

Ozone
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EXECUTIVE OFFICE OF THE PRESIDENT
OFFICE OF MANAGEMENT AND BUDGET
WASHINGTON, D.C. 20503

MAR 30 1994

MEMORANDUM FOR INTERAGENCY GROUP ON OZONE NAAQS REVIEW
(DISTRIBUTION LIST)

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FROM: Art Fraas *AF* Chris Wolz *CW*
Office of Information and Regulatory Affairs,
Office of Management and Budget

SUBJECT: Meeting of an Interagency Group on the
Environmental Protection Agency's Review of the
"National Ambient Air Quality Standard" for Ozone,
Wednesday, April 6, 1994

The Environmental Protection Agency (EPA) has asked the Office of Management and Budget (OMB) to assist in coordinating an interagency group to consult with EPA in its review of the National Ambient Air Quality Standard (NAAQS) for ozone. The interagency group would, as a first step, discuss with EPA methodological issues underlying the NAAQS review, such as the human exposure analysis and the health risk assessment that EPA is conducting for its "Staff Paper." Additional issues that might be covered later in this process could include the assessment of welfare effects and the form of the standard.

We have scheduled a meeting for the interagency group with EPA for **Wednesday, April 6, 1994**, from 9:30 a.m. to 11:30 a.m., to discuss the NAAQS review in general, and specifically the human exposure and health risk analysis. We hope you can attend this meeting.

EPA should already have sent to you several documents for discussion at this meeting, including: "Estimation of Ozone Exposures Experienced by Urban Residents Using a Probabilistic Version of NEM and 1990 Population Data," September 1993; "A Probabilistic Assessment of Health Risks Associated with Short-term and Long-term Exposure to Tropospheric ozone," February, 1994; and "Ozone NAAQS Development Project Plan," December 16, 1993.

The meeting will be held in Room 472 of the Old Executive Office Building, 17th and Pennsylvania Avenue N.W., Washington, D.C. For security clearance, please call our office on (202) 395-3084 by Monday, April 4, and leave your name and date-of-birth with our support staff.

Attached is a brief background note on the NAAQS and the review process.

Attachment

Background Information on the National Ambient Air Quality Standard for Ozone

The Clean Air Act (CAA) requires that the Environmental Protection Agency (EPA) establish standards for levels of air pollutants in the ambient air to protect the public health and welfare. (Sections 108 and 109 of the Clean Air Act) Ozone is a highly reactive gas which, in the troposphere, can produce a wide variety of harmful effects on human health and vegetation, and materials, and EPA has maintained a NAAQS for ozone (or its precursors) since 1971. The ozone NAAQS is the driving force behind a broad array of emissions control regulations both at the State and Federal level. Despite two decades of pollution control regulations and a trend towards lower nationwide ozone concentrations over the past decade, efforts to attain the NAAQS have not succeeded. EPA estimated that in 1988, about 120 million people lived in urban areas in which the current ozone NAAQS was exceeded.

Setting of the NAAQS and Statutory Constraints: The CAA requires EPA to list pollutants that cause or contribute to air pollution and "which may reasonably be anticipated to endanger public health and welfare[.]" (Section 108) Once a pollutant has been listed, EPA must review the relevant scientific literature on the effects of a pollutants and develop both primary and secondary ambient air quality standards. (Section 109) Currently, EPA has NAAQS for six pollutants: carbon monoxide (CO), sulfur dioxide, (SO₂), particulate matter (PM-10), nitrogen dioxide (NO₂), lead (Pb), and ozone (O₃).

The primary ambient air quality standard must be set at levels which with an adequate margin of safety, "are requisite to protect the public health" and the Courts have held that the CAA does not allow the consideration of economic or technical factors. EPA must also issue secondary standards, to "protect the public welfare from any known or anticipated adverse effects associated with" the pollutant. In setting secondary standards, EPA must consider a range of factors, e.g., crop damage, damage to buildings and materials and visibility impairment. In 1979, EPA revised the existing NAAQS and established the current primary and secondary NAAQS for ozone. The current ozone secondary NAAQS is set at the level of the primary NAAQS, which is 0.12 ppm averaged over one hour, not to be exceeded more than one hour per year.

Health and Welfare Effects of Ozone: At elevated concentrations, ozone can adversely affect human health, impairing normal functioning of the lungs and reducing the ability to perform physical exercise. These effects may be aggravated in sensitive individuals and those with preexisting lung disease. Symptoms associated with exposure to ozone at elevated levels include cough, chest pain, and throat irritation. There is recent data

showing some adverse health effects from exposure to ozone for six to eight hour periods at levels below the current NAAQS of 0.12 ppm; in its current review of the NAAQS, EPA will assess this information and the need for a standard (e.g., an 8-hour standard) to replace or supplement the current 1-hour standard. The chronic health effects of repeated exposure to elevated levels of ozone have been raised as a health concern, although in its 1993 review of the NAAQS, EPA concluded that the preliminary information available on chronic effects (often based on animal studies) was not sufficient to support the establishment of a chronic standard for ozone.

With regard to welfare effects, estimates based on experimental studies of major cash crops in the U.S. indicate that elevated ozone levels are responsible for several billion dollars in agricultural crop yield losses each year. Exposure to elevated levels of ozone also can cause foliar damage in many crops, as well as damage to forests and ecosystems. In its current NAAQS review, EPA will consider options to protect the public welfare, such as a standard with an averaging period that extends over a growing season.

The Ozone NAAQS and Air Pollution Control Measures: The NAAQS are the driving force behind both a number of Federal regulations to limit the emissions of air pollutants and "State Implementation Plans" (SIPs) that establish control measures for sources within each nonattainment area. SIPs must include both mandatory control measures listed in the Clean Air Act and enough additional controls as are necessary--as shown by computer modeling done to the satisfaction of EPA--to achieve attainment of the NAAQS on schedule. Billions of dollars per year are spent on air pollutant emissions control measures in the ninety-seven areas that are designated as non-attainment for the ozone NAAQS. The mandatory control measures under the Clean Air Act vary according to the severity of the ozone level in the nonattainment area, and may include:

- Control of emissions of volatile organic compounds and nitrogen oxides from **major stationary sources** under New Source Review, and Reasonable Available Control Technology requirements;
- **Transportation planning and controls**, including the Employer Trip Reduction Program, and HOV highway lanes;
- And **mobile source** (i.e., cars and trucks) emissions control requirements, including tailpipe standards, clean fuels requirements, and inspection and maintenance programs.

EPA's Review Schedule: EPA's Administrator Carol Browner has committed EPA to conduct an accelerated review of the ozone NAAQS. The CAA requires EPA to review the NAAQS every five years but after a revision in 1979, EPA completed its first review of the 1979 standard under a Court deadline on March 1, 1993. In EPA's 1993 action, EPA concluded that "revisions of the primary and secondary NAAQS are not appropriate at this time" due to a

large body of recent scientific literature that EPA had not been able to rigorously review within the time provided under the Court order. (Federal Register, March 9, 1993) EPA did commit, however, to proceed with the next review "as rapidly as possible to assess the more recent health and welfare effects information" and to publish a proposed decision in 1996, followed by promulgation of a final decision by 1997. EPA's schedule is:

MAJOR MILESTONES

TENTATIVE DATES*

CASAC subcommittee meeting on exposure assessment methods:
* December 1993

CASAC and public comment period for Criteria Document (CD)
(90 days) * February-May 1994

CASAC subcommittee meeting on risk assessment methods
* March 1994

CASAC meeting on CD * July 1994

Comment period on Staff Paper (SP)
* September-October 1994

CASAC meeting on SP * October 1994

Public comment period on revised CD (90 days)
* Early 1995

CASAC meeting on revised CD and SP
* Mid-1995

Agency development of regulatory decision/proposal; package draft
* Mid-1995 to late 1995

OMB review of proposal package
* Early 1996

Publication of proposal in the Federal Register
* Mid-1996

Public comment on proposal (90 days)
* Mid-1996

CASAC meeting to discuss review proposal
* Late 1996

Regulatory decision and final package draft completed
* Early 1997

OMB review of promulgation package
* Early 1997 to mid-1997

Publication of promulgation notice in the Federal Register
* Mid-1997

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 50

[FRL-4832-8]

Review of National Ambient Air Quality Standards for Ozone

AGENCY: U.S. Environmental Protection Agency (USEPA).

ACTION: Notice of review.

SUMMARY: This notice announces the EPA's plans to review and revise the Air Quality Criteria for Ozone and Other Photochemical Oxidants (Criteria Document) as rapidly as possible and to complete review of the national ambient air quality standards (NAAQS) for ozone (O_3) as soon as possible thereafter.

The Clean Air Act (Act) requires periodic review and, if appropriate, revision of the NAAQS and of the air quality criteria on which they are based. The EPA completed its last formal review of the air quality criteria for O_3 in 1989. Based on that review, the EPA announced a final decision on March 9, 1993 not to revise the existing O_3 NAAQS.

Since early 1989, however, a substantial number of new studies on the health and environmental effects of O_3 have appeared in the peer-reviewed literature. The EPA is moving as rapidly as possible to review them, consistent with assuring a sound, scientifically-supportable decision.

The review process includes: (1) Reviewing and revising the Criteria Document; (2) reviewing the NAAQS through development of a Staff Paper based on the revised Criteria Document; (3) external review of Criteria Document and Staff Paper drafts by the Clean Air Scientific Advisory Committee (CASAC) of the EPA's Science Advisory Board, an independent panel of scientific experts, and by the public; and (4) examining implementation ramifications of changes to the O_3 NAAQS. The EPA intends to adhere to strict schedules for external review of Criteria Document and Staff Paper drafts consistent with a full opportunity for thorough scientific and public review, and to deny any requests for extensions of the public comment periods specified in this notice.

During this NAAQS review, the EPA intends to continue implementing programs designed to meet the current standards and the requirements of the Clean Air Act Amendments of 1990 to ensure continued improvement in air quality. The EPA is also examining options for implementing alternative O_3 NAAQS to ensure a smooth transition if

a decision is made to revise the existing NAAQS.

FOR FURTHER INFORMATION CONTACT: Dr. Karen Martin, Air Quality Management Division (MD-12), U.S. Environmental Protection Agency, Research Triangle Park, N.C. 27711, telephone (919) 541-5274.

SUPPLEMENTARY INFORMATION:

Background

Based on a Criteria Document issued by the Department of Health, Education and Welfare in 1970, the EPA promulgated the first NAAQS for photochemical oxidants under section 109 of the Act (36 FR 8186) in 1971. The primary and secondary NAAQS were both set at an hourly average of 0.08 parts per million (ppm) total photochemical oxidants not to be exceeded more than 1 hour per year.

In 1977, the EPA announced (42 FR 20493) the first review and updating of the 1970 Criteria Document in accordance with section 109(d)(1) of the Act. The EPA published a Criteria Document in 1978. Based on the revised Criteria Document and taking into account public comments on revisions proposed to the primary and secondary NAAQS in 1978 (43 FR 16962), the EPA announced revisions to the 1971 standards in 1979 (44 FR 8202). The primary standard was revised from 0.08 parts per million (ppm) to 0.12 ppm; the secondary standard was set identical to the primary standard; the chemical designation of the standards was changed from photochemical oxidants to O_3 ; and the form of the standards was revised from a deterministic form to a statistical form, which defines attainment of the standards as occurring when the expected number of days per calendar year with maximum hourly average concentrations greater than 0.12 ppm is equal to or less than one.

In 1982 (47 FR 11561), the EPA announced plans to revise the 1978 Criteria Document. In 1983, the EPA announced (48 FR 38009) that review of primary and secondary standards for O_3 had been initiated. The EPA subsequently provided a number of opportunities for public review and comment on drafts of the Criteria Document and associated Staff Paper (Review of the National Ambient Air Quality Standards for Ozone: Assessment of Scientific and Technical Information—Office of Air Quality Planning and Standards Staff Paper). After reviewing the draft Criteria Document in 1985 and 1986, the CASAC sent the Administrator a "closure letter" outlining key issues and recommendations and indicating that it

was satisfied with the final draft of the 1986 Criteria Document.

Following closure, a number of scientific articles and abstracts were published or accepted for publication that appeared to be of sufficient importance concerning potential health and welfare effects of O_3 to warrant preparation of a supplement to the Criteria Document (Supplement). The CASAC, having already reviewed two drafts of the Staff Paper in 1986 and 1987, concluded that sufficient new information existed to recommend incorporation of relevant new information into a third draft of the Staff Paper.

The CASAC held a public meeting in 1988 to review a draft Supplement and the third draft Staff Paper. Major issues included: The definition of adverse health effects of O_3 ; the significance of health studies suggesting that exercising individuals exposed for 6 to 8 hours to O_3 levels at or below 0.12 ppm may experience lung inflammation and transient decreases in pulmonary function; the possibility that chronic irreversible effects may result from long-term exposures to elevated levels of O_3 ; and, the importance of analyses indicating that agricultural crop damage may be better defined by a cumulative seasonal average than by a 1-hour peak level of O_3 . In its "closure letter" of 1989, the CASAC indicated that the draft Supplement and draft Staff Paper "provide an adequate scientific basis for the EPA to retain or revise primary and secondary standards for ozone."

On October 22, 1991, the American Lung Association and other plaintiffs filed suit under section 304 of the Act to compel the EPA to complete its review of the criteria and standards for O_3 under section 109(d)(1) of the Act (*American Lung Association v. Reilly*, No. 91-cv-4114 (JRB) (E.D.N.Y.)). The U.S. District Court for the Eastern District of New York subsequently issued an order requiring the EPA to sign a **Federal Register** notice announcing its proposed decision on whether to revise the standards for O_3 by August 1, 1992 and to sign a **Federal Register** notice announcing its final decision by March 1, 1993.

On August 10, 1992 (57 FR 35542), the EPA published a proposed decision under section 109(d)(1) that revisions to the existing primary and secondary standards were not appropriate at that time. The notice explained in some detail (see 57 FR 35546) that the proposed decision would complete the EPA's review of information on health and welfare effects of O_3 assembled over a 7-year period and contained in the 1986 Criteria Document and the 1989

Supplement. The notice made clear that the Administrator did not take into account more recent studies on the health and welfare effects of O₃ because these studies had not been assessed in the 1986-1989 Criteria Document/Supplement, nor had they collectively undergone the rigorous, integrative review process (including CASAC review) required to incorporate them into a new criteria document. The proposed decision, therefore, was based on an evaluation of key studies published through early 1989 as contained in the 1986-89 Criteria Document/Supplement, the 1989 Staff Paper assessment of the most relevant information in these documents, and the advice and recommendations of the CASAC as presented both in the discussion of these documents at public meetings and in the CASAC's 1986 and 1989 "closure letters."

In view of the large number of recent scientific papers and ongoing research on the health and welfare effects of O₃, the August 10, 1992 notice also announced the EPA's intention to proceed as rapidly as possible with the next review of the air quality criteria and standards for O₃. On March 9, 1993 (58 FR 13008) the EPA published its final decision not to revise the current primary and secondary NAAQS for O₃. Because of the scientific and technical complexity of such assessments, the EPA had estimated that 2 to 3 years would be necessary to rigorously assess more than 1,000 new studies and incorporate key information into a revised criteria document, to evaluate the significance of the key information for decision-making purposes, to develop staff recommendations for the Administrator, and to provide appropriate opportunities for CASAC review and public comment. Given the potential importance of the new studies and the EPA's continuing concern about the health and welfare effects of O₃, the March 9, 1993 notice also indicated the Administrator's intention to move the review process ahead as quickly as possible and, if appropriate, to propose revisions of the standards at the earliest possible date.

Current Review Process/Schedule

Following publication of the March 9, 1993 decision, the Agency, in consultation with the CASAC and the Science Advisory Board, undertook a rigorous examination of the NAAQS review process designed to identify all measures that could be taken to accelerate its review of the criteria and standards for O₃ consistent with assuring a sound and scientifically-credible decision. As a result, the EPA

has adopted a number of measures intended to accelerate the O₃ NAAQS review. These measures include: (1) Conducting review and revision of the Criteria Document and development of the Staff Paper and associated analyses (e.g., exposure analysis and health risk assessments) in a more concurrent fashion than in the previous NAAQS reviews; (2) adhering to strict schedules for external review of Criteria Document and Staff Paper drafts consistent with a full opportunity for thorough scientific and public review; (3) establishing a highly-expedited Agency review process with senior level management oversight and involvement throughout the process, as well as early discussion of possible options with other Federal agencies, including the Office of Management and Budget; and (4) reducing the volume of information included in the revised Criteria Document by focusing on the most important new studies and setting a date beyond which new studies will not be included.

The EPA's current O₃ NAAQS review schedule incorporates the measures cited above. The Agency's target date for completion of the Criteria Document and Staff Paper is mid-1995, with proposal of changes to the O₃ NAAQS, if appropriate, in mid-1996 and promulgation in mid-1997. Table 1 outlines key milestone dates for this accelerated schedule.

As indicated in Table 1, there are a number of opportunities for public comment throughout the process. The EPA encourages involvement of interested parties and is providing this advance notice to alert potential participants in the review that adhering to the schedule will require some departures from past practices.

TABLE 1

Major milestones	Tentative dates
CASAC Subcommittee Meeting on Exposure Assessment Methods.	December 1993.
CASAC and Public Comment Period for Criteria Document (CD) (90 days).	February to May 1994. ¹
CASAC Subcommittee Meeting on Risk Assessment Methods.	March 1994.
CASAC Meeting on CD.	July 1994.
Comment Period on Staff Paper (SP) (60 days).	September to October 1994.
CASAC Meeting on SP.	October 1994.

TABLE 1—Continued

Major milestones	Tentative dates
Public Comment Period on Revised CD and SP (90 days).	Early 1995.
CASAC Meeting on Revised CD and SP.	Mid-1995.
Agency Development of Regulatory Decision/Proposal Package Draft.	Mid-1995 to late 1995.
Office of Management and Budget Review of Proposal Package.	Early 1996.
Publication of Proposal in Federal Register.	Mid-1996.
Public Comment Period on Proposal (90 days).	Mid-1996.
CASAC Meeting to Review Proposal.	Late 1996.
Regulatory Decisions and Final Package Draft Completed.	Early 1997.
Office of Management and Budget Review of Promulgation Package.	Early 1997 to mid-1997.
Publication of Promulgation Notice in Federal Register.	Mid-1997.

¹ For a notice of availability of external review draft, see 59 FR 4278, January 31, 1994.

In particular, the EPA has often granted requests for extensions of public comment periods in previous reviews; in order to meet the accelerated schedule for the O₃ NAAQS review, however, the EPA intends not to grant such extensions during this review. Accordingly, potential participants in the review should take note of the process outlined in this notice and be prepared to respond promptly when opportunities to comment are offered. Given the scientific and technical complexity of the issues likely to be involved in the O₃ review, the diversity of scientific opinion that has characterized previous reviews of the criteria and standards for O₃, and the need to ensure that its ultimate decision is soundly based, the EPA cannot, of course, provide any absolute assurance that it will meet all of the interim milestone dates indicated in Table 1. Completion of the necessary steps in a timely manner is also predicated upon the availability of adequate resources during the review process. However, the Administrator has emphasized a high priority on meeting the accelerated schedule outlined in this notice.

To that end, the review process is well under way. The EPA initiated action to update the air quality criteria for O₃ in August 1992 (57 FR 38832). It

held two peer-review workshops on draft health effects chapters of a revised Criteria Document (58 FR 35454) in July 1993. Additional workshops on draft air quality and ecological effects chapters (58 FR 48063) were held in September 1993. Since then, the EPA has discussed the schedule and process outlined in this notice with the CASAC (58 FR 59034). The EPA is also conducting exposure and risk analyses. A subcommittee of the CASAC met on December 16, 1993 to review methodologies (58 FR 63345). A further subcommittee meeting on risk analysis is planned for spring 1994.

Implementation

It is important to stress that while conducting this review, the EPA remains committed to implementing the existing O₃ NAAQS in accordance with the Clean Air Act Amendments of 1990 (CAAA). During the review, the EPA will continue to work with States to implement emission control strategies required by the CAAA to meet the existing O₃ NAAQS. These efforts include State and Federal actions to reduce emissions of volatile organic compounds and nitrogen oxides, which act as precursors to O₃ formation in the troposphere. The EPA will make every effort to maintain implementation schedules consistent with requirements of the CAAA to ensure continued improvement in air quality.

As part of the review, the EPA is examining the ramifications of any changes to the NAAQS on current implementation efforts. If appropriate, new implementation rules and guidelines will be considered for alternative NAAQS. The EPA also is reviewing options to ensure a smooth transition for implementation of any new O₃ NAAQS in the event a decision is made to revise the current O₃ NAAQS.

