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Review Of The National Ambient Air Quality Standards For Ozone Assessment Of Scientific and Technical Information OAQPS Staff Report

Preface

This document was finalized in June 1989 and reviews information from relevant studies of O_3 health and welfare effects and of exposure and risk analysis through early 1989. The assessment contained in this staff paper reflects information in the documents "Air Quality Criteria for Ozone and Other Photochemical Oxidants" (EPA-600/8-84-020F) and "Summary of Selected New Information on Effects of Ozone on Health and Vegetation: Supplement to Air Quality Criteria for Ozone and Other Photochemical Oxidants" (EPA-600/8-88/1-5a).

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This staff paper is the product of the Office of Air Quality Planning and Standards (OAQPS). Tables and Figures not otherwise cited are original to this report. The principal authors include Dr. David J. McKee, Ms. Pamela M. Johnson, Mr. Thomas R. McCurdy, and Mr. Harvey M. Richmond. This report has been improved by comments from other staff within OAQPS, the Office of Research and Development, the Office of Policy and Program Evaluation, and the Office of General Counsel within EPA. Three drafts were formally reviewed by the Clean Air Scientific Advisory Committee and comments incorporated. Particularly important in the final review of this staff paper was the technical and editorial support provided by Ms. Victoria Atwell and the clerical and editorial support of Mrs. Patricia R. Crabtree and Mrs. Barbara Miles.

Helpful comments and suggestions were also submitted by a number of independent scientists, by officials from the State environmental agencies of Illinois, Minnesota, California and Texas, by the Department of the Navy, and the Department of Energy, and by environmental and industrial groups including the Natural Resources Defense Council, the American Lung Association, the Chemical Manufacturers Association, the American Petroleum Institute, and the Motor Vehicle Manufacturers Association.

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Executive Summary

This revised staff paper evaluates and interprets the available scientific and technical information that the EPA staff believe is most relevant to the review of primary (health) and secondary (welfare) national ambient air quality standards (NAAQS) for ozone (O_3) and presents staff recommendations on alternative approaches to revising the standards. Periodic review of the NAAQS is a process instituted to ensure the scientific adequacy of air quality standards and is required by section 109 of the 1977 Clean Air Act Amendments. The assessment in this staff paper is intended to help build a bridge between the scientific review contained in the EPA O_3 criteria document (hereafter referred to as CD) (U.S. EPA, 1986), and the CD Supplement (hereafter referred to as CDS) (U.S. EPA, 1988) prepared by the Environmental Criteria and Assessment Office (ECAO) and the judgments required of the Administrator in setting ambient standards for O_3. Therefore, the staff paper is an important element in the standards review process and provides an opportunity for review by the Clean Air Scientific Advisory Committee (CASAC) and the general public on proposed staff recommendations before they are presented to the Administrator. This staff paper has been revised based upon comments received from CASAC and the public and upon staff analyses which are available for public review.

Ozone is a trace constituent formed in the atmosphere as a result of a series of complex chemical reactions involving both anthropogenic and natural hydrocarbons and nitrogen oxides, oxygen and sunlight. At ambient concentrations often measured during warmer months, O_3 can adversely affect human health, agricultural crops, forests, ecosystems, and materials. Interactions of O_3 with nitrogen oxides and sulfur oxides may also contribute to the formation of acidic vapors and aerosols which might have direct effects on human health and welfare, as well as indirect effects following their deposition on surfaces. It should be noted that new evidence indicates that co-exposure to acidic aerosols can potentiate response to O_3.

Annual average background surface O_3 concentrations in the northern hemisphere generally range between 0.03 and 0.05 ppm but are as low as 0.015 to 0.020 ppm in the tropics (U.S. EPA, 1986, p. 3-80). Stratospheric intrusion is recognized as causing locally high O_3 levels for periods lasting from minutes to hours, but these intrusions are usually worse in spring, fall, and winter. In contrast, during the photochemically active summer months intrusion is less common and less severe. Summertime hourly O_3 levels have recently been reported to be as high as 0.35 ppm in one of the nation's most heavily populated metropolitan areas. Daily daylight seasonal averages of O_3 in some rural areas have been reported to be 0.06 ppm and higher.

Primary Standard

The staff reviewed scientific and technical information on the known and potential health effects of O_3 cited in the CD and the CDS. The information includes studies of respiratory tract absorption and deposition of O_3, studies of mechanisms of O_3 toxicity, and controlled human exposure, field, epidemiological and animal toxicology studies of effects of exposure to O_3 as well as air quality information. On the basis of this review, the staff derives the following conclusions.

1) Inhaled O_3 may pose health risks as a result of (a) penetration of O_3 into various regions of the respiratory tract and absorption of O_3 in this tract (b) provocation of pulmonary response resulting from chemical interactions of O_3 along the respiratory tract, and (c) extrapulmonary effects caused indirectly by reaction of O_3 in the lungs.

2) The risks of adverse effects associated with absorption of O_3 in the tracheobronchial and alveolar regions of the respiratory tract are much greater than for absorption in the extrathoracic region (head). Increased exercise levels are generally associated with higher ventilation rates and increased oronasal or oral (mouth) breathing. Greater O_3 penetration and exposure of sensitive lung tissue occurs when individuals are heavily exercising.

3) Factors which have been demonstrated to affect susceptibility to O_3 exposure are activity level and environmental stress (e.g., humidity, high temperature). Those factors which either have not been adequately tested or remain uncertain include age, sex, preexisting disease, nutrition, and smoking status.

4) Major subgroups of the population that may be at greater risk to the effects of O_3 include: (a) any individual exercising heavily during exposure to O_3, particularly those who are otherwise healthy individuals who may experience significantly greater than group mean lung function response to O_3 exposure, and (b) individuals with preexisting respiratory disease (e.g., asthmatics and persons with allergies). The data base identifying exercising individuals as being at greater risk to O_3 exposure is much stronger and more quantitative than that for individuals with preexisting respiratory disease. This is due to the large number of clinical studies investigating effects of O_3 on exercising persons.

5) The major effects categories of concern associated with exposures to O_3 include:

   (a) alterations in pulmonary function

   (b) symptomatic effects (e.g., cough, throat irritation)

   (c) effects on work or athletic performance

(d) aggravation of preexisting respiratory disease

(e) morphological effects (lung structure damage)

(f) altered host defense systems (e.g., increased susceptibility to respiratory infection)

(g) extrapulmonary effects (e.g., effects on blood enzymes, central nervous system, liver, endocrine system).

6) An important source of applicable exposure-response information for a short-term standard is controlled human exposure and field studies, which provide concentration-response relationships between alterations in pulmonary function and O_3 exposure concentrations. Other important sources of information for standard setting are epidemiological and toxicological studies. Epidemiology has provided associations between ambient O_3 exposures and lung function decrements and aggravation of existing respiratory disease, but with greater uncertainties about the exposures involved than with controlled human exposure and field studies. Animal toxicology data provide acute and chronic exposure effects information on increased susceptibility to respiratory infection, lung structure damage, and extrapulmonary effects. Although human exposure, epidemiology, and animal toxicology studies all have limitations in assessing adverse effects and risk, it is the weight of evidence

and integration of findings from all three disciplines which should be used in assessing health effects associated with exposure to O_3.

Based on scientific and technical reviews, CASAC comments, and policy considerations, the staff makes the following recommendations with respect to primary O_3 standards:

1) Ozone should remain as the surrogate for controlling ambient concentrations of photochemical oxidants.
2) The existing form of the standard should be retained (i.e., that the NAAQS is attained when the expected number of days per calendar year with maximum 1-hour average concentrations above the level of the standard is equal to or less than one).
3) The 1-hr averaging time of the standard should be retained.
4) The range of 1-hour average O_3 levels of concern for standard-setting purposes is 0.08 to 0.12 ppm in concordance with CASAC comments (CASAC, 1986, 1987, 1988) comments. This range is based solely on 1-2 hour exposure data.
5) Because there is a good health effects data base available on 1-2 hour exposures, the staff concurs with the CASAC conclusion (McClellan, 1989) that review of the scientific basis for the 1-hr O_3 primary standard be closed out. With this portion of the review complete, and after considering CASAC's views on all

issues, the Administrator will be in a position to make a regulatory decision on how and when to best act on the 1-hour standard.

6) In response to suggestions made by CASAC (1986, 1987, 1988), staff investigated the potential need and basis for a longer-term (6-8 hour) primary standard. Although an emerging data base reporting significant lung function decrements and symptoms in subjects exposed to O_3 for 6 to 8 hours has provided some evidence of effects below 0.12 ppm O_3, staff concurs with CASAC's conclusion that ". . . such information can better be considered in the next review of the ozone standards." (McClellan, 1989). It is recommended that EPA continue review of scientific information on health effects of prolonged exposure to O_3. Once these studies have been more completely evaluated during the next CD review, the Administrator will be able to assess the need for development of a longer-term O_3 primary standard.

7) Further review and analysis also will be necessary before fully assessing the need for a separate standard to protect against chronic effects of O_3. Data on nasopharyngeal removal, dosimetry modeling and health effects based on and chronic exposure of animals will be used for future animal extrapolation and risk assessment of chronic O_3 exposures.

Secondary Standard

The staff has reviewed the scientific and technical information on the known and potential welfare effects of O_3 cited in the CD and the CDS. This information includes impacts on vegetation, natural ecosystems, materials, and symptomatic effects on humans. Based on this review, the staff derives the following conclusions:

1) The mechanisms by which O_3 may injure plants and plant communities include (a) absorption of O_3 into leaf through stomata, followed by diffusion through the cell wall and membrane, (b) alteration of cell structure and function as well as critical plant processes, resulting from the chemical interaction of O_3 with cellular components, and (c) occurrence of secondary effects including reduced photosynthesis and growth and yield and altered carbon allocation.

2) The magnitude of the O_3-induced effects depends upon the physical and chemical environment of the plant, as well as on various biological factors (including genetic potential, developmental age of plant, and interaction with plant pests).

3) The weight of the recent evidence seems to suggest that long-term averages, such as the 7-hour seasonal mean, may not be adequate indicators for relating O_3 exposure and plant response.

4) Repeated peak concentrations are the most critical element in determining plant response. Exposure indicators which emphasize peak concentrations and accumulate concentrations over time probably provide the best biological basis for standard setting (See staff paper, p. X-50).

5) There is currently a lack of exposure-response information on forest tree effects. In addition, there is a broad range of uncertainty among scientists regarding O_3 effects on forest trees. Consequently there is no consensus on the most important averaging time for perennials or on the precise role of O_3 vs. other pollutants in causing forest decline. Therefore, the staff concludes that a separate secondary standard based on protection of forest trees is not warranted at this time.

6) There appears to be no threshold level below which materials damage will not occur; exposure of sensitive materials to any non-zero concentration of O_3 (including natural background levels) can produce effects if the exposure duration is sufficiently long. However, the slight acceleration of aging processes of

materials which occurs at the level of the NAAQS is not judged to be significant or adverse. Consequently, the staff concludes that materials data should not be used as a basis for adequately defining an averaging time or concentration level for the secondary standard and that the secondary standard should be based on protection of vegetation.

7) Effects on personal comfort and well-being, as defined by human symptomatic effects, have been observed in clinical studies at O_3 levels in the range of 0.12-0.16 for 1-2 hour exposures and at somewhat lower levels in extended exposure clinical and epidemiological studies. CASAC recommended that these effects be considered health effects in developing a basis for the primary standard for O_3.

Based on scientific and technical reviews, CASAC comments, and policy considerations, the staff makes the following recommendations with respect to secondary standards:

1) In consideration of the large base of welfare information attributing effects to O_3 exposure and the limited evidence which demonstrates welfare effects from exposure to ambient levels of non-O_3 photochemical oxidants, there appears to be little evidence to suggest a change in chemical designation from O_3 to photochemical oxidants.

2) Given the lack of effects data on forests and the preliminary nature of the Lee et al. (1988c) results regarding selection of the appropriate exposure statistic for crops, the EPA staff concludes that it may be premature at this point in time to change the form of the standard and the averaging time. It is our judgment that a 1-hr averaging time standard in the range of 0.06-0.12 ppm represents the best staff recommendation that could be made to the Administrator at this time to close out the review of the scientific data. This is consistent with CASAC comments (CASAC, 1987, 1988) urging EPA to consider a 1-hr averaging time and to act on the existing state of science rather than extend the review until a more exhaustive assessment is made of alternative averaging times. With this portion of the review complete, and after considering CASAC's views on all issues, the Administrator will be in a position to make a regulatory decision on how and when to best act on the 1-hr standard.

Alternatively, EPA could continue the standard review until the information on alternative exposure indicators has matured. Additional time for review and revision of Lee et al. (1988c) would allow the scientific community the opportunity to review the alternative indicators and move toward a consensus regarding selection of the most appropriate exposure indicator. The

liability of this alternative is that it postpones action on the secondary standard and thus fails to utilize new and existing information to assess the most appropriate exposure statistic or the protection afforded by the current 1-hr standard.

Review of the National Ambient Air Quality Standards for Ozone
Assessment of Scientific and Technical Information
Staff Paper

I. Purpose

The purpose of this staff paper is to evaluate and interpret scientific information contained in the CD and the CDS and to identify critical elements to be considered in selecting primary (health based) and secondary (welfare based) national ambient air quality standards (NAAQS) for ozone (O_3). Staff conclusions and recommendations will integrate critical elements of the review of standards with other factors such as averaging times, form of standards, and margin of safety considerations.

II. Background

Since 1970 the Clean Air Act as amended has provided authority and guidance for the listing of certain ambient air pollutants which may endanger public health or welfare and the setting and revising of NAAQS for those pollutants. Primary standards must be based on health effects criteria and provide an adequate margin of safety to ensure protection of public health. As several judicial decisions have made clear, the economic and technological feasibility of attaining primary standards are not to be considered in setting them, although such factors may be considered in the development of state plans to implement the standards (Lead Industries Association v. EPA, 1980; American Petroleum Institute v. EPA, 1981). Further guidance provided in the legislative history of the Act indicates that the standards should be set at "the maximum permissible ambient air level . . . which will protect the health of any (sensitive) group of the population" (U.S. Senate, 1974). Also, margins of safety are to be provided such that the standards will afford "a reasonable degree of protection . . . against hazards which research has not yet identified" (U.S. Senate, 1974). In the final analysis, the EPA Administrator must make a policy decision in setting the primary standard based on a judgment regarding the implications of all the health effects evidence and the requirement that the standards provide an adequate margin of safety.

Secondary ambient air quality standards must be based on welfare effects and must be adequate to protect the public welfare from any known or anticipated adverse effects associated with the presence of a listed ambient air pollutant. Welfare effects, which are defined in section 302(h) of the Act, include effects on vegetation, visibility, water, crops, man-made materials, animals, economic values and personal comfort and well-being.

On April 30, 1971, the Environmental Protection Agency (EPA) published in the Federal Register (36 FR 8186) primary and

secondary national ambient air quality standards (NAAQS) for photochemical oxidants. Both standards were set at an hourly average level of 0.08 ppm not to be exceeded more than once per year.

In accordance with the provisions of sections 108 and 109 of the Clean Air Act as amended, EPA reviewed and revised the criteria upon which the original photochemical oxidants NAAQS were based. On February 8, 1979, revised standards were published (44 FR 8202) with the following changes: (1) changing the primary and secondary standards to 0.12 ppm, (2) changing the chemical designation of the standards from photochemical oxidants to ozone (O_3), (3) changing to a daily maximum standard rather than an "all-hours" standard, and (4) changing the definition of the point at which the primary and secondary standards are attained to "when the expected number of days per calendar year with maximum hourly average concentrations above 0.12 ppm is equal to or less than one."

Several factors were cited as a basis for revising the primary standard for O_3 in 1979. Among these were: (1) an adverse health effect threshold for oxidants could not be identified with certainty, (2) O_3 is a pulmonary irritant that affects respiratory mucous membranes as well as other lung tissue and impairs respiratory function at levels as low as 0.15 ppm for exercising persons (Delucia and Adams, 1977), (3) evidence suggests an elevated number of asthma attacks when peak hourly oxidant concentrations reach about 0.25 ppm (Schoettlin and Landau, 1961), (4) several studies show increased susceptibility to bacterial infection in laboratory animals exposed to O_3 plus a bacterial challenge, (5) premature aging symptoms reported in animals and (6) apparent synergistic effects on pulmonary function from exposure to 0.37 ppm O_3 and 0.37 ppm sulfur dioxide. EPA concluded that a primary standard of 0.12 ppm (one-hour average) not to be exceeded more than one day per year on

average would protect public health with an adequate margin of safety.

EPA based its decision on the secondary standard on the limited information that was available on growth and yield reduction in commercially important crops and indigenous vegetation exposed to O_3 under field conditions. These studies indicated that growth and yield responses were related to long-term (growing season) exposure of plants. On the basis of this information and available air quality data, EPA concluded that there was no evidence indicating that a significant decrease in growth and yield would result from long-term average concentrations expected to occur when the primary standard was attained. Consequently, a secondary standard more stringent than the primary standard was deemed unnecessary on the basis of O_3-related yield reduction effects on vegetation.

III. Approach

This is the final staff paper provided during current review of the NAAQS for O_3; judgments contained herein are based on scientific evidence reviewed in the CD and in the CDS prepared by the ECAO. This staff paper incorporates the results of a health risk assessment and includes the results of an exposure analysis for alternative NAAQS.

"Critical elements" have been identified which the staff believe should be considered in a review of the primary and secondary standards. Particular attention is drawn to those judgments that must be based on careful interpretation of incomplete or uncertain evidence. In such instances, the staff paper states the staff's evaluation of evidence as it relates to a specific judgment, sets forth alternatives that the staff believe should be considered, and recommends a course of action. To facilitate review, this paper is organized into sections as outlined below.

Section IV provides an overview of ambient levels of O_3 currently being experienced in various portions of the United States. This section is intended to set the stage for the remaining discussion by identifying the present air quality situation so the reader can relate available health and welfare information to O_3 levels occurring in the real world.

Section V summarizes results of the O_3 exposure analysis. The NAAQS Exposure Model (NEM) was used to estimate nationwide human exposure to O_3 given attainment of alternative standards.

Section VI deals with elements related to the health effects evidence examined in reaching conclusions regarding the primary standards; these include the following:

- most probable mechanisms of toxicity by which health effects occur,

- discussion of factors potentially affecting susceptibility to O_3 exposure,
- description of the most sensitive population groups and estimates of the size of those groups.

Section VII is a preliminary assessment of health effects and related health issues. The section:

- identifies health effects which have been attributed to O_3 and other photochemical oxidant exposures,
- discusses health effects evidence used to develop staff judgments concerning which effects are most important for the Administrator to consider in reviewing and setting primary standard(s),
- describes the health risk assessment for acute O_3 exposures,
- discusses issues related to health effects attributed to O_3 and other photochemical oxidants.

Drawing on discussions in Sections IV through VII, Section VIII identifies and assesses factors that the staff believes should be considered in selecting a pollutant indicator, averaging time, form, and level of primary standards, including margin of safety considerations. Staff recommendations also are presented in this section.

In Section IX, the effects of O_3 and other photochemical oxidants on vegetation, personal comfort, natural ecosystems, and man-made materials are identified. The section:

- describes physiological and biochemical alterations associated with welfare effects which result from exposure to O_3 and other photochemical oxidants,
- identifies welfare effects of O_3 and other photochemical oxidants,
- discusses factors affecting plant response.

Section X is a discussion of welfare effects to be considered in selecting secondary standard(s). The section:

- describes the existing scientific evidence on welfare effects attributed to O_3 and other photochemical oxidant exposures,
- describes new studies and analyses related to the issue of averaging times, and
- identifies and evaluates scientific uncertainties related to welfare effects evidence in addition to staff judgments concerning which welfare effects are important for the Administrator to consider in reviewing and setting secondary standard(s).

Drawing on discussions in Sections IX and X, Section XI identifies and assesses the factors that the staff believes should be considered in selecting averaging time(s), level(s) and form of secondary standards. Staff recommendations also are presented in this section.

IV. Ambient Ozone Concentrations in Urban and Rural Areas

This Section provides a summary of ambient O_3 air quality in urban and non-urban areas. More information on the topic appears in Appendix A. The data base used generally is 1985-1987 Storage and Retrieval of Aerometric Data (SAROAD) air quality data,[1] but data are presented for earlier time periods if 1985-1987 data are not available. Urban areas are interpreted to be Metropolitan Statistical Areas (MSAs) as defined by the U.S. Bureau of Census.

This section also briefly discusses the concept of "natural O_3 background," and provides a general estimate of its ambient concentrations for different averaging times at ground-level.

A. Urban Areas

There are 331 MSAs in the 50 states. EPA staff in the Ambient Standards Branch has identified 224 MSAs (68%) as having enough O_3 air quality data to ascertain whether or not they exceed the NAAQS. Of these, 101 areas (45%) have more than one expected exceedance per year of the current O_3 standard of 0.12 ppm. Thus, slightly less than then one-half of MSAs with sufficient data exceed the standard. Approximately 100 million people, less than one-half of the total U.S. population, live in these 101 metropolitan areas. (However, this does not mean that everyone in these areas is exposed to O_3 concentrations at or above the standard. See Section V.)

About 10% of the MSAs with sufficient data have a characteristic highest concentration (CHC)[2] of 0.16 ppm O_3 or

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[1]As has been widely reported, 1988 O_3 levels are much higher than 1985 to 1987 levels in many parts of the country. Thus, using more recent data--for the 1986-1988 time period--would result in more exceedances and higher design value than presented here (and in Appendix A). Subsequent O_3 staff papers will incorporate these new data as they become available.

[2]The CHC is a general term that encompasses more specific labels placed on peak, short-term O_3 concentrations that occur infrequently. One such CHC is the "design value," often

higher; over 5% (11) of MSAs have a CHC above 0.18 ppm O_3. There is no clear temporal trend in O_3 CHCs in most MSAs around the country, although the 1984-1985 time period generally had lower levels than previous years and 1987-1988 had higher levels. The trend in expected exceedances in MSAs likewise is indefinite. Over the 1980s, expected exceedances were relatively high in 1980, dropped in 1981-1982, and jumped radically in 1983. Expected exceedances for 1984 and 1985 were like those in 1981-1982, but increased again in 1987 and 1988.

Generally, maximum monthly mean concentrations for 1- and 8-hour daily maximum averages are in the range of 0.050-0.085 ppm. The maximum 3-month mean of 8-hour daily maximum averages is in the range of 0.045-0.065 in most urban areas of the country. There are statistically significant relationships among peak and longer-term mean indices of O_3 air quality in urban areas. These relationships can be used to estimate the impact that alternative short- term peak O_3 standards will have on long-term average levels of O_3.

B. Non-MSA Areas

Air quality data indicate that non-MSAs have a lower CHC than do MSA areas and that the CHC often is associated with O_3 transported into non-MSA areas after 4 pm. On average, a daily

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represented by the second-highest 1-hour daily maximum O_3 concentration monitored during an area's O_3 season (generally April through October). Another CHC is the characteristic largest daily maximum (CLDM), usually represented by a concentration from a mathematical distribution fitted to actual air quality data. For O_3 air quality data, a distribution often used is the two-parameter Weibull. Generally, the CLDM is the 1/n value from the Weibull, where n = O_3 season length (in days).

All three labels (CHC, design value, and CLDM) are used in this report, especially in Appendix A. The CHC mentioned above in the text is the "design value" version. See also the footnote on p. V-4.

maximum 8-hour average of 0.08 ppm is exceeded on an average of 11 days during the O_3 season in non-MSA areas. This "exceedance rate" drops to 1 for a 0.10 ppm "cutpoint" concentration and slightly less than 1 for a 0.12 ppm cutpoint concentration.

Longer-term daily maximum O_3 averages in non-MSAs usually are lower than those seen in MSA areas. The maximum monthly means for 1- and 8-hour daily maximum averages generally are 0.067 ppm and 0.058 ppm, respectively. Means as high as 0.118 ppm and 0.092 ppm for these two averaging times have been seen in non-urban areas, however. High monthly means for either averaging time occur most often in May, June, and July.

Longer-term (≥ month) air quality indicators are more closely associated with each other than with short-term indicators. In fact, correlations among long-term air quality indicators are higher than correlations among short-term indicators. This is to be expected, since long averaging time statistical measures are relatively less variable than short averaging time measures. (Figure A-8 in Appendix A provides estimates of correlations among selected air quality indicators in non-urban areas.)

Relationships can be developed among air quality indicators in non-MSA areas that can be used to estimate the impact that attaining a standard for one averaging time will have on other averaging time O_3 concentrations. Some of these are presented in Appendix A.

C. Natural Ozone Background

Ozone is a trace constituent of the atmosphere. There is controversy regarding how much of ambient O_3 monitored at ground-level is natural and how much is produced from man-made precursors. Estimates of the natural component of O_3 vary widely in the literature, and there is no standardized

terminology regarding the concept of natural O_3 background.[3] Even when a numerical estimate of background (however labeled) is provided, rarely is the averaging time provided for the estimate.

Based on a thorough review of this literature, it is obvious that natural O_3 background is a multidimensional and complex concept. Natural O_3 background concentrations vary by geographic location, altitude and season.

A working definition of natural O_3 background is:

> For a particular geographic area and averaging time, natural O_3 background is that constituent of "background O_3" (O_3 that cannot be affected by manipulating anthropogenic emissions in an area) which arises solely from photochemically-reacted biogenic precursors and from stratospheric O_3 transported downward into the area (either directly or indirectly).

Note that this definition is predicated upon the concept that natural background O_3 cannot be affected by manipulating anthropogenic emission sources. Note also that the definition does not include some constituents of natural background that are considered by many to be background O_3. Excluded constituents are (1) trapped anthropogenic-based O_3 downwashed into an area due to breakup of a morning inversion, (2) nocturnal O_3 maxima due to downward mixing of O_3-rich air from above the inversion layer, (3) transported O_3 in an urban or stationary source plume, and (4) anthropogenic-based O_3 that is formed and/or stored in the troposphere and subsequently downwashed.

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[3]In fact, a survey of the available literature that mentions background O_3 or natural O_3 background--approximately 50 articles--did not uncover a single rigorous definition of either term! Even the appellations used for the concepts vary greatly in the relevant literature. Examples include: "baseline O_3," "clean air background," "global background," "North American background," "Urban background," and "regional surface background." In addition, twelve other labels were used--all without being defined.

A reasonable estimate of the natural O_3 background concentration near sea-level in the U.S. for an annual average is 0.020-0.035 ppm. This includes a 0.010-0.015 ppm contribution (averaged over time) from the stratosphere and a 0.01 ppm contribution from photochemically-affected biogenic non-methane hydrocarbons. In addition, another 0.01 ppm is possible from the photochemical reaction of biogenic methane.

A reasonable estimate of natural O_3 background concentration for a 1-hour daily maximum at sea-level in the U.S. during the summer is on the order of 0.03-0.05 ppm.

These estimates are synthesized from the available literature, but rely most heavily on Altshuller (1986) and Kelly et al. (1984).

V. Ozone Exposure Analysis

Analysis of population exposure under alternative NAAQSs requires that significant factors contributing to total human exposure be taken into account. These factors include the temporal and spatial distribution of people and O_3 concentrations throughout an urban area as people go through their typical daily pattern of life. To the maximum extent possible with existing data, this has been done in the NAAQS Exposure Model (NEM), a simulation model designed to estimate human exposure in selected urbanized areas under user- specified regulatory scenarios (Biller et al., 1981). This chapter is a summary of information provided in Paul et al. (1986), which has been used in development of the risk assessment described in Chapter VII.

A. Overview of the Ozone NAAQS Exposure Model

The O_3 NEM model partitions all land within a selected urban area into large "exposure districts" (Paul et al., 1986). There are between three and fifteen exposure districts identified in the ten urban areas used in the O_3 NEM analysis. The number of districts identified is directly related to the number of monitors having valid air quality data in a study area.

People living within each exposure district, as estimated by the U.S. Census in 1980, are assigned to a single discrete point, the population centroid. The air quality level within each exposure district is represented by air quality at the population centroid, which is estimated for each hour of the year by monitoring data from nearby monitoring sites. Because pollutants in the ambient air are generally modified considerably when entering a building or vehicle, outdoor air quality estimates are adjusted to account for five different microenvironments: indoors at home, indoors other (all other indoor locations), inside a transportation vehicle, outdoors near a roadway, and all other outdoor locations. The air quality adjustments are made by using microenvironmental transformation factors, as explained below.

Because degree of exposure and/or susceptibility to effects of pollution may vary with age, occupation, and intensity of exercise, the total population of each study area is divided into age-occupation (A-O) groups. Each A-O group is further subdivided into three or more subgroups. A typical pattern of activity through the five microenvironments is established for each subgroup and an exercise level (high, medium, or low) for each is also specified.

Units of population analyzed by NEM are called cohorts. Each cohort is identified by exposure district of residence, by exposure district of employment or school attendance, by A-O group, and by activity-pattern subgroup. During each ten minute period of the day, each cohort is assigned to a particular exposure district and a particular microenvironment. The assignment is based upon (1) data regarding human activity patterns gathered by EPA and university "time budget" researchers, and (2) home-to-work transportation data gathered by the U.S. Census Bureau. Using these sources of information, NEM simulates ten minute movements of cohorts through different districts of the urban area and through different microenvironments, combines the movements with hourly averaged air quality data, and accumulates the resulting exposures over the "O_3 season."[1]

B. Air Quality Concentrations in Microenvironments

NEM requires that hourly air quality estimates be available for each microenvironment that every cohort passes through. The estimates are obtained by using a simple linear function relating outdoor air quality concentrations to microenvironment levels via a "transformation" relating outdoor-to-indoor levels and then

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[1]The O_3 season is that part of the year for which O_3 monitoring is undertaken as required by EPA regulations for implementation of the O_3 NAAQS (40 CFR 58, Appendix D). For three urban areas that were modeled it is the entire year; for the remaining 7 areas, it is April through October.

adding to this the pollutant concentration due to sources located in the microenvironment itself. For O_3, the transformation is essentially a multiplicative ratio derived from a review of the O_3 exposure literature (Ferdo, 1985).

The relationship is:

$$x_{m,t} = a_{m,t} + (b_m * x_t)$$

where $x_{m,t}$ = air quality in microenvironment m during hour t

$a_{m,t}$ = hourly-averaged pollutant concentration due to sources located in the microenvironment

b_m = multiplicative ratio of the microenvironment concentration value to the monitored air quality value

x_t = monitor-derived air quality value for time t

Because no significant <u>sources</u> of O_3 were identified for any of the microenvironments, $a_{m,t} = 0$ for all environments. Estimated b_m values obtained from the literature are described in Paul et al. (1985).

The x_t values are actual monitored values for the current (or "as is") situation. These monitored values are adjusted mathematically to represent a future situation when air quality in the study area just meets the O_3 NAAQS being analyzed. By definition, a NAAQS is attained when all monitors in an area have less than one expected exceedance of the standard concentration value (currently it is 0.12 ppm) in a year. The exposure analysis is based on a "just attains" scenario, where air quality levels at the monitor currently having the highest number of expected exceedances are reduced mathematically to where that monitor just attains the standard being analyzed. Air quality data at other monitors in the study area are adjusted using a non-linear approach described in Paul et al. (1986).

C. Simulation of Population Movement

Population movement in NEM is based upon information gathered by the U.S. Census Bureau regarding householders' home-work commuting patterns (Bureau of the Census, 1982). The information includes MSA- specific data on the census tract level, which itself is based upon actual location information regarding the sampled population's home and workplace. This census tract information is aggregated for exposure districts used in the NEM analysis to obtain district-to-district trip information for those cohorts that work. (Otherwise, cohorts are assumed to stay in their home districts.) Because of lack of travel data for non-work activities, we assume that all shopping is done in each cohort's residential district. The same assumption also is made regarding school-related activities.

Three one-way commuting times are used to represent non-household worker commute times: 20, 30, and 40 minutes. Most workers fall into the 20 minute commute time (representing the rather large interval of 0 to 24 minutes), since the average commuting time in the United States is about 20 minutes. Housewives/househusbands are assumed to have no commuting time.

The number of different cohorts explicitly modeled in an area is equal to the product of 54 cohort groups times the square of the number of districts identified in each study area. Thus, the number of cohort groups explicitly modeled varies between 486 and 12,150 in the ten urban area sample. (See Table V-1.)

There is no trip information available from the U.S. Census Bureau regarding weekend inter-district travel in SMSAs. (There is information available regarding weekend recreational travel, but it is not locationally specific nor is it systematic.) Consequently, all cohorts are assigned to their home districts on weekends in NEM analysis.

D. Study Areas Modeled in Ozone National Exposure Model

As mentioned, ten urban areas are used to model O_3 exposures explicitly. Results from these ten areas will be extrapolated to

Table V-1

STUDY AREAS MODELED IN OZONE-NEM

Study Area Name	MSAs Included (in whole or in part)	Study Area Population Modeled (10^6)	Total Population of Included MSAs (10^6)	2nd Highest Daily Maximum O_3 Concentration (ppm)	Number of Exposure Districts	Number of Cohorts Modeled	EPA Region Number(s)
Chicago	Aurora-Elgin, IL Chicago, IL Gary, IN Joliet, IL Lake County, IL	7.48	7.82	0.17	9	4,374	5
Denver	Boulder, CO Denver, CO	1.54	1.62	0.14	6	1,944	8
Houston	Houston, TX	2.54	2.74	0.19	7	2,646	6
Los Angeles	Anaheim, CA Los Angeles, CA Riverside, CA San Bernardino, CA	10.22	10.97	0.37	15	12,150	9
Miami	Fort Lauderdale, FL Miami, FL	1.55	2.65	0.12	4	864	4
New York	Middlesex, NJ Nassau-Sulfolk, NY Newark, NJ New York, NY Stamford, CT	13.62	13.85	0.25	10	5,400	1,2
Philadelphia	Philadelphia, PA	4.36	4.72	0.20	7	2,646	3
St. Louis	St. Louis, MO	2.20	2.38	0.18	8	3,456	5,7
Tacoma	Tacoma, WA	0.58	0.46	0.14	3	486	10
Washington, DC	Washington, DC	2.88	3.25	0.14	8	3,456	3

the nation's urban population as a whole to obtain aggregated national exposure estimates. The study areas and their base population, along with other pertinent area information, appear in Table V-1.

The ten areas vary greatly in geographical location, O_3 "design value",[2] population size (both modeled and total MSA), and number of exposure districts included. The areas were selected to obtain as widely representative a modeling data base as possible given the overall need for monitoring data completeness in an area.

For instance, study area populations modeled vary from Tacoma, with a population of almost 580,000, to New York, with a population of about 13.6 million people. Design value concentrations vary from the current standard level of 0.12 ppm to 0.37 ppm--the highest design value included in EPA's regulatory data base within the last five years.

The number of exposure districts in study areas varies between 3--the minimum thought necessary to adequately capture the variation in O_3 air quality for an area--and 15. The number of exposure districts used in an area directly translates into the number of cohort groups in the area that are tracked through time and space in O_3-NEM. Each one of these cohort groups experiences a different pattern of air quality as its weekday activities are simulated. Thus, in Los Angeles over 12,000 different patterns were modeled.

One limitation of the exposure assessment is that it does not provide good coverage of the New England area (only Stamford, CT is included). This is a limitation worthy of note since the

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[2]A design value is that measured air quality concentration value in a MSA that must be reduced to the O_3 standard level to ensure that the area meets the current O_3 NAAQS formulation of ≤ 1 expected exceedances of 0.12 ppm daily maximum 1-hour average. The value shown in Table V-1 is the second-highest 1-hour daily maximum concentration in the O_3 air quality data base for the year modeled. See the footnote on p. IV-2 for additional information regarding design values (and other CHCs).

CT is included). This is a limitation worthy of note since the New England area has high population densities and high O_3 levels. The staff was unable to adequately model the New England area because of (1) the adequacy of monitoring data suited for exposure assessment and (2) U.S. Census Bureau data--both population and transportation--are difficult to use in New England due to the political/spatial classification system used there. No other MSA in New England could be explicitly modeled for these two reasons.

E. Exercise Modeling in Ozone NAAQS Exposure Modeling

Because dose received by a person exposed to an air pollutant is highly dependent upon her or his ventilation rate, exercise level is an important consideration in exposure modeling. In O_3-NEM, four exercise levels are considered. They are listed here, along with their associated ventilation rates (in units of liters per minute, or l/min):

1. low exercise, 25 L/min or less
2. medium exercise, 26-43 L/min
3. heavy exercise, 44-63 L/min
4. very heavy exercise, 64 l/min or higher

Two broad cohort group distinctions are made in O_3-NEM with respect to exercise: exercisers and non-exercisers. Exercisers are further divided into those who exercise heavily and those who exercise very heavily. Exercisers are those cohorts who participate in any activity that requires a ventilation rate greater than 43 L/min--i.e., the last two exercise categories. Heavy exercisers participate in an activity level requiring more than 63 L/min of ventilation.

Exercise level designations used in NEM are roughly adjusted for age and body size. For example, a baby's "thrashing around" is heavy exercise for the 0 to 6 month old cohort, but the same type of activity in an adult cohort is low exercise. Exercising cohorts are emphasized in O_3-NEM because exercisers, per se, may be "sensitive" (react more) to O_3 exposure. In fact, 35 of the

54 cohort groups used in NEM are exercisers. In most of the ten study areas modeled, however, these 35 exercising cohorts constitute less than 40% of a MSA's population. Twenty cohort groups (included in the 35) participate in very heavy exercise, but contain at maximum approximately 7% of any MSA's population. Thus, both exercising and cohorts are over-sampled in O_3-NEM.

The amount of time spent in exercise for any cohort is quite small. The largest portion of any cohort group undertaking heavy exercise is 6.2% for cohort #3, which consists of children 1 to 2 year old. (This cohort constitutes only about 1.0-1.5% of a MSA's total population.) The proportion of time spent in heavy exercise by people in cohorts that undertake such exercise is less than 2%, on average.

The largest portion of any cohort group undertaking very heavy exercise is 100% for cohorts #52-54, consisting of male outdoor workers. Less than 2% of any study area's population is included in this occupational grouping. The usual proportion of exercising cohort population participating in very heavy exercise is less than 1%.

The combination of small exercising fractions and small amounts of time spent in heavy or very heavy exercise results in a very small fraction of total population-time devoted to exercise. For one modeled study area, New York, the amount of total population-time spent in exercise is:

1. 0.9% for heavy exercise (undertaking exercise at a level of 44-63 L/min).
2. 0.002% for very heavy exercise (undertaking exercise at a level of 64 or higher L/min).

The other modeled MSAs have similar, but not identical, fractions of total population-time spent in exercise.

F. Eight-Area Aggregated Estimates of Population Exposure to Alternative Ozone Standards

Although ten urban areas were modeled using O_3-NEM, only eight areas were modeled for the "headcount" risk assessment that

is described in Section VII-B.[3] The total population included in the aggregation is 25.9 million people, and the total number of one-hour exposure occurrences for this population is 125 billion person-hours annually.

Since the lung function/symptom health risk assessment currently focuses on heavily exercising individuals as a "sensitive population" with respect to O_3 exposure, we present exposure estimates for only that group. Thus, the exposure estimates that follow are for people who are exercising at a ventilation rate of between 44 and 63 L/min. Because only a small portion of total population- time is spent in this exercise category, the number of people in the aggregation drops to 9.3 million and the one-hour exposure occurrences drops to 2.6 billion person-hours. These are the "base numbers" for the exposure estimates contained later in this chapter.

In any NEM analysis, three different indicators are used to estimate exposure of people to various levels of air pollution. One unit is "occurrences of exposure:" the number of times a given level of pollution is experienced by one individual. If 30 people experience a pollutant level of 1 ppm which remains steady over a 3-hour period, population exposure can be expressed as 30 occurrences of exposure for a 3-hour averaging time or 90 occurrences for a 1-hour averaging time. A second indicator of population exposure is "people-exposed." This is simply the number of people who experience a given level of air pollution, or higher, at least one time during the time period of analysis. The third indicator of exposure used in NEM is "people at peak exposure." This is the number of people who experience their

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[3]A computer error was found in the exposure output programs of two urban areas: Chicago (for the 0.08 ppm standard) and New York (for "as is" case). The error could not be corrected in time for use in the lung function/symptom risk assessment. Consequently, exposure estimates were simply aggregated for the remaining eight urban areas and used in the assessment. The eight-area aggregate will be discussed here for purposes of continuity with Section VII-B.

highest pollutant level within a given concentration interval. Examples of the first two output measures will be provided in this Section.

Four alternative air quality scenarios are modeled. The first is the "as is" situation, which uses recent monitored air quality data to represent O_3 concentrations in the outdoor-other microenvironment. (This concentration is then modified by the microenvironmental factor to estimate O_3 levels in the other microenvironments.) Air quality for the "as is" case comes from the 1983-1985 time period, using whichever year has the most complete data base.

The remaining air quality scenarios represent the hypothetical situation when a national ambient air quality standard for O_3 is just attained in an urban area. This situation is simulated by adjusting current air quality data so that the worst monitor in the urban area has a CHC[4] equal to the NAAQS concentration level. The adjustment procedure is complex and nonlinear. (For instance, peak hourly concentrations are adjusted more-absolutely and relatively--than those near the mean of the "as is" distribution.) It utilizes regression analyses of parameters of the Weibull distribution fit to each valid monitor in the urban area. For more information regarding the air quality adjustment procedure used to simulate a just-attaining situation see Paul et al. (1986).

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[4]The CHC in this case is the characteristic largest daily maximum (CLDM) version. The worst monitor in most MSAs is located in a downwind suburb of a central city. Most of the people in the MSA never experience the high O_3 levels seen at that monitor, since they may not travel into that exposure district as they go about their daily activities. Even people living in that district do not experience the high concentrations seen at the monitor, because (1) high O_3 values often occur in the mid- to late-afternoon when many district residents are at work in other less polluted districts, and (2) most people spend most of their time indoors, and do not directly experience high outdoor O_3 concentrations. When the worst monitor in an area attains an alternative O_3 NAAQS, all other monitors experience better air quality <u>by definition</u>.

Three alternative O_3 NAAQS are simulated: 0.08, 0.10, and 0.12 ppm. The latter value, of course, is the current standard level of the O_3 NAAQS. It should be recognized that we are not concerned in our exposure analyses about how or when an alternative O_3 NAAQS is attained. That is the concern of other analyses which OAQPS and other EPA offices undertake: especially the regulatory and benefits analyses. For O_3 exposure analyses purposes, it is sufficient to simulate the just-attaining situation without being concerned about how, when, or even if that situation will occur.

Estimates appear in Table V-2 of the cumulative numbers of people (in millions) who experience increasing 1-hour O_3 exposures as they engage in heavy exercise. These estimates are based upon using "best estimate" microenvironment factors. The first column shows the O_3 concentration that is equaled or exceeded, and the other four columns show cumulative exposure distributions for the four air quality scenarios previously discussed. Because all people experience the same number of exposures at or above 0.0 ppm, the entire aggregated population base of 9.3 million exercise is shown for all scenarios.

The overall pattern of data shown in Table V-2 is common to most of the exposure results that follow. Basically, that pattern is:

1. there is a long "tail" to the current situation scenario, in that there are tabular entries for the higher O_3 concentration "cutpoints," even up to 0.34 ppm.
2. There is a short tail to the remaining scenario distributions, with very small tabular entries at or above the O_3 concentration used for the scenario standard.

What this pattern implies is that attainment of any alternative NAAQS investigated results in a dramatic reduction in peak O_3 exposures. For instance, the estimate of the number of persons exposed during heavy exercise to O_3 levels $\geq$0.12 ppm drops to 0.1

Table V-2

ESTIMATE OF THE CUMULATIVE NUMBER OF HEAVY EXERCISERS IN THE 8-AREA AGGREGATION POPULATION EXPOSED TO ONE-HOUR AVERAGE OZONE CONCENTRATION DURING THE OZONE SEASON AT HEAVY EXERCISE UNDER ALTERNATIVE AIR QUALITY SCENARIOS

(millions of people)

O_3 Conc. Equaled or Exceeded (ppm)	Air Quality Scenarios: The "As Is" Situation	Alternative NAAQS (in ppm): 0.12	0.10	0.08
0.361	0	0	0	0
0.341	*	0	0	0
0.321	*	0	0	0
0.301	0.1	0	0	0
0.281	0.1	0	0	0
0.261	0.3	0	0	0
0.241	0.5	0	0	0
0.221	0.7	0	0	0
0.201	1.3	0	0	0
0.181	1.6	0	0	0
0.161	2.6	0	0	0
0.141	3.3	0	0	0
0.121	4.0	0.1	0	0
0.101	5.2	0.3	*	0
0.081	6.9	2.2	0.9	*
0.061	8.6	6.5	4.9	2.0
0.041	9.1	8.7	7.9	6.9
0.021	9.3	9.3	9.3	9.1
0.001	9.3	9.3	9.3	9.2
0.000	9.3	9.3	9.3	9.3

*Fewer than 50,000 people.

million with attainment of a 0.12 ppm NAAQS, as compared to 4.0 million people under "as is" O_3 air quality conditions. Attaining a tighter O_3 NAAQS, either 0.10 or 0.08 ppm, reduces the number of people- occurrences $\geq$0.12 ppm to zero.

A different type of exposure statistic is depicted in Table V-3. The subject of the Table is the estimated number of people-occurrences of exposure to various O_3 levels as they undertake heavy exercise. The universe for this aggregation is 2.6 billion person-occurrences. As can be seen, the number of heavy exercising person-occurrences of exposure $\geq$.12 ppm drops radically from the "as is" situation with attainment of one of the alternative standards. It should be realized, however, that the relative proportion of heavy exercising person-occurrences estimated for O_3 exposure levels at or above 0.12 ppm is fairly small, even for the "as is" situation. For example, it is 1.7% of total heavy exercising person occurrences at $\geq$.12 ppm and is 0.2% at $\geq$0.20 ppm. With attainment of a 0.12 ppm O_3 NAAQS in all areas, the proportion of heavy exercising person occurrences of O_3 exposure above that concentration would only be 0.003%.

G. Caveats and Limitations

A number of caveats must be acknowledged concerning the O_3-NEM results. These must be recognized by the reader in her or his interpretation of exposure estimates. Probably the most important caveat is that there is considerable uncertainty concerning a number of important inputs to O_3-NEM, especially those regarding human activity and exercise patterns. Uncertainty also exists regarding (1) predicted O_3 concentrations upon attainment of alternative O_3 NAAQS, (2) microenvironment factors relating outdoor-to-indoor concentrations, and (3) extrapolation of urban area-specific exposure estimates to the nation as a whole.

Some of these uncertainties are addressed by use of the "low, "best," and "high" estimates, but not all sources of

Table V-14

Estimate of the Cumulative number of Person-Occurrences of Heavy Exercise in the 8-Area Aggregation Population Exposed to One-Hour Average Ozone During the Ozone Season at Very Heavy Exercise Under Alternative Air Quality Scenarios

(millions of person-occurrences)

O_3 Conc. Equaled or Exceeded (ppm)	Air Quality Scenario			
	The "As Is" Situation	Alternative NAAQS (in ppm)		
		0.12	0.10	0.08
0.361	0	0	0	0
0.341	*	0	0	0
0.321	*	0	0	0
0.301	0.1	0	0	0
0.281	0.3	0	0	0
0.261	0.6	0	0	0
0.241	1.4	0	0	0
0.221	2.7	0	0	0
0.201	4.3	0	0	0
0.181	6.8	0	0	0
0.161	14.4	0	0	0
0.141	24.9	0	0	0
0.121	45.2	0.1	0	0
0.101	67.4	0.7	*	0
0.081	160.9	10.5	1.7	*
0.061	343.5	77.8	39.0	8.4
0.041	758.0	439.2	352.6	209.0
0.021	1,622.4	1,655.7	1,617.0	1,544.9
0.001	2,348.3	2,348.3	2,348.3	2,326.1
0.000	2,610.3	2,610.3	2,610.3	2,610.3

*Fewer than 50,000 person-occurrences

Source: Summarized from O_3 NEM Results

uncertainty are so addressed. The Ambient Standards Branch (ASB) will attempt to explicitly analyze the uncertainties mentioned above in future O_3-NEM modeling efforts, if contract funds are available for that purpose. In addition, due to increasing interest in multiple-hour O_3 exposures it is very likely that the current O_3-NEM will not be used in the future as it only provides hourly exposure estimates. ASB's plan is to revert back to the CO-NEM and update it to handle 10-minute or "probabilistic" activity patterns. Thus, future O_3 exposure estimates may be very different than those reported here.

VI. Factors Relevant to Review of the Primary Standard(s) for Ozone

Of primary concern in this section are the health effects associated with levels of O_3 and other photochemical oxidants that are observed in the ambient air of the United States. Of the photochemical oxidants, only O_3 has been reported to exist in cities at sufficiently high concentrations to be of significant concern for human health. Since other photochemical oxidants such as hydrogen peroxide (H_2O_2) and peroxyacetyl nitrate (PAN) are known to produce health effects only at concentrations much higher than those found in the ambient air, this section will focus on the mechanisms of toxicity for O_3 and documented evidence of pulmonary and extrapulmonary effects of O_3. This approach will be based on both available human and animal data. Mechanistic studies are used to support the possibility of human health effects caused by O_3 where there is an absence of adequate data. In addition, factors affecting susceptibility and potentially susceptible groups will be discussed.

A. Ozone Absorption and Mechanisms of Effects

Ozone enters the human body through the respiratory system. During inhalation and exhalation, O_3 is removed from the air by airway surfaces through a process of dissolution and chemical reaction. The rate, amount, and sites of O_3 uptake and removal largely determine the extent to which exposure of an individual to a particular concentration of O_3 will evoke adverse health effects.

Numerous studies help to explain the quantity and sites of O_3 uptake in mammalian respiratory tracts. Nasopharyngeal removal studies reviewed in the CD (p. 9-4) suggest that: 1) the fraction of O_3 uptake depends inversely on the flow rate, 2) tracheal and exposure chamber concentrations of O_3 are positively correlated (Yokoyama and Frank, 1972; Moorman, et al., 1973; and Miller et al., 1979) and 3) approximately 50 percent of inhaled O_3 (> 72% for dogs and > 50% for rabbits) is removed in the

nasopharyngeal region of animals exposed to between 0.1 and 0.2 ppm O_3, thereby indicating the role of the nasopharynx in removing O_3 before it reaches the more sensitive lung tissues. Only one study measured O_3 uptake in the lower respiratory tract and reported 80 to 87% uptake by the lower respiratory tract of dogs (Yokoyama and Frank, 1972). There are, however, no published data currently available for human nasopharyngeal or lower respiratory tract absorption.

Inhaled O_3 not absorbed in the nasopharyngeal region can be deposited along the entire respiratory tract. Penetration into the lower respiratory tract increases in rats as inhaled O_3 concentrations increase from 0.2 to 0.8 ppm (Dungworth et al., 1975). Models have been developed to estimate lower respiratory uptake (Miller et al., 1978b, 1985; McJilton et al. 1972). One O_3 deposition model has predicted that similar patterns of O_3 deposition occur in humans, rabbits, and guinea pigs and that the junction of the conducting airways and gas exchange region receives the maximal dose (Miller et al., 1978b; 1985). This prediction is consistent with location of O_3-induced lesions as indicated by pathology data from several species of O_3-exposed animals. Recently the Miller dosimetry model has been used to estimate sensitivity to lower respiratory tract secretions and exercise of the uptake of O_3 in the human lung (Miller et al., 1985). The Miller dosimetry model is also an important tool in assessing animal toxicology data. Chapter 9 of the CD provides a detailed discussion of the use and comparison of O_3 dosimetry models.

Biologically important functional groups which react relatively rapidly with O_3 include the alkenic groups (carbon-carbon double bonds), amino groups, and sulfhydryl groups. Alkenic bonds are found in essential fatty acids and polyunsaturated fatty acids (PUFA) which are important components of the lipids in cell membranes. Oxidation of amino and sulfhydryl groups can result in protein denaturation with

concomitant loss of structural and functional integrity for the affected protein.

Although there is general agreement that the oxidative properties of O_3 cause toxic effects, the precise molecular mechanism of toxicity remains unclear. Several theories have been proposed:

1. Oxidation of polyunsaturated lipids contained mainly in cell membranes;

2. Oxidation of sulfhydryl, alcohol, aldehyde, or amine groups in low molecular weight compounds or proteins;

3. Formation of toxic compounds (ozonides and peroxides) through reaction with polyunsaturated lipids;

4. Formation of free radicals, either directly or indirectly, through lipid peroxidation; and

5. Injury mediated by some pharmacologic action, such as via a neurohormonal mechanism or release of histamine (CD, p. 9-113).

Molecular targets (e.g., carbon - carbon double bond, sulfhydryl groups) are shared across all species. Therefore, if O_3 initiates mechanisms such as those listed above in an animal which ultimately result in lung structural damage, there is reason for concern that the same mechanisms may be activated in humans with similar outcomes. This assumption is limited by differences in human and animal dosimetry which produce different doses of O_3 for equivalent exposures. Also defense and repair mechanisms are likely to provide quantitative differences in the display of toxicity resulting from equivalent doses. Nonetheless, mechanistic information strongly supports the hypothesis that equivalent effects may occur in humans and animals, albeit not necessarily at the same concentrations.

B. Factors Affecting Susceptibility to Ozone

There are numerous factors which could affect susceptibility to O_3 exposure and to alter physiological responsiveness. These factors include such individual characteristics as age, sex,

smoking status, and nutritional status. In addition, environmental stresses and exercise level during exposure can influence the extent and level of an individual's response to O_3 by increasing the volume of inhaled O_3 and by promoting penetration deeper into the lungs.

1. Age

It has been postulated that age-related changes in lung growth and development are among the factors responsible for altering individual susceptibility to O_3; thus, extent of damage may depend on the stage of lung development. Support for this hypothesis has come from several human studies, but definitive evidence is unavailable at this time.

Several clinical, field, and epidemiology studies have provided evidence of pulmonary function and/or symptomatic response in younger persons exposed to less than 0.15 ppm O_3 (McDonnell et al., 1985b,c; Avol et al., 1985a,b; Lebowitz et al., 1982, 1983; Lebowitz, 1984; Lippmann et al., 1983; Lioy et al., 1985; Bock et al., 1985; Spektor et al., 1988a). In addition, symptoms have been reported for children exposed to oxidant concentrations of 0.10 ppm and higher (Makino and Mizoguchi, 1975). Although it has been suggested that older individuals may be less susceptible to O_3 exposure than younger persons, age differences in response to O_3 have not yet been fully investigated in controlled human studies. Because children tend to exercise outdoors in the summer more often than adults, there may be more reason for concern about effects of O_3 in children.

Reisenauer et al. (1988) find that only female subjects show a rise in total respiratory resistance (R_T) when exposed to 0.3 ppm O_3 during exercise. Drechsler-Parks et al. (1984) also report no significant differences between older males and females exposed to higher O_3 levels (0.45 ppm) for 2 hours during intermittent exercise (25 L/min) and suggested that older individuals may be less responsive to O_3 than younger persons.

Bedi et al. (1988) substantiate these results in finding that older subjects, as a group, are less responsive to O_3 than younger subjects. Thus, while there continues to be no controlled human experimental evidence of additive effects from exposure to other pollutants combined with O_3 or of differential response due to sex, preliminary evidence suggests possible age-related differential response.

The few animal studies that have addressed the potential susceptibility of the young have shown a few differences between neonates and young adult animals. Generally, however, the very young rats were not more responsive than the young adult rats (CD, p. 12-36).

Conclusions regarding age as a susceptibility factor in humans will remain uncertain until the results of additional research are available (CD, p. 12-35). While more information is needed to fully understand age-related differences, it should be noted that recent studies by Reisenauer et al. (1988) and Drechsler-Parks et al. (1984) provide evidence of reduced responsiveness in older adults.

2. Sex

Due to the small number of female subjects in controlled human studies, no definitive conclusions can be drawn regarding sex differences in response to O_3 exposures. Those human studies which have evaluated lung function differences report that forced expiratory volume for 1-sec ($FEV_{1.0}$) appears to be affected more in females than in males for similar exposure concentrations and exercise levels, but the results are not conclusive. Only four studies gave enough information for limited comparative evaluation (Horvath et al., 1979; Gliner et al., 1983; Delucia et al., 1983; Lauritzen and Adams, 1985). Results of these studies suggest that lung function of women may be affected more than that of men for similar O_3 exposures and exercise levels. Adams et al. (1983) report no differences in male and female response

when exposed to 0.3 ppm, 0.6 ppm, NO_2 separately or in combination.

Most O_3 research on animals has been performed using only males. When females were used, sex comparisons were not usually made. An exception is the study by Graham et al. (1981) which reports increased pentobarbital-induced sleeping time in all females but not in male mice or rats after 5 hour exposure to 1.0 ppm O_3. Although reasons for sex differences have not yet been elucidated, these differences have been suggested in some research and thus further investigation is needed (CD, p. 12-37). Because an inadequate data base prevents drawing definitive conclusions about sex differences, staff assumes at this time that there are no differences between sexes regarding response to O_3 exposure.

3. Smoking Status

Results of studies comparing the effects of O_3 on smokers versus non-smokers remain somewhat inconclusive, although below 0.5 ppm O_3 smokers appear to be somewhat less responsive. Two studies reported greater pulmonary function changes for non-smokers than for smokers at rest during exposure to O_3 levels of 0.37 to 0.5 ppm; this finding was reversed for higher O_3 levels (0.75 ppm), with smokers showing greater response (Hazucha et al., 1973; Bates and Hazucha, 1973). Greater pulmonary function response was also reported in non-smokers than in smokers at 0.5 ppm (Kerr et al., 1975) and at 0.3 ppm (Delucia et al., 1983). Effects of O_3 were greater for non-smokers compared to smokers at 0.5 and at 0.3 ppm (Kagawa and Tsuru, 1979a); in a subsequent study exercising non-smokers showed a greater response (SG_{aw}) than exercising smokers at 0.15 ppm O_3 (Kagawa, 1983). Exercising smokers also showed slower and smaller spirometric variable changes than non-smokers exposed 2 hours at 0.5 and 0.75 ppm O_3 (Shephard et al., 1983). Although none of these studies has examined the effects of different amounts of smoking, available data supports the contention that smokers are less

responsive to O_3 than non-smokers, at least for lower O_3 exposures. Why smokers appear to be less responsive to O_3 than non-smokers is not clear, though it has been suggested that the altered lung function and increased mucus production experienced by smokers could influence O_3 deposition in the lungs (CD, p. 12-37).

4. Nutritional Status

Results of studies investigating nutritional status as a factor affecting susceptibility to O_3 are inconclusive. Some inconsistencies between human and animal data are apparent but might be explained based on different experimental approaches.

Human subjects receiving 4 to 8 times the recommended daily allowance of vitamin E showed no statistically significant differences in blood biochemistry or agglutination compared to unsupplemented subjects after 2-hour exposures to 0.5 ppm O_3 (Posin et. al., 1979; Hamburger et. al., 1979a,b). Animal studies show that vitamin E deficiency makes rats more susceptible to O_3-induced enzymatic changes (Chow et. al., 1981; Plopper et. al., 1979; Chow and Tappel, 1972) and that vitamin E alters the rate and extent of O_3 toxicity but not the lesion in the centriacinar region of the lungs (Stephens et al., 1974; Schwartz et al., 1976). Lesions have been reported to be worse in vitamin E deficient rats or in rats marginally supplemented with vitamin E when compared to highly supplemented rats (Plopper et al., 1979; Chow et al., 1981); this supports results of mortality (Donovan et al., 1977) and biochemical studies suggesting that vitamin E is protective in rats (CD, p. 12-38).

Since redistribution of vitamin E from extrapulmonary stores in humans to the lungs is slow, it is possible that such a protective effect does not occur over short-term exposures to O_3 in human studies. Another possible explanation is that an effect is only seen when subjects are vitamin E deficient. Animal studies in which effects have been reported were conducted with a vitamin E deficient group for comparison and for long-term

exposures (Sato et al., 1976, 1978, 1980), neither of which is possible in human studies. Although laboratory animal studies of vitamin E support the hypothesis that lipid peroxidation is involved in O_3 toxicity, benefits to be derived from dietary vitamin E supplementation in connection with O_3 exposure have not yet been demonstrated in humans.

5. Environmental Stresses

Subjective symptoms and physiological impairment caused by O_3 exposure can be substantially worsened by environmental stresses such as heat and high relative humidity (rh). High temperatures (31 to 40°C) and/or humid conditions (85% rh) when combined with exercise during O_3 exposure have been shown to reduce FEV_1 more than similar O_3 exposures at a more moderate temperature (25°C) and humidity (50% rh) (Folinsbee et. al., 1977a,b; Gibbons and Adams, 1984). Enhanced effects of O_3 caused by high temperatures and humidity may be the result of higher ventilation which increases the volume of inhaled O_3 and promotes deeper penetration into the lungs. There may also be an independent effect of elevated body temperature on pulmonary function (CD, p. 12-35). Since many urban areas around the U.S. tend to experience the highest O_3 levels during periods of high temperatures and humidity, environmental stress should be considered a factor of concern in assessing potential effects from O_3 exposure.

6. Exercise

Exercise stresses respiratory, cardiovascular, and musculoskeletal systems and increases ventilation and, therefore, total dose to the lungs. Exercise thus should be considered an important susceptibility factor when O_3 exposure occurs concurrently. The most apparent and well-studied effects of O_3 during exercise occur in the respiratory system. In particular, pulmonary function decrements and respiratory symptoms caused by O_3 exposure are increased by a greater work load, which is

characterized by increased frequency and depth of breathing (Folinsbee et al., 1979, 1984; McDonnell et al., 1983, 1985a,b,c; Avol et al., 1984, 1985a,b). Representative activities and associated ventilation rates are summarized in Table VI-1 (CD, p. 10-14). Higher ventilation increases total volume of O_3 breathed and could cause deeper penetration of O_3 into peripheral areas of the lung which tend to be most sensitive to injury. Because most individuals engage in some type of outdoor exercise where O_3 exposure is possible during summer months, exercise should be considered a susceptibility factor in assessing health effects of O_3.

C. Potentially Susceptible Groups

The Clean Air Act requires EPA to set standards which protect the health of individuals who are potentially susceptible to O_3 exposure. This section identifies potentially susceptible groups or subpopulations and provides a rationale for selecting these groups.

Susceptibility to any pollutant may depend upon many factors such as those previously discussed (age, sex, smoking, nutrition, environment, and exercise) but also is influenced by individual sensitivity. Greater than normal sensitivity to a particular pollutant may be conferred by numerous individual characteristics including (1) predisposition to pulmonary infection, (2) preexisting disease or nutritional deficiency, (3) some aspect of growth or decline in lung development, (4) a prior infection or immunological problem, (5) sensitization caused by prior pollutant exposure or challenge with respiratory irritants and (6) genetic variability in the population. These sensitivity and susceptibility factors should be considered in identifying groups or subpopulations which may be susceptible to O_3 exposure.

1. Individuals Having Preexisting Disease

The first major group identified in the CD (p. 12-73) as appearing to be at particular risk to O_3 exposure is that group

TABLE VI-1. ESTIMATED VALUES OF OXYGEN CONSUMPTION AND MINUTE VENTILATION ASSOCIATED WITH REPRESENTATIVE TYPES OF EXERCISE[a]

Level of work	Work performed watts	Work performed kg-m/min[b]	O_2 consumption, L/min	Minute ventilation L/min	Representative activities[c]
Light	25	150	0.65	12-16	Level walking at 2 mph; washing clothes
Light	50	300	0.96	17-23	Level walking at 3 mph; bowling; scrubbing floors
Moderate	75	450	1.25	23-30	Dancing; pushing wheelbarrow with 15-kg load; simple construction; stacking firewood
Moderate	100	600	1.54	29-38	Easy cycling; pushing wheelbarrow with 75-kg load; using sledgehammer
Moderate	125	750	1.83	35-46	Climbing stairs; playing tennis; digging with spade
Heavy	150	900	2.12	42-55	Cycling at 13 mph; walking on snow; digging trenches
Heavy	175	1050	2.47	52-57	Cross-country skiing; rock climbing; stair climbing with load; playing squash and handball; chopping with axe
Very heavy	200	1200	2.83	62-79	
Very heavy	225	1350	3.19	73-93	
Very heavy	250	1500	3.55	89-110	Level running at 10 mph; competitive cycling
Severe	300	1800	4.27	107-132	Competitive long distance running; cross-country skiing

[a]See text of Criteria Document (pp. 10-13 to 10-15) for discussion.

[b]kg-m/min = work performed each minute to move a mass of 1 kg through a vertical distance of 1 m against the force of gravity.

[c]Adapted from Astrand and Rodahl (1977).

characterized as having preexisting disease. This includes individuals with asthma, allergy, and chronic obstructive lung disease (COLD) such as emphysema. Asthmatics, who experience variable airway obstruction and/or reactivity and who may have altered immunological states (e.g., atopy) or cellular function (e.g., eosinophilia) may or may not be more sensitive to O_3 than non-asthmatics. They have not been shown to be more sensitive in numerous studies, which have shown asthmatics to have equal or lesser responsiveness. Asthma, however, is not a specific homogeneous disease and efforts to precisely define asthma have been unsuccessful. Susceptibility to O_3 of patients with COLD remains somewhat uncertain, depending on their clinical and functional state. Table VI-2 provides estimates of the number of individuals with these conditions (CD, p. 12-79).

Although several controlled human exposure studies suggest that adults with preexisting respiratory disease may not be more responsive to O_3 than healthy adults, two considerations place these subgroups at potentially higher risk to O_3 exposure according to the CD (p. 12-88). First, due to concern for the health of these persons, higher concentrations of O_3 and higher exercise levels have not been used during controlled exposure studies, thus making comparisons with healthy subjects difficult. A second consideration is that for individuals with already compromised respiratory systems, any further decrement in pulmonary function may be expected to impair the ability to perform normal activities. The same decrement in a healthy individual may go unnoticed. Increases in symptoms reported for asthmatics and allergic individuals exposed to O_3 may also be expected to interfere with normal function. Because recent studies (McDonnell et al., 1987; Eschenbacher et al., 1988; Kreit et al., 1988) have reported that both asthmatics and allergic subjects have a greater increase in airway resistance after O_3 exposure than do healthy subjects, the CD Supplement (U.S. EPA, 1988) has concluded that the order of airway responsiveness to O_3 is normal < allergic < asthmatic subjects.

TABLE VI-2

PREVALENCE OF CHRONIC RESPIRATORY CONDITIONS BY SEX AND AGE FOR 1979[a]

Condition[b]	Number of persons, in thousands							
	Total[c]	Male	Female	<17 years old	17-44 years old	45-64 years old	≥65 years old	% of U.S. population
Chronic bronchitis	7474	3289	4175	2458	2412	1547	1060	3.5
Emphysema	2137	1364	770	12[d]	127[d]	1008	990	1.0
Asthma[e]	6402	3113	3293	2225	2203	1482	488	3.0
Hay fever and other upper respiratory allergies[f]	15,620	7027	8584	3151	8278	3012	1181	7.2

[a]U.S. Department of Health and Human Services, 1981.

[b]Classified by type, according to the Ninth Revision of the International Classific of Diseases (World Health Organization, 1977).

[c]Reported as actual number in thousands; remaining subsets have been calculated from percentages and are rounded off.

[d]Does not meet standards of reliability or precision set by the National Center for Health Statistics (more than 30% relative standard error).

[e]With or without hay fever.

[f]Without asthma.

Source: O_3 Criteria Document, U.S. EPA, 1986.

2. Exercising Individuals

A second susceptible group potentially at higher risk to O_3 exposure is that group of individuals including both normal, healthy persons and those with preexisting respiratory disease whose regular outside activities cause increased minute ventilation. As stated in the CD (p. 12-71), "the most prominent modifier of response to O_3 in the general population is minute ventilation, which increases proportionately with increases in exercise workload." The increased proportion of oral breathing with increased exercise also contributes to the increase in effects since more O_3 is delivered to the lungs. Although individuals with preexisting respiratory or cardiovascular disease may not exercise as heavily as healthy persons, any increases in activity over resting levels will increase O_3 exposure and resultant effects.

Exercise has become recognized in numerous recent studies as a factor which can predispose all individuals to O_3 health effects (McDonnell et al., 1983, 1985a,b,c; Avol et al., 1983, 1984, 1985a,b; Folinsbee et al., 1978, 1984; Kulle et al., 1985). Thus, activities which increase minute ventilation out-of-doors will also increase the risk associated with exposure to O_3 of exercising individuals.

