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21 MAY 1992

ENVIRONMENTAL PROTECTION AGENCY

(40 CFR Part 50)

[AD-FDL]

Proposed Decision Not to Revise the
National Ambient Air Quality Standards for Ozone

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed Decision.

SUMMARY: In accordance with the provisions of sections 108 and 109 of the Clean Air Act (Act), as amended, the EPA has conducted a review of the criteria upon which the existing national ambient air quality standards (NAAQS) for ozone (O_3) are based. The revised criteria and supplement are being published simultaneously with the issuance of this proposed decision. The level of the existing primary and secondary standards for O_3 is currently set at 0.12 parts per million (ppm). The standards are attained when the expected number of days per calendar year with maximum hourly average concentrations above 0.12 ppm is equal to or less than 1, as determined by 40 CFR Part 50, Appendix H. As a result of the review of health and welfare criteria, the EPA proposes not to revise primary and secondary standard levels and averaging times. In view of recent studies and ongoing research on the health and welfare

1 effects of O₃, the EPA plans to proceed as rapidly as
2 possible with the next review of the air quality criteria
3 and standards for O₃.

4 DATES: The EPA will hold a public hearing on [date, time
5 and place to be added]. Written comments on this proposed
6 decision must be received by [60 days from publication of
7 today's notice].

8 ADDRESSES: Submit comments on the proposed action to:
9 Central Docket Section (A-130), Environmental Protection
10 Agency, Attn: Docket No. A-92-17, 401 M St. S.W.,
11 Washington, D.C. 20460. The docket may be inspected between
12 8:00 a.m. and 3:00 p.m. on weekdays, and a reasonable fee
13 may be charged for copying. For availability of related
14 documents, see Supplementary Information.

15 FOR FURTHER INFORMATION CONTACT: Mr. John Haines, MD-12,
16 Air Quality Management Division, Office of Air Quality
17 Planning and Standards, U.S. Environmental Protection
18 Agency, Research Triangle Park, North Carolina 27711.
19 Telephone: 919-541-5533.

20 SUPPLEMENTARY INFORMATION:

21 Availability of Related Information

22 Certain documents are available from the U.S.
23 Department of Commerce, National Technical Information
24 Service, 5285 Port Royal Road, Springfield, Virginia 22161.
25 Available documents include: Air Quality Criteria for Ozone
26 and Other Photochemical Oxidants (five volumes, EPA 600/8-
27 84-020aF thru eF, August 1986; NTIS # PB-87-142949, \$137.99

1 - paper copy and \$.00 microfiche); the Criteria Document
2 Supplement, Summary of Selected New Information on Effects
3 of Ozone on Health and Vegetation: Supplement to Air Quality
4 Criteria for Ozone and Other Photochemical Oxidants
5 (EPA/600/8-88-105F, NTIS # PB-92-, \$.00 paper copy and
6 \$.00 microfiche); and the 1989 Staff Paper, Review of the
7 National Ambient Air Quality Standards for Ozone:
8 Assessment of Scientific and Technical Information; OAQPS
9 Staff Paper (EPA-450/3-92-001, June 1989; NTIS # PB-92-, \$
10 .00 paper copy and \$.00 microfiche). (Add a \$3.00
11 handling charge per order.) A limited number of copies of
12 other documents generated in connection with this standard
13 review, such as documents pertaining to control techniques
14 for volatile organic emissions from stationary sources, are
15 available and can be obtained from: U.S. Environmental
16 Protection Agency Library (MD-35), Research Triangle Park,
17 NC 27711, telephone (919) 541-2777. These and other related
18 documents are also available in the EPA docket identified
19 above.

20 The contents of today's preamble are listed in the
21 following outline.

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I. Background

A. Legislative Requirements

1. The Standards

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

Section 109 (42 U.S.C. 7409) directs the Administrator to propose and promulgate "primary" and "secondary" NAAQS

1 - for pollutants identified under section 108. Section
2 109(b)(1) defines a primary standard as one "the attainment
3 and maintenance of which, in the judgment of the
4 Administrator, based on the criteria and allowing an
5 adequate margin of safety, [is] requisite to protect the
6 public health." A secondary standard, as defined in section
7 109(b)(2), must "specify a level of air quality the
8 attainment and maintenance of which, in the judgment of the
9 Administrator, based on [the] criteria, is requisite to
10 protect the public welfare from any known or anticipated
11 adverse effects associated with the presence of [the]
12 pollutant in the ambient air." Welfare effects are defined
13 in section 302(h) [42 U.S.C. 7602(h)] to include "effects on
14 soils, water, crops, vegetation, manmade materials, animals,
15 wildlife, weather, visibility and climate, damage to and
16 deterioration of property, and hazards to transportation, as
17 well as effects on economic values and on personal comfort
18 and well-being."

19 The U.S. Court of Appeals for the District of Columbia
20 Circuit has held that the requirement for an adequate margin
21 of safety for primary standards was intended to address
22 uncertainties associated with inconclusive scientific and
23 technical information available at the time of standard
24 setting. It was also intended to provide a reasonable
25 degree of protection against hazards that research has not
26 yet identified. Lead Industries Association v. EPA, 647

1 F.2d 1130, 1154 (D.C. Cir. 1980), cert. denied, 101 S. Ct.
2 621 (1980); American Petroleum Institute v. Costle, 665 F.2d
3 1176, 1177 (D.C. Cir. 1981), cert. denied, 102 S. Ct. 1737
4 (1982). Both kinds of uncertainties are components of the
5 risk associated with pollution at levels below those at
6 which human health effects can be said to occur with
7 reasonable scientific certainty. Thus, by selecting primary
8 standards that provide an adequate margin of safety, the
9 Administrator is seeking not only to prevent pollution
10 levels that have been demonstrated to be harmful but also to
11 prevent lower pollutant levels that he finds may pose an
12 unacceptable risk of harm, even if the risk is not precisely
13 identified as to nature or degree.

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

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

14 The process by which the EPA has reviewed the existing
15 air quality criteria and standards for O₃ under section
16 109(d) is described in a later section of this notice.

17 2. Related Control Requirements

18 States are primarily responsible for ensuring
19 attainment and maintenance of ambient air quality standards
20 once the EPA has established them. Under Title I of the Act
21 (42 U.S.C. 7410), States are to submit, for EPA approval,
22 State implementation plans (SIP's) that provide for the
23 attainment and maintenance of such standards through control
24 programs directed to sources of the pollutants involved.
25 The States, in conjunction with the EPA, also administer the
26 prevention of significant deterioration program (42 U.S.C.

1 - 7470-7479) for these pollutants. In addition, Federal
2 programs provide for nationwide reductions in emissions of
3 these and other air pollutants through the Federal Motor
4 Vehicle Control Program under Title II of the Act (42 U.S.C.
5 7521-7574), which involves controls for automobile, truck,
6 bus, motorcycle, and aircraft emissions; the new source
7 performance standards under section 111 (42 U.S.C. 7411);
8 and the national emission standards for hazardous air
9 pollutants under section 112 (42 U.S.C. 7412).

10 B. Existing Standards for Ozone

11 The principal focus of this standard review is on the
12 health and welfare effects of O₃. Ozone produced in the
13 ambient air is commonly referred to as tropospheric O₃. It
14 is chemically identical to stratospheric O₃, which is
15 produced miles above the earth's surface and provides a
16 protective shield from excess ultraviolet radiation. In
17 contrast, tropospheric O₃ produces harmful effects due to
18 its oxidative properties and its proximity to humans,
19 plants, and materials. Ozone is not emitted directly from
20 mobile or stationary sources but, like other photochemical
21 oxidants, commonly exists in the ambient air as an
22 atmospheric transformation product. Ozone formation is the
23 result of chemical reactions of volatile organic compounds
24 (VOC's), nitrogen oxides (NO_x), and oxygen (O₂) in the
25 presence of sunlight and generally at elevated temperatures.

Ozone is a highly reactive gas which produces a wide variety of harmful effects. At elevated concentrations, O₃ can adversely affect human health, vegetation, materials, economic values, and personal comfort and well-being. Through chemical reaction with other pollutants, such as sulfur dioxide (SO₂) and nitrogen dioxide (NO₂), O₃ can contribute to formation of acidic deposition and visibility impairment. Hourly average ambient O₃ levels range from 0.03 ppm in the most remote rural areas to 0.30 ppm and higher in the most polluted urban areas. A detailed discussion of formation, concentrations, and effects of O₃ can be found in the 1986 Air Quality Criteria Document (U.S. EPA, 1986), the Criteria Document Supplement (U.S. EPA, 1992), and the Staff Paper (U.S. EPA, 1989).

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 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. In preparing the Air Quality Criteria Document, the EPA provided a number of opportunities for external review and comment. The EPA made two drafts of the document available for public comment, and these drafts were peer reviewed by

1 the Subcommittee on Scientific Criteria for Photochemical
2 Oxidants of the EPA Science Advisory Board. The EPA
3 published the final revised Air Quality Criteria for Ozone
4 and Other Photochemical Oxidants on June 22, 1978.

5 Based on the 1978 revised Air Quality Criteria Document
6 and taking into account the advice and recommendations of
7 the Subcommittee, on June 22, 1978, the EPA proposed (43 FR
8 16962) revisions to the then-current primary and secondary
9 NAAQS for photochemical oxidants. The proposed changes
10 included raising the primary standard to 0.10 ppm, retaining
11 the 0.08 ppm secondary standard, changing the chemical
12 designation of the standards from photochemical oxidants to
13 O₃, and changing to standards with a statistical (i.e.,
14 expected exceedances) form rather than a deterministic form
15 (i.e., not to be exceeded more than x number of times per
16 year).

17 After taking into account public comments, the EPA
18 announced its final decision on the proposed revisions to
19 the 1971 standards. On February 8, 1979 (44 FR 8202), the
20 final rulemaking revised the level of the primary standard
21 from 0.08 ppm to 0.12 ppm, set the secondary standard
22 identical to the primary standard, changed the chemical
23 designation of the standards from photochemical oxidants to
24 O₃, and revised the definition of the point at which the
25 standard is attained to "when the expected number of days
26 per calendar year with maximum hourly average concentrations

1 - above 0.12 ppm is equal to or less than one as determined by
2 Appendix H."

3 C. Review of Air Quality Criteria and Standards for Ozone
4 and Other Photochemical Oxidants

5 In response to requirements of section 109(d) of the
6 Act, on March 17, 1982 (47 FR 11561), the EPA announced that
7 it was undertaking plans to revise the existing 1978 Air
8 Quality Criteria Document for Ozone and Other Photochemical
9 Oxidants and on August 22, 1983, announced (48 FR 38009)
10 that review of primary and secondary standards for O₃ had
11 been initiated. The EPA provided a number of opportunities
12 for review and comment on revised chapters of the Air
13 Quality Criteria Document by organizations and individuals
14 outside the Agency. On November 24, 1982 (47 FR 53119), the
15 EPA announced that its Environmental Criteria and Assessment
16 Office (ECAO) would conduct a public workshop on December
17 15-17, 1982, for authors and scientific peer reviewers to
18 discuss working draft chapters of a third revision of the
19 Air Quality Criteria Document pertaining to effects of O₃
20 and other photochemical oxidants on vegetation and materials
21 damage. The EPA announced (48 FR 50157) that a second
22 public workshop to discuss draft chapters on health effects
23 of O₃ was to be held on November 16-18, 1983. The EPA
24 carefully considered comments made at both workshops in
25 preparing the first external review draft, made available
26 (49 FR 29845) on July 24, 1984 for a 90-day public review.

