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REVIEW OF NATIONAL AMBIENT AIR QUALITY STANDARDS FOR OZONE
ASSESSMENT OF SCIENTIFIC AND TECHNICAL INFORMATION

OAQPS STAFF PAPER

Office of Air Quality Planning and Standards
U.S. Environmental Protection Agency

DISCLAIMER

This report has been reviewed by the Office of Air Quality Planning and Standards (OAQPS), U. S. Environmental Protection Agency (EPA), and approved for publication. This OAQPS Staff Paper contains the findings and conclusions of the staff of the OAQPS and does not necessarily represent those of the EPA. Mention of trade names or commercial products is not intended to constitute endorsement or recommendation for use.

Acknowledgments

The primary authors of this OAQPS Staff Paper include Dr. David J. McKee (Project Manager, health effects), Ms. Victoria V. Atwell (welfare effects), Mr. Harvey M Richmond (exposure and health risk analyses), Mr. Warren P. Freas (air quality and form of the standard), and Ms. Rosalina M. Rodriguez (economic analyses). This document has been improved by comments and support from Dr. Karen M. Martin, Mr. Eric O. Ginsburg, and Mr. John H. Haines (all of OAQPS), as well as staff of the Office of Research and Development (particularly staff of the National Center for Environmental Assessment and the National Health and Environmental Effects Laboratory-Western Ecology Division), the Office of Policy and Program Evaluation, and the Office of General Counsel, all within the U.S. Environmental Protection Agency (EPA). Of particular importance in the review of this document has been the technical and editorial support provided by Ms. Patricia R. Crabtree and Ms. Barbara Miles.

On three different occasions, draft chapters of this document were formally reviewed by the Clean Air Scientific Advisory Committee (CASAC). Helpful comments and suggestions were also submitted by a number of independent scientists and by environmental and industrial groups.

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I. PURPOSE

The purpose of this Office of Air Quality Planning and Standards (OAQPS) Staff Paper is to evaluate the key scientific information contained in the EPA document, "Air Quality Criteria for Ozone and Related Photochemical Oxidants" (U.S. EPA, 1996a; henceforth referred to as CD), and identify the critical elements that the EPA staff believes should be considered in the review of the national ambient air quality standards (NAAQS) for ozone (O_3). This Staff Paper includes factors relevant to the evaluation of current primary (health) and secondary (welfare) NAAQS, as well as staff conclusions and recommendations regarding the most appropriate alternative primary and secondary NAAQS based on current evaluation of scientific and technical information contained in the CD and this Staff Paper.

II. BACKGROUND

A. Legislative Requirements

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

Section 109 (42 U.S.C. 7409) directs the Administrator to propose and promulgate "primary" and "secondary" NAAQS for pollutants identified under section 108. Section 109(b)(1) defines a primary standard as one "the attainment and maintenance of which, in the judgment of the Administrator, based on the criteria and allowing an adequate margin of safety, [is] requisite to protect the public health."[1] A secondary standard, as defined in section 109(b)(2), must "specify a level of air quality the attainment and maintenance of which, in the judgment of the Administrator, based on [the] criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of [the] pollutant in the ambient air." Welfare effects as defined in section 302(h) [42 U.S.C. 7602(h)] include, but are not limited to, "effects on soils, water, crops, vegetation, manmade materials, animals, wildlife, weather, visibility and climate, damage to and deterioration of property, and hazards to transportation, as well as effects on economic values and on personal comfort and well-being."

The U.S. Court of Appeals for the District of Columbia Circuit has held that the "margin of safety" requirement for primary standards was intended to address uncertainties

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

associated with inconclusive scientific and technical information available at the time of standard setting. It was also intended to provide a reasonable degree of protection against hazards that research has not yet identified. Lead Industries Association v. EPA, 647 F.2d 1130, 1154 (D.C. Cir. 1980), cert. denied, 101 S. Ct. 621 (1980); American Petroleum Institute v. Costle, 665 F.2d 1176, 1177 (D.C. Cir. 1981), cert. denied, 102 S. Ct. 1737 (1982). Both kinds of uncertainties are components of the risk associated with pollution at levels below those at which human health effects can be said to occur with reasonable scientific certainty. Thus, by selecting primary standards that provide an adequate margin of safety, the Administrator is seeking not only to prevent pollution levels that have been demonstrated to be harmful but also to prevent lower pollutant levels that she finds may pose an unacceptable risk of harm, even if the risk is not precisely identified as to nature or degree.

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

Section 109(d)(1) of the Act (enacted in 1977) requires that "not later than December 31, 1980, and at 5-year intervals thereafter, the Administrator shall complete a thorough review of the criteria published under section 108 and the national ambient air quality standards . . . and shall make such revisions in such criteria and standards and promulgate such new standards as may be appropriate . . . ." Section 109(d)(2) requires that an independent scientific review committee be appointed and provides that at corresponding intervals the committee "shall complete a review of the criteria . . . and the national primary and secondary ambient air quality standards . . . and shall recommend to the Administrator any new . . . standards and revisions of existing criteria and standards as may be appropriate

. . . ." Since the early 1980's, this independent review function has been performed by the Clean Air Scientific Advisory Committee (CASAC) of EPA's Science Advisory Board.

B. History of NAAQS Reviews

1. Establishment of NAAQS for Photochemical Oxidants

On April 30, 1971, the EPA promulgated NAAQS for photochemical oxidants under section 109 of the Act (36 FR 8186). Identical primary and secondary NAAQS were set at an hourly average of 0.08 parts per million (ppm) total photochemical oxidants not to be exceeded more than 1 hr per year. Scientific and technical bases for these NAAQS were provided in the document, Air Quality Criteria for Photochemical Oxidants (U.S. DHEW, 1970). The primary standard was based in part on several epidemiology studies (Schoettlin and Landau, 1961; Motley et al., 1959; Rokaw and Massey, 1962) conducted in Los Angeles, which reported a relationship between ambient oxidant levels and aggravation of respiratory disease. The secondary standard was based on evidence of acute and chronic vegetation injury and physiological effects, including growth alterations, reduced yields, and changes in the quality of plant products (U.S. DHEW, 1970, p. 6-18).

2. Review and Revision of NAAQS for Photochemical Oxidants

In 1977, the EPA announced (42 FR 20493) that it was reviewing the 1970 Criteria Document in accordance with section 109(d)(1) of the Act and, in 1978, published a revised Criteria Document (U.S. EPA, 1978). Based on the revised Criteria Document, EPA published proposed revisions to the original NAAQS in 1978 (43 FR 16962) and final revisions in 1979 (44 FR 8202). The primary standard was revised from 0.08 ppm to 0.12 ppm; the secondary standard was set identical to the primary standard; the chemical designation of the standards was changed from photochemical oxidants to O_3; and the form of the standards was revised from a deterministic form to a statistical form, which defined attainment of the standards as occurring when the expected number of days per calendar year with maximum hourly average concentrations greater than 0.12 ppm is equal to or less than one. The revised standards were upheld on judicial appeal. American Petroleum Institute v. Costle, supra.

3. Subsequent Review of Ozone NAAQS

In 1982 (47 FR 11561), the EPA announced plans to revise the 1978 Criteria Document. In 1983, the EPA announced (48 FR 38009) that review of primary and secondary standards for O_3 had been initiated. The EPA subsequently provided a number of opportunities for public review and comment on drafts of the Criteria Document and associated Staff Paper (U.S. EPA, 1989). After reviewing the draft Criteria Document in 1985 and 1986, the CASAC sent to the Administrator a "closure letter" outlining key issues and recommendations indicating that it was satisfied with the final draft of the 1986 Criteria Document (U.S. EPA, 1986).

Following closure, a number of scientific articles and abstracts were published or accepted for publication that appeared to be of sufficient importance concerning potential health and welfare effects of O_3 to warrant preparation of a Supplement to the 1986 Criteria Document (U.S. EPA, 1992). The CASAC, having already reviewed two drafts of the Staff Paper in 1986 and 1987, concluded that sufficient new information existed to recommend incorporation of relevant new information into a third draft of the Staff Paper.