List of Subjects in 40 CFR Part 50

Environmental protection, Air pollution control, Carbon monoxide, Lead, Nitrogen dioxide, Ozone, Particulate matter, Sulfur oxides.

Dated: January 27, 1994.

Carol M. Browner,

Administrator.

[FR Doc. 94-2487 Filed 2-2-94; 8:45 am]

BILLING CODE 6560-65-P

FEDERAL COMMUNICATIONS COMMISSION

47 CFR Parts 2 and 68

[CC Docket No. 93-268, RM-7815, RM-6147; FCC 93-484]

Connection of Customer-Provided Terminal Equipment to the Telephone Network

AGENCY: Federal Communications Commission.

ACTION: Proposed rules.

SUMMARY: This Notice of Proposed Rulemaking (NPRM) proposes to amend rules which regulate the terms and conditions under which customer-provided terminal equipment may be connected to the telephone network. The proceeding was initiated by petitions for rulemaking filed by Southwestern Bell Telephone Company (SWB) and Ameritech Operating Companies (Ameritech) who ask that regulations governing switched digital services be added. The effect of the proposed rules would be to promote rapid exploitation of switched digital technology. We propose also to provide for a registration revocation procedure which should greatly enhance our ability to enforce applicable rules as well as the Telecommunications Trade Act of 1988; and we take this opportunity to propose clarifications to other rules.

DATES: Comments were to be submitted on or before January 13, 1994, and replies by January 28, 1994; however, those dates have been extended to February 10, 1994 for comments and February 25, 1994 for replies.

ADDRESSES: Office of the Secretary, Federal Communications Commission, 1919 M Street, NW., Washington, DC 20554, with copy to William H. von Alven, FCC, Mail Stop 1600B2, Washington, DC 20554.

FOR FURTHER INFORMATION CONTACT: William H. von Alven, Domestic Services Branch, Domestic Facilities Division, Common Carrier Bureau, (202) 634-1833.

SUPPLEMENTARY INFORMATION: This summarizes the NPRM in CC Docket 93-268, RM-7815, and RM-6147 (FCC 93-484) adopted October 22, 1993 and released November 22, 1993, supplemented by an Errata, and Order Extending Comment Period released January 12, 1994 (DA 94-46). Persons affected by part 68 practice and procedure are urged to review the full texts of both the NPRM and Errata, and the supporting file, which are available for inspection and copying during the

weekday hours of 9 a.m. to 4:30 p.m. in the FCC Reference Center, room 239, 1919 M St., NW., Washington, DC. Copies may be purchased from the Commission's duplicating contractor, ITS, Inc., 2100 M St., NW., suite 140, Washington, DC 20037, (202) 857-3800.

Paperwork Reduction Act

Reporting and recordkeeping activities needed to comply with the proposed rules are usual and customary.

Analysis of Proceeding

1. By this NPRM we contemplate amending parts 2 and 68 of the rules, 47 CFR parts 2 and 68. A purpose of part 68 is to maintain uniform standards for the protection of the telephone network from harms caused by the connection of terminal equipment and associated wiring. This proceeding was initiated by two petitions for rulemaking, one filed by SWB (RM-7815) and the other by Ameritech (RM-6147).

2. SWB requests that part 68 be amended to include the regulation of terminal equipment connected to the two-wire Basic Rate Access (BRA) interface and to the Primary Rate Access (PRA) interface provided by Integrated Services Digital Network (ISDN) access technology. BRA consists of one or two 64 Kbps information channels with a 16 Kbps channel for dialing and network access information. The 1.544 Mbps PRA consists of 23 64 Kbps information channels and the 64 Kbps dialing and network access channel. ISDN is in a developmental phase, being deployed these last few years in an experimental mode. The Public Notice of SWB's petition elicited comments from eight parties and reply comments from three. There was overwhelming support for including this service in part 68 in order to promote, on a nationwide and worldwide basis, rapid exploitation of this technology with minimum mandatory criteria for connection of CPE (customer premises equipment). Thus, we propose for comment technical standards for including this service in part 68 in supplement to the existing standards for non-switched leased-line digital services which were added in 1985.

3. Commenting on SWB's petition, AT&T recommends (a) that part 68 rules covering PRA not be limited to the two-wire ISDN BRA service but also authorize terminal equipment connected to the 4-wire ISDN PRA (1.544 Mbps) interface pursuant to performance and compatibility standards adopted by ANSI (American National Standards Institute); (b) that amendments to part 68 provide equipment specifications for both PRA

**OZONE NAAQS
DEVELOPMENT PROJECT PLAN**

**OFFICE OF AIR QUALITY
PLANNING AND STANDARDS**

**RESEARCH TRIANGLE PARK
NORTH CAROLINA**

December 16, 1993

Ozone NAAQS Development Project Plan

I. Introduction/Background

A. Purpose of Project Plan

There are two major purposes for this ozone (O₃) national ambient air quality standards (NAAQS) development project plan. They are:

- (1) To describe the proposed process for coordinating and implementing the scientific assessments, standards development, implementation assessments, Office of Management and Budget (OMB) coordination, and communication plan within the Administrator's accelerated schedule (final action by mid-1997).
- (2) To identify key issues in scientific assessment and standards development for the NAAQS review and lay out plans for preparing scientific and technical assessments related to decisions on the appropriate primary and secondary NAAQS.

B. Background

On April 30, 1971, the EPA promulgated primary and secondary NAAQS for photochemical oxidants under section 109 of the Act (36 FR 8186). These were set at an hourly average of 0.08 parts per million (ppm) total photochemical oxidants not to be exceeded more than 1 hour per year. On April 20, 1977, the EPA announced (42 FR 20493) the first review and updating of the 1970 Air Quality Criteria Document for Photochemical Oxidants in accordance with section 109(d)(1) of the Act. The EPA published the final revised Air Quality Criteria for Ozone and Other Photochemical Oxidants on June 22, 1978.

Based on the 1978 revised Air Quality Criteria Document (U.S. EPA, 1978) and taking into account public comments on revisions proposed to the primary and secondary NAAQS on June 22, 1978, (43 FR 16962), the EPA announced its final decision on the proposed revisions to the 1971 standards (44 FR 8202, February 8, 1979). The final rulemaking revised the level of the primary standard from 0.08 ppm to 0.12 ppm, set the secondary standard identical to the primary standard, changed the chemical designation of the standards from photochemical oxidants to O₃, and revised the definition of the point at which the standard is attained to when the expected number of days per calendar year with maximum hourly average concentrations above 0.12 ppm is equal to or less than one as determined by Appendix H.

On March 17, 1982 (47 FR 11561), the EPA announced that it was undertaking plans to revise the existing 1978 "Air Quality Criteria Document for Ozone and Other Photochemical Oxidants" and on August 22, 1983, announced (48 FR 38009) that review of primary and secondary standards for O₃ had been initiated. The EPA subsequently provided a number of opportunities for public review and comment on drafts of the Criteria Document and OAQPS Staff Paper ("Review of the National Ambient Air Quality Standards for Ozone: Assessment of Scientific and Technical Information--OAQPS Staff Paper"). The Clean Air Scientific Advisory Committee (CASAC), after reviewing the draft Criteria Document in 1985 and 1986, sent to the Administrator on October 22, 1986, a "closure letter," outlining key issues and recommendations and indicating that it was satisfied with the final draft of the 1986 Criteria Document.

Following closure, a number of new scientific findings were made that appeared to be of sufficient importance concerning potential health and welfare effects of O₃ to warrant preparation of a supplement to the Criteria Document. The CASAC, having already reviewed two drafts of the OAQPS Staff Paper in 1986 and 1987, concluded that sufficient new information existed to recommend incorporation of relevant new information into a third draft of the OAQPS Staff Paper.

The CASAC held a public meeting on December 14-15, 1988 to review the draft Supplement and draft OAQPS Staff Paper. Major issues included: the definition of adverse health effects of O₃, the significance of health studies suggesting that exercising individuals exposed for 6 to 8 hours to O₃ levels at or below 0.12 ppm may experience inflammation and transient decreases in pulmonary function, the possibility that chronic irreversible effects may result from long-term exposures to elevated levels of O₃, and the importance of analyses which indicate agricultural crop damage may be better defined by a cumulative seasonal average than by a 1-hour peak level of O₃. In its "closure letter" of May 1, 1989, the CASAC indicated that the draft Supplement and draft OAQPS Staff Paper "provide an adequate scientific basis for the EPA to retain or revise primary and secondary standards for ozone."

On October 22, 1991, the American Lung Association and other plaintiffs filed suit under section 304 of the Act to compel the EPA to complete its review of the criteria and standards for O₃ under section 109(d)(1) of the Act [American Lung Association v. Reilly, No. 91-cv-4114 (JRB) (E.D.N.Y.)]. The U.S. District Court for the Eastern District of New York subsequently issued an order requiring the EPA to sign a Federal Register notice announcing its proposed decision on whether to revise the standards for O₃ by August 1, 1992 and to sign a Federal Register notice announcing its final decision by March 1, 1993.

On August 10, 1992 (57 FR 35542), the EPA published its proposed decision under section 109(d)(1) that revisions to existing 1-hour primary and secondary standards were not appropriate at that time. The notice explained in some detail (see 57 FR 35546) that the proposed decision would complete the EPA's review of information on health and welfare effects of O₃ assembled over a 7-year period and contained in the 1986 Air Quality Criteria

Document and its Supplement. The review included an evaluation of key studies published through early 1989, the 1989 OAQPS Staff Paper assessment of the most relevant information in these documents, and the advice and recommendations of the CASAC as presented both in the discussion of these documents at public meetings and in the CASAC's 1986 and 1989 "closure letters." In the same notice, the EPA also announced its plans, in view of the large number of recent scientific papers and ongoing research on the health and welfare effects of O₃, to proceed as rapidly as possible with the next review of the air quality criteria and standards for O₃. On March 9, 1993 (58 FR 13008) EPA published its final decision not to revise the current primary and secondary NAAQS for O₃ and emphasized the Administrator's intention to proceed as rapidly as possible with a review of the scientific research published since 1989.

II. NAAQS Review Process

Based on a thorough examination of the NAAQS review process, the EPA has adopted a number of measures in an effort to accelerate the criteria and standards review for O₃. EPA's objective is to conduct these reviews as rapidly as possible consistent with assuring a scientifically supportable and legally defensible decision. The measures adopted include:

- * Reducing the volume of information on health and welfare effects included in the "criteria" for the NAAQS review by **focusing on relevant key new studies** and fixing a firm date beyond which new studies will not be included.
- * **Conducting the health/welfare effects review** (the Criteria Document) and associated exposure/risk assessments (summarized in the OAQPS Staff Paper) **in a more concurrent fashion than in previous reviews.**
- * Taking steps to ensure **adequate but significantly reduced time for peer** (i.e., CASAC) **and public reviews** of the Criteria Document and OAQPS Staff Paper, by enlisting the CASAC's cooperation in expediting their review and advice to the Administrator and not granting extensions of public comment periods.
- * Establishing a **highly expedited Agency review, issue resolution, and closure process**, as well as undertaking early discussion of possible options with other federal agencies (e.g., Office of Management and Budget).
- * **Issuing a Federal Register Notice announcing the review schedule and the above measures being taken to meet the schedule** to provide early notice to public groups interested in participating in the review process.

Overview of NAAQS Review Schedule

The scientific assessment phase of the accelerated O₃ NAAQS review schedule is shown in Figure 1. This schedule incorporates the measures described above. Given the scientific and technical complexity of the issues likely to be involved in the O₃ review, the diversity of scientific opinion that has characterized previous reviews of the criteria and standards for O₃, and the need to ensure that its ultimate decision is soundly-based, the EPA cannot, of course, provide any absolute assurance that it will meet all of the interim milestone dates indicated in Figure 1. Completion of the necessary steps in a timely manner is also predicated upon the availability of adequate resources during the review process. However, the Administrator has emphasized that she places a high priority on meeting the accelerated schedule outlined in this notice.

III. Scientific Assessment and Standards Development

A. Background and Key Issues

In order to meet the accelerated schedule shown in Figure 1, it is necessary to work as rapidly as possible on various analyses that will provide information for the Administrator's decision on whether to revise the primary and/or secondary NAAQS for O₃. Based on current scientific information and staff judgments, several options are defined below which will be used in various analytic efforts. It is recognized that additional or different options may have to be analyzed based on review of Criteria Document and OAQPS Staff Paper drafts by the CASAC and the public. However, given the time and resource requirements for certain analyses, if additional options are added, it may not be possible to conduct some analyses and still remain on the accelerated schedule.

The initial options listed below reflect the staff's best judgment, at this point in time, of the options to be used for various regulatory analyses, given the current state of scientific information. These options are not prioritized and are subject to change based on the formal review and revision of the Criteria Document.

Analytic Options for the Primary O₃ NAAQS

The basis for a short-term standard to protect against acute 1-hr exposures has been enhanced by additional research findings since the current standard was promulgated in 1979. Recent controlled human exposure and human field/epidemiology studies have strengthened the case for considering establishment of a longer averaging time primary NAAQS on the order of 8 hours to protect against acute effects and possibly provide some protection against potential chronic effects. Evidence is also growing concerning chronic effects that might require a much longer averaging time standard (e.g., seasonal mean). However, it is unlikely that the science will be sufficiently quantitative to support a separate chronic standard within the next 3-5 years. If the criteria review supports this assumption, such

chronic effects information will need to be considered as one of the factors in the Administrator's judgment about which standard(s) provide an adequate margin of safety. The initial options to be analyzed are:

- * Retain the 1-hour NAAQS of 0.12, ppm, using the current form of the standard (i.e., 1 expected exceedance allowed per year).
- * Establish a new 8-hour NAAQS in the range 0.08-0.10 ppm, with 1 expected exceedance allowed per year.
- * Establish a new 8-hour NAAQS in the range 0.06-0.08 ppm, with 5 expected exceedances allowed per year.
- * Whether or not to retain the current 1-hour NAAQS if a separate 8-hour NAAQS is established.

A key issue is whether a new 8-hour NAAQS will provide sufficient protection against 1-hour peaks of concern if changes in O₃ air quality patterns occur in the future.

Analytic Options for the Secondary O₃ NAAQS

Since early 1989, there have been several analyses of the National Crop Loss Assessment Network (NCLAN) data, as well as continued research into the effects of O₃ on trees. The NCLAN reanalyses led to the conclusion that an index that is protective of annual crops (1) cumulates hourly O₃ values over time, (2) is peak-weighted, and (3) places greatest weight on exposures that occur during certain plant growth stages. The NCLAN reanalyses identified several indices as adequate to represent the relationship between O₃ exposures and crop response. One of these, the SUM06 index, sums all hourly O₃ concentrations of 0.06 ppm and higher over a specified period of time. For protection of crops, analysis of the NCLAN data suggests using the 3 months having the highest O₃ concentrations (i.e., the maximum 3 months). For protection of trees, emerging data suggest that a longer time period (up to 5 months) may be appropriate. However, since the NCLAN data are currently the largest set of data available for considering standard-setting options, the options listed below emphasize a 3-month exposure period. In the future, information on effects of trees may suggest a longer exposure period. Alternative SUM06 levels were identified as options for analysis based on prior analyses of percent crop yield loss in a certain percentage of crop species. Since then, concern about the ability of existing air quality models to accurately assess and predict O₃ concentrations below 0.08 ppm, has led to inclusion of alternative SUM08 standards that provide the same level of crop protection as the alternative SUM06 standards under consideration. The options to be analyzed are:

- * Replace the existing 1-hour NAAQS with a new SUM06 standard where the sum of the concentrations ≥ 0.06 ppm is < 16.5 ppm-hrs for the maximum 3 calendar-month period.

- * Replace the existing 1-hour NAAQS with a new SUM06 standard where the sum of the concentrations ≥ 0.06 ppm is < 26.4 ppm-hrs for the maximum 3 calendar-month period.
- * Replace the existing 1-hour NAAQS with a new SUM08 standard where the sum of the concentrations ≥ 0.08 ppm is $< x$ ppm-hrs over the maximum 3 calendar-month period (where x is to be determined by additional analyses underway by ORD/Corvallis).
- * Replace the existing 1-hour NAAQS with a new 8-hour standard of 0.08 ppm with either 1 or 5 expected exceedances allowed per year.

B. Criteria Document Development

Overview

The previous "Air Quality Criteria for Ozone and Other Photochemical Oxidants" (O₃ Criteria Document) was released by EPA in August 1986 and a supplement, "Summary of Selected New Information on Effects of Ozone on Health and Vegetation," was released in January 1992. Review and revision of the O₃ Criteria Document will be performed by the Environmental Criteria and Assessment Office in Research Triangle Park, NC (ECAO/RTP) in accordance with sections 108 and 109 of the Clean Air Act to provide the scientific basis for reevaluation of the O₃ NAAQS. A separate draft work plan is available that discusses the structure, review process, and major scientific issues to be addressed in the revision of the O₃ Criteria Document (ECAO, 1992).

Structure

The proposed structure of the document includes first, an Executive Summary and Conclusions (Chapter 1), providing a concise presentation of key information and conclusions from all subsequent chapters. This is followed by a brief Introduction (Chapter 2) containing information on the legislative background and purpose of the document, as well as an overview of the organization of the document. Chapter 3 provides information on the chemistry, measurement, and transport of O₃ and related photochemical oxidants, whereas Chapter 4 covers environmental concentrations, patterns, and exposure estimates. This is followed by Chapter 5, dealing with environmental (e.g., ecological, agricultural, materials) effects of O₃ and related photochemical oxidants. Chapters 6, 7, and 8 discuss animal toxicological studies, human health effects, and extrapolation of animal toxicological data to humans, respectively. The last chapter (Chapter 9) will be an integrative, interpretative evaluation of health risks to the exposed population.

Status/Schedule

ECAO/RTP began revision of the O₃ Criteria Document by announcing the commencement of the review in the Federal Register (57 FR 38832). The main focus of the revised O₃ Criteria Document is on evaluation and interpretation of those new (i.e., after the 1986 Criteria Document) air quality, health, and welfare effects data identified as being pertinent to development of criteria supporting a decision on possible revision of the O₃ NAAQS. After careful assessment and evaluation of pertinent new studies by scientific experts within and outside the Agency, preliminary draft chapters have been prepared. A series of expert workshops have been held to review the draft revised O₃ Criteria Document chapters, to identify the potential need for additional information to be added to the chapters, and to evaluate the quality of the summarization and interpretation of the literature. Although the reviewers were invited experts, the workshops were announced in the Federal Register and were open to the public. The respective authors of the draft chapters are revising them on the basis of the workshop recommendations. After resolution of outstanding issues and comments, ECAO/RTP will forward the document as the first external review draft for public comment and review by the CASAC. Any necessary revisions will be made in response to public comments and CASAC recommendations in preparing the final version of the revised O₃ Criteria Document for further review by CASAC (if needed) and publication. The schedule for the revised O₃ Criteria Document preparation process is reflected in Figure 1.

C. Staff Paper Development

1. Overview and Structure

Overview

The OAQPS Staff Paper provides the Administrator, CASAC, and others involved in the standard-setting process with a concisely written assessment of the scientific literature and technical analyses which form the basis for recommendations and decisions on a range of alternative NAAQS. The preparation of the OAQPS Staff Paper and its review by the public and the CASAC is intended to ensure that the staff's interpretation of the scientific evidence in the Criteria Document is sound and is supported by the scientific community.

Structure

The OAQPS Staff Paper will provide an assessment of the key health and welfare effects information evaluated in the revised O₃ Criteria Document. Several factors are important in the staff's assessment and development of recommendations on alternative primary NAAQS. These factors include: (1) biological and statistical significance of effects; (2) adversity of effects; (3) credibility of studies; (4) type of study (e.g., human clinical, animal, or epidemiology); (5) nature and magnitude of human exposure to O₃ levels of concern; (6) nature and magnitude of health risks associated with attainment of alternative

primary NAAQS; and (7) basis for selecting appropriate averaging time(s) and form(s) of the primary NAAQS.

Similar factors are relevant for the staff's assessment and development of recommendations on alternative secondary NAAQS. These include: (1) type of welfare effect; (2) biological and statistical significance of effects; (3) extent of effects (e.g., amount of crop yield loss); (4) adversity of effects; (5) credibility of studies; (6) type of study (e.g., chamber, field); (7) assessments of crop and/or forest damage upon attainment of alternative secondary NAAQS, and (8) basis for selecting appropriate averaging time(s) and form(s) of the secondary NAAQS. The OAQPS Staff Paper will include summary discussion of the various analyses relevant to decision making on the primary and secondary NAAQS (e.g., form of the standard, exposure, health risk, crop loss, and welfare benefits).