1 On August 6, 1984 (49 FR 31337), the Agency extended the
2 comment period to November 19, 1984. Due to the length and
3 complexity of the document and requests for more time to
4 review it, on November 1, 1984, the Agency further extended
5 the comment period to January 4, 1985 (49 FR 44019).

6 On February 13, 1985 (50 FR 6049) and on April 2, 1986
7 (51 FR 11339), the EPA announced two public meetings of the
8 Clean Air Scientific Advisory Committee (CASAC) to be held
9 on March 4-6, 1985 and on April 21-22, 1986, respectively.
10 At these meetings, the CASAC reviewed external review drafts
11 of the Air Quality Criteria Document for Ozone and
12 Photochemical Oxidants. Many individuals and
13 representatives of organizations provided comments for
14 consideration. The EPA placed transcripts of the CASAC
15 meetings in the docket for the 1986 Air Quality Criteria
16 Document (ECAO CD 81-1). The EPA considered comments
17 received from the public and the CASAC members in preparing
18 the final document. The CASAC sent the Administrator a
19 "closure letter" dated October 22, 1986, indicating that it
20 was satisfied with the final draft of the Air Quality
21 Criteria Document. The letter outlined key issues and
22 recommendations; it is in the docket for today's decision
23 (A-92-17). The EPA released the 1986 draft final Air
24 Quality Criteria Document in August 1986.

25 After the CASAC meeting on March 4-6, 1985, the EPA's
26 Office of Air Quality Planning and Standards (OAQPS) began

1 work on the first draft of the Staff Paper (Review of the
2 National Ambient Air Quality Standards for Ozone:
3 Assessment of Scientific and Technical Information--OAQPS
4 Staff Paper). (The Staff Paper is an assessment of
5 scientific and technical information contained in the 1986
6 draft final Air Quality Criteria Document and other related
7 exposure and risk assessment documents, and it presents
8 staff recommendations to the Administrator regarding primary
9 and secondary standards.) At a public meeting on April 21-
10 22, 1986, the CASAC reviewed the first draft of the Staff
11 Paper. The CASAC recommended that OAQPS staff consider new
12 information on prolonged exposure effects of O₃ in a second
13 draft of the Staff Paper prior to closure. The CASAC
14 reviewed this second draft in a public meeting of the CASAC
15 held on December 14-15, 1987. Staff of the EPA's Health
16 Effects Research Laboratory (HERL) and Corvallis
17 Environmental Research Laboratory (CERL) made presentations
18 on new and emerging information on health effects of
19 prolonged exposures to O₃ and on alternative indicators of
20 crop loss damage. The CASAC concluded that sufficient new
21 information existed to recommend incorporation of relevant
22 new information into a supplement to the 1986 Air Quality
23 Criteria Document (Supplement) and in a third draft of the
24 Staff Paper.

25 In early 1988, the EPA began working concurrently on a
26 Supplement and a third draft of the Staff Paper. The ECAO

1 staff prepared a draft Supplement titled "Summary of
2 Selected New Information on Effects of Ozone on Health and
3 Vegetation: Draft Supplement to Air Quality Criteria for
4 Ozone and Other Photochemical Oxidants." The EPA made
5 available copies of both the draft Staff Paper and draft
6 Supplement to the CASAC and the public in November 1988.

7 The CASAC held a public meeting on December 14-15, 1988
8 to review the draft Supplement and draft Staff Paper. Major
9 issues included: the definition of adverse health effects
10 of O₃, the significance of health studies suggesting that
11 prolonged exposures of 6 to 8 hours to O₃ levels at or below
12 0.12 ppm may produce respiratory impairment, the possibility
13 that chronic irreversible effects may result from lifetime
14 exposures to elevated levels of O₃, and the importance of
15 considering analyses which indicate agricultural crop damage
16 may be better defined by a cumulative seasonal average than
17 by a 1-hour peak level of O₃. In its "closure letter" of
18 May 1, 1989 (reprinted as Appendix I to this notice), the
19 CASAC indicated that the draft Supplement and draft Staff
20 Paper "provide an adequate scientific basis for the EPA to
21 retain or revise primary and secondary standards for ozone."
22 The CASAC concluded that it would be some time before enough
23 new information on the health effects of multihour and
24 chronic exposure to O₃ would be published in scientific
25 journals to receive full peer review and, thus, be suitable
26 for inclusion in a criteria document. The CASAC further

1 concluded that such information could better be considered
2 in the next review of the O₃ standards. The CASAC also
3 noted that the form of the secondary standard was of
4 critical importance in protecting against O₃ effects on
5 vegetation and that a cumulative seasonal average would be
6 more appropriate than a 1-hour standard. The CASAC went on
7 to add that if a more appropriate form could not be
8 developed, the Committee was of the opinion that serious
9 consideration be given to lowering the secondary standard to
10 0.10 ppm. The CASAC strongly endorsed the need for
11 accelerated and expanded research related to multiple hour,
12 seasonal, and lifetime human exposures to O₃ as well as
13 research related to effects of O₃ on forests and ecosystems.

14 D. Decision Docket

15 On March 17, 1992, the EPA created a docket (Docket No.
16 A-92-17) for this proposed decision. The docket
17 incorporates by reference the standard review docket (Docket
18 No. OAQPS A-83-04), created in 1983, and the separate docket
19 established for criteria document revision (Docket No. ECAO-
20 CD-81-1), created in 1981.

21 E. Pending Litigation

22 On October 22, 1991, the American Lung Association and
23 other plaintiffs filed suit to compel the EPA to complete
24 its review of the criteria and standards for O₃ under
25 section 109(d)(1) of the Act. American Lung Association v.
26 Reilly, No. 91-CV-4114 (JRB) (E.D.N.Y.). The U.S. District

1 Court for the Eastern District of New York subsequently
2 issued an order requiring the EPA to sign a Federal Register
3 notice announcing its proposed decision on whether to revise
4 the standards for O₃ by August 1, 1992 and to sign a Federal
5 Register notice announcing its final decision by March 1,
6 1993. The order also requires the EPA to use rulemaking
7 procedures in making the proposed and final decisions.

8
9 II. Rationale for Proposed Decision

10 This proposed decision is based on a careful
11 examination of available information on health and welfare
12 effects of O₃ and other photochemical oxidants contained in
13 the 1986 Air Quality Criteria Document and its Supplement,
14 which includes an evaluation of key studies published
15 through early 1989; the 1989 Staff Paper assessment of the
16 most relevant information in these documents; and the advice
17 and recommendations of the CASAC as presented both in the
18 discussion of these documents at public meetings and in the
19 Committee's 1986 and 1989 "closure letters." The
20 Administrator has not taken into account more recent studies
21 that have been published in the scientific literature. As
22 discussed in Section III (Continuing Review of Air Quality
23 Criteria and Standards), some of these studies are of
24 concern because they report health effects occurring at
25 lower exposures to O₃. Additional information has also
26 become available on the welfare effects of O₃. These

1 studies were not assessed, however, in the 1986 Air Quality
2 Criteria Document nor its Supplement, nor have they
3 undergone the rigorous review process (including CASAC
4 review) required for incorporation into a new criteria
5 document.

6 Under section 109 of the Act, primary and secondary
7 NAAQS are to be based on the air quality criteria, and the
8 criteria are to be thoroughly reviewed by the CASAC. The
9 NAAQS can have profound economic, social, as well as
10 environmental consequences, and in the periodic-review
11 provisions of section 109(d), Congress obviously recognized
12 that periodic decisions on whether to revise them be based
13 on discrete sets of studies that had been rigorously
14 assessed and incorporated into air quality criteria. In
15 practice, the statutory scheme necessarily involves some
16 delay, often a substantial delay, between completion of a
17 criteria document and a final decision on whether to revise
18 the corresponding NAAQS; studies published after completion
19 of the criteria document ordinarily become the basis for the
20 next round of review. Otherwise, review and revision of
21 criteria documents would be an endless process because of
22 the continuous need to incorporate new studies.

23 The Administrator, of course, retains discretion under
24 section 109(d)(1) to revise criteria documents more
25 frequently than the statute requires. In some cases, the
26 CASAC has recommended or the EPA has decided that studies

1 published after completion of a criteria document warrant
2 deferral of a decision on whether to revise the
3 corresponding NAAQS pending supplementation of the criteria
4 document. As noted above, the CASAC made such a
5 recommendation for O₃ in 1987, and the EPA decided to follow
6 it. Aside from any statutory constraints, the exercise of
7 this discretion involves a judgment by the Administrator as
8 to whether, in the circumstances, the newer studies are of
9 such significance as to warrant substantial delay in
10 completing the review cycle. In this case, the current
11 review of the O₃ standards began a decade ago, and the EPA
12 has missed both the 1985 and 1990 deadlines for completion
13 of review cycles under section 109(d). In view of the
14 number and nature of the studies published since early 1989,
15 the substantial time that would be required to incorporate
16 them into a supplement to the 1986 Air Quality Criteria
17 Document and provide appropriate opportunities for public
18 comment, and the length of time since the EPA began its
19 current review of the standards, the Administrator believes
20 there should be no further delay in completing the current
21 review cycle. As a practical matter, the EPA has little
22 choice given the limited time available under the Court
23 order in the American Lung Association case. As discussed
24 in Section III, however, the EPA plans to proceed as rapidly
25 as possible with the next review of the criteria and
26 standards for O₃.

1 Having limited his consideration to information
2 contained in the 1986 Air Quality Criteria Document, the
3 subsequent Supplement, and the 1989 Staff Paper, and taking
4 into account the CASAC's advice and recommendations, the
5 Administrator focused on a discrete range of policy options
6 for revising or not revising the current O₃ standards. The
7 options included addressing the following questions:

8 1) Is sufficient health effects information available
9 to warrant the replacement (or supplementation) of the
10 current 1-hour primary standard with a new 6- to 8-hour
11 standard to protect against prolonged exposures and to
12 provide additional protection for the most sensitive
13 group(s)?

14 2) Is sufficient health effects information available
15 to provide the basis for the establishment of a seasonal or
16 other long-term standard to protect against possible chronic
17 effects in the exposed population?

18 3) Should the level of the current 1-hour primary
19 standard be revised from 0.12 ppm to 0.10 ppm?

20 4) Should the level of the current 1-hour secondary
21 standard be revised from 0.12 ppm to 0.10 ppm, or should a
22 new seasonal standard be established?

23 A. The Primary Standard

24 1. Basis for the Existing 1-Hour Standard

25 In selecting the level for the current 1-hour primary
26 standard in 1979, the Administrator made judgments regarding

1 lowest reported effect levels, sensitive populations, nature
2 and severity of health effects, and margin of safety. The
3 judgment of the lowest observed effect level was based
4 largely on several human clinical studies. The key study
5 was by DeLucia and Adams (1977) who reported symptoms of
6 discomfort and small but statistically nonsignificant lung
7 function decrements in vigorously exercising healthy
8 subjects acutely exposed to O_3 at concentrations as low as
9 0.15 ppm O_3 . The principal sensitive group of concern in
10 setting the 1979 O_3 primary standard was asthmatics,
11 although the EPA recognized that nonasthmatic individuals
12 engaged in exercise are also potentially vulnerable to
13 acutely irritating effects of O_3 . In addition, impaired
14 pulmonary function and symptoms were recognized as the best-
15 documented effects in human clinical studies. More severe
16 effects such as decreased resistance to respiratory
17 infection, induction of chronic respiratory disease, and
18 possible carcinogenic/mutagenic effects also had been
19 reported in animal toxicology studies, and some limited
20 epidemiology studies raised concern about possible
21 aggravation of pre-existing chronic respiratory disease.
22 However, uncertainties regarding these data limited their
23 usefulness in estimating an adverse effects level.

