source: Table 10-14, pp. 10-99 to 10-100 of the 1993 Air Quality Criteria for Oxides of Nitrogen

Table VIII-2 NITROGEN INPUT/OUTPUT RELATIONSHIPS
FOR SEVERAL ECOSYSTEMS

Site/Vegetation	Inputs (kg/ha/year)	Efflux ^a (kg/ha/year)	Reference
<u>United States</u>			
Florida, Slash pine	5.9 ^b	0	Van Miegroet et al. (1992)
Georgia, Loblolly pine	9.0 ^b	0	Van Miegroet et al. (1992)
Minnesota, Spruce	7.5 ^b	0	Van Miegroet et al. (1992)
North Carolina, Loblolly pine	15.0 ^b	0	Van Miegroet et al. (1992)
North Carolina, Oak/hickory	8.2 ^c	3.2	Cole and Rapp (1981)
North Carolina, Red spruce	27.1 ^b	11.0-20.0	Van Miegroet et al. (1992)
North Carolina, White pine	8.8 ^c	0.2	Cole and Rapp (1981)
North Carolina, White pine	7.4 ^b	0	Van Miegroet et al. (1992)
New Hampshire, N. hardwood	6.5	4.0	Bormann et al. (1977)
New Hampshire, N. hardwood	23.6	17.4	Likens et al. (1977)
New York, Deciduous	8.0 ^b	1.0	Van Miegroet et al. (1992)
New York, Red spruce	15.9 ^b	3.0	Van Miegroet et al. (1992)
Oregon, Douglas fir	2.0	1.5	Sollins et al. (1980)
Tennessee, Loblolly pine	8.7 ^b	0-2.0	Van Miegroet et al. (1992)
Tennessee, Hardwood	13.2 ^b	4.4	Kelly and Meagher (1986)
Tennessee, Hardwood	13.0	3.1	Henderson and Harris (1975)
Tennessee, Hardwood	8.7	1.8	Cole and Rapp (1981)
Tennessee, Oak forest	7.0-8.0 ^d	1.25	Kelly (1988)
Tennessee, Oak forest	11.5 ^b	3.2	Kelly and Meagher (1986)
Tennessee, Shortleaf/pine	8.7	1.8	Cole and Rapp (1981)
Tennessee, Yellow/poplar	7.7	3.5	Cole and Rapp (1981)
Washington, Douglas fir	1.7	0.6	Cole and Rapp (1981)
Washington, Douglas fir	4.7 ^b	0	Van Miegroet et al. (1992)
Washington, Red alder	70.0 ^b	71.0	Van Miegroet and Cole (1984)
Washington, Silver fir	1.3	2.7	Turner and Singer (1976)
Wisconsin, N. hardwoods	5.6	0.05	Pastor and Bockheim (1984)
<u>Canada</u>			
Ontario Maple	7.8	18.2	Foster and Nicolson (1988)
<u>Federal Republic of Germany</u>			
Norway spruce	21.8	14.9	Cole and Rapp (1981)
Beech	21.8	4.4	Cole and Rapp (1981)
<u>Netherlands</u>			
Oak	45.0	22.0	Van Breemen et al. (1987)
Oak/birch	54.0	78.0	Van Breemen et al. (1987)
Oak	56.0	28.0	Van Breemen et al. (1987)
Mixed deciduous	63.0	68.0	Van Breemen et al. (1987)

Table VIII-2 (cont'd). NITROGEN INPUT/OUTPUT RELATIONSHIPS
FOR SEVERAL ECOSYSTEMS

Site/Vegetation	Inputs (kg/ha/year)	Efflux ^a (kg/ha/year)	Reference
<u>Norway</u>			
Spruce	11.2 ^b	0	Van Miegroet et al. (1992)
<u>Sweden</u>			
Coniferous	2.1	0.6-1.0	Rosen (1982)
<u>United Kingdom</u>			
Mixed hardwood	5.8	12.6	Cole and Rapp (1981)
<u>U.S.S.R.</u>			
Norway spruce	1.1	0.9	Cole and Rapp (1981)

^aAn estimate based on nitrogen losses from the soil profile or from stream flow out of a watershed.

^bIncludes precipitation, cloud (where appropriate), particulate, and gaseous forms of nitrogen deposition.

^cIncludes nitrogen inputs from precipitation and particulate forms of deposition.

^dMean of two oak forests in eastern Tennessee.

Source: Table 10-15, pp. 10-101 to 10-102 of the 1993 Air Quality Criteria for Oxides of Nitrogen

It is clear that the critical loads concept has potential as a tool in the regulatory process. Most countries of western Europe have adopted a system for estimating critical loads and are moving closer to implementing emission reduction programs based on the concept. However, while some progress in the science has occurred in the past few years, numerous critical loads related issues remain unresolved, including and particularly, the long-term effects of nitrogen alone and in combination with other pollutants. In the United States, no national critical load level has been proposed, due in part to a lack of data on multiple forms of nitrogen deposition and a general need to examine the implications of this form of effects-based regulations.

2. Target Loads

A modification of the critical loads concept is that of target loads. As discussed above, critical loads are developed dominantly, although not purely, as a matter of science. In contrast, target loads are only partially based on science and take into account legal, social, cultural, economic and political realities. A target load is the level that is believed attainable given the above constraints. The Administrator could decide to set a target load for NO_x , however it would also be necessary to exercise significant judgement in identifying the resources of concern and the acceptable levels of impact to those resources if a critical load or target load standard is set.

3. Applicability to U.S. Situation

The information discussed in the CD and summarized in this document suggest that effects to ecosystems from deposition of atmospheric nitrogen may have already occurred in the U.S. and are therefore a very real future concern. While rough estimates of total atmospheric nitrogen loads in the United States approach levels recommended as critical loads for some European countries, further research is needed to better estimate appropriate critical loads and to develop the process for setting critical or target load values in the United States. Furthermore, the staff

feels that it would be premature to establish a long-term national ambient air quality standard based on the current tentative critical loads estimates.

D. Visibility

1. Major Categories

Air pollution can degrade the visual appearance of distant objects to an observer and reduce the range at which they can be distinguished from the background. While visibility impairment can occur naturally, it is well documented that anthropogenic air pollution in the form of fine suspended particles or NO₂ exacerbates the problem. The effects are manifested both in local impairment (e.g., plumes) and in large-scale, hazy air masses. For purposes of discussion, we are separating visibility impairment into two major categories: local scale or "reasonably attributed impairment" and large scale "regional" haze. Reasonably attributed impairment, which may be defined as a coherent, identifiable impairment, can be seen as an optical entity against the background sky or a distant object. This definition assumes that a single source or a small group of sources produce pollutants that are not widely dispersed. Thus, reasonably attributable impairment is considered a near source problem, although it can be part of a larger, regional impairment.

The second category of impairment, regional haze, is produced from a multitude of sources and impairs visibility in every direction over a large area, such as an urban area, or possibly over several states. Objects on the horizon are masked and the contrast of nearby objects is reduced. In some cases, the haze may be elevated and appear as layers of discoloration. Multiple sources may combine over many days to produce haze, which may be regional in scale. The fate of haze is a function of meteorological processes that occur concurrently on larger scales of time and distance.

2. Contributors to Visibility Reduction

Visibility impairment is caused by the scattering and absorption of light by particles and gases in the atmosphere and depends on the concentrations and properties of the gases present. Under typical ambient conditions, light scattering by particles dominates total extinction, which is related to reduction of contrast and visual range. The most significant optical effect of NO_2 , however, involves discoloration. NO_2 appears as a yellow to reddish-brown gas because it strongly absorbs blue light, allowing red wavelengths to reach the eye. The extent to which NO_2 filters out blue light is determined by the integral of NO_2 concentration along the sight path. The Criteria Document reports that less than 0.1 ppm-km NO_2 is sufficient to produce a color shift which is distinguishable in carefully controlled, color-matching tests. Reports from one laboratory using NO_2 containing sighting tubes indicate a possible visible color threshold of 0.06 ppm-km for the typical observer. These values refer to the effect of NO_2 in the absence of atmospheric aerosol because the visibility impairment caused by aerosol usually overwhelms the effect caused by NO_2 .

Although the physical properties of NO_2 are well known and its coloration effect in a controlled environment recognized, there are relatively little data available for judging the actual importance of NO_2 to visual air quality. The data needed to make such a judgment are potentially complex, including wavelength dependence of scattered light at different angles which can also cause discoloration (Charlson et al., 1978). In addition to being modified by particle scattering, discoloration of plumes and haze layers by NO_2 also is affected by a number of other factors such as sun angle, surrounding scenery, viewing angle, human perception parameters, and pollutant concentrations.

One unresolved issue concerns the relative contribution of NO_2 and particles to atmospheric discoloration. Although the color of urban haze (often termed "brown") was originally ascribed to NO_2 , more recently it has been shown that the brown

color can result from particles alone. The coloration effect of particles depends on particle size, composition, scattering angle between observer and illumination, and optical characteristics of the background target. The overall impact of aerosol haze is to reduce visual range and contrast and possibly, to change color. Nitrate aerosols do contribute to the overall impact on visibility impairment. It is difficult, however, to make a quantitative assessment of their contribution. The draft staff paper on particulate matter will consider the relative contribution of nitrate to fine particle mass.

EPA must assess whether there is any supportable relationship between NO_2 concentrations at a given point and visibility impairment due to a plume or regional haze. The present NO_2 standards are intended to protect against effects at or near ground level, and monitoring for NO_2 is generally performed at or near ground level. In the case of visibility impairment due to a plume from a stationary source, there is no reliable relationship between ground, or near ground, level concentrations at any given point and discoloration caused by the plume. The plume, trapped in an atmospheric inversion, would disperse slowly and mix to the ground far downwind of the source. Concentrations taken at ground level while a coherent plume was clearly visible would not necessarily exceed an ambient standard. For this reason it would be difficult to set a NAAQS for NO_2 , based on ground level monitoring, that would insure an acceptable level of visibility.

Another approach to establishing a visibility standard would be to monitor plume level concentrations. Because of the difficulty in making plume measurements, it may be possible to measure the discoloration itself using an optical device; e.g., a telephotometer. This instrument could measure the color contrast between the background and the plume. The measurements could be used as a possible index of the effect of NO_2 . Measuring the actual discoloration would avoid the problems encountered in the ordinary approach, where a monitor near ground level might not

pick up a violation of the standard, but a plume would be clearly visible, or where high NO_2 levels detected by remote sensing did not result in a perceptible plume. One problem in this approach is the uncertainty as to whether the discoloration that would trigger the telephotometer is caused by NO_2 or by particles. Although measurement of discoloration would be a unique way of expressing the standard, it would be worthy of consideration once these problems are resolved.

Another regulatory mechanism, provided under sections 169A and 165(d) of the Clean Air Act (CAA), may provide some control over the more noticeable brown plumes appearing in otherwise pristine areas. In December 1980, EPA promulgated a phased approach to visibility protection. Phase I applies to pristine (Class I) areas (i.e., all international parks and certain national parks and wilderness areas as described in section 162(a) of the CAA) and requires control of visibility impairment caused by visible plumes from individual sources. EPA has initiated steps to control this aspect of visibility impairment. The Agency is now beginning its development of rules to address regional haze in Class I areas.

In regard to regional haze, because the effect of NO_2 depends on the product of the pollution concentration and the viewing path length, the impression of severity is greater the farther away the viewer can see past (or around) the haze layer. (The coloration of 0.05 ppm NO_2 over 10 km is the same as 0.5 ppm over 1 km.) When NO_2 is dispersed over a large area, as in the case of urban emissions, ground level concentrations at individual points may be less than a national standard but because an observer views the entire NO_2 mass, the urban plume would appear discolored.

In summary, the scientific evidence indicates that light scattering by particles is generally the primary cause of degraded visual air quality and that aerosol optical effects alone can impart a reddish brown color to a haze layer, thus raising the question as to the appropriateness of a NAAQS for NO_2

to protect against visibility impairment. While it is clear that particles and NO_2 contribute to brown haze, it is the staff's judgement that the improvement in visual air quality to be gained by reducing NO_2 concentrations seems uncertain at best. Therefore, the staff concludes that an ambient secondary standard for NO_2 to protect visibility is not warranted at this time.

E. Non-Biological Materials

Nitrogen oxides (NO_x), including nitric oxide (NO), nitrogen dioxide (NO_2), and nitric acid (HNO_3), are known to enhance the fading of dyes; diminish the strength of fabrics, plastics, and rubber products; assist the corrosion of metals; and reduce the use-life of electronic components, paints, and masonry. Compared to studies on sulfur oxides (SO_x), however, there is only limited information available quantifying the effects of nitrogen oxides. Especially in the outdoor environment, it is difficult to distinguish a single causative agent for observed damage to exposed materials because many agents, together with a number of environmental stresses, act on a surface throughout its life. Oxides of nitrogen are deposited on material surfaces through both wet and dry deposition processes (Tombach, 1982). In addition, particles containing NO_x can be transported to a material surface through gravitational settling or inertial impaction of the particles on the surface. The rate at which deposition processes transport NO_x to the surface is dependent on the NO_x concentrations in the environment, the chemistry and geometry of the surface, the concentrations of other atmospheric constituents, and the turbulent transfer properties of the air (Lipfert, 1989). There is wide variation in the deposition of NO_x to different surfaces and NO_x species themselves are reactive and their interactions with other atmospheric constituents are complex.

It is the staff's conclusion that insufficient evidence exists that NO_2 is currently playing a significant role in damage to non-biological materials. Better experimental techniques are needed, both for investigating materials damage on the whole and

for determining the role played by NO_x . Care should be exercised in the extrapolation of data and conclusions based on chamber studies to effects expected from ambient exposures. Franey and Graedel (1985) have suggested that for any chamber study to be realistic, moisture, radiation, carbon dioxide, reduced sulfur, and a nitrogen-containing gas must be included. Therefore, we conclude that an ambient secondary standard for NO_2 to protect against materials damage is not warranted at this time.

IX. Summary of Staff Conclusions and Recommendations for Secondary Nitrogen Dioxide NAAQS

This summary of staff conclusions and recommendations for the NO_2 secondary NAAQS draws from the discussions contained in previous sections of this Staff Paper. The key findings are:

1) The staff concludes that the impact on terrestrial vegetation from short-term exposures to NO_2 under existing ambient levels is insignificant and does not warrant a change in the secondary NAAQS at this time. As presented above in section B.1.a., studies of short-term, acute effects of nitrogen additions to vegetation have demonstrated that NO_2 in mixtures with other pollutants or at higher than ambient concentrations can produce adverse effects on plant growth or reproduction. Though the sensitivity of vegetation to NO_2 varies both within and between species and can be modified by season, developmental stage, or other climate or environmental factors, taking all of these variables into account, the ambient levels of NO_2 in the United States are considered below those that evoke a short-term, acute response.

2) The staff concludes there is little potential for concentrations of nitrogen to be sufficient to acidify soils to the extent that it would cause direct phytotoxic effects in plants.