Status/Schedule

EPA will begin preparation of the OAQPS Staff Paper in early 1994. An external review draft is scheduled to be released in mid 1994 and the CASAC meeting on the first draft of the OAQPS Staff Paper is scheduled for late 1994. After revisions are made, a second CASAC meeting and, hopefully, closure are scheduled for early 1995.

Issues

Many of the policy and technical issues that will be addressed by various types of analyses and then summarized in the OAQPS Staff Paper are identified in the sections below (e.g., air quality, form of the standards, human exposure, health risk, and welfare effects assessment).

2. Air Quality and Form of Standards

Overview

This section will use ambient O₃ data to document status with respect to the current O₃ NAAQS and to provide a brief summary of U.S. O₃ trends. A brief discussion of background O₃ levels will be included based upon material from the revised O₃ Criteria Document. This background information will provide a frame of reference for the discussion of alternative standards and possible modifications to the attainment test. Because high O₃ levels in the east during the summer of 1988 received considerable attention, presentations of ambient data will be structured to illustrate the impact of 1988.

In addition to the form of the standard, this section also will present a discussion of how the attainment test could be modified, including the use of a more robust surrogate or the introduction of a "too close to call" category. Pros and cons of various options will be presented to generate discussion on the potential merits of such approaches.

Structure

Shaded U.S. maps will be used to identify areas exceeding selected NAAQS options: 1 hour (.12 ppm, 1 expected exceedance); 8 hours (.08 and .10 ppm, 1 expected exceedance); and 8 hours (.06 and .08 ppm, 5 expected exceedances). SUM06 and SUM08 areas also will be illustrated for at least two cut-points. To illustrate the impact of 1988, these results will be presented for three different time periods: 1990-92 (the most current 3-year period available); 1987-89 (the 3-year period most commonly used for the 1990 Clean Air Act Amendments classification process); and 1988-92 (the most recent 5-year period which also includes 1988). All of these maps will be determined using the same data handling procedures used for the current O₃ NAAQS.

Alternatives to the attainment test will be presented with a brief discussion of the potential advantages and disadvantages. The use of a more robust surrogate, such as the 95th percentile, the use of a fitted statistical distribution, such as tail exponential, and the use of design values rather than expected exceedances, will use material from the Ozone Design Value Study. New material will be developed for inclusion in the OAQPS Staff Paper concerning the "too close to call" approach.

Issues

With an 8-hour level as low as 0.06 ppm being considered, there will be discussion of what is background. Levels from remote sites may provide some insight but will not necessarily answer the question, and it may be necessary to develop atmospheric chemistry arguments.

The ambient data used in these analyses is based upon data in AIRS from monitors sited primarily to document 1-hour O₃ values. It is possible, particularly for SUM06, that existing networks are not adequate to fully characterize status with respect to these alternative NAAQS.

3. Human Exposure Analyses

Overview

The impact that attaining alternative O₃ NAAQS may have on human exposure will be evaluated using a new version of the Probabilistic NEM (NAAQS Exposure Model) applied to O₃ (pNEM/O₃). The alternative NAAQS that will be evaluated are listed above in section III.A. Exposure estimates will be obtained directly for nine U.S. urban areas located in each EPA Region except Region X. The nine areas display a wide variety in population, geographic area, demographic makeup, climatology, and O₃ air quality. Results from these areas will be extrapolated to the entire U.S. urban population via a functional relationship fit to the nine-area results. Uncertainty in the estimates will be explicitly addressed using Monte Carlo modeling techniques.

The exposure estimates will assist the Administrator in evaluating which alternative primary NAAQS provide an adequate margin of safety.

Issues

1. Resource priorities dictate that only 10 Monte Carlo simulations be undertaken for any one urban area. It is the staff's judgment that the value of additional information provided by conducting additional runs is not worth the cost. Use of 10 simulations is two orders of magnitude fewer simulations than are normally undertaken. Thus, the issue arises whether or not all uncertainty in input data has been adequately addressed. This issue will be quantitatively addressed in the OAQPS Staff Paper.

2. Urban areas used for NAAQS exposure assessment are generally large in size and have adequate home-to-work transportation data needed to model daily population movement within the area. In addition, a minimum of three ambient monitors are needed to adequately characterize the spatial distribution of O₃ in an area. These requirements preclude including in a PNEM/O₃ analysis small urban areas with a limited data base. Many of the excluded areas, however, have more exceedances of the 8-hr levels of interest than the 1-hr levels; in fact, many of these areas do not exceed the current 1-hr NAAQS but have scores of days with 8-hr averages >0.06, 0.08, and 0.10 ppm, which are concentration levels of interest for the longer averaging time. Thus, we are not analyzing all possible types of O₃ air quality problems that currently exist in the U.S.

This issue can only be addressed qualitatively at the current time. The OAQPS Staff Paper will include some discussion of this important issue.

3. The human activity data base used for PNEM/O₃ modeling is limited. In fact, only the mid-1980's Cincinnati data base is used for O₃ analyses. This data base is not representative of true outdoor worker activities (in any climate), and may not be representative of general population or summer camp population activities in warmer climates. Thus, the question arises of how representative are the PNEM/O₃ modeling results? This question will be addressed both quantitatively and qualitatively in the Staff Paper to the extent possible. Special analyses of "summer camper" and "outdoor worker" exposures will be used in part to address this question.

Status/Schedule

The nine-urban-area modeling work is complete. A draft report describing the PNEM model and its inputs was submitted to EPA on September 30, 1993. A draft report describing the methodology and a memo describing results for Los Angeles is being provided to a CASAC Exposure/Risk Subcommittee in November 1993 and will be reviewed by this Subcommittee at a December 16, 1993 meeting. Separate reports will be prepared in early 1994 detailing the "summer camper" and "outdoor worker" analyses mentioned above.

These reports and results for the remaining 8 urban areas will be sent to the Subcommittee and reviewed at a public Subcommittee meeting in early 1994.

The extrapolation work from the nine urban areas will be done in-house starting in early 1994 and will take about one month to complete.

4. Health Risk Assessment

Overview

The primary purpose of the O₃ health risk assessment project is to provide quantitative (probabilistic) estimates of the risk to public health associated with attainment of alternative O₃ NAAQS and qualitative information characterizing the nature and significance of various health effects. A major goal of the risk assessment work is to provide a more explicit and quantitative characterization of the uncertainties in the scientific data base. This information is designed to assist the Administrator in selecting which primary standards will protect the public health with an adequate margin of safety. The probabilistic risk assessment will not cover all of the various health effect categories for which there is some scientific evidence of effects. However, the OAQPS Staff Paper will contain a summary and evaluation of all major health effects categories for which there is scientific evidence that O₃ plays a role.

The last O₃ review included a 1-hr health risk assessment for lung function (FEV₁) and respiratory symptoms (cough, chest discomfort, any) based on 3 human clinical studies of heavily exercising individuals for "as-is" scenario and just attaining 0.08, 0.10, and 0.12 ppm, 1-hr, 1 expected exceedance NAAQS. This information was summarized in the Staff Paper, was favorably reviewed by CASAC at its 1988 meeting, and has been presented and published.

A chronic lung injury risk assessment for O₃ was conducted by Argonne National Laboratory (ANL) for OAQPS in the 1989-1990 period. This project involved elicitation of probabilistic exposure-response relationships for formation of lesions in a critical region of the lung from 6 expert health scientists. This information was not part of the O₃ NAAQS review process, but was used to inform EPA decision makers about the potential significance and magnitude of chronic effects associated with O₃ exposures. The exposure period used was the average of the 8-hr daily maximum over the O₃ season (April-October) and judgments were elicited for 1 and 10 seasons of exposure.

Structure

The three major types of inputs to the health risk assessment for O₃ are: (1) exposure-response relationships used to characterize specific health effects; (2) distributions of 1- and 8-hr O₃ concentrations upon attainment of alternative NAAQS and under the "as is" situation; and (3) distributions of population exposure, both in terms of people exposed

and occurrences of exposure, upon attainment of alternative O₃ NAAQS and under the "as is" situation obtained from the PNEM/O₃ analysis described in Section III.C.3 of this plan.

Current plans are for risk estimates to be generated for the following health endpoints:

- lung function decrements (> 10, > 15, > 20% FEV₁) and respiratory symptoms (e.g., cough, chest discomfort) associated with: 1- to 2.5-hr exposures of heavily exercising subjects, 2.5-hr exposures of moderately exercising subjects, and 6.7- to 8-hr exposures of moderately exercising subjects. (Probabilistic exposure-response relationships developed from data from published controlled human exposure studies of healthy adult subjects.)
- airway reactivity associated with 6.7-hr exposures for moderately exercising subjects. (Probabilistic exposure-response relationships developed from data from published controlled human exposure studies of healthy adult subjects.)
- development of mild and moderate lesions in the centriacinar region of the lung after 1 and 10 seasons of exposure (using mean of O₃ season daily max. 8-hr averages) for NY and LA children and outdoor workers. Plan to use probabilistic exposure-response judgments obtained during chronic risk project in 1989. The purpose is to provide additional information for judgment on adequate margin of safety associated with alternative 1- and 8-hr O₃ NAAQS, not to be used to set a separate chronic standard.

Risk estimates may be generated for inflammatory responses associated with 8-hr exposures for moderately exercising population depending on feasibility and value of information. Discussions between OAQPS, HERL, and ECAO are ongoing to develop consensus on whether there is adequate information to conduct a quantitative risk assessment and how such an assessment should be structured that utilizes both the available animal and human data on this effect category.

Based on meetings with OAQPS staff and HERL and ECAO scientists, it was concluded that there was insufficient "quantitative information" to pursue development of probabilistic risk estimates for the following health endpoints: increased asthma attacks, emergency room visits, or hospital admissions for asthmatics on high O₃ days, increased acute mortality, and increased incidence of infection. The primary limitation for quantitative risk assessment purposes of the data reporting these effects is the lack of adequate characterization of exposure to O₃ and other pollutants and the difficulty of associating any given exposure period to the reported effects. The scientific evidence for each of these effects will be discussed and evaluated in the OAQPS Staff Paper.

Two distinct types of risk measures will be developed. The first measure, "benchmark risk," focuses on the probability of risk of unhealthful air. More specifically, benchmark risk is the probability that pollutant levels will be sufficient to trigger a defined health endpoint in at least a fraction of the sensitive population if they were exposed. The second measure, "headcount risk," focuses on the number of people affected and the number of incidences of a given health effect considering individuals' personal exposures as they go about their daily activities. (Since the exposure analysis will only generate exposure occurrences, headcount risk estimates will be in terms of incidences of effects.) Together, these two types of risk measures capture important perspectives that should be considered in selecting primary O₃ NAAQS that provide an adequate margin of safety.

In an effort to improve the characterization of the health consequences and adversity of the various health endpoints associated with O₃ exposures, OAQPS and ANL will work with ECAO, HERL, and non-Agency scientists to develop a protocol to elicit expert judgments on severity and consequence of effects associated with single and repeated occurrences of 1 to 8-hr exposures (i.e., lung function, respiratory symptoms, airway reactivity, and inflammation). Experts from within and outside of the Agency will be selected and interviewed to obtain their judgments.

Status/Schedule

Development of probabilistic concentration-response relationships is underway. The computer algorithms for generating both benchmark and headcount risk estimates are being setup and tested. Risk estimates and a draft report describing the methodology for the health endpoints indicated above, with the exception of inflammation, will be available for review by the CASAC Exposure/Risk Subcommittee in early 1994. For the significance of effects project a draft report with judgments of experts is scheduled for completion by mid 1994.

Issues

(1) Given the relatively weaker state of information, it is the staff's recommendation that risk estimates not be developed for some of the health effect endpoints (e.g., increased hospital admissions for asthmatics, increased acute mortality) reported in epidemiology studies. The evidence of these effects will be addressed qualitatively in the OAQPS Staff Paper.

(2) Since there will not be exposure estimates for the adolescent population attending summer camps, with the exception of a small (< 100 person) special PNEM/O₃ analysis, it is the staff's recommendation that risk estimates for this important sensitive population group not be produced. There is inadequate information on activity patterns to conduct a more extensive "summer camp" exposure analysis.

(3) The significance of effects project is a new type of product for the risk team and the approach to tackling this problem has not been worked out. It may be difficult to keep

on the tight time schedule indicated above given the interactive nature of this project with feedback from ORD, CASAC, and other external scientists, as well as EPA policy makers, guiding development of this project.

5. Welfare Effects Assessment

Overview

In response to the CASAC closure letter (May 1989) during the last O₃ NAAQS review, EPA's Environmental Research Laboratory in Corvallis, Oregon (ERL/Corvallis): (1) continued to reanalyze the NCLAN database in search of a more appropriate exposure index to relate plant response to acute O₃ exposures and 2) developed a long term research plan titled "Effects of Tropospheric Ozone on Forest Trees" to investigate chronic O₃ exposures on tree species. In the expansion from an acute to a chronic timeframe, additional environmental stressors have the potential to impact the equation (e.g., changing soil characteristics and populations, climate, pathogens).

Research initiated under the "Tropospheric Ozone" research plan has currently produced exposure-response functions (Weibull model) for eleven forest tree species, quantifying tree response in growth and biomass to different O₃ concentrations. These response functions are from seedling and sapling studies conducted in open-top exposure chambers. These studies have not incorporated the influence of multiple environmental factors, and in only a few instances contain results from multiple year exposures. As a means of comparing NCLAN's calculated average predicted relative crop yield losses with the relative predicted biomass loss calculated for the 11 tree species, the same SUMO6 levels of 16.5, 26.4, and 39.7 ppm-hr were evaluated. (The SUMO6 of 26.4 ppm-hr corresponds to protecting 50% of the crops from greater than 10% yield loss.)

Structure

The welfare effects chapter of the OAQPS Staff Paper will provide an assessment of the welfare effects information compiled in the Criteria Document. Several factors are important in the analysis. These factors include: (1) remaining uncertainty about the relationship between acute and chronic O₃ exposure regimes on plant growth; (2) the adequacy of existing air quality and exposure indices to take into account biologically and ecologically significant O₃ regimes; (3) ecosystem-level response to O₃; (4) the extent and biological and statistical significance of the effects observed; and (5) the type and credibility of the studies conducted.

The OAQPS Staff Paper will include summary discussions of analyses relevant to decision making on the secondary NAAQS. These analyses are summarized below:

- Reanalyses of the NCLAN data to compare degree of fit for over 600 different exposure indices which were completed in 1991.

-Additional analyses currently underway at ERL/Corvallis comparing the degree of protection offered between a given SUM06 value and 1) a SUM08 and 2) an 8-hour, .08 ppm standard with either 1 or 5 expected exceedances.

-Interim risk assessment of O₃ impact on eastern U.S. forests will use Geographical Information System (GIS) to integrate and combine various data (estimations of O₃ exposure in forests, landscape and environmental factors, species ranges, empirical exposure-response functions, and model simulations of canopy O₃ uptake, growth rates and stand response over time) to provide a risk characterization of O₃ on forest species in the eastern U.S. The SUM06 values listed in Section III.A. of this project plan are evaluated along with the additional SUM06 value of 39.7 ppm-hr.

-Benefits analysis also will assess in a preliminary fashion the economic significance of O₃'s effects on crops and forests. A contract has been initiated to determine the depth of the database available on O₃ sensitive urban plant species with the potential to develop maps of possible adverse effects in O₃ nonattainment areas. These analyses will occur in the context of the existing standard.

Status/Schedule

A preliminary review of the revised O₃ Criteria Document welfare effects chapters occurred at the peer-review workshop September 27-29, 1993. See the overall review schedule for the OAQPS Staff Paper in Figure 1.

Issues

The following key issues will need to be addressed in the development of the OAQPS Staff Paper:

- How reliable are existing extrapolation methods for calculating long term O₃ impacts on trees?
- To what extent should welfare effects risk assessments be used in the secondary standard-setting process?
- How should EPA determine which welfare effects and what degree of response should be considered "adverse" effects?

6. Significant Harm Levels

Overview

Section 303 of the CAA provides authority for the Administrator to identify exposure levels for ambient air pollutants that constitute "an imminent and substantial endangerment to

the health of persons." This language provides the basis for significant harm levels (SHL) which are set forth in subpart H of 40 CFR Part 51. The SHL is to be set at a level never to be reached at any location. The CAA and EPA regulations governing adoption and submittal of SIPs require States to adopt contingency plans for emission control measures that will prevent levels from reaching the SHL. Contingency plans must specify two or more "episode criteria" (typically "Alert" and "Warning" levels) which require increasing abatement actions. EPA policy is to review the basis for SHLs and guidance/examples of episode levels during the NAAQS review. The current SHL for O₃ is 0.6 ppm, 1-hr average.

Structure

The OAQPS Staff Paper will include a section assessing the scientific evidence reviewed in the revised O₃ Criteria Document that is relevant to determining whether there is a need to revise the SHL. Review of the current episode examples contained in Appendix L of Part 51 will be addressed as part of the implementation assessments.

Status/Schedule

See the overall review schedule for the OAQPS Staff Paper in Figure 1.

D. Standards (Part 50)

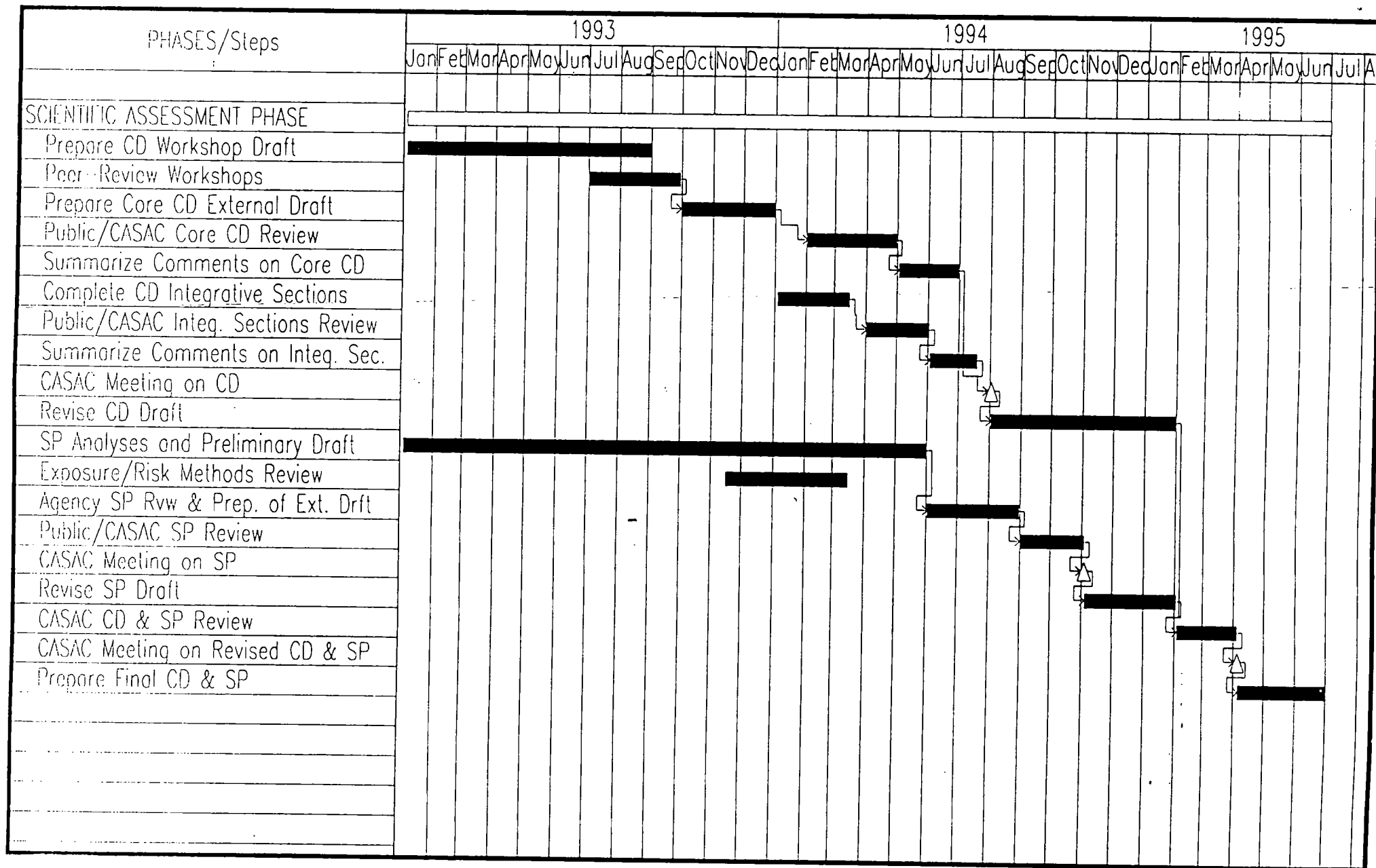
Overview

After CASAC "closure" on the revised O₃ Criteria Document and OAQPS Staff Paper in the Spring 1995, a regulatory options briefing will be prepared for the senior management oversight group and the Administrator. Once guidance has been provided, staff will develop a Federal Register notice which explains in detail the rationale for the proposed decision. Staff probably will draw heavily on the OAQPS Staff Paper and CASAC closure letter in developing the Federal Register proposal preamble.