24 . Finally, in selecting a standard level intended to
25 provide an adequate margin of safety, the Administrator
26 noted that the available quantitative information on human

1 health effects of O₃ was quite limited. For that reason and
2 because the more qualitative information in the health
3 criteria suggested the possibility of adverse effects
4 occurring below 0.15 ppm O₃, the Administrator concluded
5 "that a standard of 0.12 ppm is necessary and prudent unless
6 and until further studies demonstrate reason to doubt that
7 it adequately protects public health."

8 2. Health Effects Information Since 1979

9 Since 1979, the available information on health effects
10 caused by acute (1- to 3-hour) exposures to O₃ has expanded
11 greatly. Additional new information on prolonged (6- to 8-
12 hour) exposures began to appear in the scientific literature
13 during the late 1980's and continues to be published at this
14 time. Although some information on chronic effects was
15 available in 1979, a significant body of data from animal
16 studies has been published during the past decade confirming
17 damage caused by chronic O₃ exposures. All key information
18 available up to early 1989 has undergone careful review for
19 incorporation in the 1986 Air Quality Criteria Document, the
20 Supplement, and the 1989 Staff Paper.

21 a. Effects of 6- to 8-Hour Exposures

22 Evidence of enhanced effects from prolonged exposures
23 to O₃ began to appear in 1985. Liroy et al. (1985) and
24 Spektor et al. (1988a,b) conducted summer camp field studies
25 of children engaged in outdoor activity for periods of
26 several days to weeks, during which they were exposed to

1 ambient O₃ for several hours per day. These studies
2 indicated that statistically-significant pulmonary function
3 decrements, compared to initial baseline values, could be
4 measured even when the O₃ NAAQS were not exceeded. The
5 effects increased with exposure to increasing levels of O₃.
6 Health effects reported in the summer camp studies could be
7 attributed in part to factors such as other pollutants or
8 heat.

9 Multihour human exposure studies were conducted to
10 assess the effects of prolonged exposure to O₃ alone in a
11 controlled environment. These studies (Folinsbee et al.,
12 1989; Horstman et al., 1988, 1989) exposed subjects engaged
13 in intermittent, moderate to heavy exercise (minute
14 ventilation, $\dot{V}_E = 40$ liters/minute; e.g., brisk walking or
15 easy cycling) for 6.6 hours to O₃ levels of 0.08, 0.10, and
16 0.12 ppm. They reported small but statistically-significant
17 group mean decreases in lung function, measured as forced
18 expiratory volume (FEV_{1.0}), at all three exposures compared
19 to filtered air. (The exposure protocol was designed to
20 simulate a normal workday for a construction worker.)
21 Respiratory symptoms (e.g., cough, pain on deep inspiration)
22 were more evident at 0.12 ppm than at 0.08 ppm O₃.

23 Biochemical indicators of pulmonary inflammation (i.e.,
24 cells and other mediators of a lung inflammatory response)
25 were also reported to increase in healthy subjects exposed
26 for 6.6 hours to 0.10 ppm O₃ while engaging in intermittent,

1 moderate to heavy exercise ($\dot{V}_E = 40$ liters/minute) (Koren et
2 al., 1988a). More specifically, this and other research
3 (Koren et al., 1988b,c) demonstrate that cells and soluble
4 mediators suggestive of possible damage to pulmonary tissue
5 are increased as a result of prolonged O_3 exposures. The
6 significance of these results lies in the fact that they
7 represent indicators of inflammation in humans and potential
8 for damage in lower airways from O_3 exposures which often
9 occur in ambient air of many cities during the summer.

10 The CASAC "closure letter" of May 1, 1989, stated,
11 "While reaching closure at this time, the Committee did note
12 an emerging data base on the acute health effects resulting
13 from 6-plus hours of O_3 exposure, providing evidence of the
14 possible need for a standard with a 6-8 hour averaging time.
15 However, it was the Committee's view that it would be some
16 time before enough of this developing information would be
17 published in scientific journals to receive full peer review
18 and, thus, be suitable for inclusion in a criteria document.
19 The CASAC concluded such information can better be
20 considered in the next review of the ozone standards."
21 Although the studies cited above are of concern to the EPA,
22 they have not yet been confirmed in other laboratories.
23 Similar research is currently underway in other laboratories
24 and should be available for a subsequent review of air
25 quality criteria. For these reasons, the Administrator

1 concurs with the CASAC that this information should be
2 considered in the next review of the O₃ standards.

3 b. Effects of Seasonal or Chronic Exposures

4 Evidence of seasonal or chronic effects of O₃ has
5 accumulated in the animal toxicology literature. Chronic
6 and subchronic effects such as inflammation and structural
7 changes in respiratory tissue, increased collagen content in
8 the lungs, and impaired ability to resist respiratory
9 infection, have been well-documented in animal toxicology
10 studies. Quantitative extrapolation of these effects
11 reported in animals to human health effects remains limited
12 by inadequate knowledge of dosimetry and species sensitivity
13 differences.

14 Although the 1989 CASAC "closure letter" expressed
15 concern for the possibility that chronic irreversible
16 effects may result for people exposed to O₃ over a lifetime,
17 the CASAC concluded that such changes have not yet been
18 demonstrated. The CASAC also concluded that "there is not
19 an adequate data base on the effects of multiple hour or
20 seasonal exposures to O₃, especially as regards whether such
21 exposures may produce chronic health effects. This is
22 especially troubling since such long-term exposures to O₃
23 occur in many parts of the United States and involve many
24 millions of people It is critical that the data
25 base on health and welfare effects related to multiple hour,

1 seasonal and lifetime exposures of O₃ be increased through
2 an accelerated and expanded research effort."

3 Several chronic animal studies by the EPA, the National
4 Toxicology Program (NTP), and the Health Effects Institute
5 (HEI) are expected to be available in time for the next O₃
6 criteria review cycle. Animal toxicology studies at the EPA
7 are assessing the health effects in rats of chronic exposure
8 to O₃, and a cooperative effort between the NTP and the HEI
9 is focused on potential carcinogenic and cocarcinogenic, as
10 well as morphological effects of chronic O₃ exposures. The
11 EPA, in cooperation with New York University, is also
12 conducting an epidemiological field study to investigate the
13 effects of chronic exposure to O₃ and other irritants on
14 lung function development in healthy young adults. Results
15 of many of these studies should elucidate some, but not all,
16 of the chronic effects issues.

17 c. Effects of 1- to 3-Hour Exposures

18 The 1986 Air Quality Criteria Document reflected a
19 greatly expanded data base on effects from short-term
20 exposures to O₃ of 1 to 3 hours in healthy individuals.
21 Controlled human exposure studies (McDonnell et al., 1983;
22 Gong et al., 1986) reported small, but statistically
23 significant, transient declines in pulmonary function (e.g.,
24 reductions in lung volume and air flow), which in some cases
25 were accompanied by symptoms (e.g., cough, chest pain,
26 throat irritation, shortness of breath) during exposures to

1 - O₃ in the range of 0.12 to 0.15 ppm. These effects,
2 however, were reported only when subjects engaged in very
3 heavy exercise ($\dot{V}_E$ = 68-89 liters per minute). Such
4 exercise levels typically occur when a person engages in
5 activities like running or cycling. It should be noted,
6 however, that without heavy exercise even the most sensitive
7 subjects will not experience statistically-significant
8 decrements in lung function (FEV_{1.0}) at low-level O₃
9 exposures (around 0.12 ppm after 1 to 3 hours); and
10 furthermore, the magnitude of effects which can be measured
11 at these exposure levels, even with heavy exercise, is not
12 generally considered to be of great health significance. A
13 generally accepted relationship is that for any given
14 individual, the greater the exercise level during exposure
15 to O₃, the greater the short-term pulmonary function
16 response experienced (U.S. EPA, 1986, p. 12-81).

17 One of the key issues that emerged during review of
18 these and other studies was the high degree of variability
19 in responsiveness between individuals exposed to similar O₃
20 levels. This was evident from the number of studies
21 (Gibbons and Adams, 1984; Linn et al., 1986; Avol et al.,
22 1984; Schelegle and Adams, 1986) that found no
23 statistically-significant response at exposures (0.12 to
24 0.15 ppm O₃) and exercise levels ($\dot{V}_E$ = 55 to 86 liters per
25 minute) similar to those in the above-cited studies. In two
26 of these studies (Avol et al., 1984; Linn et al., 1986)

1 statistically significant changes in FEV_{1.0} began to appear
2 at 0.16 ppm O₃.

3 Although the group mean lung function decrements may be
4 only 1 to 4 percent for the lower level O₃ exposures, the
5 1986 Air Quality Criteria Document (U.S. EPA, 1986, p. 12-
6 22) concluded that between 5 and 20 percent of otherwise
7 healthy individuals may be more responsive to O₃ during
8 exercise and, therefore, would be at higher risk to O₃
9 exposures. For example, McDonnell et al. (1983) analyzed
10 intersubject variability in a study involving 135 healthy
11 young males who were exposed to various O₃ levels (0.12 ppm
12 to 0.4 ppm) during 2 hours of intermittent, very heavy
13 exercise. When the subjects were exposed to 0.18 ppm, the
14 study reported changes in FEV_{1.0} ranging from 0 to -23
15 percent, with a group mean of -6 percent. Subjects exposed
16 to 0.12 ppm O₃ experienced changes ranging from +7 to -16
17 percent, with a group mean decrement in FEV_{1.0} of -4
18 percent. Kulle et al. (1985) exposed each of their 20
19 subjects to various O₃ concentrations for 2 hours with
20 heavy, intermittent exercise. They reported changes in
21 FEV_{1.0} of +10 to -4 percent, with a group mean of +1 percent
22 at 0.10 ppm O₃. The response increased to +3 to -9 percent
23 (group mean of -1 percent) at 0.15 ppm O₃. At 0.2 ppm O₃,
24 the FEV_{1.0} decrements increased to +3 to -16 percent, with a
25 group mean response of -3 percent. At concentrations below
26 0.18 ppm O₃, these effects would not be noticed by most

1 healthy individuals. For these studies, the effects
2 experienced by even the most sensitive individuals acutely
3 exposed to 0.12 to 0.15 ppm O₃ ranged from -9 to -16 percent
4 decline in FEV_{1.0} with few, if any, symptoms that would be
5 considered mild to moderate by many health experts (U.S.
6 EPA, 1989, p. VII-53).

7 The EPA staff made several other observations regarding
8 health effects from short-term exposures to O₃. Exercise
9 performance is reportedly not affected in very heavily-
10 exercising ($\dot{V}_E$ = 86-88 liters per minute) individuals
11 exposed to 0.12 ppm O₃ for 1 hour; the effects are, however,
12 intensified for exposures of 0.18 to 0.24 ppm, resulting in
13 the inability of some subjects to complete the protocol
14 (Schelegle and Adams, 1986; Gong et al., 1986). Increased
15 airway reactivity to bronchoconstrictors has been observed
16 in heavily-exercising ($\dot{V}_E$ = 70 liters per minute)
17 individuals after 2-hour exposures to 0.18 ppm O₃ (McDonnell
18 et al., 1987). Increased presence of cells and other
19 mediators of lung inflammation have been reported at 18
20 hours post exposure in heavily-exercising ($\dot{V}_E$ = 64 liters
21 per minute) subjects exposed for 2 hours to 0.4 ppm O₃
22 (Koren et al., 1988a,b,c). These studies of inflammatory
23 response prompt concern that repeated or chronic exposures
24 to high levels of O₃ may result in permanent lung tissue
25 damage.