3) The staff concludes, based on existing European studies and information in the United States, that addition of nitrogen to ecosystems in the U.S. (specifically in mature forests and wetlands which host a number of endangered species adapted to

nitrogen-poor habitats) may lead to shifts in species competition and composition and may represent a significant environmental problem. However, there is not sufficient evidence to conclude that changes in plant communities have occurred in the past or are now occurring in the United States due to nitrogen inputs. Furthermore, there is only limited long-term data available on plant community composition. Additional research is needed to more accurately characterize any potential threat to mature forests and wetland species from atmospheric nitrogen. Once this information is available, the need for additional ecosystem protection should be reassessed by the Administrator.

4) The staff concludes that nitrogen deposition is a factor in chronic acidification of surface waters in certain sensitive regions. However, at this time, the staff finds insufficient evidence to quantify the magnitude or timing of the relationship between atmospheric deposition of nitrogen and its appearance in surface waters. Additional research should be conducted so that such information will be available for the next standard review. Once this relationship can be reasonably quantified, the staff recommends that the Administrator consider appropriate regulatory approaches to address the problem. These actions could take the form of a uniform national standard or regional standards which account for differences in regional sensitivities.

5) Based on the review of the available scientific information in the CD, the staff concludes that nitrogen deposition is a contributor to the episodic acidification of some streams and lakes in the United States. Thus, it can also be concluded that some freshwater ecosystems will benefit from reductions in nitrogen inputs from man-made sources. However, the scientific information reviewed in the CD is insufficient, at this time, to quantify how much of a contribution nitrogen deposition is making to the acidification problem and what levels of reduction are necessary to remedy the situation.

When additional scientific information is available that adequately quantifies the relationship between nitrogen

deposition and episodic acidification, the staff recommends that the Administrator consider appropriate regulatory approaches to address the problem. Key considerations will be the major differences evident in the occurrence, nature, location, timing of episodes at different sites, and the subsequent effects produced. Such differences will make setting a single national standard which would adequately protect all areas of the country difficult and complex. Therefore, staff believes regional approaches that take into account such variations should be considered for providing the level of control needed to address the problem of episodic acidification.

6) The staff concludes that it is very unlikely that eutrophication of freshwater systems is due to atmospheric nitrogen additions since phosphorus, not nitrogen, is generally the limiting factor for algae growth.

7) The staff concludes that both upland and direct atmospheric nitrogen deposition can significantly affect the trophic status of estuarine and coastal waters. However, it is difficult to quantify the effect that nitrogen deposition is having either within a system or across systems.

As mentioned before, once a relationship can be reasonably quantified, staff recommends that the Administrator consider appropriate regulatory approaches, including a uniform national standard or regional standards, to protect our valuable natural resources. Regional standards may provide a better mechanism to address the problem because of the variability in responses to increased nitrogen inputs between regions.

8) Because of nitrogen saturation, nitrogen levels in surface waters could increase even if nitrogen deposition does not increase. Therefore, episodic and chronic acidification of freshwater, as well as increased loadings to estuaries, could worsen even without increases in nitrogen deposition. Further research is needed, however, to determine the direct effects of nitrogen deposition on acidification or eutrophication, the rate at which this increase would occur, and in what time frame.

Therefore, staff recommends that further research be conducted to provide the information needed to evaluate this issue in its next standard review.

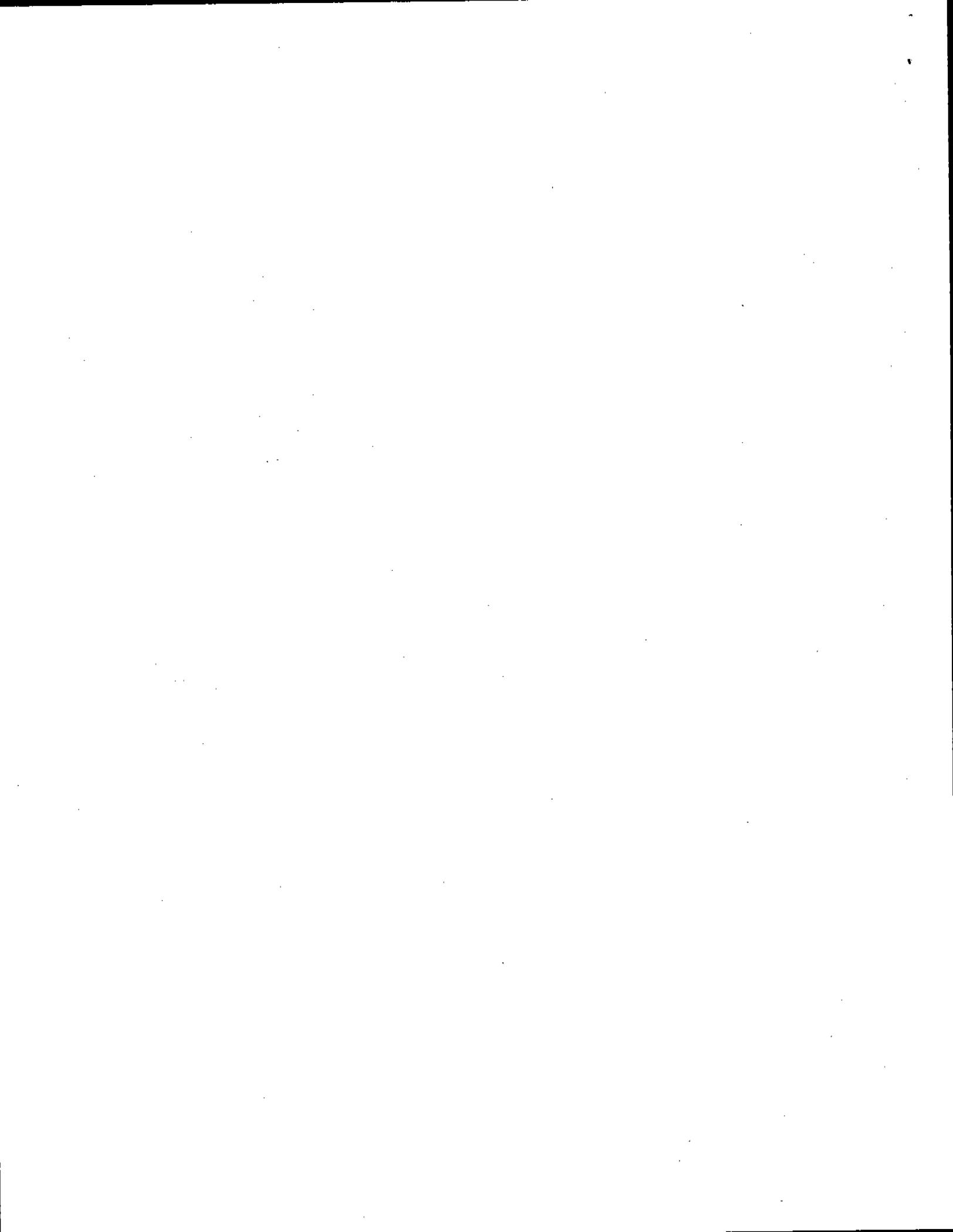
9) The staff concludes that it would be premature to consider establishment of a long-term national critical loads secondary ambient air quality standard for NO_2 given the state of the science of critical loads estimation that exists at the current time. However, this methodology has the potential to provide a useful tool for making regulatory decisions at the regional level in the future. The Acid Deposition Standard Study, which is designed to fully evaluate the issues associated with this approach, should assist the Administrator further in evaluating the potential application of both critical and target loads.

10) With regard to visibility impairment, the scientific evidence indicates that light scattering by particles is generally the primary cause of degraded visual air quality. Furthermore, aerosol optic effects alone can impart a reddish-brown color to a haze layer. While it is clear that particles and NO_2 contribute to brown haze, in the staff's judgement reducing ground level NO_2 concentrations would result in little improvement in visual air quality. Therefore, the staff concludes that an ambient secondary standard for NO_2 to protect visibility is not warranted at this time.

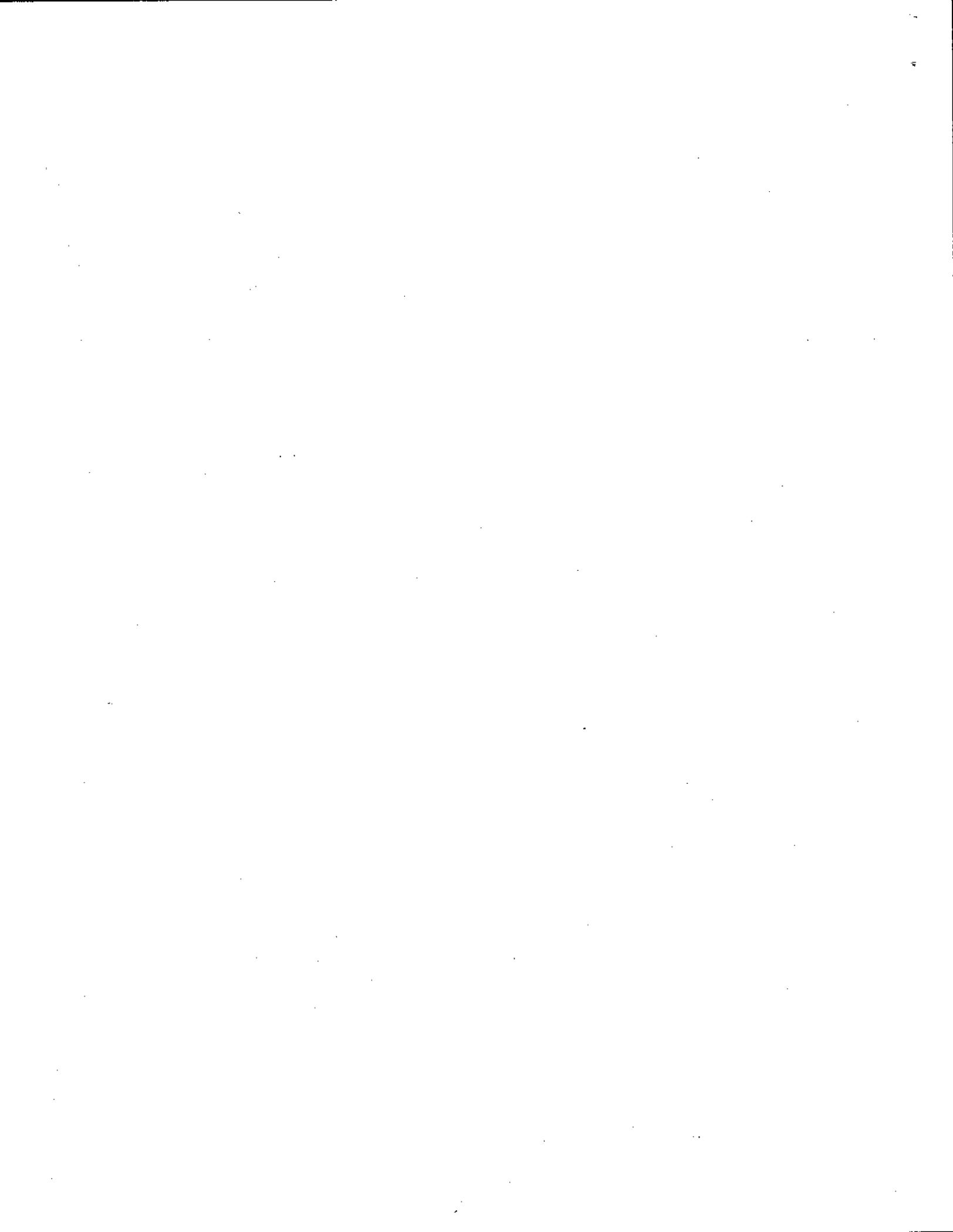
11) Based on the available data, the staff concludes that it is unlikely that NO_2 is playing a significant role in the damage to non-biological materials and therefore does not recommend consideration be given to setting a secondary standard to protect against material damage.

12) If the Administrator determines that a separate secondary standard is appropriate, the staff recommends that it be set identical to the primary standard for NO_2 . A secondary standard set within the range recommended for the primary standard would, in the staff's judgement, provide adequate protection against the direct effects of NO_2 on the environment.

13) It is clear that additional studies need to be conducted to reduce the uncertainty in the relationship between nitrogen deposition and forest health and in determining the adverse effects on surface waters and estuaries arising from the long-term accumulation of nitrogen. The staff recommends substantial efforts be undertaken to illuminate the quantitative relationships between nitrogen deposition and changes in forest nitrogen cycling, episodic and chronic acidification of surface waters, and estuarine eutrophication, either through ongoing research or new initiatives. Such efforts will provide an improved scientific basis for the next standard review.



APPENDIX A: CASAC CORRESPONDENCE





UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

September 30, 1993

EPA-SAB-CASAC-LTR-93-015

Honorable Carol M. Browner
Administrator
U.S. Environmental Protection Agency
401 M Street, S.W.
Washington, DC 20460

OFFICE OF
THE ADMINISTRATOR

Subject: Clean Air Scientific Advisory Committee Closure on the Air Quality
Criteria Document for Oxides of Nitrogen.

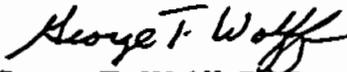
Dear Ms. Browner:

The Clean Air Scientific Advisory Committee (CASAC) of EPA's Science Advisory Board (SAB) at a meeting on July 1, 1993, completed its review of the draft document entitled National Ambient Air Quality Standards for Oxides of Nitrogen (NO_x). The Committee notes with satisfaction the improvements made in the scientific quality and completeness of the criteria document. It has been modified in accordance with the recommendations made by the CASAC in April, 1993.

The document has organized the relevant information in a logical fashion and the Committee believes that it provides a scientifically adequate basis regulatory decisions on oxides of nitrogen based on present scientific data on health effects of such exposure.

The Committee looks forward to reviewing the upcoming staff decision paper on the NO_x standard.

Sincerely,


George T. Wolff, Ph.D.
Chairman

Clean Air Scientific Advisory Committee



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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

August 22, 1995

EPA-SAB-CASAC-LTR-95-004

Honorable Carol M. Browner
Administrator
U.S. Environmental Protection Agency
401 M St., SW
Washington, DC 20460

Subject: CASAC Review of the Staff Paper for the *Review of the National Ambient Air Quality Standards for Nitrogen Dioxide: Assessment of Scientific and Technical Information*

Dear Ms. Browner:

The Clean Air Scientific Advisory Committee (CASAC) of EPA's Science Advisory Board (SAB) at a meeting on December 12, 1994, reviewed the document entitled Review of the National Ambient Air Quality Standards for Nitrogen Dioxide: Assessment of Scientific and Technical Information, Office of Air Quality Planning and Standards (OAQPS) Staff Paper. At that meeting and in subsequent written comments, the Committee made a number of recommendations for improving the document. On June 2, 1995, a revised Staff Paper was mailed to the CASAC members for review with a letter response. The resulting comments by the Committee members note with satisfaction the improvements made in the scientific quality and completeness of the staff paper. It has been modified in accordance with the CASAC recommendations.