The CASAC held a public meeting in 1988 to review a draft Supplement and the third draft Staff Paper. Major issues included the definition of adverse health effects of O_3; the significance of health studies suggesting that exercising individuals exposed for 6 to 8 hours to O_3 levels at or below 0.12 ppm may experience lung inflammation and transient decreases in pulmonary function; the possibility that chronic irreversible effects may result from long-term exposures to elevated levels of O_3; and the importance of analyses indicating that agricultural crop damage may be better defined by a cumulative seasonal average than by a 1-hr peak level of O_3. In its closure letter of 1989 (58 FR 13018), the CASAC indicated that the draft Supplement and draft Staff Paper "provide an adequate scientific basis for the EPA to retain or revise primary and secondary standards for ozone." With regard to the emerging database on exposures of 6 hours or more, CASAC concluded that such information could better be considered in the next review of the ozone NAAQS.

On October 22, 1991, the American Lung Association (ALA) and other plaintiffs filed suit under section 304 of the Act to compel the EPA to complete its review of the criteria and standards for O_3. The U.S. District Court for the Eastern District of New York

subsequently issued an order requiring the Administrator to sign a Federal Register notice announcing its proposed decision on whether to revise the standards for O_3 by August 1, 1992 and to sign a Federal Register notice announcing EPA's final decision by March 1, 1993.

On August 10, 1992 (57 FR 35542), the EPA published a proposed decision under section 109(d)(1) that revisions to the existing primary and secondary standards were not appropriate at that time. The notice explained (see 57 FR 35546) that the proposed decision would complete the EPA's review of information on health and welfare effects of O_3 assembled over a 7-year period and contained in the 1986 Criteria Document and its Supplement. The notice indicated that the Administrator had not taken into account more recent studies on the health and welfare effects of O_3 because these studies had not been assessed in the 1986 Criteria Document or its Supplement, nor had they collectively undergone the rigorous, integrative review process (including CASAC review) necessary to incorporate them into a new criteria document. Because that process and other necessary steps could not, in EPA's view, be completed in time to meet the March 1993 deadline for a final decision, the proposed decision was based on EPA's evaluation of key information published through early 1989, as contained in the 1986 Criteria Document and its Supplement; the 1989 Staff Paper assessment of the most relevant information in these documents; and the advice and recommendations of the CASAC as presented both in the discussion of these documents at public meetings and in the CASAC's 1986 and 1989 closure letters.

In view of the potential significance of the more recent scientific papers, as well as ongoing research on the health and welfare effects of O_3, the August 10, 1992 notice also announced the EPA's intention to proceed as rapidly as possible with the next review of the air quality criteria and standards for O_3. Shortly thereafter, the EPA's Environmental Criteria and Assessment Office (ECAO) formally initiated action to update the 1986 Criteria Document and its Supplement (57 FR 38832).

On March 9, 1993 (58 FR 13008), the EPA published a final decision concluding that revisions to the current primary and secondary NAAQS for O_3 were not appropriate at that time. Given the potential importance of the new studies and the EPA's continuing concern

about the health and welfare effects of O_3, the March 9, 1993 notice emphasized the Administrator's intention to complete the next review of the NAAQS as rapidly as possible and, if appropriate, to propose revisions of the standards at the earliest possible date. The Administrator subsequently adopted a substantially accelerated schedule for the next review (59 FR 5164).

The ALA sought judicial review of the March 1993 decision under section 307(b) of the Act. Noting that the Administrator intended to reconsider that decision as rapidly as possible in light of the more recent scientific information, EPA sought and was subsequently granted a voluntary remand of ALA's petition for review.

4. Current Review of Ozone NAAQS

As indicated above, ECAO initiated action to update the air quality criteria document for O_3 in August 1992 (57 FR 38832). A series of peer-review workshops was held on draft chapters of the revised Criteria Document in July 1993 (58 FR 35454) and September 1993 (59 FR 48063), and a first external review draft was made available for CASAC and public review on January 31, 1994 (59 FR 4278).

On November 18, 1993, ECAO and OAQPS discussed with CASAC (58 FR 59034) EPA's accelerated schedule for completing the O_3 NAAQS review, formally published on February 3, 1994 (59 FR 5164). In December 1993, OAQPS completed an Ozone NAAQS Development Project Plan, which identified key issues to be addressed in this Staff Paper and the basis for the initial scientific and technical assessments planned to address the issues. OAQPS also met with a subcommittee of the CASAC in December 1993 (58 FR 59034) and March 1994 to discuss methodologies used in the exposure and risk assessments summarized in this Staff Paper.

The CASAC reviewed the first external review draft of the revised Criteria Document (CD) at a public meeting held on July 20-21, 1994 and made recommendations for revisions. At a public meeting held on March 21-22, 1995, the CASAC reviewed a second external review draft of the CD and a first external review draft of the basis for the primary standard contained in this Staff Paper. Following revisions of both the CD and the Staff Paper, an external review draft of the entire Staff Paper and Chapter 5 of the CD were reviewed at a public meeting held on September 19-20, 1995. Following that meeting, letters were

forwarded by the Chairman of CASAC to the Administrator of EPA which came to closure on the draft CD and on the primary portion of the draft Staff Paper. These letters dated November 28, 1995 and November 30, 1995, respectively, are reproduced in Appendix G of this Staff Paper. Finally, at a public meeting held on March 21, 1996, the majority of the CASAC members came to closure on the secondary standard portion of the draft Staff Paper. The closure letter sent from the CASAC Chairman to the EPA Administrator dated April 4, 1996 is reproduced in Appendix G of this Staff Paper.

III. APPROACH

This Staff Paper is based on the scientific evidence in the CD. Quantitative assessments of human exposure and health risks, vegetation exposure, risk, and economic benefits, and air quality comparisons provide additional information considered by the EPA staff in evaluating the appropriateness of revising the current primary and secondary NAAQS and in assessing potential alternative NAAQS.

Critical elements are identified in this Staff Paper which the staff believes should be considered in this review of the O_3 NAAQS. Attention is drawn to judgments that must be based on careful interpretation of incomplete or uncertain evidence. In such instances, the Staff Paper provides the staff's evaluation, sets forth alternatives the staff believes should be considered, and recommends a course of action.

A. Bases for Analytic Assessments

To meet the accelerated schedule established by the Administrator for this review of the ozone NAAQS, the OAQPS Ozone NAAQS Development Project Plan identified several alternative primary and secondary standards to provide a basis for various initial analytic assessments of air quality, human exposure and health risks, and crop yield loss. In so doing, the staff recognized that additional alternatives might need to be analyzed as the review process continues; e.g., as a result of CASAC and public reviews of the CD and Staff Paper drafts.

The Plan identified the following alternative primary standards for use in initial analytic assessments:

- The current 1-hr standard at a level of 0.12 ppm, with a maximum expected exceedance rate of one per year (averaged over 3 years).
- An 8-hr standard in the range of 0.08-0.10 ppm, with a maximum expected exceedance rate of one per year (averaged over 3 years).
- An 8-hr standard in the range of 0.06-0.08 ppm, with a maximum expected exceedance rate of five per year (averaged over 3 years).

The following alternative standard was subsequently added:

- An 8-hr standard at a level of 0.07 ppm, with a maximum expected exceedance rate of one per year (averaged over 3 years).

Alternative concentration-based forms (e.g., the 2nd to 5th highest 8-hr daily maximum concentration, averaged over 3 years) for various alternative standards have also been assessed, as discussed in Section V.I.

Similarly, the Plan identified the following alternative secondary standards for use in initial analytic assessments:

- A standard with a form that is seasonal, cumulative, and peak-weighted. Specifically:
  - a SUM06 standard (which sums all hourly O_3 concentrations of 0.06 ppm and higher over a specified period of time) in the range of 16.5-26.4 ppm-hrs for the maximum 3 calendar-month period.
  - a SUM08 standard (which sums all hourly O_3 concentrations of 0.08 ppm and higher over a specified period of time) at a level equivalent in crop protection to the range of SUM06 options.
- An 8-hr secondary standard equivalent to any 8-hr primary standard that may be established.