1 Finally, although epidemiological evidence (Whittemore
2 and Korn, 1980; Holguin et al., 1985; Bates and Sizto, 1987,
3 1989; Lebowitz et al., 1982, 1983) has suggested that O₃ and
4 other photochemical oxidants may be associated with
5 increased asthma attack rates, excess respiratory hospital
6 admissions, and lung function decrements in asthmatics,
7 uncertainty associated with these data make it difficult to
8 determine a clear cause-effect relationship or an
9 appropriate exposure averaging time for the reported
10 responses.

11 3. Proposed Decision Not to Revise the Primary
12 Standard

13 The Administrator is proposing not to revise the
14 existing O₃ primary standard at this time. In reaching this
15 proposed decision, the Administrator has fully considered
16 the health effects information assessed in the 1986 Air
17 Quality Criteria Document, the Supplement that updated that
18 information, the 1989 Staff Paper, and the advice and
19 recommendations of CASAC in its 1989 "closure letter."

20 The Administrator agrees with the staff and CASAC
21 conclusions that the preliminary information on effects of
22 prolonged exposures to O₃ contained in the 1986 Air Quality
23 Criteria Document and the Supplement is not sufficient to
24 support the establishment of a new 6-8 hour standard to
25 protect against prolonged exposures, or a seasonal or other
26 long-term standard to protect against chronic effects. In

1 reaching this proposed decision, the Administrator
2 recognizes that a number of new studies, particularly on 6-8
3 hour exposures to O₃, have been published in the scientific
4 literature since completion of the air quality criteria that
5 serve as the basis for today's decision (see Section III).
6 The EPA intends to proceed with the next periodic review of
7 the air quality criteria as rapidly as possible so that the
8 implications of these new studies can be given early
9 consideration. The Administrator is also mindful that there
10 is research in progress on the chronic effects of O₃ that
11 should become available in the next 1 to 2 years. When this
12 new information has been incorporated into the air quality
13 criteria, a more informed decision can be made as to whether
14 adding a new 6-8 hour standard and/or a seasonal or other
15 long-term standard would be appropriate.

16 The Administrator also carefully considered the health
17 effects information on short-term exposures to O₃ contained
18 in the 1986 Air Quality Criteria Document and its
19 Supplement. As contrasted to the limited information on
20 health effects of O₃ available in 1979, by 1989 information
21 on 1- to 3-hour exposures had expanded greatly. The EPA
22 staff and the CASAC identified several factors that the
23 Administrator should consider in reaching a decision on
24 whether or not to revise the current primary standard to
25 protect against short-term exposures to O₃. These include:
26 a) the sensitive populations affected by O₃, b) the nature

1 and severity of the effects, and c) the protection afforded
2 by the current standards.

3 a. Sensitive Populations Affected

4 There are two groups identified as being at potential
5 risk from acute exposures to O₃ (U.S. EPA, 1986, p. 1-164).
6 As discussed in the 1986 Air Quality Criteria Document, the
7 first is that group in the general population characterized
8 as having preexisting respiratory disease (e.g., asthma or
9 chronic obstructive lung disease). These individuals are
10 not more responsive than healthy individuals in terms of the
11 magnitude of pulmonary function decrements seen at typical
12 exposure levels and durations; however, the consequent
13 impact of O₃-induced responses in their already compromised
14 respiratory systems may more noticeably impair their ability
15 to function adequately. Also, limitations on using such
16 individuals in experimental studies have prevented an
17 adequate assessment of the full range of potential responses
18 to O₃ or their health significance in these individuals.

19 The second group at increased risk to acute O₃
20 exposures is the general population of healthy individuals
21 who exercise at elevated O₃ levels and, in addition, show an
22 unusual responsiveness to O₃. Exercise increases the amount
23 of O₃ entering the airways and can cause O₃ to penetrate to
24 peripheral regions of the lung where lung tissue is more
25 sensitive and more damage can occur. Individuals who are
26 unusually responsive to O₃ experience greater decrements in

1 lung function from exposure to O_3 than the average response
2 of the groups studied. As yet, there are no means to
3 determine in advance which persons will be unusually
4 responsive to O_3 , but estimates based on subjects already
5 studied suggest 5 to 20 percent of the general population
6 may show a substantially greater response than average. It
7 is not clear whether these individuals constitute a
8 population subgroup with a specific risk factor or simply
9 represent the upper 5 to 20 percent of the O_3 response
10 distribution (U.S. EPA, 1989, p. III-12).

11 b. Nature and Severity of Effects

12 Ozone acts as a pulmonary irritant when it comes into
13 contact with the mucous or surfactant layer lining the
14 respiratory tract. Because O_3 is chemically quite reactive,
15 it tends to react rapidly with the mucous layer, thus
16 causing increased total absorption in the upper airways and
17 a reduction in O_3 reaching the more sensitive tissues deeper
18 in the lungs. Exercise, particularly heavy exercise, will
19 increase the total mass of O_3 inhaled per unit time and will
20 change patterns of O_3 deposition in the lungs, thereby
21 causing responses in persons who otherwise might not be
22 affected.

23 (1) Respiratory Function Decrements and Symptoms. The
24 principal responses associated with acute exposures to O_3
25 are respiratory function decrements and symptoms. As
26 discussed above, individuals exposed to lower levels of O_3

(e.g., 0.12 to 0.15 ppm) typically experience only mild and transient functional decrements. The available data also suggest that many responders would experience only mild to moderate reductions in lung function which may be accompanied by symptoms such as cough, chest tightness, pain on deep inspiration, and throat irritation. At higher O₃ concentrations, reductions in lung function and symptoms become more pronounced.

Most healthy individuals experiencing mild to moderate O₃-induced lung function decrements may not notice such effects due to their substantial reserve capacity; however, individuals who have preexisting respiratory disease or have hyperreactive airways may respond to O₃ exposure sufficiently to restrict normal activity or impair their performance in carrying out tasks. While such possible outcomes are a matter of concern, the staff concluded that the data on individuals with preexisting respiratory disease were limited and should only be considered in developing a margin of safety (U.S. EPA, 1989, p. VII-28).

(2) Decreased Resistance to Respiratory Infection.

This effect of O₃ has been demonstrated in experimental animal studies. The biological basis for this response appears to be that O₃ or one of its reactive products impairs or suppresses normal bactericidal functions of the pulmonary defense system components (e.g., alveolar macrophages). This results in prolonging the life of the

1 infectious agent, thus permitting its multiplication and
2 ultimately resulting in death in this animal infectivity
3 model. Because these effects have been reported in several
4 species of animals and are potentially serious, the EPA
5 remains concerned about the possibility of increased
6 susceptibility to respiratory infection in humans in
7 response to ambient O₃ exposures.

8 (3) Pulmonary Inflammation and Structural Changes in
9 Respiratory Tissue. Pulmonary inflammation and structural
10 changes in respiratory tissue have also been a focus of
11 concern. One series of studies (Koren et al., 1988 a,b,c)
12 reported biochemical and cellular indicators of pulmonary
13 inflammation in healthy adult males exposed for 2 hours to
14 0.4 ppm O₃ during intermittent, heavy exercise ($\dot{V}_E = 70$
15 liters per minute); however, acute exposures involving lower
16 concentrations have not been tested to determine the lowest
17 level causing these effects. While these studies of
18 inflammatory response prompt concern that repeated or
19 chronic exposures to high levels of O₃ may result in
20 permanent lung tissue damage, such a linkage currently
21 remains hypothetical.

22 c. Proposed Decision Not to Revise

23 Based on the staff's assessment of the health
24 information discussed above and taking into account the
25 advice and recommendations that the CASAC provided in 1989,
26 it is the Administrator's judgment that the air quality

1 criteria do not provide a sufficient basis for revising the
2 existing 1-hour primary standard. The standard level is
3 below those levels that were found in the controlled human
4 studies to be associated with major lung function decrements
5 and symptoms. In reaching this conclusion, the
6 Administrator is mindful that the mean group response
7 observed in the controlled human studies up to 0.15 ppm O₃
8 would at most be characterized as mild, and that most of the
9 responders within this population of normal healthy
10 individuals reportedly experienced only mild to moderate
11 responses under very heavy exercise. Although there is a
12 difference of opinion among the EPA's scientific advisors as
13 to the significance of decrements in lung function in the
14 range of 10 to 20 percent when accompanied by symptoms, it
15 is the Administrator's judgment that the lesser effects
16 associated with exposure to O₃ in the range of 0.12 ppm to
17 0.15 ppm observed in the controlled human studies do not
18 constitute adverse effects for purposes of section 109 of
19 the Act.

20 The Administrator also considered other sensitive
21 population groups whose response to O₃ has not been fully
22 characterized. Although some epidemiology studies
23 considered in the 1986 Air Quality Criteria Document and its
24 Supplement suggest that exposure to O₃ at ambient
25 concentrations may result in the aggravation of asthma and
26 preexisting respiratory disease, the Administrator concurred

1 with the staff view that these studies are limited by
2 uncertainties about individual exposure levels and the role
3 of other pollutants and, therefore, should not be
4 generalized to the entire population. In addition, although
5 individuals with preexisting lung disease are not more
6 responsive to O₃ than healthy persons, the same small change
7 in pulmonary function may have more impact on people whose
8 lung function is already compromised. While all of these
9 studies suggest that these sensitive groups may be at
10 somewhat greater risk at levels of 0.12 ppm O₃ and higher,
11 compared to normal healthy individuals in controlled human
12 exposure studies, in the Administrator's judgment these
13 studies do not provide a sufficient basis for lowering the
14 existing standard.

15 Finally, the Administrator took into account the O₃
16 Risk Assessment, as discussed in the 1989 Staff Paper (U.S.
17 EPA, 1989, pp. VII-44 to VII-49). This assessment evaluated
18 health effects associated with short-term exposure to O₃
19 under several different air quality scenarios. Using
20 current air quality and simulating air quality if
21 alternative 1-hour standards were just attained at 0.12,
22 0.10 and 0.08 ppm, the assessment found that by far the
23 largest reduction in short-term health effects of O₃ would
24 be achieved by bringing areas into attainment with the
25 current 1-hour standard of 0.12 ppm. Only small incremental
26 gains would be made by attaining more stringent NAAQS.

1 As discussed above and in Section III, the emerging
2 information on 8-hour and chronic or seasonal exposures is
3 also of concern. In view of this, the Administrator
4 considered to what extent attainment of the current standard
5 would reduce 8-hour and longer-term seasonal averages. Air
6 quality relational analyses indicate that multihour averages
7 of O₃ would be reduced if the current 1-hour standard is
8 attained (see U.S. EPA, 1989, Appendix A). As control
9 programs are implemented to reduce 1-hour O₃ peak levels, 8-
10 hour and longer-term seasonal averages also will be reduced
11 because most control strategies aimed at attaining the
12 existing 1-hour standard are not time-of-day specific. Such
13 programs will affect every hour of the day to a greater or
14 lesser extent and, thus, lower the entire distribution of O₃
15 air quality and not just peak concentrations. As a result,
16 the Administrator believes the major control programs
17 required by the 1990 Clean Air Act Amendments should result
18 in notable progress towards bringing the country into
19 attainment with the existing 1-hour standard and should also
20 lower O₃ levels associated with 8-hour and seasonal
21 averaging periods.

22 Given the above, and the preliminary nature of the
23 information currently assessed in the air quality criteria
24 on 6- to 8-hour exposures, the Administrator is inclined not
25 to set a new 8-hour standard or lower the level of the 1-
26 hour NAAQS to 0.10 ppm, as some have suggested. In the

1 Administrator's judgment, the more prudent course of action
2 is (1) to proceed as rapidly as possible with assessment of
3 the new studies that have not been considered in this review
4 so that a more informed decision can be made on the need for
5 additional protection from 6- to 8-hour and chronic
6 exposures, and (2) to focus on fully implementing the
7 control programs mandated by the Clean Air Act Amendments of
8 1990.