Unusual responsiveness to O_3 has been observed in some individuals. These individuals, often referred to as "responders," have not been characterized as having any particular medical problem but experience significantly greater pulmonary function decrements than the average response of the groups concurrently studied during O_3 exposure. It is not known if these individuals are a population subgroup with a specific risk factor or simply represent the upper 5 to 20 percent of the O_3 response distribution. As yet there is no means of identifying these highly responsive individuals prior to O_3 exposure.

Individual responsiveness to O_3 of apparently healthy, homogeneous groups has been shown to vary widely. For example,

during heavy exercise (minute ventilation, $\dot{V}_e$, = 45-51 L/min) and exposure to 0.4 ppm O_3, subjects showed FEV_1 decrements ranging from below 10% to as much as 40% of control values for those subjects with an average of 26% (Haak et al., 1984; Silverman et al., 1976). Mode (oral versus nasal) and pattern (rapid shallow versus slow deep) of breathing may contribute to intersubject variability but cannot fully explain it. Unsuccessful attempts have been made to determine which factors are responsible for modifying individual responsiveness (Hazucha, 1981). Factors such as prior exposure to O_3 or other pollutants, latent infections, and nutritional deficiencies may also contribute to differential response (CD, p. 12-22). The potential for these contributions is biologically plausible, but these have yet to be demonstrated in human subjects.

Although the specific factors which modify individual response to O_3 are not yet identified, it has been suggested that intersubject variability in the magnitude of effects induced by O_3 is caused by large differences in intrinsic responsiveness to O_3 (McDonnell et al., 1985a; Kulle et al., 1985). As measured by FEV_1 and FVC, individual responses to O_3 are highly reproducible for at least the 10 month period of observation and for a given individual differences tend to be much smaller than differences between subjects (McDonnell et al., 1985a; Gliner et al., 1983). Hence, some intrinsic factor appears to be responsible for individual responsiveness.

Changes in FEV_1 of exercising subjects exposed to clean air are small and normally distributed in the subject population. As O_3 concentrations increase this distribution widens and becomes skewed toward larger FEV_1 decrements, the largest changes representing the most responsive subjects (McDonnell et al., 1983; Kulle et al., 1985). Classification of subjects into "responders" and "non-responders" has been somewhat arbitrary. Examples of retrospective classification include identifying responders by using medical history and exposure test results (Hackney et al. 1975) and selecting those with greater than 10%

post-exposure decrements (Horvath et al., 1981) or with FEV_1 decrements greater than 2 standard deviations above control (Haak et al., 1984). While there are no clearly established criteria for identifying or selecting "responders," it has been suggested that between 5 and 20% of the healthy population may represent a subgroup of the population which is more responsive and, therefore, at higher risk to O_3 exposures (CD, p. 12-22). Further discussion of frequency distribution is in section VII.A.1.

VII. Assessment of Health Effects and Related Health Issues Considered in Selecting Primary Standard(s) for Ozone

This assessment of health effects attributed to O_3 and related health issues is based on the review and evaluation of health effects research literature contained in Chapters 9 to 12 of the CD as well as newer information discussed in the CDS.

A. Health Effects of Concern

For purposes of this staff paper and review of the NAAQS for O_3, the staff recommends that the following categories of health effects attributed to O_3 exposure be considered in developing the basis for the primary O_3 standard:

1. Alterations in pulmonary function
2. Symptomatic effects
3. Exercise performance
4. Bronchial Reactivity
5. Aggravation of existing respiratory disease
6. Morphological effects
7. Altered host defense systems
8. Extrapulmonary effects

Respiratory effects provide by far the strongest data base for considering adverse health effects of O_3. Extrapulmonary effects such as behavioral, blood chemistry, chromosomal, cardiovascular, reproductive, teratological, liver metabolism, and endocrine system modifications also have been associated with O_3 exposure, but most are considered to be of uncertain functional importance at this time and, thus, do not provide very strong evidence of adverse effects associated with O_3 exposures.

Evidence for the above effects has come from both human and animal studies. The strongest and most quantifiable data are provided by controlled human exposure studies, but these studies are limited to acute (short-term) exposures. Field studies are similarly limited, but permit investigation of the effects of oxidants in ambient air and allow for better characterization of exposure than epidemiological studies. Although use of most

community studies has been hampered by the difficulty in adequately characterizing exposure and numerous confounding variables, these investigations provide important supporting evidence for effects occurring in populations. Animal toxicology studies have provided evidence of some acute and chronic (long-term) exposure effects which can be detected only with invasive procedures, but uncertainties inherent in dosimetry and species sensitivity differences have limited quantitative extrapolation to humans.

Assessment of health effects attributed to O_3 requires consideration of the data base in each of the above areas of study. This section integrates research in each of these areas to provide an indication of the strength of the data base for each effect. A more thorough review and evaluation of individual studies is available in chapters 9 to 12 of the CD.

1. Alterations in Pulmonary Function

The best documented and strongest evidence of human health effects of O_3 exposure are pulmonary function decrements. Controlled human exposure, field, epidemiology, and animal toxicology studies have provided evidence that exposure to O_3 can modify such pulmonary measurements as forced expiratory volume (FEV), forced expiratory flow (FEF), forced vital capacity (FVC), vital capacity (VC), tidal volume (V_T), peak expiratory flow rate (PEFR), inspiratory capacity (IC), total lung capacity (TLC), airway resistance (R_{aw}) and breathing frequency (f_B). These and other terms are defined in Appendix B which is an abbreviated version of the glossary found in the CD.

Early controlled experimental studies of resting human subjects exposed to O_3 levels up to 0.75 ppm for 2 hours demonstrated little or no change in FVC (Silverman et al., 1976; Folinsbee et al., 1975;), FEV_1, and FRC (Silverman et al., 1976). Flow rate variables such as FEF 25% and FEF 50% showed up to 30% decreases in some subjects exposed at rest to 0.75 ppm O_3 (Bates et al., 1972; Silverman et al., 1976), while only small increases

in R_{aw} (< 17%) were reported for > 0.5 ppm O_3 exposures (Bates et al., 1972; Golden et al., 1978). More recent studies have reported occurrence of FEV and FEF decrements during resting exposures to ≥ 0.5 ppm O_3 (Folinsbee et al., 1978; Horvath et al., 1979); however, no statistically significant changes in R_{aw} and only suggestive changes in RV and TLC have been reported for similar exposures (Shephard et al., 1982). Airway resistance is not generally affected in resting subjects at these O_3 levels. Changes in pulmonary function have not been observed in resting subjects exposed to O_3 levels between 0.12 ppm (Koenig et al., 1985) and 0.3 ppm (Folinsbee et al., 1978), though some subjects exhibit O_3-induced pulmonary symptoms during resting exposures (König et al., 1980; Golden et al., 1978). In general, however, because subjects were at rest in most of the older studies, significant respiratory effects were not reported even for higher O_3 exposures.

Exercise, which causes increased minute ventilation ($\dot{V}_e$), enhances individual and group mean response to O_3 exposure. As discussed in section VI.B.6. of this staff paper, exercise increases breathing frequency and depth of breathing resulting in greater total dose of O_3 inhaled and increased penetration to the most sensitive lung tissue. As exercise levels increase to the point where $\dot{V}_e$ exceeds approximately 35 L/min, oronasal or oral breathing tends to predominate (Niinimaa et al., 1980); thus at higher exercise levels a greater portion of the inhaled O_3 will bypass the nose and nasopharynx (Niinimaa et al., 1981). Individual variability will affect the $\dot{V}_e$ at which oral or oronasal breathing predominates. The relationship between exercise and magnitude of response is illustrated quite well by Figure VII-1 prepared as Figure 12-6 for the CD (p. 12-81) showing group mean decrements in FEV_1 caused by exposure of exercising subjects to various O_3 levels based on results from 25 different studies. The curves clearly demonstrate that as exercise levels increase for a given O_3 exposure there is a resultant increase in the group mean FEV_1 decrements.

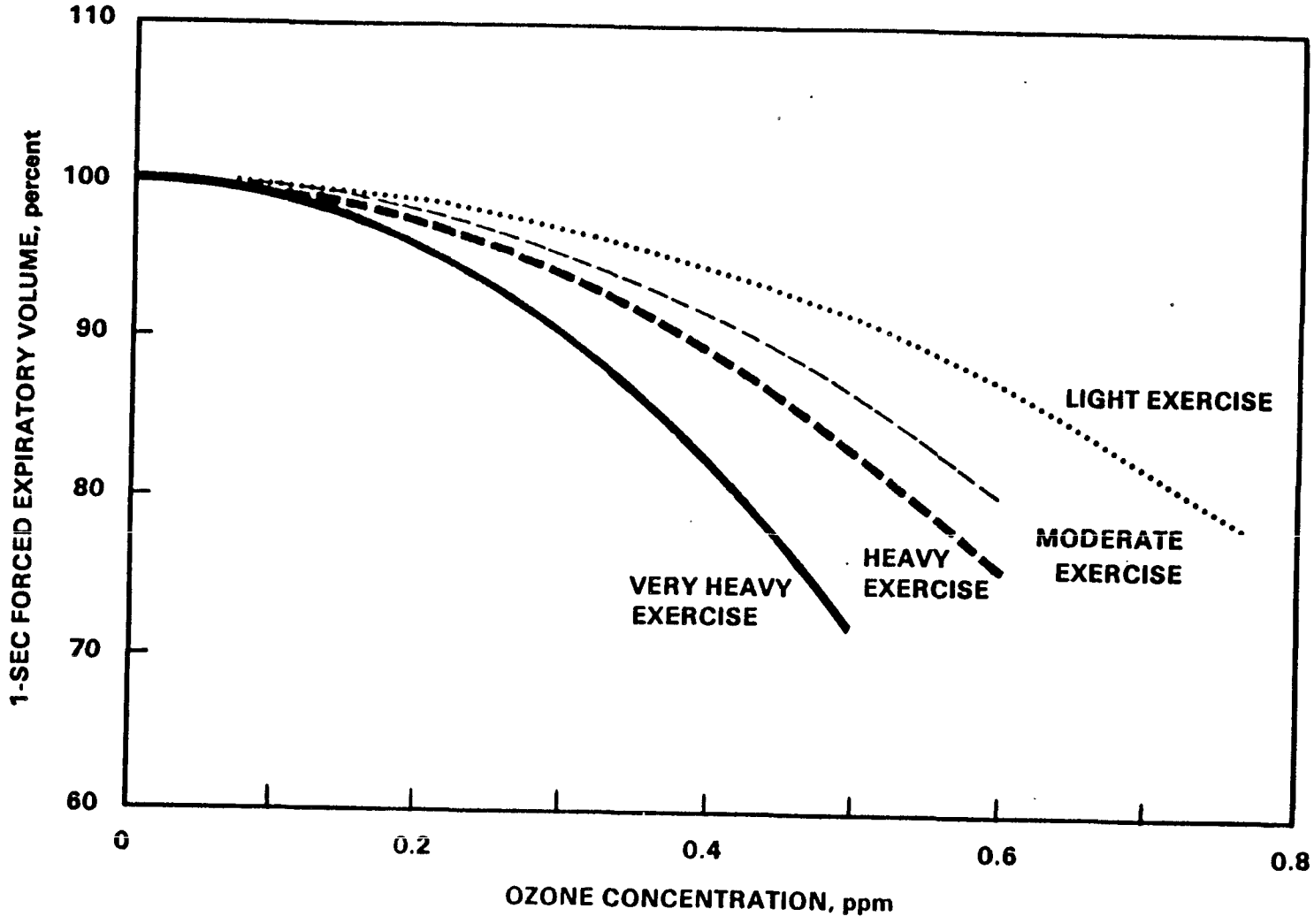


FIGURE VII-1. **Group mean decrements in 1-sec forced expiratory volume during 2-hr ozone exposures with different levels of intermittent exercise: light ($\dot{V}_E \leqslant 23$ L/min); moderate ($\dot{V}_E$ = 24-43L/min); heavy ($\dot{V}_E$ = 44-63 L/min); and very heavy ($\dot{V}_E \geqslant 64$ L/min). Concentration-response curves are taken from Figures 12-2 through 12-5.**

Source: O_3 Criteria Document, U.S. EPA, 1986

Pulmonary function decrements have been reported in controlled exposure studies of healthy exercising human adults exposed to O_3 levels between 0.12 ppm and 0.24 ppm, a range for which a concentration-response relationship has been established. During 2 hours of intermittent very heavy exercise ($\dot{V}_e$ = 64 L/min.), healthy subjects experienced group mean decrements in FEV_1 of 4.5, 6.2, and 14.5% for O_3 exposures of 0.12, 0.18, and 0.24 ppm, respectively (McDonnell et al., 1983). Positive associations between group mean magnitude of FVC (3, 4, and 12%) and FEF (7.2, 12, and 23%) decrements and O_3 exposures of (.12, .18, and .24 ppm) also were reported in the McDonnell et al. (1983) study; statistically significant increases in SR_{aw} and f_B did not occur until O_3 was $\geq$ 0.24 ppm. In a separate study, McDonnell et al. (1985b) reported a statistically significant but small decline (3.4%) in group mean FEV_1 of children (8-11 years) after 2 hours of exposure to 0.12 ppm O_3 during intermittent heavy exercise ($\dot{V}_e$ = 39 L/min). Furthermore, there was a suggestion that the small decrements in FEV_1 persisted for 16 to 20 hours after O_3 exposure ended. Findings by Lioy et al. (1985) provide epidemiological evidence that lung function decrements can persist in children up to a week following a smog episode in which 1-hour O_3 peaks were 0.135 to 0.186 ppm.

Support for O_3-induced pulmonary function changes also comes from other controlled exposure studies. Avol et al. (1984) report small but statistically significant decreases (6.1%) in FEV_1, at 0.16 ppm O_3 with a larger decrements (19.1%) at $\geq$ 0.24 ppm O_3. Decrements have been reported in FEV_1, FVC, and FEF for distance runners and distance cyclists exposed to 0.20 ppm and 0.21 ppm O_3 respectively, during 1 hour of continuous, heavy exercise ($\dot{V}_e$ = 77.5 and 81 L/min) (Adams and Schelegle, 1983; Folinsbee et al., 1984). Separate studies of continuously exercising males ($\dot{V}_e$ = 61.8 L/min) and females ($\dot{V}_e$ = 46 L/min) exposed to 0.2 ppm O_3 for about an hour showed no statistically significant FEV_1 decrements, but these negative results can be attributed in part to the small number of subjects (i.e.,

inadequate power), 8 male and 6 female respectively (Adams et al., 1981; Lauritzen and Adams, 1985). In another study involving O_3 concentrations ranging between 0.10 and 0.25 ppm, exponential decreases in FVC, FEV_1, FEF_{25-75}, SG_{aw}, IC, and TLC have been reported with exposure to increasing O_3 concentration during very heavy exercise ($\dot{V}_e$ = 68 l/min); time of exposure was related to linear decreases in FVC and FEV_1 (Kulle et al., 1985).

Field studies, which contain elements of both controlled human exposure and epidemiologic studies, provide the most quantitatively useful human exposure data available for ambient photochemical oxidants. Results from field studies are consistent with pulmonary function decrements reported in controlled O_3 exposure studies. For a 1-hour exposure to mean O_3 concentrations of 0.144 ppm, small significant group mean decreases in FVC (3.3%), $FEV_{0.75}$ (4.0%), FEV_1 (4.2%), MMFR (3.2%) and PEFR (3.9%) relative to pre-exposure levels were reported in 59 healthy continuously exercising ($\dot{V}_e$=32 L/min) adolescents (Avol et al., 1985 a, b). Based on the comparison of ambient air and control exposures in these studies, it appears that O_3 was the causative agent of the pulmonary function effects.

In a separate study of 50 healthy, adult, continuously exercising ($\dot{V}_e$ = 53 L/min) bicyclists, mean O_3 concentrations of 0.153 ppm for 1 hour produced statistically significant group mean decreases in FEV_1 (5.3%) compared to pre-exposure (Avol et al., 1984). This study showed that similar effects result in subjects exposed to comparable O_3 levels in ambient and controlled O_3 exposures. Small but statistically significant decreases in FEV_1 and FVC were also reported in exercising healthy and asthmatic adults during exposure to mean O_3 concentrations of 0.165 and 0.174 ppm (Linn et al., 1980, 1983b; Avol et al., 1983). Table VII-1 provides a summary of group mean % changes in FEV_1 for controlled exposure and field studies.

In summary, pulmonary function decrements have been reported in healthy adult subjects (18 to 45 years old) after 1 to 3 hours of exposure as a function of the level of exercise performed and

KEY HUMAN STUDIES NEAR THE CURRENT 1-HR NAAQS FOR OZONE

Ozone Concentration µg/m³	Ozone Concentration ppm	Measurement[a,b] Method	Exposure Duration	Activity[c] Level (V_E)	Percent Change in FEV_1[d]	Number, Sex, and Age of Subjects	Reference[e]
0 157	0.00 0.08	UV, UV	1 hr	CE (57)	+0.6 +1.7 (ns) (26.4 ± 6.9)	42 male 8 female	Avol et al. (1984)
0 157 196 235	0.00 0.08 0.10 0.12	UV, UV	2 hr	IE (68)	+1.0 +2.4 (ns) +1.7 (ns)	24 male (18-33 yr)	Linn et al. (1986)
0 157 196	0.00 0.08 0.10	UV, UV	2 hr	IE (68)	+1.0 +2.4 (ns) +1.7 (ns)	24 male (18-33 yr)	Linn et al. (1986)
0 157 196 235	0.00 0.08 0.10 0.12	CHEM, UV	1 & 2 hr of 6.6 hr study	CE (40)	-1.5 (1 hr) -1.0 (2 hr) -0.4 (ns) -1.1 (ns) -1.3 (ns) -1.3 (ns) -0.5 (ns) -2.7 (ns)	21 male (18-33 yr)	Horstman et al. (1988)
0 196	0.00 0.10	UV, UV	2 hr	IE (68)	+1.5 +1.1 (nd) range: +10 to -4	20 male (25.3 ± 4.1 yr)	Kulle et al. (1985)
0 196	0.00 0.10	CHEM, NBKI	2 hr	IE (67)	-0.3 -2.6 (ns)	10 male (18-28 yr)	Folinsbee et al. (1978)
0 216	0.00 0.11[f]	UV, UV	1 hr	CE (22)	-2.7 -2.9 (ns)	33 male 33 female (8-11 yr)	Avol et al. (1987)
0 235	0.00 0.12	CHEM, UV	2 hr	IE (68)	-1.0 -4.5 (p = 0.016)[g] range: +7 to -16	22 male (22.3 ± 3.1 yr)	McDonnell et al. (1983)
0 235	0.00 0.12	CHEM, UV	2 hr	IE (39)	-0.5 -3.4 (p = 0.03) range: +5 to -22	23 male (8-11 yr)	McDonnell et al. (1985)
0 235	0.00 0.12	CHEM, UV	1 & 2 hr of 6.6 hr study	CE (40)	-0.2 (1 hr) -1.2 (2 hr) -2.6 (ns) -3.8 (ns)	10 male (18-33 yr)	Folinsbee et al. (1988)
0 235	0.00 0.12	UV	1 hr (mouthpiece)	R	-1.1 0.0 (ns)	4 male 6 female (13-18 yr)	Koenig et al. (1985)
0 235 353	0.00 0.12 0.18	UV	40 min (mouthpiece)	IE (33) 30 min R + 10 min exercise	-1.0 +1.7 (ns) -0.3 (ns)	5 male 7 females (11-19 yr)	Koenig et al. (1987)

(continued on the following page)

Source: Supplement to Air Quality Criteria for O_3.

Table VII-1 (cont'd) KEY HUMAN STUDIES NEAR THE CURRENT 1-HR NAAQS FOR OZONE

Ozone Concentration µg/m³	Ozone Concentration ppm	Measurement[a,b] Method	Exposure Duration	Activity[c] Level (V_E)	Percent Change in FEV_1[d]	Number, Sex, and Age of Subjects	Reference[e]
0 235	0.00 0.12	UV	1 hr (mouthpiece)	IE (33)	-2.4 -0.6 (ns)	5 male 8 females (12-17 yr)	Koenig et al. (1988)
0 235	0.00 0.12	UV, UV	1 hr (mouthpiece)	CE (86)	+2.4 -1.8(ns)	10 male (19-29 yr)	Schelegle and Adams (1986)
0 235	0.00 0.12	UV, UV	1 hr	CE (89)	+4.1 -5.6 (p <0.02) range; +10 to -29	15 male 2 female (24 ± 3 yr)	Gong et al. (1986)
0 235 274	0.00 0.12 0.14	UV, UV	2 hr	IE (68)	+1.0 +2.8 (ns) +1.6 (ns)	24 male (18-33 yr)	Linn et al. (1986)
0 274	0.00 0.14[f]	UV, UV	1 hr	CE (31)	-0.5 -4.2 (p <0.01)	46 male 13 female (12-15 yr)	Avol et al. (1985)
0 294	0.00 0.15[f]	UV, UV	1 hr	CE(53)	+0.6 -5.3 (p <0.05)	42 male 8 female (26.4 ± 6.9 yr)	Avol et al. (1984)
0 294	0.00 0.15	UV, UV	2 hr	IE (68)	+1.5 -0.5 (nd) range: +3 to -9	20 male (25.3 ± 4.1 yr)	Kulle et al. (1985)
0 294	0.00 0.15	UV, UV	1 hr (mouthpiece)	CE (55)	+0.6 -4.5 (ns) range: +3.5 to -30.6	10 female (22.9 ± 2.5 yr)	Gibbons and Adams (1984)
0 314	0.00 0.16	UV, UV	1 hr	CE (57)	+0.6 -6.1 (p <0.05)	42 male 8 female (26.4 ± 6.9 yr)	Avol et al. (1984)
0 314	0.00 0.16	UV, UV	2 hr	IE (68)	+1.0 -2.3 (p <0.05) range: +8.9 to -35.8	24 male (18-33 yr)	Linn et al. (1986)
0 314	0.00 0.16[f]	UV, NBKI	1 hr	CE (38)	-0.1 -0.8 (ns)	27 male 21 female (28 ± 8 yr)	Linn et al. (1983); Avol et al. (1983)
0 333	0.00 0.17[f]	UV, NBKI	1 hr	CE (42)	-0.4 -3.4 (p <0.006)	45 male 15 female (30 ± 11 yr)	Linn et al. (1983); Avol et al. (1983)
0 333	0.00 0.17[f]	UV, NBKI	2 hr	IE (2XR)	+0.6 -2.1 (p <0.05)	14 male 20 female (29 ± 8 yr)	Linn et al. (1980, 1983)

(continued on the following page)

Table VII-1 (cont'd) KEY HUMAN STUDIES NEAR THE CURRENT 1-HR NAAQS FOR OZONE

Ozone Concentration µg/m³	Ozone Concentration ppm	Measurement[a,b] Method	Exposure Duration	Activity[c] Level (V_E)	Percent Change in FEV_1[d]	Number, Sex, and Age of Subjects	Reference[e]
0 353	0.00 0.18	CHEM, UV	2 hr	IE (65)	-1.0 -6.2 (p = 0.008) range: 0 to -23	20 male (23.3 ± 2.8 yr)	McDonnell et al. (1983)
0 353	0.00 0.18	UV, UV	1 hr (mouthpiece)	CE (86)	+2.4 -5.8 (p <0.05)	10 male (19-29 yr)	Schelegle and Adams (1986)
0 392	0.0 0.2	UV, UV	2 hr	IE (68)	+1.5 -3.1 (nd) range: +3 to -16	20 male (25.3 ± 4.1 yr)	Kulle et al. (1985)
0 392	0.0 0.2	UV, UV	2 hr	IE (30 for male, 18 for female subjects	+0.3 -3.1 (ns) range: +6.0 to -16.6	8 male 13 female (18-31 yr)	Gliner et al. (1983)
0 392	0.0 0.2	UV, UV	1 hr	CE (90)	+4.1 -21.6 (p <0.001) range: +10 to -46	15 male 2 female (24 ± 3 yr)	Gong et al. (1986)
0 392	0.0 0.2	UV, UV	1 hr (mouthpiece)	CE (77.5)	+1.7 -6.0 (p <0.05)	10 male (24 ± 4 yr)	Adams and Schelegle (1983)
0 392	0.0 0.2	UV, UV	75 min (mouthpiece)	CE (61.8)	-4.8 (ns)	8 male (22-46 yr)	Adams et al. (1981)
0 392	0.0 0.2	UV, UV	1 hr (mouthpiece)	CE (46)	-1.5 -8.0 (ns)	6 female (22-29 yr)	Lauritzen and Adams (1985)
0 412	0.00 0.21	UV, UV	1 hr	CE (89)	+1.9 -14.8 (p <0.05)	6 male 1 female (18-27 yr)	Folinsbee et al. (1984)
0 470	0.00 0.24	CHEM, UV	2 hr	IE (65)	-1.0 -14.5 (p <0.005) range: -1 to -36	20 male (22.9 ± 2.9 yr)	McDonnell et al. (1983)
0 470	0.00 0.24	UV, UV	1 hr	CE (60)	+0.6 -19.1 (p <0.05)	42 male 8 female (26.4 ± 6.9 yr)	Avol et al. (1984)
0 470	0.00 0.24	UV, UV	1 hr (mouthpiece)	CE (86)	+2.4 -10.5 (p <0.05)	10 male (19-29 yr)	Schelegle and Adams (1986)
0 490	0.00 0.25	UV, UV	2 hr	IE (68)	+1.5 -6.4 (nd) range: +1 to -36	20 male (25.3 ± 4.1 yr)	Kulle et al. (1985)

[a]Measurement method: CHEM = gas phase chemiluminescence, UV = ultraviolet photometry.

[b]Calibration method: NBKI = neutral buffered potassium iodide; UV = ultraviolet photometry.

[c]Minute ventilation reported in L/min or as a multiple of resting ventilation: IE = intermittent exercise; CE = continuous exercise.

[d]Pre to post difference (percent) in the group mean; statistical significance based on difference between O_3 and filtered air (0.0 ppm O_3) exposures: ns = not significant; nd = not determined.

[e]See U.S. Environmental Protection Agency (1986).

[f]Measured in ambient air (mobile laboratory).

[g]"Suggested" significance based on Bonferroni inequality correction (p <0.006).

the O_3 concentration inhaled during exposure. Group mean data pooled from numerous controlled human exposure and field studies and summarized in the CD (p. 12-80) indicate that, on average, pulmonary function decrements occur at:

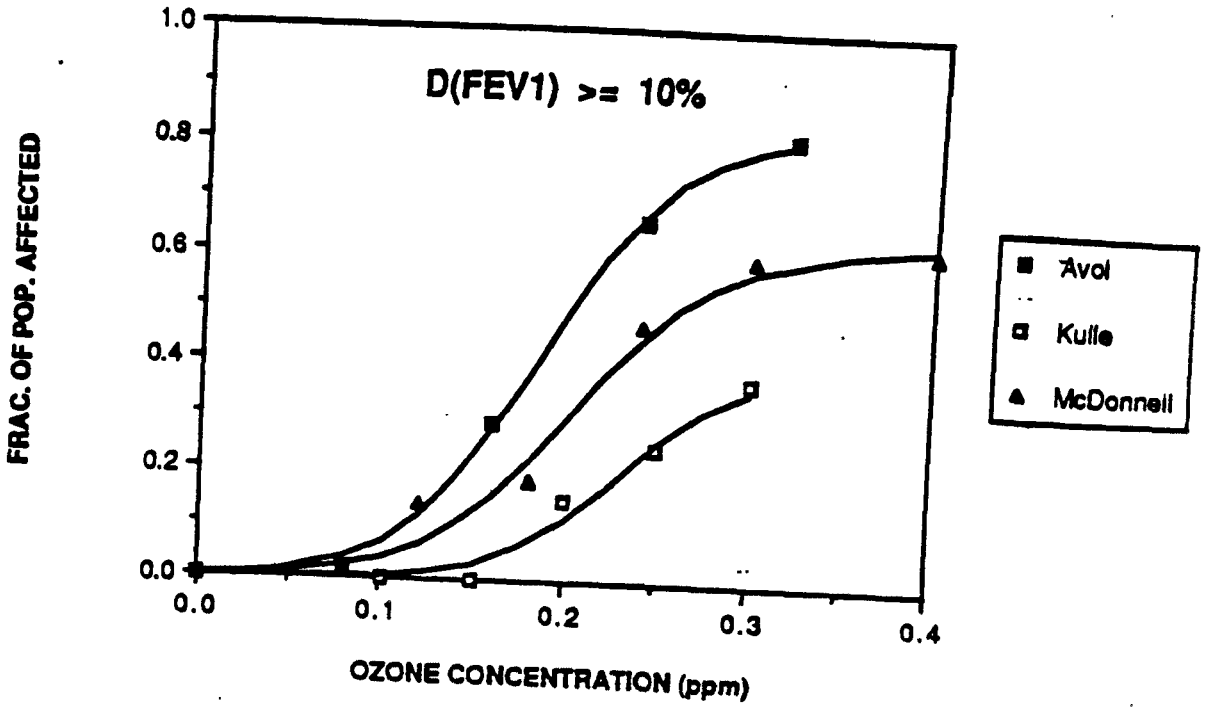
(a) $\geq$ 0.5 ppm O_3 when at rest ($\dot{V}_e$ = 5-10 L/min; e.g., sitting);

(b) $\geq$ 0.37 ppm O_3 with light exercise ($\dot{V}_e$ = 10 to 23 L/min; e.g., slow walking); (c) $\geq$ 0.30 ppm O_3 with moderate exercise ($\dot{V}_e$ = 23 to 43 L/min; e.g., brisk walking);

(d) $\geq$ 0.24 ppm O_3 with heavy exercise ($\dot{V}_e$ = 44 to 63 L/min; e.g., easy running);

(e) $\geq$ 0.18 ppm O_3 with very heavy exercise ($\dot{V}_e \geq$64 L/min; e.g., competitive running).

Although the group mean changes in lung function in the above studies are small, considerable intersubject variability in the magnitude of individual pulmonary response exists, and some subjects experienced responses which were quite large (See Section VI.C.2). Controlled exposures to 0.12 ppm O_3 during very heavy exercise have resulted in individual pulmonary function decrements up to 16% for adults (McDonnell et al., 1983) and up to 22% for children (McDonnell et al., 1985b). Individual subject data from the Avol et al., 1984, McDonnell et al., 1983, and Kulle et al., 1985 studies have been used to estimate the fraction of population which experiences $\geq$ 10% and $\geq$ 20% FEV_1 decrements due to O_3 exposure (Figure VII-2). The exposure-response relationships represented in Figure VII-2 suggest that between 2 and 20% of the heavily exercising population might exhibit $\geq$ 10% decreases in FEV_1 when exposed to O_3 levels of approximately 0.12 ppm. It is estimated that between 0 and 5% of the heavily exercising population might experience FEV_1 decrements of $\geq$ 20% when O_3 exposures are 0.12 ppm. These estimates must be considered approximate considering: (1) the amount of variability of response between the three studies analyzed, (2) the small number of subjects tested in each study



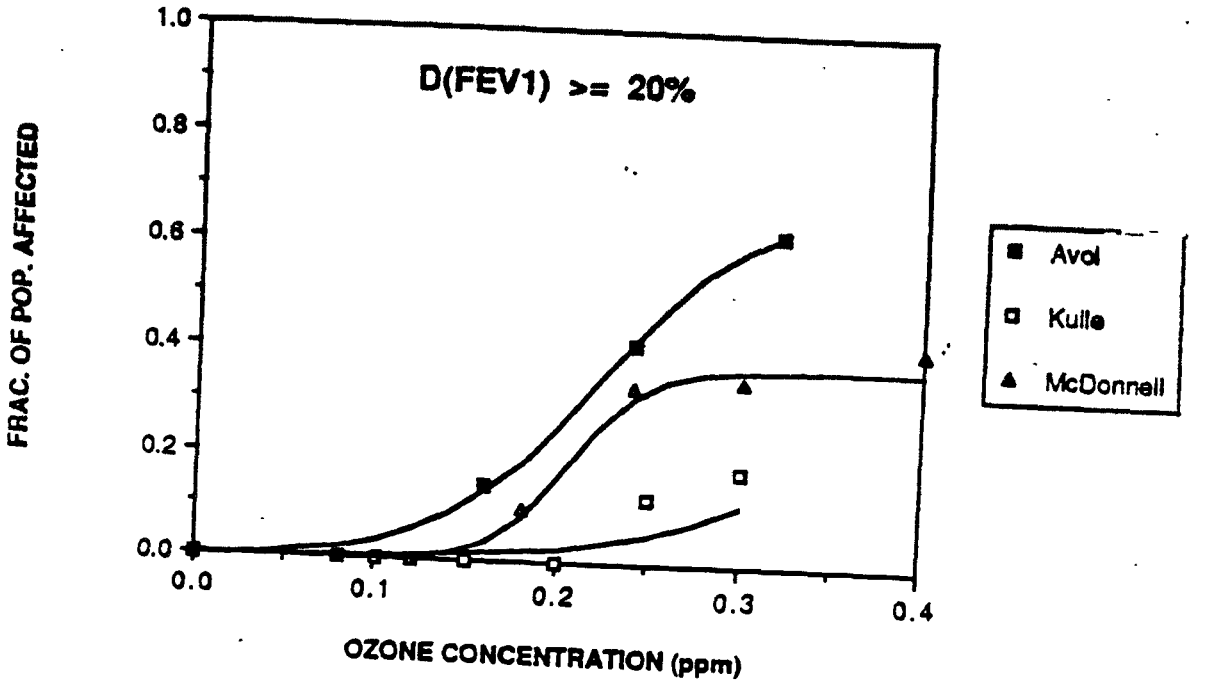


FIGURE VII-2. FRACTION OF HEAVILY EXERCISING POPULATION EXPERIENCING ≥10% AND ≥20% CHANGE IN FEV_1 DUE TO VARIOUS OZONE LEVELS. EXPOSURES WERE FOR 1-HOUR CONTINUOUS EXERCISE FOR AVOL ET AL. DATA AND FOR 2 HOURS INTERMITTENT EXERCISE FOR MCDONNELL ET AL. AND KULLE ET AL DATA.

(i.e., between 20-50 subjects at any given exposure level), (3) the fact that the subjects in these three studies were not derived from population based sampling, and (4) the use of a fitted function based on empirical, rather than biological grounds. Further discussion of this analysis can be found in Section VII.B. of the staff paper and in Hayes et al. (1987).

Recent controlled human exposure, epidemiology, and animal toxicology studies have increased concern about enhanced respiratory effects associated with multi-hour (6-8 hour) exposures to O_3. Clinical research conducted since closure on the CD has provided data which indicate lung function decrements, symptoms, and even inflammation during prolonged O_3 exposures below the current O_3 NAAQS level of 0.12 ppm. In addition, camp and field studies offer evidence which corresponds well to clinical data on lung function decrements and symptoms. New animal toxicology research is helping to make a stronger case for needing to consider time as an important variable along with concentration and $\dot{V}_e$ in assessing health effects of O_3.

Folinsbee et al. (1989) published data indicating that ten healthy non-smoking males (18-33 years) exposed to 0.12 ppm O_3 during intermittent moderate exercise. Exposures lasted 6.6 hours and consisted of six 50-minute exercise $\dot{V}_e$ = 40 L/min) periods interspersed with 10 minute rest breaks and a 35 minute lunch break. Subjects experienced progressively decreasing FEV_1 over the exposure period. At the end of O_3 exposure, group mean FEV_1 had decreased by 13.0%, FVC by 8.3%, and FEV_{25-75} by 17.4% when compared to clean air exposures. One individual experienced a 47.6% decrease in FEV_1. There were also progressive increases in symptom ratings of cough and pain on deep inspiration for subjects exposed to 0.12 ppm O_3 as well as a marked increase in methacholine airway responsiveness.

In a continuation of the Folinsbee et al. (1989) work, Horstman et al. (1988,1989) exposed each of 22 nonsmoking, healthy, male subjects to 0.0, 0.08, 0.10, and 0.12 ppm O_3 on separate days. Exposure protocol was similar to that described

above for the Folinsbee et al. (1989) study. Substantial pulmonary function decrements, respiratory symptoms, and increases in nonspecific airway reactivity were reported for all three O_3 exposures when compared to filtered air exposures. Group mean decreases in FEV_1 were 7.0% at 0.08 ppm O_3, 7.0% at 0.10 ppm O_3, and 12.3% at 0.12 ppm O_3, all of which were statistically significant ($p < .01$). Average ratings of pain on deep inspiration were low but increased significantly ($p < .05$) following exposure to all three O_3 levels compared to filtered air. Ratios ($p < .005$) of PD_{100} observed for filtered air compared to O_3 exposure were 1.56 at 0.08 ppm O_3, 1.89 at 0.10 ppm O_3, and 1.89 at 0.12 ppm O_3. These results led Horstman et al. (1988, 1989) to conclude that clinically meaningful pulmonary responses can be induced by O_3 exposures and exercise levels which simulate a 6- to 8-hour period of moderate to heavy work or play outdoors during the O_3 season.

Further support of the relationship between acute O_3 exposure and pulmonary function decrements is provided by several epidemiological studies of children and young adults (Lippmann et al., 1983; Lebowitz et al., 1982a, 1983; Lebowitz, 1984; Bock et al., 1985; Lioy et al., 1985; Spektor et al., 1988a,b). These studies report decreased peak flow or increased airway resistance for acute exposures to ambient O_3 concentrations during the study period. Lioy et al. (1985) reported that a persistent decrement in lung function of children lasted as much as a week after the end of a smog period of four days during which peak 1-hr O_3 levels were in the range of 0.135 to 0.186 ppm. The persistent decrements suggested by Lioy et al. (1985) (i.e., altered epithelial permeability and changes in airway secretion) represent a potentially more important response than the more transient effects found in controlled exposure studies; however, they recognize that elevated concentrations of inhalable particulate matter associated with a large scale photochemical smog episode also are associated with effects reported. Spektor et al. (1988a) have followed up the work of Lioy et al. (1985)

with a summer camp study of children exposed to ambient O_3 in an area which did not exceed 0.12 ppm O_3 during the study. Multiple regression analyses indicate that the most explanatory environmental variables for daily change in lung function were: 1) previous hour O_3 levels, 2) cumulative daily O_3 exposure between 9 a.m. and the time of measurement of lung function, 3) ambient temperature, and 4) humidity. Spektor et al. (1988a) concludes that the 1 hour O_3 levels have the strongest influence on lung function and that regression slopes for FVC, FEV_1, PEFR, and MMEF predict average decrements of 4.9%, 7.7%, 17% and 11%, respectively at 0.12 ppm O_3. Upper decile decrements are predicted to be 14%, 19%, 42%, and 33% for FVC, FEV_1, PEFR, and MMEF, respectively at 0.12 ppm O_3. Lioy and Dyba (1988) have proposed recently that the PEFR decrements reported in Lioy et al. (1985) resulted from total O_3 dose rather than persistence of the effect from one day to the next.

Epidemiology studies comparing incidence of chronic lung disease in communities have thus far been relatively unsuccessful due to the lack of differences in pollutant levels, inadequate control of covariables, and insufficient individual exposure data (CD, p. 11-54). While it has been concluded in the CD (p. 11-53) that not one of these epidemiological studies provides definitive quantitative data by itself due to methodological problems and confounding variables, the aggregation of studies provides reasonably good qualitative evidence of association between ambient photochemical oxidant exposure and acute pulmonary effects. The association is strengthened by the consistency of these epidemiological results with the findings of McDonnell et al. (1985b) and Avol et al. (1985a,b) who reported small decrements in pulmonary function for exercising children exposed to 0.12 ppm O_3 in purified air and adolescents exposed to 0.144 ppm O_3 in ambient air, respectively.

In conclusion, the weight of currently available evidence indicates that healthy, heavily exercising subjects can experience pulmonary function decrements during controlled

exposures of ≥ 0.12 ppm O_3 for 2 hours. Although level of exercise and individual responsiveness play a major role in determining the extent of pulmonary function loss, staff concludes that 0.12 ppm O_3 is the lowest observed effects level (LOEL) for this effect in controlled exposure studies of 2-hour exposures. Effects have been reported as low as 0.08 ppm when exposures lasted for 6.6 hours. Field and epidemiology data provide added evidence of measurable functional decrements below 0.12 pm, perhaps in part due to pollutant interactions.

The question of what degree of pulmonary function response should be considered adverse is addressed in Section VII.C.1.

2. Symptomatic Effects

Respiratory symptoms have been associated with group mean pulmonary function changes in adults acutely exposed in controlled exposures to O_3 and in ambient air containing O_3 as the predominant pollutant. Despite a close association observed between changes in group mean FEV_1 and group mean respiratory symptoms for O_3 exposures (CD, p. 12-17), Hayes et al. (1987b) report only a weak-to-moderate correlation between FEV_1 changes and symptoms severity when the analysis is conducted using individual data. It should be noted that symptoms reported are inherently more subjective than FEV_1 decrements measured.

In controlled O_3 exposures, some heavily exercising ($\dot{V}_e \geq 65$ L/min) adult subjects have experienced cough, shortness of breath, and pain on deep inspiration at 0.12 ppm O_3, although the group mean response was statistically significant for cough only (McDonnell et al., 1983). Above 0.12 ppm O_3, respiratory and non-respiratory symptoms which have been reported include throat dryness, chest tightness, substernal pain, cough, wheeze, pain on deep inspiration, shortness of breath, dyspnea, lassitude, malaise, headache, and nausea (DeLucia and Adams, 1977; Kagawa and Tsuru, 1979a,b,c; McDonnell et al., 1983; Adams and Schelegle, 1983; Avol et al., 1984; Gibbons and Adams, 1984; Folinsbee et al., 1984; Kulle et al., 1985). At 0.2 ppm O_3 and

higher, controlled exposure studies of exercising subjects have reported positive "symptom scores" with elimination of symptoms for most subjects within 24 hours post-exposure (CD, p. 12-19).

Data reported in three controlled human exposure studies (McDonnell et al., 1983; Kulle et al., 1985; Avol et al., 1984) have been analyzed to estimate the fraction of the heavily exercising population that might experience mild and moderate respiratory symptoms during exposure to various O_3 levels. Figures VII-3 and VII-4 provide the estimated exposure-response relationships. As O_3 exposure levels rise in these studies, the fraction of subjects experiencing respiratory symptoms generally increases. For example, it is estimated that for 0.12 ppm O_3 exposures, between 0 and 15% of the heavily exercising population might experience moderate cough or chest discomfort. As the O_3 exposure level rises to 0.2 ppm the estimated fraction of the population that might experience moderate cough or chest discomfort increases to a range of about 3 to 40%. As with the lung function analysis discussed in the previous section, these estimates must be considered approximate considering (1) the amount of variability of response between the three studies analyzed, (2) the small number of subjects tested in each study (i.e., between 20-50 subjects at any given exposure level), (3) the fact that the subjects in the three studies were not derived from population based sampling, and (4) the use of a fitted function based on empirical, rather than biological grounds. Section VII-B of this staff paper and Hayes et al. (1987a,b) present a more detailed discussion of the respiratory symptom risk analysis.

Comparisons of symptoms reported in controlled O_3 exposure studies have been made with those reported in field studies. One study, which provided a direct comparison of symptoms in exercising ($\dot{V}_e$ = 57 L/min) adults caused by exposure to either purified air containing 0.16 ppm O_3 or oxidant-polluted ambient air which contained 0.15 ppm O_3, showed no statistically

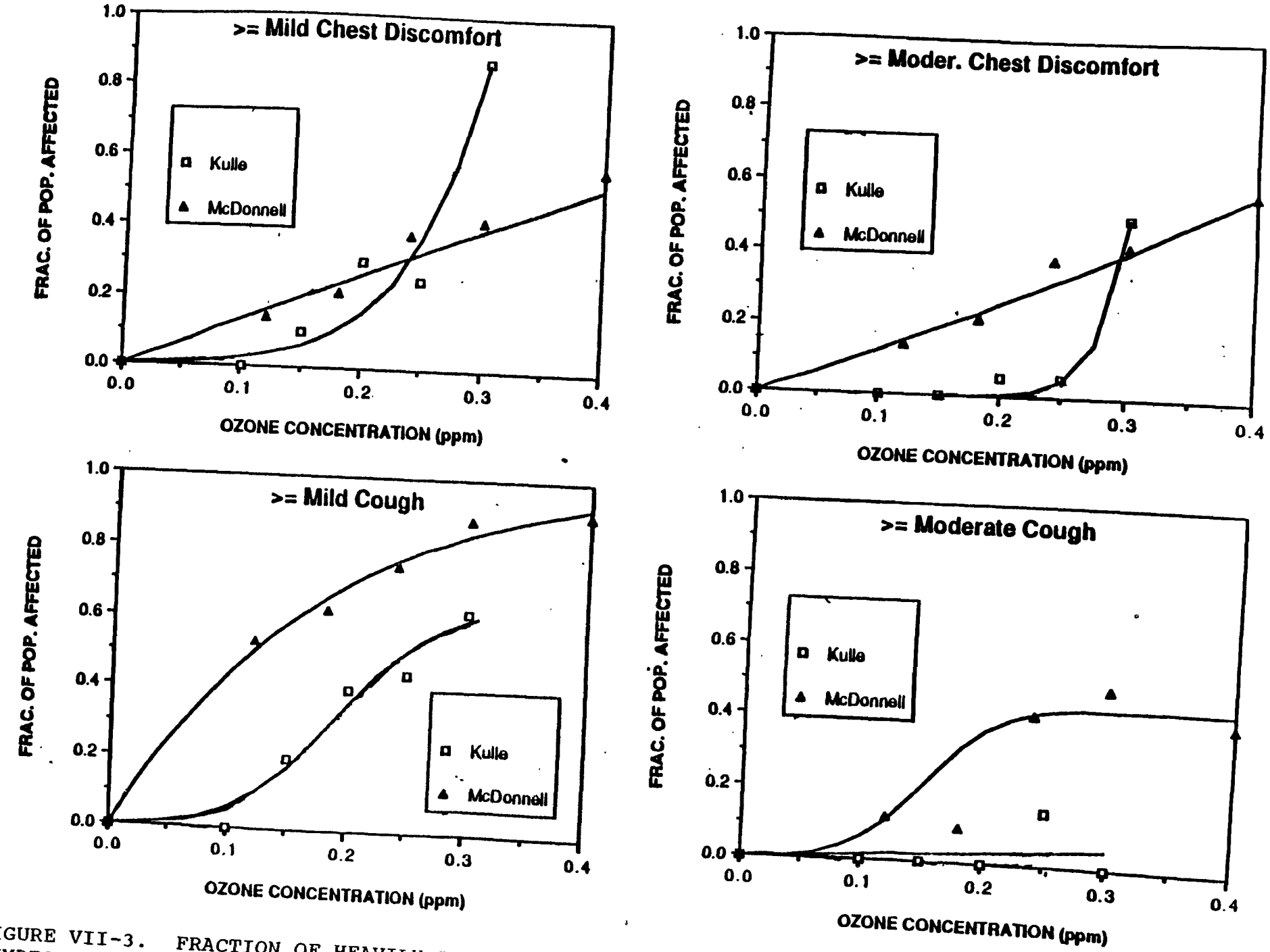


FIGURE VII-3. FRACTION OF HEAVILY EXERCISING POPULATION EXPERIENCING MILD AND MODERATE SYMPTOMS DUE TO VARIOUS OZONE LEVELS. EXPOSURE WERE FOR 2 HOURS OF INTERMITTENT EXERCISE.

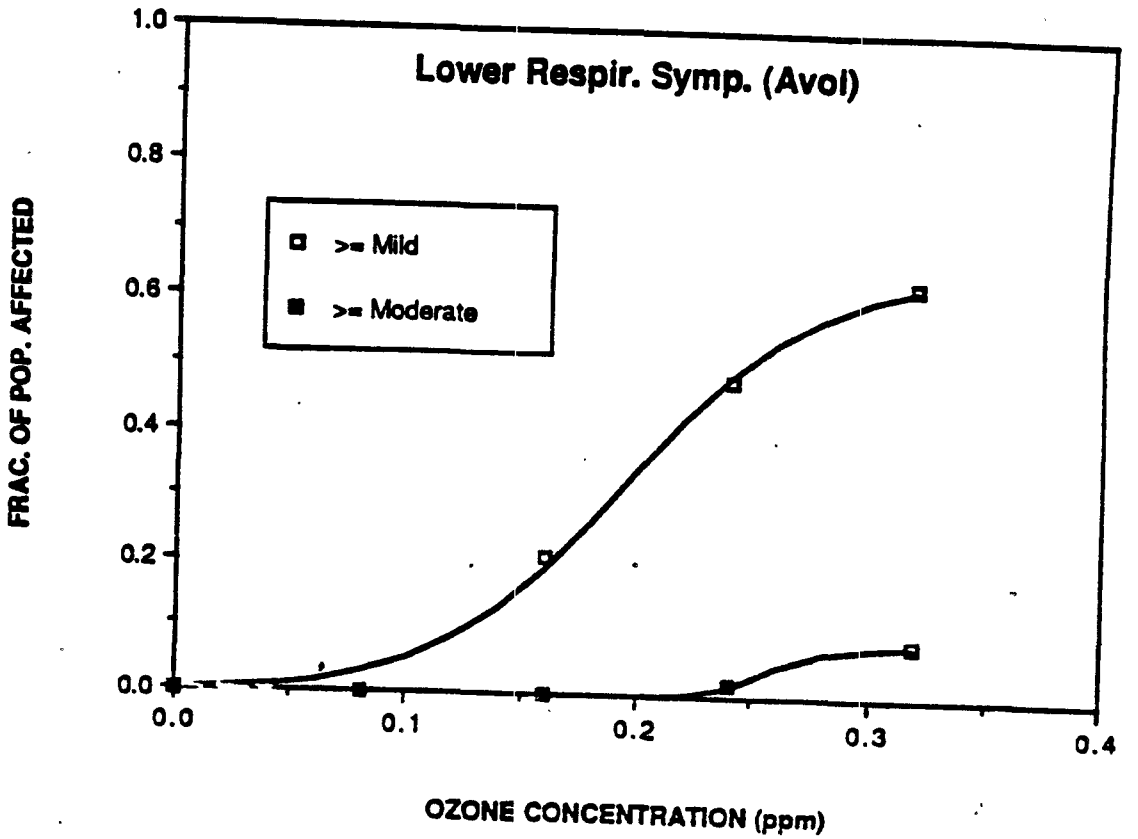


FIGURE VII-4. FRACTION OF HEAVILY EXERCISING POPULATION EXPERIENCING LOWER RESPIRATORY SYMPTOMS DUE TO VARIOUS OZONE LEVELS. EXPOSURES WERE FOR 1 HOUR WITH CONTINUOUS EXERCISE ($\dot{V}_E$=57 L/min) BASED ON AVOL ET AL. (1984).

significant differences, suggesting that increased symptoms associated with lung function impairment were caused by O_3 alone (Avol et al., 1984). Several epidemiology studies have provided evidence of qualitative associations between ambient oxidant levels > 0.10 ppm and symptoms in children and young adults such as throat irritation, chest discomfort, cough, and headache (Hammer et al., 1974; Makino and Mizoguchi, 1975). Thus, it can be concluded that most symptoms reported in individuals exposed to O_3 in purified air are similar, but not identical to, those found for ambient air exposures.

An exception is eye irritation, a common symptom associated with exposure to photochemical oxidants, which has not been reported for controlled exposures to O_3 alone. This appears to hold even at O_3 concentrations much higher than would be found in the ambient air. It is widely accepted that other oxidants such as aldehydes and peroxyacetyl nitrate (PAN) are primarily responsible for eye irritation and are generally found in atmospheres containing higher ambient O_3 levels (Altshuller, 1977; National Research Council, 1977; U.S. Environmental Protection Agency, 1978; Okawada et al., 1979).

Pulmonary function decrements have been reported in studies which did not report increases in symptoms. Children (age 8-11) intermittently exercising ($\dot{V}_e$ = 39 L/min) for 2.5 hours at 0.12 ppm O_3 showed small, but statistically significant decreases in FEV_1 but showed no changes in frequency or severity of cough compared to control (McDonnell et al., 1985a,b). Similarly, adolescents (age 12-15) continuously exercising ($\dot{V}_e$ = 31-33 L/min) during exposure to 0.144 ppm mean O_3 in ambient air showed no changes in symptoms despite statistically significant decrements in group mean FEV_1 (4%) which persisted at least one hour during resting post-exposure (Avol et al., 1985a,b). Because symptoms can be viewed as an early warning of related lung function impairment by O_3, the lack of symptoms in children and adolescents during exposures which induce functional decrements may be of concern. It is possible that children may

be at higher risk since, with no warning, they may not exhibit avoidance behavior.

Symptoms when combined with objective measures of lung function are considered useful adjuncts in assessing health effects caused by O_3 and photochemical oxidants. Group mean symptoms have been shown to be closely associated with the time-course and magnitude of group mean pulmonary function changes associated with O_3 exposures; however, only weak-to moderate correlations exist when data are analyzed on an individual basis. To the extent symptoms associated with exposure to O_3 and other photochemical oxidant exposures are associated with discomfort, interfere with normal activity, and provide subjective evidence of functional impairment, the staff in concordance with comments made by CASAC (CASAC, 1986) recommends that they should be considered adverse health effects and as such should be included in identifying a lowest observed effects level (LOEL).

3. Exercise Performance

A limited data base provides suggestive evidence of decrements in exercise performance associated with O_3 exposure. A detailed discussion of those data can be found in the CD (pp. 10-60 to p. 10-65) and is summarized in Table 10-6 (CD, p. 10-64).

Early epidemiological evidence on high school students suggested that the percentage of track team members failing to improve performance increased with increasing oxidant concentrations the hour before a race (Wayne et al. 1967). The authors concluded that the effects may have been related to increased R_{aw} or to associated discomfort which may have limited motivation to run at maximal levels. Controlled exposure studies of heavily exercising competitive runners have demonstrated decreased max $\dot{V}_e$ at 0.3 ppm O_3 (Savin and Adams, 1979) and decreased FVC, FEF, and FEF at 0.20 ppm O_3 (Adams and Schelegle, 1983). At 0.21 ppm O_3, Folinsbee et al., (1984) reported decreases in FVC, FEV, FEF, IC, and MVV at 75% max VO_2 as well as

symptoms in 7 distance cyclists exercising heavily ($\dot{V}_e$ = 81 L/min).

With regard to exercise performance studies, reductions in group mean FEV_1 (-5.6%) and mild symptoms (cough, chest soreness, shortness of breath) are reported in 17 endurance cyclists exposed at 31°C to 0.12 ppm O_3 for 1 hour during very heavy ($\dot{V}_e$ = 88 L/min) continuous exercise (Gong et al., 1986). Although no impairment of maximal performance is noted at 0.12 ppm, when Gong et al. (1986) exposed the same subjects to 0.20 ppm O_3 under the same exercise conditions, he reports that both group mean FEV_1 decrements (-21.6%) and symptoms (prominent cough and nausea) are intensified and result in impairment of maximal performance. Similarly, Schelegle and Adams (1986) report that 10 highly trained endurance athletes exposed to 0.12, 0.18, and 0.24 ppm O_3 while performing a 1-hour competitive simulation ($\dot{V}_e$ = 86 L/min) on a bicycle ergometer show a significant ($p < 0.05$) increase in the inability of subjects to complete the simulation. Even though one subject was not able to complete the simulation at any of the O_3 levels tested, five were unable to complete at 0.18 ppm O_3 and seven did not finish at 0.24 ppm O_3. Only at 0.18 and 0.24 ppm O_3 are statistically significant changes reported in FEV_1, FVC, and subjective symptoms. Schelegle and Adams (1986) report no significant changes in lung function or symptoms at 0.12 ppm O_3. Spektor et al. (1988b) offer substantiation of the effects of O_3 on respiratory function in populations engaging in continuous exercise for short periods.

Although subjective statements by individuals engaged in sports indicate possible voluntary curtailing of activities during high-oxidant episodes, increased temperature and relative humidity may be involved in provoking the symptoms and lung function decrements observed in the studies above. Controlled studies of O_3 exposure have, however, demonstrated lung function impairment and subjective symptoms which cause individuals to reduce work load and performance at 0.2 ppm O_3 and above (Adams and Schelegle, 1983; Folinsbee et al., 1984; Avol et al., 1984).

Because O_3 may be implicated at least in part in reducing exercise performance during periods of high oxidants and some individuals experience this effect at levels near the O_3 NAAQS, the staff recommends that this effect of O_3 be viewed as a matter of potential public health concern and be considered in developing a margin of safety.

4. Bronchial Reactivity and Inflammation

Bronchial reactivity is reportedly associated with exposure to O_3 (CD, p. 10-30). Using airway responsiveness to the drugs acetylcholine (ACh), methacholine, or histamine as a measure of non-specific airway sensitivity, researchers have observed increased bronchial reactivity in both healthy and asthmatic subjects exposed to O_3 levels in the range of 0.32 to 1.0 ppm. It should be noted that most of these studies were conducted on resting or only moderately exercising individuals.

In both atopic and non-atopic subjects exposed, using a noseclip, to 0.6 ppm O_3 during intermittent exercise, bronchial response to histamine and methacholine was greater when subjects were exposed to O_3 than to filtered air (Holtzman et al., 1979). Symptoms of bronchial irritation in the Holtzman et al. (1979) study were not detectable by the next day, which contrasts with results of the Golden et al. (1979) study of healthy resting subjects exposed to 0.6 ppm O_3 reporting enhanced response to histamine challenge for as much as 1 to 3 weeks after exposure.

Other studies of bronchial reactivity show statistically significant enhancement when O_3 exposures are as low as 0.32 ppm but not at 0.20 ppm. Significant increases in bronchial reactivity were reported with ACh challenge in healthy adult subjects exposed to either 0.32 ppm or 1.0 ppm O_3 (König et al., 1980). Healthy subjects showed no alterations of bronchomotor response to histamine aerosol when exposed to 0.2 ppm O_3 for 2-hr, although exposure to 0.4 ppm O_3 did enhance bronchial responsiveness to inhaled histamine (Dimeo et al., 1981). Kulle et al. (1982b) reported significantly enhanced bronchial

reactivity to methacholine of healthy subjects exposed to 0.4 ppm O_3 as compared to filtered air.

McDonnell et al. (1987) report increased reactivity to histamine following exposure of healthy subjects to 0.18 ppm O_3 during intermittent very heavy exercise (64 L/min) for 2 hours. Increased airway reactivity to methacholine following 2 hour exposures of subjects to 0.4 and 0.6 ppm O_3 during intermittent moderate exercise is reported by Seltzer et al. (1986) to be associated with neutrophil influx. Using bronchoalveolar lavage (BAL), Koren et al. (1989a,b,c) observe increased inflammation (8.2 fold increase in polymorphonuclear leukocytes, PMN's) at 18 hours post exposure in healthy adult males exposed for 2 hours to 0.4 ppm O_3 during intermittent exercise. In addition to confirmation of the Seltzer et al. (1986) findings, Koren et al. (1989a,b,c) provide evidence of stimulation of fibrogenic processes and further suggest that the inflammatory process initiated by O_3 exposure is promptly initiated (Seltzer, et al., 1986) and persists for at least 18 hours. Although the time course of the inflammatory response has not been elucidated, the Koren et al. (1989a,b,c) research demonstrates that cells and enzymes capable of causing damage to pulmonary tissues are increased as a result of O_3 exposure. Furthermore, proteins which play a role in fibrotic and fibrinolytic processes are elevated by exposure to O_3. Studies by Kehrl et al. (1987, 1989) confirm that clearance of technetium labeled DTPA from airway and alveoli into the bloodstream is accelerated after O_3 exposure and along with Koren et al. (1989a,b,c) suggest that this accelerated change is due, in part, to increased epithelial permeability in the lung. These permeability alterations are likely to be associated with acute inflammation and may allow inhaled antigens and other substances to more easily reach the submucosa.

In an extension of a 2-hour exposure study, Koren et al. (1989a,b,c) exposed ten healthy non-smoking males (18-35 yrs) to 0.1 ppm O_3 for 6.6 hours while subjects were engaged in a moderate to heavy ($\dot{V}_e$ = 40 L/min) intermittent exercise protocol

similar to Folinsbee et al. (1989). Results indicated suggestive evidence of an inflammatory response in the lower airways as evidenced by a 4.8-fold increase in percent of polymorphonuclear leukocytes (PMN's) in the bronchoalveolar lavage fluid ($p = 0.034$). Importance of these preliminary results lies in the fact that they represent evidence of inflammation in humans and potential for damage in lower airways from O_3 exposures which may occur often in ambient air.

In conclusion, there is some evidence to suggest that bronchial reactivity and inflammation occur at ambient concentrations of O_3. This is particularly true for subjects exposed to O_3 during exercise. Because the data base showing effects at low concentrations of O_3 is relatively new and small, staff recommends that this information be used to develop a margin of safety.

5. Aggravation of Existing Respiratory Disease

Some epidemiological studies suggest an association between photochemical smog and aggravation of existing respiratory disease. No clear evidence is available, however, from controlled exposure or field studies to suggest that individuals with asthma, chronic bronchitis, or emphysema have greater lung function impairment caused by O_3 or other photochemical oxidants than healthy persons. However, individuals with preexisting respiratory disease are considered to be especially "at risk" to O_3 exposure due to their already compromised respiratory systems and concern that increased symptoms or pulmonary function decrements may interfere with normal function (CD, p. 12-73).

In controlled human exposure studies, statistically significant group mean decrements in pulmonary function were not reported for adult asthmatics exposed for 2 hours at rest (Silverman, 1979) or with intermittent light exercise (Linn et al., 1978) to O_3 concentrations of 0.25 ppm or lower. Similarly, no statistically significant group mean changes in pulmonary function or symptoms were found in adolescent asthmatics exposed

to 0.12 ppm O_3 for 1-hour at rest (Koenig et al., 1985). Subjects with chronic obstructive lung disease (COLD) performing light to moderate intermittent exercise show no statistically significant group mean decrements in pulmonary function during 1- and 2-hour exposures to $\leq$ 0.30 ppm O_3 (Linn et al., 1982, 1983a; Solic et al., 1982; Kehrl et al., 1983, 1985) and only small, statistically significant group mean decrements in FEV_1 are observed for 3-hour exposures of chronic bronchitics to 0.41 ppm O_3 (Kulle et al., 1984). While these controlled exposure results indicate that individuals with pre-existing respiratory disease may not be more sensitive to O_3 than healthy subjects, experimental design considerations in these studies suggest that the issues of sensitivity and aggravation of pre-existing respiratory disease remain unresolved.

Several new studies compare effects of O_3 exposure on asthmatics with effects on healthy persons. Although no differences in O_3-induced symptoms are reported between 9 normals and 9 asthmatics exposed to 0.4 ppm O_3, Kreit et al. (1988) do report a more negative change in FEV_1, FEV_1/FVC, and FEF_{25-75} for asthmatics than for normals. SR_{aw} is not significantly increased in normals but is in asthmatics, though it should be noted that significant increase in SR_{aw} occurs in asthmatics even for air exposure. Koenig et al. (1987) compare the effects of 0.12 and 0.18 ppm O_3 on 10 asthmatics and 10 nonasthmatics exposed for 30 min. at rest followed by 10 min. exercise (32.5 L/min). No significant effects are found at 0.12 ppm O_3 while both groups show increases in respiratory resistance (R_T) at 0.18 ppm O_3. Koenig et al. (1987) conclude that there are no significant differences in response to O_3 between asthmatics and nonasthmatics. In a similar study, Koenig et al. (1988) find that 12 healthy adolescents exposed to 0.12 ppm O_3, 0.30 ppm NO_2 or a combination during intermittent moderate exercise show no significant response, while 12 asthmatic adolescents showed marginal responses in pulmonary function for .12 ppm O_3 and 0.3 ppm NO_2 but not the combination. This leads the authors to

conclude that asthmatic subjects may be slightly more responsive. However, replication of these observations will be required before this suggestion can be substantiated.

Epidemiological studies do not provide a clear concentration-response relationship between O_3 and aggravation of disease. One study (Whittemore and Korn, 1980) conducted in Los Angeles reported increased daily asthma attack rates on cool days and days with high oxidants and particulates when median daily maximum hourly oxidant levels were $\leq$ 0.15 ppm; questionable exposure assessment, lack of control for medication, pollen, respiratory infections and other pollutants limits the use of this study for developing quantitative dose-response relationship.

Despite several exposure and variable control limitations, the Houston Area Oxidants Study (Johnson et al., 1979; Javitz et al., 1983) concluded that for the study period in which the daily maximum hourly O_3 concentrations were below 0.21 ppm O_3 near the subjects' residence, 1) there was increased incidence of nasal and respiratory symptoms and increased frequency of medication use for asthmatics with increasing O_3 levels; 2) FEV_1 and FVC decreased with increasing O_3 and total oxidants; and 3) increased incidence of chest discomfort, eye irritation, and malaise occurred at high peroxyacetyl nitrate (PAN) concentrations. In a subsequent related study, (Stock et al., 1983; Holguin et al., 1985; Contant et al., 1985) increased probability of an asthma attack was associated with the occurrence of a previous attack and with exposure to increased O_3 concentrations and temperature when maximum 1-hour averages for O_3 were between 0.001 and 0.127 ppm; however, other pollutants such as SO_2 and particulates may have been involved.

In a series of studies conducted in a Tucson community, adults with asthma, allergies, or airway obstructive disease (AOD) were observed during an 11 month period in which 1-hour daily maximum O_3 concentrations were $\leq$ 0.12 ppm (Lebowitz et al., 1982, 1983; Lebowitz, 1984). After adjusting for covariables, O_3

and TSP levels were significantly associated with decreases in peak expiratory flow rate in adults with AOD, and the interaction between O_3 and temperature was significantly associated with alterations in peak flow and symptoms in asthmatics. While these results suggest an effect of O_3 in individuals with preexisting respiratory disease, interpretation is difficult due to the small sample size in relation to the number of covariates and the fact that individual exposure data were not available.

Bates and Sizto (1987) conducted a study in southern Ontario and reported that there is a consistent summer relationship between SO_4, O_3 and temperature and hospital respiratory admissions. The correlation between O_3 and hospital respiratory admissions was not affected by substituting the 8-hour average O_3 for mean hourly maximum O_3 levels. They further suggest the adverse health effects studied may not be associated with either O_3 or sulfates but rather with some species not yet monitored which they refer to as "acid summer haze." Bates and Sizto (1988) failed to confirm association between excess hospital admissions and O_3 for June 1983, the month of the highest O_3 of any month in their analyses. In a separate study conducted in Ontario, Raizenne and Spengler (1987) have preliminarily reported that lung function decrements in children attending summer camp are associated with maximum daily O_3 levels as well as temperature, fine particles, and sulfates. Over the course of a 41-day study, Raizenne and Spengler (1988) continuously measured O_3, NO_2, SO_2, and acid aerosols (H_2SO_4). In plotting individual 6-hour estimated dose for O_3 and H_2SO_4 (separately) versus percent change in PEFR, they found a negative trend in lung function as cumulative dose increases for both O_3 and H_2SO_4 although the slopes did not differ from zero ($p > .10$) when dosimetry was used.

None of these controlled human exposure or epidemiology studies alone demonstrates a clear relationship between exposure to O_3 and aggravation of preexisting respiratory disease. All of the epidemiology studies cited report effects which may be

related to inhalable particle exposure and most have inadequate characterization of exposure. However, the group of studies as a whole supports the contention that exposures to ambient levels of O_3 and other photochemical oxidants recently reported in many cities (See Appendix A) may increase the rate of asthma attacks for asthmatics. The staff believes that increased asthma attack rate is an adverse effect but recognizes the limited and uncertain data base relating this effect to O_3 exposure. Thus, in concordance with uncertainty expressed by CASAC (1986, 1987, 1988) it is recommended that data associating O_3 exposure with aggravation of existing respiratory disease be considered only in the development of a margin of safety for the primary standard unless further research provides more conclusive evidence.

6. Morphological Effects

Morphological effects of O_3 have been reported primarily in animal toxicology studies (See Table VII-2). For this reason it is important to consider the differences in dosimetry and sensitivity between humans and laboratory animals, which is discussed in Chapter 9 of the CD. The following discussion provides an indication of some of the structural changes which might occur in human lungs as a result of repeated and long-term exposures to O_3.