The document is consistent with all aspects of the scientific evidence presented in the criteria document for oxides of nitrogen. It has organized the relevant information in a logical fashion and the Committee believes that it provides a scientifically adequate basis for regulatory decisions on nitrogen dioxide. The staff paper concludes, and the CASAC concurs, that an annual primary standard of the present form and with a numerical value between 0.05 to 0.08 ppm would be supported by the present scientific data on chronic health effects of exposure to nitrogen dioxide. In addition, the CASAC concurs with the rationale that if the existing annual standard of 0.053 ppm is attained, it will provide adequate protection against the occurrence of short term 1-hour peak concentrations of 0.2 ppm or higher.

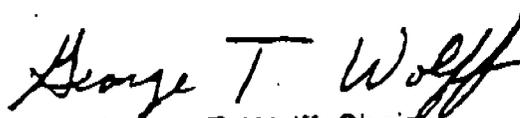


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The staff paper also concludes and the CASAC concurs, that the present primary standard protects against the direct effects of nitrogen oxide exposure to vegetation. Further, the CASAC agrees that there is insufficient scientific evidence available to warrant a secondary standard designed to protect aquatic and terrestrial ecosystems from the adverse effects of nitrogen deposition including eutrophication and acidification. Finally, CASAC concurs with the Staff conclusion that setting an NO₂ secondary standard to protect against visibility impairment is not warranted.

The Committee looks forward to receiving notice of the revised or reaffirmed nitrogen dioxide standard when it is proposed.

Sincerely,

A handwritten signature in cursive script that reads "George T. Wolff". The signature is written in black ink and is positioned above the typed name and title.

Dr. George T. Wolff, Chair
Clean Air Scientific Advisory
Committee



APPENDIX B: ANALYSIS OF CURRENT NO₂ NAAQS ATTAINMENT



APPENDIX B

The following is an excerpt from the report "Analysis of High 1 Hr NO₂ Values and Associated Annual Averages Using 1988-1992 Data" (McCurdy, 1994).

"Attaining the current NO₂ annual average NAAQS of 0.053 ppm is expected to keep 1 hour and daily exceedances of various NO₂ concentration levels below the values shown in Table 16. It should be noted that personal judgement was used to develop some of the minimum and maximum estimates since the 95PI [e.g., the 95th percentile prediction interval around the mean estimate] did not always appear for an indicator. Most of the "problem intervals" involved very small exceedances (i.e., ≤ 2), so any mis-estimates that may exist are small.

1. Non-Los Angeles CMSA Sites

As seen in Table 16, attaining the current NO₂ NAAQS results in no expected (mean) exceedances of 1 hour or daily exceedances >0.20 ppm. The worst area might have 10 exceedances of the EX20 [e.g., the number of observed 1 hour NO₂ values ≥ 0.20 ppm] indicator and 5 exceedances of the DAY20 [e.g., the number of days with ≥ 1 hour NO₂ concentrations ≥ 0.20 ppm] indicator under this situation. Multiple exceedances of EX15 [e.g., the number of observed 1 hour NO₂ values ≥ 0.15 ppm] and DAY15 [e.g., the number of days with ≥ 1 hour NO₂ concentrations ≥ 0.15 ppm] indicators can be expected, however, even if the 0.053 ppm NO₂ NAAQS is attained.

2. Los Angeles Consolidated Metropolitan Statistical Area (CMSA) Sites

Attaining the current NO₂ NAAQS in this area does not guarantee that numerous hourly or daily exceedances of a 0.15 or 0.20 ppm indicator will be prevented. The CMSA has an NO₂ pattern of numerous short-term peaks for relatively low NO₂ annual averages. This finding may indicate that more emphasis should be placed on reducing short-term NO₂ levels in that major metropolitan area. This may mean a change in the areas's NO₂ control strategy to emphasize peak hour reductions that are not

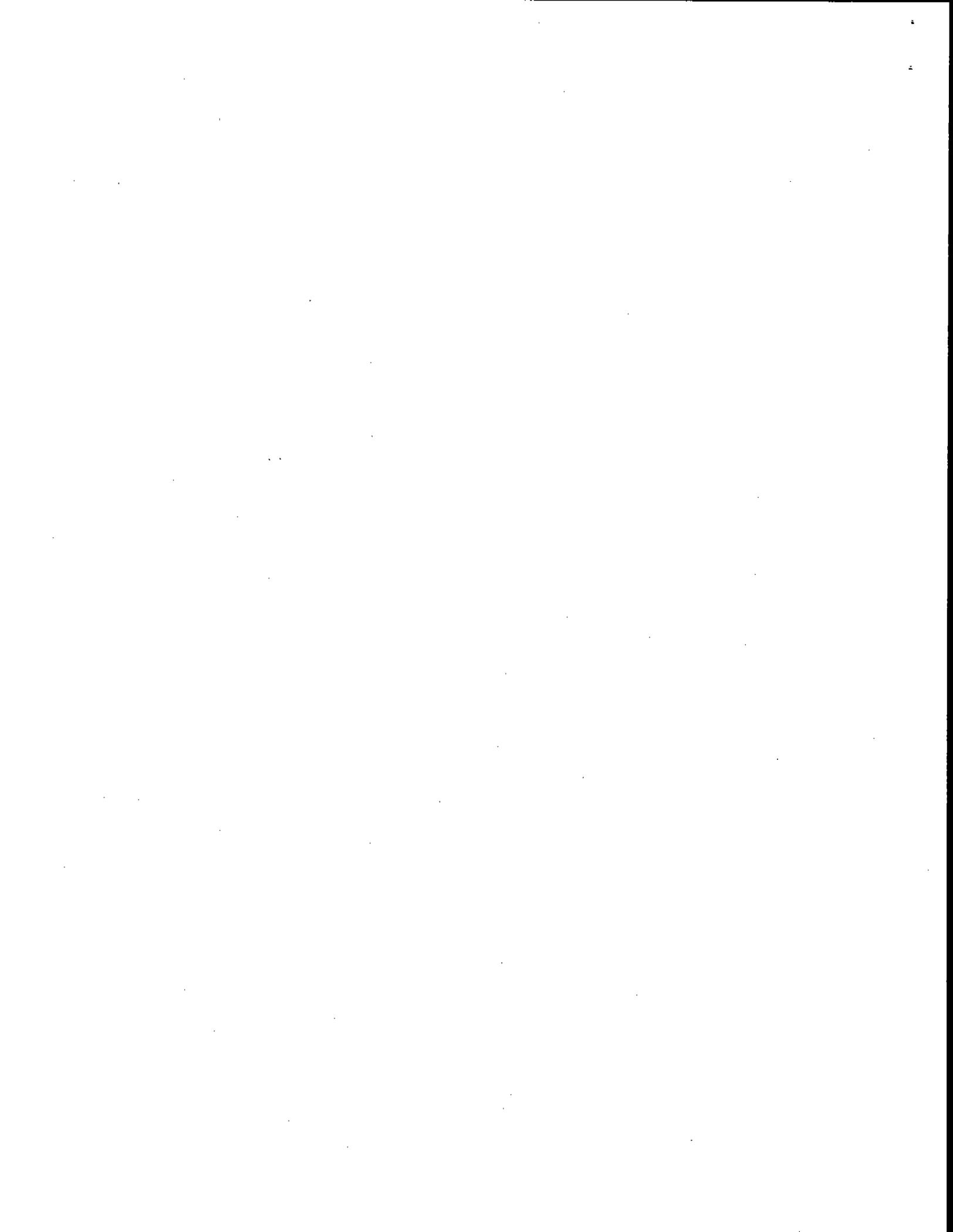
much impacted by area-wide programs, such as motor vehicle emission controls.

The peak levels that we are discussing are 0.15 and 0.20 ppm. Even the Los Angeles CMSA would not be expected to see numerous 1 hour or daily exceedances of a 0.25 ppm peak concentration if the NO₂ NAAQS were attained."

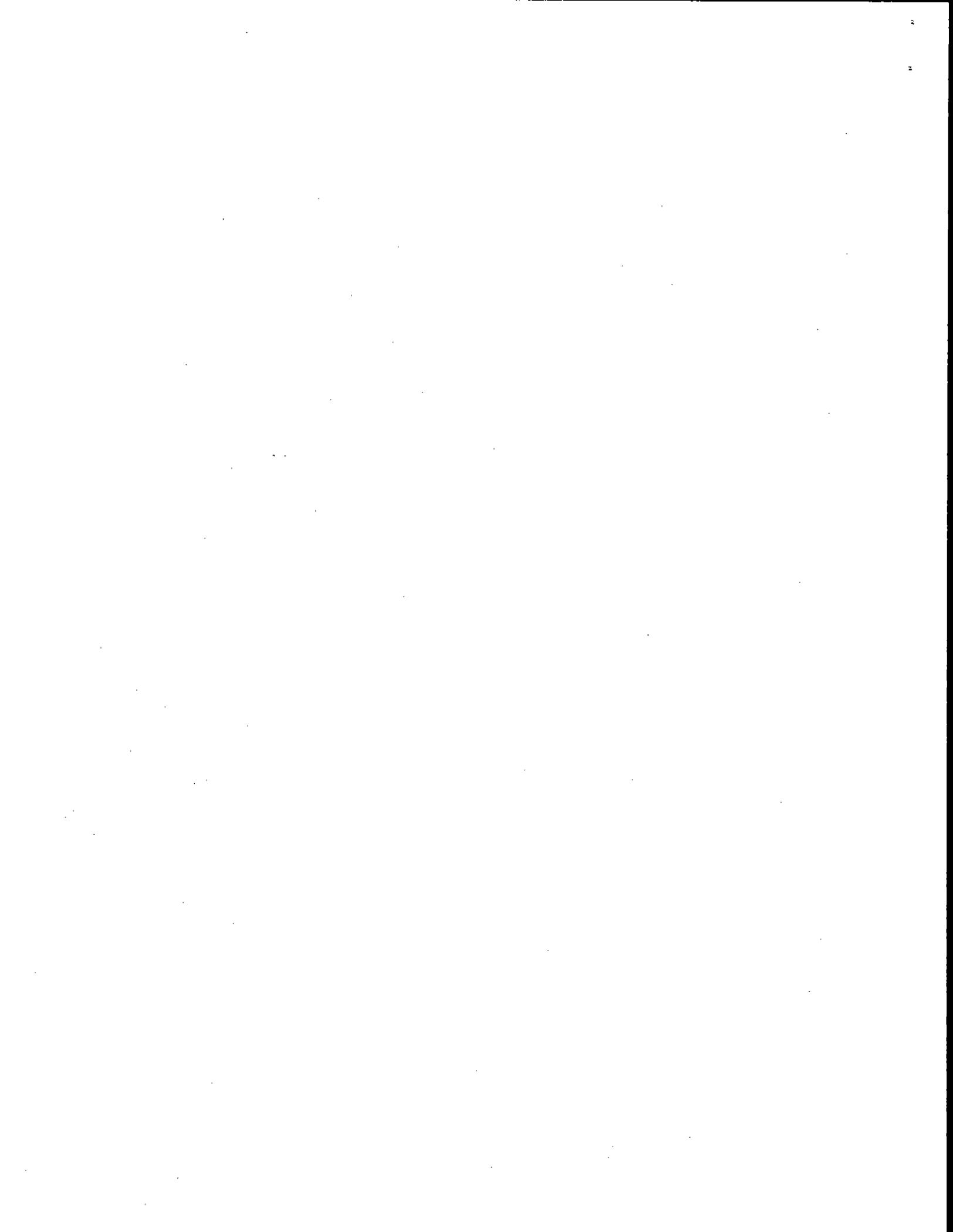
Table 16

ESTIMATED NUMBER OF EXCEEDANCES OF VARIOUS NO₂ SHORT-TERM AIR
 QUALITY INDICATORS GIVEN ATTAINMENT OF THE CURRENT NO₂ NAAQS OF
 0.053 PPM

Concentration Level (ppm)	Minimum Estimate	Mean Estimate	Maximum Estimate
A. 1 h Exceedances in Los Angeles CMSA			
0.15	10	75	195
0.20	0	13	38
0.25	0	1	17
0.30	0	0	2
B. 1 h Exceedances in Other Areas			
0.15	0	5	44
0.20	0	0	10
0.25	0	0	3
0.30	0	0	1
C. Daily Exceedances in Los Angeles CMSA			
0.15	5	27	57
0.20	0	6	21
0.25	0	1	8
0.30	0	0	2
D. Daily Exceedances in Other Areas			
0.15	0	3	9
0.20	0	0	5
0.25	0	0	2
0.30	0	0	1



APPENDIX C: DEFINITIONS



APPENDIX C

DEFINITIONS:

(These definitions were derived from the welfare discussions in the Criteria Document.)

1) Short-term exposures:

a) organismal: the organism is exposed for periods less than or equal to 24 hours in duration.

b) ecosystem: the ecosystem is exposed consistently for less than or equal to 3 years in duration.

2) Long-term exposures:

a) organismal: the organism is exposed for periods greater than 24 hours in duration and typically greater than or equal to two weeks in duration.

b) ecosystem: the ecosystem is exposed consistently for greater than three years in duration.