9 For the above reasons, the Administrator proposes not
10 to revise the existing primary standard at this time. The
11 Administrator also considered and concurs with the staff
12 recommendations that O₃ should remain as the surrogate for
13 controlling ambient concentrations of photochemical oxidants
14 and that the existing form of the standard should be
15 retained.

16 B. The Secondary Standard

17 The Administrator also proposes not to revise the O₃
18 secondary NAAQS at this time. The rationale for this action
19 is threefold: (1) the correct form and level for a new
20 standard that would offer increased or improved protection
21 of crops, and forest ecosystems are difficult to determine,
22 given the data currently reviewed by the CASAC; (2) new
23 research is currently underway to reduce this uncertainty.
24 for forest ecosystems; and (3) tightening the current 1-hour
25 standard as an interim measure would provide only marginal
26 improvement because a 1-hour averaging period is not the

1 most appropriate exposure indicator and most likely would be
2 superseded by adoption of a new standard form during the
3 next standard review. Section 109(b)(2) of the Act requires
4 the EPA to set a secondary NAAQS at a level that, in the
5 judgment of the Administrator, is requisite to protect the
6 public welfare from any known or anticipated adverse
7 effects. The term "public welfare," which is defined in
8 section 302(h) of the Act, includes, among other things,
9 effects on soils, water, crops, vegetation, wildlife,
10 visibility, manmade materials, animals, hazards to
11 transportation, and climate, as well as effects on economic
12 values and on personal comfort and well-being.

13 During the first review of O₃ NAAQS in the late 1970's,
14 the EPA carefully examined the scientific and technical
15 information evaluated in the then-revised air quality
16 criteria concerning O₃-related damage to vegetation, crops,
17 materials, and visibility. As part of this process, the EPA
18 developed a staff assessment entitled "Evaluation of
19 Alternative Secondary Ozone Air Quality Standards." Based
20 on this assessment and other relevant factors, the EPA
21 promulgated a revised secondary standard on February 8, 1979
22 (44 FR 8202) that was identical to the revised primary
23 standard of 0.12 ppm in all respects. In reaching this
24 decision, the Administrator concluded that a secondary
25 standard more stringent than the primary standard was not
26 necessary to adequately protect public welfare.

1 The current review has focused mainly on effects of O₃
2 on agricultural crops and forests. Consideration has also
3 been given to the effects of O₃ on materials and on personal
4 comfort and well-being.

5 1. Effects on Agriculture and Forests

6 a. Effect on Crops

7 The 1979 decision to revise the secondary O₃ NAAQS
8 resulted largely from a lack of evidence adequate to retain
9 a standard more stringent than the primary. The 1978
10 Criteria Document identified the need for "a set of standard
11 equations that would relate plant response to pollutant
12 concentration and duration of exposure and would also
13 incorporate the effects of all other factors that control
14 the responses of plants." The 1978 Criteria Document also
15 recognized that "Development of such equations requires a
16 data base sufficient to relate a given dose (concentration
17 of pollutant times duration of exposure) of oxidant (e.g.,
18 O₃, PAN) to some meaningful plant effect" and that "Such
19 equations are not yet available." (U.S. EPA, 1978, p. 264).

20 To address this fundamental deficiency, in 1980 the EPA
21 initiated a 5-year research program titled the National Crop
22 Loss Assessment Network (NCLAN) to define the relationships
23 between yields of major agricultural crops and O₃ exposure,
24 to assess national economic consequences of the exposure of
25 major agricultural crops to O₃, and to advance the
26 understanding of the cause-effect relationships that

1 determine crop responses to pollutant exposure. The NCLAN
2 research program on crops, completed in 1985, provided
3 valuable exposure-response information on a variety of crops
4 and strengthened the evidence for O₃-induced yield
5 reductions in important commercial crop species.

6 Because of this, the NCLAN data base became a principal
7 focus of the current assessment of yield reductions in
8 commercially important crops exposed to O₃. Yield reduction
9 or loss is defined as impairment of, or decrease in, the
10 value of the intended use of the plant. This definition
11 includes reduction in aesthetic values, changes in crop
12 quality, and occurrence of foliar injury when foliage is a
13 marketable part of the plant, as well as loss in weight or
14 bulk.

15 The EPA has analyzed data from the NCLAN to develop
16 predictive equations relating 7-hour seasonal mean O₃
17 exposures, the indicator used in the NCLAN studies, to crop
18 yield loss. These analyses suggest that a 10 percent mean
19 yield loss occurs for several species when the 7-hour
20 seasonal mean concentration of O₃ exceeds 0.04-0.05 ppm,
21 that grain crops are generally less sensitive to O₃ than
22 other crops, and that sensitivity differences within a
23 species may be as large as a difference between species. In
24 addition to differences in sensitivity among species and
25 cultivars, the available data also suggest the presence of

1 year-to-year variations in plant responses to O₃ (U.S. EPA,
2 1989, p. X-7).

3 The scientific community well recognizes that the NCLAN
4 data provide valuable exposure-response information for a
5 variety of crops. However, the adequacy of the 7-hour
6 seasonal mean as an exposure index has been questioned.
7 This seasonal exposure statistic is based on the mean 7-hour
8 daily concentration measured from 9:00 a.m. to 4:00 p.m.
9 averaged over the growing season. The use of a seasonal
10 mean to characterize exposures implies that all exposures
11 over the course of the daylight period are equally effective
12 in inducing plant responses. Several analyses, however,
13 indicate that constant concentrations have less effect on
14 plant growth responses than variable or episodic exposures
15 at equivalent cumulative doses (Musselman et al., 1983;
16 Hogsett et al., 1985). Thus, it is possible for two sites
17 with the same daytime arithmetic mean O₃ concentration to
18 have different estimated crop reductions (Larsen and Heck,
19 1984). The 7-hour seasonal mean also fails to account for
20 phenological stages of plant development, the impact of peak
21 concentrations, length of episodes, and days between peaks.

22 In addition to the NCLAN data, the 1986 Air Quality
23 Criteria Document and 1989 Staff Paper also assessed data on
24 the effects of O₃ on crop yield both under more controlled
25 conditions and under ambient air exposures. Data from the
26 controlled studies generally seem to indicate that O₃

1 concentrations of 0.10 ppm (frequently the lowest
2 concentration used in the studies) for a few hours a day,
3 over a period of several days to several weeks, induce yield
4 loss of 10-55 percent (U.S. EPA, 1989, pp. X-13 - X-15).
5 These studies further demonstrate that peak O₃
6 concentrations cause an effect. Because these studies were
7 conducted in greenhouses or growth chambers, it is difficult
8 to extrapolate the data to field conditions. However,
9 ambient air exposure studies that have been reviewed also
10 confirm that current ambient O₃ levels in many parts of the
11 country can reduce plant yield for some crops.

12 b. Forest Ecosystems

13 In addition to effects of O₃ on crops, there is
14 evidence, although regionally limited, that some forest
15 ecosystems have been adversely affected by ambient levels of
16 O₃. Among the susceptible areas are the mixed conifer
17 forests of the San Gabriel and San Bernardino mountain
18 ranges east of Los Angeles, where O₃ has been identified as
19 the agent responsible for the slow decline and death of the
20 ponderosa pine and the injury of the Jeffrey pine. The
21 decline of pines in the mixed conifer forest in the San
22 Bernardino Mountains suggests that a potential consequence
23 of O₃ stress is a change in the successional patterns and
24 composition of the forest (Miller et al., 1982). Oxidant
25 injury of eastern white pine and other native vegetation has
26 also been observed in the Eastern United States (U.S. EPA,

1 1989, p. X-25). Several studies have attributed reductions
2 in the growth of annual rings in eastern white pine to the
3 exposure of the trees to O_3 over a period of 10 to 20 years
4 (Mann et al., 1980; McLaughlin et al., 1982; Benoit et al.,
5 1982).

6 Dendrochronological studies of the decline of red
7 spruce in the northeast and of reduced growth rates of red
8 spruce, balsam fir, and fraser fir in central West Virginia
9 and western Virginia, also provide further evidence that the
10 reductions in growth and mortality measurable today probably
11 began at least 20 years ago (Johnson and Siccama, 1983;
12 Adams et al., 1985). In addition, reductions in growth
13 rates of loblolly and short leaf pine have been reported in
14 the piedmont regions of the southeastern United States
15 (McLaughlin, 1985). The magnitude of the role of O_3 in
16 these cases is unclear.

17 In regard to these most recent declines in growth,
18 there is currently no agreement as to the trigger factor
19 that precipitated the dieback, mortality, and decreased
20 growth. A number of stresses have been identified,
21 including both natural processes and air pollution (Johnson
22 and Siccama, 1983). Given the regional distribution of O_3
23 in North America, the frequent occurrence of elevated O_3
24 concentrations, and the recognized effects of O_3 on
25 agricultural productivity in the region, the potential
26 influence of O_3 on forest ecosystems is of concern. The

1 success and composition of producer species within a
2 community are the keys to "maintaining the integrity of an
3 ecosystem Any significant alterations in producers,
4 whether induced by O₃ or other stresses, can potentially
5 affect the consumer and decomposer populations of the
6 ecosystem, and can set the stage for changes in community
7 structure by influencing the nature and direction of
8 successional changes . . ." (U.S. EPA, 1986, p. 7-51).

9 While some of the same plant processes are affected in
10 trees and agriculture crop species, perennial plants,
11 because they live longer, must cope with both short- and
12 long-term stresses, the effects of which can be cumulative,
13 lasting over the years, or can be delayed, not becoming
14 apparent for many years. Likewise, effects can possibly be
15 mitigated through short- or long-term recovery or
16 replacement (U.S. EPA, 1986, p. 7-76). As a result, the
17 permanent vegetation in natural ecosystems receives much
18 greater chronic exposure than the short-lived vegetation
19 that makes up agroecosystems. The single agroecosystem has
20 little resilience to pollutant stress; the natural ecosystem
21 is initially more resistant to pollutant stress because of
22 species diversity, but the longer chronic exposures can
23 disrupt the system. As discussed more fully in the next
24 section, this difference between natural ecosystems and
25 agroecosystems raises a key issue when selecting an

1 appropriate exposure indicator for the secondary standard
2 (U.S. EPA, 1989, p. X-26).

3 In the CASAC's 1989 closure letter in the 1989 Staff
4 Paper, "the Committee took note of the lack of information
5 on the effects of O₃ on forest ecosystems and urged support
6 for research to remedy this deficiency."

7 In response to the CASAC comments and the deficiencies
8 in the data, the EPA's CERL began a Forest O₃ Research
9 Program to develop a data base on O₃ effects on forests and
10 to review alternative exposure indices for use in
11 formulating an appropriate O₃ secondary standard. The major
12 objectives of the Forest O₃ Research Plan are to (1)
13 identify the most critical aspects of O₃ exposure dynamics
14 (i.e., level, frequency, duration, time of day) through
15 mechanistic studies of O₃ uptake and the relevance of
16 environmental, genetic and cultural factors; (2) develop
17 exposure-response functions for seedlings, saplings, and
18 mature trees exposed to current and changing O₃ levels and
19 assess the role of size and age in their responses; (3)
20 parameterize a process model of tree growth using the data
21 developed in (1) and (2) to be used in stand-level models to
22 enable prediction of forest or stand-level response to
23 changing O₃ levels; and (4) produce an assessment of risk to
24 forest species of O₃ in the presence of multiple stresses.
25 The long-term chronic exposure research for forest tree
26 species is scheduled to be completed in 1995.