Despite the differences in lung structure between humans, dogs, monkeys, mice, rats, and guinea pigs, a characteristic lesion occurs at the junction of the conducting airways and the gaseous exchange tissues. In all species examined the effect is typically damage to the ciliated and Type 1 cells and hyperplasia of non-ciliated bronchiolar and Type 2 cells; an increase in inflammatory cells is also observed (CD, p. 12-58). These effects were reported after 7 days, 8 hr/day exposures to 0.2 ppm O_3 in monkeys (Dungworth et al., 1975; Castleman et al., 1977) and in rats exposed for 7 days, 8 and 24 hrs/day (Schwartz et al., 1976). Similar effects with different exposures were

TABLE VII-2. MORPHOLOGICAL EFFECTS OF OZONE IN EXPERIMENTAL ANIMALS

Effect	Ozone concentration, ppm	Exposure duration	Species	Reference
Damage to ciliated and centriacinar alveolar type 1 cells	0.2	8 hr/d x 7 d	Rat	Schwartz, et al., 1976
	0.2	8 hr/d x 7 d	Monkey	Castleman et al., 1977
	0.25	12 hr/d x 6 wk	Rat	Barry et al., 1983
	0.25	12 hr/d x 6 wk	Rat	Crapo et al., 1984
	0.5	2 hr	Rat	Stephens, et al., 1974a
	0.5	2 hr	Rat	Stephens, et al., 1974b
Hyperplasia of nonciliated bronchiolar and alveolar type 2 cells	0.2	8 hr/d x 7 d	Monkey	Castleman et al., 1977
	0.35	continuous for 1-8 d	Rat	Evans et al., 1976b
	0.5	8 hr/d x 90 d	Monkey	Eustis et al., 1981
	0.5	24 hr/d x 180 d	Rat	Moore and Schwartz, 1981
	0.8	continuous for 7 d	Rat	Lum et al., 1978
	0.8	24 hr/d x 10 d	Mouse	Ibraham et al., 1980
Inflammation	0.2	8 hr/d x 7 d	Rat	Schwartz et al., 1976
	0.2	8 hr/d x 7 d	Monkey	Castleman et al., 1977
	0.5	2 hr	Rat	Stephens et al., 1974a
	0.5	6 hr	Rat	Stephens et al., 1974b
	0.64	8 hr/d x 1 yr	Monkey	Fujinaka et al., 1985
	0.8	4 hr	Monkey	Castleman et al., 1980
Continued inflammation with remodeling of centriacinar airways and increased collagen	0.5	24 hr/d x 14 d	Rat	Last et al., 1979
	0.5	8 hr/d x 90 d	Rat	Boorman et al., 1980
	0.5	24 hr/d x 180 d	Rat	Moore and Schwartz, 1981
	0.64	8 hr/d x 361 d	Monkey	Last et al., 1984b
	1.0	8 hr/d x 18 mo	Dog	Freeman et al., 1973

reported in rats (0.26 ppm, 6 hr, endotracheal tube, Boatman et al., 1974), mice (0.5 ppm, 35 days, Zitnik et al., 1978), and guinea pigs (0.5 ppm, 6 months, Cavender et al., 1978). Inadequate data limit quantitative comparisons of human health effects with these reported for monkeys and rats, but a rough equivalency of responses has been observed under similar exposure conditions between species. Because all species tested show similar morphological responses to O_3 exposure, there is no reason to believe that humans exposed to O_3 would not respond similarly, although such effects may not necessarily occur at the same O_3 exposure concentrations or averaging times of exposure.

Changes in lung structure of monkeys and rats tend to decrease after extended exposure to O_3, although structural changes in the centriacinar region have been reported after long-term exposures of rats (Boorman et al., 1980; Moore and Schwartz, 1981; Barry et al., 1983; Crapo et al., 1984), monkeys (Eustis et al., 1981; Fujinaka, 1984; Fujinaka et al., 1985), and dogs (Freeman et al., 1973). While cell repair begins within 18 hours of exposure (Castleman et al., 1980; Evans et al., 1976 a,b,c; Lum et al., 1978), cell damage continues throughout long-term exposures but at a slower rate (CD, p. 12-47).

Increases of lung collagen content in the centriacinar interalveolar septa may be indicative of lung fibrosis and, thus, of structural damage. Thus morphological effect has been associated with biochemical changes in activity of prolyl hydroxylase and in hydroxyproline content, both related to lung collagen content and fibrosis (Last et al., 1979; Bhatnagar et al., 1983). Lung collagen content increased after exposures to O_3 concentrations as low as 0.5 ppm (Last et al., 1979) and continued to increase during long- term exposures (Last et al., 1984b). Weanling and adult rats exposed for 6 and 13 weeks, respectively, and young monkeys for one year to < 1.0 ppm O_3 also showed increased collagen content in the lungs (Last et al., 1984b). In the Last et al., (1984b) study, examination of some of the exposed weanlings and controls at six weeks post-exposure

indicated a continued increase in lung collagen content, a result demonstrating that damage continued to occur during the post-exposure period. Also, there was little apparent difference in collagen levels of rat lungs for those animals exposed continuously vs. intermittently. This would suggest that intermittent periods of clean air were insufficient for recovery.

The centriacinar inflammatory process also continues during long-term O_3 exposures and appears to be related to remodeling of the centriacinar airways (Boorman et al., 1980; Moore and Schwartz, 1981) and to increased lung collagen which appears mainly in the centriacinar regions (Last et al., 1979; Boorman et al., 1980; Moore and Schwartz, 1981; Last et al., 1984b). In addition there is morphometric (Fujinaka et al., 1985), morphologic (Freeman et al., 1973), and functional (Costa et al., 1983; Wegner, 1982) evidence of distal airway narrowing.

Several new animal toxicology studies have focused on effect of prolonged exposure and appropriate selection of an averaging time associated with respiratory damage from O_3 exposure. Rombout et al. (1989) conducted a study in which rats were exposed to O_3 levels of 0.13 to 2.0 ppm and time of exposure ranged from 1 to 54 hours. With time course of protein and albumin concentration as the endpoint, Rombout et al. (1989) concluded that the response model indicates a strong influence of time on the response, that increases with increasing concentration. Van Bree et al. (1989) exposed rats to 0.4 ppm for 12 hours on 3 consecutive nights and found that acute pulmonary injury and inflammation, as measured by lung lavage protein increase and neutrophil influx, were induced by the first exposure and showed full reversibility in spite of continued exposure. However, biochemical indices for cell proliferation and lung tissue repair continued to show increases with subsequent exposure. A polynomial model has been developed by Costa et al. (1989) to depict lung injury from the interaction of O_3 concentration (C) and exposure duration (T). The model was based on lung fluid protein values in rats exposed to O_3 levels

between 0.1 and 0.8 ppm for 2, 4 or 8 hours and suggests that impact of T on O_3 toxicity appears to be C-dependent. Further development of these models is expected as efforts continue to improve understanding of C x T relationships.

While the distal airway narrowing and lesions at the junction of the conducting airways and gaseous exchange region are similar to the changes which have been found in lungs of cigarette smokers (Niewohner et al., 1974; Cosio et al., 1980; Hale et al., 1980; Wright et al., 1983), there is no evidence of emphysema in the lungs of animals exposed to only O_3 based on the currently accepted definition for emphysema cited in the CD (p. 9-49). Many of the lung structural changes that have been reported for long-term exposures to < 1.0 ppm O_3 in several different species are considered adverse and relevant to standard setting if demonstrated in humans at ambient exposure concentrations of O_3. Although the lowest O_3 concentrations and shortest averaging times which could produce structural changes in human lungs is uncertain at this time, the staff concludes that there is a need to protect the public from O_3 exposures which may induce such adverse effects. Further analysis is needed to clarify how animal data should be used to estimate lung structural changes in humans caused by O_3 exposures. The staff suggests that these data be used in developing a margin of safety for the primary standard.

7. Effects of Ozone on Host Defense Mechanisms in Experimental Animals

Respiratory systems of mammals are protected from bacterial and viral infections by the closely interrelated particle removal (both mucociliary and phagocytic) and immunological defense systems. Numerous factors such as poor nutrition, preexisting disease, and environmental stress may influence or alter these host defense systems of individuals sufficiently to permit development of respiratory infections. Animal studies have

indicated that exposure to O_3 is one of those factors (See Table VII-3). Both in vivo (live animal) and in vitro (isolated cell) studies have demonstrated that O_3 can affect the ability of the clearance and immune systems to defend against infection. Increased susceptibility to bacterial infection has been reported in mice at 0.08 to 0.10 ppm O_3 for a single 3-hour exposure (Coffin et al., 1967; Ehrlich et al., 1977; Miller et al., 1978a) and at 0.10 ppm O_3 for subchronic exposures (Aranyi et al., 1983). Several related alterations of the pulmonary defenses caused by short-term and subchronic exposures to O_3 include: 1) impaired ability to inactivate bacteria in rabbits and mice (Coffin et al., 1968; Coffin and Gardner, 1972; Goldstein et al., 1977; Ehrlich et al., 1979); 2) delayed mucociliary clearance (Phalen et al., 1980; Frager et al., 1979; Kenoyer et al., 1981; Abraham et al., 1980); 3) immunosuppression (Campbell and Hilsenroth, 1976; Aranyi et al., 1983; Fujimaki et al., 1984); 4) significantly reduced number of pulmonary defense cells in rabbits (Coffin et al., 1968; Alpert et al., 1971); and 5) impaired macrophage phagocytic activity, less macrophage mobility, more fragility and membrane alterations, and reduced lysosomal enzymatic activity (Dowell et al., 1970; Hurst et al., 1970; Hurst and Coffin, 1971; Goldstein et al., 1971a,b; Goldstein et al., 1977; Hadley et al., 1977; McAllen et al., 1981; Witz et al., 1983; Amoruso et al., 1981). Some of these effects have been shown to occur in a variety of species including mice, rats, rabbits, guinea pigs, dogs, sheep, and monkeys.

Other studies indicate similar effects for short-term and sub-chronic exposures of mice to O_3 combined with pollutants such as SO_2, NO_2, H_2SO_4 and particles (Gardner et al., 1977; Aranyi et al., 1983; Ehrlich, 1980, 1983; Grose et al., 1980, 1982; Phalen et al., 1980; Goldstein et al., 1974). Similar to human pulmonary function response to O_3, activity levels of mice exposed to O_3 has been shown to play a role in determining the

TABLE VII-3. EFFECTS OF OZONE ON HOST DEFENSE MECHANISMS IN EXPERIMENTAL ANIMALS

Effect	Ozone concentration, ppm	Exposure duration	Species	Reference
Increased susceptibility to bacterial respiratory infection	0.08	3 hr	Mouse	Coffin et al., 1967
	0.08	3 hr	Mouse	Miller et al., 1978a
	0.1	3 hr	Mouse	Ehrlich et al., 1977
	0.1	5 hr/d x 103 d	Mouse	Aranyi et al., 1983
Impaired ability to inactivate bacteria and viruses	0.1	3 hr/d x 3 mo	Mouse	Ehrlich et al., 1979
	0.5	3 hr	Rabbit	Coffin et al., 1968
	0.5	3 hr	Rabbit	Coffin and Gardner, [illegible]
	0.5	2 hr	Rat	Goldstein et al., 197[illegible]
Delayed muco-ciliary clearance	0.4	4 hr	Rat	Kenoyer et al., 1981
	0.8	4 hr	Rat	Phalen et al., 1980
	1.0	2 hr	Sheep	Abraham et al., 1980
	1.2	4 hr	Rat	Frager et al., 1979
Immunosuppression	0.1	5 hr/d x 90 d	Mouse	Aranyi et al., 1983
	0.59	continuous for 36 d	Mouse	Campbell and Hilsenro 1976
	0.8	continuous for 14 d	Mouse	Fujimaki et al., 1984
Impaired macrophage function	0.25	3 hr	Rabbit	Hurst et al., 1970
	0.25	3 hr	Rabitt	Hurst and Coffin, 1971
	0.5	8 hr/d x 7 d	Rabbit	Dowell et al., 1970
	0.5	2 hr	Rat	Goldstein et al., 1977
	0.5	3 hr	Rabbit	Hadley et al., 1977
	0.62	4 hr	Mouse	Goldstein et al., 1971
	0.99	17 hr	Mouse	Goldstein et al., 1971
	1.0	4 hr	Rat	McAllen et al., 1981

lowest effective concentration which alters the immune defenses (Illing et al., 1980).

Although this large body of evidence clearly demonstrates that short- term and subchronic exposures to O_3 can impair the immune defense systems of animals, technical and ethical considerations have limited similar research on human subjects. Thus inferences have been drawn and models developed to assess the relevance of animal data to humans. For example, animal endpoints other than increased mortality should be appropriately compared to increased morbidity in humans, such as the increased prevalence of respiratory illness in the community (CD, p. 12-50).

Based on current understanding of physiology, metabolism, and immune defenses, it is generally accepted that the basic mechanisms of action associated with defense against infectious agents are similar in humans and animals. Similarities between human and rodent antibacterial systems have been discussed in detail (Green, 1984), but differences in lung structure and biological response will inevitably cause differences in dosimetry, sensitivity, and endpoints of response. In addition, other factors such as preexisting disease, nutrition, presence of other pollutants and environmental stresses (e.g. NO_2, SO_2, PM, high temperature or humidity) can influence the effect of exposure to O_3 and infectious agents. Despite the differences in related factors which make precise estimation of human response from animal data difficult, it is reasonable to hypothesize that humans exposed to O_3 could experience impairment of host defenses (CD, p. 12-50). The staff recommends that these data be used in developing a margin of safety.

8. Extrapulmonary Effects

Extrapulmonary effects which have been demonstrated in humans or laboratory animals following exposure to O_3 include alterations in red blood cell morphology and enzyme activity, cytogenetic effects in circulating lymphocytes, and subjective

limitations in vigilance tasks. Additionally, animal toxicology studies provide limited evidence for cardiovascular, reproductive, teratological, endocrine system, and liver metabolism effects. This wide variety of extrapulmonary effects is probably caused by oxidative reaction products of O_3. Due to the high reactivity of O_3 with biological tissue, mathematical models predict that only a small fraction of O_3 actually reaches the circulatory system (Miller et al., 1985).

Of the extrapulmonary effects reported, cytogenetic and mutational effects are probably the most controversial (CD, p. 12-51). Statistically significant increases in frequency of sister chromatid exchanges (chromosomal alterations) have been caused by in vitro exposures to 0.25 ppm for 1 hour (Guerrero et al., 1979), suggesting a mutagenic response. However, in vitro responses are not generally extrapolatable to in vivo responses, and homeostatic mechanisms are not represented. Therefore, isolated in vitro exposure studies have not been used to provide estimations of risk for exposure to O_3. In vivo animal studies have shown significant increases in the number of chromosomal and chromatid aberrations following 4 and 5 hour exposures to 0.2 and 0.43 ppm O_3, respectively (Zelac et al., 1971a,b,; Tice et al., 1978). However, other animal studies (Gooch et al., 1976) and controlled human exposures to O_3 levels as high as 0.5 ppm have shown no significant cytogenetic effects attributable to O_3 (Merz et al., 1975; McKenzie et al., 1977; McKenzie, 1982; Guerrero et al., 1979), and epidemiology studies provide no evidence of chromosomal changes induced by ambient O_3 (Scott and Burkart, 1978; Magie et al., 1982). Thus, while the animal studies are suggestive of possible cytogenetic effects from O_3, human studies have not demonstrated such effects for realistic ambient exposures to O_3.

Limited hematological and serum chemistry effects data indicate that O_3 or one of its reactive products can cross the blood gas barrier and interfere with biochemical mechanisms in human blood erythrocytes and sera (CD, p. 1-132). Much of the

data is in vitro and useful primarily in studying mechanisms of action. Ozone-induced effects such as pentobarbital- induced sleep time alterations and hormone level changes have been reported (Graham et al., 1981) and cause-effect hypotheses made. With regard to other extrapulmonary effects, exact mechanisms remain to be elucidated and physiological significance for humans remains uncertain. Nonetheless, the body of this evidence suggests that O_3 can cause effects distant from the lungs in animals, and hence possibly in humans. It is the staff's recommendation that these data be used in developing a margin of safety.

B. Pulmonary Function and Symptom Health Risk Assessment

This section summarizes an assessment (Hayes et al., 1987a,b; Whitfield, 1988) of risks for two categories of effects (pulmonary function and symptoms) associated with attainment of alternative 1-hr O_3 NAAQS. These health risk estimates characterize acute responses based on results of 1- and 2-hour controlled human exposure studies reviewed in the CD. While recent controlled human exposure research and field studies suggest that longer exposures (6-8 hours) at less strenuous levels of exercise may also pose a health threat, EPA is awaiting completion of additional research prior to extending the risk assessment to these subchronic exposures. EPA also plans to pursue risk assessment for chronic, possibly irreversible, effects on the lung observed in various animal toxicology studies upon completion of important dosimetry and lung injury research currently underway.

1. Overview of Lung Function and Symptom Risk Assessment

The objective of the pulmonary function/symptom risk assessment is to estimate the magnitude and extent of risk to the most susceptible population for these effects (i.e., heavily exercising individuals) while characterizing, as explicitly as possible, the range and implications of uncertainties in the

existing scientific data base. While the risk assessment estimates should not be viewed as demonstrated health impacts, they do represent EPA's best estimate as to the possible magnitude and extent of risk for these effects given the available scientific information. Although it does not cover all health effects caused by O_3, the risk assessment is intended as a tool that may, together with other information presented in this staff paper and the CD and CDS aid the Administrator in judging which alternative O_3 NAAQS provides an adequate margin of safety. Risk estimates for a number of urban areas, the aggregated total for those areas, and the methodology used to generate these estimates are described in detail in Hayes et al. (1987a,b) and Whitfield (1988).

The three major types of inputs to the risk assessment are:

(1) exposure-response relationships used to characterize pulmonary function and symptom effects of O_3 exposure in heavily exercising individuals developed from data obtained in three controlled human exposure studies: Avol et al. (1984), Kulle et al. (1985), and McDonnell et al. (1983);

(2) distributions of O_3 hourly concentrations upon attainment of alternative NAAQS and under the "as is" situation obtained from the O_3-NEM project (Paul et al., 1986); and

(3) distributions of population exposure, both in terms of people exposed and occurrences of exposure, upon attainment of alternative O_3 NAAQS and under the "as is" situation obtained from the O_3-NEM analysis (Paul et al., 1986).

Chapter V of this staff paper presents an overview of the O_3 exposure analysis methodology and summarizes exposure estimates for the aggregated 8-urban area population for alternative air quality scenarios. The pulmonary function and symptoms risk assessment considers the same four scenarios used in the exposure analysis: 0.08, 0.10, and 0.12 ppm, daily maximum 1-hour O_3 NAAQS with an expected exceedance rate of once per year and the "as is" situation.

Risk estimates have been developed for both pulmonary function (as measured by FEV_1 decrement) and lower respiratory symptoms (specifically, cough, chest discomfort, and as a group of symptoms). A health effect, or endpoint, can be defined in terms of a measure of biological response and the amount of change in that measure thought to be of concern. To permit EPA decision makers and the public to examine the implications of alternative definitions of adversity, risk estimates are presented for a range of different health endpoints:

(1) Pulmonary function. Two different level of O_3-induced FEV_1 decrement: (a) at least 10 percent and; (b) at least 20 percent.

(2) Lower respiratory symptoms. Cough, chest discomfort, and lower respiratory symptoms as a group for two different severity levels: (a) any symptoms (mild, moderate, or severe) and (b) moderate or severe symptoms.

Estimated exposure-response relationships derived from each of the three health studies (Avol et al., 1984; Kulle et al., 1985; and McDonnell et al., 1983) are used separately to derive independent risk estimates. While the three studies are similar in enough respects (e.g., health endpoints, young heavily exercising healthy subjects, similar 1-2 hour O_3 exposures) to make useful comparisons, there are enough differences in experimental protocol (e.g., 1-hour continuous exposure in Avol vs. 2-hour intermittent in Kulle and McDonnell and differences in exact exercise level and exposure concentration) to make statistical combination of these data bases undesirable. Estimated exposure-response relationships for FEV_1 decrement and symptomatic effects used in the risk assessment are shown in Figures VII-2 through VII-4 earlier in this staff paper. These relationships were derived by fitting a four-parameter logistic function to each data set and subtracting out the effect of exercise alone based on the response at 0 ppm (see Chapter 3 of Hayes et al., 1987a for details).

Uncertainty attributed to sampling error due to sample size considerations in the exposure-response relationships is reflected in the pulmonary function and symptom risk estimates. This uncertainty was estimated using a Bayesian approach described in Chapter 3 of Hayes et al. (1987a). Other sources of uncertainty due to differences in experimental protocol subject population, measurement error, etc. have not been quantitatively addressed. The calculation and presentation of separate risk estimates for each of the three data sets provides a rough picture of the degree of uncertainty due to these other factors.

Two distinct types of risk measures are provided by the O_3 health risk assessment. The first measure, "benchmark risk," focuses on the probability or risk of unhealthful air. The second measure, "headcount risk," focuses on the number of people affected and number of incidences of a given health effect considering individuals' personal exposures as they go about their daily activities (e.g., from indoors to outdoors, moving from place to place, and engaging in different exercise levels).

More specifically, benchmark risk is the probability that pollutant levels will be sufficient to trigger a defined health endpoint in at least a fraction of the sensitive population if they were exposed. The second measure, headcount risk, assesses the number of people or the percent of the sensitive population that would be adversely affected given the normal movement and activity patterns of the population of interest. Headcount risk also provides estimates of the number of incidences of adverse effects there would be. Staff believe that these risk measures taken together capture two important perspectives that should be considered in selecting an O_3 primary standard that provides an adequate margin of safety.

2. Benchmark Risk Results

For the O_3 pulmonary function and symptom risk assessment the benchmark risk is defined as the probability that, upon just attaining a given O_3 NAAQS or under "as is" conditions, the

maximum hourly concentration will equal or exceed the level that would cause 1, 5, or 10% of the heavily exercising population to exhibit particular health endpoints 1 or more times per year. The benchmark risk is estimated assuming the entire sensitive population is exposed while heavily exercising (defined as Regime 3 in O_3-NEM which is 3 or more 10 minute periods in an hour at a $\geq$ 44 L/min). As indicated in the risk report:

> Benchmark risk is measured in excess of that which would occur under background conditions because (a) only O_3 levels above background are amenable to human control and (b) it is difficult to reliably estimate the very small, hypersensitive fraction of the heavily exercising population that might respond at O_3 levels at or below background. While background O_3 levels can vary during the day and from day to day, for the purposes of this risk assessment, 0.04 ppm is taken to be a generally representative surrogate for the concentration level of background O_3 (Hayes et al., 1987a, p. 4-2).

Benchmark risk is calculated by combining exposure-response relationships and probability distributions of hourly O_3 ambient concentrations, either based on observed air quality data during 1981-1984 (the particular year depending on area) for the "as is" case or based on conditions of exact attainment of alternative NAAQS (0.08, 0.10, and 0.12 ppm, daily max hourly average, 1 expected exceedance per year). The benchmark risk model and more detailed discussion of the inputs to the model are contained in Hayes et al. (1987a,b).

Benchmark risk estimates are calculated for the 10 urban areas shown in Table V-1 and are presented in Hayes et al. (1987a,b). Due to differences in the degree of non-attainment of the O_3 NAAQS among the ten areas (ranging from Los Angeles to Tacoma) the benchmark risk associated with the "as is" scenario varies significantly among the urban areas. However, benchmark risk estimates for a particular endpoint, data base, and choice of benchmark case are very similar for a given NAAQS-attainment scenario across most of the 10 urban areas. Selected results for

a single urban area, St. Louis, are presented here which are generally representative of benchmark risks for the other urban areas.

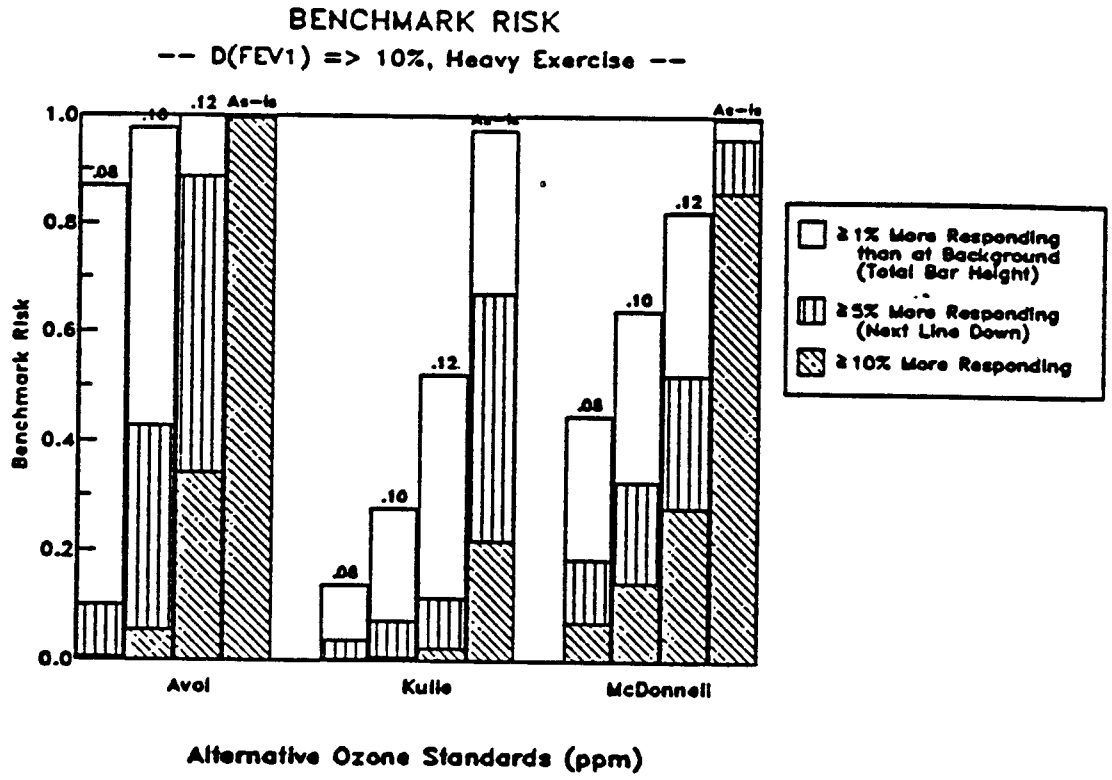
Figure VII-5 presents benchmark risks for two different degrees of FEV_1 decrement: at least 10% in the top chart and at least 20% in the bottom chart. Risk estimates are shown as three groups of bars, one group for each health data set used to generate exposure-response relationships. Each group consists of four bars: one bar for each of the three alternative O_3 NAAQS analyzed (0.08, 0.10, and 0.12 ppm, daily max hourly average, 1 expected exceedance per year) and one bar for the "as is" situation. The height of the bar represents a 1%-benchmark case, that is, "the probability that at least 1 percent or more of the heavy exercisers would respond with the specified health endpoint than under background conditions, one or more times during the O_3 season" (Hayes et al., 1987a,b). The top of the vertically-shaded portion corresponds to a 5%-benchmark case and the top of the slant-shaded portion corresponds to a 10%-benchmark case.

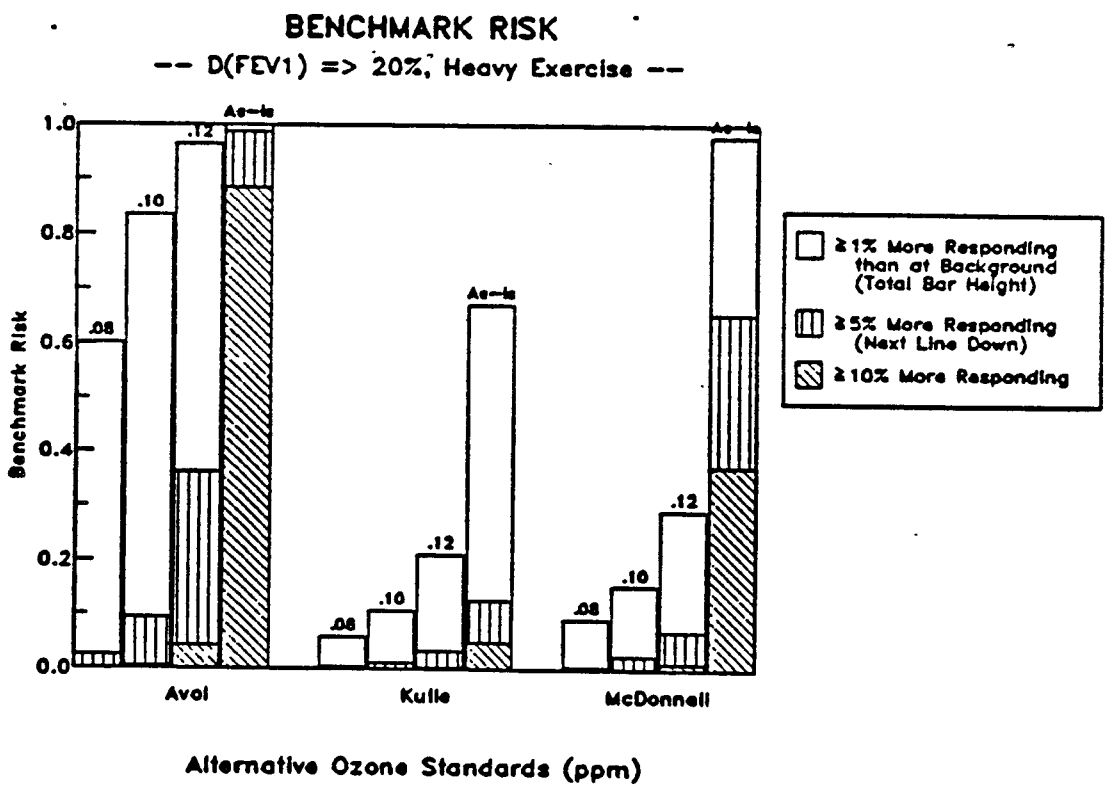
As an example of how to read the benchmark risk figures, consider the $\geq$ 10 percent FEV_1 decrement case in Figure VII-5 (top):

> Under exact attainment of the current O_3 NAAQS (0.12 ppm), the probability that at least a 10 percent FEV_1 decrement would occur in at least 5 percent more heavy exercisers than at background, at least once during the O_3 season, is nearly 0.9 for the Avol data set, about 0.1 for the Kulle data set, and about 0.5 for the McDonnell data set (Hayes et al., 1987a,b, p. 4-7).

Using the same approach, Figure VII-6 presents benchmark risk estimates for any chest discomfort (top chart) and for moderate or severe chest discomfort (bottom chart). It should be noted that risk estimates for the Avol data base are for an aggregate lower respiratory symptom score that reflects chest discomfort, cough, and other lower respiratory symptoms.

Figures VII-5 and VII-6 illustrate the importance of certain key decision parameters in interpreting risk estimates. Both

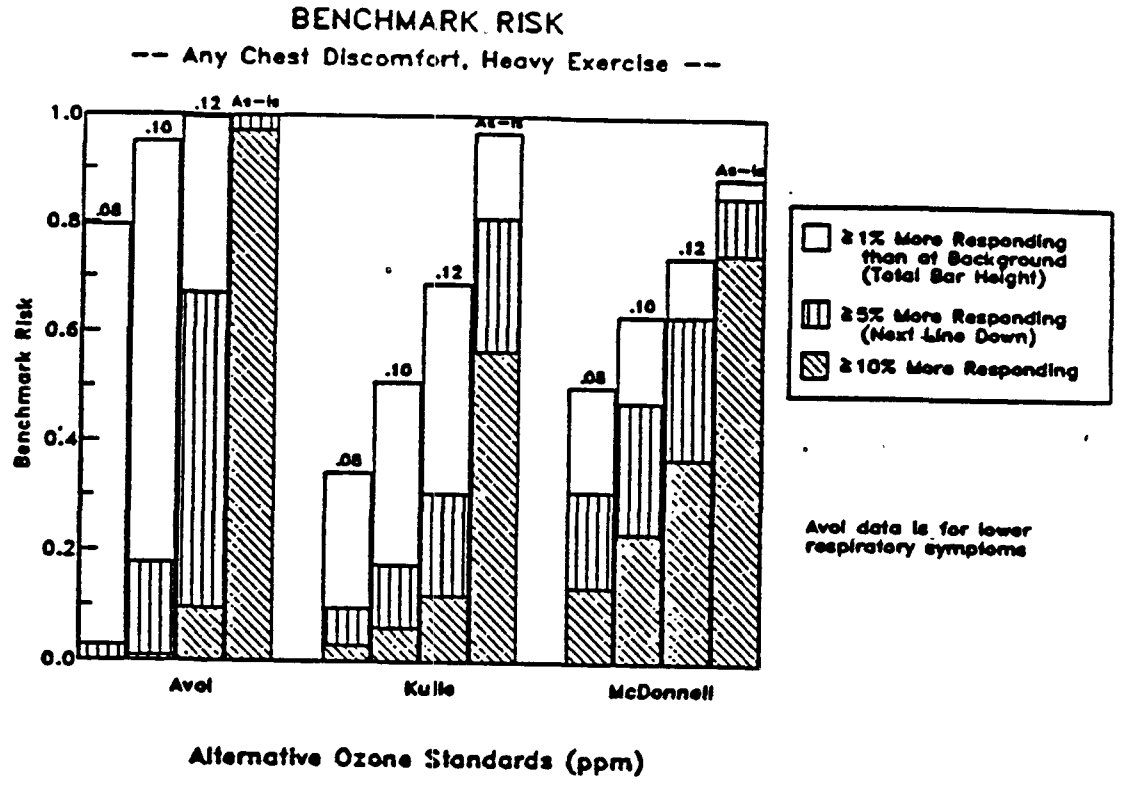


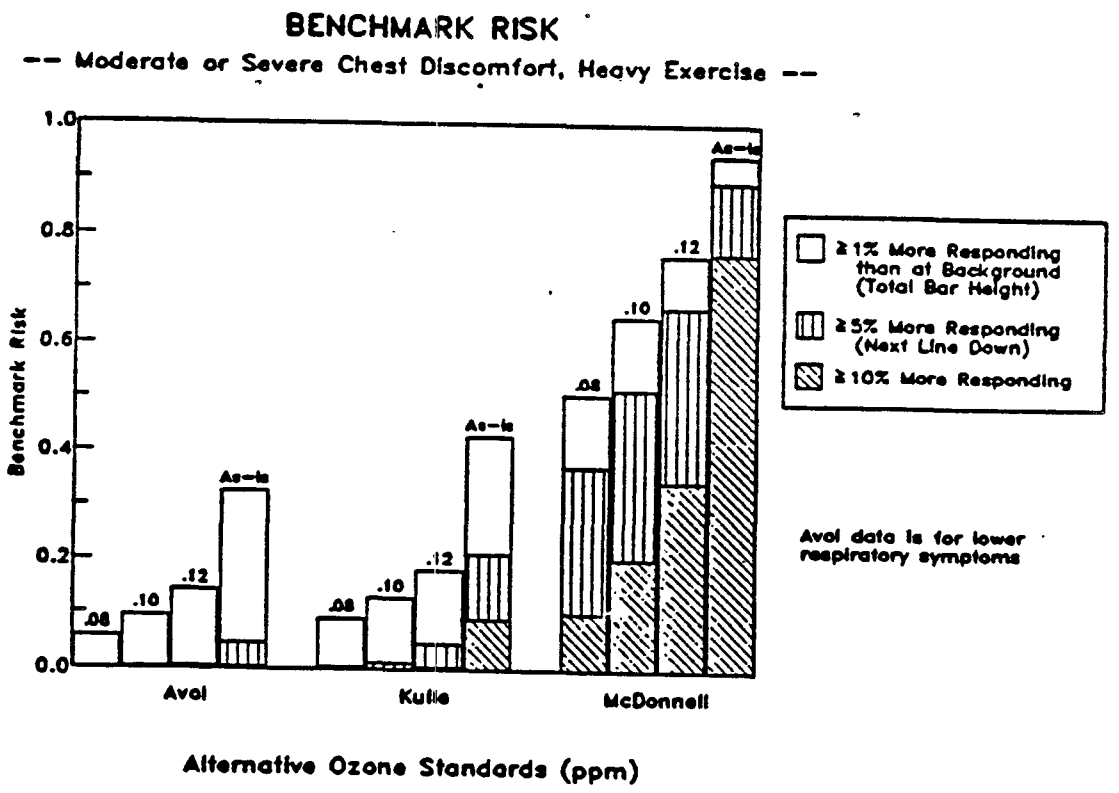


St.Louis

FIGURE VII-5. Benchmark risk in St. Louis for FEV1 decrements of ≥ 10% and ≥ 20%, under heavy exercise, for three exposure-response data sets (Avol, Kulle, and McDonnell).

Source: Hayes et al., 1987





St.Louis

FIGURE VII-6. Benchmark risk in St. Louis for chest discomfort symptoms (any and moderate/severe), under heavy exercise, for three exposure-response data sets (Avol, Kulle, and McDonnell).

Source: Hayes et al., 1987

selection of endpoint definition (e.g., ≥10% or ≥20% decrement in FEV_1) and n%-benchmark case (e.g., at least 1, 5, or 10% of the sensitive population which must experience the health effect) significantly affect the risk estimates. In addition, comparing risks across health data bases results in significant differences. FEV_1 risk estimates for a given benchmark definition, say 1%, are highest for the Avol data base and lowest for the Kulle data base. In contrast, for moderate/severe chest discomfort and lower respiratory symptoms, risk estimates are highest for the McDonnell data base and lowest for the Avol data base.

3. Headcount Risk Results

For the O_3 risk assessment headcount risk is characterized by calculating the expected headcount, that is, the expected number of people experiencing a defined effect and the expected number of incidences of that effect projected to occur during the O_3 season, given that a particular NAAQS is just attained or under "as is" air quality conditions. Headcount risk estimates the risk posed to the sensitive population (in this case individuals engaged in heavy exercise) as they go about their normal activities.

A major input to the headcount risk model is the series of population exposure distributions for "as is" air quality and the three alternative NAAQS analyzed by EPA. Using available exposure estimates, headcount risk estimates were calculated for eight of the ten urban areas listed in Table V-1. No headcount risk estimates are calculated for Chicago and New York, since EPA is in the process of revising these estimates. Appendix C of Hayes et al. (1987a,b) presents headcount risk estimates for each of the 8 urban areas.

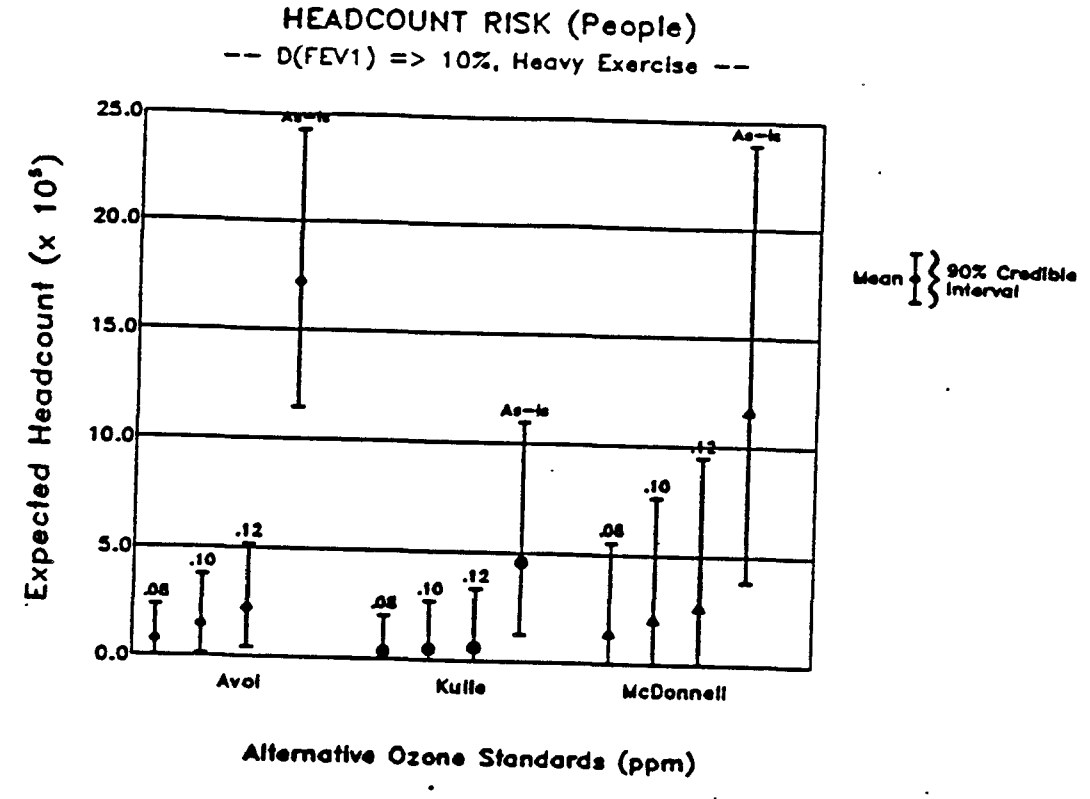
Risk estimates presented here are an aggregation of the results from the 8 urban areas. The total population living in the 8 urban areas is approximately 25.9 million. Of this population, 9.3 million people (or 36%) are estimated to exercise

sufficiently heavily to reach NEM Exercise Regime 3 (3 or more 10-minute periods in an hour at heavy ($\dot{V}_e \geq 44$ l/min) which could include one 10-minute period at very heavy ($\dot{V}_e \geq 64$ l/min) exercise).

Figure VII-7 presents risk estimated for each of the three health data sets for two levels of FEV_1 response: at least 10% decrement on top and at least 20% decrement on the bottom for the three alternative O_3 NAAQS analyzed (0.08, 0.10, and 0.12 ppm, daily max hourly average, 1 expected exceedance) and the "as is" situation. Similarly, Figure VII-8 presents expected headcount estimates for any chest discomfort (top chart) and moderate or severe chest discomfort (bottom chart). Again, it should be noted that risk estimates for the Avol data base are for lower respiratory symptoms which include chest discomfort as well as cough and other symptoms. The complete headcount risk results are contained in Hayes et al. (1987a,b), including risk estimates for cough and for expected number of incidences of each of the 6 health endpoint definitions examined, as well as estimates for each of the 8 urban areas examined.

In both Figures VII-7 and VII-8 the expected headcount in millions of people is indicated by the filled-circles and triangles and 90% credible intervals are displayed as vertical bars. The credible interval reflects uncertainty in the expected headcount attributed to uncertainty in exposure-response relationships due to sample size considerations. The 90% credible interval can be interpreted as a 0.9 probability that the headcount lies within the interval, and a 0.10 probability that it falls outside of the interval.

As with the benchmark risk results, for FEV_1 decrement the Avol data base results in the highest headcount estimates and Kulle shows the lowest estimate and for chest discomfort the McDonnell data base results in the highest headcount estimate and Avol shows the lowest estimate. One also observes a greater reduction in headcount estimate going from the "as is" situation to the 0.12 ppm NAAQS than in going from 0.12 ppm to a 0.10 or



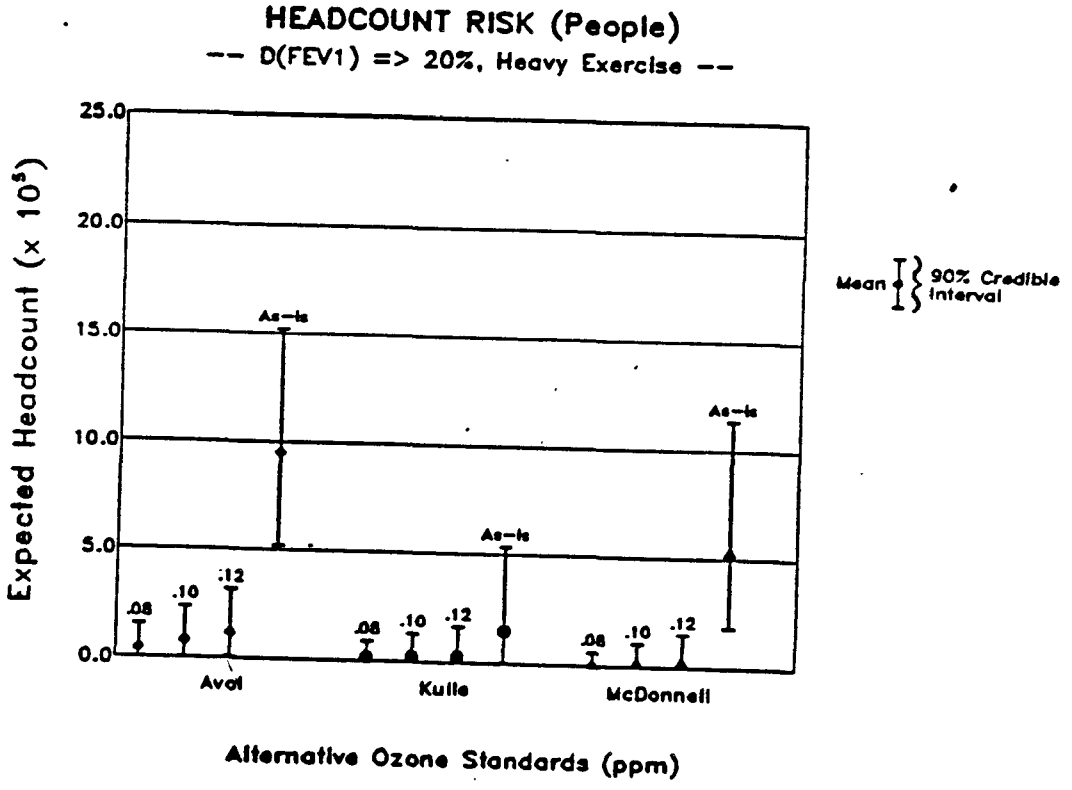
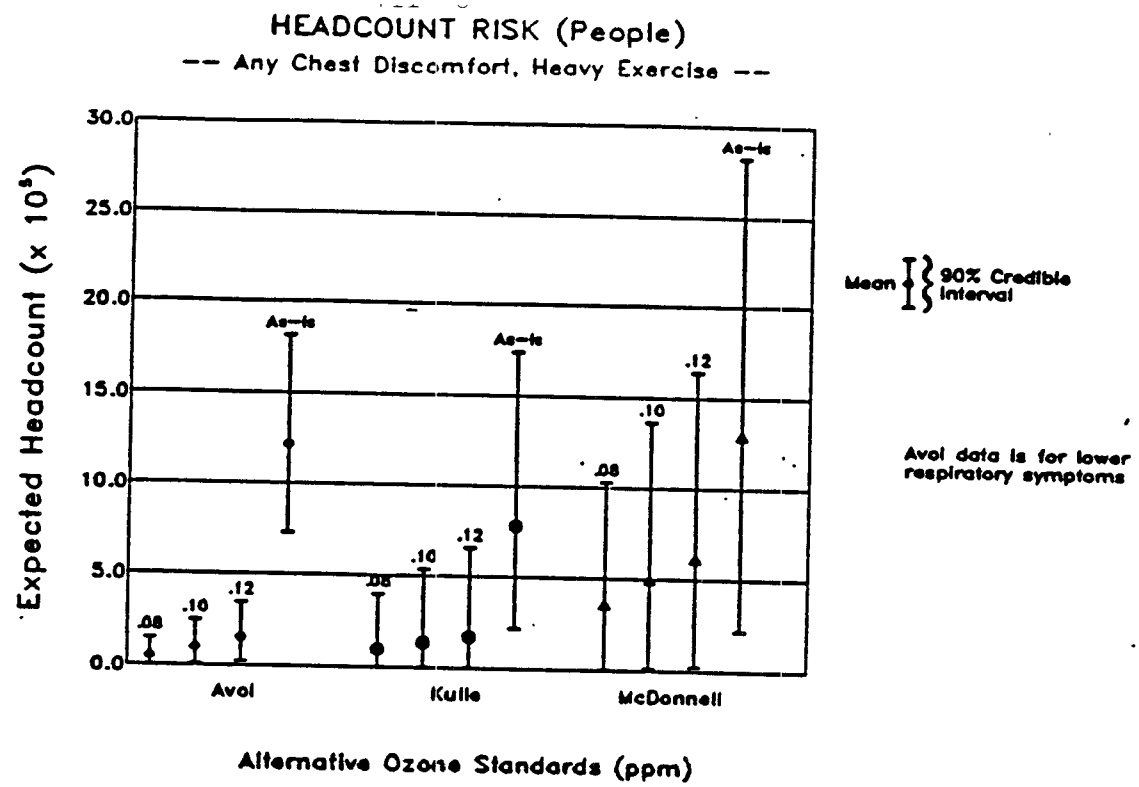
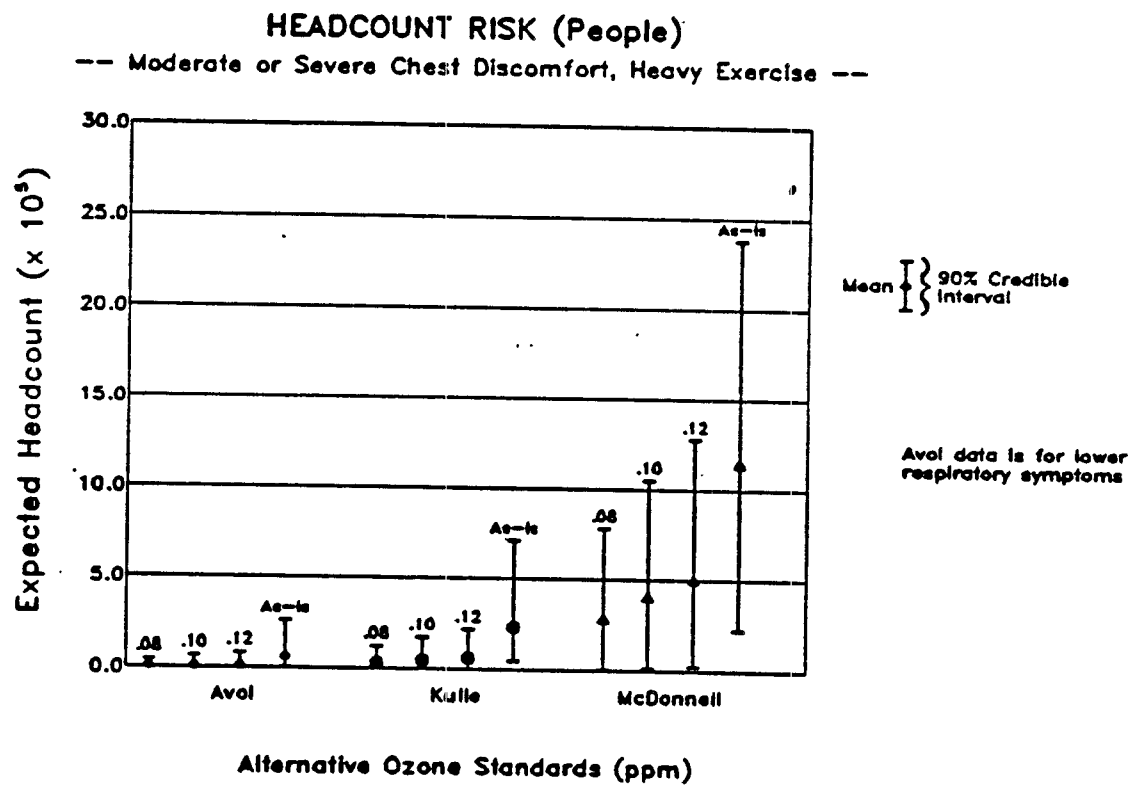


Figure VII-7. Expected headcount (pulmonary function) aggregated for eight U.S. urban areas (number of heavily exercising people responding during the ozone season).





Eight City Aggregation

FIGURE VII-8. Expected headcount (chest discomfort) aggregated for eight U.S. urban areas (number of heavily exercising people responding during the ozone season).

Source: Hayes et al.; 1987

0.08 ppm NAAQS. However, one must note that the expected headcount in Figures VII-7 and VII-8 for the "as is" case areas dominated by Los Angeles' contribution to headcount risk and should not be viewed as representative for the nation. For example, in Figure VII-7, the expected headcount for FEV_1 decrement ≥ 10% based on the Avol et al. (1984) study is about 1.7 million people, of which 1.3 million people are from Los Angeles. Headcount risk estimates can also be expressed as a percentage of the heavily exercising population. Table VII-4 presents the range of mean percentages of heavy exercisers responding 1 or more times per year upon attainment of 3 alternative NAAQS and for the "as is" situation for each of the 6 health endpoint definitions examined. The range is due both to variation in the shape of air quality distributions in the 8 urban areas and to differences in exposure-response relationships based on the three health data bases (Avol, Kulle, and McDonnell). Again, it is important to recognize the influence of the Los Angeles estimates when considering the "as-is" case. Removing Los Angeles from the "as-is" column would for example reduce the upper end of the range from 36.3% to 14.0% for FEV_1 ≥ 10%.

TABLE VII-4. Percent of Heavy Exercisers Responding Under Alternative Air Quality Scenarios

Health Endpoint		Mean Percentages of Heavy Exercisers Responding (As-Is)	Mean Percentages of Heavy Exercisers Responding Under NAAQS Attainment Across Eight U.S. Urban Areas		
			0.12 ppm	0.10 ppm	0.08 ppm
FEV_1	>10%	1.1 to 36.3	0.5 to 4.2	0.1 to 3.0	0.0 to 1.9
	>20%	0.5 to 20.7	0.2 to 2.1	0.0 to 1.3	0.0 to 0.7
Cough	Any	3.1 to 41.8	0.9 to 18.8	0.2 to 15.2	0.1 to 10.1
	Mod/Sev	0.2 to 27.7	0.1 to 4.8	0.0 to 2.9	0.0 to 1.4
Chest Discom-fort	Any	2.7 to 20.4	0.8 to 8.5	0.1 to 7.0	0.1 to 4.8
	Mod/Sev	0.2 to 18.7	0.1 to 7.1	0.0 to 5.8	0.0 to 3.8

Source: Hayes et al., 1987

4. Caveats and Limitations

A number of assumptions and limitations should be kept in mind in interpreting results of the pulmonary function and symptom risk assessment. Extrapolation of results of the Avol, Kulle, and McDonnell data sets to the heavily exercising population at large is affected by a number of considerations. These include the following:

(1) Exercise group. The CD defines heavy exercise as lung ventilation rates of 44-63 L/min and very heavy exercise as ≥64 L/min. The Avol study (57 L/min) corresponds to the mid-to-upper portion of the heavy exercise range (54 L/min is the mid-point). The McDonnell study (65 L/min) falls just in the lowest portion of the very heavy exercise range, with the Kulle study (68 L/min) some 5 percent higher. Exposure-response relationships obtained from the three data sets have been assumed to represent exposure-response in all heavy exercisers. Additional research is needed to determine the extent to which exposure-response relationships observed at 57, 64, and 65 L/min change at lower exercise levels such as 44-56 L/min. To the extent that exercise rates among many heavy exercisers are lower than in the subject studies, lung function and symptom headcount risks may be overstated. The extent to which this is so is unknown and must be regarded as an additional risk assessment uncertainty (Hayes et al., 1987a,b).

Since the December 1987 CASAC meeting, an addendum to the lung function and symptom risk assessment has been prepared (Whitfield, 1988) which combines the estimated exposure-response relationships from the same three studies discussed previously with the NEM very heavy exercise exposure estimates to generate expected headcount estimates. The resulting expected headcount estimates for very heavy exercisers are approximately 100-150 times smaller in magnitude than the heavy exercise expected headcount estimates. This is due principally to the fact that the population of very heavy exercisers is much smaller (by about two orders of magnitude) than the population of heavy exercisers.

(2) Interaction between O_3 and other pollutants. The health studies used in the risk assessment involved only O_3 exposure. It is assumed that the health effects of interest in the real world where other pollutants are present are due solely to O_3. While controlled human exposure studies have not consistently demonstrated enhancement of respiratory effects for O_3 when combined with SO_2, NO_2, CO, H_2SO_4, or other aerosols, there is some animal toxicology research suggesting additive or possibly synergistic effects. (See Section VII.C.5 for further discussion of interactions with other pollutants).

(3) Reproducibility of O_3-induced response. It is assumed that O_3-induced respiratory responses are reproducible for individuals. The CD cites both Gliner et al. (1983) and McDonnell et al. (1985a) in concluding that respiratory effects of O_3 are highly reproducible. Analysis of the Avol and Kulle data sets by Hayes et al. (1987b) also supports the reproducibility of individual responses.

(4) Age. The risk assessment has been applied to all heavily exercising individuals regardless of age. However, controlled human exposure and recent field epidemiology studies in children have reported pulmonary function, but not symptomatic, effects for O_3 exposures. Therefore, the headcount symptomatic effect estimates which rely on population exposures that include children may overstate symptom headcount estimates. Pulmonary function risk estimates are not affected, and the lack of apparent symptoms does not mean that biological processes associated with O_3 symptoms in adults are not also present in children.

(5) Sex. There is some limited evidence that women may be more responsive to O_3 in terms of FEV_1 impairment than men. The data sets used here to derive exposure-response relationships involved mostly male subjects. To the extent that women are more responsive than men, risk estimates may be understated.

(6) Smoking status. There is some limited evidence that smokers may be less responsive to O_3 than nonsmokers. The risk

assessment was applied to all heavy exercisers regardless of smoking status. To the extent that smokers are less responsive than nonsmokers, risk estimates may be overstated.

(7) Attenuation or enhancement of response. The risk assessment assumes that the O_3-induced response in any particular hour is not affected by previous O_3 exposure history. The extent of attenuation and/or enhancement of O3-induced responses due to previous O_3 exposures cannot be addressed quantitatively and must be regarded as an additional uncertainty in interpreting the risk estimates.

(8) Exposure and air quality estimates. A major input to the headcount risk is the O_3 exposure analysis estimates for the heavily exercising population. Uncertainties about human activity patterns and the procedures used to estimate O_3 concentrations upon attainment of alternative standards, as well as other uncertainties about the exposure analysis model and inputs to the model, must be regarded as additional uncertainties in interpreting the headcount estimates. As noted in Chapter V, ASB plans to use a different version of O_3-NEM in the future to address multiple-hour O_3 exposures and to more explicitly analyze various exposure-related uncertainties. When new O_3 NEM estimates are available, ASB plans to revise the risk assessment discussed in this section, assuming contract funds are available for that purpose. The benchmark risk estimates are affected by uncertainty in projecting O_3 concentrations upon attainment of alternative NAAQS at the "critical" monitor.

C. Related Health Effects Issues

The purpose of this section is to provide an overview of important issues related to the health effects of O_3 and other photochemical oxidants. Some issues such as attenuation of effects and the acute/chronic relationship involve more than one health effect, while other issues involve multiple pollutants. Detailed discussion of the individual studies associated with these issues can be found in chapters 9 to 12 of the CD.

1. Adverse Respiratory Health Effects of Acute Ozone Exposure

What constitutes an adverse respiratory health effect for acute exposure to O_3 has been a matter of some controversy and diversity of opinion. Because the NAAQS for O_3 is intended to protect the population from exposure to O_3 levels which might produce adverse health effects, it is important to identify the adverse effects of O_3. Although incapacitating effects or irreversible damage are widely accepted as being adverse, that type of response has not been reported for acute exposures of humans to O_3 levels near the current standard of 0.12 ppm.

Public health organizations, such as the American Thoracic Society (ATS), have developed general guidelines (ATS, 1985) as to what constitutes an adverse respiratory health effect with respect to interpretation of epidemiological studies. While recognizing that perceptions of "medical significance" and "normal activity" may differ among physicians and experimental subjects, the ATS (1985) defines adverse respiratory health effects as "... medically significant physiologic or pathologic changes generally evidenced by one or more of the following: (1) interference with the normal activity of the affected person or persons, (2) episodic respiratory illness, (3) incapacitating illness, (4) permanent respiratory injury, and/or (5) progressive respiratory dysfunction." Although acute O_3 exposures of human subjects have been associated only with the first of the above effects, animal studies suggest that longer-term O_3 exposures may cause more serious health effects as well.

The most commonly reported and well-established respiratory health effect of acute O_3 exposure, as discussed earlier in this chapter, is reduction in lung function. In order to address the adversity or clinical significance of this response, however, it is important to consider: (1) the magnitude of a functional change on a test-specific basis (e.g., FEV, FVC); (2) the duration or persistence of the effect; (3) the respiratory symptoms which are associated with functional changes; and (4)

limitation of activity which might result from functional losses and respiratory discomfort.

In conjunction with health scientists of the Office of Research and Development, OAQPS staff has developed Table VII-5 which shows a gradation of individual response to acute O_3 exposure. While Table VII-5 is not intended to imply exact quantitative relationships, it does categorize three variables as being mild, moderate, severe, or incapacitating depending on severity of response. Relationships among the response variables not only assist in judging the severity of effects but can be used as a general framework for assessing adversity of effects for acute O_3 exposure.

During the CASAC meeting of December 14-15, 1987 and December 14-15, 1988, and in written comments received since, a wide diversity of opinion has been expressed regarding what should be considered an adverse respiratory health effect. Most comments indicated that Table VII-5 was generally accepted as reflecting the range of effects associated with acute (1-2 hr) exposures to O_3. At the CASAC (1987) meeting some CASAC members expressed the belief that either limitation of activity or symptoms could be considered the primary determinant of adversity while others believed the more objective spirometry measurements were more appropriate. The point at which an effect can be described adverse was discussed in some detail. Some CASAC members felt that individuals would experience adverse effects when O_3 exposure induced any of the responses categorized as moderate. Other CASAC members believed that adverse effects would not be experienced until O_3 induced severe effects. A point of clarification was made that Table VII-5 refers only to healthy individuals rather than to persons with respiratory impairment. Individuals with preexisting respiratory disease may experience adverse effects even when encountering responses categorized as mild for healthy persons. Also, it was pointed out at the CASAC (1988) meeting that because children show few, if any, symptoms when exposed to ambient O_3 levels, it would be

TABLE VII-5. GRADATION OF RESPONSE FOR HEALTHY INDIVIDUALS ACUTELY EXPOSED TO OZONE

GRADATION OF RESPONSE	MILD	MODERATE	SEVERE	INCAPACITING
CHANGE IN SPIROMETRY $FEV_{1.0}$, FVC	5-10%	10-20%	20-40%	>40%
DURATION OF EFFECT	COMPLETE RECOVERY IN <30 MIN	COMPLETE RECOVERY IN <6 HR	COMPLETE RECOVERY IN 24 HOURS	RECOVERY IN >24 HOURS
SYMPTOMS	MILD TO MODERATE COUGH	MILD TO MODERATE COUGH, PAIN ON DEEP INSPIRATION, SHORTNESS OF BREATH	REPEATED COUGH, MODERATE TO SEVERE PAIN ON DEEP INSPIRATION AND SHORTNESS OF BREATH; BREATHING DISTRESS	SEVERE COUGH, PAIN ON DEEP INSPIRATION, AND SHORTNESS OF BREATH; OBVIOUS DISTRESS
LIMITATION OF ACTIVITY	NONE	FEW INDIVIDUALS CHOOSE TO DISCONTINUE ACTIVITY	SOME INDIVIDUALS CHOOSE TO DISCONTINUE ACTIVITY	MANY INDIVIDUALS CHOOSE TO DISCONTINUE ACTIVITY

inappropriate to recommend that all categories of response be seen in children before describing the effect as adverse.

Based on these comments of CASAC (1987, 1988), staff recommends that the responses identified as "mild" for healthy, adult response to O_3 not be considered an adverse respiratory health effect. Mild individual responses to O_3 exposures are viewed as involving only 5 to 10% decrements in spirometry which last 30 minutes or less accompanied by only mild to moderate cough and no limitation of activity. Mild response probably would not be considered medically significant and should not interfere with normal activity of most individuals. Staff recommends, however, that moderate, severe, and incapacitating responses be considered adverse respiratory health effects. These categories of response are more likely to be considered medically significant and could interfere with normal activities. For example, some adults with preexisting respiratory disease or heavily exercising healthy adults who experience a moderate response (i.e., lung function loss of 10-20% which persists for up to 6 hours accompanied by multiple symptoms) would tend to curtail activity. Due to lack of a clear consensus and the wide diversity of CASAC (1987,1988) opinions, staff recommends that all criteria specified for moderate response be met for an effect to be deemed adverse in healthy adults. The adverse nature of longer-term (6 to 8 hour) and chronic effects of O_3 will be discussed following completion of future research which addresses these effects.

2. Attenuation of Acute Pulmonary Effects

Attenuation of acute pulmonary response to O_3 after repeated daily exposures to O_3 is a well-established and well-documented phenomenon. Until recently, descriptive terms other than attenuation have commonly been used to describe the response, such as "adaptation" and "tolerance". These terms imply a reduced impact of repeated exposure to O_3 whereas recent evidence suggests that lung injury continues during the process of

attenuation. A thorough review of the large body of supporting evidence can be found in the CD (p. 10-47 to 10-60) and CDS . Functional decrements are generally greater on the second exposure day; by the fourth or fifth day of exposure, small or no decrements are observed (Farrell et al., 1979; Horvath et al., 1981; Kulle et al., 1982b; Linn et al., 1982). Attenuation of functional response to a particular O_3 level does not attenuate response to higher O_3 levels, nor is the attenuation process permanent. Subjects repeatedly exposed to 0.2 ppm O_3 for three consecutive days exhibited attenuation to that level of O_3 but showed no attenuation of response to higher (0.42 or 0.50 ppm) O_3 levels (Gliner et al., 1983). Once full attenuation is achieved it does not continue for more than 3 to 7 days for most subjects (Horvath et al., 1981; Kulle et al., 1982b; Linn et al., 1982), however, partial attenuation has been shown to persist for as long as 2 weeks (Horvath et al., 1981). Attenuation of symptoms correlates with magnitude of functional response and was shown in one isolated statistical result to last as long as 4 weeks (Linn et al., 1982). Thus individuals living in areas with high O_3 peaks may develop attenuated response to repeated lower O_3 levels and still respond to peak exposures, but after exposure to O_3 ceases the attenuated response eventually will return to normal.

Evidence reviewed in the CDS suggests the possibility that ambient oxidant exposure during summer months produced an "adaptation" response which persisted in human subjects for several months (Hackney et al., 1988). The authors suggested that allergy or atopy may be a risk factor for excess response and, further, that nonresponders could be at increased risk for chronic health effects of cumulative ambient O_3 exposure since they would be less likely to avoid such exposures due to lack of symptomatic adaptation or attenuation of O_3-induced deficits with sustained but not worsening protein accumulation in lavage fluid (Costa et al., 1989). However, histopathology of the animals appeared to worsen and evolve from an acute to a more chronic inflammatory pattern.

Attenuation of functional or symptomatic response does not necessarily imply attenuation of morphological or biochemical response. As indicated in the CD (p. 12-42),

> Responses to O_3, whether functional, biochemical, or morphological, have the potential for undergoing changes during repeated or continuous exposure. There is interplay between tissue inflammation, hyperresponsiveness, ensuing injury (damage), repair processes, and changes in response. The initial response followed by its attenuation may be viewed as sequential states in a continuing process of lung injury and repair.

Public health implications of attenuation, therefore, are that this response is not a protective mechanism but may in fact result in an increased O_3 dose to the deep lungs and potentially cause greater tissue damage by permitting individuals to exercise outdoors during elevated O_3 episodes.

3. Relationship Between Acute and Chronic Effects

In order to better assess attenuation of pulmonary responsiveness to O_3 and other effects of O_3 which may not be reversible, it is important to understand the relationship between acute and chronic effects. Although animal research provides some evidence, little is known with certainty regarding either long-term implications of repeated acute O_3 exposures or chronic effects of prolonged exposure of humans to O_3. Human chamber studies involving long-term exposures to O_3 have not been conducted due to concern for serious health effects which might develop in subjects.

Various pulmonary effects demonstrated in animal studies suggest that recovery from chronic exposure to O_3 is not complete even after an extended period. Monkeys exposed to 0.64 ppm O_3 for as long as a year (8 hr/day, 7 days/wk) continued to show either statistically significant or substantial decreases in lung function (e.g. static lung compliance) even after a 3 month post-exposure period (Wegner, 1982). This was interpreted as suggesting recovery was not complete even after three months. In

several studies, increases in lung collagen content occurred after short- and long-term exposures to 1.0 ppm O_3 (Last et al., 1979; Last and Greenberg, 1980; Last et al., 1981; Last et al., 1984b). Adult and weanling rats exposed for 6 and 13 weeks and monkeys exposed for one year to O_3 below 1.0 ppm showed increased lung collagen content, and even six weeks post-exposure weanling rats continued to show increases in collagen (Last et al., 1984b). This suggests that damage continued during the post-exposure period.