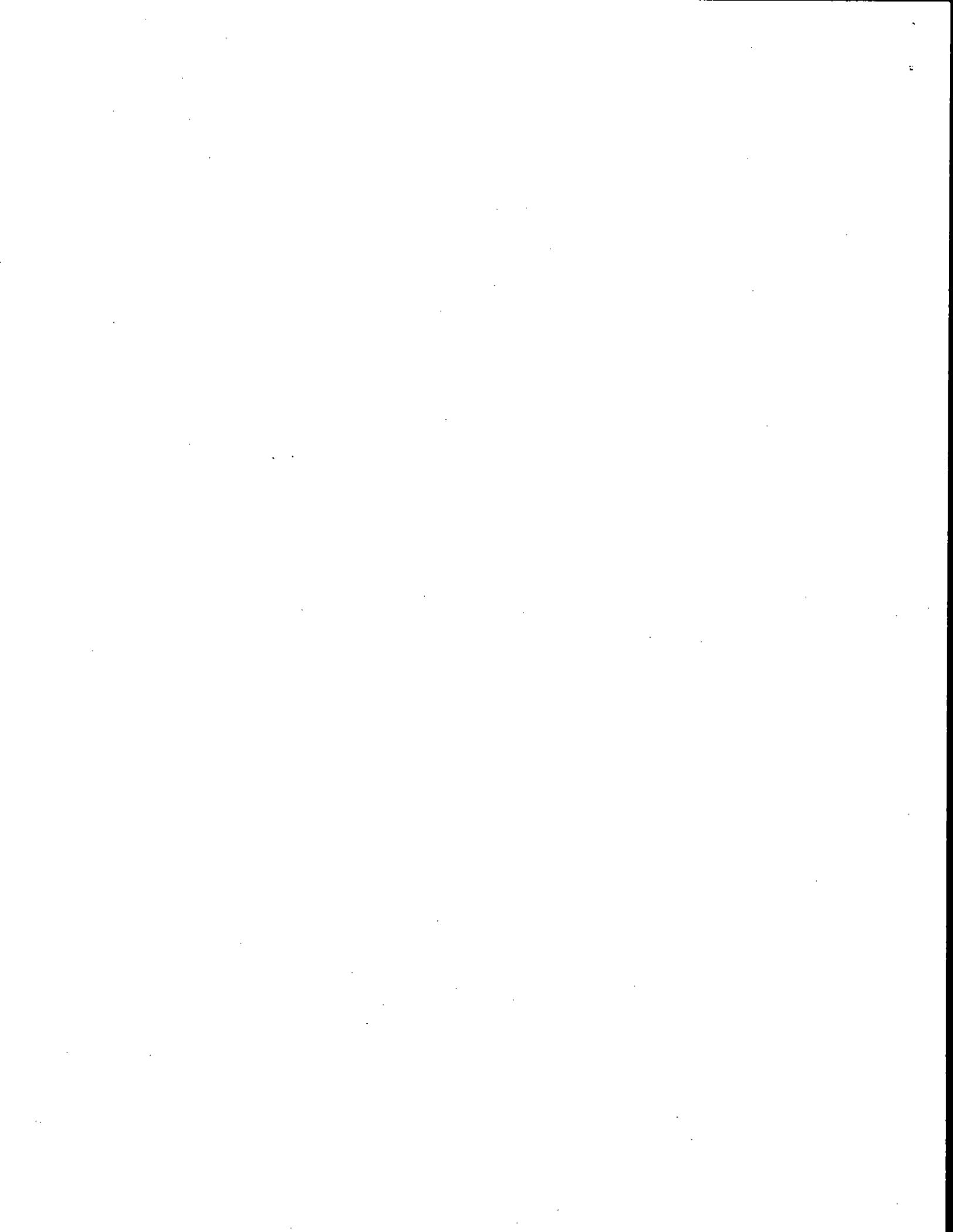
3) Assimilation: the uptake and metabolic use of nitrogen by either aquatic or terrestrial plants. While terrestrial plant species generally favor uptake of NH_4^+ over NO_3^- , the form of nitrogen used will strongly affect the effect nitrogen deposition will have on pH.

4) Nitrification: the oxidation of NH_4^+ to NO_3^- by aquatic or terrestrial bacteria or fungi and is a strongly acidifying process.

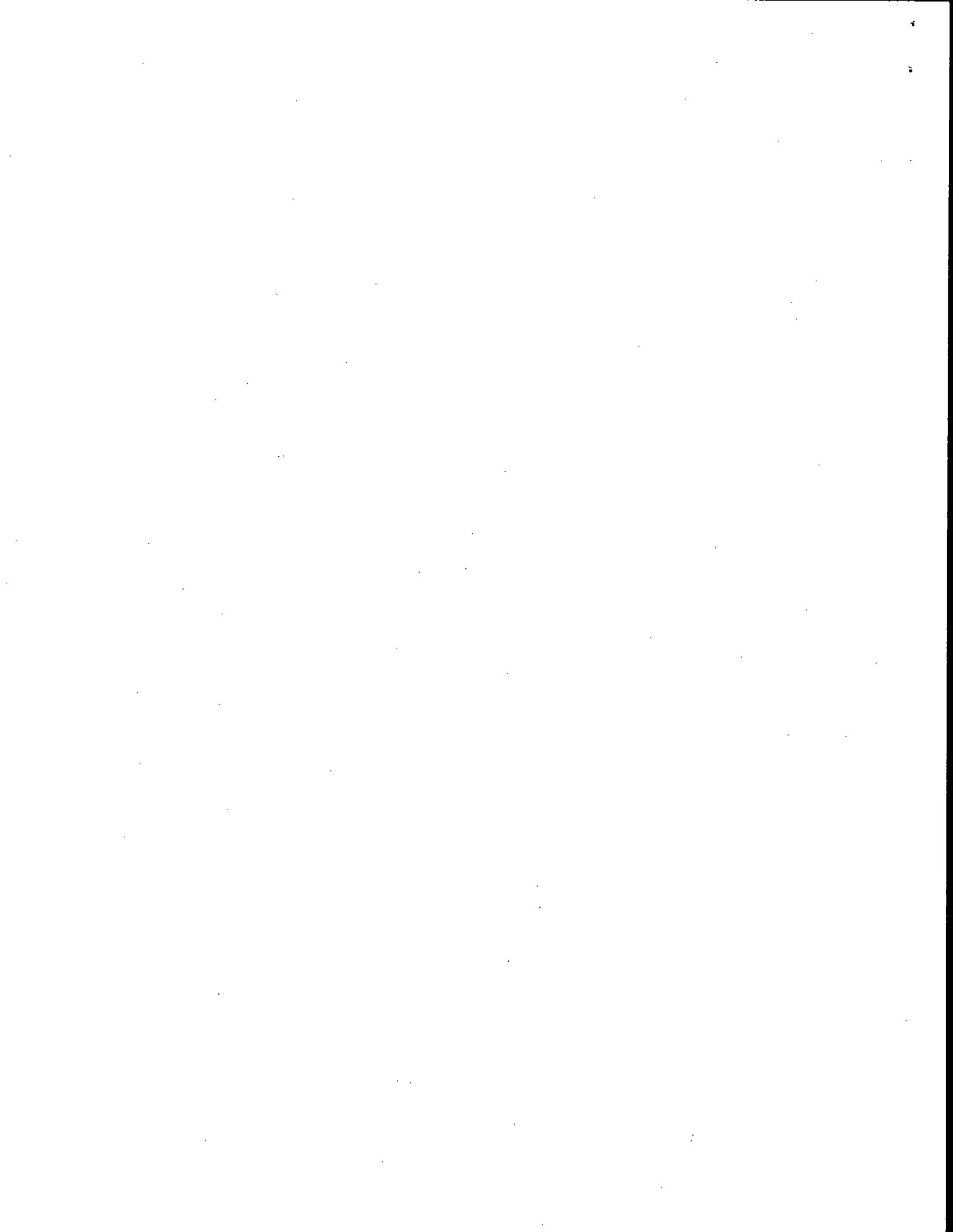
5) Denitrification: an anaerobic process which reduces NO_3^- to N_2 , NO , or N_2O . High rates of N_2O production may be a concern in some areas because of its significance as a greenhouse gas. Denitrification is also always a acidifying process.

6) Nitrogen fixation: the process by which a variety of single-celled organisms and aerobic and anaerobic bacteria fix gaseous atmospheric nitrogen into NH_4^+ . These organisms may be found singly or in symbiotic nitrogen-fixing nodules on roots of some plant species.

7) Mineralization: the bacterial decomposition of organic matter, releasing NH_4^+ that can subsequently be nitrified to NO_3^- . The effect of mineralization on the acid/base status of draining waters will depend on the form of nitrogen produced.



APPENDIX D: REFERENCES



REFERENCES

- Aber, J. D.; Nadelhoffer, K. J.; Steudler, P.; Melillo, J. M. (1989) Nitrogen saturation in northern forest ecosystems: excess nitrogen from fossil fuel combustion may stress the biosphere. *Bioscience* 39: 378-386.
- Ahlquist, M. C., and R. J. Charlson. (1969) Measurement of the wavelength dependence of atmospheric extinction due to scatter. *Atmos. Environ.* 3:551.
- Ahmed, T.; Dougherty, R.; Sackner, M. A. (1983a) Effect of NO₂ exposure on specific bronchial reactivity in subjects with allergic bronchial asthma [final report]. Warren, MI: General Motors Research Laboratories; contract report no. CR-83/07/BI.
- Ahmed, T.; Dougherty, R.; Sackner, M. A. (1983b) Effect of 0.1 ppm NO₂ on pulmonary functions and non-specific bronchial reactivity of normals and asthmatics [final report]. Warren, MI: General Motors Research Laboratories; contract report no. CR-83/11/BI.
- Alexander, M. (1977) Introduction to soil microbiology. 2nd ed. New York, NY: John Wiley & Sons; pp. 225-230; 239-246; 272-286; 355-356.
- Aranyi, C.; Fenters, J.; Erhlich, R.; Gardner, D. (1976) Scanning electron microscopy of alveolar macrophages after exposure to oxygen, nitrogen dioxide, and ozone. *Environ. Health Perspect.* 16: 180.
- Avol, E. L.; Linn, W. S.; Venet, T. G.; Hackney, J. D. (1986) Short-term health-related effects of air pollution relatable to power plants: a combined laboratory and field study [final report: year 1]. Downey, CA: Rancho Los Amigos Medical Center, Environmental Health Service; R&D series 86-RD-75.
- Avol, E. L.; Linn, W. S.; Peng, R. C.; Valencia, G.; Little, D.; Hackney, J. D. (1988) Laboratory study of asthmatic volunteers exposed to nitrogen dioxide and to ambient air pollution. *Am. Ind. Hyg. Assoc. J.* 49: 143-149.
- Bauer, M. A.; Utell, M. J.; Morrow, P. E.; Speers, D. M.; Gibb, F. R. (1986) Inhalation of 0.30 ppm nitrogen dioxide potentiates exercise-induced bronchospasm in asthmatics. *Am. Rev. Respir. Dis.* 134: 1203-1208.
- Berwick, M.; Leaderer, B. P.; Stolwijk, J. A. J.; Zgraniski, R. T. (1987) Association between nitrogen dioxide levels and lower respiratory symptoms in children exposed to unvented combustion sources. In: Seifert, B.; Esdorn, H.; Fischer, M.; Rueden, H.; Wegner, J., eds. *Indoor air '87: proceedings of the 4th international conference on indoor air quality and climate, v. 1, volatile organic compounds, combustion gases, particles and fibers, microbiological agents*; August; Berlin, Federal Republic of Germany. Berlin, Federal Republic of Germany: Institute for Water, Soil and Air Hygiene; pp. 298-303.
- Bormann, F. H.; Likens, G. E.; Melillo, J. M. (1977) Nitrogen budget for an aggrading northern hardwood forest ecosystem. *Science (Washington, DC)* 196: 981-983.
- Boyle, J. R.; Voigt, G. K. (1973) Biological weathering of silicate minerals: implications for tree nutrition and soil genesis. *Plant Soil* 38: 191-201.
- Boynton, W. R.; Kemp, W. M.; Keefe, C. W. (1982) A comparative analysis of nutrients and other factors influencing estuarine phytoplankton production. In: *Estuarine comparisons*. New York, NY: Academic Press, Inc.; pp. 69-90.

- Braun-Fahrlaender, C.; Ackermann-Liebrich, U.; Wanner, H.-U.; Rutishauser, M.; Gnehm, H. E.; Minder, Ch. E. (1989) Auswirkungen von Luftschadstoffen auf die Atemwege von Kleinkindern [Effects of air pollutants on the respiratory tract in young children]. *Schweiz. Med. Wochenschr.* 119: 1,424-1,433.
- Braun-Fahrlaender, C.; Ackermann-Liebrich, U.; Schwartz, J.; Gnehm, H. P.; Rutishauser, M.; Wanner, H.-U. (1992) Air pollution and respiratory symptoms in preschool children. *Am. Rev. Respir. Dis.* 145: 42-47.
- Brown, K. A.; Freer-Smith, P. H.; Howells, G. D.; Skeffington, R. A.; Wilson, R. B. (1988) Rapporteurs' report on discussions at the workshop on excess nitrogen deposition, Leatherhead, September 1987. In: Dempster, J. P.; Manning, W. J.; Skeffington, R. A., eds. *Excess nitrogen deposition: [papers from the workshop]; September 1987; Leatherhead, Surrey, United Kingdom.* *Environ. Pollut.* 54: 285-295.
- Brunekreef, B.; Dockery, D. W.; Speizer, F. E.; Ware, J. H.; Spengler, J. D.; Ferris, B. G. (1989) Home dampness and respiratory morbidity in children. *Am. Rev. Respir. Dis.* 140: 1,363-1,367.
- Bylin, G.; Lindvall, T.; Rehn, T.; Sundin, B. (1985) Effects of short-term exposure to ambient nitrogen dioxide concentrations on human bronchial reactivity and lung function. *Eur. J. Respir. Dis.* 66: 205-217.
- Bylin, G.; Hedenstierna, G.; Lindvall, T.; Sundin, B. (1988) Ambient nitrogen dioxide concentrations increase bronchial responsiveness in subjects with mild asthma. *Eur. Respir. J.* 1: 606-612.
- CASAC (1993) Transcript of the Clean Air Scientific Advisory Committee meeting held on July 1, 1993.
- Centers for Disease Control. (1990) Estimates of the resident population of the United States, by age, sex, and race, July 1, 1989. *Morb. Mortal. Wkly. Rep.* 38: 11.
- Chang, L. Y.; Graham, J. A.; Miller, F. J.; Ospital, J. J.; Crapo, J. D. (1986) Effects of subchronic inhalation of low concentrations of nitrogen dioxide. I. The proximal alveolar region of juvenile and adult rats. *Toxicol. Appl. Pharmacol.* 83: 46-61.
- Charlson, R. J., A. P. Waggoner, and J. F. Thielke (1978) Visibility protection for Class I areas. The technical basis. Report to Council of Environmental Quality, Washington, D.C.
- Coffin, D. L.; Gardner, D. E.; Sidorenko, G. I.; Pinigin, M. A. (1977) Role of time as a factor in the toxicity of chemical compounds in intermittent and continuous exposures. Part II. Effects of intermittent exposure. *J. Toxicol. Environ. Health* 3: 821-828.
- Cole, D. W.; Rapp, M. (1981) Elemental cycling in forest ecosystems. In: Reichle, D. E., ed. *Dynamic properties of forest ecosystems.* Cambridge, United Kingdom: Cambridge University Press; pp. 341-409. (International biological programme 23).
- Dekker, C.; Dales, R.; Bartlett, S.; Brunekreef, B.; Zwanenburg, H. (1991) Childhood asthma and the indoor environment. *Chest* 100: 922-926.
- Dijkstra, L.; Houthuijs, D.; Brunekreef, B.; Akkerman, I.; Boleij, J. S. M. (1990) Respiratory health effects of the indoor environment in a population of Dutch children. *Am. Rev. Respir. Dis.* 142: 1172-1178.