1 Additional forest tree species O₃ response data will
2 soon be available from several ongoing and future research
3 efforts, including the Southern Commercial Forest Research
4 Program begun under the National Acid Precipitation
5 Assessment Program to look at the combined effects of O₃ and
6 acid rain on forest tree species; the Southern Oxidant
7 Study, which is investigating the atmospheric chemistry
8 behind O₃ formation and the effects of regional O₃ on urban
9 ozone levels; the 1990 Clean Air Act Spatial Trends Network,
10 which will monitor a suite of atmospheric pollutant levels
11 in a nationwide network; and the Environmental Monitoring
12 and Assessment Program, which will monitor species selected
13 to serve as indicators of forest health to determine the
14 current status of forest ecosystems and determine whether or
15 not changes are taking place.

16 c. Averaging Times and Exposure Patterns of Concern

17 In terms of protecting agricultural crops and forests,
18 the most difficult issues to address are averaging times and
19 the form of the standard. Research has demonstrated that
20 there are many factors of O₃ exposure dynamics that must be
21 considered when formulating an appropriate exposure index.
22 These factors include short-term peaks, long-term chronic
23 exposures, duration between peaks, and diurnal and seasonal
24 timing of peaks. In the initial draft of the 1989 Staff
25 Paper, the EPA staff recommended that consideration be given
26 to setting both a 1-hour and a longer-term secondary

1 standard because the relationship between peak values and
2 seasonal averages is generally not predictable with a high
3 degree of confidence; therefore, a 1-hour standard may not
4 adequately reduce high chronic exposure at a particular
5 location. While the CASAC (CASAC, 1989) endorsed the
6 judgment that repeated peak exposures are critical in
7 eliciting responses in agriculture crops, the Committee's
8 views regarding the appropriateness of a separate long-term
9 standard were less clear. Instead, the CASAC challenged the
10 EPA to identify a single standard formulation that offered
11 protection from both repeated peaks of concern and long-term
12 exposures. The EPA agreed with the CASAC's recommendation
13 to identify a single standard formulation and as a first
14 step analyzed alternative monthly forms of a secondary
15 standard (U.S. EPA, 1989, Appendix A) based on air quality
16 data. The EPA found that the maximum monthly mean of the
17 daily maximum 1-hour averages related well to both repeated
18 peaks and long-term air quality indicators of concern.
19 However, there are little or no effects data for a monthly
20 exposure period.

21 Subsequently, researchers at the CERL undertook
22 additional analyses of the NCLAN data set. In an extensive
23 retrospective analysis of NCLAN data, Lee et al. (1988a) fit
24 over 600 single-index and general phenologically weighted
25 cumulative impact (GPWCI) indices to response data from
26 seven crop studies. The criterion established for

determining "best" exposure indices was that they display the smallest residual sums of square error when the yield-response data were regressed for the various O₃ exposure indices using the Box-Tidwell model.

Lee et al. (1988a) concluded that the top-performing exposure indices were those that (1) cumulate the hourly O₃ concentrations over time, (2) emphasize concentrations of 0.06 ppm and higher, and (3) place the greatest weight on exposures that occur during the plant growth stage. These findings illustrate the importance of including exposure duration, repeated peaks, and periods of increased plant sensitivity when assessing the impact of O₃ on plant growth. Although peak concentrations should be given greater weight, the authors suggested that lower concentrations are important and should also be included in the calculation of an exposure index.

In response to the CASAC recommendations (CASAC, 1987), Lee et al. (1988b) conducted additional retrospective analyses of the NCLAN data in order to evaluate selected exposure indicators. The results indicate that while the GPWCI indices best related plant response to O₃ exposure, there were other indices that were near optimal. These indices included a sigmoid-weighted integrated index (SIGMOID) centered at 0.062 ppm, which the staff concluded was too complex for use as an ambient air quality standard, and cumulative indices that sum all concentrations of 0.06

(or 0.07) ppm or higher (SUM06 and SUM07). These latter indices performed well, suggesting that ambient O₃ levels as low as 0.06 ppm are important in triggering plant response and should be included in an exposure index. These results support the conclusions reached by Lefohn et al. (1988) and Lee et al. (1987), who used the NCLAN data and cumulation indices with sigmoid and allometric weights in demonstrating the importance of peak concentrations in determining plant response.

The integrated exposure indices (SUM06 and SUM07) are functions of exposure duration and concentration that relate various yield losses calculated from experimental data to exposure "seasons." Experiments replicated in time and/or space that differ in exposure duration, but have the same SUM06 or SUM07 values, should produce identical predicted relative yield losses (Lee et al., 1988c). Because these integrated indices capture key components of exposure, they are more adequate than the 7-hour mean as descriptors of plant response; they are also attractive from a regulatory perspective because they are simple and easy to implement.

Lee et al. (1988b) also examined the relationships among the various air quality indicators, in response to the CASAC's interest in finding an indicator that correlates well with short-term peak, multiple peak, and long-term averages. Results indicate that fair to strong associations exist between the two cumulative indices (SUM06 and SUM07)

1 and the peak and mean indices: second highest daily maximum
2 (HDM2) and 7-day seasonal average (M7). The integrated
3 indices, SUM06 and SUM07, are strongly related to M7 and
4 less related to HDM2, because the relationship between SUM07
5 and HDM2 falls just below the level defined by the authors
6 (Lee et al., 1988b) as indicative of a strong association.
7 These results suggest that SUM06 and SUM07 have potential
8 for a standard that protects against adverse effects from
9 repeated peak and long-term exposures. It should be noted,
10 however, that this assessment is based solely on
11 agricultural crops because of the lack of information to
12 fully assess forest effects. Crop species are more
13 sensitive to high level, short-term peaks of O₃ than
14 perennial plants, which appear to be affected more by
15 chronic exposures to lower levels of O₃ or a combination of
16 both short-term peaks and long-term exposures. Therefore,
17 it is not clear that exposure indices based only on
18 agricultural crops will prove to be protective of perennial
19 plants.

20 2. Other Welfare Effects

21 a. Materials

22 Ozone effects on materials have been studied for the
23 last three decades. This broad data base has identified
24 several types of materials that are sensitive to O₃
25 exposure.

1 The effects of O_3 on elastomers (e.g., automobile
2 tires, protective electrical coverings, etc.) have been the
3 best documented. Ozone causes elastomers to harden, become
4 brittle or cracked, and lose physical integrity. These
5 effects increase in a dose-related fashion (i.e., the
6 product of concentration and exposure duration) and have
7 been shown to be accelerated by the presence of mechanical
8 stress, high humidity, atmospheric pressure, sunlight, and
9 other pollutants. In response, manufacturers have
10 reformulated their products to withstand greater doses of
11 O_3 , thus mitigating the effects of O_3 on elastomers.

12 The reaction of dyes to O_3 is a complex function of O_3
13 concentration, relative humidity, the presence of other
14 gaseous pollutants, the type of dye and the resistance of
15 the material in which the dye is incorporated. The
16 degradation of fibers from exposure to O_3 is poorly
17 characterized. In general, most synthetic fibers such as
18 modacrylic and polyester are relatively resistant; and
19 cotton, nylon, and acrylic fibers show variable
20 sensitivities to the gas. Anthraquinone dyes incorporated
21 into cotton and nylon fibers appear to be the most sensitive
22 to O_3 damage.

23 Paint is another material that has been investigated
24 for O_3 damage. In comparison to other materials, the effect
25 of O_3 on paints is small and has a negligible effect on the
26 useful life of the material coated.

1 Upon reviewing the available scientific technical
2 information on effects of O₃ on materials, the 1989 Staff
3 Paper concluded that "There appears to be no threshold level
4 below which materials damage will not occur; exposure of
5 sensitive materials to any non-zero concentration of O₃
6 (including natural background levels) can produce effects if
7 the exposure duration is sufficiently long. However, the
8 slight acceleration of aging processes of materials which
9 occurs at the level of the NAAQS is not judged to be
10 significant or adverse. Consequently, the staff concludes
11 that materials data should not be used as a basis for
12 defining an averaging time and concentration for the
13 secondary standard and that the secondary standard should be
14 based on protection of vegetation." (U.S. EPA, 1989, pp.
15 XI-16 to XI-17). The Administrator agrees with this staff
16 conclusion.

17 b. Personal Comfort and Well-Being

18 Effects on personal comfort and well-being, as defined
19 by human symptomatic effects, have been observed in
20 controlled human exposure studies at O₃ levels in the range
21 of 0.12-0.15 ppm for 1-3 hours of exposure at very heavy
22 exercise, and at somewhat lower levels in prolonged human
23 exposure studies (at moderate exercise), and in field
24 studies. These effects include nose and throat irritation,
25 chest discomfort, and cough. As recommended by the CASAC
26 and the EPA staff, these effects have been considered health

1 effects and have been taken into account during the review
2 of the primary standard for O₃.

3 3. Proposed Decision Not to Revise the Secondary
4 Standard

5 As previously noted, the Administrator is proposing not
6 to revise the existing O₃ secondary NAAQS at this time. In
7 reaching this proposed decision, the Administrator has
8 carefully considered the welfare effects information
9 assessed in the 1986 Criteria Document and its Supplement,
10 the 1989 Staff Paper assessment, and the advice and
11 recommendations of the CASAC (CASAC, 1989). A principal
12 reason for this proposed decision is the absence of
13 sufficient information in the 1986 Criteria Document and its
14 Supplement to specify a new form, averaging period, and
15 level of a secondary standard. Such a new standard would
16 ideally afford increased or improved protection for both
17 agro- and forest ecosystems. Research currently underway
18 will provide significant information on key aspects of O₃
19 exposure dynamics that are important for assessing the
20 effects of O₃ on forest ecosystems. When this information
21 becomes available and is incorporated into the air quality
22 criteria during the next review, a more informed judgment
23 can be made as to whether revision of the secondary standard
24 is appropriate to increase or improve the protection it
25 affords against effects of O₃ on forest ecosystems as well
26 as agricultural crops.

1 The Administrator also carefully considered the
2 available information on the effects of O₃ on agricultural
3 crops alone. Although the NCLAN studies have provided
4 extensive data on the effects of O₃ on crops, the
5 appropriateness of the seasonal mean exposure indicator used
6 in these studies has been subject to much criticism during
7 the development of revised air quality criteria. Because of
8 this and the other shortcomings of this exposure index that
9 are discussed above, the direct use of the NCLAN data for
10 standard-setting purposes would be inappropriate. The CASAC
11 recognized this and recommended that retrospective analyses
12 be undertaken in order to identify a more appropriate
13 exposure index that would offer protection from both
14 repeated O₃ peaks of concern and long-term O₃ exposures.
15 While these analyses have identified several indicators that
16 show promise, the Administrator concurs with the staff's
17 view that it would be premature to base a change in the form
18 and averaging time of the secondary standard on the
19 preliminary results presented in the Supplement to the 1986
20 Criteria Document and the Staff Paper. The CASAC also
21 recognized in its closure letter (CASAC, 1989) that further
22 work would be necessary to develop a more appropriate form
23 and averaging period for the secondary standard.

24 The Administrator also considered tightening the
25 current secondary standard as an interim measure.

26 Throughout the review of the air quality criteria and staff

1 assessment, however, no consensus was reached on an
2 appropriate range of alternative 1-hour standards. The
3 staff had great difficulty throughout the review in
4 developing and justifying alternative levels below that of
5 the current standard due to the lack of data (U.S. EPA,
6 1989, p. XI-13). In the end, the staff had to rely on the
7 preliminary results of the Lee et al. (1988b) study to
8 conclude that the upper-end of the proposed range (0.12 ppm)
9 offers little protection for vegetation (U.S. EPA, 1989, p.
10 XI-14). This is the same study that the staff concluded was
11 too preliminary to serve as a basis for recommending changes
12 in the form and averaging time of the standard. Even if the
13 results of the Lee study provided a sufficient basis for
14 revising the standard downward from 0.12 ppm to 0.10 ppm, as
15 some have suggested, it is the Administrator's judgment that
16 such a change would provide only marginal improvement
17 because a 1-hour averaging period is not the most
18 appropriate exposure indicator and most likely would be
19 superseded by adoption of a more appropriate standard form
20 during the next standard review. In the interim, it would
21 have imposed a disproportionate and largely meaningless
22 burden on States to review and make appropriate revisions in
23 applicable SIP's.