Even though the above and numerous other animal studies show structural changes caused by repeated short-term and long-term O_3 exposures, there is no evidence of emphysema in animals exposed to O_3 according to the CD (p. 9-52). However, studies of Bartlett et al. (1974) and Costa et al. (1983) provide evidence that the recoiling force of the lungs of rodents is reduced by chronic exposure to O_3. Reevaluation of three studies (P'an et al., 1972; Freeman et al., 1974; Stephens et al., 1976) cited in the 1978 criteria document (U.S. EPA, 1978), which reported emphysema in animals after prolonged exposure to <1 ppm O_3, indicates no evidence for these claims using the current definition for emphysema. No similar studies have confirmed emphysematous changes in O_3 exposed animals since 1978 (CD, p. 9-49); however, few similar studies have been reported.

The most pertinent newer research on chronic O_3 exposure is in the area of animal toxicology. Structural alterations in lung collagen have been confirmed in several studies at high O_3 concentrations. Reiser et al. (1987) have reported "abnormal" collagen synthesis during O_3 exposure. The collagen was irreversibly deposited in the lungs, suggesting that exposure to 0.61 ppm O_3 for 8 hours per day for one year may cause irreversible changes in lung collagen structure of monkeys. After exposure of rats to 0.7 ppm O_3 for 20 hours per day for 28 days, Gross and White (1986) found that collagen deposition can result in obstructive as well as restrictive changes in the lung, depending on where it is laid down. They speculate that it is

possible that the resting, nasal-breathing rats exposed to 0.7 ppm O_3 may be receiving a lower O_3 dose to the lungs than exercising, oral breathing humans exposed to lower O_3 levels. Pickrell et al. (1987) observed that rats exposed to 1.1 ppm O_3 for 19 hours per day for 11 days had a 40% increase in total lung collagen and developed interstitial pneumonia, significant weight loss, and pulmonary fibrosis based on examination 2 months after exposure was initiated. A lower level of O_3 (0.57 ppm) did not produce these effects, thus indicating that these effects may be concentration or dose dependent. Furthermore, chronic exposure of rats and monkeys to $\geq$ 0.40 ppm O_3 causes a concentration-dependent peribronchiolar inflammatory response (Barr et al., 1988; Moffatt et al., 1987).

In a chronic exposure study, Gross et al. (1989) showed that rats exposed for 12 months to an episodic profile of O_3 (2 hours at 0.25 ppm O_3 peak over 9 hours; 0.19 ppm O_3 integrated concentration) exhibited (1) functional lung changes indicative of a stiffer lung; (2) biochemical changes suggestive of increased antioxidant metabolism; and (3) no observable immunological changes. Other recent studies indicate lower levels of O_3 may be responsible for epithelial injury. Barry et al. (1985) found that rats exposed to both 0.12 and 0.25 ppm O_3 for 12 hours per day over a six week period showed statistically significant concentration dependent changes in alveolar type 1 epithelial cells suggesting that low concentrations of O_3 may cause a chronic epithelial injury in the proximal alveolar region. For chronic O_3 exposures of 0.12 - 0.30 ppm, a lesion is evident at the junction of the conducting airways and the gas exchange regions of the lungs, characterized by cell population shifts along with interstitial inflammation and thickening (Crapo et al., 1984; Barry and Crapo, 1985; Barry et al.,1985, 1988; Sherwin and Richters, 1985). This occurs without increased lung collagen (Filipowicz and McCauley, 1986; Wright et al., 1988a) unless exposure is intermittent (Tyler et al., 1988). The increased lavagable lipids found in lungs of rats (Wright et al.,

1988b) after chronic exposures to 0.15 - 0.30 ppm O_3 are consistent with cell population shifts and/or inflammation.

Exposure of monkeys by Harkema et al. (1987a,b) to 0.15 or 0.30 ppm O_3 for 6 or 90 days (8 hours/day) resulted in "quantitative changes in nasal transitional and respiratory epithelium..., ciliated cell necrosis, shortened cilia, and secretory cell hyperplasia." The authors conclude that ambient levels of O_3 can cause nasal epithelial lesions which may compromise defense mechanisms of the upper respiratory tract (Harkema et al., 1987a,b). This did not occur in a controlled exposure study of humans exposed to 0.4 ppm O_3 for 4 hours (Carson et al., 1985) despite increased neutrophils (Graham et al., 1988). The tracheal region of monkeys shows similar acute lesions as the nasal region but adaptation may occur (Hyde and Plopper, 1988).

Since CD closure, new information has become available which has improved the ability of EPA to extrapolate O_3 toxicology data to human health effects levels. An O_3 dosimetry model has been used to simulate local absorption of O_3 in the lower respiratory tracts of guinea pigs and rats (Overton et al., 1987). This model along with information on respiratory system uptake of O_3 in both rats and humans suggests that humans retain a greater fraction (97%) of inhaled O_3 than rodents (50%) (Wiester et al., 1987, 1988; Gerrity and Wiester, 1987; Gerrity, 1987; Hatch and Aissa, 1987) and enhances EPA's ability to extrapolate animal data to human health effects. According to Miller et al. (1987a,b), application of an O_3 dosimetry model to obtain intra- and interspecies dose-response curves from collective assessments of toxicological data will improve the overall risk assessment process, particularly in its quantitative aspects. While this new information indicates great strides have been made toward understanding animal-to-human dosimetry relationships, large uncertainties remain due to species sensitivity differences (Hatch et al., 1986) as evidenced by significant variations in lung tissue concentrations of antioxidant enzymes among mammalian

species (Slade et al., 1985; Bryan and Jenkinson, 1987). These differences will have to be dealt with in any animal to human extrapolation. Furthermore, extrapolation across averaging times or from high to low doses of O_3 introduces additional uncertainties into any extrapolation of these effects.

Until the degree of uncertainty associated with using animal studies to estimate human effects and effect levels is substantially reduced or methods are devised to study humans during long-term exposures to O_3, the relationship between acute and chronic response to O_3 will remain unclear.

4. Effects of Other Photochemical Oxidants

It has been postulated that oxidants such as peroxy acetyl nitrate (PAN) and hydrogen peroxide (H_2O_2) may play a role in producing health effects associated with photochemical oxidant exposures. However, relatively few controlled human exposure or animal toxicology studies routinely have investigated these pollutants, and field and epidemiology studies evaluate mixtures of pollutants thus making it difficult to judge which oxidant caused effects.

No significant effects have been reported in controlled exposures of intermittently exercising healthy young and middle-aged males to PAN concentrations of 0.25 to 0.30 ppm (Drinkwater et al., 1974; Raven et al., 1974a,b, 1976; Gliner et al., 1975). Only one study suggested a possible simultaneous effect of PAN (.3 ppm) and O_3 (.45 ppm) (Drechsler-Parks et al., 1984), but as with the other studies, this occurred at PAN concentrations much higher than those reported for relatively high oxidant areas (0.047 ppm). Except for an association of PAN with eye irritation (Okawada et al., 1979; Altshuller, 1977; Javitz et al., 1983; U.S. EPA, 1978; National Research Council, 1977) few effects of PAN have been reported. Field and epidemiological studies also have reported few relationships between health effects and PAN, while animal toxicology studies suggest that only very high PAN concentrations produce effects in animals,

such as significant alterations in host pulmonary defenses (Thomas et al., 1979, 1981). Review of the literature on effects of PAN can be found in the CD (pp. 9-205 to 9-306 and 10-80 to 10-84).

Even fewer studies on the health effects of H_2O_2 are available. No significant effects were observed in rats exposed for 7 days to 0.5 ppm H_2O_2 in the presence of ammonium sulfate; it is generally assumed to not penetrate into alveolar regions possibly due to the high solubility of H_2O_2 (Last et al., 1982). Other studies are of little more than mechanistic value.

In conclusion, the limited evidence available suggests that at levels found in ambient air, PAN and H_2O_2 are not responsible for adverse respiratory effects of photochemical air pollution. Ozone is considered to be chiefly responsible for the adverse health effects of oxidants largely due to the relative abundance compared to other oxidants (CD, pp. 12-65 to 12-66).

5. Interactions with Other Pollutants

Although controlled human exposure studies have not demonstrated consistently any enhancement of respiratory effects for O_3 when combined with SO_2, NO_2, CO, and H_2SO_4 or other particulate aerosols, animal toxicology research suggests additive or possibly synergistic effects (CD, p. 12-67). Controlled exposure studies of animals provide evidence that O_3 in combination with NO_2 increases susceptibility to bacterial infection (Ehrlich et al., 1977, 1979; Ehrlich, 1980) and morphological lesions (Freeman et al., 1974). Exposure to O_3 and H_2SO_4 has been reported to produce additive or even synergistic effects in host defense mechanisms (Gardner et al., 1977; Last and Cross, 1978; Grose et al., 1982) and collagen synthesis (Last et al., 1983). Mixtures of O_3 and $(NH_4)_2SO_4$ have produced synergistic effects on collagen synthesis and morphometry (Last et al., 1983, 1984a).

Direct comparison was made of exercising athletes exposed to equivalent O_3 levels in a chamber containing either oxidant

polluted air or O_3 in purified air (Avol et al., 1984). Results of the study showed no differences thus suggesting that O_3 is largely responsible for respiratory effects in oxidant-polluted air. However, it should be noted that combinations of oxidants with O_3 may contribute to decreased pulmonary function and increased symptomatic effects in asthmatics (Whittemore and Korn, 1980; Linn et al., 1980, 1983a; Lebowitz et al., 1982, 1983, 1985; Lebowitz, 1984; Holguin et al., 1985) and in children and adolescents (Lippmann et al., 1983; Lebowitz et al., 1982, 1983, 1985; Bock et al., 1985; Lioy et al., 1985). Interactions between O_3 and total suspended particulate matter were reported for decreased expiratory flow in children (Lebowitz et al., 1982, 1983, 1985; Lebowitz, 1984) and in adults with airway obstructive disease (Lebowitz et al., 1982, 1983). Thus it appears reasonable to conclude that O_3 may cause most of the respiratory effects in oxidant-polluted air, but effects may be exacerbated by other pollutants. Because efforts to examine pollutant interactions have been incomplete in clinical and epidemiological studies thus far, the potential for pollutant interactions warrants emphasis in the consideration of the margin of safety.

VIII. Staff Conclusions and Recommendations for Ozone Primary Standard(s)

Drawing upon the evaluation of scientific information contained in the CD and the CDS, which reviews new data made available since CASAC closure on the CD, this section provides preliminary staff conclusions and recommendations for consideration by the Administrator in selecting a pollutant indicator, averaging time, form, and level of the primary O_3 standard. Staff conclusions and recommendations contained herein are based upon the totality of scientific information and diverse effects reported in clinical, epidemiology, and toxicology research.

A. Pollutant Indicator

When the Environmental Protection Agency promulgated the NAAQS for photochemical oxidants in the Federal Register (36 FR 8186) on April 30, 1971, the scientific data base for health effects was very limited. The Schoettlin and Landau (1961) study, which associated increased incidence of asthma attacks with ambient photochemical oxidants, served an important role in developing the basis for the primary photochemical oxidants standard. Subsequent health research, however, indicated that O_3 was the predominant oxidant of concern for public health, and on February 8, 1979, the chemical designation of the primary standard was changed from photochemical oxidants to O_3 (44 FR 8202).

Since 1979, a substantial human health effects research base has established O_3 as being "...chiefly responsible for the adverse effects of photochemical air pollutants, largely because of its relative abundance compared to other photochemical oxidants (CD, p. 12-65)." As discussed in section VII.B.3. of the staff paper and section 12.6 of the CD, relatively few controlled human studies have investigated the health significance of peroxyacetyl nitrate (PAN) or hydrogen peroxide

(H_2O_2). Of the controlled human exposure studies of PAN, only one (Drechsler-Parks et al., 1984) suggested a possible simultaneous effect of O_3 and PAN. Other controlled studies have reported no significant effects for PAN exposures of 0.25 to 0.30 ppm, much higher than PAN levels commonly reported in high oxidant areas (CD, p. 12-65). Because H_2O_2 is highly soluble in aqueous media, it is believed that H_2O_2 deposits on upper airway surfaces rather than penetrating to the alveolar region (Last et al., 1982). However, investigations of H_2O_2 effects in the alveolar region have not yet been reported.

Regarding interactions with other pollutants, the CD (p. 12-67) has concluded that O_3 alone is considered responsible for observed respiratory effects reported in controlled human exposures of O_3 with SO_2, NO_2, CO, and H_2SO_4 or other particulate aerosols. Animal toxicology studies, however, have produced varied results, depending on the pollutant combination evaluated and the variable measured. Additive and/or possibly synergistic effects have been described from exposure to O_3 and NO_2 (e.g., increased susceptibility to bacterial infection, morphological lesions) and from exposure to O_3 and H_2SO_4 (e.g., host defenses effects and collagen synthesis). Although a controlled human O_3 exposure vs. ambient oxidant exposure comparison study indicated that O_3 was the principle cause of respiratory effects, several epidemiology studies suggest that combinations of oxidants may contribute to such effects as decreased lung function and exacerbation of symptoms in asthmatics and in children and young adults (CD, p. 12-68).

Despite the apparent interactions of O_3 with other pollutants as reported in toxicology and epidemiology studies, controlled human exposure and field studies have not consistently demonstrated that respiratory effects observed in combined exposure studies of O_3 and other pollutants are caused by any pollutant(s) other than O_3. This divergence in the data base does not reject the hypothesis that some portion of the human respiratory effects associated with exposure to photochemical

oxidants may be attributed to pollutants other than O_3. However, until further human research is performed to support or refute this hypothesis, the staff believe the case for controlling O_3 as a surrogate for protecting public health from human exposure to O_3 and other photochemical oxidants remains valid.

The question of whether O_3 can serve as an abatement surrogate for controlling other photochemical oxidants has been addressed in the CD (p. 12-15) with a quote from Altshuller (1983) who concluded that "... ambient air measurements indicate that O_3 may serve directionally but cannot be expected to serve quantitatively as a surrogate for the other products." This conclusion appears to apply to the subset of photochemical products of concern - O_3, PAN, PPN, and H_2O_2 - identified in the CD, even though Altshuller (1983) examined the use of O_3 as an abatement surrogate for all photochemical products. Lack of a quantitative, monotonic relationship between O_3 and other photochemical oxidants is discussed in Chapter 5 of the CD and demonstrated in Table 12-2 of the CD in which average PAN/O_3 ratios for different sites and years vary from 2 to 9. In addition, it is emphasized in the CD (p. 12-17) that no single measurement methodology can quantitatively and reliably measure all photochemical oxidants, either individually or in ambient air mixtures.

In spite of the above limitations, it is generally recognized that control of ambient O_3 levels currently provides the best means of controlling photochemical oxidants of potential health concern (O_3, PAN, PPN, and H_2O_2). This recognition along with a controlled-exposure human health data base, which implicates only O_3 among the photochemical oxidants at levels reported in ambient air, supports the recommendation that O_3 be retained as the pollutant indicator for controlling ambient concentrations of photochemical oxidants. In addition, using O_3 as a surrogate for oxidant control deemphasizes the need for monitoring of PAN, H_2O_2 and other oxidants. Unless significant additional evidence which demonstrates human health effects from

exposure to ambient levels of non-O_3 oxidants becomes available, it is the staff's recommendation that O_3 remain as the surrogate for protection of public health from exposure to all photochemical oxidants.

B. Form of the Standard

The current primary O_3 NAAQS is expressed as an hourly average which is the concentration not to be exceeded on more than 1 day per year on average. During the last standard review the decision was made to change from the deterministic to the statistical form of the standard. The deterministic form, which permitted only a single hourly exceedance of the standard level in any given year, did not adequately deal with the inherent variability in hourly O_3 concentrations due to the stochastic nature of meteorological factors affecting formation and dispersion of O_3 in the atmosphere. In addition, EPA further modified the standard so that one expected exceedance would be given a daily interpretation; that is, a day with two or more hourly values over the standard level counts as one exceedance of the standard level rather than two. These changes reduced the magnitude and increase stability of the "design value" (or characteristic highest concentration; see p. IV-2) used to evaluate precontrol air quality (Hayes et al., 1984).

If it is desired to further increase stability of the design value indicator by allowing more exceedances, the standard level would have to be reduced to preserve equality of protection for a multiple exceedances standard formulation. For example, going to a 5 expected exceedances standard would require a reduction in the standard level to the range of 0.09 to 0.11 ppm to maintain the same level of protection as the current 0.12 ppm O_3 NAAQS. (A 5 exceedances standard has a design value 80% lower than a 1 exceedance standard, on average, but there is variability in this relationship among urban areas.)

Since a multiple expected exceedances formulation for 0.12 ppm O_3 does not provide as much protection against short-term

peak concentrations as does the current O_3 NAAQS standard, it is recommended that the 1 expected exceedances form of the current O_3 NAAQS be retained for a short-term primary O_3 standard.

C. Averaging Time(s)

Exposure durations for studies reporting effects at or near ambient O_3 levels fall into three general categories--short-term (1 to 3 hours), prolonged (6 to 8 hours), and chronic (months to years). Controlled chamber and field studies of acute pulmonary effects of O_3 have reported statistically significant impairment of group mean lung function at O_3 concentrations $\leq$ 0.20 ppm for exposure durations of 1 to 2 hours (McDonnell et. al., 1983, 1985a,b,c; Kulle et al., 1985; Folinsbee et. al., 1984; Avol et al., 1983, 1984, 1985a,b; Linn et al., 1980, 1983a,b; Adams and Schelegle, 1983; Gong et al., 1986; Linn et al., 1986; Schelegle and Adams, 1986). Prolonged and chronic exposure studies have reported a variety of pulmonary and extrapulmonary effects for O_3 exposure durations ranging from less than a day to more than a year, but for ethical reasons chronic studies have been performed using only animal subjects.

As was the case during the 1978 review of the ambient O_3 standards, acute effects of O_3 documented in controlled human studies continue to provide the most quantitative and strongest support for a short-term primary O_3 standard (Table VII-1, staff paper section VII.A.). Although O_3 exposures continue in these studies for 1 to 2 hours, periods of intermittent exercise during exposure at 15 to 30 minute intervals have caused effects to occur at much lower O_3 levels than for resting subjects. It has been demonstrated that maximum impairment of lung function occurs during or immediately after the exercise period (Folinsbee et. al., 1977a,b). Although many individuals may exercise and work outdoors for extended periods, most do not exercise heavily for prolonged periods; therefore, the maximum impact of O_3 for most individuals probably occurs after a relatively short time period. While consideration may be given to standards with averaging

times of less than one hour, such a standard does not appear to be warranted. As shown in Section V, all of the alternative O_3 standards investigated reduce the probability of experiencing a high acute O_3 exposure.

With regard to the need for a separate longer-term (6-8 hr) primary O_3 standard, recent controlled human exposure, epidemiology and toxicology studies provide evidence of increased respiratory impairment caused by multi-hour O_3 exposures. Preliminary results of controlled human exposure (6.6 hour) studies indicate that O_3 levels as low as 0.08 ppm produce statistically significant lung function decrements and respiratory symptoms (Folinsbee et al., 1988; Horstman et al., 1988a,b). In a related study suggestive evidence of inflammation was reported in humans exposed for 6.6 hours to 0.10 ppm (Koren et al., 1988a,b,c).

In the case of epidemiology studies interpretation of exposure averaging times associated with this effect remains uncertain. Associations have been reported between ambient photochemical oxidant levels and both asthma attack rates (Whittemore and Korn, 1980; Holguin et al., 1985), increased respiratory hospital admissions (Bates and Sizto, 1983, 1987), and lung function decrements (Lebowitz et al., 1983; Lebowitz, 1984; Lippmann et al., 1983; Bock et al., 1985; Lioy et al., 1985). In spite of the inherent exposure and environmental uncertainties of these studies, they provide evidence of more serious health effects associated with longer-term exposures than the apparently transitory effects reported for 1 to 2 hour exposures in most controlled exposure studies. For example, Lioy et al. (1985) have reported that healthy, active children (age 7-13) experience "a persistent decrement in function lasting for as much as a week after the end of a smog period of about four days." Lioy and Dyba (1988) have proposed recently that the PEFR decrements reported in Lioy et al. (1985) may result from total O_3 dose rather than persistence of effect from one day to the next. Finally, animal studies have provided collaborating

evidence for time of exposure as an important factor in determining seriousness of effect. Until further evidence is published which confirms exacerbation of FEV_1 decrements from extended O_3 exposure or more serious effects such as inflammation and persistence of lung function effects, the basis for a separate 6 to 8 hour O_3 standard will remain inadequate.

Much uncertainty continues to exist for extrapolation to human health effects of levels and averaging times associated with chronic exposure effects reported in animal toxicology studies. Although the animal toxicology data base which documents most of the reported chronic effects provides extensive support for effects more serious than lung function decrements, uncertainties regarding dosimetry and species sensitivity must be addressed in any extrapolation to an effect level in humans. Animal studies investigating continuous and intermittent exposure to O_3 lasting weeks to years have reported changes in lung function (4 weeks to 1 year), morphology (1 week to 18 months), biochemistry (1 week to 1 year), host defenses (1 week to 3 months) and various extrapulmonary effects (up to 1 year). With respect to some of these alterations (e.g., lung function, biochemistry) the postexposure period has been reported to be one of continued damage rather than complete recovery (CD, p. 12-48). New research, recent developments in animal extrapolation, and a chronic effects risk assessment will provide a significantly improved basis for addressing the need for a separate chronic exposure standard in the future.

With respect to protection from longer-term and repeated peak exposures afforded by a 1-hour standard, Appendix A discusses relationships among air quality indicators in urban areas. These relationships have been graphically presented in Appendix A by Figures A-2 through A-4. Interpretation of these figures suggests that if the current 0.12 ppm 1-hour daily maximum O_3 standard is met by all sites in the data set, then 10% of all metropolitan statistical areas (MSAs) might have 17 or more days with an 8-hour daily maximum > 0.08 ppm O_3. Although

it is premature to suggest that 0.08 ppm O_3 is the level of concern for an 8-hour exposure, this does suggest that the current standard does permit numerous potential 8-hour exposures to 0.08 ppm O_3. On the other hand, if 0.10 ppm O_3 is determined to be the level of concern for 8-hour exposures, the current O_3 standard provides reasonable protection from multiple 8-hour exposures in most areas. The scientific basis for judgment regarding the level of concern for health effects has not been determined for 8-hour or longer-term exposures. Until adequate research has been completed and accepted for incorporation into the CD, determination of a level of concern for health effects arising from 8-hour or longer-term exposures to O_3 is premature.

In conclusion, comments made by CASAC (1986, 1987, 1988) support the need to protect the public from health effects caused by acute exposures to O_3. The staff concurs and recommends that a primary O_3 standard with a 1-hour averaging time be maintained to provide protection from acute exposures. The CASAC (1986, 1987, 1988) also discussed the need and basis for multi-hour O_3 standards; however, no consensus was reached regarding the need for alternative primary standards. New clinical, epidemiology, and toxicology research has provided additional support for the occurrence of more significant health effects resulting from 6 to 8 hour, multiple, and chronic exposures to O_3 than result from single 1-hour O_3 exposures. A CDS of new published research which addresses both acute and longer-term O_3 exposures has been prepared by the Environmental Criteria and Assessment Office (ECAO) and closure reached on the document. Staff recommends that this information be used by the Administrator in assessing the need for longer-term O_3 primary standard(s) while carefully considering the conclusions reached by CASAC in the closure letter (McClellan, 1989). Although the CASAC recognized the emerging data base on acute effects resulting from multi-hour exposures, it was the CASAC view that it would be some time before enough such information would be published and included in a criteria document. Thus, "CASAC concluded such information can

better be considered in the next review of the ozone standards" (McClellan, 1989). Also, CASAC reached "closure on the staff position paper recommending a 1-hour standard . . ." (CASAC, 1988; McClellan, 1989). With this portion of the review complete and after considering CASAC's views on all issues, the Administrator will be in a position to make a regulatory decision on how and when to best act on the 1-hour standard.

D. Level of the Primary Standard(s)

In selecting the level of the primary NAAQS for O_3, the Administrator is faced with consideration of a large and diverse health data base. Judgments regarding the scientific quality and strength of that data base already have been made in Chapters 9 to 12 of the CD. The preliminary assessment of health effects attributed to O_3 presented in Section VII of this staff paper presents a variety of health effects and discusses the seriousness of these effects. In addition to establishing a lowest observed effects level from these health data, consideration must be given to the uncertain evidence which bears on a margin of safety needed to protect public health. In addition, the Administrator must judge which of the health effects attributed to O_3 should be considered adverse before a final judgment on the level of the O_3 primary standard can be made. Although scientific literature supports the conclusion that particular O_3 concentrations and exposure patterns may pose risks to human health, scientific data can only identify the limits of a range within which a standard should be set. Specific numeric standard levels, frequency of allowable exceedances, and averaging times are largely a public policy judgment.

This section draws conclusions regarding the scientific literature used by staff to identify a lowest observed effects level for short-term health effects attributed to O_3. In addition, the more uncertain or less quantifiable evidence, which forms the basis for judgements about which standard provides an

adequate a margin of safety, is considered for use in recommending short-term O_3 standard options. Although concern has been expressed for the more serious effects reported in chronic animal exposure studies, the major uncertainties associated with dosimetry and species sensitivity differences limit direct quantitative extrapolation of effects in animals to human health effects at this time.

The strongest evidence of human health effects from acute exposure to O_3 comes from controlled human exposure studies because O_3 exposures are known accurately, other pollutants are not present, temperature and humidity are monitored, and human subjects are used. Field studies also provide strong evidence for similar reasons with the exception that other ambient pollutants are present during exposure. This exception, however, can provide support for effects occurring under real ambient conditions. The ethical limitations of controlled human exposure and field studies (e.g. restriction to short-term exposures, non-invasive techniques, and limited use of infectious agents) have prevented investigation in humans of the more serious chronic exposure effects which have been reported in animal toxicology studies. Field and epidemiology studies have the advantage of providing associations between health effects and "real world" ambient exposures to photochemical oxidant pollution; however, available epidemiology studies provide less certain exposure-response evidence for O_3 than the controlled- exposure and field studies. As a result, epidemiology data tend to be relied on less quantitatively in establishing a lowest observed effects level for O_3. Epidemiology data must be given consideration in developing the level of the NAAQS because they do provide evidence of effects experienced in more realistic environments than the artificial chamber exposure studies.

Staff observations, conclusions, and recommendations regarding health effects of O_3 are presented below. They are based upon the scientific review in chapters 9 to 12 of the CD as well as in the CDS and the preliminary assessment and analyses

discussed in Section VII of this staff paper. These conclusions are as follows:

1. Statistically significant group mean decrements in FEV_1, representing alterations in lung function, have been demonstrated in some but not all following one and two hour exposures of healthy, exercising children, adolescents and young adults to O_3 concentrations in the range of 0.12 to 0.16 ppm. As summarized in Table VII-1, statistically significant FEV_1 decrements have been reported in controlled exposure studies of intermittently, heavily exercising, healthy children and adults exposed for two hours to 0.12 ppm O_3 and of continuously heavily exercising, healthy adults exposed for one hour to 0.16 (McDonnell et al., 1983; 1985b; Avol et al., 1984). Field studies have demonstrated that statistically significant group mean FEV_1 decrements have been induced in continuously heavily exercising, healthy adolescents at mean ambient O_3 levels of 0.144 ppm and in continuously heavily exercising, healthy adults at mean ambient O_3 levels of 0.153 and 0.165 ppm (Avol et al., 1985a,b; Avol et al., 1984; Avol et al., 1983; Linn et al., 1983a).

2. Estimates of the fraction of heavily exercising adults experiencing > 10 % FEV_1 decrements when exposed to 0.12 ppm O_3 range from 2 to 14% (Figure VII-2). These estimates are based upon an analysis (Hayes et al., 1987b) of data obtained from individual subjects in three controlled exposure human studies (Avol et al., 1984; Kulle et al., 1985; McDonnell et al., 1983). Because a potentially large group of the population may experience a substantial reduction (>10%) of FEV_1 when exposed to 0.12 ppm O_3, this O_3 level should be considered as the lowest observed effects level (LOEL) but not necessarily the lowest observed adverse effects level (LOAEL) based on the staff's recommended definition of adverse effects in Section VII.C.1. which suggests that "mild" individual responses not be considered adverse.

3. Despite a close association reported (CD, p. 12-17) between changes in group mean FEV_1 and occurrence of group mean respiratory symptoms for acute O_3 exposures of adults in chambers, Hayes et al. (1987b) report only a weak-to-moderate correlation between FEV_1 changes and symptoms severity when the analysis is conducted using individual data. One reason that group mean analysis outcomes are different from results of individual data analysis may be that symptoms reported are inherently more subjective than FEV_1 decrements measured; also some individuals may experience symptomatic effects without notable changes in FEV_1 or vice versa due to differences in mechanisms causing FEV_1 decrements versus symptomatic effects.

4. Estimates of the fraction of heavily exercising adults experiencing moderate or severe chest discomfort ranged from 0 to 15% and those experiencing moderate or severe cough ranged from 0-13% when exposed to 0.12 ppm O_3 (Figure VII-3). These estimates are based upon an analysis (Hayes et al., 1987b of data obtained from individual subjects in two controlled human studies (Kulle et al., 1985; McDonnell et al., 1983).

5. The pulmonary function and symptom risk assessment for alternative O_3 hourly NAAQS (Hayes et al., 1987b; Whitfield, 1988) is one of the factors that should be considered in selecting an hourly primary NAAQS that provides an adequate margin of safety. From an air quality perspective without regard to personal exposure patterns, the benchmark risk estimates show probabilities from 0.5 to 1.0 that O_3 concentrations will exceed levels sufficient to cause $\geq$ 10% decrements in FEV_1 or $\geq$ mild symptoms (cough and chest discomfort) in 1,5, or 10% of heavily exercising individuals upon attainment of a 0.08, 0.10, or 0.12 ppm O_3 NAAQS. Benchmark risk estimates are $\leq$ 0.2 for two of the three health data bases used when the endpoint is $\geq$20% FEV_1 decrements or moderate/severe respiratory symptoms. When personal exposure patterns are considered, mean expected

headcount estimates range from 0.5 to 18.8% of the heavily exercising population for ≥ 10% FEV_1 decrements or ≥ mild respiratory symptoms depending upon health data base used and urban area examined upon attainment of a 0.12 ppm O_3 NAAQS. Mean expected headcount estimates drop to 0.1 to 7.1% of the heavily exercising population for ≥ 20% FEV_1 decrements or moderate/severe respiratory symptoms. The reader is referred to Table VII-4 for a more detailed summary of expected headcount estimates and to the discussion on pp. VII-41-43 of caveats and limitations that should be considered in interpreting the risk estimates.

6. Preliminary results indicate that prolonged (6 to 8-hour) exposures to O_3 may cause enhanced lung function decrements and respiratory symptoms as well as more persistent respiratory effects than the lung function decrements produced by single one- and two-hour exposures to O_3. Preliminary evidence of statistically significant lung function decrements and respiratory symptoms has been reported for exercising healthy persons exposed to O_3 levels of 0.08-0.12 ppm for 6.6 hours (Folinsbee et al., 1988; Horstmann et al., 1988a,b). Similar exposure protocols produced suggestive evidence of inflammation at 0.10 ppm O_3 (Koren et al., 1989a,b,c). Persistence of lung function response in children, as measured by peak expiratory flow rate (PEFR), has been reported to last for as long as a week following a smog period of about four days duration in which the O_3 concentration peaks were in the range of 0.120 to 0.185 ppm (Lioy et al., 1985). While the respiratory responses reported above appear to be primarily caused by O_3, the authors suggest that other pollutants such as acid sulfates may have contributed to the persistent lung function effects. Also, Lioy and Dyba (1988) have proposed recently that the PEFR decrements reported in Lioy et al. (1985) resulted from total O_3 dose rather than persistence of the effect from one day to the next. Although it appears to be premature to draw firm conclusions regarding

persistent or enhanced respiratory effects associated with 6 to 8 hour or longer term exposures to ambient O_3, the staff concludes that there is reason for concern about enhanced respiratory function changes beyond the transient pulmonary function decrements thus far reported in most controlled human exposure and field studies. The emerging data base of clinical and epidemiology research suggests that standards with longer-term averaging times may need to be considered as alternatives or additions to the current 1-hour averaging time primary standard after adequate information is available to make an informed judgment regarding need for such standards.

7. Work performance currently appears to be limited by exposure to O_3; however, too small a data base is available to quantify the magnitude of this impairment and evidence regarding this effect should be used only to develop an adequate margin of safety. Results of exposure to O_3 during high exercise levels indicate that discomfort may be an important factor in limiting performance (Adams and Schelegle, 1983; Folinsbee et al., 1984; Gong et al., 1986; Schelegle and Adams, 1986).

> Subjective statements by individuals engaged in various sport activities indicate that these individuals may voluntarily limit strenuous exercise during high-oxidant concentrations. However, increased ambient temperature and relative humidity are also associated with episodes of high-oxidant concentrations, and these environmental conditions may also enhance subjective symptoms and physiological impairment during O_3 exposure (CD, p. 10-65.)

While it may be difficult to differentiate performance effects caused by O_3 from those caused by other environmental conditions, exacerbation of effects caused by O_3, beyond those caused by other conditions, may prevent or curtail normal activities and should be viewed as adversely affecting individual performance; however, limitations of the data base suggest using this information only in margin of safety considerations.

8. Based on the CDS review of new clinical evidence, both allergic and asthmatic subjects have a greater increase in airway resistance after O_3 exposure than do healthy subjects (CDS, p. 64). Epidemiological data provides additional qualitative evidence of exacerbation of asthma in adults at ambient O_3 concentrations below those generally associated with symptoms or functional changes in most healthy adults. Given the nature of these data, they should be used in developing an adequate margin of safety.

> Whittemore and Korn (1980) and Holguin et al. (1985) found small increases in the probability of asthma attacks associated with previous attacks, decreased temperature, and incremental increases in oxidant and O_3 concentrations, respectively. Lebowitz et al. (1982, 1983) and Lebowitz (1984) also showed effects in asthmatics, such as decreased peak expiratory flow and increased respiratory symptoms, that were related to the interaction of O_3 and temperature. (CD, p. 12-55.)

Uncertainties concerning individual exposure and confounding environmental variables limit developing exposure-response relationships with these epidemiological data at this time and staff recommends that these data should be used to develop an adequate margin of safety. However, if it is demonstrated that an increased incidence of asthma attacks occurs as a function of O_3 exposure, this would be an effect of great medical significance and would require careful consideration.

9. Lung structure damage induced by long-term exposures to O_3 has been demonstrated in several animal species. These data should be used in establishing an adequate margin of safety. Significant morphological alterations in the centriacinar region have been reported after long-term O_3 exposures of rats (Barry et al., 1983; Crapo et al., 1984), monkeys (Eustis et al., 1981; Fujinaka et al., 1985) and dogs (Freeman et al., 1973). "There is morphometric (Fujinaka et al., 1985), morphologic (Freeman et

al., 1973), and functional evidence (Costa et al., 1983; Wegner, 1982) of distal airway narrowing" (CD, p. 12-48). Inflammation of the centriacinar region thus appears to be correlated with lung structure changes (Moore and Schwartz, 1981; Fujinaka et al., 1985) and increased lung collagen content (Last et al., 1979; 1984b; Boorman et al., 1980). This latter effect is thought to suggest lung fibrosis. The previous criteria document (U.S. EPA, 1978) cited three studies which reported emphysema in animals exposed for long periods to <1 ppm O_3; however, reevaluation based on the definition for emphysema used in the current CD does not suggest any evidence for such claims (CD, p. 12-49). The animal toxicology data should be included in establishing an adequate margin of safety.

10. Alterations of the host defense systems have been reported in animals acutely and subchronically exposed to O_3 and provide suggestive evidence for similar effects in humans. Respiratory systems of mammals are normally protected from bacterial and viral infections by the closely integrated particle removal and immunological defense systems.

> In these studies, short-term (3 hr) exposure to O_3 at concentrations of 0.08 to 0.10 ppm can increase the incidence of mortality from bacterial pneumonia (Coffin et al., 1967; Ehrlich et al., 1977; Miller et al., 1978a). Subchronic exposures to 0.1 ppm caused similar effects (Aranyi et al., 1983). (CD, p. 12-49.)

While it is generally accepted that the basic mechanisms of action are similar in animals and humans, a more meaningful endpoint in humans would be increased prevalence of acute respiratory disease in the community. It should be noted that a number of other factors may influence the O_3 levels at which respiratory disease becomes apparent (e.g., nutrition, stress, preexisting disease). Despite these factors and a lack of experimental confirmation in humans, "... it is possible that

humans exposed to O_3 could experience decrements in host defenses; but at the present time, the exact concentration at which effects might occur in man cannot be predicted, nor can the severity of the effect" (CD, p. 12-50). The staff recommends that these data be used in selecting a standard which provides an adequate margin of safety.

11. Extrapulmonary effects of O_3 reported in animal studies include alterations in red blood cell structure and enzyme activity, cytogenetic effects, and limitations in vigilance, in addition to limited evidence for cardiovascular, reproductive, teratological, mutagenic, endocrine system, and liver metabolism effects. Until further analysis better establishes the physiological significance of these effects on human health, the staff recommends that these effects data be used in selecting a standard which provides an adequate margin of safety.

12. Factors which have been demonstrated to affect susceptibility to O_3 exposure are activity level and environmental stress (e.g., humidity, high temperature). Those factors which either have not been adequately tested or remain uncertain include age, sex, nutrition, and smoking status. The Administrator should consider taking them into account in establishing an adequate margin of safety.

13. Attenuation of acute pulmonary function response caused by repeated daily exposures to O_3 is a well-established phenomenon, which should be viewed with concern for potentially increasing dose of O_3 to the lungs. Functional decrements are greater on the second exposure day, with smaller decrements on each successive day until the fourth or fifth day when small or no decrements are observed (Farrell et al., 1979; Horvath et al., 1981; Kulle et al., 1982b; Linn et al., 1982). Attenuation of functional response, however, does not necessarily imply attenuation of morphological or biochemical response to O_3.

> There is an interplay between tissue inflammation, hyperresponsiveness, ensuing injury (damage), repair processes, and changes in response. The initial response followed by its attenuation may be viewed either as sequential states in a continuing process of lung injury and repair or as a physiological adaptation to the irritative stimulus (CD, p. 12-42).

Attenuation of pulmonary function or symptomatic effects, therefore, should be seen as a human response of concern because it may result in an increase in the total O_3 dose reaching the lungs by permitting individuals to exercise more heavily outdoors when ambient O_3 levels are high, thus resulting in continued, and potentially greater, lung injury. However, the possibility exists that this response may indicate the body's ability to adapt to increased O_3 levels. Attenuation should, therefore, be considered in developing an adequate margin of safety.

14. Exacerbation of respiratory effects by interaction of other pollutants with O_3 has not been demonstrated in controlled human exposure or field studies; however, epidemiology studies suggest that respiratory effects reported near the O_3 standard may be caused by O_3 in combination with other pollutants. Furthermore, animal studies suggest that combinations of O_3 with other pollutants may act additively or synergistically, depending on the pollutants and endpoints chosen for study. While the lack of individual exposure analysis may limit development of quantitative exposure-response relationships in epidemiology studies, the large body of evidence cited in the CD (p. 12-68) supports the conclusion that ambient pollutants other than O_3 may interact with O_3 to contribute to respiratory effects observed in those studies and should be considered in developing an adequate margin of safety.

15. Two groups have been identified as being "potentially at-risk" from exposure to O_3: (1) that subgroup of the general population characterized as having preexisting respiratory

disease, and (2) those individuals whose activities outdoors result in increases in minute ventilation which includes responsive individuals who experience significantly greater decrements in lung function from exposure to O_3 than the average response of groups studied, (CD, pp. 12-88 to 12-89).

In conclusion, short-term controlled exposure studies (McDonnell et al., 1983) suggest that a group mean lowest observed effects level (LOEL) for pulmonary function and symptoms for healthy, exercising subjects is 0.12 ppm O_3 for an averaging time of one to two hours. Effects somewhat below 0.12 ppm O_3 have been reported in recent camp studies (Spektor et al., 1988a,b), but it has been suggested that these effects may be due in part to interactions with other pollutants such as acid aerosols. Analysis of clinical studies indicates that a small fraction of the population may experience measurable lung function and symptomatic effects for O_3 exposures even below 0.12 ppm (Hayes et al., 1987a,b,) while some individuals show no effects at 0.12 ppm (Linn et al., 1988). Although significant lung function decrements and symptoms have been demonstrated in a single study (Horstman et al., 1988, 1989) to occur in healthy human subjects exposed for 6.6 hours to O_3 levels as low as 0.08 ppm, staff believes a larger data base is required to establish a LOEL for prolonged exposures. Uncertainty associated with assessment of epidemiology and animal data prevents identifying a lowest observed effects level for chronic exposures at this time. Factors which staff believe should be considered in developing a 1-hour standard which provides an adequate margin of safety are:

(1) the possibility that individuals with pre-existing respiratory disease may experience effects at levels below those producing effects in healthy subjects;

(2) possible exacerbation of respiratory effects by other pollutants in combination with O_3 during ambient exposures;

(3) the possibility that attenuation of pulmonary function decrements may increase O_3 dose inhaled and result in more serious effects;

(4) possible limitation of work performance by exposure to O_3;

(5) evidence of exacerbation of asthma at ambient O_3 concentrations;

(6) evidence of bronchial reactivity in subjects exposed to $\geq$ 0.3 ppm O_3;

(7) evidence indicating that repeated acute and chronic exposures to O_3 may cause lung structure damage;

(8) evidence of increased susceptibility to infection;

(9) extrapulmonary effects of O_3 which are of uncertain biological importance;

(10) potentially "at risk" groups that have not been adequately tested; and

(11) factors which may affect susceptibility to O_3.

E. Summary of Staff Recommendations

Based upon information and discussions contained in the CD the CDS, and the staff conclusions drawn in Sections VIIIA-D, the following staff recommendations regarding the primary O_3 standard are as follows:

1. In consideration of the large base of health information attributing effects to O_3 exposure and the lack of evidence which demonstrates human health effects from exposure to ambient levels of photochemical oxidants other than O_3, staff recommends that O_3 remain as the surrogate for controlling ambient concentrations of photochemical oxidants.

2. The current primary O_3 NAAQS is attained when the expected number of days per calendar year with maximum hourly average concentrations above the level of the standard is equal to or less than one (44 FR 8202). Staff recommends that these attributes of the O_3 standard be retained.

3. Because research has demonstrated that respiratory effects of public health concern are associated with acute (1 and

2 hour) exposures it is recommended that the 1-hr averaging time be retained for the O_3 primary NAAQS. Although there are studies which have identified health effects associated with O_3 exposures having longer than one-hour averaging times, there remains great uncertainty about which exposure characteristics (e.g., level, time, repeated peaks) are most important. Recent clinical research provides evidence of enhanced respiratory effects of O_3 levels below the current NAAQS level when subjects are exposed for a 6.6 hour period (Folinsbee et al., 1988; Horstman et al., 1988). Epidemiology research suggests persistent changes in lung function, aggravation of respiratory disease, and possibly increased hospital admissions, all of which are associated with O_3 levels near the current NAAQS. In addition, data reported in animal toxicology studies provide evidence of lung structure damage, increased susceptibility to respiratory infection, and a variety of extrapulmonary effects following O_3 exposures lasting from a few hours to more than a year. The seriousness of these effects indicates a need to protect public health from ambient O_3 exposures which reasonably can be expected to induce such effects in humans. This protection could be provided by setting separate 6 to 8-hour, monthly, seasonal, or annual standards, or by setting a 1-hour standard which controls longer-term exposures of concern.

Two types of data are necessary for considering the possible need for a primary O_3 standard with an averaging time of greater than 1 hour: (1) health effects data and (2) aerometric data, both empirical and statistical. Rombout et al. (1986, 1989) published air monitoring data from the Netherlands and New Jersey on relationships between the 1-hour and 8-hour averaging times. The results of their particular data sets indicate that 1-hour maximum O_3 levels do not always predict 8-hour average O_3 concentrations above 0.10 ppm and, therefore, that the daily maximum hourly O_3 standard currently in effect "... does not adequately indicate the intensity of long duration and high concentration exposures to ozone" (Rombout et al., 1986). They

concluded from their data that a longer-term averaging time, between 7 and 10 hours, would be necessary to provide adequate protection of public health and, further, that a standard should be established for the highest daily 8-hour period based upon running 8-hour averages. Consideration of the Rombout et al. (1986, 1989) analyses and conclusions must be tempered by recognition that a very limited data base was used and that extrapolating the New Jersey data to all areas of the U.S. is subject to large uncertainty.

The New Jersey data used by Rombout et al. (1986) were for specific days, at the sites chosen, on which hourly O_3 concentrations did not exceed 0.12 ppm, the level of the current primary NAAQS. It should be noted, however, that those New Jersey sites are located in a nonattainment area and that diurnal O_3 curves at those sites, therefore, are not necessarily representative of diurnal curves (i.e., the distribution of hourly O_3 concentrations over a day) observed in areas in which the standard is actually attained.

Thus, it seems prudent to the staff that the relationships among 1-hour O_3 concentrations and other potentially appropriate exposure statistics in attainment areas or under attainment scenarios must be fully explored before adopting a longer-term standard. Whether the current 1-hour primary standard can effectively serve as a surrogate for one or more longer-term averaging times depends upon concentrations of potential concern associated with longer-term exposures. Those concentrations of potential concern must first be identified before the question of surrogacy can be resolved.

OAQPS has analyzed whether it is possible to set the short-term standard at a level which reduces the probability of experiencing an unacceptable long-term exposure. As discussed in Appendix A, there is a marginally statistically significant association between short- and longer-term O_3 indices. (For example, if the current 0.12 ppm 1-hour daily maximum O_3 standard

is met by all sites in the data set, then 10% of all metropolitan statistical areas might have 17 or more days with an 8-hour daily maximum >0.08 ppm O_3.)

Although the CASAC recognized the emerging data base on acute effects resulting from multi-hour exposures, it was the CASAC view that it would be some time before enough such information would be published and included in a criteria document. Thus, "CASAC concluded such information can better be considered in the next review of the ozone standards" (McClellan, 1989). Also, CASAC reached "closure on the staff position paper recommending a 1-hour standard . . ." (CASAC, 1988; McClellan, 1989). With this portion of the review complete and after considering CASAC's views on all issues, the Administrator will be in a position to make a regulatory decision on how and when to best act on the 1-hour standard.

4. Based on the CD the CDS, the staff assessment of the acute (1-2 hour) controlled exposure, field, epidemiology, and animal toxicology data and recommendations of the Clean Air Scientific Advisory Committee (CASAC, 1986, 1987, 1988), staff recommends that the range of 1-hour O_3 levels of concern for standard-setting purposes should remain 0.08 ppm to 0.12 ppm. The upper end of this range (0.12 ppm) represents lowest O_3 exposure concentrations in controlled exposure studies in which healthy, heavily exercising children, adolescents, and young adults have experienced statistically significant, though small group mean decrements in lung function. Analyses of individual data from these studies indicates that larger individual decrements have been observed in some subjects with cumulative frequency distributions suggesting that some fraction of subjects experience FEV_1 decrements greater than 10% at 0.12 ppm O_3. Mild cough also has been reported for individuals in these studies at the upper end of the range. On the other hand, recently published acute exposure research suggests that 0.12 ppm and even 0.14 ppm O_3 may be a no effects level in some individuals for

FEV_1 and symptoms. As discussed in Section VII.C.1., staff has recommended that mild individual responses (i.e., 5-10% FEV, decrements, 30 minute recovery, mild cough, no limitation of activity) should not be considered adverse health effects. If this response is deemed to be not adverse or if 0.12 ppm is not identified as a lowest observed effects level for O_3, then it can be argued that a standard level of 0.12 ppm does provide a margin of safety. Some field and epidemiology studies provide suggestive evidence of acute lung function impairment at similar or lower exposure levels, though these studies are limited by uncertainties concerning individual exposure levels and presence of other pollutants contributing to or causing health effects measured. Other epidemiology studies have provided evidence of aggravation of asthma and airway obstructive disease, again limited by uncertainties about individual exposure and other pollutants. Given the difficulty of extrapolating these findings to the U.S. population and the fact that the effects of O_3 on individuals with preexisting respiratory disease have not yet been fully characterized, staff believes that the upper end of the above range provides a relatively small if any margin of safety. These uncertainties and the nature of potential effects are important considerations in developing an adequate margin of safety.

With regard to the lower end of the range, neither the evidence provided by animal toxicology studies (e.g. increased susceptibility to lung infection, lung structure and biochemical changes, and extrapulmonary effects) nor other epidemiological evidence suggesting respiratory effects in children and susceptible groups provides scientific support for health effects below 0.08 ppm for short-term (1-2 hour) exposures. These data as well as estimates provided by exposure and risk analyses will be considered in evaluating the margin of safety provided by alternative 1-hour standards in the range of 0.08 to 0.12 ppm.

Health effects information discussed in Chapter VII of the staff paper and the CDS provides reason for concern about multi-

hour exposure (6-8 hours) of humans to O_3. Recently published clinical and epidemiology research suggests that extended exposure of exercising individuals to 0.08 to 0.12 ppm O_3 may produce biologically important, as well as statistically significant, lung function decrements, respiratory symptoms and inflammation. Epidemiology evidence further indicates a possible association of ambient O_3 and other pollutants with increased respiratory hospital admissions over an extended summertime exposure. Animal toxicology data show reversible epithelial injury in the lungs following sub-chronic exposure of animals to O_3 levels as low as 0.12 ppm; structural alterations in the lung and irreversible deposition of collagen in the lungs previously have been reported in animals chronically exposed to much higher levels (0.61 ppm).

Based on the health data base discussed briefly above, EPA has considered alternatives in addition to the existing 1-hour primary O_3 standard. One obvious alternative is to provide public health protection from prolonged O_3 exposures by setting a separate longer-term primary standard or standards. Great uncertainty exists at this time, however, regarding either appropriate averaging times or levels of concern. Another alternative under consideration is to set a 1-hour O_3 standard which provides adequate protection from longer-term exposures. The current 1-hour primary standard of 0.12 ppm O_3 is approximately equivalent to a 0.1 ppm, 8-hour standard. If it should be determined that protection of public health requires limiting 8-hour exposures to lower levels, then it would probably be necessary to tighten the 1-hour standard, to be useful as a surrogate for protection from longer exposures. Again, however, this decision will continue to be premature until new research has been fully evaluated and formally incorporated during the next review cycle in a criteria document because such a change would be based upon the need for protection against prolonged exposure effects.

Because there is a good health effects data base available on 1-2 hour exposures, the staff concurs with CASAC that review of this scientific information, including the staff paper and its assessment of this information be closed out. With this portion of the review complete, and after considering CASAC's views on all issues, the Administrator will be in a position to make a regulatory decision on how and when to best act on the 1-hour standard.

IX. Factors Relevant to the Review of the Secondary Standard for Ozone

This section reviews and assesses research on welfare effects attributed to O_3 and other photochemical oxidants as summarized in Air Quality Criteria for Ozone and Other Photochemical Oxidants (CD). Where possible, judgments are provided which identify adverse effects and levels of these effects for secondary-standard setting.

Important categories with respect to welfare include agricultural vegetation effects (e.g., yield loss), natural ecosystem effects, personal comfort and well-being effects, and materials damage. Because the possible indirect contributions of the photochemical oxidants to visibility degradation, climatic changes, and acidic deposition cannot at present be quantified, these atmospheric effects and phenomena are not addressed in this document (CD, p. 1-1). They have been addressed, however, in other recent air quality documents (U.S. EPA, 1982a,b). The approach taken in this staff paper is: 1) to describe each type of O_3 effect, 2) to discuss what is known regarding exposure-response relationships of O_3 exposure, and 3) to evaluate the factors which should be considered in selecting the level, averaging time and form of the secondary NAAQS for O_3. Discussions of the status of the rural O_3 exposure analysis, the O_3 economic analyses, and the welfare risk assessment are included.

A. Mechanisms of Action for Vegetation

The responses of vascular plants to O_3 may be viewed as the culmination of a sequence of physical, biochemical and physiological events. For ambient O_3 to exert a phytotoxic effect, it must first diffuse into the plant. Such an effect will occur only if a sufficient amount of O_3 reaches the sensitive cellular sites within the leaf. Ozone from the ambient air enters into the leaf space through the stomata, which can exert some control on O_3 uptake to the active sites within the

leaf. Once within the leaf, O_3 quickly dissolves in the aqueous layer on the cells lining the air spaces. Ozone or its decomposition products then diffuse through the cell wall and membrane into the cell, where it may affect cellular or organellar processes. Ozone injury will not occur if 1) the rate of O_3 uptake is sufficiently small so that the plant is able to detoxify O_3 or its metabolites or 2) the plant is able to repair or compensate for the O_3 impacts (Tingey and Taylor, 1982). The uptake and movement of O_3 to the sensitive cellular sites are subject to various physiological and biochemical controls (CD, p. 6-22).

At any point along this pathway, O_3 or its decomposition products may react with cellular components. Altered cell structure and function may result in changes in membrane permeability, carbon dioxide fixation, and many secondary metabolic processes (Tingey and Taylor, 1982). The magnitude of the O_3-induced effects will depend upon the physical environment and the chemical environment of the plant (including other gaseous air pollutants and a wide variety of chemicals), and biological factors (including genetic potential, developmental age of the plant and interaction with plant pests). Cellular injury manifests itself in a number of ways, including foliar injury, premature senescence, reduced yield or growth or both, reduced plant vigor, and sometimes death. Depending upon the intended use of a plant species (for food, forage, fiber, shelter, or amenity), any of these effects discussed above could constitute a "welfare" effect, with potentially adverse effects on society.

1. Biochemical Response

When O_3 passes into the liquid phase, it undergoes transformations that yield a variety of free radicals (e.g., superoxide and hydroxyl radicals). Whether these species result from decomposition of O_3 or reactions between O_3 and biochemicals in the extracellular fluid has not been determined. Ozone or its

decomposition products, or both, will then react with cellular components, resulting in structural and/or functional effects.

The potential for O_3, directly or indirectly, to oxidize biochemicals in vitro has been demonstrated. Ozone can oxidize several classes of biochemicals including nucleotides, proteins, some amino acids and various lipids. Data acquired from in vitro studies are best utilized to demonstrate that many cellular constituents are susceptible to oxidation by O_3. New approaches are needed to assess the full range of in vivo biochemical changes caused by O_3. (See CD, Section 6.3.1.3 for further details.)

2. Physiological Responses

Physiological responses have been more useful than biochemical changes in characterizing cell responses to oxidants (CD, p. 6-26). Many consider membranes to be the primary site of action of O_3 (Heath, 1980; Tingey and Taylor, 1982). Whether the plasma membrane or some organelle membrane is the primary site of O_3 action is open to speculation (Tingey and Taylor, 1982). The alteration in plasma membrane function, however, is clearly an early step in a series of O_3-induced events that eventually leads to leaf injury and subsequent yield loss.

The effect of O_3 on key steps in photosynthesis has been measured for several plant species as shown in Table IX-1 (CD, 6-28). Reductions in photosynthesis may reflect the direct impairment of chloroplast function or reduced CO_2 uptake resulting from O_3-induced stomatal closure, or both. Regardless of the mechanism, a sustained reduction in photosynthesis will ultimately affect growth, yield, and vigor of plants. Some examples of O_3-induced reduction in apparent photosynthesis at concentrations above and below 0.25 ppm are presented in Table IX-1. These data highlight the potential of O_3 to reduce primary productivity at various air quality levels, some of which are close to ambient.

TABLE IX-1. EFFECT OF OZONE ON PHOTOSYNTHESIS

Species	O_3 concentration, ppm[a]	Exposure duration	% inhibition	Reference
Loblolly pine	0.05	18 wk continuously	15[b]	Barnes (1972a)
Slash pine	0.05	18 wk continuously	9[b]	Barnes (1972a)
Bean	0.072	4 hr/day for 18 days	18[b]	Coyne and Bingham (1978)
Alfalfa	0.10	1 hr	4[b]	Bennett and Hill (1974)
	0.20	1 hr	10[b]	
Ponderosa pine	0.15	9 hr daily/ 60 days	25[c]	Miller et al. (1969)
	0.30	9 hr daily/ 30 days	67[c]	
Eastern white pine	0.15	19 days	10[c]	Barnes (1972a)
Eastern white pine	0.10	4 hr/day for 50 days	24[b]	Yang et al. (1983)
Sensitive	0.20	4 hr/day for 50 days	42[b]	
	0.30	4 hr/day for 50 days	51[b]	
Intermediate	0.10	4 hr/day for 50 days	Not sig. different	
	0.20	4 hr daily/50 days	14[b]	
	0.30	4 hr daily/50 days	20[b]	
Bean	0.30	3 hr	22[c]	Pell and Brennan (1973)
Black oak	0.50	4 hr daily/2 days	30 ± 10[d]	Carlson (1979)
Sugar maple	0.50	4 hr daily/2 days	21 ± 10[d]	Carlson (1979)
White pine				
Sensitive	0.7 or 0.9	3.0 or 10	100[b]	Botkin et al. (1972)
Tolerant	0.70 to 0.95	10/30 days	0[b]	
Poplar hybrid	0.90	1.5 hr	60[e]	Furukawa and Kadota (19[illegible])
Ponderosa pine	450, 700 800 ppm-hr	Cumulative dose over 1,2,3 yr	90[b]	Coyne and Bingham (1981)

[a]1 ppm = 1960 μg/m.

[b]$P < 0.05$.

[c]$P < 0.01$.

[d]Standard deviation.

[e]No statistical information.

Source: O_3 Criteria Document, U.S. EPA, 1986

For example, Miller et al. (1969) (Table IX-1) found that 3-year-old ponderosa pine seedlings sustained a 25-percent reduction in apparent photosynthesis after a 60-day exposure to an O_3 concentration of 0.15 ppm for 9 hours a day. Yang et al. (1983) exposed three clones of white pine to O_3 concentrations of 0.10, 0.20 or 0.30 ppm for 4 hours per day for 50 days in CSTR chambers. Net photosynthesis was reduced in the foliage of sensitive and intermediate clones by 14 to 51 percent in direct relation to O_3 dose and clonal sensitivity. Coyne and Bingham (1978) exposed field-grown snap beans to an O_3 concentration of 0.072 ppm for 4 hours a day for 18 days. Apparent photosynthesis was reduced 18 percent in plants treated with O_3.

In addition to depressing photosynthesis in the foliage of many plant species, O_3 inhibits the allocation and translocation of photosynthate (e.g., sucrose) from the shoots to other organs (Tingey, 1974; Jacobson, 1982), often referred to as partitioning. Tingey et al. (1971) found that, when radish plants were exposed to O_3 (0.05 ppm for 8 hrs a day, 5 days per week for 5 weeks), hypocotyl (stem) growth was inhibited 50 percent while foliage growth was inhibited only 10 percent. Ponderosa pine exposed to 0.10 ppm O_3 for 6 hours per day for 20 weeks stored significantly less sugar and starch in their roots than did control plants (Tingey, 1976). Several other studies have measured this partitioning effect of O_3 on photosynthate in carrot, parsley, sweet corn, cotton and pepper (Oshima, 1973; Bennett and Oshima, 1976; Oshima et al. 1978; Oshima et al., 1979; Bennett et al., 1979). Plants were exposed to O_3 concentrations of 0.12 to 0.25 ppm for 3 to 6 hours for 0.2 percent to 7 percent of the total growth period of all the plants. In all species but pepper, root dry weight was depressed much more than leaf dry weight. The reduction in photosynthate translocation to roots and the resulting decrease in root size indicate that the plant had fewer stored reserves, possibly rendering it more sensitive to injury from cold, heat, or water stress. When less carbohydrate is present in roots, less energy

will be available for root-related functions. An O_3-induced suppression of nitrogen fixation by root nodules could affect total biomass and agricultural yield, especially in areas where soil nitrogen is low.

Reproductive capacity (flowering and seed set) is reduced by O_3 in ornamental plants, soybean, corn, wheat and some other plants (Adedipe et al., 1972; Feder and Campbell, 1968; Heagle et al., 1972, 1974; Shannon and Mulchi, 1974). These data suggest that O_3 impairs the fertilization process in plants. This suggestion has been confirmed in tobacco and corn studies using low concentrations (0.05 to 0.06 ppm) of O_3 (Feder, 1968, Mumford, 1972). In addition to these physiological effects, which are directly related to productivity, there are many secondary metabolic responses in a plant exposed to O_3, such as increase in stress ethylene, which may contribute to the manifestation of foliar injury (CD, p. 6-31).

B. Factors Affecting Plant Response

The magnitude of plant response to O_3 exposure can be affected by biological, physical, and chemical variables. Each plant exposed to a given concentration of O_3 will respond to a different extent, depending on such biological factors as genetics, stage of development, and presence of pests or disease, as well as such physical factors as temperature, humidity, light intensity, and soil moisture. Chemical factors in the environment of plants can also modify plant response by either magnifying effects, as with multiple pollutants, or by reducing effects, as with antioxidant sprays. All of these determinants of response can overlap, thus potentially creating a complex myriad of causes for the effects observed.

1. Biological Factors

a. Plant Genetics

Differential susceptibility of individual plants to O_3 exposure is determined by genetic composition. Genetic variance

in response to O_3 is seen among species, cultivars, and individuals within a population. Although foliar injury has been used as the most common measure of comparative O_3 sensitivity, other measures, such as yield and physiological effects, substantiate differential results based on genetic differences. In fact, the relative O_3 sensitivity of cultivars within a species can vary with dose and nature of response measured (Tingey et al., 1972; Heagle, 1979b). Differences in relative sensitivity of cultivars have been reported between controlled O_3 exposures and ambient air field studies (Taylor, 1974; Meiners and Heggested, 1979; Hucl and Beyersdorf, 1982; DeVos et al., 1983). The nature of inherited O_3 sensitivity may help to explain this disparity. Changes in gene expression during plant development and due to environmental variations may explain in part the potential variability in plant response.

b. Developmental Factors

The stage of plant development plays a role in determining sensitivity to O_3. Just prior to, or at maximum, leaf expansion, plant foliage appears to be most sensitive because stomata are functional, intercellular spaces are expanded, and barriers to gas exchange are minimal (CD, p. 6-34). Stages of plant development also can be affected by O_3 exposure. Premature aging and leaf drop have been demonstrated in numerous field and controlled studies (Menser and Street, 1962; Heggestad, 1973; Hofstra et al., 1978; Pell et al., 1980; Reich, 1983; Mooi, 1980). The premature leaf drop and senescence decrease the amount of photosynthate that a leaf can contribute to plant growth. It can be concluded that the effects of O_3 on the senescence process, whenever initiated, may be responsible for many of the documented reductions in yield (CD, p. 6-34).

c. Pathogen and Pest Interactions with Ozone

Disease has been defined as the result of a complex interaction between host plant, environment, and pathogen; problems caused by both pathogens and insects have been termed disease (CD, p. 6-35). Ozone can affect the development of

disease in plant populations. Laboratory evidence suggests that O_3 (at ambient concentrations or greater for 4 or more hours) inhibits infection by pathogens and subsequent disease development (Laurence, 1981; Heagle and Letchworth, 1982). Increases, however, in diseases from "stress pathogens" have been noted. For example, plants exposed to O_3 were more readily injured by Botrytis than plants not exposed to O_3 (Manning et al., 1970a,b; Wukasch and Hofstra, 1977a,b; Bisessar, 1982). Both field and laboratory studies have confirmed that the roots and cut stumps of O_3-injured ponderosa and Jeffrey pines are more readily colonized by a root rot (Heterobasidion annosus). The degree of infection was correlated with the foliar injury (James et al., 1980; Miller et al., 1982). Studies in the San Bernardino National Forest showed that O_3-injured trees were predisposed to attack by bark beetles and that fewer bark beetles were required to kill an O_3-injured tree (Miller et al., 1982). At any stage of the disease cycle O_3 may alter the success of an invading organism by direct effects on them or by modifying the ability of the host plants to defend against attack.

2. Physical Factors

Environmental conditions before, and during, plant exposure are influential in determining plant response. The influence of environmental factors has been studied primarily under controlled conditions, but field observations have substantiated the results. Factors which can potentially influence response of plants to O_3 include light intensity, temperature, relative humidity, soil moisture, and soil fertility. Variations in one or more of these parameters can cause plants to be more or less sensitive to O_3 exposures. The CD has identified the following generalizations concerning the influence of these factors on plant response (CD, p. 6-244):

a. Light conditions conducive to stomatal opening appear to enhance O_3 injury due to increased O_3 absorption.

b. No consistent relationship between temperature and response to O_3 has been reported; however, plants do not appear to be as sensitive at extremely high or low temperatures.

c. Plant injury tends to increase with increasing relative humidity as a result of the effect of humidity on stomatal opening. McLaughlin and Taylor (1980) demonstrated that plants absorb significantly more O_3 at high humidity than at low humidity.

d. Decreasing soil moisture increases plant water stress, causing a reduction in plant sensitivity to O_3. The reduced O_3 sensitivity is apparently related to stomatal closure, which reduces O_3 uptake (U.S. EPA, 1978; Olszyk and Tibbitts, 1981; Tingey et al., 1982). Water stress, however, does not provide a permanent tolerance to O_3 (Tingey et al., 1982).

3. Chemical Factors

The chemical environment of plants can include air pollutants, herbicides, fungicides, insecticides, nematocides, antioxidants, and chemical protectants. These factors, which may influence plant response to O_3, can be grouped into two areas: multiple pollutants and chemical sprays.

a. Multiple Pollutants

Studies indicate that the joint action of O_3 and sulfur dioxide (SO_2) may cause more visible injury to plants than expected from exposure to the individual gases (Shertz et al., 1980a,b; Beckerson and Hofstra, 1979; Olszyk and Tibbitts, 1981). This synergism (injury enhancement) is most common at low concentrations of each gas and when foliar injury induced by each gas, individually, is small. At higher concentrations or when extensive injury occurs the effects tend to be antagonistic (less than additive) (CD, p. 6-245).

Field studies have investigated the influence of SO_2 on plant response to O_3 at ambient and higher concentrations for

several plant species - soybean (Heagle, et al., 1983c; Reich and Amundson, 1984), beans (Oshima, 1978; Heggestad and Bennett, 1981), and potatoes (Foster et al., 1983). Ozone altered plant yield, but SO_2 had no significant effect and did not interact with O_3 to reduce plant yield unless the SO_2 exposures were much greater than typically found in the ambient air in the U.S.

The applicability of the yield results from pollutant combination studies to ambient conditions is not known. An analysis of ambient air monitoring data (SAROAD, 1981; EPRI-SURE) indicate that most sites where the two pollutants were co-monitored had ten or fewer periods of co-occurrence occurred during the growing season (Lefohn and Tingey, 1984). Co-occurrence was defined as the simultaneous occurrence of hourly averaged concentrations of 0.05 ppm or greater for both pollutants. At this time, it appears that most of the studies of the effects on pollutant combinations (O_3 and SO_2) on plant yield have used a longer exposure duration and a higher frequency of pollutant co-occurrence than occur in the ambient air. Preliminary studies using three pollutant mixtures (O_3, SO_2, NO_2) have shown that the additions of SO_2 and NO_2 (at low concentrations) caused a greater growth reduction than O_3 alone (CD, p. 1-79).

Although Lefohn and Tingey (1984) did not observe significant co-occurrence of O_3 and NO_2 or SO_2 in their analysis of ambient air monitoring data, there is some evidence to suggest that O_3 climatology in natural ecosystems may be correlated with that of other anthropogenic pollutants (Taylor and Norby, 1985). Consequently, many forested regions of North America, where monitors are scarce, may experience elevated levels of O_3 in combination with other gases (e.g., nitrogen oxides, sulfur dioxide, nitric acid vapor, and organics) as well as various pollutants deposited in rain or cloud water (e.g., trace elements, hydrogen ion, nitrate and sulfate) (Taylor and Norby, 1985). Given the potential for these interactive effects to occur (McLaughlin, 1985), a number of new studies examining the

effects of O_3 and other acidifying substances on forest trees are being conducted as part of the Forest Response Program under NAPAP (National Acid Precipitation Assessment Program). New data will be available on this issue over the next two to five years.

b. Chemical Sprays

Chemical sprays have long been used to protect agricultural crops from pests and diseases. Fungicides, herbicides, insecticides, and nematocides control damage caused by fungus, weeds, insects, and nematodes, respectively. They also have been shown to alter sensitivity of plants to air pollutants. Studies of the effects of pesticides on O_3 sensitivity have shown differing results, with some chemicals (e.g., nematoxides, phenamiphos) increasing sensitivity and others (e.g., benomyl, carboxin) reducing sensitivity of plants to O_3 (Miller et al., 1976; Sung and Moore, 1979).