Additional seasonal, cumulative, peak-weighted forms that incorporate peak-weighting functions other than the SUMxx form have also been assessed, as discussed in Section VII of this Staff Paper.

B. Organization of Document

This Staff Paper is organized into sections as outlined below. Section IV provides a summary of air quality trends, air quality distributions, and a characterization of ozone background concentrations.

Section V presents discussions of mechanisms of human toxicity, factors which modify responses, effects of concern and effect levels, populations potentially at risk, and exposure and risk analyses. Staff judgments are made concerning which effects are important for the Administrator to consider in selecting appropriate primary standard(s).

Section VI discusses factors important in selecting primary standard(s) including alternative averaging times and forms of the standard. Drawing on these factors and on

information contained in Section V, staff conclusions and recommendations are presented for the Administrator to consider in selecting appropriate primary O_3 NAAQS.

In a similar approach for selecting appropriate secondary standard(s), Section VII of provides information on mode of vegetation response, factors that modify plant response, effects on vegetation and natural ecosystems, exposure indices, and exposure, risk, and economic benefits assessments. Based on this information, Section VIII discusses alternative forms, averaging times, and levels for the secondary NAAQS, and offers staff conclusions and recommendations for the Administrator to consider in selecting appropriate secondary O_3 NAAQS.

IV. AIR QUALITY CHARACTERIZATION

This section provides summaries of O_3 air quality trends and the spatial and temporal distribution of O_3 air quality concentrations. The concept of O_3 background is also presented, together with estimates of background concentrations at ground-level for various averaging times.

A. Air Quality Trends

States and local air pollution control agencies measured ground level hourly O_3 concentrations at 925 monitoring stations throughout the nation during 1993. Most of these monitoring sites are located in urban and suburban area locations, with far less frequent measurement in rural areas. These data constitute the ambient data base used in this staff paper to assess O_3 air quality trends, as well as to compare selected alternative standards.

The interpretation of recent O_3 trends is difficult due to the large temporal variation that results from the confounding factors of meteorology and emissions changes. Peak O_3 concentrations typically occur during hot, dry, stagnant summertime conditions. Thus, Summer 1988, as the third hottest summer on record since 1931, was highly conducive to O_3 formation with peak O_3 levels comparable to those recorded in the earlier peak year of 1983.

Meteorological conditions in 1991 and 1993 were also highly conducive to O_3 formation, especially in the eastern half of the country, although the magnitude and frequency of exceedances were significantly less than those recorded in 1988. In contrast, the years 1989 and 1992 saw meteorological conditions that were generally not as conducive to O_3 formation. These changes in meteorological conditions have led to large year-to year differences in peak O_3 concentrations. In response to the National Academy of Sciences recommendations (NAS, 1991), EPA has developed a statistical model (Cox and Chu, 1993) that adjusts for meteorological variability to detect the underlying O_3 trend.

Figure IV-1 presents the meteorologically adjusted, and unadjusted, ten year O_3 trends in 43 metropolitan areas. The 99th percentile daily maximum 1-hour concentration declined 1 percent per year or 12 percent since 1984. The national trend in the composite mean of the annual second highest daily maximum 1-hour concentration at 509 sites is shown for comparison, which coincidently, also declined by 12 percent between 1984 and 1993. The large year-to-year fluctuations in peak O_3 levels introduces a measure of instability in nonattainment statistics. Appendix A contains an expanded discussion of both O_3 air quality trends, variability in nonattainment status, and the relationship among alternative averaging times, air quality statistics, and standards.

B. Air Quality Distributions

This section provides a brief overview of how both 1-hour and 8-hour O_3 concentrations vary across the country and among differing monitoring environments. Figure IV-2 displays a map of those counties with 1-hour daily maximum, 1 expected exceedance O_3 design values greater than 0.12 ppm based on 1991-93 air quality monitoring data. The bar chart to the right of the map indicates the number of people living in the corresponding shaded counties. Figure IV-3 shows the spatial distribution of counties with 8-hour daily maximum, 1 expected exceedance design values greater than 0.08 ppm, based on 1991-93 data also. Figure IV-4 depicts those counties with a 3-month SUM06 exposure index value greater than 25 ppm-hours. These SUM06 values are based only on the daylight hours, 8:00 am - 8:00 pm Local Standard Time (LST) in 1990. In each of these maps, the county air quality status was determined by the peak design value site in each county. For the one exceedance standard options, the design value is simply the fourth highest concentration measured during 1991-93, since if the fourth highest value is reduced to the level of the standard, there will be only three days above the level of the standard, or 1 exceedance per year. Similarly, the SUM06 exposure index design value is simply the index value itself. Additional air quality comparisons are presented in Appendix A, including an examination of

Figure IV-1. Metropolitan area O_3 trends adjusted for meteorological variability, 1984-93.

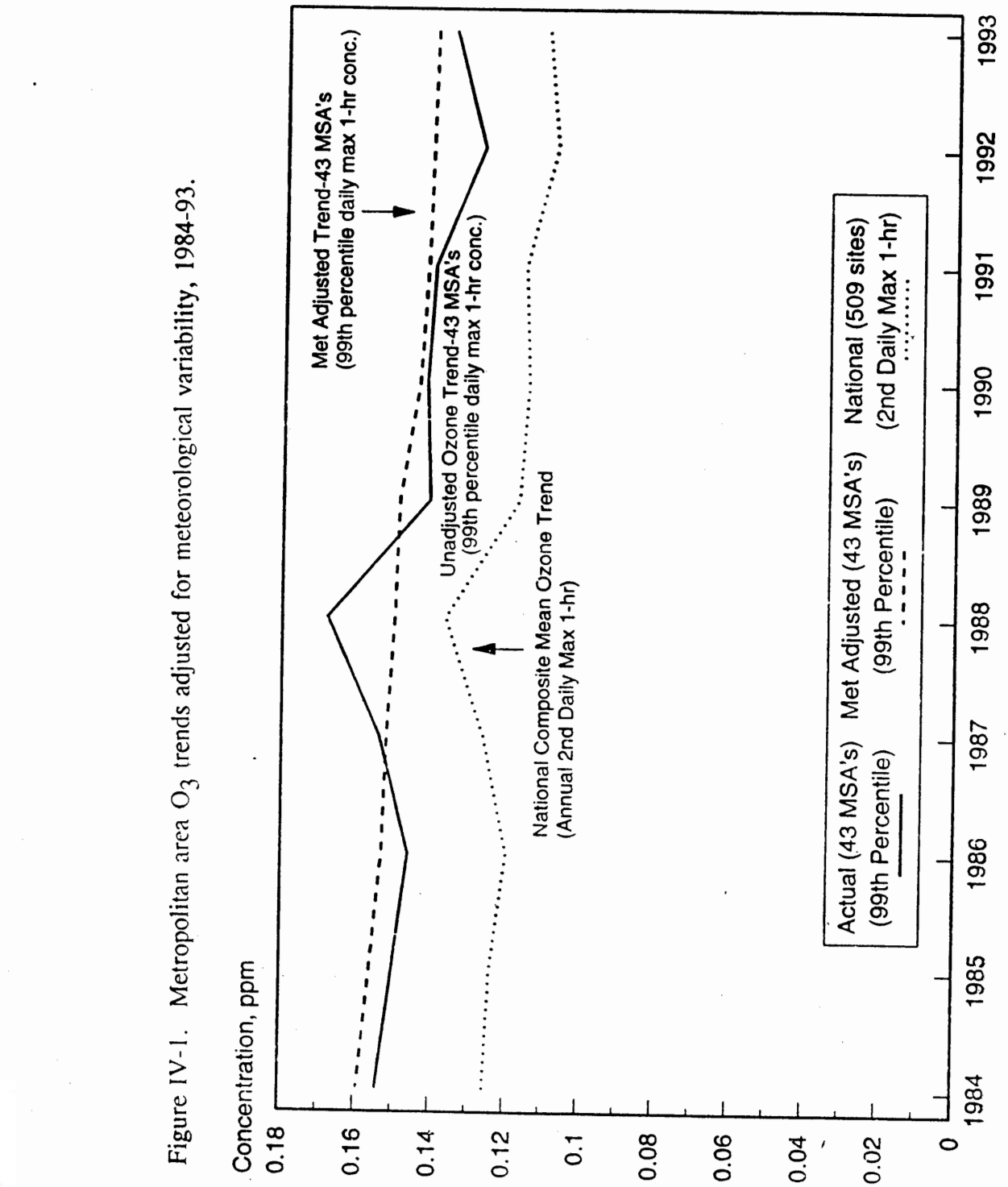


Figure IV-2. Spatial distribution of counties with 1-hour daily maximum, 1 expected exceedance design values greater than 0.12 ppm based on 1991-93 air quality data.