- ²Dockery, D. W.; Spengler, J. D.; Neas, L. M.; Speizer, F. E.; Ferris, B. G., Jr.; Ware, J. H.; Brunekreef, B. (1989a) An epidemiologic study of respiratory health status and indicators of indoor air pollution from combustion sources. In: Harper, J. P., ed. Combustion processes and the quality of the indoor environment: transactions of an international specialty conference; September 1988; Niagara Falls, NY. Pittsburgh, PA: Air & Waste Management Association; pp. 262-271. (A&WMA transactions series:TR-15).
- ²Dockery, D. W.; Speizer, F. E.; Stram, D. O.; Ware, J. H.; Spengler, J. D.; Ferris, B. G., Jr. (1989b) Effects of inhalable particles on respiratory health of children. *Am. Rev. Respir. Dis.* 139: 587-594.
- Draaijers, G. P. J.; Ivens, W. P. M. F.; Bos, M. M.; Bleuten, W. (1989) The contribution of ammonia emissions from agriculture to the deposition of acidifying and eutrophying compounds onto forests. *Environ. Pollut.* 60: 55-66.
- Driscoll, C. T.; Schaefer, D. A.; Molot, L. A.; Dillon, P. J. (1989) Summary of North American data. In: Malanchuk, J. L.; Nilsson, J., eds. The role of nitrogen in the acidification of soils and surface waters. Gotab, Sweden: Nordic Council of Ministers; pp. 6-1 - 6-45.
- Driscoll, C. T.; Schaefer, D. A. (1989) Background on nitrogen processes. In: Malanchuk, J. L.; Nilsson, J., eds. The role of nitrogen in the acidification of soils and surface waters. Miljorapport, Sweden: Nordic Council of Ministers; pp. 4-1 - 4-12.
- Driscoll, C. T.; Schafran, G. C. (1984) Short-term changes in the base neutralizing capacity of an acid Adirondack lake, New York. *Nature (London)* 310: 308-310.
- Driscoll, C. T.; Van Dreason, R. (n.d.) Seasonal and long-term temporal patterns in the chemistry of Adirondack lakes. *Water Air Soil Pollut.*: in press.
- ²D'Elia, C. F.; Sanders, J. G.; Boynton, W. R. (1986) Nutrient enrichment studies in a coastal plain estuary: phytoplankton growth in large-scale, continuous cultures. *Can. J. Fish. Aquat. Sci.* 43: 397-406.
- D'Elia, C. F.; Taft, J.; Smullen, J. T.; Macknis, J. (1982) Nutrient enrichment. In: Chesapeake Bay Program technical studies: a synthesis. Annapolis, MD: U.S. Environmental Protection Agency; pp. 36-102. Available from: NTIS, Springfield, VA; PB84-111202.
- Ehrlich, R.; Henry, M. C. (1968) Chronic toxicity of nitrogen dioxide: I. effect on resistance to bacterial pneumonia. *Arch. Environ. Health* 17: 860-865.
- Ehrlich, R.; Findlay, J. C.; Fenters, J. D.; Gardner, D. E. (1977) Health effects of short-term inhalation of nitrogen dioxide and ozone mixtures. *Environ. Res.* 14: 223-231.
- Ekwo, E. E.; Weinberger, M. M.; Lachenbruch, P. A.; Huntley, W. H. (1983) Relationship of parental smoking and gas cooking to respiratory disease in children. *Chest* 84: 662-668.
- Ellenberg, H. (1988) Floristic changes due to nitrogen deposition in central Europe. In: Nilsson, J.; Grennfelt, P., eds. Critical loads for sulphur and nitrogen: report from a workshop; March; Skokloster, Sweden. Copenhagen, Denmark: Nordic Council of Ministers; pp. 375-383.

- Elwood, J. W.; Sale, M. J.; Kaufmann, P. R.; Cada, G. F. (1991) The Southern Blue Ridge Province. In: Charles, D. F., ed. *Acidic deposition and aquatic ecosystems: regional case studies*. New York, NY: Springer-Verlag; pp. 319-364.
- Euler, G.L.; Abbey, D.E.; Hodgkin, J.E.; Magie, A.R. (1988) Chronic obstructive pulmonary disease symptom effects of long-term cumulative exposure to ambient levels of total oxidants and nitrogen dioxide in California Seventh-day Adventist residents. *Arch. Environ. Health* 43: 279-285.
- Feller, M. C. (1987) The influence of acid precipitation on stream chemistry in a small forested basin in southwestern British Columbia. In: Swanson, R. H.; Bernier, P. Y.; Woodward, P. D., eds. *Forest hydrology and watershed management: proceedings of the Vancouver symposium*; August. IAHS-AISH publication no. 167.
- Ferguson, P.; Robinson, R. N.; Press, M. C.; Lee, J. A. (1984) Element concentrations in five *Sphagnum* species in relation to atmospheric pollution. *J. Bryol.* 13: 107-114.
- Fisher, T. R.; Harding, L. W., Jr.; Stanley, D. W.; Ward, L. G. (1988a) Phytoplankton, nutrients, and turbidity in the Chesapeake, Delaware, and Hudson estuaries. *Estuarine Coastal Shelf Sci.* 27: 61-93.
- Fisher, F. M.; Zak, J. C.; Cunningham, G. L.; Whitford, W. G. (1988b) Water and nitrogen effects on growth and allocation patterns of creosotebush in the northern Chihuahuan Desert. *J. Range Manage.* 41: 387-391.
- Florey, C. du V.; Melia, R. J. W.; Chinn, S.; Goldstein, B. D.; Brooks, A. G. F.; John, H. H.; Craighead, I. B.; Webster, X. (1979) The relation between respiratory illness in primary schoolchildren and the use of gas for cooking: III—nitrogen dioxide, respiratory illness and lung infection. *Int. J. Epidemiol.* 8: 347-353.
- Florey, C. du V.; Melia, R.; Goldstein, B.; Morris, R.; John, H. H.; Clark, D. (1982) Stickstoffdioxid im Innenraum—Epidemiologische Auswirkungen im Vereinigten Koenigreich [The epidemiology of indoor nitrogen dioxide in the U. K.]. In: Aurand, K.; Seifert, B.; Wegner, J., eds. *Luftqualitaet in Innenraeumen* [Indoor air quality]. Stuttgart, Federal Republic of Germany: Gustav Fischer Verlag; pp. 209-218. (Schriftenreihe des Vereins fuer Wasser-, Boden- und Lufthygiene no. 53).
- Focht, D. D. (1974) The effect of temperature, Ph, and aeration on the production of nitrous oxide and gaseous nitrogen—zero-order kinetic model. *Soil Sci.* 118: 173-179.
- Folinsbee, L. J. (1992) Does nitrogen dioxide exposure increase airways responsiveness? *Toxicol. Ind. Health* 8: 1-11.
- Foster, N. W.; Nicolson, J. A. (1988) Acid deposition and nutrient leaching from deciduous vegetation and podzolic soils at the Turkey Lakes Watershed. *Can. J. Fish. Aquat. Sci.* 45(suppl. 1): 96-100.
- Fowler, D.; Cape, J. N.; Unsworth, M. H. (1989) Deposition of atmospheric pollutants on forests. *Philos. Trans. R. Soc. London B* 324: 247-265.
- Frampton, M. W.; Finkelstein, J. N.; Roberts, N. J., Jr.; Smeglin, A. M.; Morrow, P. E.; Utell, M. J. (1989) Effects of nitrogen dioxide exposure on bronchoalveolar lavage proteins in humans. *Am. J. Respir. Cell Mol. Biol.* 1: 499-505.
- Frampton, M. W.; Morrow, P. E.; Cox, C.; Gibb, F. R.; Speers, D. M.; Utell, M. J. (1991) Effects of nitrogen dioxide exposure on pulmonary function and airway reactivity in normal humans. *Am. Rev. Respir. Dis.* 143: 522-527.

- Franey, J. P.; Graedel, T. E. (1985) Corrosive effects of mixtures of pollutants. *J. Air Pollut. Control Assoc.* 35: 644-648.
- Freeman, B. A.; Mudd, J. B. (1981) Reaction of ozone with sulfhydryls of human erythrocytes. *Arch. Biochem. Biophys.* 208: 212-220.
- Freeman, G.; Crane, S. C.; Furiosi, N. J.; Stephens, R. J.; Evans, M. J.; Moore, W. D. (1972) Covert reduction in ventilatory surface in rats during prolonged exposure to subacute nitrogen dioxide. *Am. Rev. Respir. Dis.* 106: 563-579.
- Fujimaki, H.; Shimizu, F.; Kubota, K. (1982) Effect of subacute exposure to NO₂ on lymphocytes required for antibody responses. *Environ. Res.* 29: 280-286.
- Galloway, J. N.; Schofield, C. L.; Hendrey, G. R.; Peters, N. E.; Johannes, A. H. (1980) Sources of acidity in three lakes acidified during snowmelt. In: Drablos, D.; Tollan, A., eds. *Ecological effects of acid precipitation: proceedings of an international conference*. Mysen, Norway: Johs. Grefslie Trykkeri A/S; pp. 264-265.
- Gardner, D. E.; Coffin, D. L.; Pinigin, M. A.; Sidorenko, G. I. (1977a) Role of time as a factor in the toxicity of chemical compounds in intermittent and continuous exposures. Part I. Effects of continuous exposure. *J. Toxicol. Environ. Health* 3: 811-820.
- Gardner, D. E.; Miller, F. J.; Blommer, E. J.; Coffin, D. L. (1977b) Relationships between nitrogen dioxide concentration, time, and level of effect using an animal infectivity model. In: Dimitriadis, B., ed. *International conference on photochemical oxidant pollution and its control: proceedings, v. I; September 1976; Raleigh, NC. Research Triangle Park, NC: U.S. Environmental Protection Agency, Environmental Sciences Research Laboratory; pp. 513-525; EPA report no. EPA-600/3-77-001a. Available from: NTIS, Springfield, VA; PB-264232. (Ecological research series).*
- Gardner, D. E.; Miller, F. J.; Blommer, E. J.; Coffin, D. L. (1979) Influence of exposure mode on the toxicity of NO₂. *Environ. Health Perspect.* 30: 23-29.
- Gardner, D. E.; Graham, J. A.; Illing, J. W.; Blommer, E. J.; Miller, F. J. (1980) Impact of exposure patterns on the toxicological response to NO₂ and modifications by added stressors. In: *Proceedings of the US-USSR third joint symposium on problems of environmental health; October 1979; Suzdal, USSR. Research Triangle Park, NC: National Institute of Environmental Health Sciences; pp. 17-40.*
- Gardner, D. E.; Miller, F. J.; Illing, J. W.; Graham, J. A. (1982) Non-respiratory function of the lungs: host defenses against infection. In: Schneider, T.; Grant, L., eds. *Air pollution by nitrogen oxides: proceedings of the US-Dutch international symposium; May; Maastricht, The Netherlands. Amsterdam, The Netherlands: Elsevier Scientific Publishing Company; pp. 401-415. (Studies in environmental science 21).*
- Glezen, W. P. (1989) Antecedents of chronic and recurrent lung disease: childhood respiratory trouble. *Am. Rev. Respir. Dis.* 140: 873-874.
- Goings, S. A. J.; Kulle, T. J.; Bascom, R.; Sauder, L. R.; Green, D. J.; Hebel, J. R.; Clements, M. L. (1989) Effect of nitrogen dioxide exposure on susceptibility to influenza A virus infection in healthy adults. *Am. Rev. Respir. Dis.* 139: 1075-1081.

- Gold, D. R.; Tager, I. B.; Weiss, S. T.; Tosteson, T. D.; Speizer, F. E. (1989) Acute lower respiratory illness in childhood as a predictor of lung function and chronic respiratory symptoms. *Am. Rev. Respir. Dis.* 140: 877-884.
- Goldstein, E.; Eagle, M. C.; Hoepflich, P. D. (1973) Effect of nitrogen dioxide on pulmonary bacterial defense mechanisms. *Arch. Environ. Health* 26: 202-204.
- Goldstein, B. D.; Melia, R. J. W.; Chinn, S.; Florey, C. du V.; Clark, D.; John, H. H. (1979) The relation between respiratory illness in primary schoolchildren and the use of gas for cooking. II. Factors affecting nitrogen dioxide levels in the home. *Int. J. Epidemiol.* 8: 339-345.
- Goldstein, B. D.; Melia, R. J. W.; Florey, C. du V. (1981) Indoor nitrogen oxides. *Bull. N. Y. Acad. Med.* 57: 873-882.
- Graham, J. A.; Gardner, D. E.; Blommer, E. J.; House, D. E.; Menache, M. G.; Miller, F. J. (1987) Influence of exposure patterns of nitrogen dioxide and modifications by ozone on susceptibility to bacterial infectious disease in mice. *J. Toxicol. Environ. Health* 21: 113-125.
- Hantschel, R.; Kaupenjohann, M.; Horn, R.; Zech, W. (1990) Water, nutrient and pollutant budgets in damaged Norway spruce stands in NE-Bavaria (F.R.G.) and their changes after different fertilization treatments. *Water Air Soil Pollut.* 49: 273-297.
- Hasselblad, V.; Eddy, D. M.; Kotchmar, D. J. (1992) Synthesis of environmental evidence: nitrogen dioxide epidemiology studies. *J. Air Waste Manage. Assoc.* 42: 662-671.
- Hauhs, M. (1989) Lange Bramke: an ecosystem study of a forested catchment. In: Adriano, D. C.; Havas, M., eds. *Acidic precipitation: volume 1, case studies*. New York, NY: Springer-Verlag; pp. 275-305. (Adriano, D. C.; Salomons, W., eds. *Advances in environmental science*).
- Haydon, G. B.; Davidson, J. T.; Lillington, G. A.; Wasserman, K. (1967) Nitrogen dioxide-induced emphysema in rabbits. *Am. Rev. Respir. Dis.* 95: 797-805.
- Hazucha, M. J.; Ginsberg, J. F.; McDonnell, W. F.; Haak, E. D., Jr.; Pimmel, R. L.; House, D. E.; Bromberg, P. A. (1982) Changes in bronchial reactivity of asthmatics and normals following exposures to 0.1 ppm NO₂. In: Schneider, T.; Grant, L., eds. *Air pollution by nitrogen oxides: proceedings of the US-Dutch international symposium; May; Maastricht, The Netherlands*. Amsterdam, The Netherlands: Elsevier Scientific Publishing Company; pp. 387-400. (*Studies in environmental science* 21).
- Hazucha, M. J.; Ginsberg, J. F.; McDonnell, W. F.; Haak, E. D., Jr.; Pimmel, R. L.; Salaam, S. A.; House, D. E.; Bromberg, P. A. (1983) Effects of 0.1 ppm nitrogen dioxide on airways of normal and asthmatic subjects. *J. Appl. Physiol.: Respir. Environ. Exercise Physiol.* 54: 730-739.
- Hazucha, M. J.; Seal, E.; Folinsbee, L. J.; Bromberg, P. A. (1992) Lung function response of healthy subjects following sequential exposures to NO₂ and O₃. *Am. Rev. Respir. Dis.* 145: A456.