Antioxidants, which are commonly used to reduce rubber cracking and food spoilage, have been reported to reduce vegetation injury caused by O_3 (Kendrick et al., 1962). The addition of antioxidants to insecticides, herbicides and fungicides has been shown to reduce O_3 injury to vegetation (Rubin et al., 1980; Koiwai et al., 1977; Gilbert et al., 1977). Ethylenediurea (EDU), a widely used antioxidant, prevented visible injury in bean plants exposed for 150 minutes to 0.8 ppm O_3 (Carnahan et al., 1978). Reduced bronzing, delayed leaf drop, and increased yield have also been attributed to EDU (Hofstra et al., 1978; Weidensaul, 1980). Extensive testing of EDU indicates that treatment of flowers, herbs, and woody vegetation with EDU can reduce O_3 injury (Cathey and Heggestad, 1982a,b; McClenahan, 1979). In conclusion, many chemicals can apparently protect against O_3 injury, but thus far none appears to be sufficiently practical to be used solely for this purpose (CD, pp. 6-68 to 6-71).

c. Heavy Metals

Heavy metals, mainly distributed in heavily industrialized areas, along heavily traveled highways, and on crop land

fertilized with sludge, may have important effects on vegetation and ecosystems. Heavy metals may penetrate the cuticle and cause direct toxic effects on plants or penetrate the soil and affect plant roots, which in turn may contaminate the food chain. The effects of O_3 in combination with heavy metals have been studied in several plant species. Zinc (Zn) and cadmium (Cd) reacted synergistically with O_3 (0.30 ppm for 6 hours) in producing visible injury and loss of chlorophyll in garden cress and lettuce (Czuba and Ormrod, 1974). Exposure to the combination of Cd and O_3 induced earlier development of necrosis and chlorosis and the injury was observed at lower O_3 plus Cd levels than for the individual treatments (Czuba and Ormrod, 1981).

Low concentrations of Cd and nickel (Ni) have been shown to enhance O_3 phytotoxicity on the growth peas (Ormrod, 1977). The interaction of Cd and O_3 was influenced by both concentration and environmental conditions. Tomato plants grown at 0.25 and 0.75 mg Cd/ml developed only slight foliar injury when exposed to O_3 (0.20 ppm for 3 hours) under cloudy skies; whereas the Cd treatment alone had no significant effect (Harkov et al., 1979). In full sun there was extensive O_3 injury and the joint response was synergistic. Quaking aspen treated with 10 μg Cd/ml for 30 days displayed significantly more foliar injury when exposed to ambient air in New Jersey or exposed to 0.20 ppm O_3 for 2.5 hours (Clark and Brennan, 1980). When plants were exposed to 0.03 ppm O_3, the Cd enhancement of injury was not apparent. Although it is not possible at the present time to assess the risk from the joint action of gaseous and heavy metal pollutants to vegetation, the limited data available indicates that heavy metals can increase the phytotoxic reactions of O_3 (CD, p. 6-67).

X. Assessment of Welfare Effects and Related Welfare Issues Considered in Selecting Secondary Standard(s) for Ozone

Of the phytotoxic compounds commonly found in the ambient air, O_3 is the most prevalent, impairing crop production and injuring native vegetation and ecosystems more than any other air pollutant (Heck et al., 1980). Some of the effects of O_3 reported in the literature occur at O_3 levels at or below natural background concentrations in many areas of the country (see Section IV. for further discussion of background values). Ozone has also been shown to damage elastomers, textile fibers and dyes and certain types of paints. Other photochemical oxidants of importance to effects on vegetation, ecosystems and materials are nitrogen dioxide (NO_2) and peroxyacetyl nitrates. Air Quality Criteria for Oxides of Nitrogen (U.S. EPA, 1982) and Review of the NAAQS for NO_2: Assessment of Scientific and Technical Information (U.S. EPA, 1984) previously assessed the phytoxicity of NO_2, and thus NO_2 will not be discussed in this staff paper. In addition, while at a given dose the peroxyacetyl nitrates are more phytotoxic than O_3 (p. X-22), they generally occur at significantly lower ambient concentrations. Because phytotoxic concentrations of peroxyacetyl nitrates are less widely distributed than those of O_3 (CD, p. 6-1), the focus of this staff paper will be on the effects of O_3.

The objective of this section of the staff paper is to assess the current basis for the O_3 secondary NAAQS as contained in Chapters 6, 7 and 8 of the CD. In addition, the section will summarize new analyses that address key issues of concern for the secondary standard: relationships of various air quality indicators, crop loss estimates, averaging times and forest response to O_3. Key new studies that relate to the issue of averaging time(s) will also be discussed to determine whether new effects information suggests any change in existing secondary NAAQS for O_3.

A. Vegetation Effects

1. Types of Exposure Effects

Plant response to O_3 exposure is quite varied and may be expressed as biochemical, physiological, visible injury, growth, yield, reproductive and ecosystem effects. When reviewing the current data base for vegetation effects, it is apparent that the bulk of the scientific research on crops that has been completed since the last standard review focuses on reduction in growth and yield from various long-term (days, months) exposures. In contrast, the majority of short-term (few hours) exposure studies focus on foliar injury or physiological changes as the response measure. While biochemical and physiological alterations are the basis of all subsequent effects as described in section IX A.1. of this paper, visible foliar injury provides one of the earliest manifestations of short-term effects of O_3. However, these effects are not always well correlated with reduction in growth and yield (CD, p. 6-141).

This section will attempt to assess the data with regard to foliar injury effects and reductions in growth and yield. For purposes of this staff paper, greater emphasis will be placed on damage or yield loss than on injury. Injury includes all plant reactions, such as reversible changes in plant metabolism (e.g., altered photosynthesis), leaf necrosis, altered plant quality, or reduced growth, which do not impair yield or intended use of a plant (Guderian, 1977). Damage or yield loss, on the other hand, involves any effect which reduces the quantity, use, or value (e.g., aesthetic) of a plant or any impairment in the intended use of a plant. Although foliar injury may have some limitations in evaluating plant response, its presence is usually an indicator of elevated O_3 concentrations. Growth and yield losses provide an important measure of the effects of O_3 because such losses impair the intended use of the plant and generally constitute damage, whereas foliar injury may or may not be considered damage. These growth and yield loss effects have become the focus of most of the exposure response models and

assessments to be discussed later, thus providing the strongest and best documented evidence of vegetation effects of O_3 exposure.

a. Visible Foliar Injury Effects

The first documented observation of O_3 injury to vegetation in the field was by Richards et al. (1958), who described O_3 stipple on grape vine leaves. Other studies soon after confirmed that foliar injury was being caused by O_3 from nearby cities (Heggestad and Middleton, 1959; Daines et al., 1960). Numerous subsequent studies have reported vegetation injury at rural sites caused by O_3 transported long distances from urban centers (Heck et al., 1969; Kelleher and Feder, 1978; Skelly, 1980; Edinger et al., 1972).

Of the many approaches taken to estimate the O_3 levels and exposure times required to induce these foliar injury effects, most have involved short-term exposures (less than one day) and have measured visible injury as the response variable. Various plant species were exposed to a range of O_3 levels and exposure durations by Heck and Tingey (1971), who evaluated the resulting data by regression analysis. Data for several species are summarized in Table X-1 (CD, p. 6-224) to illustrate the range of O_3 levels and exposure times required to induce 5 and 20% foliar injury on sensitive, intermediate, and less sensitive species.

Limiting-value analysis is an alternative approach to estimating O_3 levels and exposure durations which induce foliar injury. Such an analysis, which was performed on more than 100 studies of agricultural crops and 18 studies of tree species, yielded the following range of concentration and exposure durations that were likely to induce visible injury (Jacobson, 1977):

1. Agricultural crops:
   0.20 to 0.41 ppm for 0.5 hr
   0.10 to 0.25 ppm for 1.0 hr
   0.04 to 0.09 ppm for 4.0 hr

Table X-1. Ozone Concentrations for Short-Term Exposure that Produce 5 or 20 Percent Injury to Vegetation Growth Under Sensitive Conditions[a]

Exposure time, hr	Ozone Concentrations that may Produce 5% (20%) Injury: Sensitive plants	Intermediate plants	Less sensitive plants
0.5	0.35 to 0.50 (0.45 to 0.60)	0.55 to 0.70 (0.65 to 0.85)	≥ 0.70 (0.85)
1.0	0.15 to 0.25 (0.20 to 0.35)	0.25 to 0.40 (0.35 to 0.55)	≥ 0.40 (0.55)
2.0	0.09 to 0.15 (0.12 to 0.25)	0.15 to 0.25 (0.25 to 0.35)	≥ 0.30 (0.40)
4.0	0.04 to 0.09 (0.10 to 0.15)	0.10 to 0.15 (0.15 to 0.30)	≥ 0.25 (0.35)
8.0	0.02 to 0.04	0.07 to 0.12	≥ 0.20 (0.30)

[a]The concentrations in parenthesis are for the 20% injury level. Table is from U.S. Environmental Protection Agency (1986, p. 6-224).

Source: O_3 Criteria Document, U.S. EPA, 1986

2. Trees and shrubs:
   0.20 to 0.51 ppm for 1.0 hr
   0.10 to 0.25 ppm for 2.0 hr
   0.06 to 0.17 ppm for 4.0 hr

Foliar injury in sensitive plants provides a basis for a good understanding of the occurrence of elevated O_3 concentrations in a given area. Among the plants that have been identified as indicators of pollutants are milkweed (Duchelle and Skelly, 1981), eastern white pine (Benoit et al., 1982), ponderosa and Jeffrey pine (Miller, 1973) Bel W-3 tobacco (Posthumus, 1976) and lichens (Sigal and Nash, 1983). Although the presence of visible foliar symptoms on vegetation cannot be directly related to effects on growth or yield, they do indicate that elevated levels of O_3 have occurred. The detection of visible symptoms is an indication that additional studies should be undertaken to determine if effects on growth and yield are occurring (CD, pp. 6-84).

There are a few studies in which short-term exposures have resulted in growth and yield reduction: exposure of cherry belle radish to 0.25 ppm O_3 for three hours resulted in a 38 percent reduction in root dry weight (Adedipe and Ormrod, 1974); exposure to 0.10 ppm O_3 for two hours resulted in a 9 percent reduction in the average of three growth responses of capri petunia (Adedipe, 1972); a 16 percent reduction in leaflet area growth rate was reported in pinto bean exposed to 0.05 ppm O_3 for 12 hours (Evans, 1974). In addition, some cultivars of crops such as spinach and tobacco experience yield losses due to extensive foliar injury at 0.10 ppm for 2-hours (Menser and Hodges, 1972). Thus, although a few studies relate short-term exposures and yield loss, there is very little yield loss data based on exposures that are easy to relate to the 1-hr average of the current secondary standard.

Despite the importance of visible symptoms, it must be recognized that long-term exposure to low pollutant

concentrations may adversely affect plant health without producing visible symptoms. Chronic injury from this type of exposure may be represented by reductions in growth, and/or yield, or premature senescence resulting from changes in photosynthesis, respiration, chlorophyll content or other processes (Dochinger et al., 1970; Feder, 1978; Heck, 1966; Posthumus, 1976).

Because plant growth and production depend on photosynthetically functional leaves, various studies have been conducted to assess the relationship between foliar injury and yield for species in which the foliage is not part of the yield. Some research has demonstrated significant yield loss with little or no foliar injury (Tingey et al., 1971; Tingey and Reinert, 1975; Kress and Skelly, 1985a,b,c; Adedipe et al., 1972), while others have reported significant foliar injury without yield loss (Heagle et al., 1974; Oshima et al., 1975). Relative sensitivities of two potato cultivars were reversed when judged by foliar injury versus yield reductions (Pell et al., 1980). Foliar injury in field corn was reported at lower O_3 concentrations than yield loss, but, at higher O_3 levels, yield loss was increased to a greater extent than foliar injury; similarly, it was reported that foliar injury was not a good predictor of yield loss for wheat (Heagle et al., 1979a,b). Therefore, while the presence of foliar injury is significant in and of itself, no precise relationship exists between foliar injury and yield loss for species of plants for which foliage is not part of the yield (CD, p. 6-141).

b. Growth and Yield Effects

As stated previously, the bulk of the evidence since the last standard review has focused on reduction in growth and yield from various long-term exposures. The data base has been summarized previously (Chapter 6, CD) and consists of open top chamber studies, greenhouse and other controlled experiments, and various ambient air exposures. This section will assess the

strengths and weaknesses of these various approaches. Most of these studies have characterized the exposure-response relationship in terms of the seasonal daily daylight mean O_3 concentration, although various averaging times have been used.

For purposes of assessing exposure-response relationships derived from the existing studies, yield loss is defined as an impairment of, or decrease in, the value of the intended use of the plant. This concept includes reduction in aesthetic values, changes in crop quality, and occurrence of foliar injury when foliage is the marketable part of the plant. The actual amount of yield loss due to decreased aesthetic value or appearance may be more difficult to quantify than yield loss in weight or bulk but is extremely important for crops such as tobacco, spinach, and ornamentals. Such effects occur at concentrations as low as 0.041 ppm for several weeks or 0.10 ppm for 2 hours, and can constitute a yield loss when marketability of the plant is decreased (Menser and Hodges, 1972).

Most of the recent studies of O_3-induced yield loss have measured effects on the weight of the marketable plant organ. These effects will be the primary focus of this section. Studies conducted to estimate the impact of O_3 on the yield of various crop species have been grouped into two types, depending on the experimental design and statistical methods used to analyze the data: (1) studies that developed predictive equations relating O_3 exposure to plant response and (2) studies comparing discrete treatment levels to a control using analyses of variance. The advantage of the regression approach is that it permits the estimation of the O_3 impact on plant yield over the range of concentrations, not just at the treatment means as is the case with the analysis of variance methods.

(1) Open-Top Chamber Studies

Data from a series of studies conducted by the National Crop Loss Assessment Network (NCLAN) have been analyzed to develop predictive equations relating 7-hour seasonal mean O_3 exposures to crop yield loss. Examples of the relationship between O_3

concentration and plant yield are shown in Figure X-1 (CD, p. 6-229) and X-2 (CD, p. 6-230). These cultivars/species were selected because they illustrate the kinds of exposure response that occur and the type of year-to-year variation in plant response to O_3 that may occur. The derived regression equations can be used to determine the concentrations that would be predicted to cause a specific yield loss or to estimate the predicted yield loss that would result from a specific O_3 concentration. Both of the approaches cited above have been used to summarize the data on crop response to O_3 by use of the Weibull (Rawlings and Cure, 1985) function.

As an example of response, the O_3 concentrations that would be predicted to cause a 10- to 30-percent yield loss have been estimated (Table X-2 (CD, p. 6-232). These cutpoints were selected to illustrate an effect and do not imply an effects threshold. Other cutpoints could have been selected. A brief review of these data in the table suggests that: (1) a 10% mean yield loss is predicted for several species when the 7-hour seasonal mean concentration of O_3 exceeds 0.04-0.05 ppm; (2) grain crops were generally less sensitive to O_3 than other crops; (3) sensitivity differences within a species may be as large as differences between species. In addition to differences in sensitivity among species and cultivars, the data in Figure X-1 and X-2 illustrate year to year variations in plant response to O_3.

Although linear regression equations have been used to estimate yield loss, there appear to be systematic deviations from the data for some species and cultivars even though the equations had moderate to high coefficients of determination (R^2). The use of plateau-linear, polynomial equations and the recently developed Weibull model (Heck et al., 1983) appeared to fit the data better. On the basis of available data, it is recommended that curvilinear exposure response functions be used to describe and analyze plant response to O_3 (CD, p. 7-106).

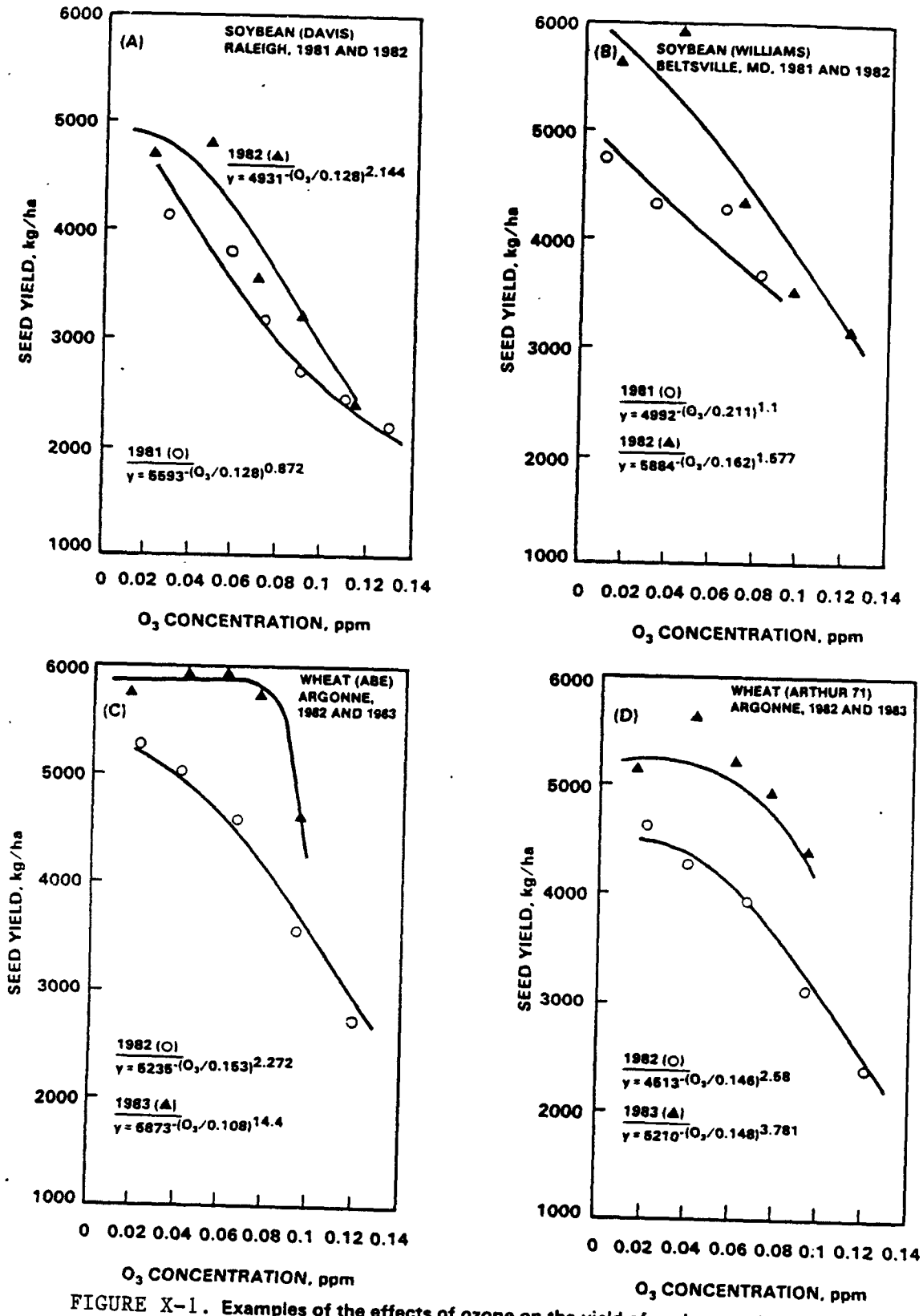


FIGURE X-1. Examples of the effects of ozone on the yield of soybean and wheat cultivars. The O_3 concentrations are expressed as 7-hr seasonal mean concentrations. The cultivars were selected as examples of O_3 effects and of year-to-year variations in plant response to O_3.

Source: Soybean data from Heck et al. (1984b); wheat data from Kress et al. (1985).

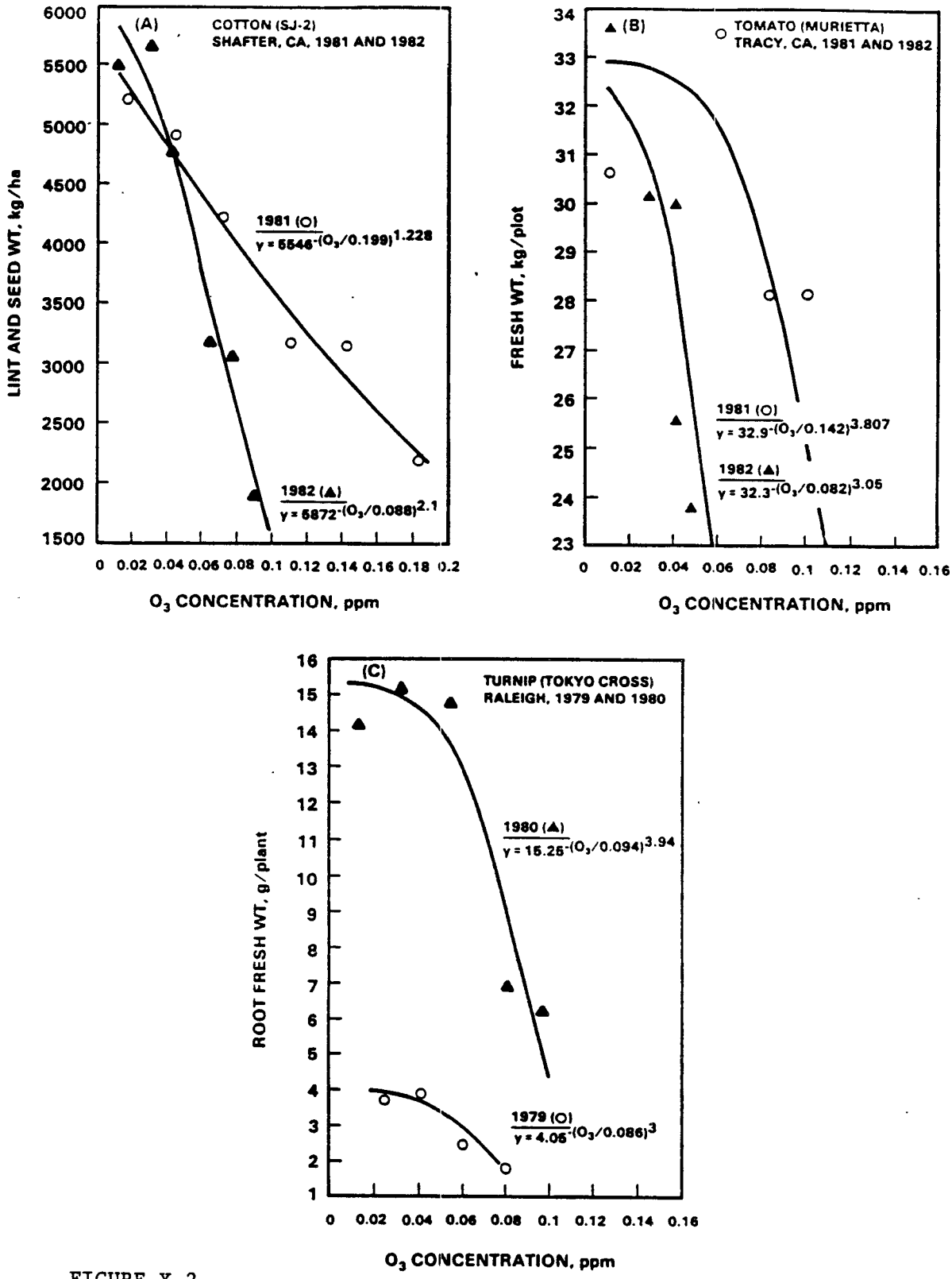


FIGURE X-2. Examples of the effects of ozone on the yield of cotton, tomato, and turnip. The O_3 concentrations are expressed as 7-hr seasonal mean concentrations. The species were selected as examples of O_3 effects and of year-to-year variations in plant response to O_3.

Source: Cotton and tomato data from Heck et al. (1984b); turnip data from Heagle et al. (1985).

TABLE X-2. SUMMARY OF OZONE CONCENTRATIONS PREDICTED TO CAUSE 10 PERCENT AND 30 PERCENT YIELD LOSSES AND SUMMARY OF YIELD LOSSES PREDICTED TO OCCUR AT 7-hr SEASONABLE MEAN OZONE CONCENTRATIONS OF 0.04 and 0.06 ppm[a]

Species	O_3 concentrations, ppm, predicted to cause yield losses of:		Percent yield losses predicted to occur at 7-hr seasonal mean O_3 concentration of:	
	10%	30%	0.04 ppm	0.06 ppm
Legume crops				
Soybean, Corsoy	0.048	0.082	6.4	16.6
Soybean, Davis (81)	0.038	0.071	11.5	24.1
Soybean, Davis (CA-82)	0.048	0.081	6.4	16.5
Soybean, Davis (PA-82)	0.059	0.081	2.0	10.4
Soybean, Essex	0.048	0.099	7.2	14.3
Soybean, Forrest	0.076	0.118	1.7	5.3
Soybean, Williams	0.039	0.093	10.4	18.1
Soybean, Hodgson	0.032	0.066	15.4	18.4
Bean, Kidney	0.033	0.063	14.9	28
Peanut, NC-6	0.046	0.073	6.4	19.4
Grain crops				
Wheat, Abe	0.059	0.095	3.3	10.4
Wheat, Arthur 71	0.056	0.094	4.1	11.7
Wheat, Roland	0.039	0.067	10.3	24.5
Wheat, Vona	0.028	0.041	28.8	51.2
Wheat, Blueboy II	0.088	0.127	0.5	2.8
Wheat, Coker 47-27	0.064	0.107	2.2	8.4
Wheat, Holly	0.099	0.127	0.0	0.9
Wheat, Oasis	0.093	0.135	0.4	2.4
Corn, PAG 397	0.095	0.126	0.3	1.5
Corn, Pioneer 3780	0.075	0.111	1.4	5.1
Corn, Coker 16	0.133	0.175	0.0	0.3
Sorghum, DeKalb-28	0.108	0.186	0.0	2.7
Barley, Poco	0.121	0.161	0.0	0.5
Fiber crops				
Cotton, Acala SJ-2 (81)	0.044	0.096	8.3	16.2
Cotton, Acala SJ-2 (82)	0.032	0.055	16.1	35.1
Cotton, Stoneville	0.047	0.075	4.6	16.2
Horticultural crops				
Tomato, Murrieta (81)	0.079	0.108	0.8	3.7
Tomato, Murrieta (82)	0.040	0.059	10.3	31.2
Lettuce, Empire	0.053	0.075	0.0	16.8
Spinach, America	0.046	0.082	6.8	17.2
Spinach, Hybrid	0.043	0.082	2.6	9.2
Spinach, Viroflay	0.048	0.080	6.0	16.7
Spinach, Winter Bloom	0.049	0.080	5.8	16.5
Turnip, Just Right	0.043	0.064	7.7	24.9
Turnip, Pur Top W. G.	0.040	0.064	10.1	26.5
Turnip, Shogoin	0.036	0.060	13.0	29.7
Turnip, Tokyo Cross	0.053	0.072	3.3	15.6

[a]The yield losses are derived from Weibull equations and are based on the control yields in charcoal-filtered air.

Source: Derived from Heck et al. (1984b).

Although NCLAN provides valuable dose response information on a variety of crops, the program has limitations that must also be considered. The adequacy of the 7-hr seasonal mean has been questioned on several grounds. Recent evidence has suggested that the diurnal cycle of O_3 in natural ecosystems may differ markedly from that of urban airsheds where the highest O_3 concentrations are in the mid-afternoon. In remote locations it is likely that maximum O_3 concentrations shift into the late afternoon and evening hours (Taylor and Norby, 1985; McCurdy, 1987). In addition, the use of the seasonal mean as well as other mean statistics in characterizing exposure implies that all exposures over the course of the daylight period are equally effective in eliciting a plant response and minimizes the contribution of peak concentrations. Thus, while the 7-hr seasonal mean may contain all hourly concentrations for the 7-hr period, it treats all concentrations the same (CD, p. 6-10). An infinite number of hourly distributions can be used to generate the same 7-hr seasonal mean; some containing many peaks and others containing none. In fact, Larsen and Heck (1984) have emphasized that it is possible for two air sampling sites with the same daytime arithmetic mean O_3 concentration to have different estimated crop reductions.

Several authors have reported the greater importance of concentration compared to exposure duration in causing injury (e.g., Heck et al., 1966; Bennett, 1979, Heck and Tingey, 1971, Reinert and Nelson, 1979, Larsen and Heck, 1984). More recent findings indicate that constant concentrations have less effect on plant growth responses than variable or episodic exposures at equivalent cumulative doses (Musselman et al., 1983; Hogsett et al., 1985). Hogsett found that over the period of 3 cuttings (133 days), alfalfa growth was reduced more when exposed to an episodic O_3 profile than to a regime of daily O_3 peaks, both with equivalent long-term means. Generally, assessment of human physiological responses as well as vegetation responses to O_3 have stressed the importance of episodic exposures (high

concentrations over short periods of time) in eliciting a biological response (Rogers, 1985).

There are several additional concerns regarding the NCLAN data. First, there is the lack of validation of the model. That is, however, a common deficiency among all models. A second concern that has been identified is that the use of the Weibull model might bias the estimated yield losses in the low dose range of the curve. Finally, the most serious limitation of the NCLAN data stems from the inadequate sampling of environmental conditions and in inadequate number of test sites. The problem of inadequate sampling is an easy criticism to level at almost any agricultural study that has the objective of making an inference over time and space. The limited time and resources meant that subjective judgments had to be made regarding which species were to be studied. The primary criteria for these decisions were the relative economic importance of the species and their sensitivity to O_3. While the limitations mentioned above create uncertainties that should be considered in any application of the results, the staff concludes that with appropriate caveats, the NCLAN data, as discussed, provide useful information on crop loss due to O_3 exposure. A more detailed analysis of the NCLAN crop loss data which addresses some of the key uncertainties, particularly regarding exposure dynamics, is discussed on p. X-46.

(2) Greenhouse and Controlled Environment Studies

The effects of O_3 on plant yield may be affected by a host of genetic and environmental factors. In addition to the use of regression approaches in the studies previously discussed, various other approaches have been used to investigate the effects of O_3 on crop yield under more controlled (to various degrees) conditions, as shown in Tables 6-22 and 6-23 of the CD (CD, p. 6-129 to 6-137). These studies were designed to test whether specific O_3 treatments were different from the control, using analysis of variance. To summarize the data, the lowest O_3 concentration that significantly reduced yield was taken from

each study (Table X-3; CD, p. 6-235); this concentration was frequently the lowest concentration used in the study. Although it is difficult to estimate a "no-effect" exposure concentration, the data generally seem to indicate that O_3 concentrations of 0.10 ppm (frequently the lowest concentration used in the study) for a few hours a day for several days to several weeks induced yield losses of 10-55 percent.

One weakness of studies conducted under more controlled conditions (greenhouse, growth chamber) is that it is difficult to extrapolate data from the chamber to field conditions. The more controlled chamber data, however, do serve to strengthen the demonstration of O_3 effects in the field. Concentrations of 0.10 ppm and above appear to cause yield reductions consistently, although exceptions can be found. In studies which used concentrations below 0.10 ppm, the response varied among species (Table 6-22; CD, p. 6-130). Concentrations of 0.05 ppm in extended or repeated exposures have been shown to cause yield reductions in some species or cultivars, no effects in others, and increased yield in others (Table 6-23; CD, p. 6-134). Although these studies seem to suggest that a higher O_3 concentration was required to cause an effect than was estimated from the regression studies, it should be noted that the concentrations derived from the regression studies were based on a 10% yield loss, while the studies using analysis of variance indicate that 0.10 ppm frequently induced substantial losses (10 to 55 percent).

(3) Ambient Air Exposure Studies

Ambient air exposure studies conducted to date demonstrate that O_3 in many areas of the country can reduce plant yield. Although the most severe effects appear to occur in areas with the highest O_3 concentrations such as the South Coast Air Basin and the San Bernadino Mountains in California, other agricultural areas in the country can be impacted as well. Recently, open-top chamber studies evaluating the yield of plants grown in the

TABLE X-3. OZONE CONCENTRATIONS AT WHICH SIGNIFICANT YIELD LOSSES HAVE BEEN NOTED FOR A VARIETY OF PLANT SPECIES EXPOSED UNDER VARIOUS EXPERIMENTAL CONDITIONS

Plant species	Exposure duration	Yield reduction, % of control	O_3 concentration, ppm	Reference
Alfalfa	7 hr/day, 70 days	51, top dry wt	0.10	Neely et al. (1977)
Alfalfa	2 hr/day, 21 day	16, top dry wt	0.10	Hoffman et al. (1975)
Pasture grass	4 hr/day, 5 days/wk, 5 wk	20, top dry wt	0.09	Horsman et al. (1980)
Ladino clover	6 hr/day, 5 days	20, shoot dry wt	0.10	Blum et al. (1982)
Soybean	6 hr/day, 133 days	55, seed wt/plant	0.10	Heagle et al. (1974)
Sweet corn	6 hr/day, 64 days	45, seed wt/plant	0.10	Heagle et al. (1972)
Sweet corn	3 hr/day, 3 days/wk, 8 wk	13, ear fresh wt	0.20	Oshima (1973)
Wheat	4 hr/day, 7 day	30, seed yield	0.20	Shannon and Mulchi (1974)
Radish	3 hr	33, root dry wt	0.25	Adedipe and Ormrod (1974)
Beet	2 hr/day, 38 days	40, storage root dry wt	0.20	Ogata and Maas (1973)
Potato	3 hr/day, every 2 wk, 120 days	25, tuber wt	0.20	Pell et al. (1980)
Pepper	3 hr/day, 3 days/wk, 11 wk	19, fruit dry wt	0.12	Bennett et al. (1979)
Cotton	6 hr/day, 2 days/wk, 13 wk	62, fiber dry wt	0.25	Oshima et al. (1979)
Carnation	24 hr/day, 12 days	74, no. of flower buds	0.05-0.09	Feder and Campbell (1968)
Coleus	2 hr	20, flower no.	0.20	Adedipe et al. (1972)
Begonia	4 hr/day, once every 6 days for a total of 4 times	55, flower wt	0.25	Reinert and Nelson (1979)
Ponderosa pine	6 hr/day, 126 days	21, stem dry wt	0.10	Wilhour and Neely (1977)
Western white pine	6 hr/days, 126 days	9, stem dry wt	0.10	Wilhour and Neely (1977)
Loblolly pine	6 hr/day, 28 days	18, height growth	0.05	Wilhour and Neely (1977)
Pitch pine	6 hr/day, 28 days	13, height growth	0.10	Wilhour and Neely (1977)
Poplar	12 hr/day, 5 mo	+1333, leaf abscission	0.041	Wilhour and Neely (1977)
Hybrid poplar	12 hr/day, 102 days	58, height growth	0.15	Patton (1981)
Hybrid poplar	8 hr/day, 5 day/wk, 6 wk	50, shoot dry wt	0.15	Patton (1981)
Red maple	8 hr/day, 6 wk	37, height growth	0.25	Dochinger and Townsend (1979)
American sycamore	6 hr/day, 28 days	9, height growth	0.05	Kress and Skelly (1982)
Sweetgum	6 hr/day, 28 days	29, height growth	0.10	Kress and Skelly (1982)
White ash	6 hr/day, 28 days	17, total dry wt	0.15	Kress and Skelly (1982)
Green ash	6 hr/day, 28 days	24, height growth	0.10	Kress and Skelly (1982)
Willow oak	6 hr/day, 28 days	19, height growth	0.15	Kress and Skelly (1982)
Sugar maple	6 hr/day, 28 days	12, height growth	0.15	Kress and Skelly (1982)

Source: O_3 Criteria Document, U.S. EPA, 1986

presence of O_3 (ambient air) versus charcoal-filtered air have demonstrated losses in tomato (33 percent), bean (26 percent), soybean (20 percent), snapbean (0 to 22 percent), sweet corn (9 percent), several tree species (12 to 67 percent), and forbes, grasses and sedges (9 to 33 percent) (Table X-4; CD, pp. 6-145 and 6-146). Field studies in the San Bernadino Forest during the last 30 years indicate that ambient O_3 reduced the height growth of ponderosa pine by 25 percent, annual radial growth by 37 percent and the total volume of wood produced by 84 percent (Miller et al., 1982). Oxidant-induced changes in forest ecosystems will be discussed in more detail in Section B.

Antioxidant chemical protectants appear to provide another objective method of estimating the impact of ambient O_3 on crop production (Toivonen et al., 1982). Some limitations, however, must be kept in mind. The chemical itself may alter plant growth and therefore results must be interpreted carefully. Also, since the chemical may not be effective against all concentrations of all pollutants, an underestimation of yield loss may result (Manning et al., 1974).

Ethylenediurea (EDU), developed as a chemical protectant to prevent oxidative effects of O_3, has been used extensively to reduce visible O_3 injury in greenhouse and field studies and to estimate O_3-induced yield loss. Several studies have reported estimates of the impact of O_3 on yield by comparing yield data from plots with and without EDU treatment (Table X-5; CD, p. 6-237). For a seven-week study in which ambient O_3 exceeded 0.15 and 0.08 ppm on five separate days at each concentration, EDU treatment reduced foliar injury of onions and increased yield by 37.8% (Wakasch and Hofstra, 1977). During a June-to-August study in which ambient O_3 exceeded 0.08 ppm on 15 days with a maximum of 0.14 ppm, EDU treatment increased yield of the Tiny Tim tomato by 30% but had no effect on the New Yorker cultivar (Legassicke and Ormrod, 1981). Other studies with beans (Toivonen et al., 1982), tobacco (Bissessar and Palmer, 1984), and potatoes (Bissessar, 1982) provided evidence of EDU increasing yield by

TABLE X-4

EFFECTS OF AMBIENT AIR IN OPEN-TOP CHAMBERS, OUTDOOR CSTR CHAMBERS, OR GREENHOUSES ON GROWTH AND YIELD OF SELECTED CROPS

Plant species	O_3 concn., ppm	Exposure duration	Percent reduction from control	Location of study	Monitoring method[a]	Calibration method[b]	Fumigation facility[c]	Reference
Tomato (Fireball 861 VR)	0.035 (0.017-0.072)	99 day average (0600-2100)	33[d], fruit fresh wt	New York	Mast	NBKI	OT	MacLean and Schneider (1976)
Bean (Tendergreen)	0.041 (0.017-0.090)	43 day average (0600-2100)	26[d], pod fresh wt; 24[d], number of pods		Mast	NBKI	OT	
Snap bean (3 cultivars: Astro, BBL 274, BBL 290)	0.042	3 mo average (0900-2000)	1, pod wt	Maryland	Not given	Not given	OT	Heggestad and Bennett (1981)
Soybean (4 cultivars: Cutler, York, Clark, Dare)	>0.05	31% of hr (8:00 a.m. to 10:00 p.m.) from late June to mid-September over three summers; 5% of the time the concentration was above 0.08 ppm	20[d], seed wt; 10[d], wt/100 +2, % protein content, 4% oil content	Maryland	Mast	NBKI, known O_3 source	OT	Howell et al. (1979) Howell and Rose (1980)
Forbs, grasses, sedge §	0.052	1979, 8 hr/day average 1000-1800), April-September	32, total above ground biomass	Virginia	Chem	Known O_3 source, UV	OT	Duchelle et al. (1983)
	0.051	1980, 8 hr/day average (1000-1800), April-September	20, total above ground biomass	Virginia	Chem			
	0.035	1981, 8 hr/day average (1000-1800), April-Sepbember	21, total above ground biomass					
Snap bean (Gallatin 50)	>0.05	Average 170 hr over 60 days exposure (1972-1974) (6 crops)	+5, pod fresh wt	Maryland	Mast	1% NBKI, Chem	OT,	Heggestad et al. (1980)
(BBL 290)	>0.05	Average 170 hr over 60 days exposure (1972-1974) (6 crops)	14[d], pod fresh wt	Maryland	Mast	1% NBKI, Chem	OT	Heggestad et al. (1980)
(Astro)	>0.05	Average 170 hr over 60 days exposure (1972-1974) (6 crops)	3, pod fresh wt	Maryland	Mast	1% NBKI, Chem	OT	Heggestad et al. (1980)

TABLE X-4 (cont.)

EFFECTS OF AMBIENT AIR IN OPEN-TOP CHAMBERS, OUTDOOR CSTR CHAMBERS, OR GREENHOUSES ON GROWTH AND YIELD OF SELECTED CROPS

Plant species	O_3 concn., ppm	Exposure duration	Percent reduction from control	Location of study	Monitoring method[a]	Calibration method[b]	Fumigation facility[c]	Reference
(Astro)	>0.05	Average 160 hr over 60 days exposure (1975-1976) (2 crops)	6, pod dry wt	Maryland	Mast	1% NBKI, Chem	OT	Heggestad et al. (1980)
Snap bean (Gallatin 50)	>0.05	Average 160 hr over 60 days exposure (1975-1976) (2 crops)	+1, pod dry wt	Maryland	Mast	1% NBKI Chem	OT	Heggestad et al. (1980)
(BBL 290)	>0.05	Average 160 hr over 60 days exposure (1975-1976) (2 crops)	10, pod dry wt	Maryland	Mast	1% NBKI Chem	OT	Heggestad et al. (1980)
(BBL 274)	>0.05	Average 160 hr over 60 days exposure (1975-1976) (2 crops)	22[d], pod dry wt	Maryland	Mast	1% NBKI Chem	OT	Heggestad et al. (1980)
Sweet corn (Bonanza)	>0.08	58% of hr (0600-2100) between 1 July and 6 September	9[d], ear fresh wt; 10[d], no. seeds/ear	California	Mast	UV	OT	Thompson et al., 1976a
(Monarch Advance)	0.08		28[d], ear fresh wt; 42[d]%, no. seeds/ear					

[a]Chem = chemiluminescence; Mast = Mast oxidant meter (coulombmetric); UV = ultraviolet spectrometry.

[b]NBKI = neutral buffered potassium iodide; UV = ultraviolet spectrometry.

[c]OT = open-top chamber; CSTR = continuous stirred tank reactor.

[d]Significant at $p = 0.05$.

[e]Total above ground biomass, 3 yr average; NF and open plot versus CF α significant at $p = 0.05$

Source: O_3 Criteria Document, U.S. EPA, 1986

TABLE X-5. EFFECTS OF OZONE ON CROP YIELD AS DETERMINED BY THE USE OF CHEMICAL PROTECTANTS[a]

Species	Yield reduction, % of control[b]	O_3 exposure, ppm	Reference
Beans (green)	41	>0.08 for total of 27 hr over 3.5 months	Manning et al. (1974)
Onion	38	>0.08 on 5 days out of 48	Wukasch and Hofstra (1977b)
Tomato	30	>0.08 on 15 days over 3 months	Legassicke and Ormrod (1981)
Bean (dry)	24	>0.08 on 11 days (total of 34 hr) over 3 months	Temple and Bisessar (1979)
Tobacco	18	>0.08 on 14 days during the summer	Bisessar and Palmer (1984)
Potato	36	>0.08 ppm on 18 days (total of 68 hr) over 3 months	Bisessar (1982)
Potato	25	-[c]	Clarke et al. (1983)

[a]All the species were treated with the antioxidant, EDU, except the bean study by Manning et al. (1974) which used the systemic fungicide, benomyl.

[b]Yield reduction was determined by comparing the yields of plants treated with chemical protectants (control) to those that were not treated.

[c]This study was run over 2 years when the O_3 doses were 65 and 110 ppm-hr, respectively, but the yield loss was similar both years.

Source: O_3 Criteria Document, U.S. EPA, 1986

35%, 18%, and 35.5% respectively. These and other studies demonstrate the effectiveness of antioxidants to improve crop yield and to enable O_3-induced crop loss to be estimated for several crop species. The data clearly show that O_3 concentrations occurring during these studies reduced crop yields between 10 and 40% although O_3 levels only occasionally exceeded 0.08 ppm. A new study, (SP, P. X-57) which uses the EDU technique to investigate the impact of ambient O_3 pollution on field grown soybeans, concludes that soybeans do not sustain yield losses due to O_3 pollution in New Jersey.

2. Related Vegetation Issues

a. Empirical Models Used to Develop Exposure Response Relationships

Empirical response models for the effects of O_3 on plants are mathematical functions that describe a relationship between O_3 exposure and a biological response. These models, which quantitatively define the entire relationship between the range of exposures, have been used both for crop production forecasting and to interface biological systems with economic models.

Categories of empirical exposure response models include physiological, injury, growth and yield-loss models. Physiological response models are used as research tools to summarize relationships or allow comparisons among species, while injury models generally estimate the magnitude of foliar injury incurred from pollutant exposures. Growth models quantify pollutant-induced changes in biomass accumulation. Yield loss models are most difficult to develop but are needed to estimate production and economic losses since they provide direct relationships between yield and pollutant exposure.

The three types of yield-loss models which describe plant response in the absence of a known functional relationship between O_3 levels and yield are linear, plateau-linear, and Weibull functions. Non-linear models, which have been used in describing situations for which response to O_3 appears to have a

threshold, are the plateau-linear model and Weibull model. The plateau-linear model incorporates a threshold value but does not allow curvature of any increase in yield followed by a decrease. The Weibull model, however, can take on a plateau shape followed by curved gradual decreases.

National Crop Loss Assessment Network (NCLAN) yield losses have been modeled by Heck et al. (1983, 1984), who used a three parameter Weibull function. The Weibull function chosen has a flexible form, which covers the range of observed biological responses, is biologically realistic, has parameters with clear interpretations, and offers a method of summarizing species responses by developing a common proportional model (Rawlings and Cure, 1985). The Weibull model subsequently has been used with NCLAN data previously modeled with other functional forms (CD, p. 6-165).

b. Statistics Used to Characterize Ozone Exposures

Important factors needed to characterize O_3 exposures adequately in vegetation exposure studies include time, concentration, and dynamic nature (i.e., constant or variable). Which of the components of exposure actually causes plant response, however, remains unclear. Although many studies tend to report exposures as mean O_3 concentrations, the exposure statistic can be peak hourly, daily, weekly, monthly, or seasonal means; number of hours above selected concentration; or number of hours above selected concentration intervals. None of these statistics has adequately characterized the relationship among concentration, exposure duration, interval between exposures and plant responses. The lack of correlation between exposure statistics and ambient air measurements has posed a major problem for those trying to assess the effects of O_3 exposure.

The implication inherent in the use of mean O_3 concentrations is that all O_3 concentrations are equally effective in causing plant responses. Use of the mean statistic minimizes the importance of peak concentrations by treating low-level long-term exposures the same as high concentration short-

term exposures. Studies with beans and tobacco, however, have shown that a given dose (concentration x time) distributed over a short period induced more injury than did the same dose distributed over a longer period (Heck et al., 1966). Concentration was shown to be approximately twice as effective as time of exposure at causing foliar injury in tobacco plants (Tonneijck, 1984). The results of several other studies also have supported the greater importance of concentration in comparison to time in determining plant response (Bennett, 1979: Heck and Tingey, 1971; Henderson and Reinert, 1979; Reinert and Nelson, 1979; Amiro et al., 1984). Thus, a judgment must be made as to whether greater protection of plants from O_3 exposure is provided by limiting short-term peak exposures, long-term average exposures, or both.

Not only are concentration and time important but the dynamic nature of the O_3 exposure is also important; i.e., whether the exposure is at a constant or variable concentration. Musselman et al. (1983) recently demonstrated that although constant concentrations cause the same type of plant response as variable concentrations at equivalent doses, the constant concentrations had less effect on plant growth responses. This finding has been confirmed by other studies. Ashmore found significant yield reductions in radishes exposed to ambient O_3 when the maximum O_3 concentration exceeded 0.06 ppm at least 10% of the days when the crop was growing. Initial studies have compared the response of alfalfa to daily peak and episodic O_3 exposure profiles which had the equivalent total O_3 dose over the growing season (Hogsett et al., 1985) alfalfa yield was reduced to a greater extent in the episodic that the daily peak exposure. The plants that displayed the greater growth reduction (in the episodic exposure) have a significantly lower 7-hour seasonal mean concentration, thus raising the possibility that the 7-hour seasonal mean may not properly consider peak concentrations. Studies with SO_2 also showed that plants exposed to variable concentrations exhibited a greater plant response than those

exposed to a constant concentration (McLaughlin et al., 1979). In addition, the mean does not specifically include an exposure duration component. Thus, it cannot distinguish between two exposures to the same concentration, but of different durations.

c. Exposure and Response to Peroxyacetyl Nitrate

Response to peroxyacetyl nitrate (PAN) exposure has been assessed by use of the limiting-value method to estimate the lowest PAN concentration and exposure duration required to produce visible injury in plants (Jacobson, 1977). The range of PAN levels and exposure durations which this analysis suggests will produce foliar injury are: 1) 0.20 ppm for 0.5 hr; 2) 0.10 ppm for 1.0 hr; and 3) 0.035 ppm for 4.0 hr. (CD, p. 6-215). To reduce the likelihood of foliar injury to some plants, more recent studies have suggested that PAN exposures may need to be 30 to 40% lower than the values cited above (Tonneijck, 1984).

Few studies are available which report the effects of PAN on growth and yield. A study of the effects on non-vascular plants indicates that growth, photosynthesis, and respiration of algae can be adversely affected by exposure to PAN (Gross and Dugger, 1969). Fumigation of lichens with 0.05 ppm and 0.10 ppm PAN for several days inhibited photosynthesis (Sigal and Taylor, 1979). Vascular plants which have been studied to determine the effects of PAN on growth and yield include radishes, oats, tomatoes, pinto beans, beets, and barley. These plants were exposed in greenhouse studies to PAN concentrations up to 0.04 ppm for 4 hours per day, twice per week, from germination to maturity, with no significant effects (Taylor et al., 1983). In the same study, similar exposures of lettuce and Swiss chard produced yield losses of 13% and 23%, respectively, without visible foliar injury. Although it is possible for severe PAN exposure to make some crops unmarketable, it is unlikely that PAN concentrations sufficient to cause visible injury or reduced yield will occur in the United States except perhaps in some areas of California and a few other localized areas.

d. Economic Assessments of Agriculture

Evidence from the plant science literature clearly demonstrates that O_3 at ambient levels will reduce the yields of some crops (CD, p. 6-246). The fact that such reductions in U.S. agriculture could adversely affect human welfare has resulted in numerous attempts to assess, in monetary terms, the losses from ambient O_3 or the benefits of O_3 control to agriculture. Most of the past 1986 economic assessments focus on specific regions of the country, primarily California and the Corn Belt (Illinois, Indiana, Iowa, Ohio and Missouri). This regional emphasis may be attributed to the relative availability of data on crop response and air quality for selected regions, as well as the national importance of these agricultural regions. Of the regional assessments evaluated in the CD (1986, p. 6-247), Howitt et al., 1984a,b; Rowe et al., 1984; Adams and McCarl, 1985; and Mjelde et al., 1984 are judged to be adequate on the basis of the adequacy of the plant science, aerometric and economic data and the assumptions used in each assessment. Most regional studies however abstract from the interdependencies that exist between regions, which limits their utility in evaluating secondary NAAQS (CD, p. 6-252).

National-level studies can overcome this limitation of regional analyses by accounting for economic linkages between groups and regions. This requires additional data and more complex models and frequently poses more difficult analytical problems. Most of the national assessments conducted since 1978 suffer from either plant science and aerometric data problems, incomplete economic models, or both. As a result of the limitations, caution should be applied in using these estimates to evaluate the efficiency of alternative secondary NAAQS. Two of the studies, both based on NCLAN data, will be briefly summarized here. Both are judged to be adequate in terms of the three critical areas of data inputs (CD, p. 6-252) and together they provide reasonably comprehensive estimates of the economic consequences of changes in ambient levels of O_3 on agriculture.

In the first of these studies, Kopp et al. (1984) measured the national economic effects of changes in ambient O_3 level on the production of corn, cotton, soybean, wheat and peanuts. In addition to accounting for price effects on producers and consumers, the assessment methodology places emphasis on developing producer level responses to O_3 induced yield changes (from NCLAN data) in 200 production regions. The results of the Kopp et al. (1984) study indicated that a reduction in O_3 from 1978 regional ambient levels to a seasonal 7-hr average of approximately 0.04 ppm would result in a $1.2 billion net benefit in 1978 dollars. On the other hand, an increase in O_3 to an assumed ambient concentration of 0.08 ppm (seasonal 7-hr average) across all regions produced a net loss of approximately $3.0 billion (CD, p. 1-87).

The second study, by Adams et al. (1984b) is a component of the NCLAN program. The results were from an economic model of the U.S. agriculture sector individual farms models for 55 production regions. Using NCLAN data, the analysis examined yield changes for six major crops (corn soybeans, wheat, cotton, grain, sorghum and barley) that together account for over 75 percent of U.S. crop acreage. The estimated annual benefit (in 1980 dollars) from O_3 adjustments are substantial, but make up a relatively small percentage of total agricultural output (about 4 percent). In this analysis a 25 percent reduction in O_3 from 1980 ambient levels resulted in benefits of $1.7 billion. A 25 percent increase in O_3 resulted in an annual loss (negative benefit) of 2.363 billion. When adjusted for differences in years and crop coverages, these estimates are quite close to the Kopp et al. (1984) benefit estimates (CD, p. 1-88).

While the estimates from both of Kopp et al. (1984) and Adams et al. (1984b) were derived from conceptually sound economic models and the most defensible plant science and aerometric data available at the time of CD closure, there are several sources of uncertainty including the following:

- the issue of exposure dynamics and whether the 7-hr seasonal mean is an appropriate exposure statistic,
- the lack of environmental interactions in the experiments, particularly water stress,
- the lack of rural monitoring sites in the SAROAD system of EPA, which is necessary for extensive validation of Kriging data,
- the economic models themselves contain many potential sources of uncertainty including the effects of benefits estimates of market-distorting factors such as the Federal Farm Programs. In addition to the revised NCLAN economic assessment, which will be published in 1988, there have been other analyses that explicitly consider the Federal Farm Programs (see SP, p. XI-10).

Despite these uncertainties these two studies, in combination with the underlying NCLAN data on yield effects, provide the most comprehensive economic information to date on which to base decisions regarding the economic efficiency of alternative secondary standards (CD, p. 1-88).

B. Natural Ecosystem Effects

The previous section discussed the responses of individual species of agricultural plants, trees and other native vegetation to O_3. The responses, which are well documented, include: (1) injury to foliage, (2) reductions in growth, (3) losses in yield, (4) alterations in reproductive capacity, and (5) increased susceptibility to pests and pathogens. This section discusses the effects of O_3 stress on simple and complex plant communities to illustrate that such effects, because of the interconnections and relationships among ecosystem components, can produce perturbations in ecosystems. Stresses placed on plant communities and the ecosystems of which they are a part can produce changes that are long lasting and that may be irreversible. No attempts have been made to examine the effects

of PAN on ecosystems since there are little data, and trees and other woody plants appear to be resistant to PAN (CD, p. 7-1).

Evidence indicates that any impact of O_3 on ecosystems will depend on the responses to O_3 of the "producer" community. Producer species (trees and other green plants) are of particular importance in maintaining the integrity of an ecosystem since producers provide, through the process of photosynthesis, all of the new organic matter (food, energy) added to an ecosystem. Ozone-induced changes in photosynthesis influence energy flow and mineral nutrient cycling. Alteration of these processes in ecosystems can set the stage for changes in community structure by influencing the nature and direction of successional changes (Woodwell, 1970; Bormann, 1985), with possibly irreversible consequences (see e.g., Odum, 1985, Bormann, 1985). The sequence of responses outlined by Bormann is given in Table X-6 to assist in understanding ecosystem effects (CD, p. 7-4).

1. Forest Ecosystems

Anthropogenic stress on forested ecosystems may result in reductions in regional tree growth and a decline of stands in more susceptible forest types (Johnson and Siccama, 1983). Atmospheric pollutants, in particular oxidants and acid deposition are important regional factors that are thought to play a major role in the array of stresses affecting forests (Smith 1981; Bormann 1982, 1985). Observations of naturally occurring symptoms or reduced growth as a result of acidic deposition are limited at the present time. New research results on the effects of acidic deposition on forest are expected emerge over the next two to five years.

While the nature and magnitude of the effects of atmospheric pollution on North American forest are still relatively unknown, there is evidence that some forest types are negatively affected by ambient levels of O_3. Among the more susceptible forested areas are the mixed conifer forests of the San Gabriel and San Bernardino mountain ranges east of Los Angeles, which have been

TABLE X-6. CONTINUUM OF CHARACTERISTIC ECOSYSTEM RESPONSES TO POLLUTANT STRESS

Phase	Response characteristics
0	No response occurs. Manmade pollutants are absent or constitute insignificant stress. Plant growth occurs under natural conditions.
I	Ecosystems serve as sinks for pollutants. Species and/or ecosystem functions are relatively unaffected. Self-repair occurs.
II	Sensitive species or individuals are subtly and adversely affected. A reduction in photosynthesis, a change in reproductive capacity, or a change in predisposition to insect or fungus attack may occur.
III	Decline occurs in the populations with sensitive species; some individuals will be lost. Their effectiveness as functional members of the ecosystem diminishes. Ultimately, species could be lost from the system.
IV	Large plants, trees, and shrubs of all species die. The basic structure of the forest ecosystem is changed. Biotic regulation is affected as forest layers are peeled off: first trees, tall shrubs, and, under the most severe conditions, short shrubs and herbs. The ecosystem is dominated by weedy species not previously present and by small scattered shrubs and herbs.
V	The ecosystem collapses. The loss of species and changes in ecosystem structure, nutrients, and soil so damage the system that self-repair is impossible.

Source: Adapted from Bormann (1985).

exposed to oxidant pollution since the early 1950's (Miller, 1973). Ozone was first identified as the agent responsible for the slow decline and death of ponderosa pine trees in these forests in 1962 (Miller et al., 1963). Later, Jeffrey pine was also found to be injured by O_3 exposure. Oxidant injury of eastern white pine has been observed for many years in the eastern United States (although some genotypes of white pine appear to be more tolerant). It was first reported as needle blight in the early 1900s but in 1963 was shown to be the result of acute and chronic ozone exposure (Berry and Ripperton, 1963).

More recently, oxidant injury of eastern white pine in the Blue Ridge Mountains of Virginia has been reported by Hayes and Skelly (1977), Skelly 1980, and Benoit et al. (1982) and on the Cumberland Plateau of east Tennessee by Mann et al. (1980) and McLaughlin et al. (1982). Ozone injury in natural plant communities has been reported by Treshow and Stewart (1973) and by Duchelle et al. (1983). In addition, there is evidence from laboratory studies of visible injury, negative effects on physiological function and reduced productivity as a result of oxidant pollution (Guderian, 1977; Smith, 1981). However, because of the disparity between effects observed in the laboratory and those observed in the field, results from controlled laboratory and greenhouse studies are not easily extrapolated to field conditions and there is very little experimental evidence directly linking ambient ozone concentrations with decline of tree productivity in the field. This section will discuss the effects of O_3 on plant processes and growth and the limited data available on ecosystem response.

a. Effects on Plant Processes

A discussion of individual tree response is the first step in explaining ecosystem response. In forest ecosystems, trees play a critical role. As producers, trees influence the structure, energy flow and nutrient cycling of forest ecosystems (Ehrlich and Mooney, 1983). According to the CD, while some of the same plant processes are affected in trees and agricultural

crop species, perennial plants, because they live longer, must cope with both short- and long-term stresses, the effects of which can be cumulative, lasting over the years, or can be delayed, not becoming apparent for many years. Likewise, effects can possibly be mitigated through short-or long-term recovery or replacements (CD, p. 7-23). Therefore, the permanent vegetation in natural ecosystems receives much greater chronic exposure than the short-lived vegetation that makes up agroecosystems. The single agroecosystem has little resilience to pollutant stress; the natural ecosystem is initially more resistent to pollutant stress because of species diversity, but the longer chronic exposures can disrupt the system. These differences between natural ecosystems and agroecosystems raise a key issue in the debate on the O_3 secondary standard regarding the adequacy of the current 1-hr standard and other exposure indicators under consideration to protect trees as well as crops.

In regard to the effects of O_3 on individual tree response, inhibition or reduction in the rate of photosynthesis is possibly the most significant effect of O_3 entry into the leaves of sensitive plants, although other mechanisms such as increased foliar leaching have been suggested (Taylor and Norby, 1985). Ozone inhibits photosynthesis, decreases formation of organic compounds needed for plant growth, and can alter the transport and allocation of the decreased products of photosynthesis so that sugar storage and root growth are reduced. Trees in which O_3 has been shown to reduce photosynthesis are loblolly pine and slash pine (Barnes, 1972), ponderosa pine (Miller et al., 1969; Coyne and Bingham, 1981), eastern white pine, (Barnes, 1972, Yang et al., 1983; Botkin et al., 1972) black oak, sugar maple (Carlson, 1979), and a poplar hybrid (Furukawa and Kadota, 1975) (Table IX-1, CD, p. 6-28 or SP, pg. IX-4).

Miller et al. (1969), Coyne and Bingham (1981), and Yang et al. (1983) relate visible injury symptoms and reduced tree growth to the effect of O_3 on photosynthesis. Miller et al. (1969) found that exposure of 3-year old ponderosa pine seedlings under

controlled conditions to concentrations of 0.15 and 0.30 ppm 9 hr/day for 30 days reduced photosynthesis by 10 and 70 percent, respectively. In addition, it was noted that a reduction in photosynthesis was accompanied by a decrease in the sugar content of injured needles. Tingey et al. (1976) observed that the amounts of soluble sugars, starch, and phenols tended to increase in the tops and decrease in the plant roots of ponderosa pine seedlings exposed to 0.10 ppm O_3 for 6 hours per day for 20 weeks. The sugars and starches stored in the tree roots were significantly less when compared with the controls (CD, p. 7-14).

Coyne and Bingham (1981) measured photosynthesis and stomatal conductance of attached ponderosa pine needles in relation to cumulative O_3 dose. The decline in photosynthesis and stomatal function normally associated with aging was accelerated as O_3 injury symptoms increased. Premature senescence and abscission of needles occurred soon after photosynthesis reached its lowest level. In a study of white pine, Yang et al. (1983) also observed that decrease in rates of photosynthesis due to O_3 exposure was closely associated with visible needle injury, premature senescence and reduction in biomass (CD, p. 7-13). Thus, the impact of O_3 on important plant processes such as photosynthesis does seem to be reflected in the occurrence of other symptoms of O_3 injury.

b. Effects on Growth

The limited observations of tree response to O_3 in the field and the chamber data that is currently available (Table X-7) provide the strongest evidence to date of tree response to O_3. Studies made along the Blue Ridge Parkway support the view that exposure to O_3 reduces growth in sensitive trees (Benoit et al., 1982). Eastern white pine located in experimental plots situated along the Blue Ridge Parkway were studied to determine the radial growth increment during the 1955 to 1978 period. Growth of trees classified as sensitive was 25 percent less than in tolerant trees; growth was 15 percent less in trees classified as intermediate in sensitivity. Mean radial increments for all

trees during the last 10 years of the study were smaller than for the previous 24 years. During the period of the study, concentrations of 0.05 to 0.07 ppm of O_3 were recorded on a recurring basis, with episodic peaks of 0.12 ppm or higher occurring (Benoit et al., 1982).

Hayes and Skelly (1977) monitored total oxidants and recorded oxidant related injury on eastern white pine in three rural Virginia sites between April 1975 and March 1976. Injury was associated with total oxidant peaks of 0.08 ppm for 1-hr or more. Peak 1-hr ozone concentrations of 0.17 have been measured in the Blue Ridge Mountains (Skelly, 1980). Monthly 8-hr average O_3 concentrations ranged from 0.035 to 0.065 ppm during the oxidant season (April - October) and peak hourly concentrations ranged from 0.08 to 0.13 ppm (Skelly et al., 1984). Increased injury symptoms were observed on pine trees previously classified as sensitive or intermediately sensitive after an O_3 episode. Hayes and Skelly (1977) suggested that continued exposure of white pine to acute and chronic oxidant concentrations could influence their vigor and reproductive ability, ultimately resulting in replacement by tolerant species.

A steady decline in annual ring increments was also noted on the Cumberland Plateau of east Tennessee during the years 1962 to 1979 (McLaughlin et al., 1982). A reduction of 70 percent in average annual growth and 90 percent in average bole growth was observed in sensitive white pine when compared to both trees classified as tolerant to O_3 and trees of intermediate sensitivity. Decline was attributed primarily to chronic exposure to O_3, which frequently occurred at phytotoxic concentrations in the area. For the years 1975-1979 the incidence rates for hourly concentrations $\geq$0.08 ppm ranged from 129-339 hours above 0.08 ppm. Maximum 1-hr concentrations range from 0.12 to 0.2 ppm during this time period. The reduction in growth on the Blue Ridge Parkway and on the Cumberland Plateau, as in the case of the San Bernardino Mountains, was correlated with the predisposing symptoms of chronic decline, which includes

the following sequence of events and conditions: (1) premature senescence and loss of older needles; (2) reduced storage capacity of carbohydrates in the fall and resupply capacity in the spring to support new needle growth; (3) shorter new needles, resulting in lower gross photosynthetic productivity; (4) reduced availability of photosynthate for external usage (including repair of chronically stressed tissues of older needles); and (5) premature casting of older needles (McLaughlin et al., 1982). Degeneration of feeder roots and mycorrhizae usually precedes the onset of above ground symptoms (Manion, 1981). Decreases in nutrient and water uptake may also occur. These changes produce weakened trees. Weakened trees are, in turn, predisposed to attack by root rot fungi, to defoliation by insects, and to attack by the pine beetle. Growth reductions in trees (Table X-7) grown under controlled conditions have also been observed (Mooi, 1980; Kress et al., 1982a,b,c; Kress and Skelly, 1982; Jensen, 1979; McClenahen, 1979; Jensen and Dochinger, 1974; Jensen and Masters, 1975) (CD, p. 6-134).

Injury by O_3 to native herbaceous vegetation growing in the Virginia mountains was also observed (Duchelle et al., 1983). Ambient O_3 concentrations were shown to reduce growth and productivity of graminoid and forb vegetation in the Shenendoah National Park. For each year of the study, biomass production was greatest for vegetation grown in filtered-air chambers. The total 3-year cumulative dry weight of plants in filtered chambers was significantly greater than that in non-filtered and open-air plots. Common milkweed and common blackberry were the only two native species to develop foliar injury. Ozone episodes occurred several times during the period of the study. Peak hourly concentrations ranged from 0.08 to 0.12 ppm; however, monthly hourly average concentrations ranged from 0.03 to 0.06 ppm.