Status/Schedule

Work on the Federal Register proposal notice will begin in the Spring 1995 when closure is obtained on the revised O₃ Criteria Document and OAQPS Staff Paper.

Projected Ozone NAAQS Review Schedule: Scientific Assessment Phase





Air Quality Criteria for Ozone and Related Photochemical Oxidants

Review

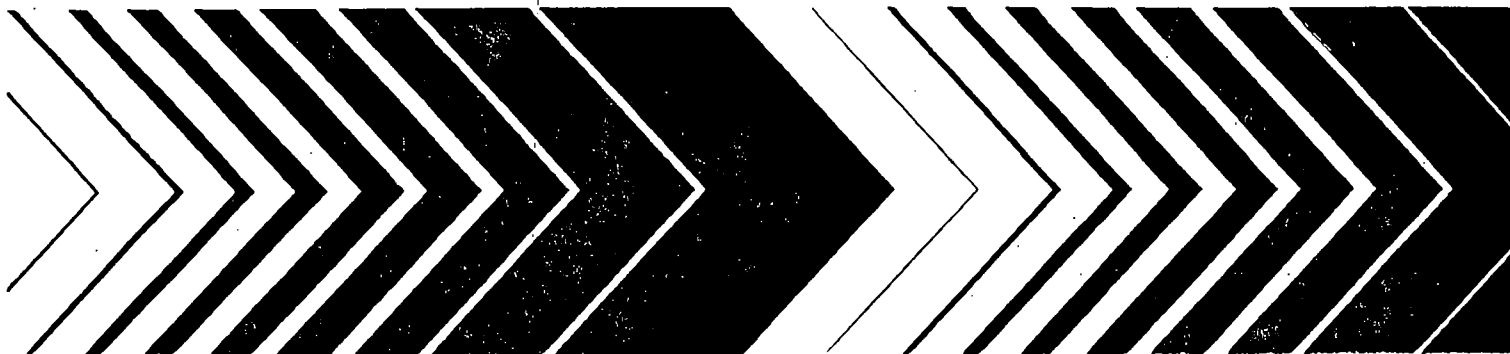
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Chapter 1. Executive Summary

(Do Not
Quote or Cite)

NOTICE

This document is a preliminary draft. It has not been formally released by EPA and should not at this stage be construed to represent Agency policy. It is being circulated for comment on its technical accuracy and policy implications.



Air Quality Criteria for Ozone and Related Photochemical Oxidants

Chapter 1. Executive Summary

and

Chapter 9. Integrative Summary of Ozone Health Effects

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Environmental Criteria and Assessment Office
Office of Health and Environmental Assessment
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PREFACE

In 1971, the U.S. Environmental Protection Agency (EPA) promulgated National Ambient Air Quality Standards (NAAQS) to protect the public health and welfare from adverse effects of photochemical oxidants. In 1979, the chemical designation of the standards was changed from photochemical oxidants to ozone (O_3). This document, therefore, focuses primarily on the scientific air quality criteria for O_3 and, to a lesser extent, for other photochemical oxidants like hydrogen peroxide and the peroxyacyl nitrates.

The EPA promulgates the NAAQS on the basis of scientific information contained in air quality criteria documents. The previous O_3 criteria document, *Air Quality Criteria for Ozone and Other Photochemical Oxidants*, was released in August 1986 and a supplement, *Summary of Selected New Information on Effects of Ozone on Health and Vegetation*, was released in January 1992. These documents were used as the basis for a March 1993 decision by EPA not to revise the existing 1-h NAAQS for O_3 . That decision, however, did not take into account some of the newer scientific data that became available after the 1986 criteria document. This revised air quality criteria document for O_3 and related photochemical oxidants critically evaluates and assesses the latest scientific data associated with exposure to concentrations of these pollutants found in ambient air. Emphasis is placed on presentation of health and environmental effects data; however, other scientific data are presented and evaluated in order to provide a better understanding of the nature, sources, distribution, measurement, and concentrations of O_3 and related photochemical oxidants and their precursors in the environment. The document is not an exhaustive literature review; rather it assesses the most pertinent literature available through 1993.

The chapters in this volume summarize and interpret key information drawn from the other, more detailed chapters (Chapters 2 through 8) of the present document, which were prepared and peer reviewed by experts from various Federal and State governmental offices, academia, and private industry for use by EPA to support decision making regarding potential risks to public health and welfare. The Environmental Criteria and Assessment Office of EPA's Office of Health and Environmental Assessment acknowledges with appreciation the contributions provided by these authors and reviewers as well as the diligence of its staff and contractors in the preparation of this document at the request of the Office of Air Quality Planning and Standards.

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1. EXECUTIVE SUMMARY

1.1 INTRODUCTION

The external review draft document *Air Quality Criteria for Ozone and Related Photochemical Oxidants* evaluates the latest scientific information useful in deriving criteria that form the scientific basis for U.S. Environmental Protection Agency (EPA) decisions regarding the National Ambient Air Quality Standards (NAAQS) for ozone (O₃). This Executive Summary concisely summarizes key conclusions from the document, which comprises nine chapters. Following this Executive Summary is a brief Introduction (Chapter 2) containing information on the legislative and regulatory background for review of the O₃ NAAQS. Chapter 3 provides information on the chemistry, sources, emissions, measurement, and transport of O₃ and related photochemical oxidants and their precursors, whereas Chapter 4 covers environmental concentrations, patterns, and exposure estimates of O₃ and oxidants. This is followed by Chapter 5, dealing with the environmental effects; Chapters 6, 7, and 8 discuss, respectively, animal toxicological studies, human health effects, and the extrapolation of animal toxicological data to humans. The last chapter, Chapter 9, provides an integrative, interpretative evaluation of health effects associated with exposure to O₃. The following subsections follow the chapter organization of the external review draft.

1.2 LEGISLATIVE AND REGULATORY BACKGROUND

The EPA is required under Sections 108 and 109 of the Clean Air Act to periodically evaluate the air quality criteria that reflect the latest scientific information relevant to review of the O₃ NAAQS. These air quality criteria, contained in the criteria document, are useful for indicating the kind and extent of all identifiable effects on public health or welfare that may be expected from the presence of O₃ and related photochemical oxidants in ambient air. The last criteria document was released in 1986, and a supplement was released in 1992. These documents were used as the basis for a March 1993 decision by EPA to not revise the existing 1-h NAAQS for O₃ at that time. That decision, however, did not take into consideration more recent scientific information that has been published since the last literature review in early 1989. The purpose of this revised criteria document, therefore, is to summarize the pertinent information contained in the previous O₃ criteria document and to critically evaluate and assess the more recent scientific data associated with exposure to the concentrations of O₃ and related photochemical oxidants that are found in ambient air.

1.3 TROPOSPHERIC OZONE AND ITS PRECURSORS

Tropospheric Ozone Chemistry

Ozone is found in the stratosphere, the "free" troposphere, and the planetary boundary layer (PBL) of the earth's atmosphere. Ozone occurs in the stratosphere as the result of chemical reactions initiated by short-wavelength radiation from the sun. In the "free"

1 troposphere, O_3 occurs as the result of incursions from the stratosphere; upward venting
2 from the PBL (the layer next to the earth's surface) through certain cloud processes; and
3 photochemical formation from precursors, notably methane (CH_4), carbon monoxide (CO),
4 and nitrogen oxides (NO_x). In the PBL, O_3 occurs as the result of downward mixing from
5 the stratosphere and free troposphere and as the result of photochemical processes occurring
6 within the PBL.
7

8 The photochemical production of O_3 and other oxidants found at the earth's surface
9 (in the PBL, troposphere, or ambient air, used interchangeably in this summary) is the result
10 of atmospheric physical processes and complex, nonlinear chemical processes involving two
11 classes of precursor pollutants, reactive volatile organic compounds (VOCs) and NO_x . The
12 only significant initiator of the photochemical production of O_3 in the polluted troposphere is
13 the photolysis of nitrogen dioxide (NO_2), yielding nitric oxide (NO) and a ground-state
14 oxygen atom that reacts with molecular oxygen to form O_3 . The O_3 thus formed reacts with
15 NO, yielding O_2 and NO_2 . These cyclic reactions attain equilibrium in the absence of
16 VOCs. In the presence, however, of VOCs, which are abundant in polluted ambient air, the
17 equilibrium is upset, resulting in a net increase in O_3 . Methane is the chief VOC found in
18 the free troposphere and in relatively "clean" areas of the PBL. The VOCs found in polluted
19 ambient air are more complex and more reactive than CH_4 , but, as with CH_4 , their
20 atmospheric oxidative degradation is initiated through attack on the VOCs by hydroxyl (OH)
21 radicals. As in the CH_4 oxidation cycle, the conversion of NO to NO_2 during the oxidation
22 of VOCs is accompanied by the production of O_3 and the efficient regeneration of the OH
23 radical. The O_3 and peroxyacyl nitrates (PANs) formed in polluted atmospheres increase
24 with the NO_2/NO concentration ratio.
25

26 At night, in the absence of photolysis of reactants, the simultaneous presence of O_3 and
27 NO_2 results in the formation of the nitrate radical, NO_3 . Reactions with NO_3 radicals appear
28 to constitute major sinks for alkenes, cresols, and some other compounds, although alkyl
29 nitrate chemistry is not well characterized.
30

31 Most inorganic gas-phase processes, that is, the nitrogen cycle and its interrelationships
32 with O_3 production, are well understood. The chemistry of the VOCs in ambient air is not
33 as well understood. It is well-known, however, that the chemical loss processes of gas-phase
34 VOCs, with concomitant production of O_3 , include reaction with OH and NO_3 radicals and
35 O_3 , and photolysis. Reaction with the OH radical is the only important atmospheric reaction
36 (loss process) for alkanes, aromatic hydrocarbons, and the higher aldehydes and ketones that
37 lack $>C=C<$ bonds; and the only atmospheric reaction of alcohols and ethers. Photolysis
38 is the major loss process for formaldehyde and acetone. Reactions with OH and NO_3
39 radicals and with O_3 are all important loss processes for alkenes and for carbonyls containing
40 $>C=C<$ bonds.
41

42 Uncertainties in the atmospheric chemistry of the VOCs can affect quantification of the
43 NO-to- NO_2 conversion and of O_3 yields; and can present difficulties in representation of
44 mechanisms, products, and product yields in O_3 air quality models. Major uncertainties in
45 current understanding of the atmospheric chemistry of the VOCs include (1) chemistry of
46 alkyl nitrate formation, (2) products and reaction rates for $>C_4$ alkanes and for branched

1 alkanes, (3) mechanisms and products of alkene-O₃ reactions, and (4) products of aromatic
2 hydrocarbons in both urban and rural atmospheres.

3
4 It should be noted that the atmospheric chemical processes involved in the
5 photooxidation of VOCs and the formation of O₃ and other photochemical oxidants can lead
6 also to the formation of OH radicals and of particulate-phase organic compounds. The OH
7 radicals produced can not only oxidize VOCs but can also react with NO₂ and sulfur dioxide
8 (SO₂) to form nitric and sulfuric acids, respectively, which become incorporated into aerosols
9 as particulate nitrate and sulfate.

10 11 **Meteorological Influences on Ozone Formation and Transport**

12
13 The surface energy (radiation) budget of the earth strongly influences the dynamics of
14 the PBL. The redistribution of energy through the PBL creates thermodynamic conditions
15 that influence vertical mixing. Growing evidence indicates that the strict use of mixing
16 heights in modeling is an oversimplification of the complex processes by which pollutants are
17 redistributed within urban areas; and that it is necessary to treat the turbulent structure of the
18 atmosphere directly and acknowledge the vertical variations in mixing. Energy balances
19 therefore require study so that more realistic simulations can be made of the structure of the
20 PBL.

21
22 Day-to-day variability in O₃ concentrations depends heavily on day-to-day variations in
23 meteorological conditions, such as the degree of mixing that occurs between release of a
24 pollutant or its precursors and their arrival at a receptor; the occurrence of inversion layers
25 (layers in which temperature increases with height above ground level); and the transport of
26 O₃ left overnight in layers aloft and subsequent downward mixing of that O₃ to the surface.

27
28 The transport of O₃ and its precursors beyond the urban scale (≤ 50 km) to neighboring
29 rural and urban areas has been well documented and was described in the 1986 EPA criteria
30 document for O₃. Episodes of high O₃ concentrations in urban areas are often associated
31 with high concentrations of O₃ in the surroundings. Areas of O₃ accumulation are
32 characterized by (1) synoptic-scale subsidence of air in the free troposphere, resulting in
33 development of an elevated inversion layer; (2) relatively low wind speeds associated with
34 the weak horizontal pressure gradient around a surface high pressure system; (3) a lack of
35 cloudiness; and (4) high temperatures.

36
37 Ultraviolet (UV) radiation from the sun plays a key role in initiating the photochemical
38 processes leading to O₃ formation and affects individual photolytic reaction steps. Still, there
39 is little empirical evidence in the literature linking day-to-day variations in observed UV
40 radiation levels with variations in O₃ levels. An association, however, between tropospheric
41 O₃ concentrations and tropospheric temperature has been demonstrated. Empirical data from
42 four urban areas, for example, show an apparent upper bound on O₃ concentrations that
43 increases with temperature. A similar qualitative relationship exists at a number of rural
44 locations.

1 The relationship between wind speed and O₃ buildup varies from one part of the
2 country to another.

3
4 Statistical techniques (e.g., regression techniques) can be used to help identify real
5 trends in O₃ concentrations, both intra- and interannual, by normalizing meteorological
6 variability. In the Southern Oxidant Study, for example, regression techniques were
7 successfully used to forecast O₃ levels to ensure that specialized measurements were made on
8 appropriate days.

9 10 **Precursors**

11 12 *Nitrogen Oxides Emissions*

13
14 Anthropogenic NO_x is associated with combustion processes. The primary pollutant
15 emitted is NO formed at high combustion temperatures from the nitrogen and oxygen in air
16 and from nitrogen in the combustion fuel. Emissions of NO_x in 1991 in the United States
17 totaled 21.39 Tg. The two largest single NO_x emission sources are electric power generation
18 and highway vehicles. Because a large proportion of anthropogenic NO_x emissions come
19 from distinct point sources, published annual estimates are thought to be very reliable.

20
21 Natural NO_x sources include stratospheric intrusion, oceans, lightning, soils, and
22 wildfires. Lightning and soil emissions are the only two significant natural sources of NO_x
23 in the United States. Combined natural sources contribute about 2.2 Tg of NO_x to the
24 troposphere over the continental United States. Uncertainties in natural NO_x inventories are
25 much larger, however, than for anthropogenic NO_x emissions.

26 27 *Volatile Organic Compound Emissions*

28
29 Hundreds of VOCs, commonly containing from 2 to about 12 carbon atoms, are
30 emitted by evaporative and combustion processes from a large number of source types.
31 Total U.S. VOC emissions in 1991 were estimated at 21.0 Tg. The two largest source
32 categories were industrial processes (10.0 Tg) and transportation (7.9 Tg). Emissions of
33 VOCs from highway vehicles accounted for almost 75% of the transportation-related
34 emissions; studies have shown that the majority of these VOC emissions come from about
35 20% of the automobiles in service, many, perhaps most, of which are older cars that are
36 poorly maintained. The accuracy of VOC emission estimates is difficult to determine, both
37 for stationary and mobile sources.

38
39 Vegetation emits significant quantities of VOCs into the atmosphere, chiefly
40 monoterpenes and isoprene, but also oxygenated VOCs, according to recent studies. The
41 most recent biogenic VOC emissions estimate for the United States showed annual emissions
42 of 29.1 Tg/year.

43
44 Uncertainties in both biogenic and anthropogenic VOC emission inventories prevent
45 establishing the relative contributions of these two categories.

Concentrations of Volatile Organic Compounds in Ambient Air

The VOCs most frequently analyzed in ambient air are the nonmethane hydrocarbons (NMHCs). Morning concentrations (6:00 a.m. to 9:00 a.m.) have been measured most often because of the use of morning data in the Empirical Kinetics Modeling Approach (EKMA) and in air quality simulation models. Measurements made in 22 cities in 1984 and 19 cities in 1985 showed median NMHC concentrations ranging from 0.39 ppm C to 1.27 ppm C for 1984; and from 0.38 ppm C to 1.63 ppm C in 1985. Overall median values from all urban sites were about 0.72 ppm C in 1984 and 0.60 ppm C in 1985.

Concurrent measurements of anthropogenic and biogenic NMHCs have shown that biogenic NMHCs usually constituted much less than 10% of the total NMHCs. For example, average isoprene concentrations ranged from 0.001 to 0.020 ppm C and terpenes from 0.001 to 0.030 ppm C.

Concentrations of Nitrogen Oxides in Ambient Air

Measurements of NO_x made in 22 and 19 U.S. cities in 1984 and 1985, respectively, showed median 6:00-to-9:00 a.m. NO_x concentrations ranging from 0.02 to 0.08 ppm in most of these cities. Nonurban NO_x concentrations, reported as average seasonal or annual NO_x, range from <0.005 to 0.015 ppm.

Ratios of 6:00-to-9:00 a.m. nonmethane organic compounds (NMOC) to NO_x are higher in southeastern and southwestern U.S. cities than in northeastern and midwestern U.S. cities, according to data from EPA's multicity studies conducted in 1984 and 1985. Median ratios ranged from 9.1 to 37.7 in 1984; in 1985, median ratios ranged from 6.5 to 53.2 in the cities studied. Rural NMOC/NO_x ratios tend to be higher than urban ratios. Morning (6:00-to-9:00 a.m.) NMOC/NO_x ratios are used in the EKMA type of trajectory model. The correlation of NMOC/NO_x ratios with maximum 1-h O₃ concentrations, however, was weak in a recent analysis.

Source Apportionment and Reconciliation

Source apportionment (now regarded as synonymous with receptor modeling) refers to determining the quantitative contributions of various sources of VOCs to ambient air pollutant concentrations. Source reconciliation refers to the comparison of measured ambient VOC concentrations with emissions inventory estimates of VOC source emission rates for the purpose of validating the inventories.

Recent findings showed that vehicle exhaust was the dominant contributor to ambient VOCs in seven of eight U.S. cities studied. Whole gasoline contributions have been estimated to be equal to vehicle exhaust in one study and 20% of vehicle exhaust in a second study.

Estimates of biogenic VOCs at a downtown site in Atlanta in 1990 indicated a lower limit of 2% (24-h average) for the biogenic percentage of total ambient VOCs at that location (isoprene was used as the biogenic indicator species). The percentage varies during the 24-h

1 period because of the diurnal (e.g., temperature, light intensity) dependence of isoprene
2 concentrations.

3
4 Source reconciliation data have shown disparities between emission inventory estimates
5 and receptor-estimated contributions. For biogenics, emission estimates are greater than
6 receptor-estimated contributions. The reverse has been true for natural gas contributions
7 estimated for Los Angeles, Columbus, and Atlanta; and for refinery emissions in Chicago.
8

9 **Analytical Methods for Oxidants and Their Precursors**

10 ***Oxidants***

11
12
13 Current methods used to measure O_3 are chemiluminescence (CL); UV absorption
14 spectrometry; and newly developed spectroscopic and chemical approaches, including
15 chemical approaches applied to passive sampling devices (PSDs) for O_3 .
16

17 The CL method has been designated as the reference method by EPA. Detection limits
18 of 0.005 ppm and a response time of < 30 s are typical of currently available commercial
19 instruments. A positive interference from atmospheric water vapor was reported in the
20 1970s and has recently been confirmed. Proper calibration can minimize this source of
21 error.
22

23 Commercial UV photometers for measuring O_3 have detection limits of about
24 0.005 ppm and a response time of < 1 min. Because the measurement is absolute, UV
25 photometry is also used to calibrate O_3 methods. A potential disadvantage of UV photometry
26 is that atmospheric constituents that absorb 254 nm radiation, the wavelength at which O_3 is
27 measured, will cause a positive interference in O_3 measurements. Interferences have been
28 reported in two recent studies, but assessment of the potential importance of such
29 interferences (e.g., toluene, styrene, cresols, nitrocresols) is hindered by lack of absorption
30 spectra data in the 250 nm range and by lack of aerometric data for the potentially interfering
31 species.
32

33 Calibration of O_3 measurement methods (other than PSDs) is done by UV spectrometry
34 or by gas-phase titration (GPT) of O_3 with NO. Ultraviolet photometry is the reference
35 calibration method approved by EPA. Ozone is unstable and must be generated in situ at
36 time of use to produce calibration mixtures.
37

38 Peroxyacetyl nitrate and the higher PANs are normally measured by gas
39 chromatography using an electron capture detector (GC-ECD). Detection limits have now
40 been extended to 1 to 5 ppt. The preparation of reliable calibration standards is difficult
41 because PAN is unstable (explosive and subject to surface-related decomposition), but several
42 methods are available.
43
44

Volatile Organic Compounds

Traditionally, NMHCs and other NMOCs have been measured by methods that employ a flame ionization detector (FID) as the sensing element that measures a change in ion intensity resulting from the combustion of air containing organic compounds. The method recommended by EPA for total NMOC measurement involves the cryogenic preconcentration of NMOCs and the measurement of the revolatilized NMOCs using FID. The main technique for speciated NMOC/NMHC measurements is cryogenic preconcentration followed by GC-FID. Systems for sampling and analysis of VOCs have now been developed that require no liquid cryogen for operation, yet provide sufficient resolution of species.