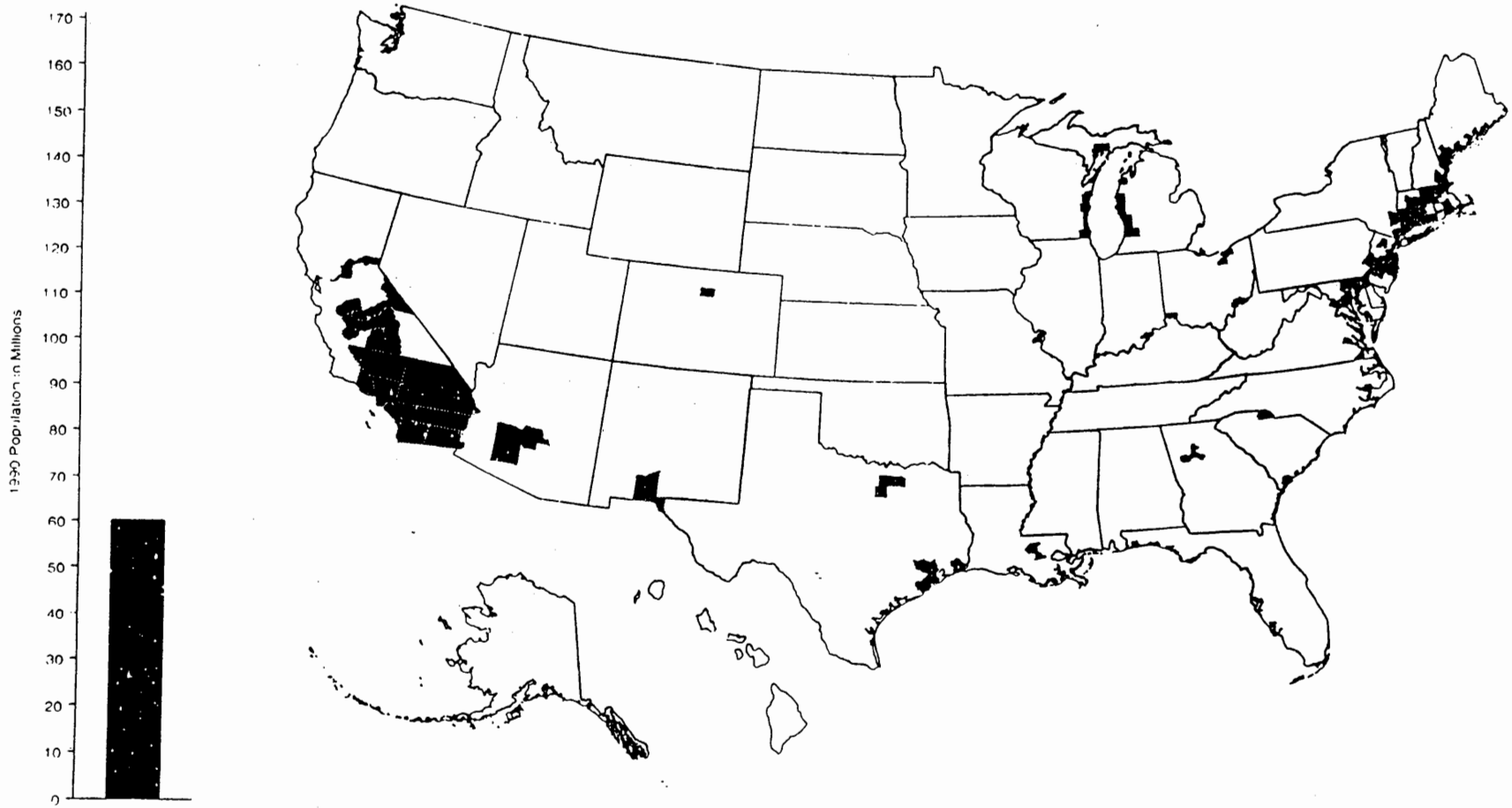
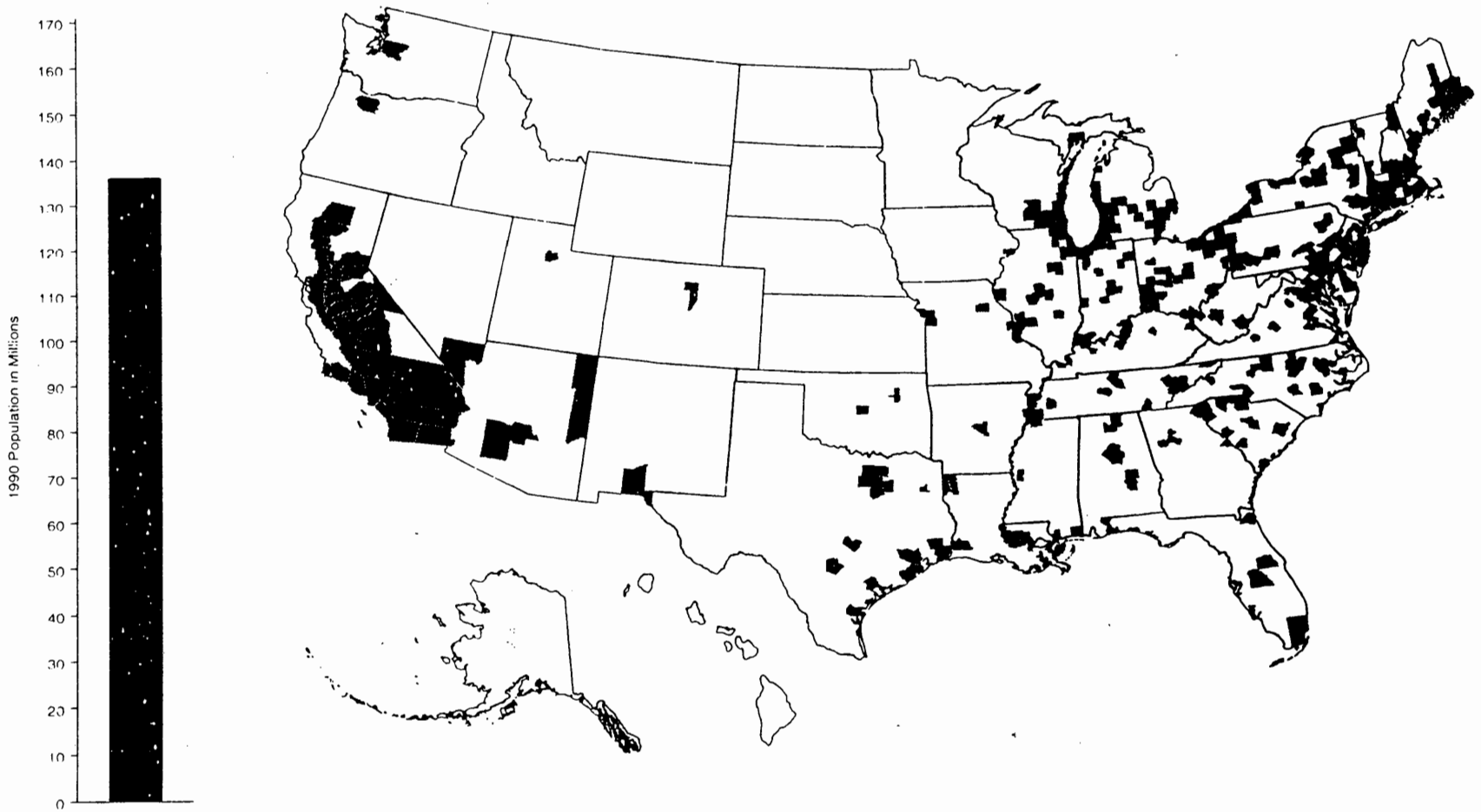


Figure IV-3. Spatial distribution of counties with 8-hour daily maximum, 1 expected exceedance design values greater than 0.08 ppm based on 1991-93 air quality data.