TABLE X-7. EFFECTS OF OZONE ADDED TO FILTERED AIR ON THE YIELD OF SELECTED TREE CROPS

Plant species	O_3 concn., ppm	Exposure duration	Yield, % from control[a]	Monitoring method[b]	Calibration method[c]	Fumigation facility[d]	Reference
Poplar (Dorskamp)	0.041	12 hr/day, 5 mo	+14[e], stem length; 12 stem dry wt; +1333, no. of dropped leaves; 6, total dry wt	Chem	NBKI	GH-CH	Mooi (1980)
(Zeeland)	0.041	12 hr/day, 5 mo	2, stem length; 4, stem dry wt; +692, no. of dropped leaves; 0, total dry wt	Chem	NBKI	GH-CH	Mooi (1980)
American Sycamore (16-SYC-19)	0.05	6 hr/day, 28 days	9[e], height growth	Chem	1% NBKI	CH	Kress et al. (1982b)
(16-SYC-23)	0.05	6 hr/day, 28 day	2, height growth	Chem	1% NBKI	CSTR	Kress et al. (1982b)
American Sycamore (16-SYC-19)	0.05	6 hr/day, 28 days	11, height growth	Chem	Constant source, NBKI, UV	CSTR	Kress and Skelly (1982)
(16-SYC-23)	0.05	6 hr/day, 28 day	9[e], height growth	Chem	Constant source, NBKI, UV	CSTR	Kress and Skelly (1982)
Sweetgum	0.05 0.10 0.15	6 hr/day, 28 days	+9[e], height growth; 10, total dry wt 29[e], height growth; 26[e], total dry wt 45[e], height growth; 42[e], total dry wt	Chem	Constant source, NBKI, UV	CSTR	Kress and Skelly (1982)
American Sycamore	0.05 0.10 0.15	6 hr/day, 28 days	+4[e], height growth; 23, total dry wt 27[e], height growth; 61[e], total dry wt 21[e], height growth; 69[e], total dry wt	Chem	Constant source, NBKI, UV	CSTR	Kress and Skelly (1982)
White ash	0.05 0.10 0.15	6 hr/day, 28 days	+12, height growth; +22[e], total dry wt 9, height growth; 9[e], total dry wt 15, height growth; 17[e], total dry wt	Chem	Constant source, NBKI, UV	CSTR	Kress and Skelly (1982)
Green ash	0.05 0.10 0.15	6 hr/day, 28 days	2[e], height growth; 14, total dry wt 24[e], height growth, 28, total dry wt 30[e], height growth; 33, total dry wt	Chem	Constant source, NBKI, UV	CSTR	Kress and Skelly (1982)

TABLE X-7. (cont.) EFFECTS OF OZONE ADDED TO FILTERED AIR ON YIELD OF SELECTED TREE CROPS

Plant species	O_3 concn., ppm	Exposure duration	Yield, % from control[a]	Monitoring method[b]	Calibration method[c]	Fumigation facility[d]	Reference
Willow oak	0.05 0.10	6 hr/day, 28 days	1, height growth; 2, total dry wt 4, height growth; 11, total dry wt	Chem	Constant source, NBKI, UV	CSTR	Kress and Skelly (1982)
Sugar maple	0.05 0.10 0.15	6 hr/day, 28 days	5, height growth; 2, total dry wt +8[e], height growth; 7, total dry wt 12[e], height growth; 41[e], total dry wt	Chem		CSTR	Kress and Skelly (1982)
Yellow poplar	0.05 0.10 0.15	6 hr/day, 28 days	+60[e], height growth; +41, total dry wt +8, height growth; +5, total dry wt 12, height growth; +18, total dry wt	Chem		CSTR	
Yellow poplar	0.10	12 hr/day, 48 days	19[ng], relative growth rate	Chem	Not given	CSTR	Jensen (1981)
Cottonwood	0.10		59[ng], relative growth rate				
White ash	0.10		no significant effects				
White ash	0.10 0.20 0.30 0.40	4 hr/day, 1 day/wk, 9 wk	+13, total height; +7, shoot dry wt 0, total height; +5, shoot dry wt 0, total height; 11, shoot dry wt 0, total height; 14, shoot dry wt	Not given	Not given	Not given	McClenahen, (1979)
Black cherry	0.10 0.20 0.30 0.40		+16, total height; +15, shoot dry wt +5, total height; 4, shoot dry wt +3, total height; 4, shoot dry wt 28[e], total height; 15, shoot dry wt	Not given	Not given	Not given	McClenahen, (1979)
Hybrid poplar (NS 207 + NE 211)	0.15	8 hr/day, 5 days/wk, 6 wk	50[e], dry wt new shoots from terminal cuttings 62[e], dry wt new shoots from basal cuttings	Not given	Not given	GH-CH	Jensen and Dochinger (1974)

TABLE X-7. (cont.) EFFECTS OF OZONE ADDED TO FILTERED AIR ON YIELD OF SELECTED CROPS

Plant species	O_3 concn., ppm	Exposure duration	Yield, % from control[a]	Monitoring method[b]	Calibration method[c]	Fumigation facility[d]	Reference
Hybrid poplar (207)	0.20 0.20	7.5 hr/day, 5 day/wk, 6 wk	5, height 8, height	Not given	Not given	CH	Jensen (1979)
Yellow birch	0.25	8 hr/day, 5 day/wk,	9, height 15 wk	MAST	NBKI	GH-CH	Jensen and Masters (1975)
White birch	0.25		34, height				
Bigtooth aspen	0.25		+7, height				
Eastern cottonwood	0.25						
Red maple (163 ME)	0.25	8 hr/day, 6 wk	18, height	MAST	1% NBKI	CH	Dochinger and Townsend (1979)
(167 NB)			32, height				
(128 OH)			37[e], height				
Loblolly pine (4-5 x 523)	0.05	6 hr/day, 28 days	6, height growth	Chem	1% NBKI	CH	Kress et al. (1982a)
(14-5 x 517)	0.05						
Loblolly pine	0.05 0.10 0.15	6 hr/day, 28 days	18[e], height growth; 14[e], total dry wt 27[e], height growth; 22[e], total dry wt 41[e], height growth; 28[e], total dry wt	Chem	Constant source, NBKI, UV	CSTR	Kress and Skelly (1982)
Pitch pine	0.05 0.10 0.15	6 hr/day, 28 days	4, height growth; 8, total dry wt 13[e], height growth; 19[e], total dry wt 26[e], height growth; 24[e], total dry wt				

TABLE X-7. (cont.) EFFECTS OF OZONE ADDED TO FILTERED AIR ON YIELD OF SELECTED CROPS

Plant species	O_3 concn., ppm	Exposure duration	Yield, % from control[a]	Monitoring method[b]	Calibration method[c]	Fumigation facility[d]	Reference
Virginia pine	0.05 0.10 0.15	6 hr/day, 28 days	5, height growth; +2, total dry wt 11, height growth; 3, total dry wt 14, height growth; 13, total dry wt	Chem	Constant source, NBKI, UV	CSTR	Kress and Skelly (1982)
White spruce	0.25	8 hr/day, 5 day/wk, 15 wk	5, height	Mast	NBKI	GH-CH	Jensen and Masters (1975)
Japanese larch	0.25		+6, height	Mast	NBKI	GH-CH	

[a] + = an increase above the control; ng = statistical data not given.
[b] Chem. = chemiluminescence; Mast = Mast meter (coulometric); UV = ultraviolet spectrometry.
[c] NBKI = neutral buffered potassium iodide.
[d] GH = greenhouse; CSTR = continuous stirred tank reactor; CH = manufactured chamber other than CSTR or GC; GH-CH = CH in greenhouse.
[e] Significant at p = 0.05.

Source: O_3 Criteria Document, U.S. EPA, 1986

Treshow and Stewart (1973) conducted one of the few studies investigating the impact of air pollution on natural plant communities. Grassland, oak, aspen, and conifer communities in Salt Lake Valley, Utah were studied. Two dominant species, cheatgrass and aspen, considered key to community integrity were found to be sensitive. In both cases single 2-hr exposures to 0.15 ppm O_3 caused severe injury. Removal of the dominant species from plant communities could result in a shift to another species. In a companion study conducted in portable plastic chambers, O_3 exposures of 0.15 to 0.3 ppm for 3-hr per day 5 days/week throughout the growing season reduced root and top growth and fewer seeds were produced (Harward and Treshow, 1973).

c. Ecosystem Responses: The San Bernardino Study

The interdisciplinary study of the decline of the pine and mixed conifer forest in the San Bernardino Mountains is the most comprehensive and best documented report available on the effects of oxidants on an ecosystem (Miller et al., 1982). While San Bernardino may be regarded as a worst case scenario due to the high levels of O_3 found in Southern California, it still provides valuable information on the potential consequences of ecosystem exposure to O_3.

Many of the major ecosystem processes were shown by the study to be affected directly or indirectly. Ozone associated stress on trees decreased photosynthesis, affected directly or indirectly translocation of carbon (energy), mineral nutrients, and water, and reduced trunk diameter, tree height and seed production in ponderosa and Jeffrey pine (Miller et al., 1982). Foliar injury of O_3 sensitive ponderosa and Jeffrey pines was observed when 24-hr O_3 concentrations ranged from 0.05 to 0.06 ppm. During the period of the study, average 24-hr O_3 concentrations during the months of May through September ranged from a background of 0.03 ppm to a maximum of 0.10 to 0.12 ppm. A comparison of radial growth of ponderosa pine from 1910 to 1940, a period of low pollution (<0.03 ppm), with the years 1941 to 1971, a period of high pollution (0.03 to 0.12 ppm), indicated

that O_3 exposure reduced the average annual radial growth by approximately 40 percent, height by 25 percent, and wood volume by 84 percent in trees less than 40 years of age. The marketable volume of trees 30 years of age was reduced by 83 percent in the areas with the highest O_3 concentrations (Miller and Elderman, 1977). In addition, stressed pines also became more susceptible to root rot and pine beetle as a result of weakening by photochemical oxidants (Stark and Cobb, 1969).

Ozone-induced stress on sensitive ponderosa and Jeffrey pine and to a lesser extent on sensitive white fir, black oak, increase cedar and sugar pine, accompanied by fire, brought about the removal of the pine forest overstory. A shift in dominance to self-perpetuating fire adapted, O_3 tolerant shrub and oak species resulted. These mixtures provide fewer commodity and amenity values than the former pine forest (Miller et al., 1982).

The case of San Bernardino suggests that a potential consequence of O_3 stress is a change in the composition and successional patterns of some plant communities (Woodwell, 1974). With regard to forests in eastern North America, both the extent of the decline and the causal mechanisms remain controversial (Taylor and Norby, 1985). Changes in the growth patterns of eastern white pine have been attributed to stress resulting from O_3 exposure that began 15 to 20 years earlier (Miller and Elderman, 1977; Miller et al., 1982; McLaughlin et al., 1982). More recently, dendroecological studies of the decline of red spruce in the northeast (Johnson and Siccama, 1983) and of reduced growth rates of red spruce, balsam fir and frasier fir in central West Virginia and western Virginia also provide further evidence that the reductions in growth and mortality measurable today probably began at least 20 years ago. In addition, reductions in growth rates of loblolly and short leaf pine have been reported in the piedmont regions of the southeastern U.S. (McLaughlin, 1985).

In regard to these most recent declines, there is currently no agreement as to the trigger factor that precipitated the

dieback, mortality and decreased growth. A number of stresses have been identified including natural processes and air pollution (Johnson and Siccama, 1983). Given the regional distribution of O_3 in North America, the frequent occurrence of elevated O_3 concentrations, and the recognized effects of O_3 on agricultural productivity in the region, the potential influence of O_3 on forest ecosystems should be given high priority (Taylor and Norby, 1985).

2. Interrelated Ecosystems

a. Aquatic Ecosystems

It is extremely important to consider that an adverse impact on a forest or agricultural ecosystem may in turn adversely affect adjacent aquatic systems. A variety of linkages for energy and nutrient exchange exist. Disruptions induced by air pollution stress on terrestrial ecosystems often trigger dysfunctions in neighboring aquatic ecosystems, such as streams, lakes, and reservoirs. Sediments resulting from erosion can change the physical character of stream channels, causing changes in bottom deposits, erosion of channel banks, obstruction of flow, and increased flooding. They can fill in natural ponds and reservoirs. Finer sediments can reduce water quality, affecting public and industrial water supplies and recreational areas. Turbidity caused by increased erosion can also reduce the penetration of light into natural waters. This, in turn, can reduce plant photosynthesis and lower supplies of dissolved oxygen, leading to changes in the natural flora and fauna (Bormann and Smith, 1980). Significant forest alterations, therefore, may have a regional impact on nutrient cycling, soil stabilization, sedimentation, and eutrophication of adjacent or nearby aquatic systems. Interfacing areas, such as wetlands and bogs, may be especially vulnerable to impact (CD, p. 7-47).

b. Agricultural Ecosystems

Natural and agricultural ecosystems possess the same basic functional components, require energy flow and mineral cycling

for maintenance, and are subject to the dominating influences of climate and substrate. Natural ecosystems vary in diversity from simple systems with few species to complex systems with many species. Their populations also vary in genetic composition, age, and species diversity. They are self-regulating and self-perpetuating. Agroecosystems, on the other hand, are usually highly manipulated monocultures of similar genetic and age composition and are unable to maintain themselves without the addition of nutrients, energy, and human effort; opportunistic native and imported species may invade the sites. The manipulation of monocultures is designed to concentrate ecosystem productivity into a particular species to maximize its yield (e.g., corn, wheat, soybeans) for the benefit of humans (Cox and Atkins, 1979). If any of the species, varieties, or cultivars is very sensitive to O_3, its market value can be destroyed. When this occurs, efforts are made to find a resistant cultivar, as with tobacco, or to grow a crop less sensitive to O_3 stress. Cost alone would prevent replacement of the variety of species in a natural ecosystem.

C. Materials Damage

Research over a period of more than two decades has shown that O_3 has the ability to react with both manmade and natural materials. While some research has been done on the effects of total oxidants on materials, the only components of total oxidants that have been studied individually are O_3 and NO_2. In spite of the research focus on O_3, however, the amount of damage from O_3 to actual in-house materials remains poorly characterized.

The materials known to be most susceptible to O_3 attack are elastomers, textile fibers and dyes, and certain types of paint. Ideally, the nature and amount of O_3 damage to the materials could be approximated by physical damage functions in combination with ambient air concentrations. The economic impact of O_3 related damage could then be estimated by using accelerated

repair and replacement costs. Because little recent work has been reported on the effects on nonbiological materials, however, it is necessary to rely on older studies which, in many cases, do not provide sufficient information to assess the amount and costs of oxidant related damage.

1. Elastomers

The effects of O_3 on elastomers are the best documented. Natural rubber and synthetic polymers/copolymers of butadiene, isoprene, and styrene account for the bulk of elastomer production for products such as automobile tires and protective electrical coverings. The mechanism of O_3 damage on elastomers shares similarities and differences with simple oxidation from atmospheric oxygen. Ozone damage, usually in the form of cracking, tends to be more of a surface phenomenon than simple oxidation. It is greatly accelerated by mechanical stress, which produces fresh surface areas at crack boundaries. Simple oxidation, on the other hand, is slower; it occurs more in the bulk of a material, and it is less affected by the degree of stress (Mueller and Stickney, 1970). At pollutant concentrations and stress levels normally encountered outdoors (and in many indoor environments), the elastomer hardens or becomes brittle and cracked, losing its physical integrity. High humidity and mechanical stress greatly affect the formation, depth of cracking, and, in automotive tires, the adhesion piles (Davies, 1979; Wenghoefer, 1974) (CD, p. 8-3).

O_3 affects natural rubber and other elastomers in a dose related fashion. Dose is defined in materials research as the product of concentration and duration of exposure. The importance of O_3 dose was demonstrated by Bradley and Haagen-Smit (1951), who used a specially formulated O_3 sensitive natural rubber (NR). Samples exposed to 20,000 ppm cracked almost instantaneously, and those exposed to lower concentrations took a proportionately longer time to crack. Cracking occurred at a

rate of 0.02 to 0.03 ppm-hr over the entire range of concentrations.

Haynie et al. (1976) exposed samples of a tire sidewall to O_3 at concentrations of 0.08 and 0.5 ppm for 250 to 1000 hr under 10-20 percent strain. From these and other data, they estimated that at the O_3 standard of the time (0.08 ppm, 1-hr average), and at the annual NO_x standard of 0.05 ppm, it would take 2-5 years for a crack to penetrate cord depth. In addition to stress, factors affecting the cracking rate include atmospheric pressure, humidity, sunlight, and other atmospheric pollutants. Veith and Evans (1980) found a 16-percent difference in cracking rates reported from laboratories located at various geographic locations (CD, p. 8-48).

Ozone has been found to affect the adhesion of piles (rubber-layered strips) in tire manufacturing. Exposures to O_3 concentrations of 0.05 to 0.15 for a few hours significantly decreased adhesion in an NR/SBR blend, causing a 30 percent reduction at the highest O_3 level. This adhesion problem worsened at higher relative humidities. Wenghoefer (1974) showed that O_3 (up to 0.15 ppm), especially in combination with high relative humidity (up to 90 percent), caused greater adhesion losses than heat and NO_2 did, with or without high relative humidity (CD, p. 8-49).

2. Textile Fibers and Dyes

The effects of O_3 on dyes have been known for nearly three decades. In 1955, Salvin and Walker exposed certain red and blue anthraquinone dyes to 0.1 ppm O_3 and noted fading which until that time had been thought to be caused by NO_2. Subsequent work by Schmitt (1960, 1962) confirmed the fading action caused by O_3 and the importance of relative humidity in the absorption and reaction of vulnerable dyes. Later Beloin (1972, 1973) noted the acceleration in fading of certain dyes at an O_3 concentration of 0.05 ppm and a relative humidity of 90 percent (CD, p. 8-49).

Both the type of dye and the material in which it is incorporated are important factors in the resistance a fabric has to O_3. Haylock and Rush (1976, 1978) showed that anthraquinone dyes on nylon fibers were sensitive to fading from O_3 at a concentration of 0.2 ppm for 16 hrs, while Haynie et al. (1976) and Upham et al. (1976) found no effects from O_3 concentrations of 0.1 to 0.5 on royal blue acetate, red rayon-acetate, or plum cotton. Field studies by Nipe (1981) and laboratory work by Kamath et al. 1982 showed a positive association between O_3 levels and dye fading of nylon materials at an O_3 concentration of 0.2 ppm and at various relative humidities. In summary, dye fading is a complex function of O_3 concentration, relative humidity, and the presence of other gaseous pollutants. At present, the available research is insufficient for quantifying the amount of damage to fibrous materials attributable to O_3 alone. Anthraquinone dyes incorporated into cotton and nylon fibers appear to be the most sensitive to O_3 damage (CD, p. 8-50).

The degradation of fibers from exposure to O_3 is poorly characterized. In general, most synthetic fibers such as modacrylic and polyester are relatively resistant; and cotton, nylon, and acrylic fibers show variable sensitivities to the gas. Ozone reduces the breaking strength of these fibers, and the degree of reduction depends on the amount of moisture present. Under laboratory conditions, Bogaty et al. (1952) found a 20 percent loss in breaking strength in cotton textiles under high-moisture conditions after exposure to a 0.06 ppm concentration of O_3 for 50 days. They equated these conditions to a 500- to 600-day exposure under natural conditions. Kerr et al. (1969) found a net loss of 9 percent in breaking strength of moist cotton fibers exposed to O_3 at a concentration of 1.0 ppm for 60 days. The limited research in this area indicates that O_3 in ambient air may have a minimal effect on textile fibers, but additional research is needed to verify this conclusion (CD, p. 8-50).

3. Paints

The effects of O_3 on paint are small in comparison with those of other factors (Campbell et al., 1974). Past studies have shown that, of various paints, only vinyl and acrylic coil coatings are affected (Haynie et al., 1976), and that this impact has a negligible effect on the useful life of the material coated. Preliminary results of current studies have indicated a statistically significant effect of O_3 and relative humidity on latex house paint, but final results are needed before conclusions can be drawn.

Pigments in artists' paints have also been tested under controlled conditions for 3 months at an average exposure level of 0.4 ppm of O_3. While fading occurred in anthraquinone-based pigments, no quantitative information on dose-response relationships is available.

4. Conclusion

Among the various materials studied, research has narrowed the type of materials most likely to affect the economy from increased O_3 exposure. These include elastomers and textile fibers and dyes. Among these, natural rubber, used for tires is probably the most economically important. While the limitations of McCarthy et al. (1983) preclude the reliable estimation of damage costs, the figures indicate the magnitude of potential damage from exposures to O_3 in ambient air (CD, p. 8-46).

D. Effects on Personal Comfort and Well-Being

The Clean Air Act requires that secondary NAAQS for a pollutant specify a level of air quality which is adequate to protect public welfare from any known or anticipated adverse effects. Section 302(h) includes personal comfort and well-being in referring to effects on welfare. Those effects of a pollutant on humans which are not identified as being adverse health effects but do affect personal comfort and well-being are covered by this provision. Symptoms are defined generally as subjective

evidence of disease or physical disturbance but are not necessarily adverse health effects.

Symptomatic effects associated with human exposure to O_3 and other photochemical oxidants may contribute to a reduction in personal comfort and well-being. Similar, but not identical, symptoms have been reported for clinical O_3 and community photochemical oxidant exposures. Eye irritation, for example, is commonly associated with ambient photochemical oxidant levels of about 0.10 ppm but does not occur during controlled O_3 exposures at much higher levels than found in ambient air. Symptoms such as nose and throat irritation, chest discomfort, cough and headache have been reported at > 0.10 ppm O_3 in epidemiology studies (Hammer et al., 1974; Makino and Mizoguchi, 1975; Okawda et al., 1979) and at $\geq$ 0.12 ppm in controlled studies (McDonnell et al., 1983; Avol et al., 1984; Kulle et al., 1985). Impairment of athletic performance in high school students may have been caused by irritative symptoms associated with 0.12 ppm oxidants (Wayne et. al., 1967). Other symptoms commonly reported in clinical O_3 studies are throat dryness, difficulty or pain during deep inspiration, chest tightness, substernal soreness or pain, wheezing, lassitude, malaise, and nausea. CASAC recommended that these effects be considered health effects in developing a basis for the primary standard for O_3.

E. Related Welfare Effects Information and Issues

In its public meetings of April 20-21, 1986, December 14-15, 1987, and December 14-15, 1988 on the CD, staff paper, and NAAQS-related analyses, the Clean Air Scientific Advisory Committee (CASAC) discussed preliminary new information relevant to the possible need for additional standard(s) to protect public health from exposure to O_3. At the time of the meetings much of the information was unpublished and not incorporated into the CD. Subsequently this new information base has been reviewed and largely incorporated by ECAO into the CDS. For a more detailed

discussion and review of individual studies the reader is referred to that document.

The purpose of this section is to provide an overview of the key issues of concern for the secondary standard: relationships of various air quality indicators, crop loss estimates, averaging times and forest response to O_3. This section will summarize new analyses that attempt to address these issues and discuss key new studies that relate to the issue of averaging time(s). The review of new data in this section and in ECAO's Summary of Selected New Information on Effects of Ozone on Health and Vegetation will provide input to staff conclusions and recommendations in Chapter XI. For convenience of readers, citations for new research reviewed in this section are included in as part of the reference list at the end of this staff paper. The CDS provides a more current reference list and discussion of O_3 welfare studies published since closure of the CD.

Past research, as well as current scientific opinion indicate that the current air quality standard is not fully protective of all vegetation (Reich and Amundson, 1984; CASAC, 1986). Yet because of uncertainties in the data base for crops, and the paucity of data regarding forest response to O_3, the averaging time, the level of the standard and even the need for a separate secondary standard remain difficult issues to resolve.

The issue of averaging times and form of the standard has proven to be one of the most difficult issues associated with the review of the O_3 secondary standard. EPA's previous recommendation (CD) that serious consideration be given to setting both a 1-hr and a longer-term secondary standard was based on two findings:

- That forest trees, because they are perennials, experience chronic O_3 stress due to exposure to long-term O_3 concentrations
- That the relationship between peak values and seasonal averages is generally not predictable with any degree of confidence; therefore the 1-hr standard may not

adequately reduce the probability of a high chronic exposure.

While CASAC members (CASAC, 1987) strongly endorsed the judgment that repeated peaks are critical in eliciting plant response, the Committee's views regarding the need for a long-term standard were less clear. Although some members expressed concern that long-term, low level O_3 exposures could adversely affect vegetation, there was reluctance to suggest a long-term standard in the range suggested by the data (0.04-0.06 ppm) because of the obvious conflict with background levels in many areas of the country. The CASAC challenged EPA to identify a single standard formulation that would offer protection from both repeated peaks of concern and long-term exposures. While monthly standard and was specifically mentioned for consideration, it was acknowledged that this exposure statistic would not be ideal in terms of fully capturing extreme situations or biological response.

In response to these CASAC comments several analyses were undertaken and were presented at the December, 1987 CASAC meeting. While CASAC found the results of the new analyses interesting and encouraged EPA to pursue the analysis of alternative exposure indicators with Corvallis, they rejected EPA's recommendation to continue the standard review until the analyses on alternative exposure indicators is complete. Rather, they endorsed the idea of making a decision on retaining the 1-hr standard with the information currently available. There was some difference of opinion among the committee members as to whether the current standard or a lower level would be adequate to protect vegetation. EPA has recently completed an initial draft (Lee et al., 1988c) of the analysis of alternative exposure indicators requested by CASAC. These results are considered preliminary and will be undergoing peer-review. In addition to the analysis, a few new studies have been completed which look at various aspects of the exposure dynamics issue. The analysis and

the new studies, as well as CASAC's response to them at the December 1988 meeting, are discussed in Section E.3.

1. Air Quality Analyses

Air quality analyses and, in particular, relationships between alternative air quality indicators, are critical in the selection of an averaging time for the secondary standard. While previous analyses have indicated that relationships between peak and mean statistics are generally not stable or predictable with any degree of confidence (Johnson et al., 1986; Lefohn, 1984; Heck et al., 1984a, 1984b; Larsen and Heck, 1983) none of the analyses specifically examined the type of compromise indicator that was suggested by CASAC (CASAC, 1986). An analysis by McCurdy (1987) is the latest effort to examine relationships between various types of air quality indicators. These results have been provided in separate reports and summarized in Appendix A.

For purposes of this discussion, it is interesting to note that after examining several short-term peak, multiple peak, and long-term average indicators, McCurdy found that all three exposure patterns of interest are highly correlated with two monthly forms of the standard: maximum monthly mean of the 1-hr daily maximums and maximum monthly mean of the 8-hr daily maximums (SP, p. A-22). The first indicator performed better than the second, but because they are highly correlated with each other ($r = .95$), either one could be used. It appears from this analysis that if a single secondary O_3 NAAQS which provides reasonable protection from both repeated peaks and long-term exposures of concern is desired, it should be the maximum monthly mean of the daily maximum 1-hr averages (max monthly mean). It should be noted that this exposure statistic is largely based on purely statistical concerns, as there is little or no data for a one month exposure period. The same is likely to be true for other similar compromise exposure statistics.

2. Crop Loss Estimates

As stated above, the max monthly mean appears to be an appropriate statistic in terms of the protection it affords from repeated peaks and long-term average concentrations of concern; however, there is little or no effects data for a one month exposure period. This makes it difficult to arrive at crop loss estimates for a monthly indicator.

In an effort to arrive at crop loss estimates for a monthly indicator, Larsen has analyzed new exposure indicators in his existing crop loss model. This model (Larsen and Heck, 1984) was adapted from the Larsen-Heck lognormal plant injury model (Larsen and Heck, 1976) to estimate the impact of O_3 on crop yield. This model uses the "effective mean" O_3 concentration (defined in the next paragraph) to adjust for the greater effect of peak O_3 concentrations. While calculation of the effective mean is somewhat cumbersome for standard-setting, the Larsen-Heck crop reduction model does allow one to look at other exposure indicators and the crop loss estimates associated with them.

The "effective mean" O_3 concentration can be defined by comparing it with the arithmetic mean. Estimates of crop loss as a function of an arithmetic mean O_3 concentration essentially assume that crop loss is proportional to the summation of all (seasonal daytime) 1-hr average O_3 concentrations. Some studies suggest (Larsen and Heck, 1984) that peak 1-hr average O_3 concentrations cause a much greater than proportional effect, suggesting that effects may be proportional to a new dose parameter called air pollution "impact," a parameter that is the summation of all (seasonal daytime) 1-hr average O_3 concentrations raised to the 2.66 power. The "effective mean" is the 2.66th root of impact divided by the number of hourly O_3 concentrations that are exponentiated:

$$m_e = [(\Sigma c_h^{2.66})/n]^{1/2.66}$$

The arithmetic mean can be calculated by merely replacing 2.66 with 1 in this equation, thus showing the parallel construction of the arithmetic and effective means.

Larsen et al. (1988) discussed several crop reduction dose-response mathematical models and concluded that the lognormal, Weibull, and Box-Tidwell models are all closely related. When fitted to data from the National Crop Loss Assessment Network (NCLAN), all three models produce similar dose-response plots. The Larsen-Heck dose-response mathematical model was used to estimate percent crop reduction for soybeans: the above "effective mean" O_3 concentration was the "dose" and the lognormal model was used to estimate the "response." This model was used to estimate soybean percent crop reduction at each agricultural site in the National Aerometric Data Bank (NADB) for each year of years 1981-1985.

Larsen et al. (1988) calculated fourteen O_3 concentration parameters for each site year of data. The potential ambient standards that would limit crop reduction to 5, 10, 15 or 20 percent of agricultural sites are summarized in Table X-8. The three of these parameters that correlate best with crop reduction are the effective mean O_3 concentration (1 percent of the variance unexplained), an arithmetic mean O_3 concentration (4 percent unexplained), and the maximum 1 month mean of daily maximum 1-hr O_3 concentrations (15 percent unexplained). Potential O_3 standards can be selected to achieve a desired response. For instance, if no more than 15 percent crop reduction were desired at any agricultural NADB site, a second highest daily maximum 1-hr O_3 standard of 0.099 ppm could be used. A maximum monthly mean standard (of 1-hr daily maximum) of 0.072 would be required to achieve the same protection. Table X-8 indicates that 65% of agricultural NADB sites achieved this potential standard in 1981-1985 as opposed to 40% of sites that achieved the second high daily max standard of 0.099 ppm.

Table X-8. Potential Ambient Ozone Standards that Would Llimit Soybean Crop Reduction to 5, 10, 15, or 20 Percent

Ozone Parameter	Ambient ozone standard that would limit soybean crop reduction to stated percentage				Percentage of agricultural NADB sites that achieved this potential standard in years 1981-1985				Percentage of variance unexplained by regression of soybean crop reduction against this ozone parameter
	5%	10%	15%	20%	5%	10%	15%	20%	
Second highest daily maximum 1-h ozone conc (ppm)	.059	.079	.099	.119	2	10	40	74	42
Maximum 1-month mean of daily maximum 1-h ozone conc (ppm)	.048	.060	.072	.085	5	26	65	91	15
Maximum 1-month mean of daily maximum 8-h ozone conc (ppm)	.042	.052	.062	.073	5	26	64	89	17
Maximum 3-month mean of daily maximum 8-h ozone conc (ppm)	.034	.044	.054	.064	3	15	48	85	24
Number of days/yr that 8-h daily max. conc exceeds .08 ppm	-	-	6	25	-	-	17	61	27
Summer daytime arithmetic mean ozone conc (ppm)	.035	.045	.055	.065	9	36	70	93	4
Summer daytime effective mean ozone conc (ppm)	.042	.052	.063	.073	14	45	77	94	1

Source: Larsen et al., 1987a

As mentioned earlier the "effective mean" O_3 concentration adjusts for the greater effect of peak O_3 concentrations. Several sites may have identical arithmetic mean values, but the higher the peak concentration at one of these sites, the higher will be the effective mean value and the expected effects at that site.

The exponent of 2.66 on the concentration term for the effective mean was derived from injury studies and then applied to yield studies without validating its applicability to yield (CD, 6-159). Larsen et al. (1988) did an additional analysis on this issue and concluded that crop reduction is probably closely analogous to growth suppression, leaf dry weight reduction, and percent leaf injury; thus, the average exponent of 2.66 for these three effects was assumed to be a good approximation to use in this model for crop reduction. Also, because of the low variability of O_3 concentrations measured at agricultural sites, estimated effects would not change much if an exponent far from 2.66 was used.

The approach taken by Larsen et al. (1988) differs from that of Lee et al. (1987a,b; 1988a,b,c). Larsen's estimated crop reduction was calculated from a lognormal model that expresses O_3 exposure as a function of the effective mean times the exposure duration, i.e., the total impact (Larsen and Heck, 1985). By definition, the estimated crop loss is totally determined by the effective mean once the exposed duration is fixed. Since there is no biological variation in the data, (as in Lee et al., 1987a,b; 1988a,b) correlations between the estimated crop loss and the exposure indices are, in fact, measures of association between the (transformed) effective mean and the other exposure indices. Thus, it is difficult to tell whether mean indices are better correlated with plant response than other exposure indicators based on Larsen et al (1988).

3. Averaging Times

a. NCLAN/CERL Reanalysis

The discrepancy between the seasonal mean exposure indicator used in the NCLAN studies and the repeated peak exposures identified in the CD as being most important for plant response make it difficult to evaluate the vegetation effects data base for O_3. Accordingly, OAQPS requested that the O_3 team at Corvallis Environmental Research Laboratory (CERL) conduct a more detailed analysis of the crop yield response data from the National Crop Loss Assessment Network (NCLAN) and from the Corvallis laboratory.

Lee et al., (1987a,b) reanalyzed several NCLAN data sets (soybean, wheat, cotton, and alfalfa) in an effort to determine which exposure index produces the most accurate exposure response relationship. Although the most widely used exposure index to assess O_3 effects on crops is the 7-hr seasonal mean, current information about agricultural crops suggests that short-term, high concentration exposures appear to be more detrimental to crop production than long-term, low concentration exposures. The seasonal mean ignores many factors known to affect vegetation growth including phenological response, peak concentrations, length of episodes and days between peaks (CD, 1986).

Lee et al. (1987a,b) examined several exposure statistics that emphasize peak concentrations more than lower concentrations as alternatives to the seasonal mean. These include: 1) summation of all concentrations above a cut-off level, 2) number of hourly concentrations above a cutoff level, 3) summation of all concentrations raised to a power greater than 1. In conjunction with 3), an exponential weighting scheme over time was used to account for differences in response at various stages of phenological development; this is described as phenologically weighted cumulative impact statistics (PWCI). Exposure indices that included all the data performed (24 hr) performed better than the indices that used only 7 hours of data. The 7-hr seasonal mean was never "best" and was near optimal in only 5 of

14 cases. The exposure indices that emphasized peaks performed better than those that gave equal weighting to all concentrations; indices that accumulated the exposures performed better than those that averaged the exposures.

Because of the extreme importance of this type of analysis in resolving the issue of averaging time, and form of the standard for the secondary standard, Corvallis has conducted increasingly more detailed assessments of alternative exposure statistics using various weighting schemes for relating exposure to plant response. In a more extensive retrospective analysis of NCLAN data, Lee et al., (1988a,b) fit 24 common and 589 general phenologically weight cumulative impact (GPWCI) functions to the response data from seven crop studies. The criteria established for determining "best" exposure indices were those that displayed the smallest residual sums of square error when the yield response data were regressed on the various O_3 exposure indices using the Box-Tidwell model.

The "best" exposure index was a GPWCI with sigmoid weighting. Cumulative indices (with concentration thresholds) performed as well as the GPWCIs while mean indices did not perform as well. The authors concluded that,

> "While no single index was deemed "best" in relating O_3 exposure to plant response, the top performing indices were those indices that (1) cumulate the hourly O_3 concentrations over time, (2) emphasize concentrations of 0.06 ppm and higher either by continuous sigmoid weights or by discrete 0-1 weights of the threshold indices and 3) phenologically weight the exposure such that greatest weight occurs during the plant growth stage. When assessing the impact of O_3 on plant growth, these findings illustrate the importance of repeated peaks, and the time of increased sensitivity in assessing the impact of O_3 on plant growth."

Although peak concentrations should be given greater weight, the authors suggested that lower concentrations should also be included in the calculation of an exposure index.

Tingey et al. (1988) is essentially a condensation of the paper by Lee et al. (1988a,b) and therefore the conclusions are basically the same. However, the paper does show the importance of exposure duration and the limitation of the seasonal mean to specifically incorporate duration. For example, the mean does not distinguish among exposures to the same concentration (mean) but of different durations (e.g., 10, 50, or 100 days).

The most recent analyses by Lee et al. (1988c), developed in response to a request by CASAC (December, 1987), evaluates selected ambient O_3 air quality indicators and estimates the exposure levels associated with agricultural losses. The results of this initial draft analysis are discussed in detail in Lee et al. (1988c). This section will highlight key points regarding the methodology and the results. Ideally, the selection of the most appropriate exposure indices would be derived from scientific principles and be validated by experimental data specifically designed to identify the most appropriate exposure indices. Such an approach, however, would delay resolving the selection of exposure indices for years until the necessary experiments were designed and conducted. Since the likelihood of the Agency funding another major crops research program in the near future is small, EPA has developed an alternative approach of conducting a retrospective analysis of existing plant-response data. While recognizing the limitations of the original NCLAN data, CASAC has requested further analysis of the exposure dynamics issue through retrospective analyses. The major limitation of such analyses is that the specific studies were not designed for developing exposure indices or testing exposure hypotheses; consequently, the usability of the data is somewhat limited.

Plant yield and hourly O_3 monitoring data were obtained for twelve NCLAN field experiments (soybean, winter wheat, corn, sorghum and cotton). The twelve NCLAN studies represented five species with differing cultivars for a total of 17 individual cases. Crops were grown according to standard agricultural

practices and exposed to a range of concentration in open-top chambers according to NCLAN protocols. These crop yield data were regressed against various exposure indices using the Weibull model (defined in Appendix A of Lee et al., 1988b).

Selection criteria for determining the "best" exposure index was based on the minimum residual sum of squares (RSS) for each of the 17 cases. Ranking and selection of exposure indices was performed separately for each case. For each individual case, the relative performance of an index was measured as the ratio of its RSS to the minimum RSS. For overall comparison among species and cultivars, the exposure indices were evaluated according to three criteria: (1) the average score calculated as the mean relative RSS's; and (3) percent variation (i.e., range/mean score x 100).

Based on the criterion of minimum RSS, no single exposure index performed "best" for all seventeen cases. The best overall fits are obtained using indices that:

1. cumulate hourly concentrations overtime,
2. place greater weight on concentrations of 0.06 ppm or higher either by continuous sigmoid weight or by discrete 0 - 1 weights, and
3. weight hourly concentrations according to the phenological stage of plant development.

The results indicate that while the generalized phenological cumulative impact (GPWCI) indices best related plant response to O_3 exposure, there were other indices that were near optimal. These indices included a sigmoid-weighted integrated index (SIGMOID) centered at 0.062 ppm which is much too complex for use as an ambient air quality standard. The cumulative censored indices that integrated concentrations of 0.06 (or 0.07) ppm or higher (SUM06 and SUM07) also performed well, suggesting that ambient O_3 levels below 0.08 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. (1988a,b) and Lee et al. (1987a,b; 1988b) who used 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) used to characterize experimental exposures are functions of exposure duration and concentration. Correspondingly the exposure levels associated with various yield losses calculated from experimental data are functionally related to exposure "seasons." An implicit assumption here is that exposures of varying duration, with equal values of SUM06 (or SUM07) cause the same biological response. According to Lee et al. (1988c) experiments replicated in time and/or space, differing in exposure duration, should produce identical predicted relative yield losses using SUM06 or SUM07. Therefore, the discrepancy between exposure "seasons" and between experimental and ambient averaging times are accounted for in the integrated indices. Thus, these results indicate the integrated indices capture the key components of exposure, are adequate descriptors of plant response, and are simple and easy to implement from a regulatory perspective.

The magnitude of the O_3-induced yield losses varied among sites, years and species/cultivars. The NCLAN studies used in this analysis represent both O_3 sensitive and tolerant species/cultivars, and multiple years, and experimental sites. Crop yield losses for 16 NCLAN studies are contained in Table 3.3. The predicted relative yield losses for the four exposure indices across the 16 cases are shown for the mean, the 25th, 50th, 75th, and 85th percentiles.

As can be seen in Table X-9, median crop losses of 18.9% are expected in agricultural sites experiencing O_3 exposures with a HDM2 value of 0.12 ppm. Lowering the concentration to 0.10 ppm would result in an estimated median crop loss of 12.8%, whereas going to 0.08 ppm would result in an estimated median crop loss of 7.9%. Table X-10 presents the exposure levels associated with mean and percentiles of predicted relative yield loss of 5 to 30%

TABLE X-9

Percentiles and mean predicted relative yield losses (PRYLs) associated with various levels of the four exposure indices, HDM2, M7, SUM06, and SUM07, for the 16 NCLAN cases. The distribution of PRYLs for the 16 NCLAN cases are characterized by the 25th, 50th, 75th, and 85th percentiles and the mean.

HDM2 (ppm)	Percentiles of PRYLs 25th	50th	MEAN	75th	85th
0.04	0.1%	1.7%	6.3%	9.8%	15.0%
0.06	0.4%	4.2%	9.9%	15.9%	21.7%
0.08	1.1%	7.9%	13.7%	22.1%	28.0%
0.10	2.2%	12.8%	17.8%	28.8%	33.8%
0.12	3.9%	18.9%	22.3%	34.8%	39.7%
0.16	12.6%	32.6%	33.0%	46.0%	50.5%
0.20	38.6%	48.7%	48.6%	58.3%	60.4%

M7 (ppm)	Percentiles of PRYLs 25th	50th	MEAN	75th	85th
0.02	0.1%	0.2%	3.4%	0.6%	1.4%
0.03	0.3%	2.3%	5.6%	5.9%	7.6%
0.04	0.9%	4.4%	8.3%	10.4%	12.3%
0.05	1.8%	7.5%	11.5%	16.0%	17.6%
0.06	3.1%	11.3%	15.3%	22.3%	23.5%
0.07	6.7%	15.9%	19.8%	27.2%	30.9%
0.08	11.5%	22.0%	25.1%	32.4%	38.7%

SUM06 (ppm)	Percentiles of PRYLs 25th	50th	MEAN	75th	85th
10	0.2%	2.2%	5.1%	4.9%	13.4%
20	0.9%	9.0%	12.0%	12.6%	30.6%
30	5.4%	16.5%	20.2%	22.3%	46.5%
40	14.6%	27.9%	31.4%	42.8%	61.1%
50	28.2%	37.6%	43.8%	55.5%	85.3%

SUM07 (ppm)	Percentiles of PRYLs 25th	50th	MEAN	75th	85th
10	0.3%	3.7%	6.7%	7.2%	17.2%
20	2.0%	11.7%	14.3%	16.4%	33.7%
30	8.6%	19.4%	22.7%	26.2%	47.4%
40	21.5%	30.9%	32.8%	40.9%	58.5%
50	30.2%	40.2%	43.8%	53.7%	83.4%

Source: Lee et al. (1988c)

TABLE X-10.

Exposure levels associated with predicted relative yield losses (PRYLs) of 5 to 30% for the four exposure indices, HDM2, M7, SUM06, and SUM07, for the 16 NCLAN studies. Separate calculations are performed using the 50th, 75th, and 85th percentiles, and the mean PRYLs for the 16 NCLAN studies.

A) Exposure levels with Mean PRYLs of 5 to 30%.

	Mean Predicted Relative Yield Loss					
	5%	10%	15%	20%	25%	30%
HDM2 (ppm)	0.032	0.061	0.087	0.110	0.131	0.149
M7 (ppm)	0.027	0.045	0.059	0.070	0.080	0.089
SUM06 (ppm)	9.7	17.1	23.7	29.8	34.3	38.8
SUM07 (ppm)	7.5	14.4	20.9	26.8	32.3	37.2

B) Exposure levels with 50th Percentile PRYLs of 5 to 30%.

	50th Percentile Predicted Relative Yield Loss					
	5%	10%	15%	20%	25%	30%
HDM2 (ppm)	0.065	0.089	0.107	0.123	0.137	0.152
M7 (ppm)	0.042	0.057	0.068	0.077	0.085	0.093
SUM06 (ppm)	14.1	21.3	28.0	33.1	37.5	42.2
SUM07 (ppm)	11.6	17.9	24.3	30.5	34.9	39.2

C) Exposure levels with 75th Percentile PRYLs of 5 to 30%.

	75th Percentile Predicted Relative Yield Loss					
	5%	10%	15%	20%	25%	30%
HDM2 (ppm)	0.023	0.041	0.057	0.073	0.089	0.104
M7 (ppm)	0.028	0.039	0.048	0.056	0.066	0.075
SUM06 (ppm)	10.2	16.6	22.4	27.6	31.3	33.7
SUM07 (ppm)	6.9	13.0	18.5	23.7	28.8	32.6

D) Exposure levels with 85th Percentile PRYLs of 5 to 30%.

	85th Percentile Predicted Relative Yield Loss					
	5%	10%	15%	20%	25%	30%
HDM2 (ppm)	<0.020	0.026	0.040	0.055	0.070	0.087
M7 (ppm)	0.026	0.035	0.045	0.054	0.062	0.069
SUM06 (ppm)	3.7	7.5	10.9	13.8	16.7	19.6
SUM07 (ppm)	2.9	5.8	8.7	11.7	14.7	17.8

Source: Lee et al. (1988c)

for the four indicators across all sites. If EPA decided to protect against median crop losses in the range of 15%, the HDM2 value would need to be 0.10 ppm. Alternative standards for limiting crop reductions to 15% could be achieved using the cumulative censored indices. Restricting the exposures with SUM06 to less than 28 ppm (or sum 07 less than 24 ppm) would protect against a 15% reduction.

It is important to note that using the median predicted relative yield losses (PRYL's) to estimate the associated O_3 exposure levels will protect against typical crop losses but not necessarily against the maximum losses that can occur. For example, the median crop loss of 18.9% expected in agricultural sites experiencing a HMD2 value of 0.12 ppm (Table X-9) could go as high as 34.8% if a more protective strategy such as the 75th percentile of PRYL's were used rather than the median PRYLs. A broad range of percentiles of PRYL's are presented for each indicator so that the reader has an appreciation of the range of expected yield losses.

Finally, Lee et. al. (1988c) presents information on the relationships among the various air quality indicators, in response to CASAC's interests 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 censored indices (SUM06 and SUM07) and the peak and mean indices (HDM2 and M7). The integrated indices, SUM06 and SUM07, are strongly related to M7, and less related to HDM2. The relationship between SUM07 and HDM2 just falls below the level defined by the authors (Lee et al., 1988c) as indicative of a strong association. Thus SUM06 and SUM07 correlate well with a long-term index (M7) and a short-term index (HDM2) and relate well to biological response. These results suggest SUM06 and SUM07 have potential for a standard that protects against adverse effects from repeated peak and long-term exposures.

In regard to the exposure period of interest, an air quality analysis of 83 non-urban site-years of ambient O_3 data indicate an averaging time of five months from May to September (153 days) should be used to capture 90% of the hourly concentrations of 0.06 ppm or higher. This period includes the EPA-defined O_3 season for most sites, represents the period when the second highest daily maximum 1-hr concentration generally occurs, and coincides with the agricultural season for the majority of crops grown in the United States.

b. New Studies

Although only a limited number of studies have been conducted with the specific objective of developing or evaluating various exposure indices, several studies have attempted to use existing exposure response data for evaluating a range of exposure indices. The results of these retrospective analyses have provided useful concepts and the conclusions are in general agreement. However, because the studies were not specifically designed to evaluate various indices, the differences in and among exposure treatments may be relatively small. As a result, the power of these studies is less than desirable.

In a small retrospective analysis, Lefohn et al. (1988a) has come to similar conclusions as Lee et al. (1987a,b) using different NCLAN data sets with the Weibull and linear models. As in Lee et al. (1987a,b), he is comparing the use of several indices of exposure in describing the relationship between O_3 and reduction in agricultural crop yield. Prior to these studies, no attempt had been made to determine which exposure response models best fit the data sets examined. Lefohn, et al. (1988a) used hourly mean concentration data based on 2-3 measurements per hour to develop indices of exposure from soybean and winter wheat studies conducted in open-top chambers at the Boyce Thompson field site. The efficiency of using seasonal mean and cumulative indices (i.e., number of occurrences equal to or above specific hourly mean concentrations, sums of all hourly mean

concentrations equal to or above a selected level, and the weighted sum of all hourly mean concentrations) to describe the relationship between exposure to O_3 and reductions in the yield of agricultural crops has been evaluated. In most cases, the Weibull model, a functional form used extensively by the NCLAN program, tends to fit the data based on cumulative indices better than on seasonal mean indices. This conclusion is similar to what Lee et al. (1987a,b) found with different models and data sets. In addition, two other recent papers (Hogsett et al., 1988 and Musselman et al., 1988) support the same conclusion that 1) mean indices are not among the best exposure indicators and 2) the preferred (yield best statistical fit to the data) exposure indices cumulate the exposure impact over the growing season and preferentially weight the peak concentrations. While the exposure indicators developed in these analyses can be potentially complex, Hogsett et al. (1988) has recently found that there are other simple indices such as summation of all concentrations above 0.06 (SUM06) and 0.08 ppm (SUM08) which are almost as good as the complex PWCI statistics. These potential exposure statistics were evaluated in greater depth in a new analysis (Lee et al., 1988b) previously discussed on p. X-48.

The paper by Lefohn et al. (1988a) has generated discussion about the interpretation of the data (Runeckles, 1988; Parry and Day, 1988). Both commenters felt that the data presented was insufficient to support the conclusion that peak-weight exposure indices should be used in developing exposure response functions, although Runeckles did point out that peak-weighted indices performed at least as well as mean indices. The commenters also criticized the compilation of two years of wheat data with markedly different exposure durations into a single model. In response, Lefohn et al. (1988b) stated that the wheat data support the need to include a cumulative component in an exposure index and that "the cumulative index is more relevant to use in the standard setting process than seasonal means, which ignore the length of the exposure period."

These conclusions on the importance of cumulative peak concentrations in causing plant response are consistent with the data presented in the CD as well as other recent studies. Wang et al. (1986) characterized O_3 exposure of three tree species as the number of daily occurrences above 0.08 ppm and 0.12 ppm over a four month growing season. The authors concluded that O_3 significantly impaired the growth of hybrid poplar in the absence of visible injury. In a three year study with quaking aspen, Wang et al. (1986) found that plant growth was reduced 12 to 24% although the current national air quality standard was exceeded in only one of those years. Adomait et al. (1987) characterized O_3 exposure of white bean as the cumulative O_3 concentration above a threshold of 0.08 ppm for a one month period. In addition, Reich and Amundson (1986) stated that the O_3 induced decrease in growth of several tree species was directly related to reduced photosynthesis, which was impaired by the cumulative O_3 dose.

Although the CD and the weight of the new evidence since the CD review (with the exception of papers reporting results from NCLAN) seem to suggest the importance of peaks, another issue of concern is how to treat the peaks in the development of exposure response indicators. While Lefohn et al. (1988a) recommended that a weighting scheme which gives greater weight to higher concentrations be used in developing exposure indicators, others support the removal of some dose from each hourly O_3 value so that low levels which are less likely to effect plant growth are eliminated (McCool et al., 1986). Basically, three weighting approaches have been used: 1) a concentration threshold in which the concentrations, number of occurrences or hours above the threshold cumulated; 2) an exponential weighting in which all concentrations are raised to a specific exponential power and 3) a sigmoid weighting in which all concentrations are weighted with a multiplicative weighting factor (which depends on concentration). The functional weighting approach using either exponential or sigmoid weighting seems preferable to the

threshold approach because it considers the contribution of all concentrations in eliciting a response.

While several studies (Lee et al., 1987a,b and 1988b; Hogsett et al., 1988; Lefohn et al., 1988a; Musselman et al., 1988) have focused on developing new exposure statistics, there have been several attempts to evaluate the statistic used most often in the current O_3 data base - the 7-hr seasonal mean. According to Smith et al. (1987) the open-top chamber method on which predictive models such as NCLAN are based do not reflect the episodic nature of O_3 pollution or some of the more important environmental conditions that influence plant growth. The authors concluded that the failure of the NCLAN predictive model to account for such conditions might explain why they found no effect of ambient O_3 on yield of field grown soybeans in New Jersey while the NCLAN model predicted losses of around 19%.

Heagle et al. (1986, 1987) examined the seasonal mean statistic in studies on soybean and tobacco. Both studies added O_3 in constant or in variable amounts which were proportional to ambient O_3 concentrations. Interestingly enough, while there were greater fluctuations and higher peak concentrations with the proportional method than with the constant addition method, the two methods of addition gave similar seasonal mean concentrations. Regression of yield response on O_3 concentrations showed no significant differences between the two types of additions. This finding may be partly explained by the fact that the 7-hr seasonal mean, even with the proportional addition, may fail to accurately reflect the elevated exposures. In fact, one of the reasons the 7-hr seasonal mean was originally selected is because it was less sensitive to variations in O_3 patterns (Cure, 1986). Therefore, the conclusion in these studies that differences in peak O_3 concentrations do not significantly affect yield responses in soybean and tobacco may be unwarranted.

Rawlings et al. (1988) conducted additional analyses of the soybean (Heagle et al. 1986) and tobacco (Heagle et al., 1987)

data sets to evaluate various exposure indices and the influence of exposure duration. The results from the cotton and soybean data suggested that the peaks should be given greater weight. In contrast, the 7-hr studies with tobacco suggested that the arithmetic mean was sufficient and that the peaks should not be weighted differently. According to Rawlings, these results must be viewed with caution because the differences between the constant and proportional O_3 additions were relatively small. Consequently the power of this experiment as well as Heagle et al. (1986, 1987) for determining the "best" exposure index is limited.

Finally, Adams and McCarl (1985) compared various alternative second high daily maxima (0.08 and 0.16 ppm) standards against the current standard. The study was based on 1980 data on the effects of O_3 on wheat, corn, and soybeans in the Corn Belt. Their analyses showed that 0.08 ppm standard would provide additional benefits of $700 million dollars annually to the Corn Belt, while a relaxation of the current standard to 0.16 ppm would cost the,U.S. farm economy an additional $2.225 billion annually.

The information reviewed in this section suggests a recognition on the part of plant scientists that exposure dynamics is an important issue. The weight of the new evidence supports the conclusion that a cumulative O_3 exposure index is needed that reflects the total exposure that the plant experiences. The conclusions found in the recent literature discussed above regarding the importance of cumulative peak concentrations in causing vegetation responses are consistent with the data presented in the recent CD. Staff conclusions and policy recommendations based on this new evidence are discussed in Chapter XI.

4. Forest Risk Assessment

Assessing the problem of forest damage due to O_3 has proven to be a difficult task in the review of the secondary standard

for O_3. While the potential contribution of O_3 and other atmospheric pollutants has been receiving increasing scientific and regulatory interest, the numerous and complex interactions between O_3, environmental factors, and health and productivity of forests make it difficult to distinguish O_3 effects from natural forest processes (Peterson and Violette, 1986). Although the nature and magnitude of the effects and the precise contribution of various atmospheric pollutants is not well understood, there is evidence that some sensitive forest types are negatively affected by O_3. The effects data, summarized in Chapter X of this staff paper, is somewhat scarce at this point in time and tends to focus more on controlled studies of seedlings or saplings. The results of these controlled laboratory or greenhouse studies are not easily extrapolated to field conditions, thus making current extrapolation (e.g., seedlings to mature trees, stands to populations) difficult.

a. Overview of Forest Risk Assessment

While significant new research is underway to improve our knowledge of the exposure-response relationships of various tree species and O_3 and to develop our extrapolation modeling capabilities, at this point in the standard review we must assess a data base that has considerable uncertainty associated with it. Standard statistical procedures cannot quantify this type of uncertainty because of the absence of valid statistical samples across the relevant populations. This type of uncertainty, however, can be represented using appropriate decision analytic procedures to elicit judgmental probabilities (Walsten and Whitfield, 1986; Peterson and Violette, 1986). EPA decided to use this approach as a tool to better quantify the range of scientific judgment and uncertainties regarding O_3-caused risks to forest resources.

This section will briefly describe the methods and preliminary results of a study which assesses the uncertainty around estimates of forest-tree growth decrement and foliar injury due to O_3 exposures. This information is meant to

supplement scientific information and data by presenting an interpretation of current scientific knowledge regarding O_3 caused forest effects. It is extremely important to recognize that the results presented here are not a substitute for continued scientific research; clearly such research is needed and the results of this work confirm that fact. However, because the magnitude of forest damage caused by O_3 is likely to remain uncertain in the near term, policy analysts and regulators may find information regarding the uncertainty among forest experts useful in formulating policy options and better focusing research efforts.

b. Protocol Summary

For judgmental probabilities regarding forest damage to be useful to policy makers they must satisfy three criteria (Peterson and Sueker, 1987):

1) Judgments should be made over the appropriate forest response parameters for policy assessment - in this case the endpoints of concern are growth decrement and foliar injury,

2) Judgments should be obtained from experts who are likely to possess interpretations of O_3 associated forest effects that span the range of respected opinion, and

3) Judgments should be relatively stable over the course of the study.

To satisfy these criteria, a protocol for probability encoding was developed by the investigators and reviewed by EPA. While detailed information on the protocol is contained in the report (Petersen et al., 1987) it is useful to summarize several of the basic assumptions made in the protocol:

1) Total above ground growth decrement and foliar injury were selected as response parameters. Several other response parameters were considered, but rejected as being beyond the scope of this study.

2) While numerous O_3-exposure measures were reviewed, two were selected: the maximum 3-month mean for 24- and 7-hour (0900-1600) daily averages. The selection of exposure statistics for

this analysis was extremely difficult. While new data indicate the importance of repeated peaks in eliciting plant response (at least for crops), several experts expressed concern about their ability to relate their judgments to a repeated peak statistic not commonly used in the effects literature. Rather, they seemed more comfortable with a mean statistic, which was defined for purposes of the study as including the episodic as well as the low-level exposures seen by the trees. These very real concerns strongly influenced the selection of the exposure statistics for this analysis.

3) Trees in each O_3 treatment "population" were assumed to be exposed to O_3 exposures represented by the 0.035, 0.055 and 0.085 O_3 maximum 3-month means for the 24- and 7-hour daily averages. The concentrations were chosen to express the range of O_3 exposures over which growth decrement or foliar injury could be detected in U.S. forests. Trees in the "control" population were assumed to be exposed to concentrations ranging between 0.020 and 0.030 ppm O_3 to represent naturally occurring background conditions.

4) The following assumptions were made about the conditions of the forests in the hypothetical studies:

a. The forests were mixed/monoculture type and of uneven/even aged composition.

b. Forest trees were exposed to one of the O_3 exposures from germination to the time growth measurements were taken.

c. Ozone sensitive genotypes were distributed in forests according to the experts' beliefs.

d. Except in the use of plantation trees, trees were entirely wild type genetically.

e. Site conditions, including slope, aspect, elevation, soil type, air temperature and humidity, and pest and pathogen populations were distributed across the control and experimental populations as the experts believed they were in the real world.

f. Growth effects were measured on all trees (no sampling problems) at all locations. Measurements were assumed to reveal effects on all age classes within a true population.

g. Effects concerned only the species in question and not second-order effects involving other species.

c. Scientific Experts

In order for the judgments to be useful to decision makers, they must be obtained from knowledgeable experts in the area of O_3 effects on forests. The experts were selected to provide judgments regarding O_3 associated forestry effects spanning the range of respected opinion. Establishing the range of opinion and selecting the experts to be encoded may appear to be very subjective; however, the field was narrowed considerably by focusing on those individuals whose careers had been devoted to conducting air pollution research (with a focus on O_3) in the area of forest effects. Recent reviews of O_3 associated forest effects and references to the published literature also facilitated the selection of experts. Several offices within EPA (OAQPS, OPPE, ORD) collaborated to develop a set of potential candidates from which the five experts were selected. Names of participating experts are provided in Table X-11 to facilitate the interpretation of the range of responses by other forest scientist and policy makers. All experts selected by EPA agreed to participate with the understanding that their judgments would remain anonymous. Particular responses or judgments for experts A through E are identified only by randomly assigned code letters.

d. Credible Judgments

To ensure that an expert's judgments were relatively stable and accurately represented his uncertainty, a number of steps were followed. First, a draft of the protocol was developed and reviewed by the staff at EPA and the consultants prior to the encoding session. Following the revision of the protocol, two

Table X-11

Forest Response Experts

Expert	Affiliation
Sam Linzon	Ontario Ministry of Environment
Samuel McLaughlin	Oak Ridge National Laboratory
Paul Miller	USDA Forest Service
John Skelly	Pennsylvania State University
David Tingey	U.S. EPA, Corvallis Environmental Research Laboratory

Souce: Peterson et al., 1987

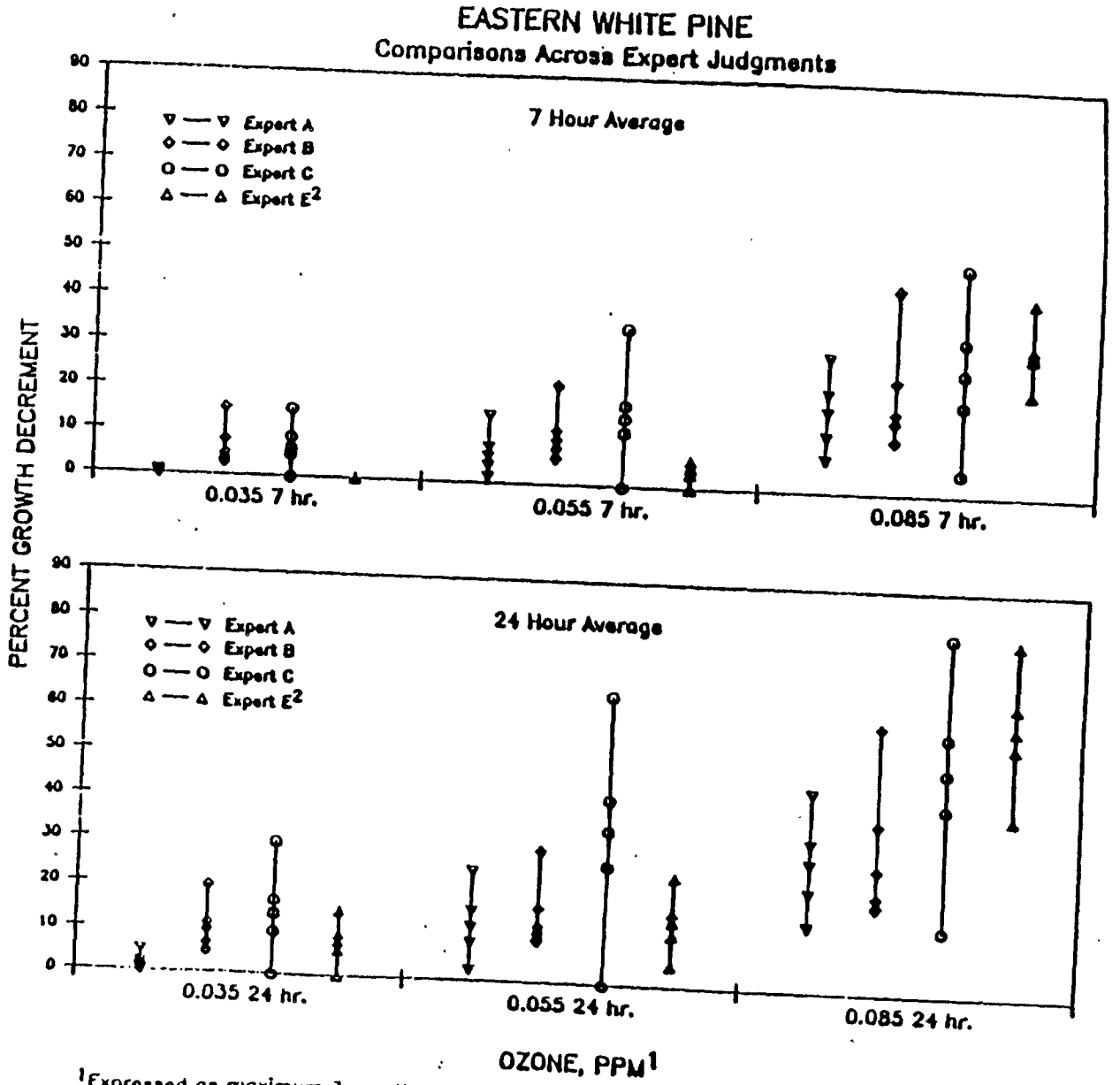
separate meetings to encode the judgmental exposure-response relationships were held with each expert. The first meetings were conducted during November-December 1986; the second during February-March 1987. During the first meeting, which lasted 6 to 8 hours, experts reviewed the protocol, discussed relevant literature and research into factors determining exposure-response functions and estimated probability distributions for selected tree species. At the second meeting, the results of the first session, which had been forwarded to the experts by mail, were carefully reviewed and an additional probability encoding session was completed. Draft final results were distributed to the experts and discussed in a telephone conference with each expert.

e. Results

The findings suggest a wide range of opinion regarding tree sensitivity to O_3 among credible forest scientists. Three different sets of results were developed as a result of the analysis. The first set, discussed in sections 3.1 through 3.6 of the report, present 152 cumulative probability distributions for growth decrement estimated by Experts A through E. These judgments dimension the uncertainty over growth decrement in 19 different tree species due to six different O_3 exposures. Cumulative probability distributions across tree species and experts reflect different underlying probability models. Some cumulative probability distributions are virtually linear, others reflect the normal cumulative probability model, and others a "hockey stick" distribution. This variability prevented fitting a single probability model to the cumulative probability distributions developed in this study. For purposes of description, individual judgments within any given distribution were linked using cubic spline interpolation.

Figure X-3 summarizes these judgements across experts for eastern white pine, deemed to be the most sensitive species by Experts A, B, C and E at all O_3 exposures. The vertical axes in the figure represent percent growth decrement relevant to

Figure X-3.



[1]Expressed as maximum 3 month mean.
[2]Expert E estimated responses at 0.035, 0.045 and 0.075 ppm O_3 7 and 24 hour averages.

Source: Peterson et al., 1987

background conditions. The horizontal axes represent the six different O_3 exposure levels. The vertical bars at each O_3 concentration represent each expert's maximum credible confidence interval, assumed to be 98%, for growth decrement. The symbols within each bar indicate respectively the .01, .25, .50, .75, and .99 probability fractiles. Thus, in Figure X-3, the maximum credible interval for Expert C's judgment of growth decline at exposures characterized by the 0.035 ppm O_3 7-hour average statistic ranges between a lower bound (F=0.01) growth decrement of 0% to an upper bound (F=0.99) growth decrement of 15%. The three intermediate symbols represent growth decrement at cumulative probability fractiles .25, .50, and .75. Figure X-3 illustrates a number of important points:

- First, there is a wide range of uncertainty expressed in the experts' judgments. A and E are consistently the most confident, C the least.
- Second, uncertainty tends to increase as O_3 exposures increase.
- Third, there is no agreement across experts that growth decrement due to O_3 will occur (within the maximum credible interval of 98%) until O_3 exposures associated with the 0.085 ppm 7 hour average statistic are reached.
- Fourth, 2 of 4 experts believe that there will be little, if any, growth decrement at the 0.035 7-hr average.

The second set of results presents exposure-response functions for O_3 caused growth decrements over the range of O_3 exposures considered in the study. The experts were unanimous in their belief that exposures of 0.035 ppm O_3 7-hr average would not cause measurable growth decrement in the tree species considered. These exposure-response functions are summarized in Figures 3.15 through 3.30 of the report. Figures 3.31 and 3.32 (Peterson et al., 1987) presents upper and lower bound judgments across experts, providing the largest possible credible interval

and the broadest interpretation of uncertainty. As can be seen for eastern white pine, this interval is often quite large. For example, extreme risk estimates range from 0 to 65% growth decrement at 0.055 ppm O_3, 24-hr average.

The third set of results focus on the judgements on foliar injury to forest trees. Although the experts indicated considerable uncertainty with respect to growth and yield effects, their lack of confidence in risk estimates for foliar injury were even more pronounced.

Peterson et al. (1987) was designed to explicitly present the scientific uncertainty in estimates of O_3 induced forest decline. It is intended to augment scientific research by reframing effects data so that non-scientists can better evaluate the probability of different levels of forest damage. The study does not, however, replace effects research as it did not generate any new hypotheses or data. What it does is confirm what many felt was the case - that there is a wide range of uncertainty among credible forest scientists regarding the role of O_3 and forestry effects. Further scientific research is needed before confident estimates of growth decrement can be attributed to O_3. Much of this research will be conducted during the next several to five years under NAPAP's Forest Response Program and EPA's research program, the Effects of Tropospheric Ozone on Forest Types.

XI. Staff Conclusions and Recommendations Regarding the Secondary Standard

Drawing upon the evaluation of scientific information contained in the CD and CDS, this section provides preliminary staff conclusions that should be considered by the Administrator in selecting the pollutant indicator, form, averaging time and level of the O_3 secondary standard. The primary focus of this discussion will be vegetation and ecosystem effects, as this data base provides the best support for the secondary standard for O_3. Materials damage and effects on personal comfort and well-being will be covered in the final section. Risk and benefit analyses covering vegetation effects are currently underway as part of a continuing program to assess secondary standard effects.

A. Pollutant Indicator

On February 8, 1979, the chemical designation of the O_3 primary and secondary standards was changed from photochemical oxidants to O_3 (44 FR 8202). EPA changed the designation of the standard to O_3 since the Federal Reference Method (FRM) for determining compliance specifically measured O_3 as a surrogate for total oxidants, and because a substantial vegetation effects research base has established O_3 as being chiefly responsible for the adverse effects of photochemical air pollutants, largely because of its relative abundance compared to other photochemical oxidants.

The toxicities of the peroxyacetyl nitrates, of hydrogen peroxide (H_2O_2), and of formic acid are less well documented than the toxicity of O_3. These oxidants have been the focus of considerably less research because the levels at which they occur in the ambient air, even in urban areas, appear to warrant much less concern (CD, p. 7-1).

Peroxyacetyl nitrate, perhaps the best studied of the non-O_3 oxidants, is produced by photochemical reactions similar to those that produce O_3. Controlled exposures of plants to PAN have indicated that PAN can cause foliar injury and yield losses in

sensitive cultivars of leafy vegetable crops (p. X-22). However, a comparison of PAN concentrations likely to cause either visible injury or reduced yield with measured ambient concentrations (CD, Chapter 5) indicates that it is unlikely that PAN related effects occur in plants in the U.S. except in some areas of California and possibly in a few other localized areas. Because phytotoxic concentrations of PAN and the other photochemical oxidants generally occur at significantly lower ambient concentrations and are less widely distributed than O_3, the focus of this standard review will be the effects of O_3.