24 Given the above information, the Administrator believes
25 that the most prudent course of action is to retain the
26 current secondary standard until sufficient information can

1 be assessed and incorporated into the air quality criteria
2 to provide the basis for specifying a form, averaging time,
3 and level for a new standard that would be more protective
4 of both agro- and forest ecosystems.

5
6 III. Continuing Review of Air Quality Criteria and
7 Standards

8 As previously noted, a large number of new studies on
9 the health and welfare effects of O₃ have been published in
10 the scientific literature, since completion of the 1986 Air
11 Quality Criteria Document, its Supplement, and the 1989
12 Staff Paper that serve as the basis for today's decision.
13 Among the most pertinent of the new studies are those which
14 address: the effects of prolonged O₃ exposures in
15 controlled human experiments, the impact of O₃ on
16 susceptible subpopulations (e.g., individuals with
17 preexisting respiratory disease), chronic exposure effects
18 in animals, analysis of indicators of yield loss in
19 agricultural crops, and effects of O₃ on forest tree
20 species. Concisely summarized below are results reported in
21 a sample of potentially important new studies. These
22 studies have been selected from among numerous new
23 publications, all of which must be assessed as part of the
24 upcoming air quality criteria review.

25 Further clinical studies investigating the effects of
26 multihour exposures to O₃ have appeared in the literature

1 since 1988-1989. Extensions of earlier studies of multihour
2 exposures suggest that lung function decreased, airway
3 resistance response to methacholine was increased, and pain
4 upon deep breath was experienced by healthy, young, male
5 subjects exposed to O₃ in the range of 0.08 to 0.12 ppm for
6 6.6 hours (Horstman et al., 1990; McDonnell et al., 1991).
7 In a study of older subjects ($\geq$ 50 years old), Horvath et
8 al. (1991) reported that subjects exposed on two consecutive
9 days to 0.08 ppm O₃ for 6.6 hours showed little reduction in
10 lung function after the first day and even less on the
11 second day, thus supporting earlier studies showing that
12 responsiveness to O₃ is variable in the population and
13 decreases with advancing age of the subjects. Devlin et al.
14 (1991) have extended their work (Koren et al., 1988a,b,c) on
15 lung inflammation by evaluating additional health endpoints,
16 increasing the number of subjects, and adding a lower O₃
17 concentration. Human subjects were exposed to 0.08 and 0.10
18 ppm O₃ for 6.6 hours with moderate exercise, and their lungs
19 were lavaged 18 hours later. As reported earlier, the
20 investigators observed that 0.10 ppm O₃ increased a specific
21 class of white blood cells, protein, and some cellular
22 mediators. At 0.08 ppm O₃, a specific class of white blood
23 cells and fewer of the cellular mediators increased, but
24 protein did not. The investigators concluded that
25 inflammation occurred at both O₃ levels. In addition, they
26 examined a basic function of the primary defense cells of

1 the lungs, phagocytosis by alveolar macrophage, which
2 involves engulfing other cells or particles. They reported
3 that 6.6-hour exposure to both 0.08 and 0.10 ppm O₃
4 decreased one class of phagocytosis of microorganisms.

5 Some of the newer studies appearing in the literature
6 have addressed the effects of O₃ susceptible subpopulations.
7 For example, Molfino et al. (1991) exposed atopic asthmatic
8 subjects to 0.12 ppm O₃ for 1 hour and reported increased
9 bronchial responsiveness to allergens which was not the
10 result of changes in baseline airway function. This study
11 is preliminary and awaits confirmation, which is currently
12 underway. In an epidemiological investigation of children
13 with physician-confirmed asthma, Krzyzanowski et al. (1992)
14 reported a significant relationship between 8-hour O₃ levels
15 and lung function decrements, which increased as temperature
16 and PM-10 increased. In a retrospective epidemiology study,
17 Cody et al. (1992, in press) reported that when temperature
18 was controlled for in a multiple regression analysis, a
19 significant relationship between hospital visits for asthma
20 and O₃ was identified.

21 Generally, recent animal toxicology studies have
22 reported findings which apparently confirm and, in a few
23 cases, extend knowledge of the health effects of O₃. A few
24 key examples are described here. In a reanalysis and
25 extension of the early results of Costa et al. (1989),
26 Highfill et al. (1992) reported that significant interaction

exists between concentration (C) and exposure duration (T) in eliciting an increase in lavageable protein (an index of inflammation) in the lungs of guinea pigs and rats. This interaction was best explained using the product of CxT in an exponential relationship and provided initial evidence that cumulative dose may determine response over an extended exposure day. Numerous studies of host defenses have been conducted that report effects on the pulmonary immune system, anti-viral defenses, and clearance mechanisms. The effects cited appear to confirm and expand understanding, but their quantitative extrapolation to humans is unclear. More data from the EPA chronic rat study are being reported, beyond the subchronic phase discussed in the Supplement (Huang et al., 1988; Grose et al., 1989). Chang et al. (1992, in press) reported that a 78-week exposure to an urban pattern of O₃ (low continuous baseline, with a ramped spike; integral of 0.19 ppm under the spike) caused several structural changes in the lungs, which they interpreted as epithelial inflammation and interstitial fibrosis. Some of the changes observed persisted over a 17-week post-exposure period in the clean air. Costa et al. (1991) and Tepper et al. (1991) reported that rats from the same study had pulmonary function changes consistent with restrictive lung lesions (e.g., fibrosis). Similar types of effects were earlier observed in rats and monkeys, as are discussed in the Supplement.

Over the next 1-2 years, several ongoing studies are likely to be completed and provide data relevant to the air quality criteria for O_3 . The key classes of animal studies involve the issues of carcinogenesis and extrapolation. A cooperative effort between the NTP and the HEI is focused on potential carcinogenic, cocarcinogenic, and morphological effects of chronic exposure to O_3 . The EPA is conducting research to enable quantitative animal-to-human extrapolation, which could increase the use of animal data in the air quality criteria. Currently, research involves measuring (1) uptake of labeled O_3 in the respiratory tracts of humans and animals, and (2) dose of labeled O_3 to sensitive lung cells (i.e., alveolar macrophages) in humans and animals. These studies are correlating indicators of injury (lung function decrements, inflammation, and quantitative cell damage) with regional lung dose of labeled O_3 . Significant breakthroughs in the extrapolation of dose-effect relationships across species are expected. Human clinical studies by the EPA are expected to elucidate critical exposure factors (e.g., patterns of multihour exposures) and mechanisms of lung inflammation to improve risk interpretability of this response. Additional human clinical, field, and epidemiological studies by the EPA and academic institutions are expected to provide results useful to improve characterization of susceptible subpopulations.

1 In summary, numerous publications on the health effects
2 of O₃ have appeared since closure on the Supplement and the
3 1989 Staff Paper. Principally, they confirm what was known
4 earlier and discussed in these documents. In some
5 instances, new findings have been reported, such as effects
6 of 6.6-hour exposures to 0.08 ppm O₃ on human lung
7 inflammation and alveolar macrophage function as well as
8 increased responsiveness of asthmatics compared to healthy
9 people. This extends information evaluated in the
10 Supplement and 1989 Staff Paper, which discussed
11 observations of inflammation as low as 0.10 ppm for 6.6 hour
12 exposures and identified asthmatics as a susceptible
13 subpopulation based primarily on the concern that small
14 decrements in lung function may have exaggerated impacts in
15 people with compromised lungs, compared to normal lungs.

16 With regard to welfare effects, the most relevant of
17 the new studies published after the 1986 Criteria Document
18 or its Supplement address two key areas: 1) the
19 identification of exposure indices which characterize the
20 elements of O₃ exposure important in plant response and the
21 appropriate form and level of the index to protect
22 vegetation, and 2) the effects of O₃ on tree species.
23 Studies published on the appropriate exposure index for the
24 O₃ secondary standard have concluded that 1) O₃ impacts on
25 crop yield are the consequence of the season-long cumulative
26 exposure rather than a single peak event and thus the index

1 should reflect the cumulative nature of the plant growing
2 season exposure; 2) peak concentrations should be
3 preferentially weighted to reflect their greater biological
4 impact; 3) no single exposure index is best for all crop
5 species or all data sets; 4) a 7-hour seasonal mean would
6 not be a good exposure index to use because it implies
7 certain assumptions and is insensitive to peak
8 concentrations and exposure durations; and 5) the crop yield
9 response is not well-represented by an extreme value
10 statistic such as the current standard, 2HDM.

11 Additionally, three studies (Tingey et al., 1989; Lee et
12 al., 1991; Tingey et al., 1991) judged indices that cumulate
13 all hourly values equal to or greater than 0.06ppm (SUM06)
14 as best for relating O₃ exposures to plant response, along
15 with the indices that weight all hourly values with a
16 sigmoid function (SIGMOID or W126). The level of 24.4 ppm-
17 hr (total cumulation of hourly ppm averages over the maximum
18 3-month exposure period) is given as the SUM06 necessary to
19 limit crop loss to 10 percent in half of the cases of crop
20 O₃ exposure in the U.S. (Tingey et al., 1991). This level
21 is based on 49 NCLAN studies involving 12 crops. Research
22 on the appropriate form and level of an index for the
23 protection of trees is now also beginning to be addressed.
24 (Hogsett and Tingey, 1990; Lefohn et al., 1992).

25 The second area of relevant new research is that of the
26 effects of O₃ on forest tree species. Hogsett et al. (1989)

1 observed reduced shoot and root growth in the season
2 following O₃ exposure, indicating that effects are
3 cumulative and are effecting the carbon reserves that a tree
4 utilizes in bud/shoot elongation and spring root growth. In
5 the first year results of a multiyear study, Andersen et al.
6 (1991) similarly reports that "ponderosa pine seedlings
7 exposed to 122 and 169 ppm-hr O₃ for one season have
8 significantly less root starch reserves available just prior
9 to and during bud break the following year and spring root
10 growth is decreased following O₃ exposure." These are
11 exposure regimes typical of rural monitoring sites in the
12 eastern U.S. It is not yet known how these effects will
13 translate over time in multiyear exposures. Hogsett and
14 Tingey (1990) report that growth effects in trees exposed to
15 episodic regimes are greater than in trees experiencing a
16 high-elevation exposure regime, thus indicating the
17 importance of peak events and their episodic occurrence in
18 the response of trees as well as crops.