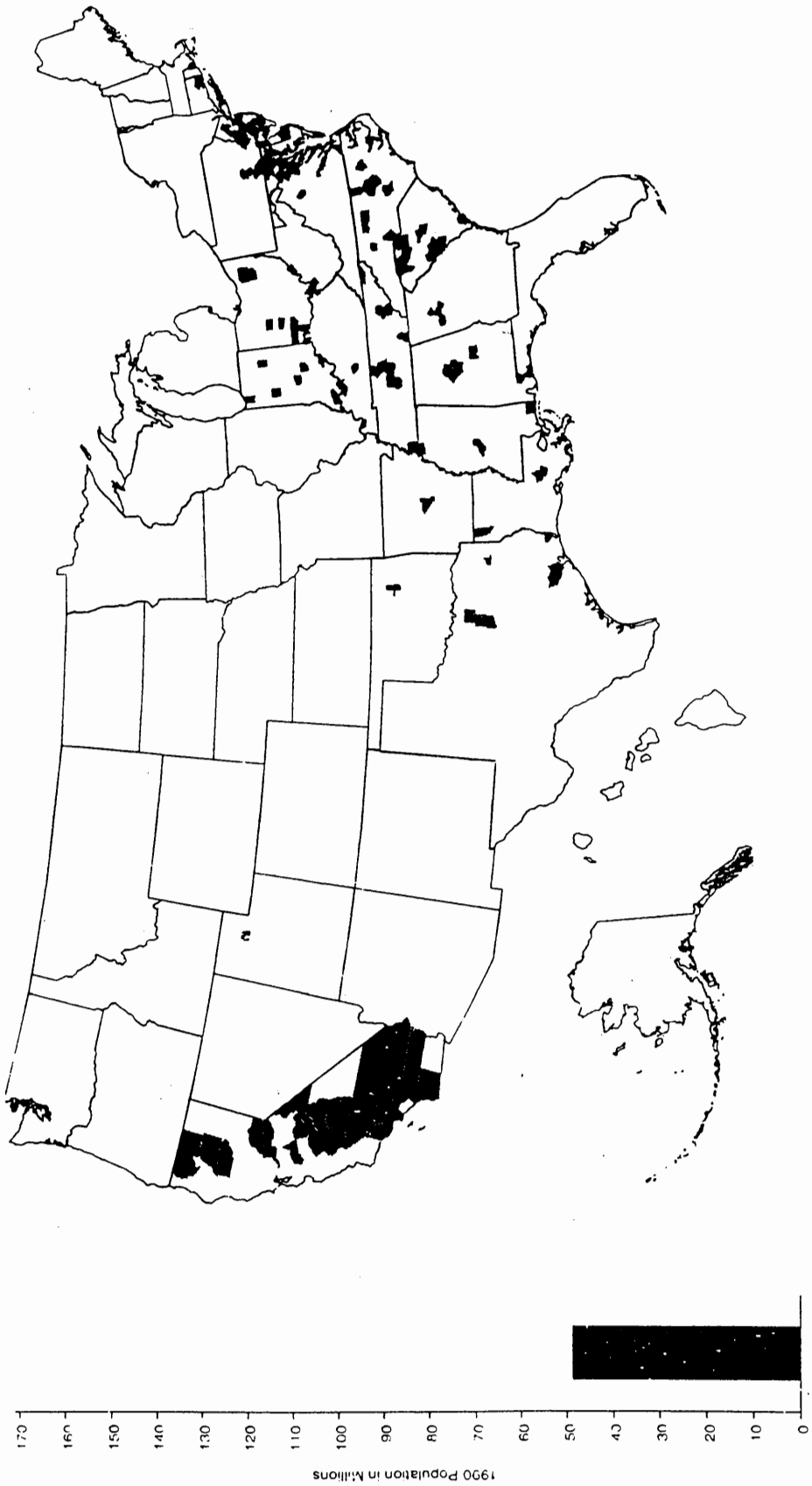


Figure IV-4. Spatial distribution of counties with highest 3-month Sum06 exposure index values greater than 25 ppm-hours in 1990 (based on 8:00 am - 8:00 pm LST hours only).

alternative levels, forms and interrelationships among alternative standards. These comparisons are summarized on a county, metropolitan area, and nonattainment area basis.

The staff has also examined the differences in air quality concentration distributions among differing monitoring site location environments, particularly for the secondary standard comparisons. Figure IV-5 presents histograms of the hourly O_3 concentrations for the peak 3-month summer period at urban and downwind sites in Chicago, a site downwind of Atlanta, and a site at higher elevation in Albuquerque. All hourly concentrations equal to, or greater than, 0.06 ppm are displayed with darker shading. The values of three alternative exposure indices for alternative secondary standards have been computed and are displayed for each site. There are distinct differences among these sites, with downwind sites exhibiting a greater frequency of higher concentrations.

C. Ozone Background

Ozone is a naturally occurring, 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 has historically been no standardized terminology regarding the concept of O_3 background.[2] Even when a numerical estimate of background (however labeled) is provided, rarely is the averaging time provided for the estimate.

Based on a review of the available literature, it is obvious that "natural" O_3 background is a multidimensional and complex concept. Background O_3 concentrations vary by geographic location, altitude and season. For the purposes of this document, background ozone is defined as the ozone concentrations that would be observed in the U.S. in the