The question of whether O_3 can serve as an abatement surrogate for controlling other photochemical oxidants is addressed by Altshuller (1983) who concluded that "the ambient air measurements indicate that O_3 may serve directionally but cannot be expected to serve quantitatively as a surrogate for the other products." This conclusion appears to apply to the subset of photochemical products of concern - O_3, PAN, PPN, and H_2O_2 - identified in the CD, even though Altshuller (1983) examined the use of O_3 as an abatement surrogate for all photochemical products. Lack of a quantitative, monotonic relationship between O_3 and other photochemical oxidants is discussed in Chapter 5 of the CD in which average PAN/O_3 ratios for different sites and years vary from 9 to 3. In addition, no single measurement methodology can quantitatively and reliably measure O_3 and other photochemical oxidants, either individually or in ambient air mixtures.

In spite of the above limitations, it is generally recognized that control of ambient O_3 levels currently provides the best means of controlling photochemical oxidants of potential welfare concern (O_3, PAN, PPN, and H_2O_2). This recognition along with a controlled-exposure, welfare data base which implicates only O_3 among the photochemical oxidants at levels commonly reported in ambient air, supports the recommendation that O_3 be retained as the pollutant indicator for controlling ambient concentrations of photochemical oxidants. Unless significant

additional evidence which demonstrates welfare effects from exposure to ambient levels of non-O_3 oxidants becomes available, it is the staff's conclusion that O_3 remains a reasonable surrogate for protection of public health from exposure to photochemical oxidants.

B. Form of the Standard and Averaging Time(s)

The current secondary O_3 NAAQS is expressed as an hourly average which is the concentration not to be exceeded on more than 1 day per year on average. During the last standard review the decision was made to change from the deterministic to the statistical form of the standard. The deterministic form, which permitted only a single hourly exceedance of the standard level in any given year, did not adequately deal with the variations in O_3 concentrations which are largely due to the random nature of meteorological factors affecting formation and dispersion of O_3 in the atmosphere. In addition, EPA further modified the standard so that one expected exceedance would be given a daily interpretation; that is, a day with two hourly values over the standard level counts as one exceedance of the standard level rather than two. It is recommended that the statistical form (i.e., a number of expected exceedances allowed per year) be retained for the secondary standard.

In selecting the most appropriate averaging time to protect vegetation and ecosystems from exposure to O_3, questions immediately arise concerning which components of an exposure are most important in causing vegetation responses. The studies conducted thus far offer some useful information in assessing the answer to this question, but to date research has not yet clearly defined which components of exposure are the most critical in eliciting plant responses (CD, p. 6-7). In light of these uncertainties, the selection of an averaging time which correlates well with the effects of concern is a difficult task. Not only is there little consensus in the scientific community as to the most appropriate summary statistic, but researchers are

currently using many different exposure statistics in their studies, thus making comparisons between studies extremely difficult. This section assesses the information available on which to base a judgment regarding the averaging time for the secondary standard.

Much of the research over the last three years has been driven by these concerns related to the issue of averaging time(s). In particular, the discrepancy between the seasonal mean exposure indicator used in the NCLAN studies and the repeated peak exposures identified as being most important for plant response have motivated several retrospective analyses which have attempted to investigate alternative exposure indices using existing experimental data. Based on the weight of the new evidence from analyses of plant response data from NCLAN and other research programs, EPA concludes that exposure indices that "best" related to plant response were those indices that cumulated hourly concentrations over time and gave greater weights to higher concentrations (Lee et al., 1987a,b; 1988b; Hogsett et al., 1988; Lefohn et al., 1988). These results support the conclusions of the CD and the CDS on the importance of peak concentrations and the cumulative impact of O_3 in relating plant response to exposure.

EPA has responded directly to CASAC's challenge (CASAC, 1986) to identify a single standard formulation that would offer protection from both repeated peaks and long-term exposures of concern in a series of analyses designed to identify the most appropriate exposure indicator. The most recent analysis (Lee, et al., 1988c) proceeded along two lines: (1) regression analysis of plant response data against various exposure indices to determine indices that "best" depict biological response, and (2) ambient air quality analysis of ambient O_3 data to identify indices that correlate well with various exposure patterns. While the results of these analyses have been discussed in some detail previously (SP, p. X-47), the key conclusions also provide valuable insight regarding the issue of averaging times.

Among the indicators Lee et al. (1988c) analyzed was the second highest daily maximum 1-hr concentration (HDM2) because it is a close approximation of the current air quality standard and the 7-hr seasonal mean (M7), because it is the statistic most often used in the current data base for crop loss. According to Lee et al. (1988c), neither the single peak indices or the long-term averages are biologically relevant. The HDM2 and M7 indices do not adequately describe the temporal variations in exposure and do not relate well to plant response. Thus, a major shortcoming of noncumulative indices is the exclusion of exposure duration.

The importance of this factor in determining plant response is readily apparent when examining yield loss results for the wheat cultivar VONA, which is extremely sensitive to O_3. In the VONA wheat results, there is greater variation in the predicted relative yield losses (PRYLS) when the noncumulative indices such as HDM2 and M7 (Figures 3.1, 3.2, Lee et al., 1988c) were used than when yield loss was estimated using the SUM06 and SUM07 (Figure 3.3, 3.4, Lee et al., 1988c) exposure indices. The lower year-to-year variability in PRYLS when the cumulative indices (SUM06 and 07) are used are largely explained by differences in exposure duration, a factor which is not captured by either the HDM2 or the M7. Larsen et al. (1988) also points to the limitations of the present 1-hr O_3 NAAQS in controlling crop reduction because it forces some sites to control more than is necessary in order to protect against a given level of crop loss. Similarly, Cure et al. (1986) have alluded to some limitations of the M7 such as the fact that the seasonal mean characterizations of O_3 exposure were much less sensitive than the 1-hr max to yearly concentration variations in O_3 patterns. This latter point supports the conclusion of Lee et al. (1988c) that the mean does not specifically include exposure duration and that a mean tends to minimize the contributions of peak concentrations to the response.

The results of Lee et al. (1988c) indicate that while the generalized phenological cumulative impact (GPWCI) indices best related plant response to O_3 exposure, there were other indices that were near optimal. These indices included sigmoid-weighted integrated index (SIGMOID centered at 0.062 ppm) which are much too complex to implement in the air quality management system of air pollution control. The cumulative censored indices that integrated concentrations of 0.06 (or 0.07) ppm or higher (SUM06 and SUM07) also performed well, suggesting that ambient O_3 levels below 0.08 ppm are important in triggering plant response and should be included in an exposure index. The cumulative censored indices are adequate descriptors of exposure which relate well to biological response, and are simple and easy to implement from a regulatory perspective. As stated earlier, these results support the conclusions reached by Lefohn et al. (1988) and Lee et al. (1987a,b; 1988b) who used NCLAN data and cumulative indices with sigmoid and allometric weights in demonstrating the importance of peak concentrations in determining plant response. In addition, the results indicate that fair to strong associations exist between the cumulative censored indices (SUM06 and 07) and the peak and mean indices. The integrated indices SUM06 AND SUM07, are strongly related to M7, and somewhat less related to HDM2. Thus, the relationship between SUM07 and HDM2 falls just below the level defined by the authors (Lee et al., 1988c) as indicative of a strong association.

These results suggest the cumulative indices SUM 06 and SUM 07 correlate well with a long-term index (M7) and a short-term index (HDM2) and relate well to biological response. Thus these indicators have potential for setting a standard that protects against adverse effects from repeated peak and long-term exposures. While these indicators appear to be promising, EPA staff concludes that the preliminary nature of the methodology and results make a decision to change the averaging time and form of the standard premature at this point in time. EPA further believes that the analysis should be reviewed further by the

scientific community and CASAC before the results are used in deciding whether a new secondary standard form and averaging time are needed.

The analysis, although preliminary, does provide CASAC with an additional piece of information regarding the crop loss expected at alternative 1-hr standard levels. As pointed out previously, these results are derived differently than Larsen et al. (1988) in that actual air quality and plant response data were used in deriving exposure-response functions. Nevertheless, the two analyses represent different approaches to estimating crop loss for alternative exposure indicators and provide EPA with information that was not available previously. While the form of the 1-hr standard has definite limitations, EPA staff concludes that establishing a 1-hr averaging time standard in an appropriate range (level of the standard is discussed in the next section) represents the best staff recommendation that could be made to the Administrator at this time to close out the review of the scientific data. With this portion of the review complete, and after considering CASAC's views on all issues, the Administrator will be in a position to make a regulatory decision on how and when to best act on the 1-hr standard. This is consistent with CASAC comments (December 1987 and 1988 meetings) urging EPA to take action on a 1-hr standard rather than waiting for a more complete assessment of alternative averaging times.

While EPA recommends closing on the scientific review of information relevant to the 1-hr standard, there is another course of action that could be considered - that is, continuing the standard review until the information on alternative exposure indicators has matured. This option may appeal to those who are in support of retaining a 1-hr standard at an appropriate level but feel that the Lee et al. (1988c) analysis should be peer-reviewed and published before relying on it in a standard decision or that more information is necessary before a specific level of the 1-hr standard can be justified. For example, the current draft analysis by Lee et al., (1988c) does not estimate

economic losses because of the cost and time involved in such an effort. The views of CASAC and the Administrator regarding the need for such an analysis would have to be taken into consideration.

Secondly, the option of continuing the review may appeal to those who do not find the current form of the standard to be a suitable air quality control target and would prefer to use one of the cumulative indicators suggested by Lee et al. (1988c) once the analysis has been finalized. Additional time for review and revision of this analysis would allow the scientific community an opportunity to review the alternative indicators and move toward a consensus regarding selection of the most appropriate exposure indicator. The liability of this alternative is that it postpones action on the secondary standard and thus, fails to utilize new and existing data to assess the most appropriate exposure indicator or the protection afforded by the current 1-hr standard.

While evidence exists to support the hypothesis that for agricultural crops, cumulative, repeated peak exposures appear to be more important than long-term, low level concentrations, there is still considerable uncertainty regarding the O_3 distribution patterns that affect trees. Some CASAC members objected to the attempt in the previous staff paper to draw similarities between the responses of trees and crops to O_3 and to the suggestion that the exposure pattern of interest for crops might be the same for trees - that is, repeated peaks over time. Perhaps there are no lessons from the years of research on crops that can be applied to forestry. On the other hand, it seems incumbent upon staff to examine the total data base, look at similarities and differences between crops and trees, and lay out plausible hypotheses that will spur debate on the key issues which science has not yet resolved. It is with this in mind that staff examines the data on exposure of trees to O_3 and notes a good many studies that report the exposure period in terms of hours above given thresholds.

McLaughlin et al. (1982) reported that patterns of annual growth in which pine along the Cumberland Plateau reflected a loss in capacity for short-term recovery and eventual loss of tree vigor. The primary cause of the observed decline was chronic exposure to elevated concentrations of O_3, possibly accompanied by low levels of SO_2 and other pollutants. Reports of oxidant injury on white pine in rural Virginia (Hayes and Skelly, 1977) and on the Cumberland Plateau (Mann et al., 1980), indicate that the injury appears to be associated with total oxidants concentrations of 0.08 ppm or higher. Hayes and Skelly (1977) reported concentrations which equalled or exceeded 0.08 ppm for 104 hours at one of the rural sites they monitored in Western Virginia. Skelly and Johnston (1978) also reported that O_3 concentrations during July of 1977 were above 0.08 ppm 30 percent of the time at the same location. In addition, relatively new evidence with slash pine Hogsett et al. (1985) suggests that regimes containing peak O_3 concentrations elicit a greater response than regimes containing mostly lower concentrations over similar time periods. Also, Wang et al. (1986) concluded that the 20 days when O_3 exceeded concentrations of 0.08 ppm and the 1 day when the concentration exceeded 0.12 ppm was responsible for significantly impairing the growth of hybrid poplar.

While assessment of the above evidence suggests the potential importance of repeated peak exposures in the response of trees to O_3, further research is needed to confirm or deny this hypothesis. As Peterson et al. (1987) point out, there is currently considerable uncertainty regarding the effects of O_3 on forest trees. In addition, there is considerable uncertainty regarding the O_3 distribution patterns that effect trees. Consequently, EPA staff recommend that until these scientific uncertainties can be resolved, no separate secondary standard should be established for forest trees and that the secondary standard should be based on protection of vegetation.

C. Level of Standard

A critical factor in determining the level of the standard is deciding which effects are to be considered adverse. There are two important dimensions to this question. The first is what should be measured. The second is how big does the change in the selected measure have to be to be considered adverse. Answers to the question of what should be measured fall, broadly speaking, into one of two groups, physical measures and economic measures. The most commonly proposed physical measure for commercial crops is a change in yield. In addition, the bulk of the data available since the last standard review uses growth and yield loss as the response measure. While a change in yield loss is the most commonly proposed physical effect measure for commercial crops, a broader range of effects could be considered adverse.

It is common to segregate the effects of air pollutants on vegetation, into "injury" and "damage" categories (Guderian et al., 1985). Injury encompasses all measurable plant reactons, such as reversible changes in metabolism, reduced photosynthesis, leaf necrosis, leaf drop, altered quality or reduced growth that does not influence all effects that reduce the intended use or value of the plant or plant system. Value or intended use must be viewed from the perspectives of both; (1) value to society or human welfare and (2) value to the organism itself (i.e., reproduction) or the surrounding community. From this perspective damage is equivalent to an "adverse" effect. In this context, yield loss is easily identified as an adverse effect.

Concerning the magnitude of the change that is of concern, one could pick some absolute value (e.g., tons) or percentage (e.g., 1%, 10%); but there is an element of arbitrariness in the selection of any particular numerical value. Clearly, the answer to the question of what constitutes an adverse effect is a policy judgment. Previous attempts to answer this question by imposing a specified percentage yield decrement to be avoided were criticized by members of the scientific community at the O_3 CASAC meeting of April 1986. Some CASAC members argued that 10 percent

loss in yield may not be a meaningful criterion for all crops and that economic significance needs to be considered when determining the adversity of a given effect. For example, a 10 percent decrement on a low valued crop does not have the same welfare effect as a 10 percent decrement on a high valued crop. An economic measure of adversity can serve to weight yield effects according to their relative impact on the well-being of agricultural producers and consumers.

For these reasons, a measure of adversity such as percent or absolute decrement in crop value is useful in addition to a physical effect measure such as percent yield and growth decrement. However, the range of economic effects resulting from O_3 reductions will not be uniform across agricultural producers, consumers, income groups, and geographic areas. Thus, a single economic measure of adversity may be less appropriate than a more extensive analysis that considers the distribution of economic effects.

Clearly, the answer to the question of what constitutes an adverse effect is a policy judgment. The definition of "adverse" ultimately involves an interplay among scientists and society, represented by the Administrator. While the role of the scientist is to identify or describe the effect and explain why it is adverse, the Administrator must determine if he is willing to accept the impact/consequence of the effect or take action (i.e., expend money and resources) to limit the consequences of the adverse effect. Information on both physical effect measures and measures of economic welfare should be useful in making this policy judgment.

Two national models currently exist that have used NCLAN crop-response data, along with adequate aerometric and economic information, to estimate the magnitude and distribution of agricultural economic benefits associated with the control of O_3 (Adams et al., 1984 and Kopp et al., 1985). Both of these models measure benefits as the change in producer and consumer surpluses resulting after O_3 control. These models can be used to

calculate economic measures of adversity (such as percent change in farmer profits), as well as to describe the distribution of economic impacts.

A major shortcoming of the Adams et al. (1984) and Kopp et al. (1985) models is that neither model accounted for on-going U.S. agricultural subsidy policies. Agricultural subsidies tend to distort prices such that they do not reflect the true value of additional production and thus make the value of increased production difficult to assess. The forum of agricultural price support policy can have a substantial impact on the benefits realized from any reduction in O_3. Measures of economic benefits must be based on realistic assumptions concerning the features of agricultural policy and benefit measures must be interpreted as being conditional upon the existence of the policy being assumed.

The original Kopp-Adams measures of benefits may be misleading because they ignore crop surpluses caused by agricultural subsidies. McGartland (1987) argued that increased production due to decreased O_3 could exacerbate crop surplus problems, and that the Adams et al., 1988 and Kopp et al., 1988 models substantially overestimated the economic benefits from O_3 control. Madariaga (1988) argued that additional surpluses are not likely to occur under current provisions in the law. The Food Security Act of 1985 allows for subsidy reductions and requires acreage restriction increases to counter additional surplus production. However, despite the provisions of this Act, future farm policies and the resulting economic effects on agriculture are uncertain.

Because of CASAC's interest in economic measures of adversity, EPA conducted an illustrative analysis (Table XI-1) which presents national benefit estimates for nine crops, three air quality scenarios, and three measures of benefits using a modified version of the Kopp et al., 1988 model. The nine crops that have been studied were selected based on economic importance and data availability. The three crops soybeans, corn, and wheat alone account for more than one-half of the value of all U.S.

sales of agricultural crops. The estimates in Table XI-1 represent benefits from reducing seasonal ambient O_3 levels (7-hr means) to 60, 45, and 30 ppb under three different assumptions regarding agricultural policies. In response to CASAC's recommendations, three separate measures of economic benefits are presented. Measure (1), the original Adams/Kopp approach, assumes no agricultural market distortions. Measure (2), the approach suggested by McGartland, accounts for market distortions but assumes no acreage restriction increases or other policy adjustments to counter additional surplus production. Measure (3), the approach suggested by Madariaga, is a cost savings measure of benefits that takes both market distortions and policy adjustments to increased surpluses into account. The benefit estimates for soybeans, peanuts, barley, sorghum, oats, and alfalfa were calculated as the simple change in producer and consumer surpluses after O_3 control, since the prices of these crops are not supported.

Given present agricultural policy, the benefits of controlling O_3 are partly dissipated by the costs of the additional excess production of farm crops. This can be seen by comparing measures 1 and 2 in Table XI-1. An alternative policy that effectively prevented an increase in excess production would permit the realization of greater net economic benefits from O_3 control. This can be seen by comparing measure 3 with measure 2 in the table.

The total benefit estimates presented in Table XI-1 can be used as measures of the agricultural welfare impacts resulting from given O_3 changes. Adversity may better characterized using reduced economic value and physical effect decrements. Nevertheless, the choice of a specific threshold level of economic benefits that is to be considered as significant, is still somewhat arbitrary and open to subjective judgment. Further, distributional concerns may be of importance. Results from the modified Kopp et al. model indicate that the distributional impacts on agricultural producers and consumers

from reducing O_3 will be non-neutral and will depend on farm policies. For example, corn, wheat, and cotton producers would benefit substantially from O_3 reductions if producer prices for these crops were fixed by agricultural policies. In contrast, it was estimated that soybean producers would actually lose profits after O_3 reductions since increased production would cause large price declines. The big gainers in this case would be soybean consumers. Any use of economic information to assess the welfare impacts from changing O_3 levels should take these considerations

Table XI-1. U.S. Agricultural Welfare Benefits from Reducing Rural Ambient Ozone (7-hr seasonal means) to 60, 45, and 30 ppb for Three Alternative Benefit Measures (millions of 1986 $)

		Crop Benefits					
Ozone Reduction (ppb)	Benefit Measure	Soybeans	Corn	Wheat	Cotton	Other Crops*	Total
60	(1)	4	0	0	6	N/A	10
60	(2)	4	0	0	6	N/A	10
60	(3)	4	8	3	9	N/A	24
45	(1)	248	77	169	639	N/A	1133
45	(2)	248	30	101	617	N/A	996
45	(3)	248	314	435	642	N/A	1639
30	(1)	927	415	426	1351	103	3222
30	(2)	927	105	259	1250	103	2542
30	(3)	927	1019	1000	1107	103	4156

NA = Not Available
*Other Crops include: peanuts, barley, sorghum, oats, and alfalfa.

into account. In part, because the views of CASAC regarding the use of economic information in determining adversity are so divergent, EPA plans to provide the Administrator with information on physical effects and economic measures of

adversity to use at his discretion in reaching a final decision on the O_3 secondary NAAQS.

During the last SP review the only approach available for analyzing estimated crop loss for alternative averaging times was that of Larsen et al. (1988). The preliminary results of Lee et al. (1988c) provide an alternative approach for evaluating alternative exposures indices and the crop losses associated with them. It should be noted that while Lee et al. (1988c) is confined to a subset of the NCLAN data, three of the crops studied (corn, soybeans, and wheat) account for more than one-half of the value of agricultural production in the U.S. In addition, the cultivars selected were the dominant cultivars in the crop growing regions at the time of the study. Therefore, these results provide the information necessary to assess the appropriate range for a 1-hr secondary standard.

CASAC's past comments regarding the range for the 1-hr standard reflect a division of opinion among the Committee members. At the April 1986 meeting the Committee indicated that the current standard was not protective of vegetation, but no alternative level was suggested. At the December 1987 meeting there was support expressed for the current 0.12 ppm standard as well as for 0.10 ppm. Based on CASAC's written comments as well as comments made at the December 1987 meeting, a range of 0.08 - 0.12 ppm appears to capture the range of opinion expressed.

In light of the results of Lee et al. (1988c), it appears that the upper-end of this range offers very little protection for vegetation. As can be seen in Table X-9 (SP, p. X-52), median crop losses of 18.9% are expected in agricultural sites experiencing O_3 exposures with a HDM2 value of 0.12 ppm. Thus, half of the crops in this study experienced losses of 18.9% or greater upon attainment of the current standard. If EPA decides to use a more protective strategy such as the 75th percentile rather than the median PRYL's, the crop loss in agricultural sites attaining the 0.12 ppm standard could go as high as 34.8% (Table X-9, SP, p. X-52). In addition, a clear majority of CASAC

members at the December 1988 meeting felt that the current standard was not protective of vegetation. Therefore, staff concludes that 0.06 ppm - 0.10 ppm is an appropriate range for the 1-hr standard.

Lowering the HDM2 to 0.10 ppm would result in an estimated median crop loss of 12.8%, which could go as high as 28.8% when using the 75th percentile of PRYL's (Table X-9, SP, p. X-52). Similarly, lowering the HMD2 to 0.08 would result in an estimated median crop loss of 7.9% which would go as high as 22.1% when using the 75th percentile of PRYL's. EPA concludes that in order to be protective of anything other than median crop losses, the bottom end of the range needs to be lowered to 0.06 ppm. This would allow for a 15.9% or greater yield loss in 25% of the crops at 0.06 ppm as compared to 22.1% or greater loss in 25% of the crops at 0.08 ppm.

While these results are preliminary, they do provide additional information on which to base a decision on the level of a 1-hr standard. CASAC comment on the methodology and the results of this analysis help determine how these results should be factored into the range. In the judgment of EPA staff, a range of 0.06-0.10 ppm takes into consideration the results of Lee et al. (1988c) as well as CASAC comments regarding the upper end of the range.

D. Staff Conclusions

Based upon the assessment in Section XI.A-E, the staff conclusions regarding the welfare effects of O_3 are as follows:

1. In consideration of the large base of welfare information attributing effects to O_3 exposure and the limited evidence which demonstrates welfare effects from exposure to ambient levels of non-O_3 photochemical oxidants, there appears to be little evidence to suggest a change in chemical designation from O_3 to photochemical oxidants.

2. Because the bulk of the data available since the last standard review uses growth and yield loss as the response

measure, this parameter could be utilized to evaluate different levels of the secondary standard. Alternatively a broader range of effects could be considered adverse including: foliar injury, premature senescence, reduced photosynthesis, altered carbon allocation and reduced plant vigor. Although there is no precise relationship between these effects and growth and yield reduction, these more subtle effects are often early warning signals of potentially harmful effects on plant vigor. In addition, economic measures of adversity such as changes in producer and consumer surpluses resulting after O_3 control may be useful in evaluating alternative levels of the secondary standard.

3. The weight of the recent evidence seems to suggest that long-term averages, such as the 7-hour seasonal mean, may not be adequate indicators for relating O_3 exposure and plant response.

4. Repeated peak concentrations accumulated over time are the most critical element in determining plant response for agricultural crops. Exposure indicators which emphasize peak concentrations and accumulate concentrations over time probably provide the best biological basis for standard setting. Unfortunately, the analysis currently available on such indicators is preliminary. EPA seeks CASAC advice and comment on the methodology as well as the suggested exposure indicators (SUM06 and 07).

5. There is currently a lack of exposure-response information on forest tree effects. In addition, there is a broad range of uncertainty among scientists regarding O_3 effects on forest trees. Consequently there is no consensus on the most important averaging time for perennials or on the precise role of O_3 vs. other pollutants in causing forest decline. Therefore, staff concludes that a separate secondary standard for protection of forest trees is not warranted.

6. Given the lack of effects data on forests and the preliminary nature of the Lee et al. (1988c) results regarding selection of the appropriate exposure statistic for crops, the

EPA staff concludes that it may be premature at this point in time to change the form of the standard and averaging time. It is our judgement that a 1-hr averaging time standard in the range of 0.06 - 0.10 ppm represents the best staff recommendation that could be made to the Administrator at this time to close out the review of the scientific data. With this portion of the review complete, and after considering CASAC's views on all issues, the Administrator will be in a position to make a regulatory decision on how and when to best act on the 1-hr standard. This is consistent with CASAC comments (December 1987 and December 1988 meetings) urging EPA to consider a 1-hr averaging time and act on the existing state of science rather than extend the review until a more exhaustive assessment is made of alternative averaging times.

Alternatively, EPA could continue the standard review. Additional time for review of information on alternative exposure indicators would allow the scientific community the opportunity to move toward a consensus regarding selection of the most appropriate exposure indicator. The liability of this alternative is that it postpones action on the secondary standard and thus fails to utilize new and existing data to assess the most appropriate exposure indicator or the protection afforded by the current 1-hr standard.

7. There appears to be no threshold level below which materials damage will not occur; exposure of sensitive materials to any non-zero concentration of O_3 (including natural background levels) can produce effects if the exposure duration is sufficiently long. However, the slight acceleration of aging processes of materials which occurs at the level of the NAAQS is not judged to be significant or adverse. Consequently, the staff concludes that materials data should not be used as a basis for defining an averaging time and concentration for the secondary standard and that the secondary standard be based on protection of vegetation.

8. Effects on personal comfort and well-being, as defined by human symptomatic effects, have been observed in clinical studies at O_3 levels in the range of 0.12 - 0.16 ppm and at somewhat lower levels in extended exposure epidemiological studies. These effects include nose, and throat irritation, chest discomfort, cough, and headache. In addition, cough, chest pain on deep inspiration, chest tightness, wheezing, lassitude, malaise, and nausea have been reported in controlled O_3 exposure studies. CASAC recommended that these effects be considered health effects in developing a basis for the primary standard for O_3.

APPENDIX A: AIR QUALITY

This Appendix characterizes existing O_3 ambient air quality in the United States. Emphasis is placed upon five indices of air quality:

- the expected number of exceedances of the current 0.12 ppm O_3 NAAQS. (Hereafter often cited as the 1-hour ExEx index.)
- the second-highest 1-hour daily maximum O_3 concentration. (Hereafter often cited as the 2nd-high daily max index.)
- the number of days in an O_3 season with a 8-hour daily maximum average greater than a specified concentration level. Four concentrations are used: 0.06, 0.08, 0.10, and 0.12 ppm. (Hereafter often cited as 8hrDM>.06, etc.)
- the maximum monthly mean of 1-hour daily maximum O_3 concentrations. (Hereafter often cited as the max monthly mean index.)
- the maximum 3-month mean of 8-hour daily maximum averages. Hereafter often cited as the 3-month mean indicator.)

The first two indicators are those normally used by EPA to ascertain if an area violates the current O_3 NAAQS. The remaining three indicators are potentially useful formulations for considering possible alternative longer-term primary and secondary O_3 NAAQS. The 8-hour indices are useful for describing how many days have an 8-hour exposure greater than an O_3 concentration level thought by some researchers to cause adverse welfare effects (Lee, et al., 1987a,b) or adverse health effects (Lioy et al., 1985). The fourth indicator is a possible surrogate for both short- and long-term standard formulations in that it is relatively closely associated with them in a statistical sense. The last indicator--the 3 month mean--is a simplified formulation of the 7-hour long-term mean used by NCLAN researchers in investigating O_3 impacts on agricultural crops (Heck and Tingey, 1971). All of these indicators have been extensively reviewed in previous Ambient Standards staff analyses (McCurdy 1987, McCurdy and Atherton 1988); these analyses form the basis for all results presented in this Appendix unless otherwise noted.

The material presented here expands upon and updates information presented in Chapter 5 of the CD and numerous air quality reports. The data generally come from 1985-1987 information contained in EPA's AIRS-AQ subsystem (Aerometric

Information Retrieval System-Air Quality), but data are presented for other time periods also.[1]

A. Discussion of the First Two Air Quality Indicators

The focus on expected number of exceedances (the 1-hour ExEx indicator) is due to the formal definition of the O_3 NAAQS:

> The standard is attained when the expected number of days per calendar year with maximum hourly concentrations above 0.12 parts per million (235 $\mu g/m^3$) is equal to or less than 1. (40 CFR 50.9, 1984)

The expected number of exceedances is determined by a mathematical formula explained in Appendix H of 40 CFR 50 (1984), and repeated here in simplified form to remove a term:

$$\text{1-Hour ExEx} = \frac{v * (N - z)}{n}$$

where:
- v = number of measured 1-hour daily maximum values >0.12 ppm
- N = number of days in the O_3 season
- z = number of days assumed to have a 1-hour daily maximum $\leq$0.12 ppm
- n = number of days having a valid 1-hour daily maximum O_3 value

This formula, and its application, is described in an EPA guidance document (Curran, 1979).

The second air quality indicator of interest here is one of the variant approaches to defining the characteristic highest concentration (CHC). For our purposes, the CHC is that daily maximum air quality value that is expected to be exceeded no more than one time per year (during the "O_3 season," actually) on average. There are a number of ways to choose the CHC, ranging from using fitted statistical distributions to a tabular "look-up" approach (Curran, 1979). A modified version of the look-up approach is used here because it is the simplest method and is used by most states in analyzing O_3 air quality (AMTB, 1981). The CHC under this approach is the n+1 highest observed daily maximum value where n equals the number of years of valid data.[2]

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[1]See the footnote on p. IV-1 regarding more recent O_3 air quality data.

[2]A valid year of data is one that contains a valid daily maximum one-hour average for 75% of all the days in the state-defined O_3 season. A valid day has data for 75% of the hours

Thus, if there are three years of data, the CHC is the 4th-highest value in the ranked order data set. If there is one year of data, it is the second-highest value.

B. Ambient Ozone Air Quality in Urban Areas

There are 331 Metropolitan Statistical Areas (MSAs) in the 50 states. Ozone air quality data for every MSA were reviewed against the 75% completeness criteria noted below in the footnote. Sites meeting these criteria were determined to have "valid data" and were used to determine which site in a MSA had the highest CHC for the time period. This site is often called the "design value" site in a MSA. Each MSA is represented by only one year of data in most of the analyses reported here. It will be made clear where multiple years of data were used.

The 1985-1987 data were supplemented in some areas with valid 1980-1984 data when recent valid data were not available for a MSA. This was done in 37 MSAs. Including these areas, valid O_3 data are available for 224 MSAs--about 68% of all U.S. metropolitan areas.

1. Expected Exceedances of the Existing Ozone NAAQS

The data indicate that 101 MSAs (45.1% of those with data) had more than one expected exceedance per year of the O_3 NAAQS. Thus, a significant proportion of U.S. urban areas exceeded the existing O_3 standard concentration level during the time period analyzed.

Approximately 100 million people live in these high O_3 MSAs.[3] That is slightly below one-half of the total U.S. population (using a 1988 estimated population of 230 million people). Over 16 million people live in MSAs that had 20 or more estimated expected exceedances in at least one year between 1985

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between 9 am and 9 pm (9 hours). An O_3 season is a fixed period of months that is expected to contain most, if not all, high O_3 days in the year for each state; it varies from 4 months (June through September) in northern-tier states to 12 months in some southern states. The defined O_3 season may be shortened in a number of States, especially those with a 12 month season, because exceedances are rare in the winter-time, even in the South.

[3]This does not mean that all persons living in these MSAs experience an O_3 concentration >0.12 ppm as they go about their daily lives. People living in these MSAs have the potential for doing so, but many people living in even moderately polluted O_3 areas never experience O_3 levels greater than 0.12 ppm. For a discussion of this, see Paul, et al. (1986).

and 1987. Over 18 million people live in MSAs that had between 10 and 20 expected exceedances.

Part of the cumulative frequency distribution of 1-hour ExEx data is shown in Table A-1, along with similar information for two other air quality indicators that will be discussed later. The median number of 1-hour expected exceedances is 1.1; however, there is a lot of variability in the data. Although not directly observable in Table A-1, 37% of the MSAs investigated[4] have 0 exceedances. The mean of the sample, almost 5 exceedances, is much greater than the median due to the very high number of expected exceedances in the worst areas. The worst MSA is Los Angeles, with about 125 exceedances.

Expected exceedances data for the 1979-1987 time period were investigated in a sample of the 224 MSAs to determine if a temporal trend existed in the 1-hour ExEx indicator. A MSA was included in this trend analysis if it had 3 or more valid years of data in the 1979-1987 period. Under this criterion, 128 MSAs had sufficient data for trend analysis. An area was considered to have a trend in its expected exceedance index if the slope coefficient for the temporal variable was significant at $p<.10$.[5] Of the 128 areas, only 1 (<1%) had an increasing trend in expected exceedances, while 19 (15%) had a decreasing trend. If the rejection significance level were tightened to $p<.05$, no MSA had an increasing trend while only 9 MSAs (7%) had a decreasing trend. However, it should be emphasized that the trend results are quite crude because of the lack of sufficient data over a longer time period. In addition, O_3 air quality was

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[4]The sample size for the data shown in Table A-1, and many of the Tables that follow, is 224 MSAs. While the base time period is the same, 1985-1987, 2 MSAs did not have data coverage sufficient enough to calculate longer-term means. Since this sample is used to develop relationships among air quality indicators, the smaller sample size is used for the relationship analyses presented later.

[5]The trend indicator needs further explanation. With only a maximum of 9 years of data for trend analysis purposes, it is analytic overkill to undertake sophisticated time series analyses to determine whether or not a statistically significant temporal trend is occurring in the MSA sample. The "degrees of freedom" just are not there to fit a time series or a multivariate model to the data. It was therefore decided to do a very simple trend analysis using a linear regression approach.

The trend regression used "annual MSA expected exceedances" as the dependent variable and "year" as the independent variable. If the slope coefficient of year was significantly different than 0 (at $p<.10$) for a MSA during the 1979-1987 time period, that MSA was said to have a trend in ozone air quality.

Table A-1

CUMULATIVE FREQUENCY DESCRIPTIVE STATISTICS ASSOCIATED WITH PEAK AND MULTIPLE-HOUR OZONE AIR QUALITY INDICATORS IN URBAN AREAS

Statistic	Expected Exceedances of 0.12 ppm 1-Hour Daily Maximum	2nd-High 1-Hour Daily Maximum (ppm)	Observed Exceedances of 0.08 ppm 8-Hour Daily Maximum
Mean	4.6	.126	14.9
Std. Dev.	13.1	.036	20.9
Minimum	0.0	.039	0
Median	1.1	.119	9
75th Percent.	4.0	.140	18
90th Percent.	10.0	.167	29
95th Percent.	14.8	.180	38
Maximum	125.3	.340	150
Sample Size	224	224	224

Note: Std. Dev. = Standard Deviation
Percent. = Percentile

Source: McCurdy and Atherton (1988).

quite bad in 1988 over much of the United States, and using 1988 data in the regression analyses might change many of the significant down-trending results.

Additional information on trend in the 1-hour ExEx indicator is available. EPA's Monitoring and Data Analysis Division (MDAD) undertakes air quality trends analyses of all NAAQS pollutants on an annual basis (MDAD, 1988).

MDAD's trend analyses used 242 sites to investigate if a trend existed in the number of expected exceedances over the ten-year time period 1977-1986. The composite average of the expected number of exceedances decreased 38% during that time. 1979, 1980, and 1983 values are higher than the other years.

2. Second-Highest 1-hour and 8-hour Daily Maximum Air Quality Indicators

Cumulative frequency descriptive data for these two air quality indicators appear in Table A-1, previously introduced. The Table indicates that the median 2nd-high 1-hour design value for the sample of 224 MSAs is slightly lower than the current O_3 NAAQS level. The mean is higher than the current standard, 0.126 ppm, because of very high design values seen in a few MSAs. For instance, 10% of the MSAs have a 2nd-high daily max >0.167 ppm. The worst 5% have a design value >0.180 ppm. The highest design value sites are located in MSAs near or adjacent to the New York, Los Angeles, and Chicago major metropolitan areas.

Looking at the temporal trend in the 2nd-high daily max indicator, the <u>composite</u> <u>average</u> of this indicator decreased 13% between 1979 and 1986 (MDAD, 1988). This decrease is modest in the post-1983 time period, and 1983 2nd-High values are higher than adjacent years' values. Thus, the trend in this indicator is not monotonic. If trend in individual MSAs is investigated, rather than a composite average, it is obvious that there is a cyclical pattern in 2nd-high daily max at most sites.

As seen in Table A-1, there are many expected exceedances of an 8-hour daily maximum >0.08 ppm. There also is wide variability in their number, from 0 for 63 MSAs (28% of the sample) to 150 for the worst MSA. The mean is about 15, and the standard deviation of the sample is larger than the mean.

3. Alternate 8-Hour Daily Maximum Air Quality Indicators

In previous O_3 Staff Papers, the 8hrDM indicator of interest focused on a concentration level of 0.08 ppm. (See Table A-1.) Recent interest has focused upon alternative concentration levels, such as 0.06, 0.10, and 0.12 ppm. Descriptive statistical data for these concentration levels appear in Table A-2.

Table A-2

CUMULATIVE FREQUENCY DESCRIPTIVE STATISTICS ASSOCIATED WITH VARIOUS 8-HOUR OZONE AIR QUALITY INDICATORS IN URBAN AREAS

Statistic	Observed Exceedances of 0.06 ppm 8-hour Daily Maximum	Observed Exceedances of 0.08 ppm 8-hour Daily Maximum	Observed Exceedances of 0.10 ppm 8-hour Daily Maximum	Observed Exceedances of 0.12 ppm 8-hour Daily Maximum
Mean	47.4	14.9	4.2	1.4
Std. Dev.	24.9	20.9	11.8	6.8
Minimum	0	0	0	0
Median	48	9	1	0
75th Percent.	61	18	4	0
90th Percent.	77	29	10	2
95th Percent.	87	38	14	5
Maximum	183	150	111	74
Sample Size	204*	224	224	224
No. of MSAs ≥ 1	197	161	49	30
% of Sample	96.6	71.9	21.9	13.4

NOTE: Std. Dev. = Standard Deviation
Percent. = Percentile

*This indicator cannot be computed for many of the MSAs in California. These MSAs are expected to have many days with an 8-hr DM > .06 ppm, which would greatly affect the statistics shown for this indicator.

Source: McCurdy and Atherton (1988).

Note the great differences among the alternative 8-hour indicators in the mean number of days above the various concentration "cutpoints." The average MSA has 3 times as many days with an 8-hour daily maximum average over 0.10 ppm as it does over 0.12 ppm. It also has about 3.5 times as many days above 0.08 ppm as it does over 0.10 ppm.

Finally, Table A-2 shows that the average MSA has about 3 times as many days with an 8-hour DM >.06 ppm than days >.08 ppm. However, this ratio is suspect because 8hrDM>.06 data are missing for many California MSAs. Where the data are available, the mean value for 8hrDM>.06 indicator would be much higher, as would the ratio. (The median statistics are messed up for the same reason.) Relationships among the various 8-hour air quality indicators are more fully discussed in subsequent sections of this Appendix.

Note the last two lines of Table A-2. They indicate the number and relative portion of MSAs having one or more 8-hour DM average greater than the cutpoint concentration shown. Only 22% of the sample has one or more 8hrDM>.10 ppm, for instance, while 72% has one or more 8hrDM>.08 ppm.

4. Longer-term Air Quality Indicators

Two longer-term indicators of interest are the max monthly and 3-month mean indices. The daily maximum averaging time used for these indicators is 1-hour and 8-hour, respectively. Cumulative frequency descriptive information for these two indicators is shown in Table A-3.

There is moderate sample variability in the two indicators, as evidenced by the small difference between the mean and median values and the small standard deviations relative to their means. The large 3-month means of the top 5% MSAs in the sample should be noted. The median value is 0.055 ppm. The worst area has a maximum 3-month mean of 8-hour daily maximum averages of 0.132 ppm--higher than the current 1-hour daily maximum NAAQS! (The area is Los Angeles.)

5. Relationships Among Air Quality Indicators in Urban Areas

As an introduction to relationship analyses among air quality indicators, we consider Figure A-1. Depicted are linear and non-parametric correlation coefficients among the air quality

Table A-3

CUMULATIVE FREQUENCY DESCRIPTIVE STATISTICS ASSOCIATED WITH LONGER-TERM OZONE AIR QUALITY INDICATORS IN URBAN AREAS

Statistic	Maximum Monthly Mean for 1-Hour Daily Maximums (ppm)	Maximum 3-Month Mean for 8-Hour Daily Maximums (ppm)
Mean	.071	.056
Std. Dev.	.017	.012
Minimum	.025	.016
Median	.069	.055
75th Percent.	.079	.061
90th Percent.	.086	.067
95th Percent.	.099	.072
Maximum	.178	.132
Sample Size	224	222*

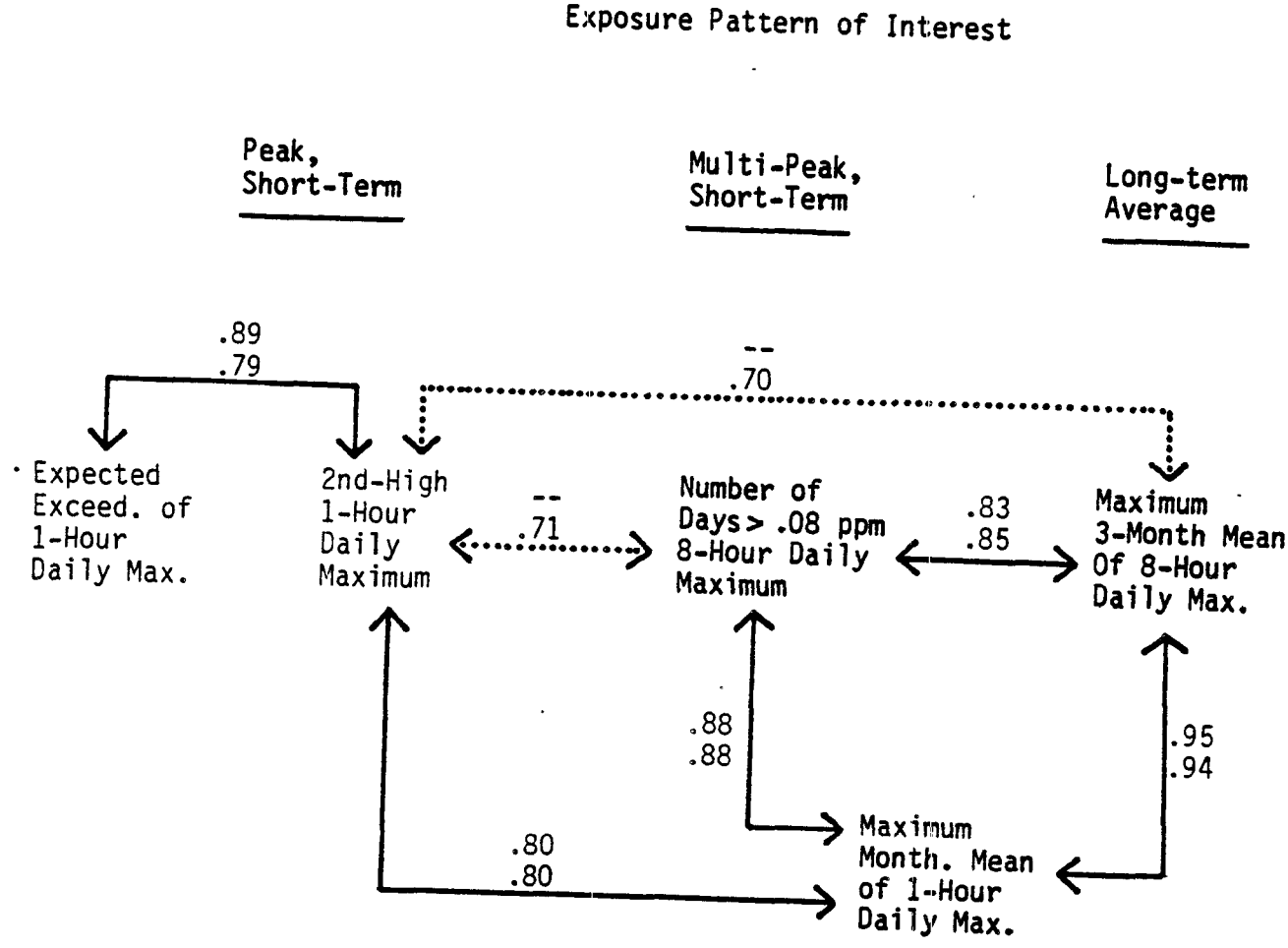
Note: Std. Dev. = Standard Deviation
Percent. = Percentile

*This indicator cannot be computed for two MSAs due to lack of a valid 3-month mean for the MSA design value monitor.

Source: McCurdy and Atherton (1988).

Figure A-1

CORRELATIONS AMONG SHORT- AND LONG-TERM AIR QUALITY INDICATORS IN MSAs (USING 2nd HIGH)



Source: McCurdy and Atherton (1988).

The sample size is 224. The top decimal along the lines refers to the Spearman non-parametric correlation coefficient. The bottom decimal refers to the Pearson correlation coefficient. Only coefficients $>|.50|$ are depicted. A dotted line is used when the coefficient is less than $|.75|$. All coefficients are significant at $p < .05$.

indicators of interest.[6] Solid lines depict a correlation coefficient >|.75|. Only the relations between (1) the 2nd-high daily max and 3-month mean indicators and (2) the 2nd-high daily max and observed number of days with an 8-hour daily max >.08 ppm are <|.75|. Note that the max monthly mean index is highly correlated with both short-term and long-term air quality indicators. This may indicate that the max monthly indicator could be used as a surrogate for the other averaging times. If a single O_3 NAAQS formulation is desired, from a purely descriptive statistical perspective it appears that it could be the maximum monthly mean of the daily maximum 1-hour averages. However, if EPA is concerned about different effects from short-term, multiple peak, and long-term exposures, the Agency should establish multiple standards specific to the exposure times and patterns causing the damage. If different effects cannot explicitly be attributed to different exposure times or patterns, then using a single air quality indicator may be an acceptable compromise as it is easier to implement than multiple standards.

Note that two peak, short-term indicators of interest are depicted in Figure A-1. The second-high "design value" indicator is the prime short-term index shown in that figure. In Figure A-2, the prime short-term air quality indicator used is the "expected exceedances index." For the most part, it is more highly correlated with the other indicators than the design value index. The correlation coefficients improve dramatically for the ExEx-number of days >.08 ppm daily max link in particular. This is probably not too surprising given that both indicators have the same general form--a "count" of daily maximum observations greater than a specified cutpoint. What may be surprising is the modest improvement in correlation coefficients for the peak, short-term to long-term average link. There is no explanation for this improvement, and it could be an artifact of the analyses.

Additional relational analyses among the air quality indicators of interest are discussed in McCurdy and Atherton (1988) and in earlier analyses (McCurdy 1987). The objective of these analyses was to simulate attainment of one air quality indicator in all areas and investigate how this affected the subsequent cumulative frequency distribution of other air quality indicators. The simulation was accomplished by fitting a logistic or exponential equation to the frequency distribution of stratified data sets. The rationale and procedure used for the stratification is described in McCurdy (1987).

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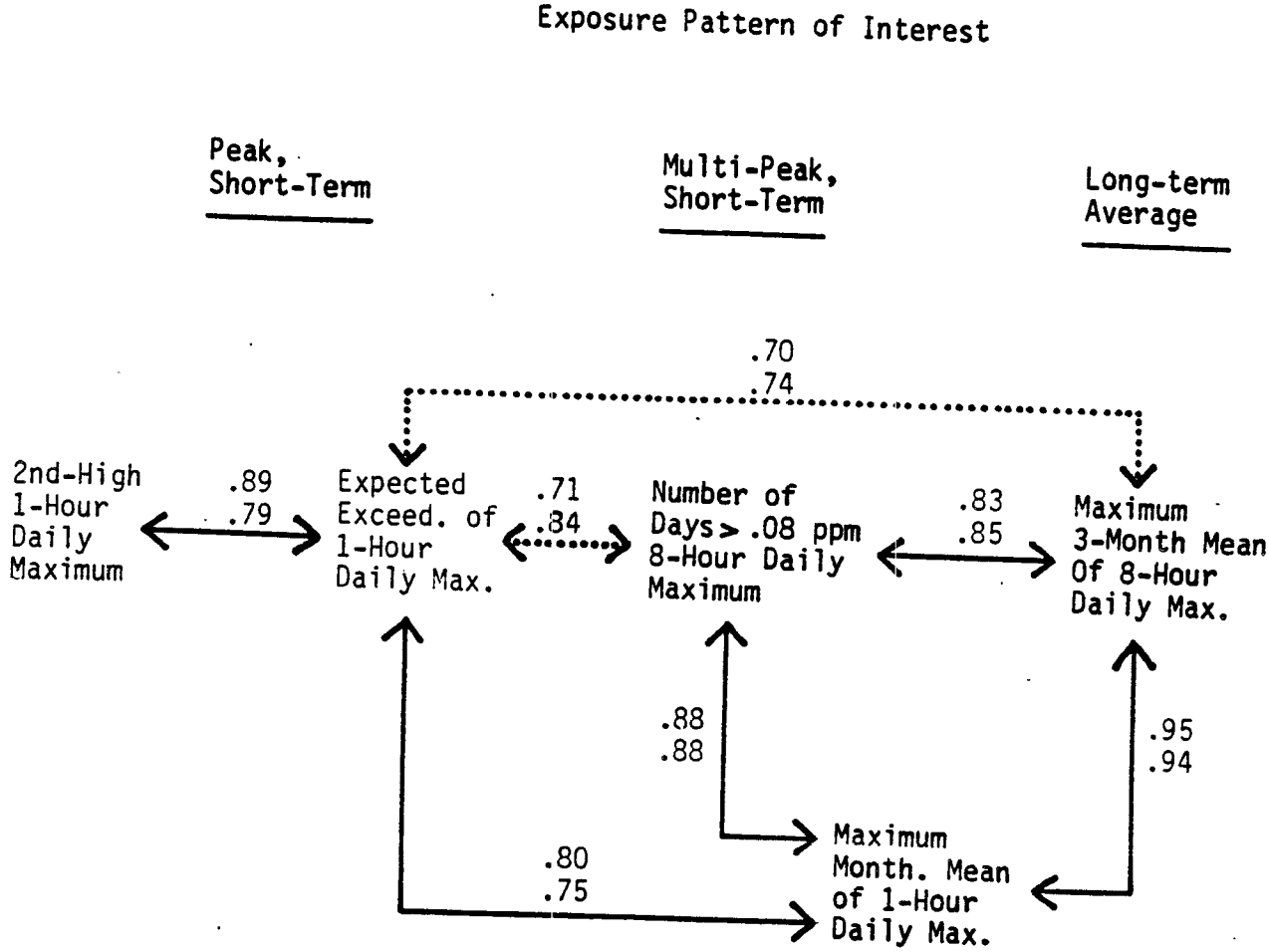
[6]The coefficients shown are the Pearson product-moment correlation coefficient (r) and the Spearman rho coefficient. All coefficients depicted in Figure A-1 (and in Figure A-2) are significantly different than zero at $p<.05$.

Figure A-2

CORRELATIONS AMONG SHORT- AND LONG-TERM AIR QUALITY INDICATORS IN MSAs (USING ExEx)



Source: McCurdy and Atherton (1988).

The sample size is 224. The top decimal along the lines refers to the Spearman non-parametric correlation coefficient. The bottom decimal refers to the Pearson correlation coefficient. Only coefficients $>|.50|$ are depicted. A dotted line is used when the coefficient is less than $|.75|$. All coefficients are significant at $p < .05$.

Figure A-3

PROPORTION (IN PERCENT) OF URBAN AREAS EXCEEDING EXPECTED NUMBER OF DAYS WITH AN 8-HOUR DAILY MAXIMUM AVERAGE >.08 ppm FOR FIVE 1-HOUR DAILY MAXIMUM STANDARDS

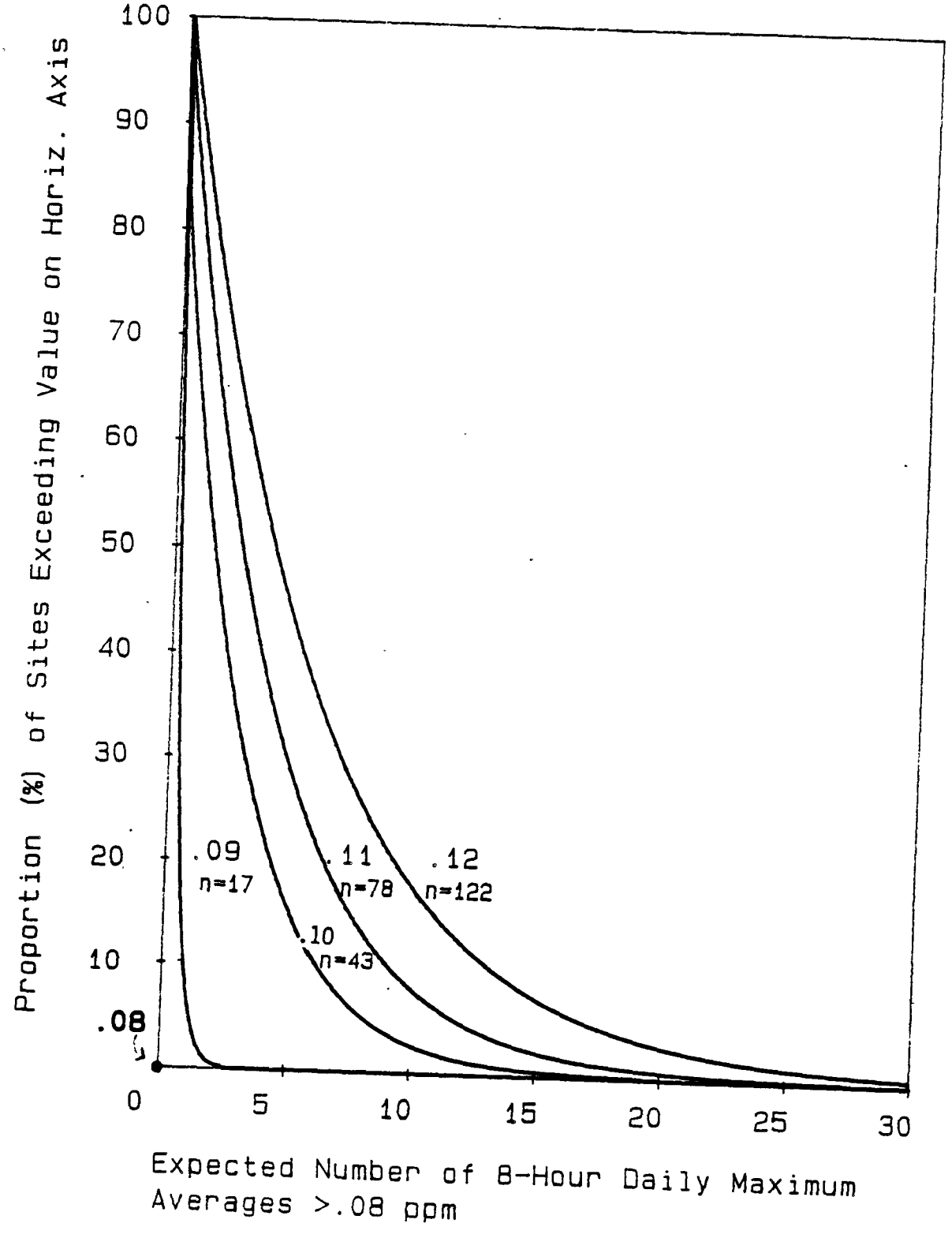


Figure A-4

PROPORTION (IN PERCENT) OF URBAN AREAS EXCEEDING EXPECTED NUMBER OF DAYS WITH AN 8-HOUR DAILY MAXIMUM AVERAGE >.06 ppm FOR FOUR 1-HOUR DAILY MAXIMUM STANDARDS

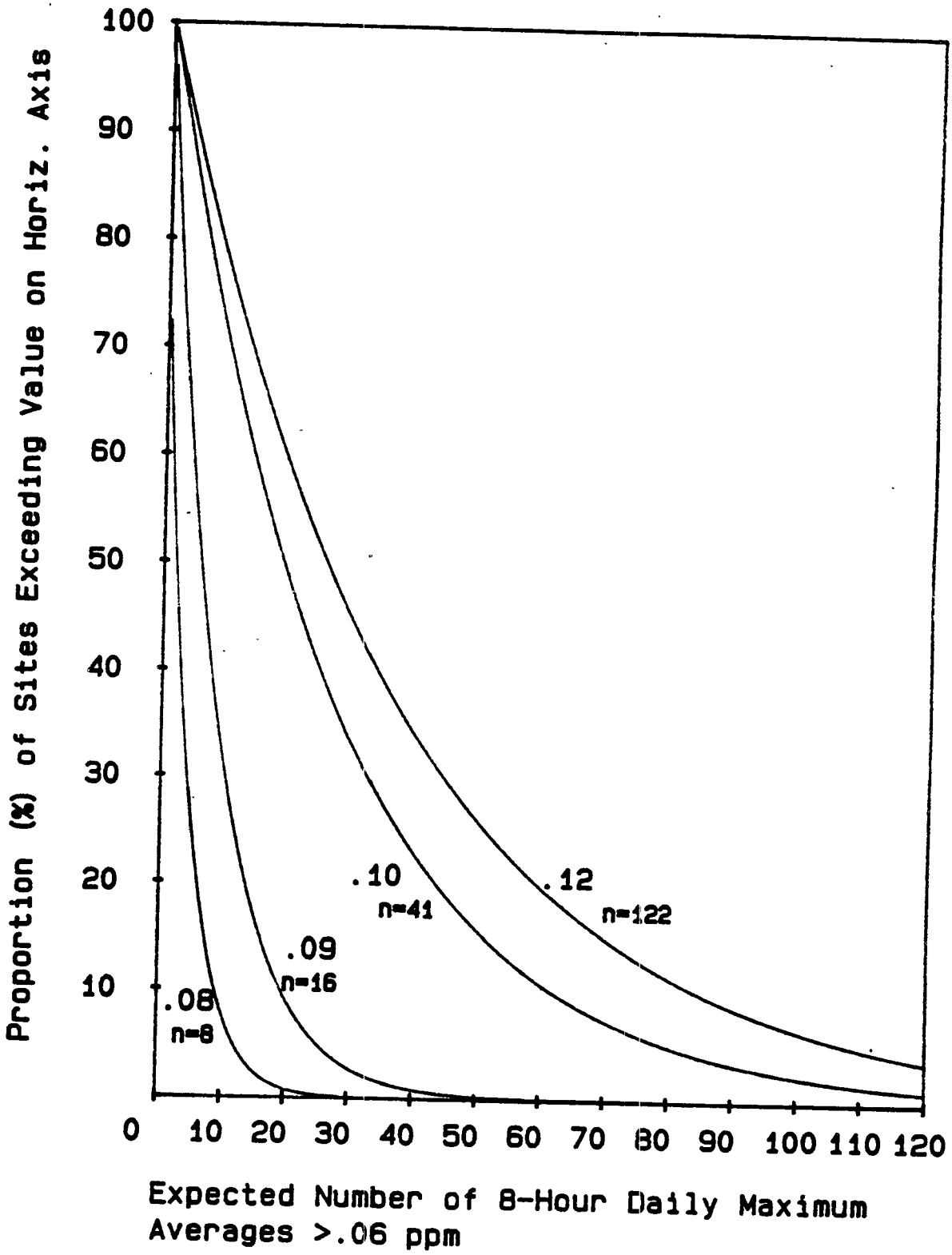
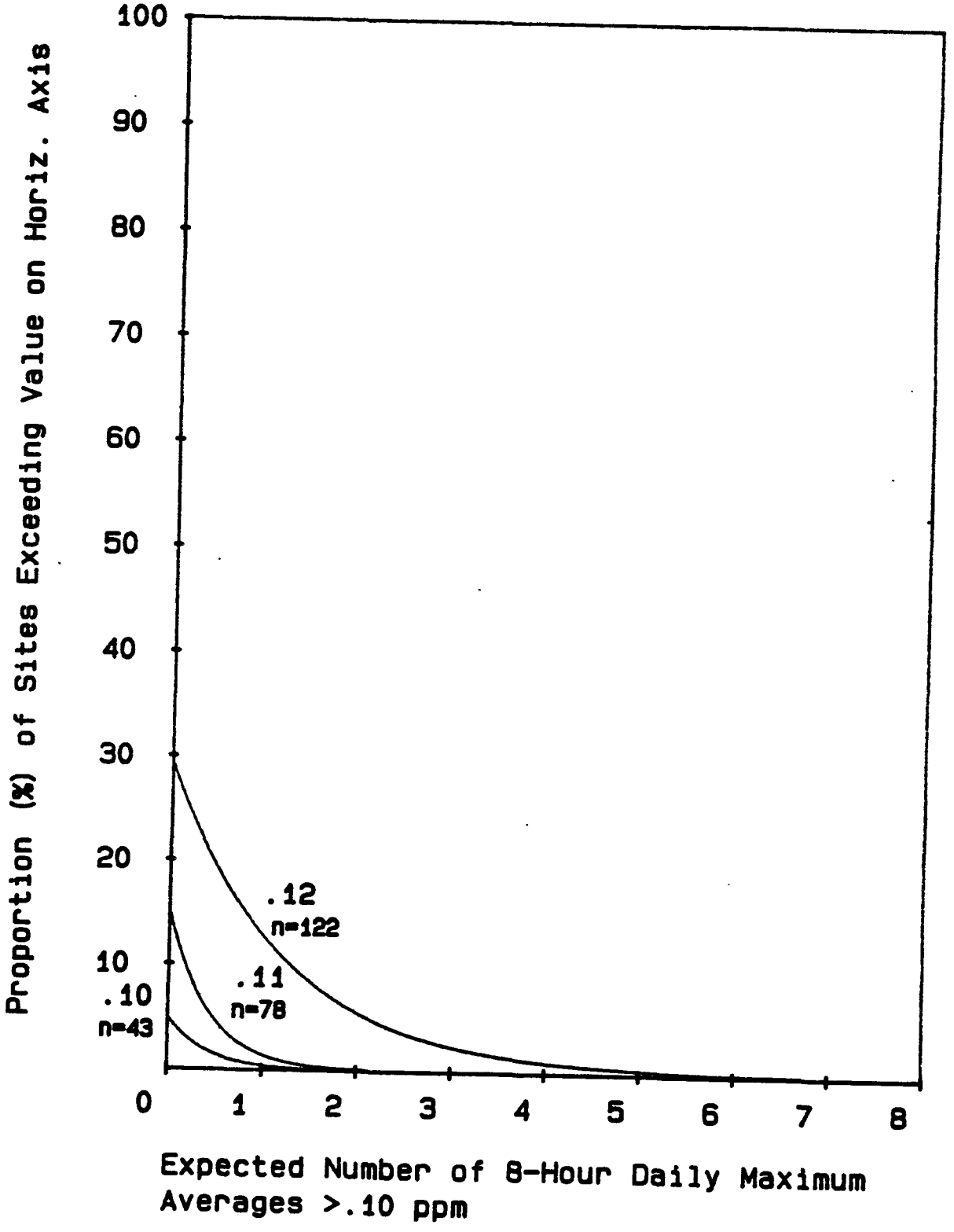


Figure A-5

PROPORTION (IN PERCENT) OF URBAN AREAS EXCEEDING EXPECTED NUMBER OF DAYS WITH AN 8-HOUR DAILY MAXIMUM AVERAGE >.10 ppm FOR THREE 1-HOUR DAILY MAXIMUM STANDARDS



The relationships among certain air quality indicators are graphically depicted in Figures A-3 through A-5. Figure A-3 relates the number of 8-hour daily maximum averages >.08 ppm (x axis) to five alternative 1-hour standards: 0.08, 0.09, 0.10, 0.11 and 0.12 ppm. Since the x-axis variable is an interval-scale expected exceedance variable in this case, the curves follow a negative exponential shape.

An example or two may aid the reader in reading Figure A-3 and subsequent curves in this Section. The Figure indicates that if a 0.12 ppm 1-hour daily maximum standard is met by all sites in the data set, 10% of all MSAs might have 13 or more days with an 8-hour daily maximum >.08 ppm, on average. At least one area might have as many as 30 days. Fifty percent of the MSAs might have 4 or more days with an 8-hour ExEx indicator >.08 ppm, on average. If the 1-hour daily maximum air standard was lowered to 0.10 ppm and attained by all areas in the data set, 10% of the MSAs may have 6 or more days over 8hrDM > .08 ppm, on average. If a 0.08 ppm daily max 1-hour standard were attained, no area would be expected to have any day with an 8-hour ExEx value >.08 ppm.

Figure A-4 is like Figure A-3 except that the 8-hour average cutoff concentration is now 0.06 ppm. Note that there are many more expected exceedances of 0.06 ppm than 0.08 ppm for any 1-hour daily maximum standard (compare A-4 with A-3). The ratio is anywhere between 8:1 and 20:1 for the various 1-hour standards analyzed.

It should be noted that there is no 0.11 ppm 1-hour daily max standard depicted in Figure A-4. It so closely followed the 0.10 ppm curve that it was deleted for the sake of clarity. Thus, it can be said that there is no practical difference between 0.10 and 0.11 ppm 1-hour NAAQS with respect to reducing the number of days with a 8-hour daily maximum >.06 ppm.

In order to reduce the expected number of days with an 8-hour O_3 daily max >.06 ppm down to, say, 20 per year in the worst 10% of urban areas would require a 1-hour NAAQS of about 0.09 ppm. To further reduce these days to 10 per year may require a 1-hour O_3 NAAQS of 0.08 ppm.

Figure A-5 depicts the relationship between alternative 1-hour daily max standards and the number of days with an 8-hour daily max average > .10 ppm. If the current O_3 standard would be attained everywhere, the 10% worst area probably would have only slightly more than 1 day per year with an 8-hour daily max >.10 ppm, on average. There would be less than 1 day per year on average in 98% of all areas if an O_3 NAAQS of 0.11 ppm or lower were attained everywhere.

As previous CASAC reviewers of relationships like those depicted in Figures A-3 to A-5 have recognized (CASAC 1988), there is uncertainty associated with the estimates just discussed. This uncertainty results from two factors. The first is a lack of fit between the observed data and the cumulative distribution function fitted to it (i.e., the 2-parameter exponential). The second is due to sample variability. Both sources of uncertainty have been addressed for some of the relationships presented. (Because of the tedious nature of the statistical tests available for goodness-of-fit and confidence intervals around predictions from a cumulative distribution exponential function, it was impossible to test very many of the relationships depicted in the three Figures.) The uncertainty analyses undertaken are described in McCurdy and Atherton (1988). Excerpts are discussed next.

The goodness-of-fit tests indicate that the tested fitted distributions adequately describe an exponential cumulative distribution function, at least down to the 0.09 ppm 1-hour daily maximum standard.[7] (Many of the empirical "functions" become a single point below a 0.10 ppm 1-hour daily maximum and cannot be tested.)

The tests of uncertainty due to sample variability used standard confidence interval estimation techniques (for a linear regression line fitted to the log-transformed cdf data). It was discovered that the 95 percentile confidence intervals:

1. are ± 25-30% for the 0.12 ppm 1-hour daily maximum standard.

2. are ± 42-129% for the 0.10 ppm standard.

3. overlap for standards having an interval of 0.01 ppm (e.g., 0.12 and 0.11 ppm).

4. do not overlap for the 0.10 and 0.12 ppm standards using the current (1985-1987) data set.

It was also found that the 95 percentile prediction interval for the various standards is very wide (± 105%) and includes 0. Thus, there may be no relationship in any individual urban area between, say, the 0.12 ppm 1-hour daily maximum and number of

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[7]The null hypotheses is that the estimated cumulative distribution function (cdf) represented by G(x) is not significantly different than the "true" cdf represented by a 2-parameter exponential distribution ($F_n(x)$. The test then is $F_n(x) - G(_x) = 0$, using the Kolmogorov D statistic to evaluate the hypothesis at $p < .05$ for a one-sided test.

exceedances of the 8-hour daily max >.08 ppm. Additional work is underway to refine these uncertainty estimates.