- Hedberg, K.; Hedberg, C. W.; Iber, C.; White, K. E.; Osterholm, M. T.; Jones, D. B. W.; Flink, J. R.; MacDonald, K. L. (1989) An outbreak of nitrogen dioxide-induced respiratory illness among ice hockey players. *JAMA J. Am. Med. Assoc.* 262: 3,014-3,017.
- Henderson, G. S.; Harris, W. F. (1975) An ecosystem approach to characterization of the nitrogen cycle in a deciduous forest watershed. In: Bernier, B.; Winget, C. H., eds. *Forest soils and forest land management: proceedings of the fourth North American forest soils conference; August 1973; Quebec, PQ, Canada.* Quebec, PQ, Canada: Les Presses de L'Universite Laval; pp. 179-183.
- Henriksen, A. (1988) Critical loads of nitrogen to surface waters. In: Nilsson, J.; Grennfelt, P., eds. *Critical loads for sulphur and nitrogen: report from a workshop; March; Skokloster, Sweden.* Copenhagen, Denmark: Nordic Council of Ministers; pp. 385-412.
- Henriksen, A.; Brakke, D. F. (1988) Increasing contributions of nitrogen to the acidity of surface waters in Norway. *Water Air Soil Pollut.* 42: 183-201.
- Hicks, B. B.; Draxler, R. R.; Albritton, D. L.; Fehsenfeld, F. C.; Dodge, M.; Schwartz, S. E.; Tanner, R. L.; Hales, J. M.; Meyers, T. P.; Vong, R. J. (1991) Atmospheric processes research and process model development. In: Irving, P. M., ed. *Acidic deposition: state of science and technology, volume I, emissions, atmospheric processes and deposition.* Washington, DC: The U.S. National Acid Precipitation Assessment Program. (State of science and technology report no. 2).
- Howarth, R. W.; Marino, R.; Lane, J.; Cole, J. J. (1988) Nitrogen fixation in freshwater, estuarine, and marine ecosystems. 1. Rates and importance. *Limnol. Oceanogr.* 33: 669-687.
- Howes, B. L.; Dacey, J. W. H.; Goehring, D. D. (1986) Factors controlling the growth form of *Spartina alterniflora*: feedbacks between above-ground production, sediment oxidation, nitrogen and salinity. *J. Ecol.* 74: 881-898.
- Husar, R. B. (1986) Emissions of sulfur dioxide and nitrogen oxides and trends for eastern North America. In: *Acid deposition: long-term trends.* Washington, DC: National Academy Press; pp. 48-92.
- Hyde, D.; Orthofer, J.; Dungworth, D.; Tyler, W.; Carter, R.; Lum, H. (1978) Morphometric and morphologic evaluation of pulmonary lesions in beagle dogs chronically exposed to high ambient levels of air pollutants. *Lab. Invest.* 38: 455-469.
- Islam, M. S.; Ulmer, W. T. (1979a) Beeinflussung der Lungenfunktion durch ein Schadstoffgemisch aus Ozon (O₃), Schwefeldioxyd (SO₂) und Stickstoffdioxyd (NO₂) im MAK-Bereich (Kurzzeitversuch) [The influence of acute exposure against a combination of 5.0 ppm SO₂, 5.0 ppm NO₂ and 0.1 ppm O₃ on the lung function in the MAK (lower toxic limit) area (short-time test)]. *Wiss. Umwelt* 3: 131-137.
- Islam, M. S.; Ulmer, W. T. (1979b) Die Wirkung einer Langzeitexposition (8h/Tag ueber 4 Tage) gegen ein Gasmisch von SO₂ + NO₂ + O₃ im dreifachen MIK-Bereich auf die Lungenfunktion und Bronchialreagibilitaet bei gesunden Versuchspersonen [The effects of long-time exposures (8 h per day on 4 successive days) to a gas mixture of SO₂ + NO₂ + O₃ in the threefold MIC range (maximum emission concentration) on lung functions and reactivity of the bronchial system of healthy persons]. *Wiss. Umwelt* 4: 186-190.
- Ito, K. (1971) [Effect of nitrogen dioxide inhalation on influenza virus infection in mice]. *Nippon Eiseigaku Zasshi* 26: 304-314.

- Jaakkola, J. J. K.; Paunio, M.; Virtanen, M.; Heinonen, O. P. (1991) Low-level air pollution and upper respiratory infections in children. *Am. J. Public Health* 81: 1,060-1,063.
- Jakab, G. J. (1987a) Modulation of pulmonary defense mechanisms by acute exposures to nitrogen dioxide. *Environ. Res.* 42: 215-228.
- Jakab, G. J. (1987b) Modulation of pulmonary defense mechanisms by acute exposures to nitrogen dioxide. *Experientia Suppl.* 51: 235-242.
- Jefferies, R. L.; Perkins, N. (1977) The effects on the vegetation of the additions of inorganic nutrients to salt marsh soils at Stiffkey, Norfolk. *J. Ecol.* 65: 867-882.
- Johannessen, M.; Skartveit, A.; Wright, R. F. (1980) Streamwater chemistry before, during and after snowmelt. In: Drablos, D.; Tollan, A., eds. *Ecological impact of acid precipitation: proceedings of an international conference; March; Sandefjord, Norway.* Oslo, Norway: SNSF project; pp. 224-225.
- Johnson, D. W.; Cole, D. W.; Gessel, S. P.; Singer, M. J.; Minden, R. V. (1977) Carbonic acid leaching in a tropical, temperate, subalpine and northern forest soil. *Arct. Alp. Res.* 9: 329-343.
- Johnson, D. W.; Lindberg, S. E., eds. (1992) *Atmospheric deposition and forest nutrient cycling: a synthesis of the integrated forest study.* Ecosystems. New York, NY: Springer-Verlag.
- Johnson, D. W.; Todd, D. E. (1988) Nitrogen fertilization of young yellow poplar and loblolly pine plantations at differing frequencies. *Soil Sci. Soc. Am. J.* 52: 1468-1477.
- Karon, J. M.; Dondero, T. J., Jr.; Workshop Group. (1990) HIV prevalence estimates and AIDS case projections for the United States: report based upon a workshop. *Morb. Mortal. Wkly. Rep.* 39 (November 20): 12.
- Kaufmann, P. R.; Herlihy, A. T.; Mitch, M. E.; Messer, J. J.; Overton, W. S. (1991) Stream chemistry in the eastern United States: 1. synoptic survey design, acid-base status, and regional patterns. *Water Resour. Res.* 27: 611-627.
- Keller, M. D.; Lanese, R. R.; Mitchell, R. I.; Cote, R. W. (1979a) Respiratory illness in households using gas and electricity for cooking: I. survey of incidence. *Environ. Res.* 19: 495-503.
- Keller, M. D.; Lanese, R. R.; Mitchell, R. I.; Cote, R. W. (1979b) Respiratory illness in households using gas and electricity for cooking: II. symptoms and objective findings. *Environ. Res.* 19: 504-515.
- Kelly, J. M. (1988) Annual elemental input/output estimates for two forested watersheds in eastern Tennessee. *J. Environ. Qual.* 17: 463-468.
- Kelly, J. M.; Meagher, J. F. (1986) Nitrogen input/output relationships for three forested watersheds in eastern Tennessee. In: Correll, D. L., ed. *Watershed research perspectives.* Washington, DC: Smithsonian Institution Press; pp. 360-391.
- Kerr, H. D.; Kulle, T. J.; McIlhany, M. L.; Swidersky, P. (1979) Effects of nitrogen dioxide on pulmonary function in human subjects: an environmental chamber study. *Environ. Res.* 19: 392-404.
- Kleinmann, M. T.; Mautz, W. J. (1991) The effects of exercise on dose and dose distribution of inhaled automotive pollutants. Cambridge, MA: Health Effects Institute; research report number 45.

- Koenig, J. Q.; Covert, D. S.; Morgan, M. S.; Horike, M.; Horike, N.; Marshall, S. G.; Pierson, W. E. (1985) Acute effects of 0.12 ppm ozone or 0.12 ppm nitrogen dioxide on pulmonary function in healthy and asthmatic adolescents. *Am. Rev. Respir. Dis.* 132: 648-651.
- Koenig, J. Q.; Pierson, W. E.; Marshall, S. G.; Covert, D. S.; Morgan, M. S.; Van Belle, G. (1987a) The effects of ozone and nitrogen dioxide on lung function in healthy and asthmatic adolescents. Cambridge, MA: Health Effects Institute; research report no. 14.
- Koenig, J. Q.; Covert, D. S.; Marshall, S. G.; Van Belle, G.; Pierson, W. E. (1987b) The effects of ozone and nitrogen dioxide on pulmonary function in healthy and in asthmatic adolescents. *Am. Rev. Respir. Dis.* 136: 1152-1157.
- Lee, J. A.; Press, M. C.; Woodin, S. J. (1986) Effects of NO₂ on aquatic ecosystems. In: *Environment and quality of life: study on the need for an NO₂ long-term limit value for the protection of terrestrial and aquatic ecosystems*. Luxembourg: Commission of the European Communities; pp. 99-119.
- Levy, H., II; Moxim, W.J. (1987) Fate of US and Canadian combustion nitrogen emissions. *Nature (London)* 328: 414-416.
- Likens, G. E., ed. (1985) *An ecosystem approach to aquatic ecology: Mirror Lake and its environment*. New York, NY: Springer-Verlag.
- Likens, G. E.; Bormann, F. H.; Johnson, N. M.; Fisher, D. W.; Pierce, R. S. (1970) Effects of forest cutting and herbicide treatment on nutrient budgets in the Hubbard Brook watershed-ecosystem. *Ecol. Monogr.* 40: 23-47.
- Likens, G. E.; Bormann, F. H.; Pierce, R. S.; Eaton, J. S.; Johnson, N. M. (1977) *Biogeochemistry of a forested ecosystem*. New York, NY: Springer-Verlag, Inc.
- Liljelund, L.-E.; Torstensson, P. (1988) Critical load of nitrogen with regards to effects on plant composition. In: Nilsson, J.; Grennfelt, P., eds. *Critical loads for sulphur and nitrogen: report from a workshop*; March; Skokloster, Sweden. Copenhagen, Denmark: Nordic Council of Ministers; pp. 363-373.
- Lindberg, S. E.; Lovett, G. M.; Richter, D. D.; Johnson, D. W. (1986) Atmospheric deposition and canopy interactions of major ions in a forest. *Science (Washington, DC)* 231: 141-145.
- Linn, W. S.; Hackney, J. D. (1984) Short-term human respiratory effects of nitrogen dioxide: determination of quantitative dose-response profiles, phase II. exposure of asthmatic volunteers to 4 ppm NO₂. Atlanta, GA: Coordinating Research Council, Inc.; report no. CRC-CAPM-48-83-02. Available from: NTIS, Springfield, VA; PB85-104388/XAB.
- Linn, W. S.; Shamoo, D. A.; Spier, C. E.; Valencia, L. M.; Anzar, U. T.; Venet, T. G.; Avol, E. L.; Hackney, J. D. (1985a) Controlled exposure of volunteers with chronic obstructive pulmonary disease to nitrogen dioxide. *Arch. Environ. Health* 40: 313-317.
- Linn, W. S.; Solomon, J. C.; Trim, S. C.; Spier, C. E.; Shamoo, D. A.; Venet, T. G.; Avol, E. L.; Hackney, J. D. (1985b) Effects of exposure to 4 ppm nitrogen dioxide in healthy and asthmatic volunteers. *Arch. Environ. Health* 40: 234-239.

- Linn, W. S.; Shamoo, D. A.; Avol, E. L.; Whynot, J. D.; Anderson, K. R.; Venet, T. G.; Hackney, J. D. (1986) Dose-response study of asthmatic volunteers exposed to nitrogen dioxide during intermittent exercise. *Arch. Environ. Health* 41: 292-296.
- Linsey, G. A.; Schindler, D. W.; Stainton, M. P. (1987) Atmospheric deposition of nutrients and major ions at the Experimental Lakes Area in northwestern Ontario, 1970 to 1982. *Can. J. Fish. Aquat. Sci.* 44 (suppl. 1): 206-214.
- Lipfert, F. W. (1989) Dry deposition velocity as an indicator for SO₂ damage to materials. *JAPCA* 39:446-452.
- Logofet, D. O.; Alexandrov, G. A. (1984) Modelling of matter cycle in a mesotrophic bog ecosystem: II. dynamic model and ecological succession. *Ecol. Modell.* 21: 259-276.
- Lovett, G. M. (1992) Atmospheric deposition and canopy interactions of nitrogen. In: Johnson, D. W.; Lindberg, S. E., eds. *Atmospheric deposition and forest nutrient cycling: a synthesis of the integrated forest study*. New York, NY: Springer-Verlag; pp. 152-166.
- Lovett, G. M.; Reiners, W. A.; Olson, R. K. (1982) Cloud droplet deposition in subalpine balsam fir forests: hydrological and chemical inputs. *Science (Washington, DC)* 218: 1303-1304.
- Margolis, P. A.; Greenberg, R. A.; Keyes, L. L.; Lavange, L. M.; Chapman, R. S.; Denny, F. W.; Bauman, K. E.; Boat, B. W. (1992) Lower respiratory illness in infants and low socioeconomic status. *Am. J. Public Health* 82: 1119-1126.
- McComb, A. J.; Atkins, R. P.; Birch, P. B.; Gordon, D. M.; Lukatelich, R. J. (1981) Eutrophication in the Peel-Harvey estuarine system, Western Australia. In: Neilson, B. J.; Cronin, L. E., eds. *Estuaries and nutrients*. Clifton, NJ: Humana Press; pp. 323-342.
- McCurdy, T. R. (1994) Analysis of high 1 hour NO₂ values and associated annual averages using 1988-1992 data. Report of the Office of Air Quality Planning and Standards, Durham, NC. Available in Docket A-93-06.
- Melia, R. J. W.; Florey, C. du V.; Altman, D. G.; Swan, A. V. (1977) Association between gas cooking and respiratory disease in children. *Br. Med. J.* 2: 149-152.
- Melia, R. J. W.; Florey, C. du V.; Chinn, S. (1979) The relation between respiratory illness in primary schoolchildren and the use of gas for cooking: I--results from a national survey. *Int. J. Epidemiol.* 8: 333-338.
- Melia, R. J. W.; Florey, C. du V.; Chinn, S.; Goldstein, B. D.; Brooks, A. G. F.; John, H. H.; Clark, D.; Craighead, I. B.; Webster, X. (1980) The relation between indoor air pollution from nitrogen dioxide and respiratory illness in primary schoolchildren. *Clin. Respir. Physiol.* 16: 7P-8P.
- Melia, R. J. W.; Florey, C. du V.; Morris, R. W.; Goldstein, B. D.; John, H. H.; Clark, D.; Craighead, I. B.; Mackinlay, J. C. (1982) Childhood respiratory illness and the home environment: II. association between respiratory illness and nitrogen dioxide, temperature and relative humidity. *Int. J. Epidemiol.* 11: 164-169.
- Melia, J.; Florey, C.; Sittampalam, Y.; Watkins, C. (1983) The relation between respiratory illness in infants and gas cooking in the UK: a preliminary report. In: *Air quality 6th world congress: [proceedings of the International Union of Air Pollution Prevention Associations]*; May; Paris, France. SEPIC (APPA); pp. 263-269.