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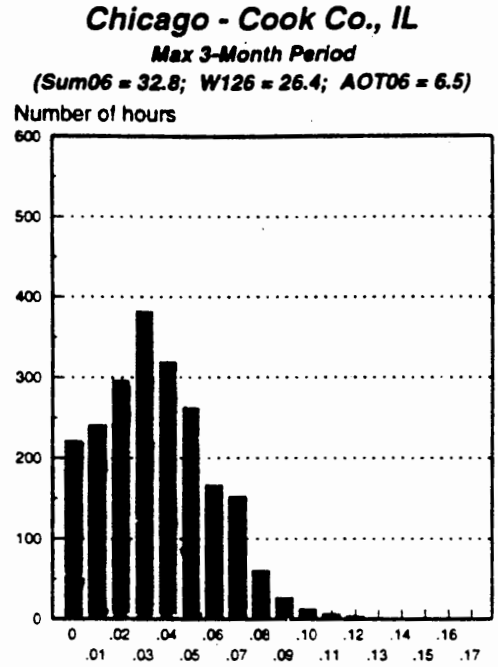
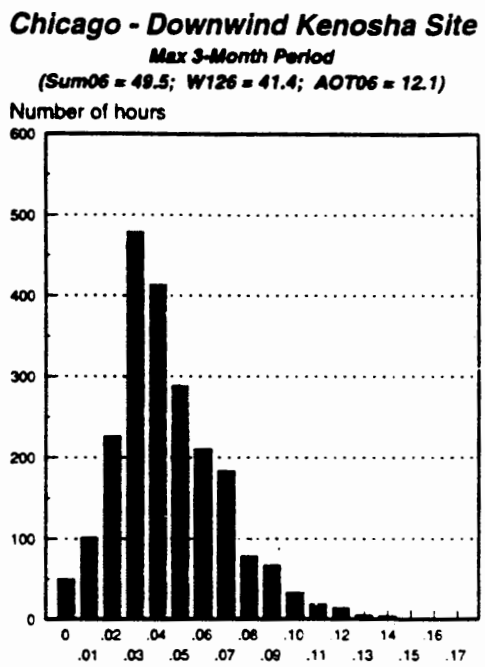
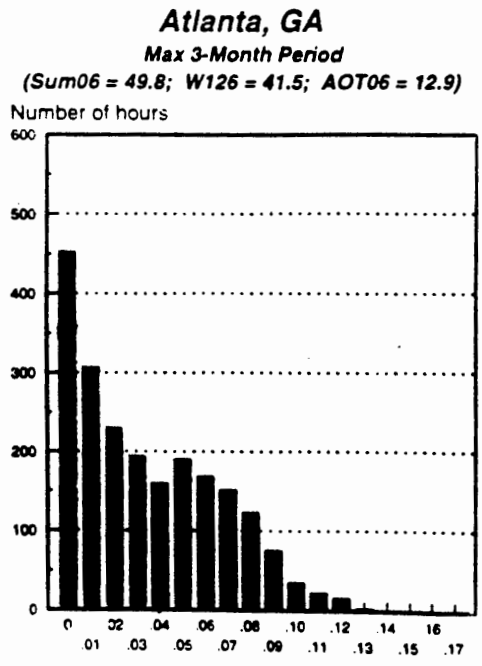
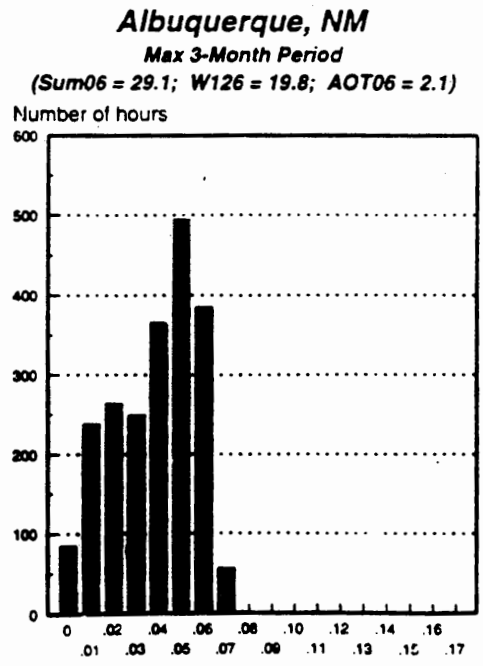
[2] In fact, a survey of the available literature that mentions background ozone or natural ozone background--approximately 50 articles--did not uncover a single rigorous definition of either term! Even the appellations used for the concepts vary greatly in the relevant literature. Examples include: "baseline ozone," "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.

Figure IV-5. Hourly frequency distributions for the maximum 3-month O_3 period.









absence of anthropogenic or biogenic emissions of VOCs and NO_x in North America. During the summertime O_3 season in the U.S., daily 1-hr. maximum background O_3 is typically between 0.03 to 0.05 ppm. Part of this background is due to natural sources and part of it is due to long-range transport of anthropogenic or biogenic emissions.

The natural component of the background originates from three sources: stratospheric O_3 which is transported down to the troposphere, O_3 formed from the photochemically-initiated oxidation of biogenic and geogenic methane and carbon monoxide, and the photochemically-initiated oxidation of biogenic VOCs. The magnitude of this natural part cannot be precisely determined for two reasons. First, the part due to long-range transport of anthropogenic precursor emissions is not known. Second, NO_x plays an important role in the oxidation of methane, carbon monoxide and the biogenic VOCs and it is not possible to determine amount of O_3 that would have been formed just due to natural NO_x emissions. However, some estimates can be made.

On the basis of O_3 data from isolated monitoring sites (CD, Ch. 4), a reasonable estimate of the O_3 background concentration near sea level in the U.S. for an annual average is 0.020 to 0.035 ppm. This estimate includes a 0.005 to 0.015 ppm contribution (averaged over time) from stratospheric intrusions into the troposphere and a 0.01 ppm contribution from photochemically-initiated oxidation of methane and carbon monoxide. The remainder is due to the photochemically-initiated oxidation of biogenic VOCs and long-range transport.

Similarly, a reasonable estimate of the background O_3 concentration near sea level in the U.S. for a 1-hour daily maximum during the summer is usually in the range of 0.03 to 0.05 ppm. At clean sites in the Western U.S., the maximum annual hourly values are in the range of 0.060 to 0.075 ppm. Such elevated O_3 levels may be occurring at higher altitudes due to stratospheric O_3 intrusion. Summertime daily maxima of less than 0.03 are also observed due to precipitation scavenging. These estimates are synthesized from the available literature, but rely most heavily on Altshuller (1986), Kelly et al. (1982, 1984), and Lefohn

and Foley (1992). Based on the diurnal profiles presented for O_3 at rural sites in Kelly et al. (1982, 1984), it is reasonable to estimate that the 8-hour daily maximum O_3 during the summer is also in the range of 0.03 to 0.05 ppm.

V. SCIENTIFIC AND TECHNICAL BASIS FOR PRIMARY NAAQS

A. Introduction

This section presents critical information for the review of the primary NAAQS for O_3. This information includes identification of: (1) the principal mechanisms of toxicity which help to establish a link between O_3 exposure and resultant health effects; (2) specific health effects associated with O_3 exposure and estimates of lowest observed effects levels; (3) factors which may modify the extent and nature of responses to O_3 experienced by individuals; (4) a qualitative discussion of populations potentially at risk to O_3 exposures; and (5) which effects may be of public health concern (i.e., "adverse" effects). Further, this section presents quantitative estimates of exposure and risk to help inform judgments as to which primary standard(s) for O_3 would protect public health with an adequate margin of safety. Finally, alternative forms of primary standards are discussed.

B. Mechanisms of Toxicity

Ozone enters the human body through the respiratory tract where it reacts with polyunsaturated fatty acids (PUFAs), various electron donors (e.g., ascorbate and vitamin E), and the thiol, aldehyde, and amine groups of low molecular weight biochemicals and proteins. Mechanisms which explain biochemical and physiological effects of human exposure to O_3 are complex and often involve the direct action of O_3 on macromolecules in the lungs. However, they also can involve the reaction of secondary biochemical products resulting from the generation of free radical-precursor molecules, the release of endogenous mediators of physiological response, and the reactive oxygen intermediates and proteinases associated with the activities of inflammatory cells that subsequently infiltrate into O_3-damaged lungs.

One hypothesis, based on the high reactivity of O_3, suggests that O_3 does not penetrate beyond the surface lining fluids of the lungs except in those terminal airway regions with minimal thickness of the lining, where epithelial cells might be unprotected by either mucus or surfactant (Pryor, 1992). In a review of pathological effects of O_3 (Pryor, 1991),

it was suggested that O_3-induced cellular damage is more likely to result from reactions of the more stable, less reactive ozonide, aldehyde, and hydroperoxide reaction products of O_3 with surface-lining fluid components than from direct reaction of O_3 with intracellular components. A wide variety of toxicological effects has been linked to O_3 exposure, including lung inflammation, effects on host defense mechanisms, morphological effects, pulmonary function decrements, changes in lung biochemistry, and genotoxicity and carcinogenicity. Although these effects may have different physiological mechanisms, each effect is initiated by the preliminary interactions of O_3 and O_3-reaction products with fluids and epithelial cells in the respiratory tract.