Numerous ratio analyses were undertaken among the urban O_3 air quality indicators. For instance, the mean ratio of the 2nd-highest 8-hour daily max to the 2nd highest 1-hour daily max is 0.811 (s.d.=.082), with a range for the 224 area data set of 0.467 to 0.940. These statistics are similar to those found by other investigators.

Other relational analyses focused upon exceedances of various 8-hour cutpoints vis-a-vis exceedances of the current O_3 NAAQS. One such analysis fitted curves having the general form 8hr>(c) = a * (1 ExEx ** b), where (c) represents the cutpoint concentration of interest (e.g., .08 ppm, etc). The fitted curves for R (c >.06) are depicted in Figure A-6. While the curves show the general relationship between the two air quality indices for alternative cutpoints, they are misleading at ExEx=0 (at x=0). At ExEx=0, the predicted number of exceedances of the alternative 8-hour cutpoints as depicted in Figure A-6 are all 0. In reality, that is only true for R(.12); at an ExEx or x of 0, the observed data indicate the following exceedances:

	R(.06)	R(.08)	R(.10)	R(.12)
Minimum	0	0	0	0
Median	37	4	0	0
Mean	36.1	5.0	0.3	0
Maximum	102	23	6	0

Thus, the equations in general underestimate the number of 8-hour exceedances for the 0.06, 0.08, and 0.10 ppm cutpoints for 0 exceedances of the 1-hour daily maximum NAAQS.[8]

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[8]While a statistic such as R^2 is not readily available for non-linear equations using the PC statistical package available to us, one index of goodness-of-fit of the equation might be to divide the "explained" sum-of-squares due to the regression equation by the total sum-of-squares. Using this index, the following values are obtained:

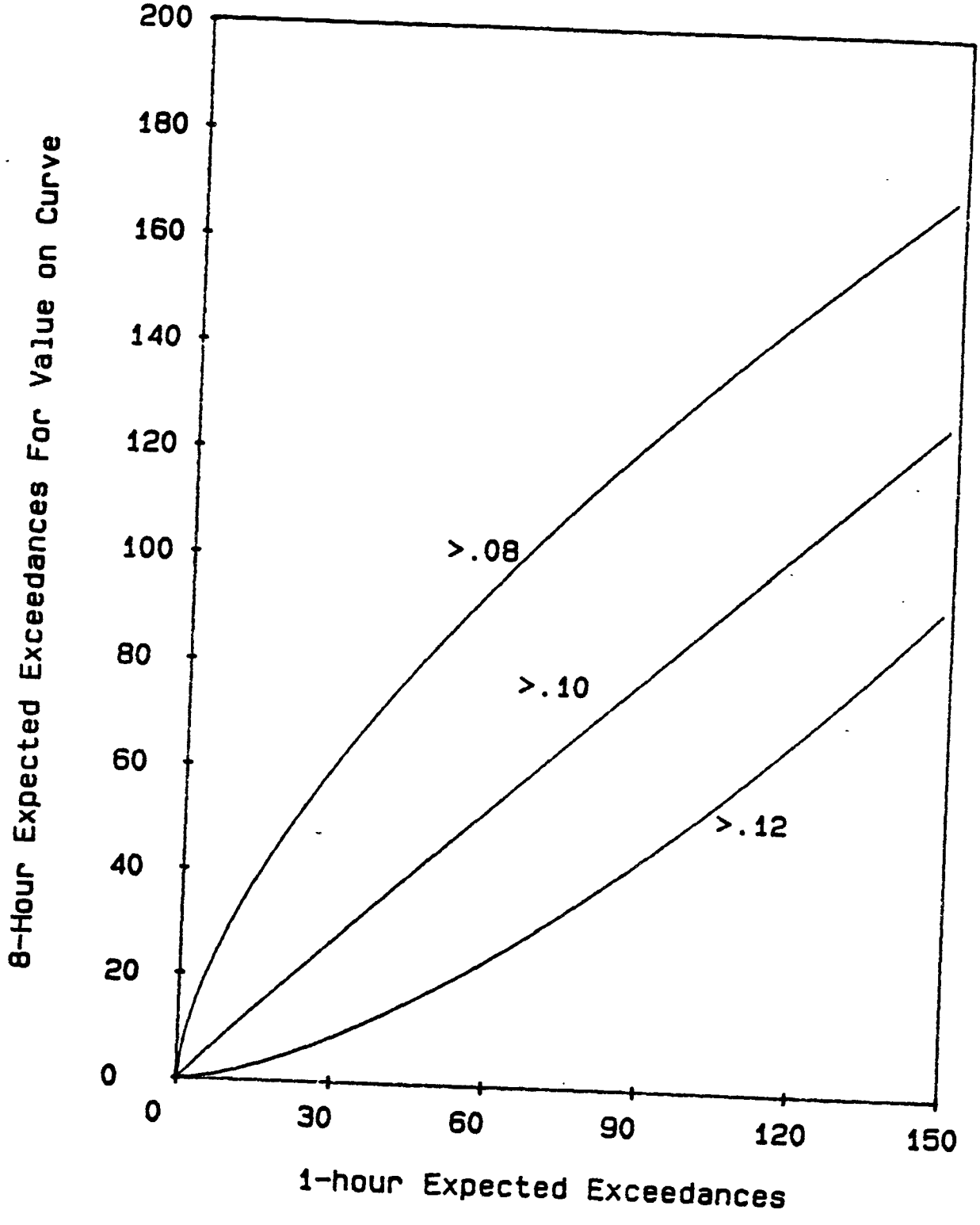
R(.06): 66% R(.08): 83% R(.10): 96% R(.12): 98%

Another measure of the fitting ability of the equations is to investigate the 95% confidence intervals for the two unknown parameters, a and b. The predicted intervals were compared to the estimated a and b values to give the following relative confidence intervals for a and b, respectively:

R(.06): ±19%, ±51% R(.08): ±16%, ±7% R(.10): ±13%, ±3%
R(.12): ± 21%, ±3%

Figure A-6

GENERALIZED RELATIONSHIPS OF THE CURRENT OZONE NAAQS AND THREE ALTERNATIVE 8-HOUR AVERAGES



What this means is that you can have many exceedances of alternative 8-hour daily maximum averages without equaling or exceeding the current NAAQS. Attaining the 1-hour NAAQS is no guarantee that alternative 8-hour daily maximums in the range of 0.06 to 0.08 ppm will be attained. This conclusion is reinforced by a detailed analysis of specific days in 21 MSAs having 1-hour and 8-hour exceedances of the cutpoints mentioned above (McCurdy and Atherton 1988). Results of this analysis appear in Table A-4. If a relative frequency viewpoint of probability is taken, the results indicate that if a day exceeds the current NAAQS concentration level of 0.12 ppm then it is very likely (> 96%) to exceed a 0.06 or 0.08 ppm 8-hour daily maximum (DM) cutpoint. It is less likely (059%) to exceed a 0.10 ppm 8-hour DM cutpoint. On the other hand, a day can experience an exceedance of various 8-hour DM cutpoints without exceeding the 0.12 ppm 1-hour DM level. Only 26% of days exceeding an 8-hour DM cutpoint of 0.06 ppm also exceed a 1-hour 0.12 ppm concentration. This increases to 51% for a 0.08 ppm cutpoint and to 91% for a 0.10 ppm cutpoint. Thus, it is very possible to have days exceeding the lower 8-hour cutpoints investigated without exceeding the current O_3 standard level of 0.12 ppm.

6. Consecutive Daily Exceedances of the Current 1-Hour Standard Level

A number of CASAC members were interested in knowing how likely it is to have two or more days in a row with a violation of the current 1-hour O_3 standard level of 0.12 ppm. An analysis of this multiple-day phenomenon was undertaken for most of the MSAs in our sample of 224. See Table A-5. The total number of days in the sample with a 1-hour DM >.12 ppm is 1,002. About 58% of these "violations" are single-day episodes. Almost 17% are two-day episodes, while about 12% are three-day episodes. Four-day episodes constitute about 7% of violation-days, and greater-than-four-day episodes are 6% or so of violation-days. Thus almost 75% of days having a 1-hour DM >.12 ppm occur singly or in pairs.

Perhaps a brief explanation of the data appearing in Table A-5 is in order. The table provides information on both episode length (the left-hand column) and how often that episode occurs in an individual MSA (the second column). In addition, it provides data on how many MSAs had a particular episode-length/frequency combination. For instance, look at the 2-day episode length portion of the table. The first row indicates that 28 MSAs only had one 2-day episode. The next row indicates that 11 MSAs had two 2-day episodes, and so forth. The fourth column simply is the product of the number of MSAs times the episode-length/frequency combination. (For example, for the first row of 2-day episodes: 2 x 1 x 28 = 56.)

Table A-4

PERCENT OF DAYS EXCEEDING THE CURRENT OZONE NAAQS AND 3 ALTERNATIVE 8-HOUR AVERAGE DAILY MAXIMUM "CUTPOINTS"

	Alternative 8-Hour Average Daily Maximum Cutpoints (in ppm)		
	0.06	0.08	0.10
Percentage of days that are:			
1. >.12 ppm 1-hour DM* & >8-hour cutpoint shown	99.7	96.7	58.6
2. >8-hour cutpoint shown & >.12 ppm 1-hour DM	26.3	51.3	91.0
3. >8-hour cutpoint shown & <.12 ppm 1-hour DM	73.7	48.7	9.0

*DM = Daily Maximum

Table A-5

ESTIMATED FREQUENCY OF DAILY OZONE EPISODES BY LENGTH OF THE EPISODES

Length of Episodes (Days)	Frequency of Episodes in an MSA	Number of Occurrences of That Frequency	Total Episode-Days	Percent of Total Episode Days
1	-	579	579	57.5
2				16.6
	1	28	56	
	2	11	44	
	3	3	18	
	4	2	16	
	7	1	14	
	9	1	18	
3				12.3
	1	13	39	
	2	5	30	
	3	1	9	
	4	1	12	
	5	1	15	
	6	1	18	
4				7.2
	1	7	28	
	3	1	12	
	4	2	32	
>4	(All)	8	62	6.2
TOTAL	-	-	1002	100.0

One final item with respect to Table A-5: the >4 category lumps all episode frequencies. Looking at >4 episodes in more detail for all years of data indicates that only a few areas of the country have episodes of that length. Those areas are the South Coast Air Basin of California and the greater New York City region. The South Coast recently has had episode lengths of up to 30 days, and lengths of 5-8 days are relatively frequent. The maximum episode length in the New York area is 8-10 days; that area has only a few episodes per year >4 days in length. The vast majority of MSAs do not have >4-day episodes.

C. Ambient Ozone Air Quality in Non-Urban Areas

The non-urban areas data base includes monitoring information for 86 "rural" or "remote" sites located in 35 different states. The data come from the 1982-1985 time period, with almost 80% of it coming from 1984 (35%) and 1985 (43%).

With two exceptions, the sites used are those listed in Johnson et al. (1986). In that study, 88 monitoring sites with valid ambient air quality data were identified as having one of the following SAROAD land use codes:

32 - rural agricultural

41 - strictly remote

45 - other remote

These codes are thought to adequately represent rural areas (code 32) and forested areas (codes 41 and 45).

It is important to realize that the majority of sites chosen are located within a U.S. Census Bureau-defined Metropolitan Statistical Area (MSA). This fact does not contravene the land use coding classification. Rural areas are found within an MSA, as are forested areas; truly remote areas, however, are not, except in unusual circumstances. The sites used for the non-urban analyses reported in this Section are described in McCurdy (1987). See that report for additional information on sites included, year of analysis, site coverage by State, and month of highest 1-hour and 8-hour averages.

The air quality indicators to be discussed for non-urban areas are those used previously. Our discussion begins with short-term peak indicators.

1. 1-Hour ExEx Indicator

A one expected exceedance indicator was calculated for each nonurban monitoring site. For the 86 sites:

- 70% attained the 0.12 ppm O_3 standard in the years analyzed.
- 76% had fewer than 1.5 expected exceedances of a 0.12 ppm daily maximum one-hour value.
- the largest number of expected exceedances at a site was 27.3 days.[9]

Cumulative frequency distribution statistics for the one expected exceedances air quality indicator is depicted in Table A-6. (The other indicators shown in the Table will be discussed below.) As can be seen, there is a lot of variability in the data.

The non-urban distribution is very different than the urban distribution (compare Tables A-1 and A-6). There are many fewer expected exceedances of the current O_3 standard in rural/remote areas than in urban areas. This is to be expected, given the urban origin of precursor pollutants that ultimately result in high ambient O_3 concentrations.

2. Second-High Daily Maximum

Cumulative frequency distribution data for the 2nd-high daily max indicator in rural/remote areas is presented in Table A-6. There is not much variability in the data, especially when compared to that seen for the same indicator in urban areas. While non-urban areas have similar, but somewhat lower, mean 2nd-high "design values," there is a large difference in this air quality indicator for the worst areas. The maximum 2nd-high indicator seen in rural/remote areas is 0.16 ppm. It is 0.37 for urban areas.

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[9]This site is Kern County, CA and it is very different than most of the other rural/remote sites. It is the "outlier," so to speak, for many of the air quality indicators investigated. However, the County is definitely an agricultural area. Johnson et al. (1986) indicates that it has the second-highest amount of crop acreage and harvested acreage of the 84 counties in the sample (two counties have two sites). Thus, the site belongs in the sample and it indicates how much variability in air quality is possible at rural sites.

Table A-6

DESCRIPTIVE CUMULATIVE FREQUENCY STATISTICS ASSOCIATED WITH PEAK OZONE AIR QUALITY INDICATORS

Statistic	Expected Exceedances of 0.12 ppm 1-Hour Daily Maximum	2nd-High 1-Hour Daily Max. (ppm)	Expected Number of Daily Maxs > .08 ppm
Mean	1.3	.11	21.1
Std. Dev.	3.4	.02	18.1
Minimum	0.0	.06	0
Median	0.0	.11	17
75th Percentile	1.4	.12	29
90th Percentile	3.5	.14	48
95th Percentile	6.8	.15	58
Maximum	27.3	.16	132
Sample Size	86	86	85*

Note: Std. Dev. = Standard Deviation

*The sample size changes because one site has a missing value for the indicator.

Source: McCurdy (1987)

3. 8-Hour Expected Exceedances

Cumulative frequency data for this indicator are seen in Table A-6. There is relatively a lot of variability in this indicator. Note the particularly large number of expected exceedances for the worst site (Kern County, CA).

The data indicate that it is possible for rural and remote areas to experience many days with a maximum 8-hour average >0.08 ppm. The median area, for instance, has 17 days over that cutpoint concentration.

Although not shown here, the number of 8-hour expected exceedances drop dramatically as the cutpoint concentration increases. For instance, the median non-urban area has 2 days over a 0.10 ppm daily max and no days over a 0.12 ppm cutpoint (McCurdy, 1987). The mean values for these two indicators is 5.7 and 1.4, respectively.

4. Longer-term Air Quality Indicators

Data for the max monthly and 3-month mean indicators appear in Table A-7. The 1-hour monthly mean is significantly higher than the 8-hour 3-month mean for any percentile level chosen. The two distributions are statistically significantly different at $p<.05$, using a paired t-test.

5. Discussion of the 3-Month Mean and Other Multiple-Hour Indicators

The 3-month indicator used here is different from that often used to characterize O_3 exposure to agricultural crops. Most of these long-term studies, such as those conducted by the National Crop Loss Assessment Network (NCLAN), use a "growing season" fixed 7-hour (9am - 4pm) daily average exposure statistic.

This would be a difficult standard formulation to implement because of variability in growing season across the country. While a three month period seems biologically relevant because it approximates the average growing season for most crops in the U.S., there is considerable uncertainty as to whether the fixed daily daylight period captures the time interval of greatest plant sensitivity. It certainly does not capture the time period of maximum O_3 in a day for many rural and remote sites (McCurdy, 1984). This is depicted in Figure A-7, which compares cumulative frequency distributions of the 8-hour daily maximum, 7-hour fixed time period, and 1-hour daily maximum peak air quality

Table A-7

DESCRIPTIVE CUMULATIVE FREQUENCY STATISTICS ASSOCIATED WITH LONGER-TERM AIR QUALITY INDICATORS (in ppm)

Statistic	Maximum Monthly Mean for 1-hr Daily Max. Ave.	Maximum 3-Month Mean for 8-hr Daily Max. Ave.
Mean	.066	.054
Std. Dev.	.012	.009
Minimum	.048	.029
Median	.067	.054
75th Percentile	.073	.059
90th Percentile	.079	.063
95th Percentile	.086	.067
Maximum	.118	.086
Sample Size	86	85*

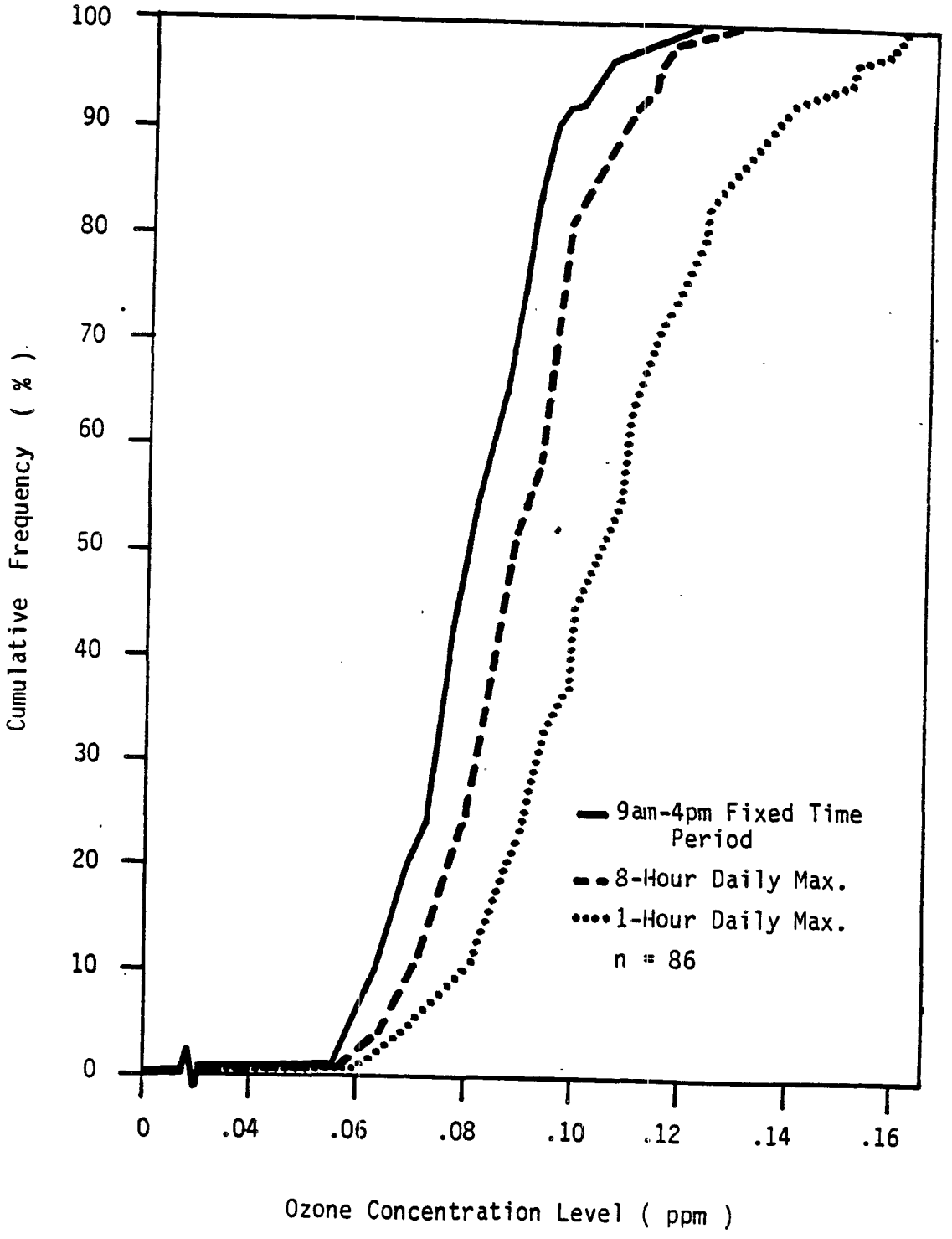
Notes: Std. Dev. = Standard Deviation

*The sample size changes because one site has a missing value for the indicator.

Source: McCurdy (1987)

Figure A-7

CUMULATIVE FREQUENCY DISTRIBUTION OF THREE PEAK AIR QUALITY INDICATORS



indicators. A paired difference-in means test among all possible pairs of the three peak air quality indicators denotes that their means are significantly different (at $p<0.05$). The indicators also are highly correlated (r's >0.80).

There is also a large difference in long-term means obtained from the two different averaging times. For example, the highest monthly average of 7-hour fixed "daily daylight" indicators ranges from 0.023 to 0.071 ppm, with a mean of 0.053 ppm. These values respectively are 109%, 66%, and 25% lower than corresponding values for maximum monthly 8-hour daily max indicators (McCurdy 1987).

The same pattern holds for 3-month means. Third-quarter 7-hour fixed averages data for non-MSA areas are analyzed in McCurdy (1985). The mean for the 59 sites investigated was 0.045 ppm (standard deviation = 0.010 ppm), with a sample range of 0.021-0.061 ppm. Compare these figures with those appearing in Table A-7. The 3-month mean data are considerably higher than the 7-hour mean data, except for the standard deviation value. Undoubtedly the differences are statistically different, although an explicit test of this difference could not be undertaken. Since the fixed time period does not capture the maximum 7-hour (or 8-hour) O_3 exposure for many days and probably does not include all periods of plant biological activity, EPA thinks that a more-encompassing long-term air quality indicator is needed than the one used by NCLAN and others. The maximum 3-month mean of the 8-hour daily maximums is one such indicator. There may be others, and an on-going analysis of this and other indicators may uncover a better exposure statistic from a plant dose-response perspective (Lee et al., 1988).

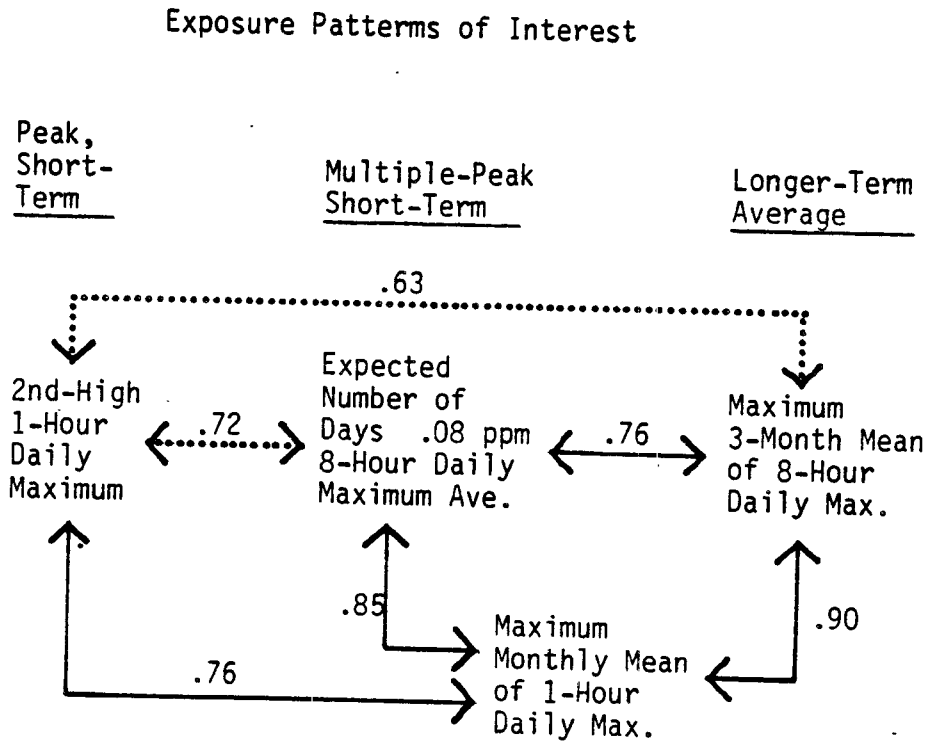
6. Relationships Among Air Quality Indicators in Non-Urban Areas

Correlations among the four indicators of interest are shown in Figure A-8. All coefficients are lower than their corresponding coefficients in urban areas (see Figures A-1 and A-2).

Relationships between pairs of air quality indicators in non-urban areas are shown in Figures A-9 through A-13. These figures follow the approach and format used in Figures A-3 through A-5 for urban areas. Figure A-9 indicates that attaining the current standard in non-urban areas does not guarantee that 8-hour daily maximums will not be experienced. In fact, in 10% of these "attaining areas," as many as 17 days can occur with an 8-hour daily maximum > 0.08 ppm. This number drops to 8 days for a 0.10 ppm 1-hour daily maximum standard.

Figure A-8

CORRELATIONS AMONG SHORT-TERM, MULTIPLE-PEAK, AND LONGER-TERM AIR QUALITY INDICATORS IN NON-URBAN AREAS

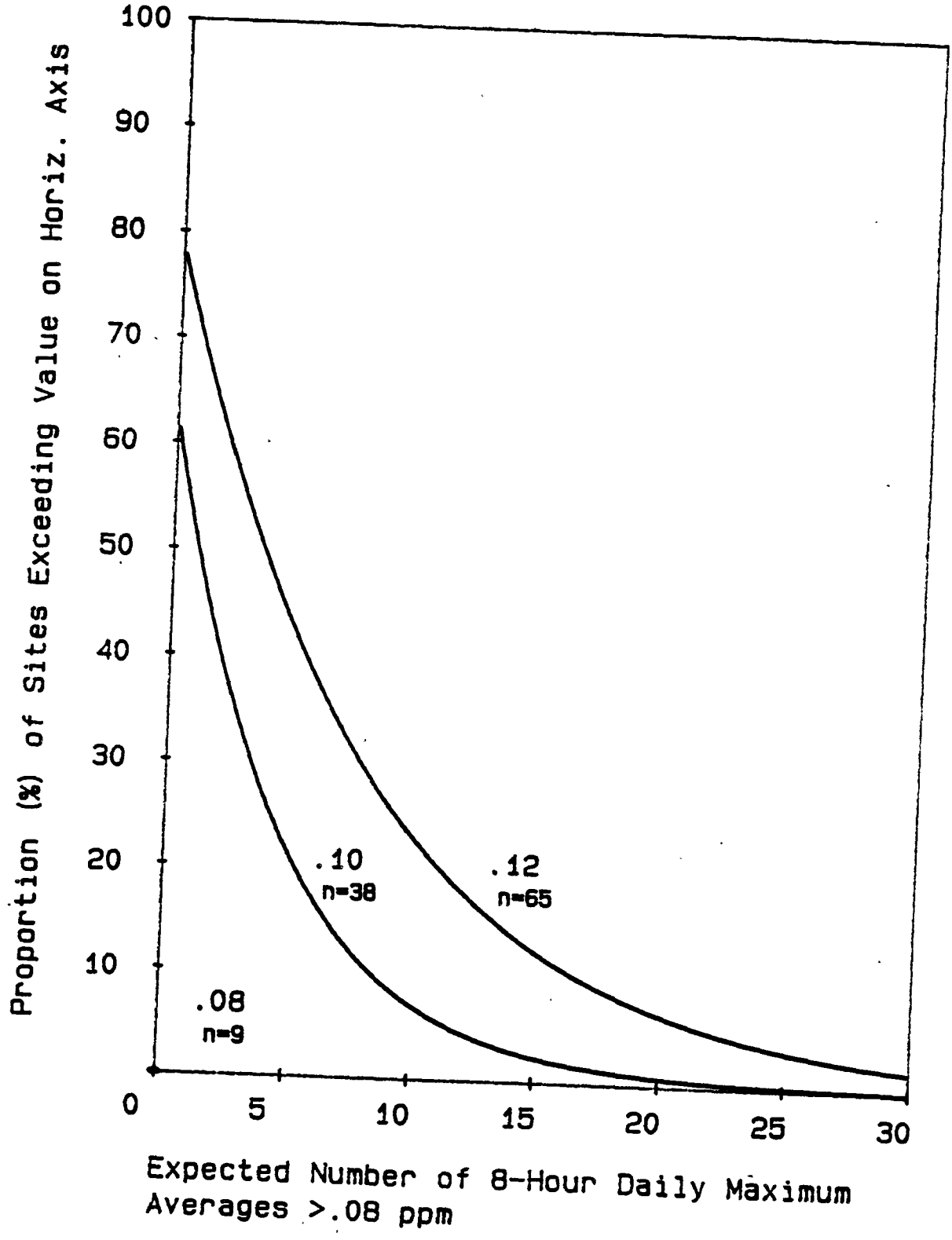


Source: McCurdy (1987a).

The sample size for all indicators is 86. A dotted line is used when the correlation coefficient between the two variables is less than |0.75.| All coefficients are significant at $p<.05$.

Figure A-9

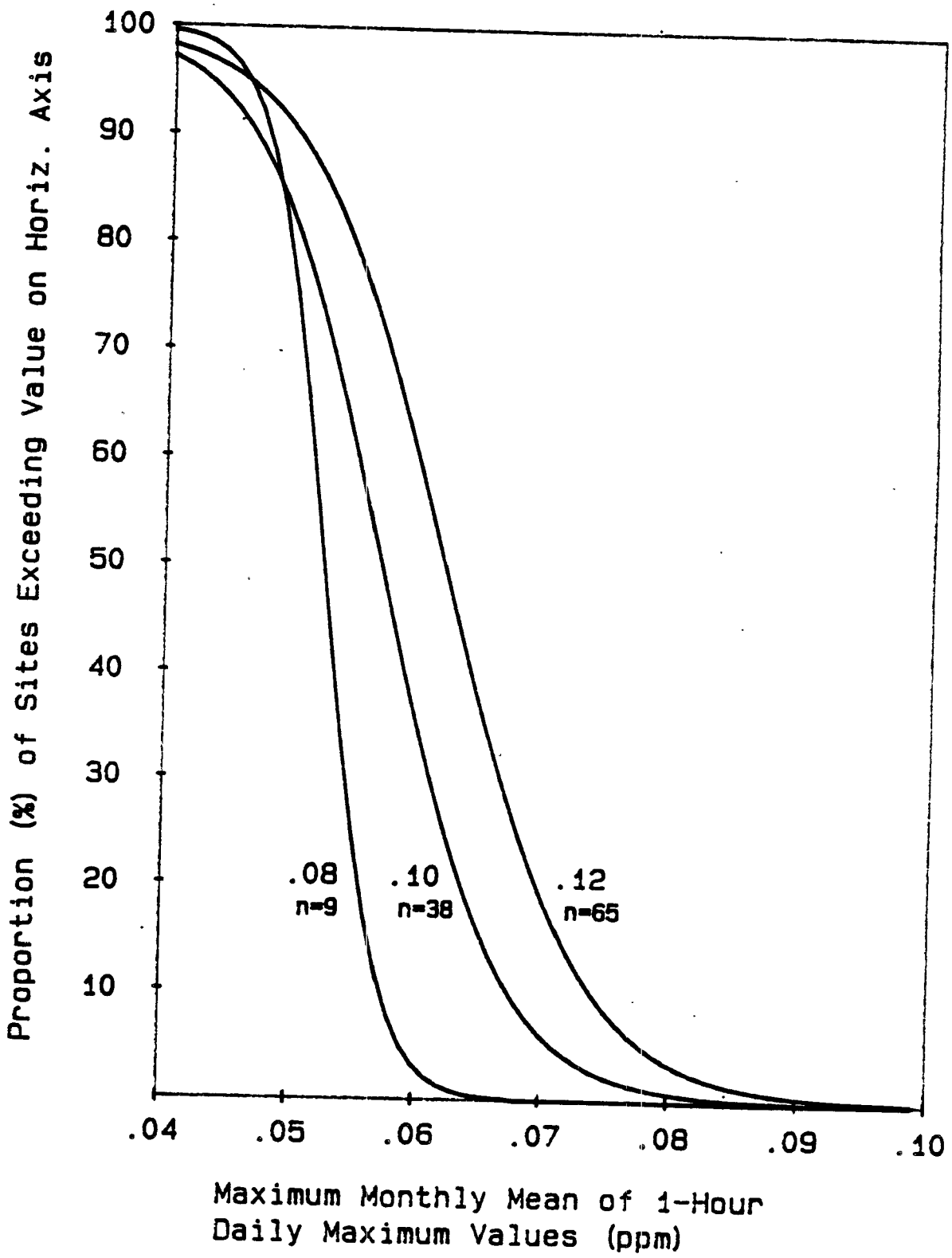
PROPORTION (IN PERCENT) OF RURAL/REMOTE SITES EXCEEDING SPECIFIED EXPECTED NUMBER OF 8-HOUR DAILY MAXIMUM AVERAGES >.08 ppm FOR THREE 1-HOUR DAILY MAXIMUM STANDARDS*



*Attained at all sites in the data set.

Figure A-10

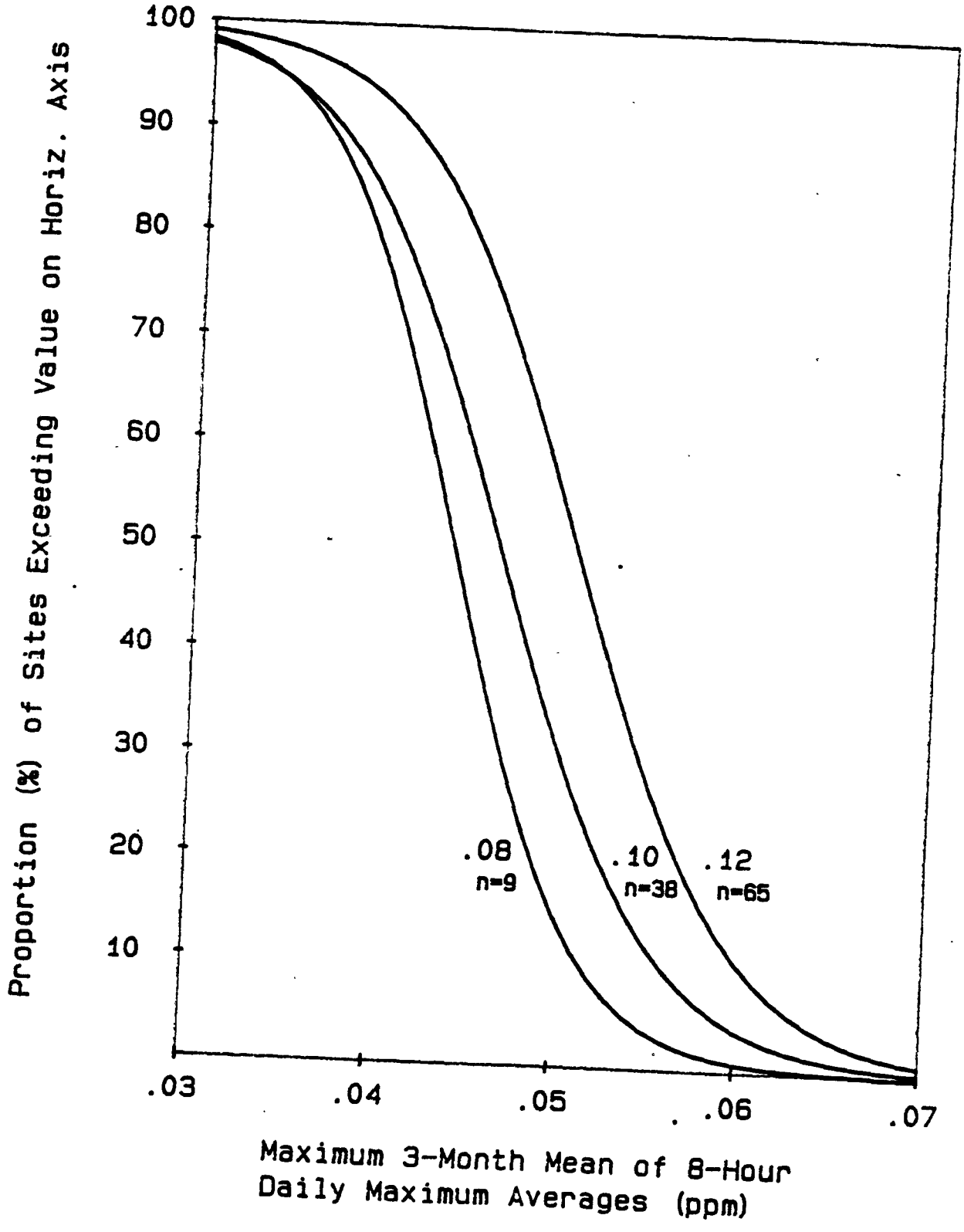
PROPORTION (IN PERCENT) OF RURAL/REMOTE SITES EXCEEDING SPECIFIED MAXIMUM MONTHLY 1-HOUR DAILY MAXIMUM VALUES FOR THREE 1-HOUR DAILY MAXIMUM NAAQS*



*Attained at all sites in the data set.

Figure A-11

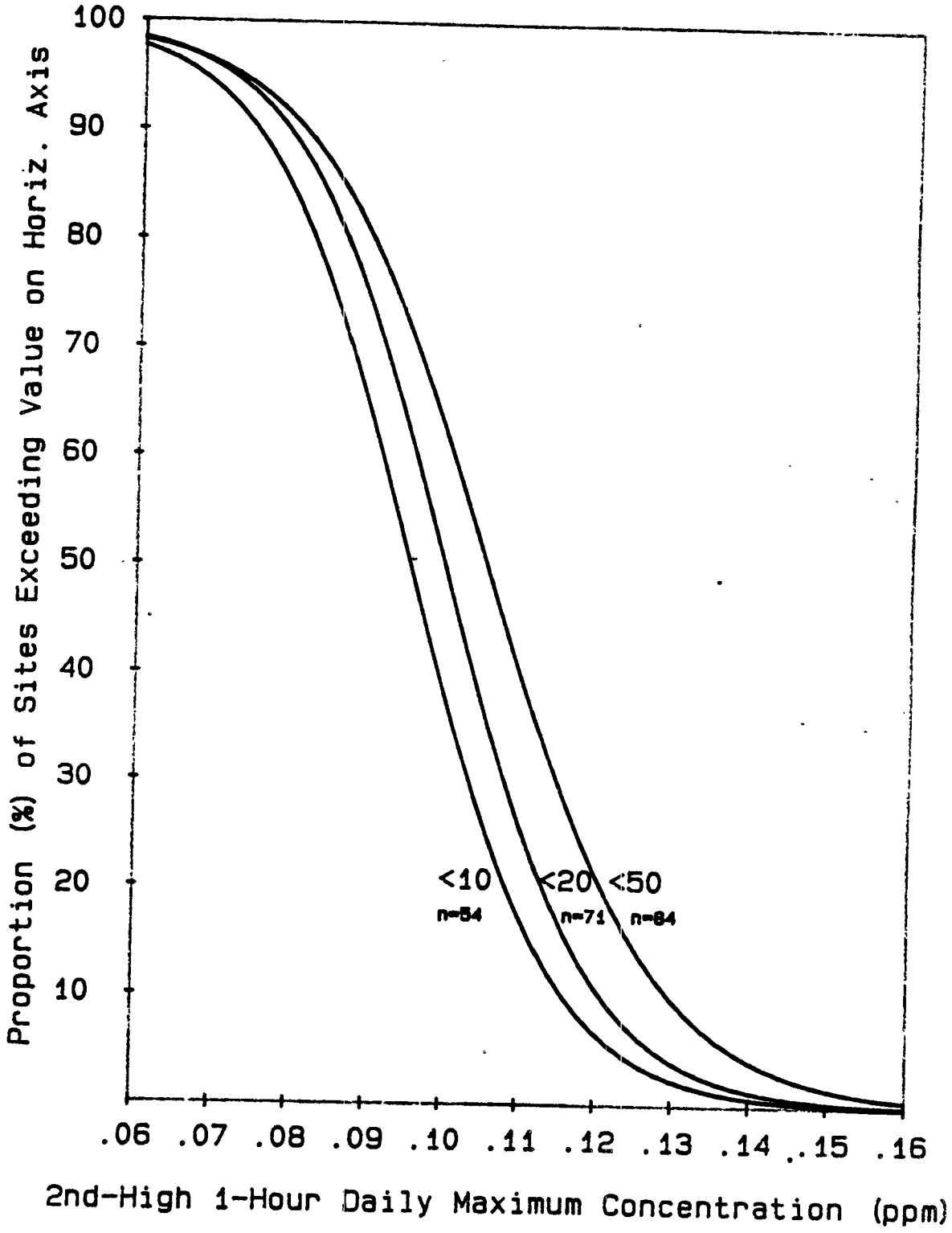
PROPORTION (IN PERCENT) OF RURAL/REMOTE SITES EXCEEDING SPECIFIED THREE-MONTH 8-HOUR DAILY MAXIMUM AVERAGES FOR THREE 1-HOUR DAILY MAXIMUM NAAQS*



*Attained at all sites in the data set.

Figure A-12

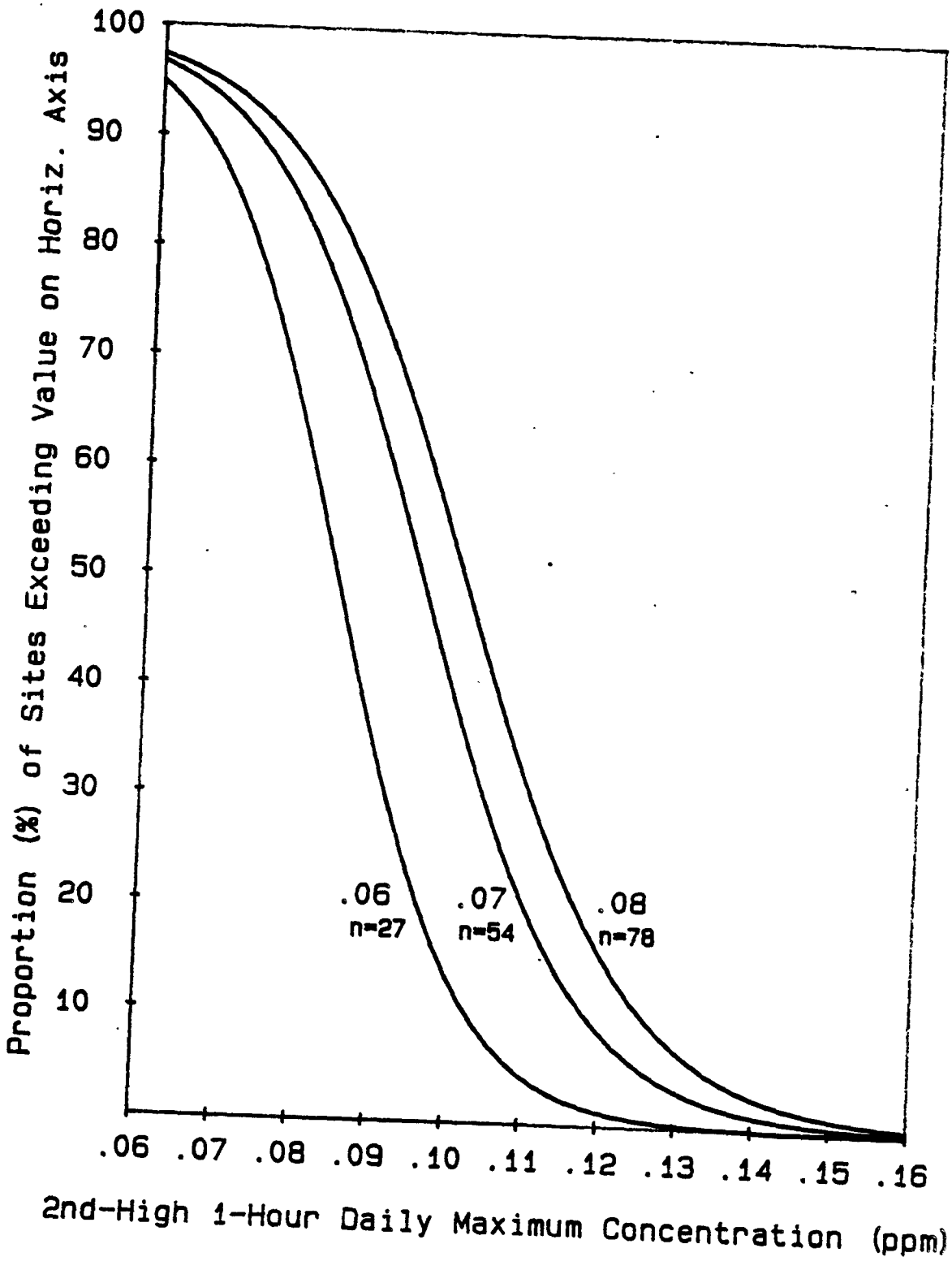
PROPORTION (IN PERCENT) OF RURAL/REMOTE SITES EXCEEDING SPECIFIED SECOND HIGH 1-HOUR DAILY MAXIMUM VALUES FOR THREE EXPECTED NUMBER OF 8-HOUR DAILY MAXIMUM AVERAGES >.08ppm STANDARDS *



*Attained at all sites in the data set.

Figure A-13

PROPORTION (IN PERCENT) OF RURAL/REMOTE SITES EXCEEDING SPECIFIED NUMBER OF SECOND HIGH 1-HOUR DAILY MAXIMUM VALUES FOR THREE MAX MONTHLY MEAN 1-HOUR DAILY MAXIMUM STANDARDS*



*Attained at all sites in the data set.

The curves shown in Figure A-9 are almost identical to those shown in Figure A-3. This may indicate that air quality distributions are practically the same for urban and non-urban areas if the characteristic highest concentrations[10] are similar.

The proportion of non-urban sites exceeding various max monthly means given attainment of alternative 1-hour standards is depicted in Figure A-10. It indicates that 10% of non-urban sites attaining the 0.12 ppm O_3 standard could have a max monthly mean of 0.073 ppm or higher. If a 0.10 ppm 1-hour standard were attained, the mean for the highest 10% of non-urban sites may be $\geq$0.068 ppm.

Figure A-11 depicts the relationship between attaining alternative 1-hour daily max standards and the maximum 3-month 8-hour daily max indicator in non-urban areas. Attaining a 0.12 ppm 1-hour O_3 standard might keep all but 10% of non-urban areas under a 0.06 ppm 3-month mean. The worst 10% 3-month mean drops to 0.056 ppm for a 0.10 ppm standard and to 0.052 ppm for a 0.08 ppm standard.

Figure A-12 indicates what may happen to short-term peaks when exceedances of a 8-hour daily maximum average are regulated. Attempting to reduce short-term peaks by reducing the number of days with an 8-hour daily maximum average >0.08 ppm is inefficient. It takes a large reduction in expected exceedances of a 0.08 ppm daily max to effectuate a small reduction in the 1-hour design value.

A slightly more efficient relationship is shown in Figure A-13. This Figure relates alternative max monthly mean standards to 1-hour design values. Going from a 0.08 ppm to a 0.07 ppm max monthly standard may reduce the design value from 0.125 to 0.118 ppm in the worst 10% of non-urban sites. A drop to a 0.06 ppm max monthly standard might result in a very large reduction in 1-hour daily maximum CHC. For the worst 10% of non-urban sites, the CHC could be as low as 0.102 ppm.

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[10]CHC; in this case, the 2nd-highest 1-hour daily maximum design value.

D. Summary

This Appendix presented descriptive O_3 data for five air quality indicators associated with peak, multiple-peak, and longer-term averaging times. Analyses were also performed on relationships between selected pairs of the 5 air quality indicators.

The data and relational information were presented separately for metropolitan statistical areas and for non-urban areas. The sample size used for most of the analyses was 224 and 86 for urban and non-urban areas, respectively.

It was shown that there are large differences between urban and nonurban sites in their frequency distribution for some air quality indicators, especially at the upper tail. In addition, while the relational curves are similarly shaped for the two types of areas, they are not equivalent numerically.

Finally, it was suggested that a maximum monthly mean of one-hour daily maximum O_3 concentrations could function as a surrogate air quality indicator of both short- and long-term exposure averaging times. Using this, or any other, surrogate is at best a compromise, however, as compared to the more direct and rigorous approach of using multiple averaging time standards to protect against adverse effects specific to these averaging times.

APPENDIX B: GLOSSARY OF PULMONARY TERMS AND SYMBOLS*+

Acetylcholine (ACh): A naturally occurring substance in the body having important parasympathetic effects; often used as a bronchoconstrictor.

Aerosol: Solid particles or liquid droplets that are dispersed or suspended in a gas, ranging in size from 10^{-4} to 10^{-2} micrometers (μm).

Air spaces: All alveolar ducts, alveolar sacs, and alveoli. To be contrasted with AIRWAYS.

Airway conductance (Gaw): Reciprocal of airway resistance. Gas = (1/Raw).

Airway resistance (Raw): The (frictional) resistance to airflow afforded by the airways between the airway opening at the mouth and the alveoli.

Airways: All passageways of the respiratory tract from mouth or nares down to and including respiratory bronchioles. To be contrasted with AIR SPACES.

Allergen: A material that, as a result of coming into contact with appropriate tissues of an animal body, induces a state of allergy or hypersensitivity; generally associated with idiosyncratic hypersensitivities.

Alveolar-capillary membrane: A fine membrane (0.2 to 0.4 μm) separating alveolus from capillary; composed of epithelial cells lining the alveolus, a thin layer of connective tissue, and a layer of capillary endothelial cells.

Alveolus: Hexagonal or spherical air cells of the lungs. The majority of alveoli arise from the alveolar ducts which are lined with the alveoli. An alveolus is an ultimate respiratory unit where the gas exchange takes place.

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*References: Bartels, H.; Dejours, P.; Kellogg, R.H.; Mead, J. (1973) Glossary on respiration and gas exchange. J. Appl. Physiol. 34: 549-558.

American College of Chest Physicians - American Thoracic Society (1975) Pulmonary terms and symbols: a report of the ACCP-ATS Joint Committee on pulmonary nomenclature. Chest 67: 583-593.

+Adapted from Appendix A, Volume V of the CD.

Asthma: A disease characterized by an increased responsiveness of the airways to various stimuli and manifested by slowing of forced expiration which changes in severity either spontaneously or as a result of therapy. The term asthma may be modified by words or phrases indicating its etiology, factors provoking attacks, or its duration.

Breathing pattern: A general term designating the characteristics of the ventilatory activity, e.g., tidal volume, frequency of breathing, and shape of the volume time curve.

Bronchiole: One of the finer subdivisions of the airways, less than 1 mm in diameter, and having no cartilage in its wall.

Bronchiolitis: Inflammation of the bronchioles which may be acute or chronic. If the etiology is known, it should be stated. If permanent occlusion of the lumens is present, the term bronchiolitis obliterans may be used.

Bronchitis: A non-neoplastic disorder of structure or function of the bronchi resulting from infectious or noninfectious irritation. The term bronchitis should be modified by appropriate words or phrases to indicate its etiology, its chronicity, the presence of associated airways dysfunction, or type of anatomic change. The term chronic bronchitis, when unqualified, refers to a condition associated with prolonged exposure to nonspecific bronchial irritants and accompanied by mucous hypersecretion and certain structural alterations in the bronchi. Anatomic changes may include hypertrophy of the mucous-secreting apparatus and epithelial metaplasia, as well as more classic evidences of inflammation. In epidemiologic studies, the presence of cough or sputum production on most days for at least three months of the year has sometimes been accepted as a criterion for the diagnosis.

Bronchoconstrictor: An agent that causes a reduction in the caliber (diameter) of airways.

Bronchodilator: An agent that causes an increase in the caliber (diameter) of airways.

Bronchus: One of the subdivisions of the trachea serving to convey air to and from the lungs. The trachea divides into right and left main bronchi which in turn form lobar, segmental, and subsegmental bronchi.

Carbon monoxide (CO): An odorless, colorless, toxic gas formed by incomplete combustion, with a strong affinity for hemoglobin and cytochrome; it reduces oxygen absorption capacity, transport, and utilization.

Carboxyhemoglobin (COHb): Hemoglobin in which the iron is associated with carbon monoxide. The affinity of hemoglobin for CO is about 300 times greater than for O_2.

Chronic obstructive lung disease (COLD): This term refers to diseases of uncertain etiology characterized by persistent slowing of airflow during forced expiration. It is recommended that a more specific term, such as chronic obstructive bronchitis or chronic obstructive emphysema, be used whenever possible. Synonymous with chronic obstructive pulmonary disease (COPD).

Closing capacity (CC): Closing volume plus residual volume, often expressed as a ratio of TLC, i.e. (CC/TLC%).

Closing volume (CV): The volume exhaled after the expired gas concentration is inflected from an alveolar plateau during a controlled breathing maneuver. Since the value obtained is dependent on the specific test technique, the method used must be designated in the text, and when necessary, specified by a qualifying symbol. Closing volume is often expressed as a ratio of the VC, i.e. (CV/VC%).

Conductance (G): The reciprocal of RESISTANCE. See AIRWAY CONDUCTANCE.

FEV_t/FVC: A ratio of timed (t = 0.5, 1, 2, 3 s) forced expiratory volume (FEV_t) to forced vital capacity (FVC). The ratio is often expressed in percent 100 x FEV_t/FVC. It is an index of airway obstruction.

Forced expiratory flow (FEFx): Related to some portion of the FVC curve. Modifiers refer to the amount of the FVC already exhaled when the measurement is made. For example:

$FEF_{75\%}$ = instantaneous forced expiratory flow after 75% of the FVC has been exhaled.

$FEF_{200-1200}$ = mean forced expiratory flow between 200 ml and 1200 ml of the FVC (formerly called the maximum expiratory flow rate (MEFR).

$FEF_{25-75\%}$ = mean forced expiratory flow during the middle half of the FVC [formerly called the maximum mid-expiratory flow rate (MMFR)].

FEF_{max} = the maximal forced expiratory flow achieved during an FVC.

Forced expiratory volume (FEV): Denotes the volume of gas which is exhaled in a given time interval during the execution of a forced vital capacity. Conventionally, the times used are 0.5, 0.75, or 1 sec, symbolized $FEV_{0.5}$, $FEV_{0.75}$, $FEV_{1.0}$. These values are often expressed as a percent of the forced vital capacity, e.g. ($FEV_{1.0}$/VC) X 100. Forced inspiratory

Forced vital capacity (FVC): Vital capacity performed with a maximally forced expiratory effort.

Functional residual capacity (FRC): The sum of RV and ERV (the volume of air remaining in the lungs at the end-expiratory position). The method of measurement should be indicated as with RV.

Gas exchange: Movement of oxygen from the alveoli into the pulmonary capillary blood as carbon dioxide enters the alveoli from the blood. In broader terms, the exchange of gases between alveoli and lung capillaries.

Gas trapping: Trapping of gas behind small airways that were opened during inspiration but closed during forceful expiration. It is a volume difference between FVC and VC.

Hemoglobin (Hb): A hemoprotein naturally occurring in most vertebrate blood, consisting of four polypeptide chains (the globulin) to each of which there is attached a heme group. The heme is made of four pyrrole rings and a divalent iron (FE^{2+}-protoporphyrin) which combines reversibly with molecular oxygen.

Histamine: A depressor amine derived from the amino acid histidine and found in all body tissues, with the highest concentration in the lung; a powerful stimulant of gastric secretion, a constrictor of bronchial smooth muscle, and a vasodilator that causes a fall in blood pressure.

Hypoxia: Any state in which the oxygen in the lung, blood, and/or tissues is abnormally low compared with that of normal resting man breathing air at sea level. If the P_{02} is low in the environment, whether because of decreased barometric pressure or decreased fractional concentration of O_2, the condition is termed environmental hypoxia. Hypoxia when referring to the blood is termed hypoxemia. Tissues are said to be hypoxic when the P_{02} is low, even if there is no arterial hypoxemia, as in "stagnant hypoxia" which occurs when the local circulation is low compared to the local metabolism.

Inspiratory capacity (IC): The of IRV and TV.

Lung volume (V_L): Actual volume of the lung, including the volume of the conducting airways.

Maximal aerobic capacity (max V " O_2): The rate of oxygen uptake by the body during repetitive maximal respiratory effort. Synonymous with maximal oxygen consumption.

Maximum expiratory flow rate (MEFR): Synonymous with $FEF_{200-1200}$.

Maximum mid-expiratory flow rate (MMFR or MMEF): Synonymous with $FEF_{25-75\%}$.

Maximum ventilation (max V_E): The volume of air breathed in one minute during repetitive maximal respiratory effort. Synonymous with maximum ventilatory minute volume.

Minute ventilation (V_E): Volume of air breathed in one minute. It is a product of tidal volume (V_T) and breathing frequency (f_B). See VENTILATION.

Mucociliary transport: The process by which mucus is transported, by ciliary action, from the lungs.

Mucus: The clear, viscid secretion of mucous membranes, consisting of mucin, epithelial cells, leukocytes, and various inorganic salts suspended in water.

Nasopharyngeal: Relating to the nose or the nasal cavity and the pharynx (throat).

Nitrogen oxides: Compounds of N and O in ambient air; i.e., nitric oxide (NO) and others with a higher oxidation state of N, of which NO_2 is the most important toxicologically.

Oxidant: A chemical compound that has the ability to remove, accept, or share electrons from another chemical species, thereby oxidizing it.

Particulates: Fine solid particles such as dust, smoke, fumes, or smog, found in the air or in emissions.

Pathogen: Any virus, microorganism, or etiologic agent causing disease.

Peak expiratory flow (PEF): The highest forced expiratory flow measured with a peak flow meter.

Peroxyacetyl nitrate (PAN): Pollutant created by action of UV component of sunlight on hydrocarbons and NO_x in the air; an ingredient of photochemical smog.

Pulmonary edema: An accumulation of excessive amounts of fluid in the lung extravascular tissue and air spaces.

Pulmonary emphysema: An abnormal, permanent enlargement of the air spaces distal to the terminal nonrespiratory bronchiole, accompanied by destructive changes of the alveolar walls and without obvious fibrosis. The term emphysema may be modified by words or phrases to indicate its etiology, its anatomic subtype, or any associated airways dysfunction.

Residual volume (RV): That volume of air remaining in the lungs after maximal exhalation. The method of measurement should be indicated in the text or, when necessary, by appropriate qualifying symbols.

Resistance flow (R): The ratio of the flow-resistive components of pressure to simultaneous flow, in cm H_2O/liter per sec. Flow-resistive components of pressure are obtained by subtracting any elastic or inertial components, proportional respectively to volume and volume acceleration. Most flow resistances in the respiratory system are nonlinear, varying with the magnitude and direction of flow, with lung volume and lung volume history, and possibly with volume acceleration. Accordingly, careful specification of the conditions of measurement is necessary; see AIRWAY RESISTANCE, TISSUE RESISTANCE, TOTAL PULMONARY RESISTANCE, COLLATERAL RESISTANCE.

Respiratory frequency (f_R): The number of breathing cycles per unit of time. Synonymous with breathing frequency (f_B).

Specific airway conductance (SGaw): Airway conductance divided by the lung volume at which it was measured. SRaw = Raw x TGV.

Sulfur dioxide (SO_2): Colorless gas with pungent odor, released primarily from burning of fossil fuels, such as coal, containing sulfur.

STPD conditions (STPD): Standard temperature and pressure, dry. These are the conditions of a volume of gas at 0°C, at 760 torr, without water vapor. A STPD volume of a given gas contains a known number of moles of that gas.

Synergism; A relationship in which the combined action of effect of two or more components is greater than the sum of effects when the components act separately.

Tidal volume (TV): That volume of air inhaled or exhaled with each breath during quite breathing, used only to indicate a subdivision of lung volume. When tidal volume is used in gas exchange formulations, the symbol V_T should be used.

Total lung capacity (TLC): The sum of all volume compartments or the volume of air in the lungs after maximal inspiration. The method of measurement should be indicated, as with RV.

Total pulmonary resistance (R_L): Resistance measured by relating flow-dependent transpulmonary pressure to airflow at the mouth. Represents the total (frictional) resistance of the lung tissue (R_{ti}) and the airways (Raw). $R_L = R_{aw} + R_{ti}$.

Trachea: Commonly known as the windpipe; a cartilaginous air tube extending from the larynx (voice box) into the thorax (chest) where it divides into left and right branches.

Ventilation: Physiological process by which gas is renewed in the lungs. The word ventilation sometimes designates ventilatory flow rate (or ventilatory minute volume) which is the product of the tidal volume by the ventilatory frequency. Conditions are usually indicated as modifiers; i.e.,

V_E = Expired volume per minute (BTPS),
and
V_E = Inspired volume per minute (BTPS).

Ventilation is often referred to as "total ventilation" to distinguish it from "alveolar ventilation" (see VENTILATION, ALVEOLAR).

Ventilation, alveolar (V_A): Physiological process by which alveolar gas is completely removed and replaced with fresh gas. Alveolar ventilation is less than total ventilation because when a total volume of gas leaves the alveolar spaces, the last part does not get expelled from the body but occupies the dead space, to be reinspired with the next inspiration. Thus the volume of alveolar gas actually expelled completely is equal to the tidal volume minus the volume of the dead space. This truly complete expiration volume times the ventilatory frequency constitutes the alveolar ventilation.

Vital capacity (VC): The maximum volume of air exhaled from the point of maximum inspiration.

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

May 1, 1989

OFFICE OF
THE ADMINISTRATOR

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 Clear 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 <u>not</u> 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." The opinion of the CASAC Ozone Review Committee was divided with regard to the upper range of the standard with eight individuals favoring a range with an upper value of 0.12 ppm, three individuals favored an upper bound in the range of 0.10-0.12 ppm, four individuals favored an upper bound value no higher than

0.10 ppm, and one individual abstained from offering an opinion. Several individuals who supported an upper value of 0.12 ppm as well as all of the other individuals who favored a lower value for the upper end of the range expressed the view that at 0.12 ppm there was little or no margin of safety. As you are aware, the margin of safety is intended to provide protection against adverse effects which have not yet been uncovered by research and effects whose medical significance is a matter of disagreement. Finally, several members of the subcommittee favored development of a standard with a more statistically robust upper bound on the annual distribution of ozone concentrations rather than reliance on the current expected exceedance form of the standard. While the Committee offers no further advice on what form the Agency should consider, we would caution you against any form which alters the degree of health protection afforded by the current standard.

CASAC had substantial discussion of the issue of what are or are not adverse health effects. This discussion was aided by the presentation of this issue in the staff position paper. Within CASAC there was diversity of opinion; some members felt that healthy individuals experience adverse effects when ozone exposure induced any of the responses categorized as moderate (i.e., >10% decrement in FEV_1 or mild to moderate respiratory symptoms) in the staff position paper, while a few members believed that adverse effects would not be experienced until ozone induced more severe effects (i.e., >20% decrement in FEV_1 and moderate to severe respiratory symptoms). The view of some individuals on this matter was influenced by recognition that resolution of the adverse health effect issue represents a blending of scientific and policy judgments and, thus, we feel it appropriate to inform you of the range of our views on this matter.

Of particular concern to CASAC is the potential for effects arising from exposures to ozone with daily peak concentrations at or near 0.12 ppm for periods of 6-8 hours and with co-exposure to other pollutants. This concern is due to air quality analyses which have shown that even in areas which do not repeatedly exceed the ozone standard, ozone concentrations can remain close to 0.12 ppm for several hours per day for extended periods of time in summer. There was concern based on recent controlled human exposure, epidemiology and toxicology studies, that such prolonged exposures could result in increased respiratory impairment. Further, for people exposed to these ozone concentrations over a lifetime, the possibility that chronic irreversible effects may result is of concern, although such changes have not been demonstrated.

The Committee noted that the Criteria Document Supplement failed to cite and discuss a group of "ecological" epidemiological studies of the effects of ozone on various measures of human health such as hospitalizations for respiratory illnesses or exacerbation of chronic respiratory problems. Although these studies have obvious limitations in establishing cause and effect relationships, they have

certain strengths which can aid in regulatory decision-making. Studies of this type should be discussed and evaluated in future criteria documents as a complementary source of information.

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

CASAC did not reach a consensus opinion on endorsement of the staff position paper recommendation of "a 1-hour averaging time standard in the range of 0.06-0.12 ppm" for a secondary standard. The CASAC Ozone Welfare Effects Subcommittee that considered this matter reached a divided opinion; two favored a range with an upper value of 0.12 ppm, three favored an upper value of less than 0.12 ppm, and five favored an upper value of 0.10 ppm. The Committee noted that the form of the standard was of critical importance in protecting against ozone effects on vegetation. The Committee was of the opinion that a cumulative seasonal standard would be more appropriate than a 1-hour standard and felt that such a standard could be developed. CASAC favored issuance of a cumulative seasonal standard form assuming its development would not further delay the standard setting process. If this form of standard cannot be developed in time for the current review, the Committee is of the opinion that you should give serious consideration to setting a 1-hour secondary standard with a maximum concentration of 0.10 ppm. The Committee took note of the lack of information on the effects of ozone on forest ecosystems and urged support for research to remedy this deficiency.

In closing, I would like to briefly comment on CASAC's failure to reach a consensus as to the appropriate range for setting the ozone standards. This lack of consensus is reflective of major deficiencies in our knowledge regarding health and welfare effects of long-term exposure (beyond a few hours) to ozone. The data base is very large and adequate for knowledgeable individuals to reach agreement on the effects of acute exposure to ozone in the range appropriate for setting a 1-hour standard. However, there is not an adequate data base on the effects of multiple hour or seasonal exposures to ozone, especially as regards whether such exposures may produce chronic health effects. This is especially troubling since such long-term exposures to ozone occur in many parts of the United States and involve many millions of people and thousands of acres of crop and forest lands. As a result, there continues to be concern for the public health

and welfare threat which may be posed by chronic exposure to ozone. It is critical that the data base on health and welfare effects related to multiple hour, seasonal and lifetime exposures of ozone be increased through an accelerated and expanded research effort. This must be done so that future considerations of ozone standards will derive from a stronger scientific base.

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

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

Sincerely,

Roger O. McClellan

Roger O. McClellan, D.V.M.
Chairman, Clean Air Scientific Advisory Committee

ROM:ewb

United States
Environmental Protection
Agency

Office of the Administrator
Science Advisory Board
Washington, DC 20460

EPA-SAB-CASAC-89-019
May 1989

Report of the Clean Air Scientific Advisory Committee (CASAC)

Review of the NAAQS for Ozone: Closure on the OAQPS Staff Paper (1988) and the Criteria Document Supplement (1988)

ABSTRACT

This is the report of the EPA's Clean Air Scientific Advisory Committee (CASAC) on its review of the Agency's draft documents: "Air Quality Criteria Document Supplement (1988)" and the "Review of the National Ambient Air Quality Standards for Ozone: Assessment of Scientific and Technical Information (1988)". These documents were reviewed in public session on December 14-15, 1988, with the Committee reaching the conclusion that the documents provide an adequate scientific and technical basis for EPA to retain or revise primary and secondary national ambient air quality standards for ozone.

NOTICE

This report has been written as part of the activities of the Science Advisory Board, a public advisory group providing extramural scientific information and advice to the Administrator and other officials of the Environmental Protection Agency. The Board is structured to provide a balanced expert assessment of scientific matters related to problems facing the Agency. This report has not been reviewed for approval by the Agency; and, hence, the contents of this report do not necessarily represent the views and policies of the Environmental Protection Agency or other agencies in the Federal Government. Mention of trade names or commercial products does not constitute a recommendation for use.

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16. ABSTRACT

This paper evaluates and interprets the updated scientific and technical information that EPA staff believes is most relevant to the review of primary (health) national ambient air quality standards for ozone as well as secondary (welfare) standards for ozone. This staff paper provides staff conclusions and recommendations based on the scientific information contained in the 1986 ozone criteria document and its supplement. This assessment is intended to bridge the gap between the scientific review in the 1986 criteria document and supplement and the judgments required of the Administrator in setting ambient air quality standards for ozone.
The major recommendations of the staff paper include the following: (1) Ozone should remain as the surrogate for controlling ambient concentrations of photochemical oxidants; (2) The current form of the ozone standards should be retained (i.e., the standard is attained when expected number of days/year with maximum hourly average concentrations above the level of the standard is equal or greater than one; (3) The 1-hour standards should be retained, although serious consideration should be given to future development of alternative forms of both primary and secondary standards; (4) The range of consideration for primary standard should be 0.08 to 0.12 parts per million (ppm) and for welfare standard the range should be 0.06 to 0.10 ppm.

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