- Menzel, D. B. (1976) The role of free radicals in the toxicity of air pollutants (nitrogen oxides and ozone). In: Pryor, W. A., ed. *Free radicals in biology: v.II*. New York, NY: Academic Press, Inc.; pp. 181-202.
- Miller, F. J.; Graham, J. A.; Raub, J. A.; Illing, J. W.; Menache, M. G.; House, D. E.; Gardner, D. E. (1987) Evaluating the toxicity of urban patterns of oxidant gases. II. Effects in mice from chronic exposure to nitrogen dioxide. *J. Toxicol. Environ. Health* 21: 99-112.
- Mochitate, K.; Takahashi, Y.; Ohsumi, T.; Miura, T. (1986) Activation and increment of alveolar macrophages induced by nitrogen dioxide. *J. Toxicol. Environ. Health* 17: 229-239.
- Mohsenin, V. (1987a) Airway responses to nitrogen dioxide in asthmatic subjects. *J. Toxicol. Environ. Health* 22: 371-380.
- Mohsenin, V. (1987b) Effect of vitamin C on NO₂-induced airway hyperresponsiveness in normal subjects: a randomized double-blind experiment. *Am. Rev. Respir. Dis.* 136: 1408-1411.
- Mohsenin, V. (1988) Airway responses to 2.0 ppm nitrogen dioxide in normal subjects. *Arch. Environ. Health* 43: 242-246.
- Moore, D. R. J.; Keddy, P. A.; Gaudet, C. L.; Wisheu, I. C. (1989) Conservation of wetlands: do infertile wetlands deserve a higher priority? *Biol. Conserv.* 47: 203-217.
- Moorhead, D. L.; Reynolds, J. F.; Whitford, W. G. (1986) A conceptual model for primary productivity, decomposition and nitrogen cycling in the Chihuahuan creosotebush desert. *Tree Physiol.* 2: 215-222.
- Morris, D. P.; Lewis, W. M., Jr. (1988) Phytoplankton nutrient limitation in Colorado mountain lakes. *Freshwater Biol.* 20: 315-327.
- Morrow, P. E.; Utell, M. J. (1989) Responses of susceptible subpopulations to nitrogen dioxide. Cambridge, MA: Health Effects Institute; research report no. 23.
- Munger, J. W.; Eisenreich, S. J. (1983) Continental-scale variations in precipitation chemistry. *Environ. Sci. Technol.* 17: 32A-42A.
- National Institutes of Health. (1985) The definition of emphysema: report of a National Heart, Lung, and Blood Institute, Division of Lung Diseases workshop. *Am. Rev. Respir. Dis.* 132: 182-185.
- National Institutes of Health. (1991) Guidelines for the diagnosis and management of asthma. Bethesda, MD: U.S. Department of Health and Human Services, National Heart, Lung, and Blood Institute, National Asthma Education Program; publication no. 91-3042.
- Neas, L. M.; Ware, J. H.; Dockery, D. W.; Spengler, J. D.; Ferris, B. G., Jr.; Speizer, F. E. (1990) The association of indoor nitrogen dioxide levels with respiratory symptoms and pulmonary function in children. In: *Indoor air '90: proceedings of the 5th international conference on indoor air quality and climate, volume 1, human health, comfort and performance; July-August; Toronto, ON, Canada. Ottawa, ON, Canada: International Conference on Indoor Air Quality and Climate, Inc.; pp. 381-386.*
- Neas, L. M.; Dockery, D. W.; Ware, J. H.; Spengler, J. D.; Speizer, F. E.; Ferris, B. G., Jr. (1991) Association of indoor nitrogen dioxide with respiratory symptoms and pulmonary function in children. *Am. J. Epidemiol.* 134: 204-219.

- Nilsson, J.; Grennfelt, P., eds. (1988) Critical loads for sulphur and nitrogen: report from a workshop; March; Skokloster, Sweden. Copenhagen, Denmark: Nordic Council of Ministers.
- Nixon, S. W.; Pilson, M. E. Q. (1983) Nitrogen in estuarine and coastal marine ecosystems. In: Carpenter, E. J.; Capone, D. G., eds. Nitrogen in the marine environment. New York, NY: Academic Press; pp. 565-648.
- Ogston, S. A.; Florey, C. du V.; Walker, C. H. M. (1985) The Tayside infant morbidity and mortality study: effect on health of using gas for cooking. *Br. Med. J.* 290: 957-960.
- Orehek, J.; Grimaldi, F.; Muls, E.; Durand, J. P.; Viala, A.; Charpin, J. (1981) Reponse bronchique aux allergenes apres exposition controlee au dioxyde d'azote [Bronchial response to allergens after controlled NO₂ exposure]. *Bull. Eur. Physiopathol. Respir.* 17: 911-915.
- Overton, J. H., Jr. (1984) Physicochemical processes and the formulation of dosimetry models. In: Miller, F. J.; Menzel, D. B., eds. Fundamentals of extrapolation modeling of inhaled toxicants: ozone and nitrogen dioxide. Washington, DC: Hemisphere Publishing Corporation; pp. 93-114.
- Parker, R. F.; Davis, J. K.; Cassell, G. H.; White, H.; Dziedzic, D.; Blalock, D. K.; Thorp, R. B.; Simecka, J. W. (1989) Short-term exposure to nitrogen dioxide enhances susceptibility to murine respiratory mycoplasmosis and decreases intrapulmonary killing of *Mycoplasma pulmonis*. *Am. Rev. Respir. Dis.* 140: 502-512.
- Pastor, J.; Bockheim, J. G. (1984) Distribution and cycling of nutrients in an aspen-mixed-hardwood-Spodosol ecosystem in northern Wisconsin. *Ecology* 65: 339-353.
- Peake, E.; Davidson, C. I. (1990) Wet and dry deposition of air pollutants in Alberta. In: Legge, A. H.; Krupa, S. V., eds. Acidic deposition: sulphur and nitrogen oxides. Boca Raton, FL: Lewis Publishers, Inc.; pp. 381-412.
- Pearlman, M. E.; Finklea, J. F.; Creason, J. P.; Shy, C. M.; Young, M. M.; Horton, R. J. M. (1971) Nitrogen dioxide and lower respiratory illness. *Pediatrics* 47: 391-398.
- Port, C. D.; Ketels, K. V.; Coffin, D. L.; Kane, P. (1977) A comparative study of experimental and spontaneous emphysema. *J. Toxicol. Environ. Health* 2: 589-604.
- Postlethwait, E. M.; Mustafa, M. G. (1981) Fate of inhaled nitrogen dioxide in isolated perfused rat lung. *J. Toxicol. Environ. Health* 7: 861-872.
- Postlethwait, E. M.; Mustafa, M. G. (1989) Effect of altered dose rate on NO₂ uptake and transformation in isolated lungs. *J. Toxicol. Environ. Health* 26: 497-507.
- Rebmann, H.; Huenges, R.; Wichmann, H. E.; Malin, E. M.; Huebner, H. R.; Roell, A.; Hoerz, G.; Hub, R.; Walter, C.; Doeller, G.; Gerth, H.-J. (1991) Krupp und Luftschadstoffe: Ergebnisse einer zweijahrigten prospektiven Laengsschnittstudie [Croup and air-pollution: results of a two-year prospective longitudinal study]. *Zentralbl. Hyg. Umweltmed.* 192: 104-115.
- Redfield, A. C. (1934) On the proportions of organic derivatives in sea water and their relation to the composition of plankton. In: James Johnstone memorial volume. Liverpool, United Kingdom: Liverpool University Press; pp. 176-192.

- Richter, D. D.; Johnson, D. W.; Todd, D. E. (1983) Atmospheric sulfur deposition, neutralization, and ion leaching in two deciduous forest ecosystems. *J. Environ. Qual.* 12: 263-270.
- Richters, A.; Damji, K. S. (1988) Changes in T-lymphocyte subpopulations and natural killer cells following exposure to ambient levels of nitrogen dioxide. *J. Toxicol. Environ. Health* 25: 247-256.
- Richters, A.; Damji, K. S. (1990) The relationship between inhalation of nitrogen dioxide, the immune system, and progression of a spontaneously occurring lymphoma in AKR mice. *J. Environ. Pathol. Toxicol. Oncol.* 10: 225-230.
- Riggan, P. J.; Lockwood, R. N.; Lopez, E. N. (1985) Deposition and processing of airborne nitrogen pollutants in Mediterranean-type ecosystems of southern California. *Environ. Sci. Technol.* 19: 781-789.
- Ro, C. U.; Tang, A. J. S.; Chan, W. H.; Kirk, R. W.; Reid, N. W.; Lusic, M. A. (1988) Wet and dry deposition of sulfur and nitrogen compounds in Ontario. *Atmos. Environ.* 22: 2763-2772.
- Robison, T. W.; Duncan, D. P.; Forman, H. J. (1990) Chemoattractant and leukotriene B₄ production from rat alveolar macrophages exposed to nitrogen dioxide. *Am. J. Respir. Cell Mol. Biol.* 3: 21-26.
- Roger, L. J.; Horstman, D. H.; McDonnell, W. F.; Kehrl, H.; Seal, E.; Chapman, R. S.; Massaro, E. J. (1985) Pulmonary effects in asthmatics exposed to 0.3 ppm NO₂ during repeated exercise. *Toxicologist* 5: 70.
- Roger, L. J.; Horstman, D. H.; McDonnell, W.; Kehrl, H.; Ives, P. J.; Seal, E.; Chapman, R.; Massaro, E. (1990) Pulmonary function, airway responsiveness, and respiratory symptoms in asthmatics following exercise in NO₂. *Toxicol. Ind. Health* 6: 155-171.
- Rombout, P. J. A.; Dormans, J. A. M. A.; Marra, M.; Van Esch, G. J. (1986) Influence of exposure regimen on nitrogen dioxide-induced morphological changes in the rat lung. *Environ. Res.* 41: 466-480.
- Rosen, K. (1982) Supply, loss and distribution of nutrients in three coniferous forest watersheds in central Sweden. Umea, Sweden: Swedish University of Agricultural Sciences, Department of Forest Soils; report no. 41.
- Royal Society. (1983) The nitrogen cycle of the United Kingdom. London, United Kingdom: The Royal Society; pp. 45-46.
- Sagai, M.; Ichinose, T.; Kubota, K. (1984) Studies on the biochemical effects of nitrogen dioxide. IV. Relation between the change of lipid peroxidation and the antioxidative protective system in rat lungs upon life span exposure to low levels of NO₂. *Toxicol. Appl. Pharmacol.* 73: 444-456.
- Samet, J. M.; Tager, I. B.; Speizer, F. E. (1983) The relationship between respiratory illness in childhood and chronic air-flow obstruction in adulthood. *Am. Rev. Respir. Dis.* 127: 508-523.
- Samet, J. M.; Utell, M. J. (1990) The risk of nitrogen dioxide: what have we learned from epidemiological and clinical studies? *Toxicol. Ind. Health* 6: 247-262.
- Samet, J. M.; Lambert, W. E.; Skipper, B. J.; Cushing, A. H.; Hunt, W. C.; Young, S. A.; McLaren, L. C.; Schwab, M.; Spengler, J. D. (1993) Health outcomes. In: Nitrogen dioxide and respiratory illness in children, part I. Cambridge, MA: Health Effects Institute; research report no. 58.
- Saul, R. L.; Archer, M. C. (1983) Nitrate formation in rats exposed to nitrogen dioxide. *Toxicol. Appl. Pharmacol.* 67: 284-291.

- Schenker, M. B.; Samet, J. M.; Speizer, F. E. (1983) Risk factors for childhood respiratory disease: the effect of host factors and home environmental exposures. *Am. Rev. Respir. Dis.* 128: 1,038-1,043.
- Schlesinger, R. B. (1987a) Effects of intermittent inhalation exposures to mixed atmospheres of NO_2 and H_2SO_4 on rabbit alveolar macrophages. *J. Toxicol. Environ. Health* 22: 301-312.
- Schlesinger, R. B. (1987b) Intermittent inhalation of nitrogen dioxide: effects on rabbit alveolar macrophages. *J. Toxicol. Environ. Health* 21: 127-139.
- Schlesinger, R. B.; Driscoll, K. E.; Vollmuth, T. A. (1987) Effect of repeated exposures to nitrogen dioxide and sulfuric acid mist alone or in combination on mucociliary clearance from the lungs of rabbits. *Environ. Res.* 44: 294-301.
- Schofield, C. L.; Galloway, J. N.; Hendry, G. R. (1985) Surface water chemistry in the ILWAS basins. *Water Air Soil Pollut.* 26: 403-423.
- Schuurkes, J. A. A. R. (1986) Atmospheric ammonium sulphate deposition and its role in the acidification and nitrogen enrichment of poorly buffered aquatic systems. *Experientia* 42: 351-357.
- Schuurkes, J. A. A. R. (1987) Acidification of surface waters by atmospheric deposition [Ph.D. thesis]. Nijmegen, The Netherlands: Catholic University.
- Schuurkes, J. A. A. R.; Elbers, M. A.; Gudden, J. J. F.; Roelofs, J. G. M. (1987) Effects of simulated ammonium sulphate and sulphuric acid rain on acidification, water quality and flora of small-scale soft water systems. *Aquat. Bot.* 28: 199-226.
- Schwartz, J.; Spix, C.; Wichmann, H. E.; Malin, E. (1991) Air pollution and acute respiratory illness in five German communities. *Environ. Res.* 56: 1-14.
- Selgrade, M. K.; Mole, M. L.; Miller, F. J.; Hatch, G. E.; Gardner, D. E.; Ju, P. C. (1981) Effect of NO_2 inhalation and vitamin C deficiency on protein and lipid accumulation in the lung. *Environ. Res.* 26: 422-437.
- Sevanian, A.; Hacker, A. D.; Elsayed, N. (1982) Influence of vitamin E and nitrogen dioxide on lipid peroxidation in rat lung and liver microsomes. *Lipids* 17: 269-277.
- Shy, C. M.; Creason, J. P.; Pearlman, M. E.; McClain, K. E.; Benson, F. B.; Young, M. M. (1970a) The Chattanooga school children study: effects of community exposure to nitrogen dioxide. 1. Methods, description of pollutant exposure, and results of ventilatory function testing. *J. Air Pollut. Control Assoc.* 20: 539-545.
- Shy, C. M.; Creason, J. P.; Pearlman, M. E.; McClain, K. E.; Benson, F. B.; Young, M. M. (1970b) The Chattanooga school children study: effects of community exposure to nitrogen dioxide. II.
- Shy, C. M.; Niemeier, L.; Truppi, L.; English, T. (1973) Re-evaluation of the Chattanooga school children studies and the health criteria for NO_2 exposure [revised draft]. Research Triangle Park, NC: U.S. Environmental Protection Agency, Human Studies Laboratory.
- Shy, C. M.; Kleinbaum, D. G.; Morgenstern, H. (1978) The effect of misclassification of exposure status in epidemiological studies of air pollution health effects. *Bull. N. Y. Acad. Med.* 54: 1,155-1,165.