Stainless steel canisters have become the containers of choice for collection of whole-air samples for NMHC/NMOC data. Calibration procedures for NMOC instrumentation require the generation, by static or dynamic systems, of dilute mixtures at concentrations expected to occur in ambient air.

Preferred methods for measuring carbonyl species (aldehydes and ketones) in ambient air are spectroscopic methods; on-line colorimetric methods; and the most common method in current use for measuring gas-phase carbonyl compounds in ambient air, which is the high-performance liquid chromatography (HPLC) method employing 2,4-dinitrophenylhydrazine (DNPH) derivatization in a silica gel cartridge. Use of an O₃ scrubber has been recommended to prevent interference in this method by O₃ in ambient air. Several methods are available for preparing stable calibration mixtures.

Oxides of Nitrogen

Nitric oxide and NO₂ comprise the NO_x compounds involved as precursors to O₃ and other photochemical oxidants.

The most common method of NO measurement is the gas-phase CL reaction with O₃, which is essentially specific for NO. Commercial NO monitors have detection limits of a few parts per billion by volume (ppbv) in ambient air but may not have sensitivity sufficient for surface measurements in rural or remote areas, or for airborne measurements. Direct spectroscopic methods for NO exist that have very high sensitivity and selectivity for NO, but their complexity, size, and cost restrict these methods to research applications. No PSDs presently exist for measurement of NO.

Chemiluminescence analyzers are the method of choice for NO₂ measurement, even though they do not measure NO₂ directly. Minimum detection levels for NO₂ have been reported to be 5 to 13 ppb, but more recent evaluations have indicated detection limits of 0.5 to 1 ppbv. Reduction of NO₂ to NO is required for measurement. In practice, selective measurement of NO_x by this approach has proved difficult, and the NO₂ value inferred from such measurements may be significantly in error.

Several spectroscopic approaches to NO₂ detection have been developed but share the drawbacks of spectroscopic NO methods. Passive samplers for NO₂ exist, but are still in the developmental stage for ambient air monitoring.

1 Calibration of methods for NO measurement is done using standard cylinders of NO in
2 nitrogen. Calibration of methods for NO₂ measurement include use of cylinders of NO₂ in
3 nitrogen or air, use of permeation tubes, and GPT.

4 5 **Ozone Air Quality Models**

6 7 *Models and Their Components*

8
9 Photochemical air quality models are used to predict how O₃ concentrations change in
10 response to prescribed changes in source emissions of NO_x and VOCs. They operate on sets
11 of input data that characterize the emissions, topography, and meteorology of a region and
12 produce outputs that describe air quality in that region.

13
14 Two kinds of photochemical models are recommended in guidelines issued by EPA:
15 (1) the use of EKMA is acceptable under certain circumstances, and (2) the grid-based Urban
16 Airshed Model (UAM) is recommended for modeling O₃ over urban areas. The 1990 Clean
17 Air Act Amendments mandate the use of three-dimensional (grid-based) air quality models
18 such as UAM in developing State Implementation Plans for areas designated as extreme,
19 severe, serious, or multistate moderate. General descriptions of EKMA and of grid-based
20 models were given in the 1986 EPA criteria document for O₃.

21
22 The EKMA-based method for determining O₃ control strategies has some limitations,
23 the most serious of which is that predicted emissions reductions are critically dependent on
24 the initial NMHC/NO_x ratio used in the calculations. This ratio cannot be determined with
25 any certainty and is expected to be quite variable in an urban area.

26
27 Grid-based, photochemical air quality models have their limitations as well. These are
28 pointed out subsequently.

29
30 Spatial and temporal characteristics of VOC and NO_x emissions are major inputs to a
31 grid-based photochemical air quality model. Greater accuracy in emissions inventories is
32 needed for biogenics and for both mobile and stationary source components. Grid-based air
33 quality models also require as input the three-dimensional wind field for the photochemical
34 episode being simulated.

35
36 A chemical kinetic mechanism (a set of chemical reactions), representing the important
37 reactions that occur in the atmosphere, is used in an air quality model to estimate the net rate
38 of formation of each pollutant simulated as a function of time.

39
40 Dry deposition is an important removal process for O₃ on both urban and regional
41 scales and is included in all urban- and regional-scale models. Wet deposition is generally
42 not included in urban-scale photochemical models, since O₃ episodes do not occur during
43 periods of significant clouds or rain.

44
45 Concentration fields of all species computed by the model must be specified at the
46 beginning of the simulation ("initial conditions"). These initial conditions are determined

1 mainly with ambient measurements, either from routinely collected data or from special
2 studies; but interpolation can be used to distribute the surface ambient measurements.

4 *Use of Ozone Air Quality Models*

6 Photochemical air quality models are used for control strategy evaluation by first
7 demonstrating that a past episode, or episodes, can be adequately simulated and then
8 reducing hydrocarbon or NO_x emissions or both in the model inputs and assessing the effects
9 of these reductions on O₃ in the region. The adequacy of control strategies based on grid-
10 based models depends in part on the nature of input data for simulations and model
11 validation, on input emissions inventory data, and on the mismatch between model output
12 and the current form of the NAAQS for O₃. Uncertainties in models can obviously affect
13 their outputs. Uncertainties exist in all components of grid-based O₃ air quality models:
14 emissions, meteorological modules, chemical mechanisms, deposition rates, and
15 determination of initial conditions.

17 Grid-based models that have been widely used to evaluate control strategies for O₃ or
18 acid deposition, or both, are (1) the Urban Airshed Model (UAM), (2) the California
19 Institute of Technology/Carnegie Institute of Technology (CIT) model, (3) the Regional
20 Oxidant Model (ROM), (4) the Acid Deposition and Oxidant Model (ADOM), and (5) the
21 Regional Acid Deposition Model (RADM).

23 *Conclusions*

25 Urban air quality models are becoming readily available for application and have been
26 applied in recent years in several urban areas. Significant progress has also been made in the
27 development of regional models and the integration of state-of-the-art prognostic
28 meteorological models as drivers.

30 Although there are still many uncertainties in photochemical air quality modeling, prime
31 among which are emission inventories, models are nevertheless essential for regulatory
32 analysis and constitute one of the major tools for attacking the O₃ problem. Grid-based
33 O₃ air quality modeling is superior to the available alternatives for O₃ control planning, but
34 the chances of its incorrect use must be minimized.

37 **1.4 ENVIRONMENTAL CONCENTRATIONS, PATTERNS, AND** 38 **EXPOSURE ESTIMATES**

40 Ozone is measured at concentrations above the minimum detectable level at all
41 monitoring locations in the world. In this section of the document, hourly average
42 concentration and exposure information was summarized for urban, rural forested, and rural
43 agricultural areas in the United States.

45 Because O₃ produced from urban area emissions is transported to more rural downwind
46 locations, elevated O₃ concentrations can occur at considerable distances from urban centers.

1 Urban O₃ concentration values are often depressed because of titration by NO. Because of
2 the absence of chemical scavenging, O₃ tends to persist longer in nonurban than in urban
3 areas and exposures may be higher than in urban locations.
4

5 Trends

6

7 Ozone hourly average concentrations have been recorded for many years by the state
8 and local air pollution agencies who report their data to the EPA. The 10-year (1983 to
9 1992) composite average trend for the second highest daily maximum hourly average
10 concentration during the O₃ season shows that the 1992 composite average for the trend sites
11 is 21 % lower than the 1983 average. The 1992 value is the lowest composite average of the
12 past 10 years. The 1992 composite average is significantly less than all the previous nine
13 years, 1983 to 1991. The relatively high O₃ concentrations in 1983 and 1988 were likely
14 attributable in part to hot, dry stagnant conditions in some areas of the country that were
15 especially conducive to O₃ formation.
16

17 Between 1991 and 1992, the composite mean of the second highest daily maximum
18 1-h O₃ concentrations decreased 7% and the composite average of the number of estimated
19 exceedances of the O₃ standard decreased by 23%. Nationwide VOC emissions decreased
20 3% between 1991 and 1992. The composite average of the second daily maximum
21 concentrations decreased in 8 of the 10 EPA Regions between 1991 and 1992, and remained
22 unchanged in Region VII. Except for Region VII, the 1992 regional composite means are
23 lower than the corresponding 1990 levels.
24

25 Surface Concentrations

26

27 Published data provides evidence showing the occurrence, at some sites, of multihour
28 periods within a day of O₃ at levels of potential health effects. Although most of these
29 analyses were made using monitoring data collected from sites in or near nonattainment
30 areas, in one analysis of five sites (two in New York state, two in rural California, and one
31 in rural Oklahoma), none of which was in or near a nonattainment area, O₃ concentrations
32 showed only moderate peaks but showed multihour levels above 0.10 ppm.
33

34 On the basis of O₃ data from isolated monitoring sites, the EPA has indicated that a
35 reasonable estimate of natural O₃ background concentration near sea level in the United
36 States today, for an annual average, is from 0.020 to 0.035 ppm. This estimate included a
37 0.010- to 0.015-ppm contribution from the stratosphere and a 0.01-ppm contribution from
38 photochemically affected biogenic NMHCs. An additional 0.010 ppm is possible from the
39 photochemical reaction of biogenic methane. The EPA concluded that a reasonable estimate
40 of natural O₃ background concentration for a 1-h daily maximum at sea level in the United
41 States during the summer is on the order of 0.03 to 0.05 ppm. This estimate may be low,
42 however, because available data from sites appearing to be isolated from anthropogenic
43 sources in the western United States indicate that maximum hourly concentrations can reach
44 0.06 and 0.075 ppm with infrequent occurrences below 0.02 ppm (i.e., lack of scavenging).
45
46

Diurnal Variations

Diurnal patterns of O_3 may be expected to vary with location, depending on the balance among the many factors affecting O_3 formation, transport, and destruction. Although they vary with locality, diurnal patterns for O_3 typically show a rise in concentration from low or levels near minimum detectable amounts to an early afternoon peak. The diurnal pattern of concentrations can be ascribed to three simultaneous processes: (1) downward transport of O_3 from layers aloft, (2) destruction of O_3 through contact with surfaces and through reaction with NO at ground level, and (3) in situ photochemical production of O_3 .

Seasonal Patterns

Seasonal variations in O_3 concentrations in urban areas usually show the pattern of high O_3 in late spring or in summer and low levels in the winter; however, weather conditions in a given year may be more favorable for the formation of O_3 and other oxidants than during the prior or following year.

Average O_3 concentrations tend to be higher in the second versus the third quarter of the year for many isolated rural sites. This observation has been attributed to either stratospheric intrusions or an increasing frequency of slow-moving, high-pressure systems that promote the formation of O_3 . However, for several clean rural sites, the highest exposures have occurred in the third quarter rather than in the second. For rural O_3 sites in the southeastern United States, the daily maximum 1-h average concentration was found to peak during the summer months.

Spatial Variations

Concentrations of O_3 vary with altitude and with latitude. There appears to be no consistent conclusion concerning the relationship between O_3 exposure and elevation.

Indoor Ozone

Until the early 1970s, very little was known about the O_3 concentrations experienced inside buildings, and to date, the database on this subject is not large and a wide range of indoor/outdoor O_3 concentration relationships can be found in the literature; reported indoor/outdoor values for O_3 are highly variable. Indoor/outdoor O_3 concentration ratios generally fall in the range from 0.1 to 0.7 and indoor concentrations of O_3 will almost invariably be less than outdoors.

Estimating Exposure

Both fixed-site monitoring information and human exposure models are used to estimate risks associated with O_3 exposure. Because, for most cases, it is not possible to estimate population exposure solely from fixed-station data, several human exposure models have been developed. Some of these models include information on human activity patterns (i.e., the microenvironments people visit and the times they spend there). These models also

1 contain submodels depicting the sources and concentrations likely to be found in each
2 microenvironment, including indoor, outdoor, and in-transit settings.

3
4 Few data are available for individuals using personal exposure monitors. Results from
5 a pilot study demonstrated that fixed-site ambient measurements may not adequately represent
6 individual exposures. Models based on time-weighted indoor and outdoor concentrations
7 explained only 40% of the variability in personal exposures.

8
9 Two distinct types of O₃ exposure models exist: those that focus narrowly on predicting
10 indoor O₃ levels and those that focus on predicting O₃ exposures on a community-wide basis.

11 Peroxyacyl Nitrates

12
13 Peroxyacetyl nitrate and peroxypropionyl nitrate (PPN) are the most abundant of the
14 non-O₃ oxidants in ambient air in the United States, other than the inorganic nitrogenous
15 oxidants such as NO₂, and possibly nitric acid (HNO₃). The concentrations of PAN that are
16 of most concern are those to which vegetation could potentially be exposed, especially during
17 daylight hours in agricultural areas. Most of the available data on concentrations of PAN
18 and PPN in ambient air are from urban areas. The levels to be found in nonurban areas will
19 be highly dependent upon the transport of PAN and PPN or their precursors from urban
20 areas because the concentrations of the NO_x precursors to these compounds are considerably
21 lower in nonurban than in urban areas.

22 Co-occurrence

23
24 Studies of the joint occurrence of gaseous NO₂/O₃ and SO₂/O₃ at rural sites have
25 concluded that the periods of co-occurrence represent a small portion of the potential plant
26 growing period. For human ambient exposure considerations, in most cases, the
27 simultaneous co-occurrence of NO₂/O₃ and SO₂/O₃ was infrequent. Some researchers have
28 reported the joint occurrence of O₃, nitrogen, and sulfur in forested areas, combining
29 cumulative exposures of O₃ with data on dry deposition of sulfur and nitrogen. One study
30 reported that several forest landscapes with the highest dry deposition loadings of sulfur and
31 nitrogen tended to experience the highest average O₃ concentrations and largest cumulative
32 exposure. Although the authors concluded that the joint concentrations of multiple pollutants
33 in forest landscapes were important, nothing was mentioned about the hourly co-occurrences
34 of O₃ and SO₂ or O₃ and NO₂. Acid sulfates, which are usually composed of sulfuric acid,
35 ammonium bisulfate, and ammonium sulfate, have been measured at a number of locations in
36 North America. The potential for O₃ and acidic sulfate aerosols to co-occur at some
37 locations in some form (i.e., simultaneously, sequentially, or complex-sequentially) is real
38 and requires further characterization. For human ambient exposures, the simultaneous
39 co-occurrence of NO₂ and O₃ was infrequent.

40
41 In one study, the relationship between O₃ and hydrogen ions in precipitation was
42 explored using data from sites that monitored both O₃ and wet deposition simultaneously and
43 within one minute latitude and longitude of each other. It was reported that individual sites
44 experienced years in which both hydrogen ion deposition and total O₃ exposure were at least
45
46

1 moderately high. With data compiled from all sites, it was found that relatively acidic
2 precipitation occurred together with relatively high O₃ levels approximately 20% of the time,
3 and highly acidic precipitation occurred together with a high O₃ level approximately 6% of
4 the time. Sites most subject to relatively high levels of both hydrogen ions and O₃ were
5 located in the eastern portion of the United States, often in mountainous areas.
6

7 The co-occurrence of O₃ and acidic cloudwater in high-elevation forests has been
8 characterized. The frequent O₃-only and pH-only single-pollutant episodes, as well as the
9 simultaneous and sequential co-occurrences of O₃ and acidic cloudwater, have been reported.
10 Both simultaneous and sequential co-occurrences were observed a few times each month
11 above cloud base.
12

13 14 **1.5 ENVIRONMENTAL EFFECTS OF OZONE AND RELATED** 15 **PHOTOCHEMICAL OXIDANTS** 16

17 Ozone is the gaseous pollutant most injurious to agricultural crops, trees, and native
18 vegetation. Exposure of vegetation to O₃ can inhibit photosynthesis, alter carbon
19 (carbohydrate) allocation, and interfere with mycorrhizal formation in tree roots. Disruption
20 of these important physiological processes can suppress the growth of crops, trees, shrubs,
21 and herbaceous vegetation by decreasing their capacity to form the carbon (energy)
22 compounds needed for growth and maintenance and their ability to absorb the water and
23 mineral nutrients they require from the soil. In addition, loss of vigor increases
24 susceptibility of trees and crops to insects and pathogens and impairs their ability to
25 reproduce. The following section summarizes key environmental effects associated with
26 O₃ exposure.
27

28 **Effects on Agroecosystems** 29

30 *Methodologies Used in Vegetation Research* 31

32 Most of the knowledge concerning the effects of O₃ on vegetation comes from the
33 exposure-response studies of important agricultural crop plants and some selected forest and
34 urban tree species, mostly as seedlings. A variety of methodologies have been used, ranging
35 from field exposures without chambers to open-top chambers and to exposures conducted in
36 chambers under highly controlled climates. In general, the more controlled conditions are
37 most appropriate for investigating specific responses and for providing the scientific basis for
38 interpreting and extrapolating results.
39

40 *Mode of Action* 41

42 Photosynthesis provides plants with the energy and structural building blocks necessary
43 for their existence. The photosynthetic capacity of a plant (i.e., carbohydrate production) is
44 an important aspect of plant response to stresses in natural environments and is strongly
45 associated with leaf nitrogen content and with water movement. Continued acquisition of
46 nitrogen and water uptake are necessary, if photosynthesis is to occur, and involves the

1 allocation of carbohydrates from the leaves to the roots. Leaf photosynthetic capacity is age
2 dependent. As the plant grows, the canopy structure changes, altering the amount and angle
3 of light hitting a leaf. Allocation of carbohydrates and nutrients to new leaves is especially
4 important in stimulating growth production.

5
6 Ozone exposure results in a leaf-mediated plant response. Leaves are important
7 regulators of plant stress response and function. As the major regulators of anatomical and
8 morphological development of the shoot, they control the allocation of carbohydrates to the
9 whole plant. The many regulatory systems contained in leaves change both as a function of
10 leaf development and in response to different environmental stresses.

11
12 Only O₃ that enters the plant through stomata (openings in the leaves) can impair plant
13 processes. In addition, an effect will occur only if sufficient O₃ reaches sensitive sites within
14 leaf cells. Ozone injury will not be detected if (1) the rate of uptake is small enough for the
15 plant to detoxify or metabolize O₃ or its derivatives, or (2) the plant is able to repair or
16 compensate for its impacts at a rate equal to or greater than the rate of uptake. (If the rate
17 of repair is very slow, there may be an effect.)

18
19 The uptake and movement of O₃ to sensitive cellular sites are subject to various
20 biochemical and physiological controls. The magnitude of O₃-induced effects depends upon
21 the physical environment of the plant, including both macro-and microclimatic factors, and
22 the chemical environment of the plant, including other gaseous pollutants and biological
23 factors. Visible foliar injury is usually the first indication of cellular plant response. Once
24 sufficient O₃ enters the leaf, the plant can experience reduced photosynthesis, altered
25 carbohydrate production and allocation, reduced plant vigor, reduced growth or yield or
26 both, and sometimes death. The alterations in the biochemical and physiological processes
27 mentioned above may occur with or without visible injury to the plant.

28
29 Reductions in photosynthesis caused by O₃ are likely to be accompanied by a shift in
30 growth pattern that favors shoots. Plants compensate for differences in resource imbalances
31 by allocating energy, water, and minerals to maintain optimal growth. Allocation of these
32 resources to leaf repair or to new leaf formation and an overall reduction in carbohydrate
33 formation decrease the availability of carbohydrates for both stem and root growth.
34 Alteration of the normal allocation pattern affects all aspects of plant growth and
35 reproduction.