19 Because of the potential significance of these studies,
20 as well as other ongoing research efforts, the EPA is
21 planning to proceed as rapidly as possible with the next
22 periodic review of the air quality criteria and standards
23 for O₃. Under the process established in sections 108 and
24 109 of the Act and refined by the EPA and the CASAC, the EPA
25 will begin by announcing the commencement of the review in
26 the Federal Register. After carefully assessing and

1 evaluating the pertinent new studies, the EPA will then
2 prepare a preliminary draft of a revised criteria document
3 and subject it successively to review at expert peer-review
4 workshops; by the public; and by the CASAC. Once the CASAC
5 has reviewed the first external review draft of the revised
6 criteria document, thus providing a preliminary basis for
7 review of the existing standards, the EPA staff will prepare
8 a draft staff paper assessing the most significant
9 information contained in the draft criteria document and
10 develop recommendations for revisions, if appropriate, to
11 the standards. The first draft of the staff paper and the
12 second external review draft of the criteria document will
13 then be made available for public and CASAC review.
14 Typically at this point, the criteria document is of
15 sufficient quality for the CASAC to reach "closure" and will
16 provide the basis for completing the staff paper that in
17 turn will be reviewed by the CASAC. The CASAC will then
18 submit its advice and recommendations to the Administrator.
19 The overall process will take an estimated 2-3 years.
20 Although the process is lengthy and rigorous, the EPA
21 believes it is both necessary and appropriate given
22 applicable statutory requirements, volume of material
23 requiring careful evaluation, and extraordinary
24 environmental, economic, and social importance of O₃ NAAQS.
25
26

1 IV. Federal Reference Method

2 The EPA is not proposing any revisions to the Federal
3 reference measurement method for O₃ described in Appendix D
4 to 40 CFR Part 50, as amended on February 18, 1975 (40 FR
5 7042) and further amended on February 8, 1979 (44 FR 8221).
6

7 V. Regulatory and Environmental Impact Analysis

8 Under Executive Order 12291, the EPA must judge whether
9 an action is a "major" regulation for which a Regulatory
10 Impact Analysis (RIA) is required. The EPA has judged the
11 proposed O₃ NAAQS decision is not a major action because
12 there are no additional costs or environmental impacts as a
13 result of not revising the standards. The EPA, therefore,
14 has deemed unnecessary the preparation of either a RIA or an
15 Environmental Impact Statement.
16

17 VI. Impact on Small Entities

18 Under the Regulatory Flexibility Act (RFA), 5 U.S.C.
19 601 et seq., the EPA must prepare initial and final
20 regulatory flexibility analyses assessing the impact of
21 certain rules on small entities. These requirements are
22 inapplicable to rules or other actions for which the EPA is
23 not required by the Administrative Procedure Act (APA), 5
24 U.S.C. 551 et seq., or other law to publish a notice of
25 proposed rulemaking. 5 U.S.C. 603(a), 604(a). The EPA is
26 following rulemaking procedures in deciding whether to

1 revise the O₃ standards in light of the court order in the
2 American Lung Association case and the importance of the
3 issue. Under section 307(d) of the Act, as the EPA
4 interprets it, neither the APA nor the Act requires
5 rulemaking procedures where the Agency decides to retain
6 existing NAAQS without change. Accordingly, the EPA has
7 determined that the impact assessment requirements of the
8 RFA are inapplicable to this proposed decision.

9
10 VII. Other Reviews

11 This proposed decision was submitted to the Office of
12 Management and Budget (OMB) for review. Any written
13 comments from OMB and the EPA written responses to these
14 comments are available for public inspection at the EPA's
15 Central Docket Section (Docket No. A-92-17), South
16 Conference Center, Room 4, Waterside Mall, 401 M Street, SW,
17 Washington, DC.

18
19 List of Subjects in 40 CFR Parts 50

20 Air pollution control, Carbon monoxide, Ozone, Sulfur
21 oxides, Particulate matter, Nitrogen dioxide, Lead.

22
23
24
25 _____
Date

Administrator

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Appendix I

May 1, 1989

The Honorable William K. Reilly
Administrator
U. S. Environmental Protection Agency
401 M Street, SW
Washington, DC 20460

Dear Mr. Reilly:

I am pleased to transmit via this letter the advice of the Clean Air Scientific Advisory Committee (CASAC) concerning the National Ambient Air Quality Standards for Ozone. CASAC has reviewed and offered comments directly to EPA staff on the EPA criteria document "Air Quality Criteria for Ozone and Other Photochemical Oxidants (1986)," the draft "Criteria Document Supplement (1988)," and the Office of Air Quality Planning and Standards staff position paper "Review of the National Ambient Air Quality Standards for Ozone Assessment of Scientific and Technical Information (1988)" and related support documents.

CASAC previously reached closure on the 1986 Criteria Document. At a meeting held on December 14-15, 1988, CASAC came to closure on the "Criteria Document Supplement (1988)" and the 1988 Staff Position Paper and concluded that they provide an adequate scientific basis for EPA to retain or revise primary and secondary standards for ozone. While reaching closure at this time, the Committee did note an emerging data base on the acute health effects resulting from 6-plus hours of ozone exposure, providing evidence of the possible need for a standard with a 6-8 hour averaging time. However, it was the Committee's view that it would be some time before enough of this developing information would be published in scientific journals to receive full peer review and, thus, be suitable for inclusion in a criteria document. CASAC concluded such information can better be considered in the next review of the ozone standards.

CASAC did not reach a consensus opinion on endorsement of the staff position paper recommendation that "the range of 1-hour average ozone levels of concern for standard setting purposes is 0.08-0.12 ppm for a primary standard."

1 The opinion of the CASAC Ozone Review Committee was divided
2 with regard to the upper range of the standard with eight
3 individuals favoring a range with an upper value of 0.12
4 ppm, three individuals favored an upper bound in the range
5 of 0.10-0.12 ppm, four individuals favored an upper bound
6 value no higher than 0.10 ppm, and one individual abstained
7 from offering an opinion. Several individuals who supported
8 an upper value of 0.12 ppm as well as all of the other
9 individuals who favored a lower value for the upper end of
10 the range expressed the view that at 0.12 ppm there was
11 little or no margin of safety. As you are aware, the margin
12 of safety is intended to provide protection against adverse
13 effects which have not yet been uncovered by research and
14 effects whose medical significance is a matter of
15 disagreement. Finally, several members of the subcommittee
16 favored development of a standard with a more statistically
17 robust upper bound on the annual distribution of ozone
18 concentrations rather than reliance on the current expected
19 exceedance form of the standard. While the Committee offers
20 no further advice on what form the Agency should consider,
21 we would caution you against any form which alters the
22 degree of health protection afforded by the current
23 standard.
24

25 CASAC had substantial discussion of the issue of what
26 are or are not adverse health effects. This discussion was
27 aided by the presentation of this issue in the staff
28 position paper. Within CASAC there was diversity of
29 opinion; some members felt that healthy individuals
30 experience adverse effects when ozone exposure induced any
31 of the responses categorized as moderate (i.e., >10%
32 decrement in FEV₁ or mild to moderate respiratory symptoms)
33 in the staff position paper, while a few members believed
34 that adverse effects would not be experienced until ozone
35 induced more severe effects (i.e., >20% decrement in FEV₁
36 and moderate to severe respiratory symptoms). The view of
37 some individuals on this matter was influenced by
38 recognition that resolution of the adverse health effect
39 issue represents a blending of scientific and policy
40 judgments and, thus, we feel it appropriate to inform you of
41 the range of our views on this matter.
42

43 Of particular concern to CASAC is the potential for
44 effects arising from exposures to ozone with daily peak
45 concentrations at or near 0.12 ppm for periods of 6-8 hours
46 and with co-exposure to other pollutants. This concern is
47 due to air quality analyses which have shown that even in
48 areas which do not repeatedly exceed the ozone standard,
49 ozone concentrations can remain close to 0.12 ppm for
50 several hours per day for extended periods of time in
51 summer. There was concern based on recent controlled human
52 exposure, epidemiology and toxicology studies, that such

1 prolonged exposures could result in increased respiratory
2 impairment. Further, for people exposed to these ozone
3 concentrations over a lifetime, the possibility that chronic
4 irreversible effects may result is of concern, although such
5 changes have not been demonstrated.
6

7 The Committee noted that the Criteria Document
8 Supplement failed to cite and discuss a group of
9 "ecological" epidemiological studies of the effects of ozone
10 on various measures of human health such as hospitalizations
11 for respiratory illnesses or exacerbation of chronic
12 respiratory problems. Although these studies have obvious
13 limitations in establishing cause and effect relationships,
14 they have certain strengths which can aid in regulatory
15 decision-making. Studies of this type should be discussed
16 and evaluated in future criteria documents as a
17 complementary source of information.
18

19 While reaching closure on the staff position paper
20 recommending a 1-hour standard, CASAC urged that the Agency
21 provide increased support for research that will prove an
22 improved scientific basis for evaluating the need for
23 standards with multi-hour or seasonal averaging times.
24 Clearly, the obvious, research on this critical
25 environmental health issue must be supported now in order
26 for results to be available for consideration in the next 5-
27 year review cycle. CASAC has enumerated these research
28 needs in some detail in a September 1987 submission to the
29 Agency. The Committee feels these research recommendations
30 are still valid and should be incorporated as expeditiously
31 as possible into the Agency research program.
32

33 CASAC did not reach a consensus opinion on endorsement
34 of the staff position paper recommendation of "a 1-hour
35 averaging time standard in the range of 0.06-0.12 ppm" for a
36 secondary standard. The CASAC Ozone Welfare Effects
37 Subcommittee that considered this matter reached a divided
38 opinion; two favored a range with an upper value of 0.12
39 ppm, three favored an upper value of less than 0.12 ppm, and
40 five favored an upper value of 0.10 ppm. The Committee
41 noted that the form of the standard was of critical
42 importance in protecting against ozone effects on
43 vegetation. The Committee was of the opinion that a
44 cumulative seasonal standard would be more appropriate than
45 a 1-hour standard and felt that such a standard could be-
46 developed. CASAC favored issuance of a cumulative seasonal
47 standard form assuming its development would not further
48 delay the standard setting process. If this form of
49 standard cannot be developed in time for the current review,
50 the Committee is of the opinion that you should give serious
51 consideration to setting a 1-hour secondary standard with a
52 maximum of 0.10 ppm. The Committee took note of the lack of

1 information on the effects of ozone on forest ecosystems and
2 urged support for research to remedy this deficiency.
3

4 In closing, I would like to briefly comment on CASAC's
5 failure to reach a consensus as to the appropriate range for
6 setting the ozone standards. This lack of consensus is
7 reflective of major deficiencies in our knowledge regarding
8 health and welfare effects of long-term exposure (beyond a
9 few hours) to ozone. The data base is very large and
10 adequate for knowledgeable individuals to reach agreement on
11 the effects of acute exposure to ozone in the range
12 appropriate for setting a 1-hour standard. However, there
13 is not an adequate data base on the effects of multiple hour
14 or seasonal exposures to ozone, especially as regards
15 whether such exposures may produce chronic health effects.
16 This is especially troubling since such long-term exposures
17 to ozone occur in many parts of the United States and
18 involve many millions of people and thousands of acres of
19 crop and forest lands. As a result, there continues to be
20 concern for the public health and welfare threat which may
21 be posed by chronic exposure to ozone. It is critical that
22 the data base on health and welfare effects related to
23 multiple hour, seasonal and lifetime exposures of ozone be
24 increased through an accelerated and expanded research
25 effort. This must be done so that future considerations of
26 ozone standards will derive from a stronger scientific base.
27

28 CASAC recognizes that your statutory responsibility to
29 set standards requires public health policy judgments in
30 addition to determinations of a strictly scientific nature.
31 While the Committee is willing to further advise you on the
32 ozone standards, we see no need, in view of the already
33 extensive comments provided, to review the proposed ozone
34 standards prior to their publication in the Federal
35 Register. In this instance, the public comment period will
36 provide sufficient opportunity for the Committee to provide
37 any additional comments or review that may be necessary.
38

39 CASAC would appreciate being kept informed of progress
40 on establishing revised or new ozone standards and plans for
41 research on ozone effects. Please do not hesitate to
42 contact me if CASAC can be of further assistance on this
43 matter.
44

45 Sincerely,
46
47

48
49 Roger O. McClellan, D.V.M.
50 Chairman, Clean Air Scientific
51 Advisory Committee
52