- Silsbee, D. G.; Larson, G. L. (1982) Water quality of streams in the Great Smoky Mountains National Park. *Hydrobiologia* 89: 97-115.
- Simpson, J. C.; Olsen, A. R. (1990) Wet deposition temporal and spatial patterns in North America, 1987. Research Triangle Park, NC: U.S. Environmental Protection Agency, Atmospheric Research and Exposure Assessment Laboratory; EPA report no. EPA-600/4-90-019. Available from: NTIS, Springfield, VA; PB90-251836.
- Skeffington, R. A.; Wilson, E. J. (1988) Excess nitrogen deposition: issues for consideration. *Environ. Pollut.* 54: 159-184.
- Smith, R. A.; Alexander, R. B.; Wolman, M. G. (1987) Water-quality trends in the nation's rivers. *Science* (Washington, DC) 235: 1607-1615.
- Smith, R. L. (1980) *Ecology and field biology*. 3rd ed. New York, NY: Harper & Row, Publishers; pp. 11-199.
- Smith, S. V. (1984) Phosphorus versus nitrogen limitation in the marine environment. *Limnol. Oceanogr.* 29: 1149-1160.
- Smith, W.; Anderson, T.; Anderson, H. A.; Remington, P. L. (1992) Nitrogen dioxide and carbon monoxide intoxication in an indoor ice arena—Wisconsin, 1992. *Morb. Mortal. Wkly. Rep.* 41: 383-385.
- Smullen, J. T.; Taft, J. L.; Macknis, J. (1982) Nutrient and sediment loads to the tidal Chesapeake Bay system. In: Chesapeake Bay Program technical studies: a synthesis. Annapolis, MD: U.S. Environmental Protection Agency; pp. 150-251. Available from: NTIS, Springfield, VA; PB84-111202.
- Sollins, P.; Grier, C. C.; McCorison, F. M.; Cromack, K., Jr.; Fogel, R.; Fredriksen, R. L. (1980) The internal element cycles of an old-growth Douglas-fir ecosystem in western Oregon. *Ecol. Monogr.* 50:261-285.
- Speizer, F. E.; Ferris, B., Jr.; Bishop, Y. M. M.; Spengler, J. (1980) Respiratory disease rates and pulmonary function in children associated with NO₂ exposure. *Am. Rev. Respir. Dis.* 121: 3-10.
- Steele, G. D.; Winchester, D. P.; Menck, H. R. (1991) Patterns of care: United States (provisional, 1985, 1988). Commission on Cancer—American College of Surgeons and the American Cancer Society; National Cancer Data Base.
- Sullivan, T. J.; Christophersen, N.; Muniz, I. P.; Seip, H. M.; Sullivan, P. D. (1986) Aqueous aluminum chemistry response to episodic increases in discharge. *Nature* (London) 323: 324-327.
- Summers, P. W.; Bowersox, V. C.; Stensland, G. J. (1986) The geographical distribution and temporal variations of acidic deposition in eastern North America. *Water Air Soil Pollut.* 31: 523-535.
- Suzuki, T.; Ikeda, S.; Kanoh, T.; Mizoguchi, I. (1986) Decreased phagocytosis and superoxide anion production in alveolar macrophages of rats exposed to nitrogen dioxide. *Arch. Environ. Contam. Toxicol.* 15: 733-739.
- Swank, W. T.; Waide, J. B. (1988) Characterization of baseline precipitation and stream chemistry and nutrient budgets for control watersheds. In: Swank, W. T.; Crossley, D. A., Jr., eds. *Forest hydrology and ecology at Coweeta*. New York, NY: Springer-Verlag; pp. 57-79. (Billings, W. D.; Golley, F.; Lange, O. L.; Olson, J. S.; Remmert, H., eds. *Ecological studies: analysis and synthesis*, v. 66).

- Tallis, J. H. (1964) Studies on southern Pennine peats: III. the behaviour of *Sphagnum*. *J. Ecol.* 52: 345-353.
- Tamm, C. O.; Popovic, B. (1974) Intensive fertilization with nitrogen as a stressing factor in a spruce ecosystem. I. Soil effects. Stockholm, Sweden: Royal College of Forestry. (Studia forestalia suecica nr. 121).
- Tombach, I. (1982) Measurement of local climatological and air pollution factors affecting stone decay. In: Conservation of historic stone buildings and monuments: report of the Committee on Conservation of Historic Stone Buildings and Monuments. Washington, D.C.: National Academy Press; pp. 197-210.
- Tschaplinski, T. J.; Johnson, D. W.; Norby, R. J.; Todd, D. E. (1991) Biomass and soil nitrogen relationships of a one-year-old sycamore plantation. *Soil Sci. Soc. Am. J.* 55: 841-847.
- Turner, R. S.; Cook, R. B.; Van Miegroet, H.; Johnson, D. W.; Elwood, J. W.; Bricker, O. P.; Lindberg, S. E.; Hornberger, G. M. (1990) Watershed and lake processes affecting surface water acid-base chemistry. Washington, DC: National Acid Precipitation Assessment Program. (Acidic deposition: state of science and technology report 12).
- Turner, J.; Singer, M. J. (1976) Nutrient distribution and cycling in a sub-alpine coniferous forest ecosystem. *J. Appl. Ecol.* 13: 295-301.
- Tyler, M. (1988) Contribution of atmospheric nitrate deposition to nitrate loading in the Chesapeake Bay. Annapolis, MD: Department of Natural Resources, Chesapeake Bay Research & Monitoring Division; report no. AD-88-7.
- Ulrich, B. (1980) Production and consumption of hydrogen ions in the ecosphere. In: Hutchinson, T. C.; Havas, M., eds. Effects of acid precipitation on terrestrial ecosystems: proceedings of the NATO conference on effects of acid precipitation on vegetation and soils; May 1978; Toronto, ON, Canada. New York, NY: Plenum Press; pp. 255-282.
- U.S. Bureau of the Census. (1991) Statistical abstract of the United States: 1991. 111th ed. Washington, DC: U.S. Bureau of the Census; pp. 111 and 123.
- U.S. Department of Health and Human Services. (1990) Vital and health statistics: current estimates from the National Health Interview Survey, 1989. Hyattsville, MD: Public Health Service, National Center for Health Statistics; DHHS publication no. (PHS) 90-1504. (Series 10: data from the National Health Survey no. 176).
- U.S. Environmental Protection Agency. (1971) Air quality criteria for nitrogen oxides. Washington, DC: U.S. Environmental Protection Agency, Air Pollution Control Office; EPA report no. AP-84. Available from: NTIS, Springfield, VA; PB-197333/BE.
- U.S. Environmental Protection Agency. (1982) Air quality criteria for oxides of nitrogen. Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; EPA report no. EPA-600/8-82-026. Available from: NTIS, Springfield, VA; PB83-131011.
- U.S. Environmental Protection Agency. (1985) Ambient water quality criteria for ammonia - 1984. Washington, DC: Criteria and Standards Division; EPA report no. EPA-440/5-85-001. Available from: NTIS, Springfield, VA; PB85-227114.
- U.S. Environmental Protection Agency. (1982b) Review of the National Ambient Air Quality Standards for Nitrogen Oxides: Assessment of Scientific and Technical Information. OAQPS Staff Paper. Office of Air Quality

- Planning and Standards; EPA report no. EPA-450/5-82-002. Available from: NTIS, Springfield, VA.
- U.S. Environmental Protection Agency. (1993a) Air quality criteria for oxides of nitrogen. Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; EPA report no. EPA-600/8-91/049F. Available from: NTIS, Springfield, VA.
- U.S. Environmental Protection Agency. (1993b) National air quality and emissions trends report, 1992. Research Triangle Park, NC: Office of Air Quality Planning and Standards; EPA report no. EPA-454/R-93-031. Available from: NTIS, Springfield, VA.
- Van Breemen, N.; Van Dijk, H. F. G. (1988) Ecosystem effects of atmospheric deposition of nitrogen in the Netherlands. In: Dempster, J. P.; Manning, W. J.; Skeffington, R. A., eds. Excess nitrogen deposition: [papers from the workshop]; September 1987; Leatherhead, Surrey, United Kingdom. *Environ. Pollut.* 54: 249-274.
- Van Breemen, N.; Mulder, J.; Van Grinsven, J. J. M. (1987) Impacts of acid atmospheric deposition on woodland soils in the Netherlands: II. nitrogen transformations. *Soil Sci. Soc. Am. J.* 51: 1634-1640.
- Van Miegroet, H.; Cole, D. W. (1984) The impact of nitrification on soil acidification and cation leaching in red alder ecosystem. *J. Environ. Qual.* 13: 586-590.
- Van Miegroet, H.; Cole, D. W.; Foster, N. W. (1992) Nitrogen distribution and cycling. In: Johnson, D. W.; Linberg, S. E., eds. Atmospheric deposition and forest nutrient cycling: a synthesis of the integrated forest study. New York, NY: Springer-Verlag; pp. 178-196. (Billings, W. D.; Golley, F.; Lange, O. L.; Olson, J. S.; Remmert, H., eds. Ecological studies: analysis and synthesis: v. 91).
- Vedal, S.; Schenker, M. B.; Munoz, A.; Samet, J. M.; Batterman, S.; Speizer, F. E. (1987) Daily air pollution effects on children's respiratory symptoms and peak expiratory flow. *Am. J. Public Health* 77: 694-698.
- Vermeer, J. G. (1986) The effect of nutrients on shoot biomass and species composition of wetland and hayfield communities. *Acta Oecol. Oecol. Plant.* 7: 31-41.
- Von Nieding, G.; Wagner, H. M. (1979) Effects of NO₂ on chronic bronchitics. *Environ. Health Perspect.* 29: 137-142.
- Von Nieding, G.; Wagner, H. M.; Krekeler, H.; Smidt, U.; Muysers, K. (1970) Absorption of NO₂ in low concentrations in the respiratory tract and its acute effects on lung function and circulation. Presented at: the second international clean air congress; December; Washington, DC; paper no. MB-15G.
- Von Nieding, G.; Wagner, M.; Krekeler, H.; Smidt, U.; Muysers, K. (1971) Grenzwertbestimmung der akuten NO₂-Wirkung auf den respiratorischen Gasaustausch und die Atemwegswiderstaende des chronisch lungenkranken Menschen [Minimum concentrations of NO₂ causing acute effects on the respiratory gas exchange and airway resistance in patients with chronic bronchitis]. *Int. Arch. Arbeitsmed.* 27: 338-348.
- Von Nieding, G.; Krekeler, H.; Fuchs, R.; Wagner, M.; Koppenhagen, K. (1973) Studies of the acute effects of NO₂ on lung function: influence on diffusion, perfusion and ventilation in the lungs. *Int. Arch. Arbeitsmed.* 31: 61-72.

- Von Nieding, G.; Wagner, M.; Loellgen, H.; Krekeler, H. (1977) Zur akuten Wirkung von Ozon auf die Lungenfunktion des Menschen [The acute effect of ozone on the pulmonary function of man]. VDI Ber. (270): 123-129.
- Von Nieding, G.; Wagner, H. M.; Krekeler, H.; Loellgen, H.; Fries, W.; Beuthan, A. (1979) Controlled studies of human exposure to single and combined action of NO₂, O₃, and SO₂. Int. Arch. Occup. Environ. Health 43: 195-210.
- Von Nieding, G.; Wagner, H. M.; Casper, H.; Beuthan, A.; Smidt, U. (1980) Effect of experimental and occupational exposure to NO₂ in sensitive and normal subjects. In: Lee, S. D., ed. Nitrogen oxides and their effects on health. Ann Arbor, MI: Ann Arbor Science Publishers, Inc.; pp. 315-331.
- Wagner, H. M. (1970) Absorption von NO und NO₂ in MIK- und MAK-Konzentrationen bei der Inhalation [Absorption of NO and NO₂ in mik- and mak-concentrations during inhalation]. Staub Reinhalt. Luft 30: 380-381.
- Ware, J. H.; Dockery, D. W.; Spiro, A., III; Speizer, F. E.; Ferris, B. G., Jr. (1984) Passive smoking, gas cooking, and respiratory health of children living in six cities. Am. Rev. Respir. Dis. 129: 366-374.
- Wigington, P. J., Jr.; Davies, T. D.; Tranter, M.; Eshleman, K. (1990) Episodic acidification of surface waters due to acidic deposition. Washington, DC: National Acid Precipitation Assessment Program. (Acidic deposition: state of science and technology report 12).
- Williams, M. W.; Melack, J. M. (1991) Precipitation chemistry in and ionic loading to an alpine basin, Sierra Nevada. Water Resour. Res. 27: 1563-1574.
- Wisheu, I. C.; Keddy, P. A. (1989) The conservation and management of a threatened coastal plain plant community in eastern North America (Nova Scotia, Canada). Biol. Conserv. 48: 229-238.
- Woodin, S. J.; Lee, J. A. (1987) The fate of some components of acidic deposition in ombrotrophic mires. Environ. Pollut. 45: 61-72.
- World Health Organization. (1987) The effects of nitrogen on vegetation. In: Air quality guidelines for Europe. Copenhagen, Denmark: Regional Office for Europe; pp. 373-385. (WHO regional publications, European series no. 23).
- Yamamoto, I.; Takahashi, M. (1984) Ultrastructural observations of rat lung exposed to nitrogen dioxide for 7 months. Kitasato Arch. Exp. Med. 57: 57-65.
- Zemba, S. G.; Golomb, D.; Fay, J. A. (1988) Wet sulfate and nitrate deposition patterns in eastern North America. Atmos. Environ. 22: 2751-2761.

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16. ABSTRACT This paper evaluates and interprets the updated scientific and technical information that EPA staff believes is most relevant to the review of the primary (health) and secondary (environment; welfare) national ambient air quality standards (NAAQS) for nitrogen dioxide. This assessment is intended to bridge the gap between the scientific review in the 1993 revised criteria document and the judgements required of the Administrator in setting ambient air quality standards for nitrogen dioxide. The major conclusions of the staff paper include the following: (1) the Administrator should consider setting the level of the annual primary standard within the range of 0.05 to 0.08 ppm NO ₂ to provide adequate protection against the health effects associated with long-term NO ₂ exposure; (2) air quality analyses indicate that a standard selected from the lower portion of the range suggested would effectively limit the frequency and magnitude of 1-hour peak NO ₂ concentrations of potential health concern; (3) the annual secondary standard for NO ₂ should be set identical to the primary standard in order to provide adequate protection against the direct effects of NO ₂ on the environment; (4) additional research is needed to determine the quantitative relationships between nitrogen deposition and changes in forest nitrogen cycling, episodic and chronic acidification of surface waters, and estuarine eutrophication.		
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