36 37 *Factors That Modify Plant Response*

38
39 Plant response to O₃ may be modified by a variety of biological, chemical, and physical
40 factors. Both the impact of environmental factors on plant response to O₃ and the effects of
41 O₃ on the responses of plants to environmental factors have to be considered when
42 determining the impact of oxidants on vegetation in the field.

43
44 Biological factors include those both within and external to the plant. Within the plant,
45 its genetic composition and stage of development play critical roles in plant response to O₃ as
46 well as other stresses. Different varieties or cultivars, as well as different individuals of a

species, due to differences in their genetic composition, are known to differ greatly in their responses to a given O₃ exposure.

The magnitude of the response of a particular species or variety depends on such environmental factors as temperature and humidity, soil moisture and nutrition, exposure to other pollutants or agricultural spray chemicals, and interaction with plant pests and pathogens. In other words, the plant's present and past environmental milieu, which includes the temporal exposure pattern and stage of development, dictates the plant response. The corollary is also true: exposure to O₃ can modify plant response to other environmental variables. For example, exposure to O₃ reduces the ability of trees to withstand winter injury caused by freezing temperatures and influences the success of pest and fungal infections by decreasing tree vigor.

Effects Based Air Quality Exposure Indices

Environmental scientists for many years have attempted to characterize and mathematically represent plant exposures to O₃. A variety of averaging times have been used to characterize exposure-response in plants. Though most studies have characterized exposure by using mean concentrations such as seasonal, monthly, weekly, daily, or peak hourly means, other studies have used cumulative measures (e.g., the number of hours above selected concentrations). None of these statistics completely characterizes the relationships among O₃ concentration, exposure duration, interval between exposures, and plant response.

The use of a mean concentration with long averaging times (1) implies that all concentrations of O₃ are equally effective in causing plant responses, and (2) minimizes the contributions of the peak concentrations to the response. Present evidence suggests that higher concentrations, episodic peak occurrences, and duration of exposure have a relatively greater role in producing plant effects than mean concentrations; therefore, an index that cumulates hourly concentrations during the season and gives greater weight to higher concentrations appears to be a more appropriate index for relating ambient exposures to growth or yield effects. From the toxicological perspective, it is the peak occurrences or concentrations above an unidentified effect level that are most likely to have an impact. Effects on vegetation appear when the amount of pollutant that has entered a plant exceeds its ability to repair or compensate for the impact.

An index of ambient exposure that relates well to plant response should directly or indirectly incorporate both environmental influences, (e.g., temperature, humidity, and soil-moisture status) and exposure dynamics.

No experimental studies have been designed specifically to evaluate the adequacy of the various peak-weighted indices that have been proposed. In retrospective analyses when O₃ is the primary source of variation in response, year-to-year variations in plant response are minimized by peak-weighted, cumulative exposure indices. However, a number of different forms of peak-weighted, cumulative indices have been examined for their ability to properly order yield responses from the large number of studies of the National Crop Loss Assessment Network (NCLAN) program. These exposure indices (i.e., SUM00, SUM06, SIGMOID,

1 and W126) all performed equally well, and it is not possible to distinguish between them on
2 the basis of statistical fits of the data.

3 4 *Exposure-Response of Plant Species*

5
6 The number of crop species/cultivars for which information regarding O₃ exposure
7 responses are available encompasses a mere fraction of the total of cultivated crops and even
8 a smaller fraction of native plants. The emphasis of experimental studies has usually been on
9 the more economically important crop plants and tree species, as seedlings.

10
11 Crop species usually are monocultures that are fertilized and in many cases watered.
12 Therefore, because crop plants are usually grown under optimal conditions, their sensitivity
13 to O₃ exposures undoubtedly varies from that of native trees, shrubs, and herbaceous
14 vegetation.

15
16 The concept of limiting values was used in both the 1978 and 1986 criteria documents
17 to summarize visible foliar injury. Limiting values are defined as concentrations and
18 durations of exposure below which visible injury does not occur. The limit for visible injury
19 indicating reduced plant performance was an O₃ exposure of 0.05 ppm for several hours per
20 day for greater than 16 days. When the exposure period was decreased to 10 days, the
21 O₃ concentration required to cause injury was increased to 0.1 ppm. A short, 6-day
22 exposure further increased the concentration to 0.30 ppm. The exposure and concentration
23 periods apply today for those crops where appearance or aesthetic value (e.g, spinach,
24 cabbage, lettuce) is considered important. Limiting values for foliar injury to trees and
25 shrubs range from 0.06 to 0.1 ppm for 4 h.

26
27 Several conclusions were drawn in the 1986 criteria document from the various
28 experimental approaches used to estimate crop yield loss.

- 29
30 1. Ambient O₃ concentrations are sufficiently elevated in several regions of the country to
31 impair growth and yield of plants. This is clearly indicated by comparison of data
32 obtained from crop yield in charcoal-filtered and unfiltered (ambient) exposures. These
33 elevated levels are further supported by data from studies using chemical protectants.
34 These response data make possible extrapolation to plants not studied experimentally.
35 Both approaches mentioned above indicate that effects occur with only a few exposures
36 above 0.08 ppm.
37
38 2. Several plant species exhibited growth and yield effects when the mean O₃ concentration
39 exceeded 0.05 ppm for 4 to 6 h/day for at least 2 weeks.
40
41 3. Data from regression studies conducted to develop an exposure-response function for
42 estimating yield loss indicated that at least 50% of the species/cultivars tested could be
43 predicted to exhibit a 10% yield loss at 7-h seasonal mean O₃ concentrations of 0.05 ppm
44 or less.

45
46 Based on research published since the 1986 criteria document, the following
47 conclusions can be drawn.

1. Monitoring data from 80 to 200 nonurban sites for a period of 10 years were analyzed to determine the ambient 7-h growing-season average O₃ concentrations. For periods of 3 and 5 mo, concentrations ranged from 0.05 to 0.06 ppm and 0.047 to 0.054 ppm, respectively.
2. Open-top chamber studies comparing yields at ambient O₃ exposures with those in filtered air, and retrospective analyses of crop data, indicate that current ambient O₃ concentrations at some sites in the United States are sufficient to reduce the yield of major crops. These results also indicate that visible injury that reduces the market value of certain crops and ornamentals (spinach, petunia, geranium, for example) occurs at O₃ concentrations ranging from 0.04 to 0.10 ppm for 4 h.
3. A growing season SUMO6 exposure of 26.4 ppm · h, corresponding to a 7-h growing season mean of 0.049 ppm, is estimated to prevent a 10% yield loss from O₃ in 50% of the 54 experimental cases analyzed within this document. It is estimated that a 12-h growing season mean of 0.045 ppm would restrict yield losses to 10% in major crop species.

Effects on Natural Ecosystems

Most of the information dealing with the possible responses of ecosystems to O₃ stress is based on the studies in the San Bernardino Forest ecosystem. This mixed conifer forest ecosystem in southern California has been studied more than any other ecosystem in the United States, over 20 years. Chronic O₃ exposures for a period of 50 or more years have been associated with major changes in the ecosystem.

Data from an inventory conducted from 1968 through 1972 indicated that for 5 mo of the year, trees were exposed to O₃ concentrations greater than 0.08 ppm for more than 1,300 h. Concentrations rarely decreased below 0.05 ppm at night near the crest of the mountain slope, approximately 5,500 ft. In addition, during the years 1973 to 1978, average 24-h O₃ concentrations ranged from a background of 0.03 to 0.04 ppm in the eastern part of the San Bernardino Mountains to a maximum of 0.10 to 0.12 ppm in the western part during May through September. Although PAN and NO₂ were present in these studies, symptoms of PAN injury on forest herb-layer plant species could not be distinguished from those caused by O₃, and NO₂ concentrations were too low to cause injury.

Ecosystem responses to stress are hierarchical. They begin with the response of the most sensitive individuals of a population. Stresses, whose primary effects occur at the molecular level (within the leaves), must be propagated progressively up through more integrated levels of organ physiology (e.g., leaf, branch, root) to whole plant physiology, to populations within the stand (community), and then to the landscape level to produce ecosystem effects.

Therefore, to understand the effects of a stress, one must utilize a framework of hierarchical scales. This is particularly true of low-level stresses because only a small fraction of stresses at the molecular level become disturbances at the tree, stand, or landscape

1 level. Insect defoliation, for example, may severely reduce the growth of one or several
2 branches, whereas growth of the tree appears not to be affected. The time required for a
3 stress to be propagated from one level to the next determines how soon the effects of the
4 stress can be observed or measured.

5
6 Foliar injury is usually the first visible indication of O₃ exposure in plants. In the San
7 Bernardino forest, injury was first observed on ponderosa pine, with Jeffrey pine, white fir,
8 black oak, incense cedar, and sugar pine following in decreasing order of sensitivity. Foliar
9 injury on sensitive ponderosa and Jeffrey pine was observed when 24-h-average
10 O₃ concentrations were 0.05 to 0.06 ppm.

11
12 The biochemical changes within the leaves were expressed as (1) visible foliar injury;
13 (2) premature needle senescence; (3) a reduction in photosynthesis; (4) a reduction in
14 carbohydrate production; (5) altered carbohydrate allocation, reduced plant vigor; and (6) a
15 reduction in growth and reproduction.

16
17 Tree growth is the culmination of biochemical and physiological processes, during
18 which plants use, accumulate, and store carbon compounds (energy) to build and maintain
19 their structure. Within the leaves during the process of photosynthesis, carbon dioxide
20 absorbed from the atmosphere is converted to carbohydrates. The water and minerals
21 necessary for growth are absorbed from the soil through the roots. Growth and seed
22 formation depend not only on photosynthesis and the uptake of water and mineral nutrients,
23 but also on subsequent metabolic processes and the allocation of carbohydrates to the rest of
24 the plant. Impairment of any of these processes can decrease plant vigor and affect plant
25 growth and reproduction.

26
27 Trees each year require energy to grow new leaves and needles, to produce new fine
28 roots, and to increase radial growth. Factors that inhibit photosynthesis and limit
29 carbohydrate formation shift carbohydrate allocation to new leaves, whereas factors that limit
30 nitrogen or water availability will shift allocation to the roots. Increased carbohydrate
31 allocation to new needles and to repair foliage injured by O₃ can place a drain on
32 carbohydrate reserves. Reduced tree vigor, the result of altered carbohydrate allocation,
33 increases susceptibility of trees to insect pests and fungal pathogens and reduces the
34 formation of mycorrhizae required for water and nutrient uptake.

35
36 Premature needle senescence, as observed in ponderosa and Jeffrey pine, not only
37 decreases the amount of foliage available to carry on photosynthesis, but alters
38 microorganismal succession on conifer needles, thus altering the detritus-forming process and
39 subsequent nutrient cycling.

40
41 The primary effect on the more susceptible members of the San Bernardino forest
42 community (e.g., ponderosa and Jeffrey pine) was that they were no longer able to compete
43 effectively for essential nutrients, water, light, and space. As a consequence of altered
44 competitive conditions in the community, there was a decline in the sensitive species,
45 permitting the enhanced growth of more tolerant species. In addition, changes in the
46 dominant individuals in the ecosystem altered the processes of energy flow and nutrient
47 cycling. These changes that began with the biochemical changes within the leaves of the

1 sensitive conifers were ultimately transferred upward to the whole ecosystem, from the
2 individual, to the population, to the community, and finally, to the ecosystem.

3
4 Functional ecosystem changes associated with the deaths in the conifer populations
5 altered the processes of carbohydrate (energy) flow, mineral nutrient cycling, and water
6 movement. The altered processes also changed, either directly or indirectly, the functioning
7 of other living ecosystem components and ultimately changed the vegetational community
8 patterns. The continuum of changes beginning with injury to the individual trees resulted in
9 ecosystem breakdown and a shift in dominance from ponderosa and Jeffrey pine to
10 O₃-tolerant shrub and oak species.

11
12 The effects that a stress at one level of organization will have on a higher level are
13 determined by variability and compensation. Variability in response to stress can mean that
14 not all trees are equally susceptible to O₃, as was observed, in the San Bernardino Forest, in
15 the Appalachian Mountains, and the Cumberland Plateau of eastern Tennessee. Individuals
16 of Ponderosa, Jeffrey, and eastern white pine were designated as being sensitive,
17 intermediate or tolerant, based on their responses to O₃ exposure. Compensation means that
18 plants tolerant to the stress are able either to detoxify or metabolize O₃ entering the leaves or
19 to repair the injury.

20
21 Variability and compensation can also occur at the population level and, all populations
22 do not respond equally. Plant populations can respond in four different ways: (1) no
23 response, the individuals are tolerant to the stress; (2) mortality of all individuals and local
24 extinction of the extremely sensitive population, the most severe response; (3) physiological
25 accomodation, resulting in growth and reproductive success of tolerant individuals; and
26 (4) differential response, some individuals of the population exhibit better growth and
27 reproductive success due to genetically determined traits.

28
29 The properties of variability and compensation at the individual level determine (1) the
30 severity of the response, (2) whether the effect will be propagated to the next level, and
31 (3) the length of time required for the effect to be expressed functionally and structurally. In
32 most instances, the time required for response at the individual level is a period of at least
33 3 to 5 years. Variability and compensation at the population level determine whether there
34 will be an ecosystem response. These properties help to explain the differences in responses
35 between the San Bernardino Forest and the forest ecosystems in the eastern United States.

36
37 Forest stands differ greatly in age, species composition, stability, and capacity to
38 recover from disturbance. In addition, the position in the stand or community of the most
39 sensitive species is extremely important. Ponderosa and Jeffrey pine are controller species.
40 Their removal altered energy flow, nutrient cycling, and water movement in the San
41 Bernardino forest ecosystem and changed the ecosystem. The removal of the eastern white
42 pine, on the other hand, did not alter the functional ecosystem processes sufficiently to bring
43 about a recognizable change within the ecosystems. For this reason, data dealing with the
44 responses of one forest type (e.g., San Bernardino) may not be applicable to other forest
45 types such as those found in the Appalachian Mountains.

1 Individual eastern white pine trees varied in sensitivity to O₃, and, because of
2 physiological accommodation and compensation, effects at the population level were not
3 reported in the forest stands in the Appalachian Mountains. Plantation plantings of eastern
4 white pine (a monoculture) on the Cumberland Plateau, however, did exhibit overall growth
5 reductions.
6

7 Mycorrhizae (fungus roots) function in the uptake of water and minerals (e.g., nitrogen,
8 phosphorus) and are required for optimal growth by most plants. Nitrogen is a critical
9 element in the process of photosynthesis and is usually the element in shortest supply in both
10 agricultural and natural ecosystems. Increased carbohydrate allocation to new needles and to
11 foliage repair limits allocation to the roots and reduces formation of the mycorrhizae needed
12 to take up water, nitrogen, and other nutrients. Mycorrhizae can also protect plants from
13 attack by root pathogens.
14

15 Effects on Materials

16

17 Over four decades of research show that O₃ damages certain materials such as
18 elastomers, textile fibers, and dyes. The amount of damage to actual in-use materials and
19 the economic consequences of that damage are poorly characterized.
20

21 Natural rubber and synthetic polymers of butadiene, isoprene, and styrene, used in
22 products like automobile tires and protective outdoor electrical coverings, account for most
23 of the elastomer production in the United States. The action of O₃ on these compounds is
24 well known, and concentration-response relationships have been established and corroborated
25 by several studies. These relationships, however, must be correlated with adequate exposure
26 information based on product use. For these and other economically important materials,
27 protective measures have been formulated to reduce the rate of oxidative damage. When
28 antioxidants and other protective measures are incorporated in elastomer production, the
29 O₃-induced damage is reduced considerably, although the extent of reduction differs widely
30 according to the material and the type and amount of protective measures used.
31

32 Both the type of dye and the material in which it is incorporated are important factors
33 in the resistance of a fabric to O₃. Some dyed fabrics, such as royal blue rayon-acetate, red
34 rayon-acetate, and plum cotton, are resistant to O₃. On the other hand, anthraquinone dyes
35 on nylon fibers are sensitive to fading from O₃. Field studies and laboratory work show a
36 positive association between O₃ levels and dye fading of nylon materials. At present, the
37 available research is insufficient to quantify the amount of damage to materials attributable to
38 O₃ alone.
39

40 The degradation of fibers from exposure to O₃ is poorly characterized. In general,
41 most synthetic fibers like modacrylic and polyester are relatively resistant, whereas cotton,
42 nylon, and acrylic fibers have greater but varying sensitivities to O₃. Ozone reduces the
43 breaking strength of these fibers, and the degree of reduction depends on the amount of
44 moisture present. The limited research in this area indicates that O₃ in ambient air may have
45 a minimal effect on textile fibers, but additional research is needed to verify this conclusion.
46

1 A number of artists' pigments and dyes are sensitive to O₃ and other oxidants. Many
2 organic pigments in particular are subject to fading or other color changes when exposed to
3 O₃. Although most, but not all, modern fine arts paints are more O₃ resistant, many older
4 works of art are at risk of permanent damage due to O₃-induced fading.
5

6 A great deal of work remains to be done to develop quantitative estimates of the
7 economic damage to materials from photochemical oxidants. Most of the available studies
8 are now outdated in terms of the O₃ concentrations, technologies, and supply-demand
9 relationships that prevailed when the studies were conducted. Additionally, little is known
10 about the physical damage functions, so cost estimates have been simplified to the point of
11 not properly recognizing many of the scientific complexities of the impact of O₃.
12
13

14 1.6 TOXICOLOGICAL EFFECTS OF OZONE

15 Respiratory Tract Effects of Ozone

16 *Biochemical Targets of Ozone Interaction*

17
18 Knowledge of molecular targets provides a basis for understanding mechanisms of
19 effects and strengthening animal-to-human extrapolations. Ozone reacts with polyunsaturated
20 fatty acids and sulfhydryl, amino, and some electron-rich compounds. These elements are
21 shared across species. Several types of reactions are involved, and free radicals may be
22 created. Based on this knowledge, it has been hypothesized that the O₃ molecule is unlikely
23 to penetrate the liquid linings of the respiratory tract to reach the tissue, raising the
24 possibility that reaction products exert effects.
25
26

27 *Lung Inflammation and Permeability Changes*

28
29 Ozone disrupts the barrier function of the lung, resulting in (1) the entry of compounds
30 in the airspaces into the blood and (2) the entry of serum components (e.g., protein) and
31 white blood cells (especially polymorphonuclear leukocytes) into the airspaces and lung
32 tissue. This latter impact reflects the initial stage of inflammation. These cells can release
33 biologically active mediators that are capable of a number of actions, including damage to
34 other cells in the lung. In lung tissue, this inflammation can also increase the thickness of
35 the air-blood barrier.
36
37

38 Increases in permeability and inflammation have been observed at levels as low as
39 0.1 ppm (2 h/day, 6 days; rabbits). After acute exposures, the influence of the time of
40 exposure (from 2 h to several hours) increases as the concentration of O₃ increases.
41 Long-term exposure effects are discussed under lung morphology.
42

43 The impacts of these changes are not fully understood. At higher O₃ concentrations
44 (e.g., 0.7 ppm, 28 days), the diffusion of oxygen into the blood decreases, possibly because
45 the air-blood barrier is thicker; cellular death may result from the enzymes released by the
46 inflammatory cells; and host defense functions may be altered by mediators.

Effects on Host Defense Mechanisms

Ozone exposure results in alterations of all defense mechanisms of the respiratory tract, including mucociliary and alveolobronchiolar clearance, functional and biochemical activity of the alveolar macrophage, and immunologic competence. These effects can cause susceptibility to bacterial respiratory infections.

Mucociliary clearance, which removes particles and cellular debris from the conducting airways, is slowed by acute, but not repeated exposures to O₃. Ciliated epithelial cells that move the mucous blanket are altered or destroyed by acute and chronic exposures. Neonatal sheep exposed to O₃ do not have normal development of the mucociliary system. Such effects could prolong the retention of unwanted substances (e.g., inhaled particles) in the lungs, allowing them to exert their toxic potential for a longer period of time.

Alveolar clearance mechanisms, which center on the functioning of alveolar macrophages, are altered by O₃. Short-term exposure to levels as low as 0.1 ppm (2 h/day, 1 to 4 days; rabbits) accelerates clearance, but longer exposures do not. Even so, after a 6-week exposure of rats to an urban pattern of O₃, the retention of asbestos fibers in a region protected by alveolar clearance is prolonged.

Alveolar macrophages engulf and kill microbes, as well as clear the deeper regions of the lungs of nonviable particles. They also participate in immunological responses, but little is known about the effects of O₃ on this function. Acute exposures of rabbits to levels as low as 0.1 ppm decrease the ability of alveolar macrophages to ingest particles. This effect is displayed in decreases in the ability of the lung to kill bacteria after acute exposure of mice to levels as low as 0.4 ppm.