Mechanisms leading to O_3-induced lung function decrements and symptoms are probably the best understood of the mechanisms of O_3 toxicity in humans. The CD (Sec. 7.2.1.1) identifies several such mechanisms, including: (1) O_3 delivery to the tissue (i.e., inhaled concentration of O_3, breathing pattern, airway geometry); (2) O_3 reactions with the airway lining fluid and/or epithelial cell membranes; (3) local tissue responses, including injury and inflammation; and (4) stimulation of neural afferents (bronchial C- fibers) and the resulting reflex responses and symptoms. The cyclooxygenase inhibitors block production of prostaglandin E_2 (PGE_2) and interleukin-6 (IL-6), as well as reduce lung volume responses; however, cyclooxygenase inhibitors don't reduce neutrophilic inflammation and levels of cell damage markers such as lactate dehydrogenase. More detailed discussions of biochemical targets of O_3 interaction and of the mechanisms of acute pulmonary response can be found in the CD in Chapters 6 and Chapter 7, respectively.

C. Health Effects of Ozone

The following discussion of O_3 health effects is presented as a summary of the most important conclusions and is based on the review and evaluation of health effects research literature which has been discussed in much greater detail in Chapters 6 through 9 of the CD. This section of the Staff Paper integrates information from human clinical, epidemiological, and animal toxicological studies, as appropriate, within each subsection on effects. Furthermore, the effects on healthy individuals and on individuals with impaired respiratory systems (e.g., asthmatics) are discussed in the context of acute, prolonged, and, finally, chronic exposures within the following subsections.

A wide array of health effects has been attributed to short-term (1 to 3 hrs), prolonged (6 to 8 hrs), and long-term (months to years) exposures to O_3. Those acute health effects induced by short-term exposures to O_3 concentrations as low as 0.12 ppm, generally occur while subjects are engaged in heavy (e.g., running) exercise, include: transient pulmonary function responses, transient respiratory symptoms and effects on exercise performance, increased airway responsiveness, transient pulmonary inflammation, and increased hospital admissions and emergency room visits for respiratory causes. Similar health effects have been observed following prolonged exposures to O_3, at concentrations of O_3 as low as 0.08 ppm and at lower levels of exercise than for short-term exposures. Although chronic effects such as structural damage to pulmonary tissue and impaired host defense mechanisms have been established in a substantial number of laboratory animal studies, there remains little of no evidence of association between ambient O_3 exposures and carcinogenicity and/or genotoxicity at this time.

Prior to completion of the previous review of scientific criteria in 1989, there was a substantial data base defining the health effects of O_3. Key human and laboratory animal studies in that database are listed in Table V-1. Since 1989 numerous new studies have greatly expanded the information on O_3 health effects, particularly on prolonged exposures of 6- to 8-hrs. A selected group of recent key human and laboratory animal studies has been summarized in Table V-2. Each of these tables includes only a small fraction of the total data base linking O_3 exposure with health effects in humans. Inclusion criteria for these studies are mainly the adequacy of scientific credibility, as determined and discussed in the CD, and the relevance to regulatory decision making, as determined by staff, the CASAC, and public review.

1. Pulmonary Function Responses

A variety of pulmonary function responses has been observed in healthy and impaired humans acutely exposed to O_3. These responses include reductions in forced vital capacity (FVC), forced expiratory volume in 1 sec (FEV_1), and forced expiratory flow at 25 to 75% of FVC ($FEF_{25-75\%}$), which are usually measured by having an individual exhale forcefully into a spirometer designed to measure expiratory flow rates. Another acute response to O_3, airway resistance (R_{aw}), is typically measured in a body plethysmograph.

TABLE V-1. KEY HEALTH STUDIES SUPPORTING THE CURRENT 1-HOUR NATIONAL AMBIENT AIR QUALITY PRIMARY STANDARD FOR OZONE

O_3 Concentration, ppm	Health Effect	Reference
Ambient air containing 0.01-0.14 daily 1-hr max over days to weeks	Decrements in lung function in children, adolescents and adults exercising outdoors	Berry et al. (1991) Bock et al. (1985) Higgins et al. (1990) Kinney et al. (1989) Lioy and Dyba (1989) Lioy et al. (1985) Lippmann et al. (1983) Raizenne et al. (1987, 1989) Spektor et al. (1988a,b; 1991)
≥0.12 (1-3 hr) or ≥0.08 (6.6 hr) (chamber exposures)	Decrements in lung function (reduced ability to take a deep breath), increased respiratory symptoms (cough, shortness of breath, pain upon deep inspiration), increased airway responsiveness and increased airway inflammation in heavily exercising adults	Adams et al. (1981) Avol et al. (1983, 1984) Devlin et al. (1991) Folinsbee and Horvath (1986) Folinsbee et al. (1978, 1984, 1988) Gibbons and Adams (1984) Gliner et al. (1983) Horstman et al. (1990) Koren et al. (1989a,b, 1991) Kulle et al. (1985) Lauritzen and Adams (1985) Linn et al. (1980, 1983a,b, 1986, 1988) McDonnell et al. (1983, 1991) Seltzer et al. (1986)

O_3 Concentration, ppm	Health Effect	Reference
≥ 0.12 (1-3 hr) (chamber exposures)	Decrements in lung function in heavily exercising children and adolescents	Avol et al. (1985a,b,c, 1987) Koenig et al. (1987, 1988) McDonnell et al. (1985)
≥ 0.12 (1-3 hr) (chamber exposures)	Effects are similar in individuals with preexisting disease except for a greater increase in airway responsiveness for asthmatic and allergic subjects	Koenig et al. (1985, 1987, 1988) Kreit et al. (1989) McDonnell et al. (1987)
≥ 0.12 (1-3 hr) (chamber exposures)	Older subjects (>50 yr old) have smaller and less reproducible changes in lung function	Bedi and Horvath (1987) Bedi et al. (1988, 1989) Drechsler-Parks et al. (1987, 1989, 1990) Reisenauer et al. (1988)
≥ 0.18 (1-3 hr) (chamber exposures)	Reduced exercise performance in heavily exercising adults	Adams and Schelegle (1983) Folinsbee et al. (1984) Gong et al. (1986) Linder et al. (1988) Schelegle and Adams (1986)
≥ 0.12 (1-3 hr) (chamber exposures)	Attenuation of lung function response with repeated exposure	Avol et al. (1988) Farrell et al. (1979) Hackney et al. (1976, 1989) Horvath et al. (1981) Kulle et al. (1982) Linn et al. (1982, 1988)

O_3 Concentration, ppm	Health Effect	Reference
≥0.12 with chronic, repeated exposure (chamber exposures)	Changes in lung structure, function, and biochemistry in laboratory animals that are indicative of airway irritation and inflammation with possible development of chronic lung disease	Amdur et al. (1978) Barry et al. (1983, 1985, 1988) Boorman et al. (1980) Castleman et al. (1977, 1980) Chow et al. (1981) Costa et al. (1983) Crapo et al. (1984) Eustis et al. (1981) Filipowicz and McCauley (1986a,b) Fujinaka et al. (1985) Grose et al. (1989) Last et al. (1979,1984) Moore and Schwartz (1981) Mustafa et al. (1985) Plopper et al. (1979) Rao et al. (1985a,b) Schwartz et al. (1976) Sherwin and Richters (1985) Tyler et al. (1988) Wegner (1982) Wright et al. (1988)
≥ 0.08 (3 hr) or ≥ 0.10 with chronic repeated exposure (chamber exposures)	Increased susceptibility to bacterial respiratory infections in laboratory animals	Coffin et al. (1972) Ehrlich et al. (1977) Miller et al. (1978) Aranyi et al. (1983)

TABLE V-2. KEY HEALTH STUDIES PUBLISHED SINCE THE LAST REVIEW OF THE PRIMARY NATIONAL AMBIENT AIR QUALITY STANDARD FOR OZONE