Both the pulmonary and systemic immune system are affected by O₃, but in a poorly understood way. It appears that the part of the immune system dependent on T-cell function is more affected than that part dependent on B-cell function.

Dysfunction of host defense systems results in enhanced susceptibility to bacterial lung infections. For example, exposure as low as 0.08 ppm for 3 h can overcome the ability of mice to resist infection with streptococcal bacteria, resulting in mortality. However, prolonged exposures (weeks, months) do not cause greater effects on infectivity.

Effects on antiviral defenses are more complex and less well understood. Only high concentrations (1.0 ppm, 3 h/day, 5 days; mice) increase viral-induced mortality. Apparently, O₃ does not impact antiviral clearance mechanisms. Although O₃ does not affect acute lung injury from influenza virus infection, it does enhance later phases of the course of an infection (i.e., postinfluenzal alveolitis).

Morphological Effects

Ozone causes similar types of alterations in lung structure in all laboratory animal species studied, from rats to monkeys. In the lungs, the most affected cells are the ciliated

1 epithelial cells of the airways and Type 1 epithelial cells of the gas exchange region. In the
2 nasal cavity, ciliated cells are also affected.

3
4 The centriacinar region (the junction of the conducting airways and gas exchange
5 regions) is the primary target, possibly because this area is predicted to receive the greatest
6 dose of O₃. The ciliated cells can be killed and are replaced by nonciliated cells (i.e., cells
7 not capable of clearance functions that also have increased ability to metabolize some foreign
8 compounds). Mucous-secreting cells are affected, but to a lesser degree. Type 1 cells,
9 across which gas exchange occurs, can be killed. They are replaced by Type 2 cells, which
10 are thicker and produce more lipids. An inflammatory response also occurs in the tissue.
11 The tissue is thickened further in later stages when collagen (a structural protein increased in
12 fibrosis) and other elements accumulate.

13
14 The distal airway is remodeled. More specifically, bronchiolar epithelium replaces the
15 cells present in alveolar ducts. Concurrent inflammation may play a role. This effect has
16 been observed at 0.25 ppm (8 h/day, 18 mo) in monkeys; at a higher concentration, this
17 remodeling persists after exposure stops.

18
19 The progression of effects during and after a chronic exposure is complex. Over the
20 first few days of exposure, inflammation peaks and then drops considerably, plateauing for
21 the remainder of exposure, after which it largely disappears. Epithelial hyperplasia increases
22 rapidly over the first few days and rises slowly or plateaus thereafter; when exposure ends, it
23 begins to return toward normal. In contrast, fibrotic changes in the tissue between the air
24 and blood increase very slowly over months of exposure, and after exposure ceases, the
25 change sometimes persists or increases further.

26
27 The pattern of exposure can make a major difference in effects. Monkeys exposed to
28 0.25 ppm O₃ (8 h/day) every other month of an 18-mo period had equivalent changes in lung
29 structure, more fibrotic changes, and more of certain types of pulmonary function changes
30 than monkeys exposed every day over the 18 mo. From this work and rat studies, it appears
31 that natural seasonal patterns may be of more concern than more continuous exposures.
32 Thus, long-term animal studies with uninterrupted exposures may underestimate some of the
33 effects of O₃.

34
35 There is no evidence that O₃ causes emphysema.

36 37 *Effects on Pulmonary Function*

38
39 Pulmonary function changes in animals resemble those observed in humans after acute
40 exposure.

41
42 During acute exposure, the most commonly observed alteration is an increased
43 frequency of breathing and decreased tidal volume (i.e., rapid, shallow breathing). This has
44 been reported at exposures as low as 0.2 ppm for 3 h (rats). Typically, higher
45 concentrations (around 1 ppm) are required to affect breathing mechanics (compliance and
46 resistance). Extended characterizations of pulmonary function show types of changes

generally seen in humans. For example, there are decreased lung volumes at levels ≥ 0.5 ppm (a few hours; rats).

When rats are exposed to O_3 for 2 h/day for 5 days, the pattern of attenuation of pulmonary function responses is similar to that observed in humans. Other biochemical indicators of lung injury did not return to control values by Day 5, and morphological changes increased in severity over the period of exposure. Thus, attenuation did not result in protection against all the effects of O_3 .

Long-term exposures have provided mixed results on pulmonary function, including no or minimal effects, restrictive effects, or obstructive effects. When changes occurred and postexposure examinations were performed, pulmonary function recovered.

Biochemical Effects

In acute and short-term exposure studies, a variety of lung lipid changes occur, including an increase in arachidonic acid, the further metabolism of which produces a variety of biologically active mediators that can affect host defenses, lung function, the immune system, and other functions.

The level of lung antioxidant metabolism increases after O_3 exposure, probably as a result of the increase in the number of cells (Type 2 cells) rich in antioxidant enzymes.

Collagen (the structural protein involved in fibrosis) increases in O_3 -exposed lungs in a manner that has been correlated to structural changes (e.g., increased thickness of the tissue between the air and blood after prolonged exposure). Some, but not all, studies found that the increased collagen persists after exposure ceases.

Generally, O_3 enhances lung xenobiotic metabolism after both short- and long-term exposure, possibly as a result of morphological changes (increased numbers of nonciliated bronchiolar epithelial cells). The impact of this change is dependent on the xenobiotics involved. For example, the metabolism of benzo[a]pyrene to active metabolites was enhanced by O_3 .

Genotoxicity and Carcinogenicity of Ozone

The chemical reactivities of O_3 give it the potential to be a genotoxic agent.

In vitro studies are difficult to interpret because the culture systems used allowed the potential formation of artifacts, and often high or very high concentrations of O_3 were used. Generally, in these studies, O_3 causes DNA strand breaks, sometimes is weakly mutagenic, and causes cellular transformation and chromosomal breakage. The latter finding has been investigated in vivo with mixed results in animals. A well-designed human clinical study found no such effect.

The few earlier long-term carcinogenic studies, with or without coexposure to known carcinogens, are either negative or ambiguous.

1 The National Toxicology Program (NTP) completed chronic rat and mouse cancer
2 bioassays using commonly accepted experimental approaches and designs. Both males and
3 females were studied. Animals were exposed for 2 years (6 h/day, 5 days/week) to 0.12,
4 0.5, and 1.0 ppm O₃ or for a lifetime to the same levels (except 0.12 ppm). Following their
5 standard procedures for determination of weight-of-evidence for carcinogenicity, the NTP
6 reported "no evidence" in rats, "equivocal evidence" in male mice, and "some evidence" in
7 female mice. The increases in adenomas and carcinomas were observed only in the lungs.
8 There was no concentration response. One of the reasons for the designation of "some
9 evidence" in female mice was that when the 2-year and lifetime exposure studies were
10 combined, there was a statistically significant increase in total tumors at 1.0 ppm. It is not
11 justified to extrapolate these mouse data to humans at the present time because rats were not
12 affected, only a high level of O₃ (1.0 ppm) caused a limited degree of carcinogenic activity
13 in one strain of mice, there was no concentration response, and there is inadequate
14 information to provide a mechanistic support for the finding in mice.
15

16 In a companion NTP study, male rats were treated with a tobacco carcinogen and
17 exposed for 2 years to 0.5 ppm O₃. Ozone did not affect the response and therefore had no
18 tumor promoting activity.
19

20 Systemic Effects of Ozone

21
22 Ozone causes a variety of effects on tissues/organs distant from the lung. Because
23 O₃ itself is not thought to penetrate the lung, these systemic effects are either secondary to
24 lung alterations or result from reaction products of O₃. Effects have been observed on
25 clinical chemistry, white blood cells, red blood cells, the circulatory system, the liver,
26 endocrine organ(s), and the central nervous system. Most of these effects cannot be
27 adequately interpreted at this time and have not been investigated in humans, but it is of
28 interest to note that O₃ exposures causing effects on the respiratory tract of animals cause a
29 wide array of effects on other organs also.
30

31 Several behavior changes occur in response to O₃. For example, 0.12 ppm (6 h, rats)
32 decreases wheel-running activity, and 0.5 ppm (1 min) causes mice to avoid exposure.
33 These effects are not fully understood, but they may be related to lung irritation or decreased
34 ability to exercise.
35

36 Although cardiovascular effects, such as slowed heart rate and decreased blood
37 pressure, occur in O₃-exposed rats, some observed interactions with thermoregulation prevent
38 qualitative extrapolation of these effects to humans at this time.
39

40 Developmental toxicity studies in pregnant rats summarized in the 1986 O₃ criteria
41 document showed that levels up to about 2.0 ppm did not cause birth defects. Rat pups from
42 females exposed to 1.0 ppm O₃ during certain periods of gestation weighed less or had
43 delays in development of behaviors (e.g., righting, eye opening). No "classical"
44 reproductive assays with O₃ were found.
45

1 Other studies have indicated that O₃ can affect some endocrine organs (i.e., pituitary-
2 thyroid-adrenal axis and parathyroid gland). It appears that the liver has less ability to
3 detoxify drugs after O₃ exposure, but assays of liver enzymes involved in xenobiotic
4 metabolism are not consistent with each other.

5 6 **Interactions of Ozone with Other Co-occurring Pollutants**

7
8 Animals studies of the effects of O₃ in combination with other air pollutants show that
9 antagonism, additivity, and synergism can result, depending upon the animal species,
10 exposure regimen, and health endpoint. Thus, they clearly demonstrate the major
11 complexities and potential importance of interactions, but do not provide a scientific basis for
12 predicting the results of interactions under untested ambient exposure scenarios.

13 14 15 **1.7 HUMAN HEALTH EFFECTS OF OZONE AND RELATED** 16 **PHOTOCHEMICAL OXIDANTS**

17
18 This section summarizes key health effects associated with exposure to O₃, the major
19 component of photochemical oxidant air pollution that is clearly of most health concern to the
20 human population. Another, often co-occurring photochemical oxidant component of "smog"
21 is PAN, but this compound has been demonstrated to be primarily responsible for induction
22 of smog-related eye irritation (stinging of eyes). Limited pulmonary function studies have
23 shown no effects of PAN at concentrations below 0.13 to 0.30 ppm, which are much higher
24 than the generally encountered ambient air levels in most cities.

25 26 **Controlled Human Studies of Acute Ozone Effects**

27 28 *Effects on Lung Function*

29
30 Controlled studies in healthy adult subjects have demonstrated O₃-induced decrements
31 in pulmonary function, characterized by alterations in lung volumes and flow, airway
32 resistance, and airway responsiveness. Respiratory symptoms, such as cough and pain on
33 deep inspiration, are associated with these changes in lung function.

34
35 Ozone-induced decreases in lung volume, specifically forced vital capacity (FVC) and
36 forced expiratory volume in 1 s (FEV₁), can be largely attributed to decreases in inspiratory
37 capacity, or the ability to take a deep breath; although at higher exposure concentrations,
38 there is clearly an additional component that is not volume dependent. They recover to a
39 large extent within 2 to 6 h; normal baseline function is typically reestablished within 24 h,
40 but not fully with more severe exposures.

41
42 Ozone causes increased airway resistance and may cause reductions in expiratory flow
43 and the FEV₁/FVC ratio.
44

Ozone causes an increase in airway responsiveness to nonallergenic stimuli (e.g., histamine or methacholine) in healthy and asthmatic subjects. There is no clear evidence of a relationship between O₃-induced lung volume changes and changes in airway responsiveness.

Inflammation and Host Defense Effects

Controlled studies in healthy adult subjects also indicate that O₃ causes an inflammatory response in the lungs characterized by elevated levels of neutrophils (a type of white blood cell), increased epithelial permeability, and elevated levels of biologically active substances (e.g., prostaglandins, proinflammatory mediators, and cytokines).

Inflammatory responses to O₃ can be detected within 1 h after a single 1-h exposure with exercise to concentrations ≥ 0.30 ppm; the increased levels of some inflammatory cells and mediators persist for at least 18 h. The temporal response profile is not adequately defined, although it is clear that the time course of response varies for different mediators and cells.

Lung function and respiratory symptom responses to O₃ do not seem to be correlated with airway inflammation.

Ozone also causes inflammatory responses in the nose, marked by increased numbers of neutrophils (neutrophilia) and protein levels suggestive of increased permeability.

Alveolar macrophages removed from the lungs of human subjects after 6.6 h exposure to 0.08 and 0.10 ppm O₃ have a decreased ability to ingest microorganisms, indicating some impairment of host defense capability.

Ozone Exposure-Response Relationships

Functional, symptomatic, and inflammatory responses to O₃ increase with increasing exposure dose of O₃. The major determinants of the exposure dose are O₃ concentration (C), exposure duration (T), and the amount of ventilation ($\dot{V}_E$). Of these factors, C has more influence on the response than T or $\dot{V}_E$.

Exercise increases response to O₃ by increasing $\dot{V}_E$ (greater mass delivered), tidal volume, inspiratory flow (greater percentage delivery), and the intrapulmonary O₃ concentration.

Repeated daily exposures to relatively high levels of O₃ doses ($C \times T \times \dot{V}_E$) causing substantial reductions in FEV₁ ($\geq 20\%$ decrement) typically cause exacerbation of the lung function and respiratory symptom responses on the second exposure day. However, attenuation of these responses occurs with continued exposures for a few days. Most inflammatory responses also attenuate; for example, neutrophilia is absent after five consecutive exposures.

Multihour exposures (e.g., for up to 7 h) to O₃ concentrations as low as 0.08 ppm cause small but significant (1) decrements in lung function, (2) increases in respiratory

1 symptoms, and (3) increases in neutrophils and protein levels. Ozone C is a more important
2 factor than exercise $\dot{V}_E$ or T in predicting responses to multihour low-level O_3 exposure.
3 There is now clear evidence of a response plateau in terms of lung volume response to
4 prolonged O_3 exposure. This evidence suggests that for a given combination of exercise and
5 O_3 concentration (i.e., dose rate), there is a response plateau; continued exposure (i.e.,
6 increased T) at that dose rate will not increase response. Therefore, quantitative
7 extrapolation of responses to longer exposure durations is not valid.

8 9 *Mechanisms of Acute Pulmonary Responses*

10
11 The mechanisms leading to the observed pulmonary responses induced by O_3 are
12 beginning to be better understood. The available descriptive data suggest a number of
13 mechanisms leading to the alterations in lung function and respiratory symptoms, including
14 (1) O_3 delivery to the tissue (i.e., the inhaled concentration, breathing pattern, and airway
15 geometry; (2) O_3 reactions with the airway lining fluid and/or epithelial cell membranes;
16 (3) local tissue responses, including injury and inflammation; and (4) stimulation of neural
17 afferents (bronchial C fibers) and the resulting reflex responses and symptoms. The
18 cyclooxygenase inhibitors block production of prostaglandin E_2 and interleukin-6 as well as
19 reduce lung volume responses; however, these drugs do not reduce neutrophilic inflammation
20 and levels of cell damage markers such as lactate dehydrogenase.

21 22 *Effects on Exercise Performance*

23
24 Maximal oxygen uptake, a measure of peak exercise performance capacity, is reduced
25 in healthy young adults if preceded by O_3 exposures sufficient to cause marked changes in
26 lung function (i.e., decreases of at least 20%) and increased subjective symptoms of
27 respiratory discomfort. Limitations in exercise performance may be related to increased
28 symptoms, especially those related to breathing discomfort.

29 30 *Factors Modifying Responsiveness to Ozone*

31
32 Many variables have the potential for influencing responsiveness to O_3 ; most, however,
33 are inadequately addressed in the available clinical data to make definitive conclusions.

34
35 Active smokers are less responsive to O_3 exposure, which may reverse following
36 smoking cessation, but these results should be interpreted with caution.

37
38 The possibility of age-related differences in response to O_3 has been explored, although
39 young adults historically have provided the subject population for controlled human studies.
40 Children and adolescents have similar lung volume responses to O_3 as young adults, but lack
41 respiratory symptoms at levels to which they have been exposed. Pulmonary function
42 responsiveness in adults appears to decrease with age, whereas symptom rates remain similar
43 to young adults. Group mean lung function responses of adults over 50 years of age are less
44 than those of children, adolescents, and young adults.

45
46 The available data have not conclusively demonstrated that men and women respond
47 differently to O_3 . Likewise, pulmonary function responses of women have been compared

1 during different phases of the menstrual cycle, but the results are conflicting. If gender
2 differences exist for lung function responsiveness to O₃, they are not based on hormonal
3 changes, differences in lung volume, or the ratio of FVC to V_E.

4
5 There is no compelling evidence, to date, suggesting that any ethnic or racial groups
6 have a different distribution of responsiveness to O₃.

7
8 Seasonal and ambient factors may vary responsiveness to O₃, but further research is
9 needed to determine how they affect individual subjects. Individual sensitivity to O₃ may
10 vary throughout the year, related to seasonal variations in ambient O₃ concentrations.

11
12 The specific inhalation route appears to be of minor importance in exercising adults.
13 Exposure to O₃ by oral breathing (i.e., mouthpiece) yields similar results as oronasal
14 breathing (i.e., chamber exposures).

15 16 *Population Groups at Risk from Ozone Exposure*

17
18 Population groups that have demonstrated responsiveness to ambient concentrations of
19 O₃ consist of exercising healthy and asthmatic individuals, including children, adolescents,
20 and adults.

21
22 Available evidence from controlled human studies on subjects with preexisting disease
23 suggests that (1) mild asthmatics have similar lung volume responses, but greater airway
24 resistance responses to O₃ than nonasthmatics; and (2) moderate asthmatics may have, in
25 addition, greater lung volume responses than nonasthmatics.

26
27 Of all the other population groups studied, those with preexisting limitations in
28 pulmonary function and exercise capacity (e.g., chronic obstructive pulmonary disease,
29 chronic bronchitis, ischemic heart disease) would be of primary concern in evaluating the
30 health effects of O₃. Unfortunately, limitations of (1) subject selection, (2) standardized
31 methods of subject characterization, and (3) range of exposure hamper the ability to make
32 definitive conclusions regarding the relative responsiveness of most chronic disease subjects.

33 34 *Effects of Ozone Mixed with Other Pollutants*

35
36 No significant enhancement of respiratory effects has been consistently demonstrated
37 for simultaneous exposures of O₃ mixed with SO₂, NO₂, sulfuric or nitric acid, particulate
38 aerosols, or with multiple combinations of these pollutants. It is fairly well established that
39 simultaneous exposure of healthy adults and asthmatics to mixtures of O₃ and other pollutants
40 for short periods of time (<2 h) induces pulmonary function responses not significantly
41 different from those following O₃ alone when studies are conducted at the same
42 O₃ concentration. Exposure to PAN has been reported to induce greater pulmonary function
43 responses than exposure to O₃ alone, but at PAN concentrations (>0.27 ppm) much higher
44 than ambient levels. Unfortunately, only a limited number of pollutant combinations and
45 exposure protocols have been investigated, and subject groups are small and are
46 representative of only small portions of the general population. Thus, much is unknown
47 about the relationships between O₃ and the complex mix of pollutants found in the ambient air.

1 Prior exposure to O₃ in asthmatics may cause an increase in response to other pollutant
2 gases, especially SO₂. Likewise, prior exposure to other pollutants can enhance responses to
3 O₃ exposure.
4

5 **Controlled Human Studies of Ambient Air Exposures**

6

7 Mobile laboratory studies of lung function and respiratory symptoms in a local subject
8 population exposed to ambient photochemical oxidant pollution provide quantitative
9 information on exposure-response relationships for O₃. A series of these studies from
10 Los Angeles, CA, have demonstrated pulmonary function decrements at O₃ concentrations of
11 0.14 ppm in exercising healthy adolescents, and increased respiratory symptoms and
12 pulmonary function decrements at 0.15 ppm in heavily exercising athletes and at 0.17 ppm in
13 lightly exercising healthy and asthmatic subjects. Comparison of the observed effects in
14 exercising athletes with controlled chamber studies at comparable O₃ concentrations showed
15 no significant differences in lung function and symptoms, suggesting that coexisting ambient
16 pollutants have a minimal contribution to the measured responses under typical summer
17 ambient conditions in Southern California.
18

19 **Field and Epidemiology Studies of Ambient Air Exposures**

20

21 Individual-level field studies and aggregate level time-series studies have addressed the
22 acute effects of O₃ on lung function decrements and increased morbidity and mortality in
23 human populations exposed to real-world conditions of O₃ exposure.
24

25 Camp and exercise studies of lung function provide quantitative information on
26 exposure-response relationships linking lung function declines with O₃ exposure occurring in
27 ambient air. Combined statistical analysis of six recent camp studies in children yields an
28 average relationship between decrements in FEV₁ and previous-hour O₃ concentration of
29 -0.64 mL/ppb. Two key studies of lung function measurements before and after well-
30 defined outdoor exercise events in adults have yielded exposure-response slopes of
31 -0.40 and -1.35 mL/ppb. The magnitude of pulmonary function declines with
32 O₃ exposure is consistent with the results of controlled human studies.
33

34 Daily life studies support a consistent relationship between O₃ exposure and acute
35 respiratory morbidity in the population. Respiratory symptoms (or exacerbation of asthma)
36 and decrements in peak expiratory flow rate are associated with increasing ambient O₃,
37 particularly in asthmatic children; however, concurrent temperature, particles, acidity
38 (hydrogen ions), aeroallergens, and asthma severity or medication status may also contribute
39 as independent or modifying factors. Aggregate results show greater responses in asthmatic
40 individuals than in nonasthmatics, indicating that asthmatics constitute a sensitive group in
41 epidemiologic studies of oxidant air pollution.
42

43 Summertime daily hospital admissions for respiratory causes in various locations of
44 eastern North America have consistently shown a relationship with ambient levels of O₃,
45 accounting for approximately one to three excess respiratory hospital admissions per hundred
46 ppb O₃ per million persons. This association has been shown to remain even after

1 statistically controlling for the possible confounding effects of temperature and copollutants
2 (e.g., hydrogen ions, sulfate, and particles less than 10 μm), as well as when considering
3 only concentrations below 0.12 ppm O_3 .
4

5 Two recent time-series epidemiologic studies indicate a small but statistically significant
6 association between O_3 and total daily mortality in Los Angeles, CA, and New York City,
7 NY, where peak 1-h maximum O_3 reached concentrations greater than 0.2 ppm during the
8 study period. The results were significant even after controlling for the potentially
9 confounding effects of temperature and particles. A third study in regions with lower
10 (≤ 0.15 ppm) maximum 1-h O_3 concentrations (St. Louis, MO, and Kingston-Harriman, TN)
11 did not detect a significant O_3 association with mortality.
12

13 Only suggestive epidemiologic evidence exists for health effects of chronic ambient
14 O_3 exposure in the population. All of the available studies of chronic respiratory system
15 effects in exposed children and adults are limited by a simplistic assignment of exposure or
16 by their inability to isolate potential effects related to O_3 from those of other pollutants,
17 especially particles.
18
19

20 **1.8 EXTRAPOLATION OF ANIMAL TOXICOLOGICAL DATA TO** 21 **HUMANS** 22

23 There have been significant advances in O_3 dosimetry since 1986 that better enable
24 quantitative extrapolation with marked reductions in uncertainty. Experiments and models
25 describing the uptake efficiency and delivered dose of O_3 in the respiratory tract (RT) of
26 animals and humans are beginning to present a clearer picture than has previously existed.
27

28 The total RT uptake efficiency of rats at rest is approximately 50%. Within the RT of
29 the rat, 50% of the O_3 taken up by the RT is removed in the head, 7% in the larynx/trachea,
30 and 43% in the lungs.
31

32 In humans at rest, the total RT uptake efficiency is between 80 and 95%. Total RT
33 uptake efficiency falls as flow increases. As tidal volume increases, uptake efficiency
34 increases and flow dependence lessens. Pulmonary function response data and O_3 uptake
35 efficiency data in humans generally indicate that the mode of breathing (oral versus nasal
36 versus oronasal) has little effect on upper RT or on total RT uptake efficiency, though one
37 study suggests that the nose has a higher uptake efficiency than the mouth.
38

39 When all of the animal and human in vivo O_3 uptake efficiency data are compared,
40 there is a good degree of consistency across data sets. This agreement raises the level of
41 confidence with which these data sets can be used to support dosimetric model formulations.
42

43 Several mathematical dosimetry models have been developed since 1986. Generally,
44 the models predict that net O_3 dose to lung lining fluid plus tissue gradually decreases
45 distally from the trachea toward the end of the tracheobronchial region, and then rapidly
46 decreases in the pulmonary region.

1 When the dose of O₃ to lung tissue is computed theoretically, it is found to be very low
2 in the trachea, to increase to a maximum in the terminal bronchioles of the first generation
3 pulmonary region, and then to decrease rapidly, moving further into the pulmonary region.
4 The increased tidal volume and flow, associated with exercise in humans, shifts O₃ dose
5 further into the periphery of the lung and causes a disproportionate increase in distal lung
6 dose.

7
8 Predictions of delivered dose have been used to investigate O₃ responses in the context
9 of intra- and interspecies comparisons. In the case of intraspecies comparisons, for example,
10 the distribution of predicted O₃ tissue dose to a ventilatory unit in a rat as a function of
11 distance from the bronchoalveolar duct junction is very consistent with the distribution of
12 alveolar wall thickening. In the case of interspecies comparisons (using the delivered
13 O₃ dose to the proximal alveolar regions), although the tachypneic responses (i.e., rapid,
14 shallow breathing) differ markedly between rats and humans, there is similarity of dose-
15 response patterns in inflammation among species, with humans and guinea pigs more
16 responsive than rats and rabbits. In other words, the quantitative relationship between animal
17 and human responses is dependent on the animal species and the endpoint.

18
19 In summary, there is an emerging consistency among a variety of O₃ dosimetry data
20 sets and between the experimental data and theoretical predictions of O₃ dose. The
21 convergence of experimental data with theoretical predictions lends a degree of confidence to
22 the use of theoretical models to predict total and regional O₃ dose. The use of O₃ dosimetry
23 data and models is beginning to bear fruit in attempts to extrapolate effects between animals
24 and humans. The data and models have thus far helped demonstrate that humans may be
25 more responsive to O₃ than rats with respect to inflammatory responses. Thus, chronic
26 effects data in rats may not accurately reflect the degree to which comparably exposed
27 humans would respond.

30 1.9 INTEGRATIVE SUMMARY OF OZONE HEALTH EFFECTS

31
32 This section summarizes the primary conclusions derived from an integration of the
33 known health effects of O₃ provided by animal toxicological, human clinical, and
34 epidemiological studies.

36 1. *What are the health effects of short-term (<8 h) exposures to ozone?*

37
38 Acute O₃ exposure of laboratory animals and humans causes changes in pulmonary
39 function, including tachypnea (rapid, shallow breathing), decreased lung volumes and flows,
40 and increased airway responsiveness to nonspecific stimuli. Increased airway resistance
41 occurs in both humans and laboratory animals, but typically at higher exposure levels than
42 other functional endpoints. In addition, adult human subjects experience O₃-induced
43 symptoms of airway irritation such as cough or pain on deep inspiration. The changes in
44 pulmonary function and respiratory symptoms occur as a function of exposure concentration,
45 duration, and level of exercise. Recovery of pulmonary function and the absence of

O₃-induced symptoms is usually complete within 24 h of the end of exposure, although other responses may persist somewhat longer.

- Pulmonary function decrements are generally observed in healthy subjects (8 to 45 years of age) after 1 to 3 h of exposure as a function of the level of exercise performed and the O₃ concentration inhaled during the exposure. Group mean data from numerous controlled human exposure and field studies indicate that, in general, statistically significant pulmonary function decrements beyond the range of normal measurement variability (e.g., 3 to 5% for FEV₁) occur

- (1) at >0.5 ppm when at rest,
- (2) at >0.37 ppm with light exercise (slow walking),
- (3) at >0.30 ppm with moderate exercise (brisk walking),
- (4) at >0.18 ppm with heavy exercise (easy jogging), and
- (5) at >0.16 ppm with very heavy exercise (running).

For a number of studies, small group mean changes (e.g., <5%) in FEV₁, the medical significance of which is a matter of controversy, have been observed at lower O₃ concentrations than those listed above. For example, data from one specific study indicate that FEV₁ decrements occur with very heavy exercise in healthy adults at 0.15 to 0.16 ppm O₃, and data from two studies indicate that such effects may occur in healthy adults at levels as low as 0.12 ppm. Also, pulmonary function decrements have been observed in children and adolescents at concentrations of 0.12 and 0.14 ppm O₃ with heavy exercise. Pulmonary function decrements were observed at 0.12 ppm O₃ in healthy young adults undergoing heavy exercise in a recent study. Some individuals within a study may experience FEV₁ decrements in excess of 15% under these exposure conditions, even when the group mean decrement is less than 5%.

- For exposures of healthy subjects performing moderate exercise during longer duration exposures (6 to 8 h), 5% group mean decrements in FEV₁ were observed at

- (1) 0.08 ppm O₃ after 5.6 h,
- (2) 0.10 ppm O₃ after 4.6 h, and
- (3) 0.12 ppm O₃ after 3 h.

For these same subjects, 10% group mean FEV₁ decrements were observed at 0.12 ppm O₃ after 5.6 and 6.6 h. As in the shorter duration studies, some individuals experience changes larger than those represented by the group mean changes.

- An increase in the incidence of cough has been reported at O₃ concentrations as low as 0.12 ppm in healthy adults during 1 to 3 h of exposure with very heavy exercise. Other respiratory symptoms, such as

1 pain on deep inspiration, shortness of breath, and lower respiratory scores
2 (a combination of several symptoms), have been observed at 0.16 to
3 0.18 ppm O₃ with heavy and very heavy exercise. Respiratory symptoms
4 have also been observed following exposure to 0.08, 0.10, and 0.12 ppm
5 O₃ for 6.6 h with moderate levels of exercise.
6

- 7 • Increases in nonspecific airway responsiveness have been observed after
8 1 to 3 h of exposure to 0.40 ppm, but not 0.20 ppm, O₃ at rest, and have
9 been observed at concentrations as low as 0.18 ppm, but not to 0.12 ppm,
10 O₃ during exposure with very heavy exercise. Increases in nonspecific
11 airway responsiveness during 6.6-h exposures with moderate levels of
12 exercise have been observed at 0.08, 0.10, and 0.12 ppm O₃.
13

14 Acute O₃ exposure of laboratory animals and humans disrupts the barrier function of
15 the lung epithelium, permitting materials in the airspaces to enter lung tissue, allowing cells
16 and serum proteins to enter the airspaces (inflammation), and setting off a cascade of
17 responses.
18

- 19 • Increased levels of neutrophils and protein in lung lavage fluid have been
20 observed following exposure of humans to 0.20, 0.30, and 0.40 ppm with
21 very heavy exercise and have not been studied at lower concentrations for
22 1- to 3-h exposures. Increases in protein and/or neutrophils have also been
23 observed at 0.08 and 0.10 ppm O₃ during 6.6-h exposures with moderate
24 exercise; lower concentrations have not been tested.
25

26 Acute O₃ exposure of laboratory animals and humans impairs alveolar macrophage
27 clearance of viable and nonviable particles from the lungs and decreases the effectiveness of
28 host defenses against bacterial lung infections in animals and perhaps humans. The ability of
29 alveolar macrophages to engulf microorganisms is decreased in humans exposed to 0.08 and
30 0.10 ppm for 6.6 h with moderate exercise.
31

32 *2. What are the health effects of repeated, short-term exposures to ozone?*

33

34 During repeated acute exposures, some of the O₃-induced responses are partially or
35 completely attenuated. Over a 5-day exposure, pulmonary function changes are typically
36 greatest on the second day, but return to control levels by the fifth day of exposure. Most of
37 the inflammatory markers (e.g., neutrophil influx) also attenuate by the fifth day of exposure,
38 but markers of cell damage (e.g., lactate dehydrogenase enzyme activity) do not attenuate
39 and continue to increase. Attenuation of lung function decrements is reversed following 7 to
40 10 days without O₃. Some inflammatory markers are also reversed during this time period,
41 but others still show attenuation even after 20 days without O₃. The mechanisms and
42 impacts involved in attenuation are not known, although the underlying cell damage continues
43 throughout the attenuation process. In addition, attenuation may alter the normal distribution
44 of O₃ within the lung, allowing more O₃ to reach sensitive regions, possibly affecting normal
45 lung defenses (e.g., neutrophil influx in response to inhaled microorganisms).
46
47

1 **3. *What are the health effects of long-term exposures to ozone***
2

3 Exposure to O₃ for months and years causes structural changes in several regions of the
4 respiratory tract, but effects may be of the greatest importance in the centriacinar regions
5 where the alveoli and conducting airways meet because this region is typically affected in
6 most chronic diseases of the human lung. This information on O₃ effects in the distal lung is
7 derived from animal toxicological studies because, to date, such data are not available in
8 humans. Epidemiological studies attempting to associate chronic health effects in humans
9 with long-term O₃ exposure have yet to provide unequivocal evidence that such a linkage
10 exists.

11
12 Chronic exposure of one strain of female mice to high O₃ levels (1 ppm) caused a
13 small, but statistically significant increase in lung tumors. There was no concentration-
14 response relationship and rats were not affected. Genotoxicity data are either negative or
15 weak. Given the nature of the database, the effects in one strain of mice cannot yet be
16 qualitatively extrapolated to humans. Ozone did not show tumor-promoting activity in a
17 chronic rat study (at 0.5 ppm O₃).

18
19 **4. *What are the health effects of binary pollutant mixtures containing ozone?***
20

21 Combined data from laboratory animal and controlled human exposure studies on
22 O₃ support the hypothesis that coexposure to pollutants, each at low-effect levels, may result
23 in effects of significance. The data from human studies of O₃ in combination with NO₂,
24 SO₂, sulfuric acid, nitric acid, or CO show no more than an additive response on lung
25 spirometry or respiratory symptoms. The larger number of laboratory animal studies with
26 O₃ in mixture with NO₂ and sulfuric acid show that effects can be additive, synergistic, or
27 even antagonistic, depending upon the exposure regimen and the endpoint studied. This issue
28 of exposure to copollutants remains poorly understood, especially with regard to chronic
29 effects.

30
31 **5. *What population groups are at-risk as a result of exposure to ozone?***
32

33 Identification of population groups that may show increased susceptibility to O₃ are
34 based on their (1) biological responses to O₃; (2) physiological status (e.g., preexisting lung
35 disease); (3) activity patterns; (4) personal exposure history; and (5) personal factors (e.g.,
36 age, nutritional status).

37
38 The predominant information on the health effects of O₃ noted above comes from
39 studies on healthy, nonsmoking, exercising subjects, 8 to 45 years of age. These studies
40 demonstrate that among this group, there is a large variation in sensitivity and responsiveness
41 to O₃, with at least a 10-fold difference between the most and least responsive individuals.
42 Individual sensitivity to O₃ may also vary throughout the year, related to seasonal variations
43 in ambient O₃ exposure. The specific factors that contribute to this large intersubject
44 variability, however, remain undefined. Although differences may be due to the dosimetry
45 of O₃ in the respiratory tract, available data show little effect on O₃ deposition after
46 inhalation through the nose or mouth.

1 Controlled studies on mild asthmatics suggest that they have similar lung volume
2 responses but greater airway resistance changes to O₃ than nonasthmatics. Furthermore,
3 limited data from studies of moderate asthmatics suggest that they may have greater lung
4 volume responses than nonasthmatics. Daily life studies reporting an exacerbation of asthma
5 and decrease in peak expiratory flow rates, particularly in asthmatic children, appear to
6 support the controlled studies; however, those studies are confounded by concurrent
7 temperature, particle or aeroallergen exposure, and asthma severity of the subjects or their
8 medication use. In addition, field studies of summertime daily hospital admissions for
9 respiratory causes show a consistent relationship between asthma and ambient levels of O₃ in
10 various locations in the northeastern United States, even after controlling for independent
11 contributing factors.

12
13 Other population groups with preexisting limitations in pulmonary function and exercise
14 capacity (e.g., chronic obstructive pulmonary disease, chronic bronchitis, ischemic heart
15 disease) would be of primary concern in evaluating the health effects of O₃. Unfortunately,
16 not enough is known about the responses of these individuals to make definitive conclusions
17 regarding their relative responsiveness to O₃. Indeed, functional effects in these individuals
18 with reduced lung function may have greater clinical significance than comparable changes in
19 healthy individuals.

20
21 Currently available data on personal factors or personal exposure history known or
22 suspected of influencing responses to O₃ are the following.

- 23
24 • Human studies have identified a decrease in pulmonary function
25 responsiveness to O₃ with increasing age, although symptom rates remain
26 similar. Toxicological studies are not easily interpreted but suggest that
27 young animals are not more responsive than adults.
- 28
29 • Available toxicological and human data have not conclusively demonstrated
30 that males and females respond differently to O₃. If gender differences
31 exist for lung function responsiveness to O₃, they are not based on
32 differences in baseline pulmonary function.
- 33
34 • There is no compelling evidence to date to suggest that any ethnic or racial
35 group has a different distribution of responsiveness to O₃. However, data
36 are not adequate to rule out the possibility of such differences.
- 37
38 • Information derived from O₃ exposure of smokers is limited. The general
39 trend is that smokers are less responsive than nonsmokers. This reduced
40 responsiveness may wane after smoking cessation.
- 41
42 • Although nutritional status (e.g., vitamin E deficiency) makes laboratory
43 rats more susceptible to O₃-induced effects, it is not clear if vitamin E
44 supplementation has an effect in human populations. Such supplementation
45 has no or minimal effects in animals. The role of such antioxidant vitamins
46 in O₃ responsiveness, especially their deficiency, has not been well studied.

1 Based on information presented in this document, the population groups that have
2 demonstrated responsiveness to ambient concentrations of O₃ consist of exercising healthy
3 and asthmatic individuals, including children, adolescents, and adults.



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
Office of Air Quality Planning and Standards
Research Triangle Park, North Carolina 27711

November 15, 1993

Dr. George T. Wolff, Ph.D.
Principal Scientist
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Argonaut A-205-H
485 W. Milwaukee
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Dear Dr. Wolff:

As discussed with Randy Bond, in preparation for the December 16, 1993 meeting of the Clean Air Scientific Advisory Committee's (CASAC's) subcommittee on ozone (O_3) exposure and risk, I want to lay out the topics that we would like to discuss with the subcommittee. These include:

1. The objectives and overall approach used by the Ambient Standards Branch (ASB) in estimating exposures and risks associated with alternative O_3 national ambient air quality standards (NAAQS).
2. The specific exposure modeling methodology (pNEM/ O_3) employed by ASB.
3. The results obtained by applying pNEM/ O_3 to the Los Angeles urban area.

To facilitate the discussion, I am transmitting the enclosed materials directly to you and the subcommittee members that you identified, as requested by Randy Bond. You will find three documents that pertain to the last two topics: An International Technology-Air Quality Services draft report entitled Estimation of Ozone Exposures Experienced by Urban Residents Using a Probabilistic Version of NEM and 1990 Population Data (September 1993) which describes in detail the exposure modeling methodology that ASB proposes to use in the O_3 standard review; and two memos by Tom McCurdy, one is "Los Angeles Ozone Exposure Estimates" (November 4, 1993) which describes results of applying the methodology to people living and working in that urban area, and

the other is "Testing pNEM/O₃ Runs to Determine if a Set of 10 Runs is 'Representative'" (April 19, 1993) which provides additional information regarding the inherent variability in multiple runs of pNEM/O₃.

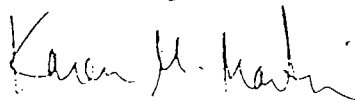
With respect to the report and the first memo, we request that subcommittee members review them in detail in order to provide us with specific advice and recommendations as to the technical soundness of the exposure methodology that we plan to employ. In order to avoid delays later in the review process, we believe that it is important to identify now any areas in the methodology and its application that require further refinement. Toward this end, we ask that the subcommittee members specifically address the following topics:

1. The type of O₃ exposure indicators that are presented, as well as the format used to summarize results of the modeling exercises.
2. The air quality adjustment model that is used to simulate attainment of alternative O₃ standards.
3. The commuting methodology used to simulate home-to-work trips that allows us to "move" people around an urban area.
4. The Nagda, Rector, and Koontz mass-balance model used to estimate indoor concentrations, including the air exchange rate data needed to solve that model.
5. The breathing rate algorithms used to simulate ventilation by people undergoing various exercise levels as they go through their daily activities.

During the course of your review, please feel free to raise other topics as well that pertain to the methodology or its application.

Thank you for agreeing to participate in this important project. We look forward to receiving the subcommittee's comments and recommendations at the December meeting. By copy of this letter, I am also forwarding the enclosed materials to the members of CASAC for their information.

Sincerely,



Karen M. Martin, Ph.D.
Chief

Ambient Standards Branch

cc: Subcommittee on Ozone Exposure and Risk:

Dr. Stephen Ayres
Dr. Stephen D. Colome
Dr. Morton Lippman
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"PRELIMINARY DRAFT"

ESTIMATION OF OZONE EXPOSURES EXPERIENCED BY URBAN RESIDENTS USING A PROBABILISTIC VERSION OF NEM AND 1990 POPULATION DATA

by

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