O_3 Concentration, ppm	Health Effect	Reference
Ambient air containing 0.01-0.154 daily 1-hr max over days to weeks	Decrements in lung function (FEV_1) in children, adolescents, and adults exposed to O_3 outdoors	Avol et al. (1990, 1991) Braun-Fahrlander et al. (1994) Castillejos et al. (1992) Hoek et al. (1993a,b) Kilburn et al. (1992) Krzyzanowski et al. (1992) Lebowitz et al. (1991) Raizenne et al. (1987, 1989) Raizenne and Spengler (1989) Schmitzberger et al. (1993) Schwartz et al. (1994a,b,c) Spektor et al. (1991) Spektor and Lippmann (1991) Stern et al. (1989, 1994) Thurston et al. (1995)
	Exacerbation of respiratory symptoms (e.g., cough, chest pain) in individuals with preexisting disease (e.g., asthma) with low ambient exposure, decreased temperature, and other environmental factors resulting in increased summertime hospital admissions and emergency department visits for respiratory causes.	Burnett et al. (1994) Cody et al. (1992) Thurston et al. (1992, 1994, 1995) Weisel et al. (1995) White et al. (1994)
≥0.12 (1-3 hr) ≥0.08 (6.6 hr) (chamber exposures)	Decrements in lung function (reduced ability to take a deep breath), increased respiratory symptoms (cough, shortness of breath, pain upon deep inspiration), increased airway responsive-ness and increased airway inflammation in exercising adults	Devlin et al. (1991, 1990) Folinsbee et al. (1991, 1994, 1995) Frampton et al. (1993) Gross et al. (1991) Hazucha et al. (1992, 1987) Horstman et al. (1990, 1995) Koren et al. (1991) McKittrick et al. (1995) McDonnell et al. (1991) Linn et al. (1994)
≥0.12 (1-3 hr) ≥0.08 (6.6 hr) (chamber exposures)	Effects are similar in individuals with preexisting disease except for a greater increase in airway responsiveness for asthmatic and allergic subjects	Koenig et al. (1988) Kreit et al. (1989)

O_3 Concentration, ppm	Health Effect	Reference
≥0.12 (1-3 hr) ≥0.08 (6.6 hr) (chamber exposures)	Older subjects (>50 yr old) have smaller and less reproducible changes in lung function.	Drechsler-Parks et al. (1990) Horvath et al. (1991) Seal et al. (1993, 1994)
	Attenuation of response with repeated exposure	Hackney et al. (1989) van Bree et al. (1994)
≥0.12 with prolonged, repeated exposure (chamber exposures)	Changes in lung structure, function, elasticity, and biochemistry in laboratory animals that are indicative of airway irritation and inflammation with possible development of chronic lung disease	Catalano et al. (1995a,b) Chang et al., (1991, 1992, 1995) Costa et al., (1995) Harkema et al. (1989, 1993, 1994) Harkema and Mauderly (1994) Hiroshima et al. (1989) Hotchkiss et al. (1989a,b) Hyde et al. (1992) Last et al. (1993a,b, 1994) National Toxicology Program/Health Effects Institute (1995) Parks and Roby (1994) Pinkerton et al. (1992, 1993, 1995) Pino et al. (1992a,b,&c) Plopper et al. (1991, 1994a,b) Radhakrishnamurthy (1994) Schultheis et al. (1991) Szarek (1994) Tan et al. (1992) Tepper et al. (1994, 1995) Tyler et al. (1991) Van Bree et al. (1992)
	Increased susceptibility to bacterial respiratory infections in laboratory animals	Gilmour et al. (1993a,b) Jakab and Bassett (1990) Jakab et al. (1995) Selgrade et al. (1990)

Neurogenic inhibition of maximal inspiration, possibly caused by stimulation of C-fiber afferents, is believed to be the cause of decreased FVC and inspiratory capacity in humans (CD, Sec. 9.3.1.1). Although a large body of evidence generally suggests that healthy individuals and those with impaired respiratory systems have similar functional responses, one recent study (Horstman et al. (1995)), not yet replicated, reported that asthmatics had a greater change in lung function than healthy individuals.

The strongest and most quantifiable exposure-response data on pulmonary function responses to O_3 have come from controlled human exposure studies. The magnitude and time course of spirometry responses to O_3 depend upon the O_3 concentration (C), the "exercise" level (minute ventilation, $\dot{V}_E$), and the duration of exposure (T). One of the best demonstrations of the impact of various "exercise" levels and O_3 concentrations on group mean FEV_1 following 2-hr exposures is summarized by Hazucha (1987) in Figure V-1 (CD, Figure 9-1). This figure clearly shows that FEV_1 decrements are enhanced by increased levels of "exercise" and/or increased levels of O_3 exposure.

In experimental studies, increased ventilation rates are brought about by having the subjects engage in activities typically identified as "exercise." This exercise is meant to simulate any type of activity involving exertion that increases the ventilation rate. Thus, while experimental studies typically report "exercise" levels, the broader term "exertion" will be used throughout this Staff Paper when referring to the types of normal activities in which people engage that result in similar increased ventilation rates. The staff intends that the term exertion be understood to encompass a much broader class of activities than is typically associated with the term exercise, as discussed in Section V.D.1 of this Staff Paper.

Numerous experimental studies of exercising adults have demonstrated decrements in lung function for exposures of 1-3 hrs at $\geq$0.12 ppm O_3 (Adams et al., 1981; Avol et al., 1983, 1984; Folinsbee and Horvath, 1986; Folinsbee et al., 1978, 1984, 1988; Gibbons and Adams, 1984; Gliner et al., 1983; Kulle et al., 1985; Lauritzen and Adams, 1985; Linn et al., 1980, 1983, 1986, 1988; McDonnell et al., 1983; Seltzer et al., 1986) and for exposures of 6.6 hrs at $\geq$0.08 ppm O_3 (Folinsbee et al., 1988, 1991, 1994; Hazucha et al., 1992; Horstman et al., 1990, 1995; Horvath et al., 1991; Koren et al., 1988, 1989, 1991; Linn et

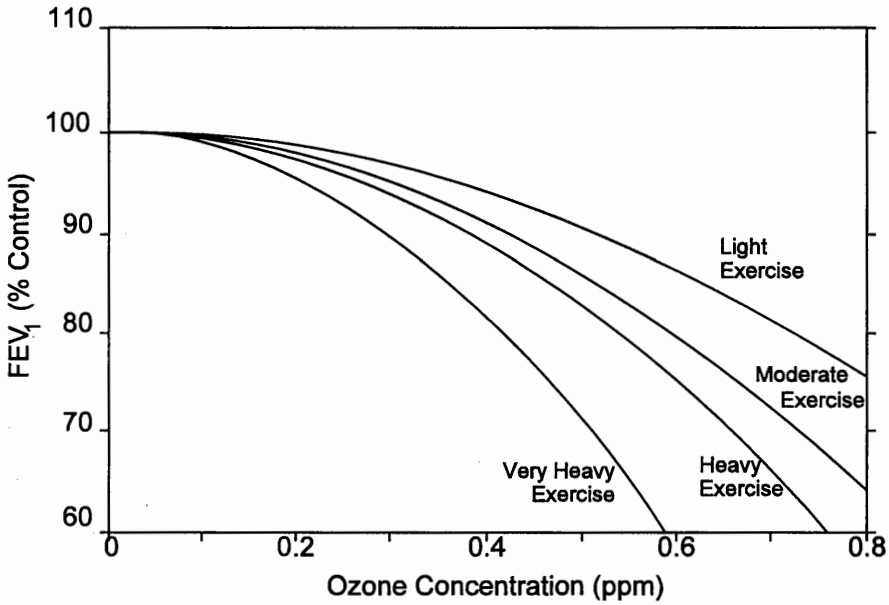


Figure V-1. Mean predicted changes in forced expiratory volume in 1 sec following 2-hr exposures to ozone with increasing levels of intermittent exercise.

Source: Hazucha (1987); CD, Figure 9-1, p. 9-16.

al., 1994; McDonnell et al., 1991). These studies provide conclusive evidence that O_3 levels commonly monitored in the ambient air induce FEV_1 decrements in exercising adults.

For short-term exposures of 1 to 2 hr, subjects exposed to higher O_3 concentrations (e.g., > 0.25 ppm) during intermittent heavy exertion tend to experience rapid responses indicative of a plateau (See Figure V-2.A). In contrast, lower O_3 concentrations with lighter exertion tend to induce responses which progress slowly and may not reach a plateau during the period of exposure. McDonnell and Smith (1994) plotted predicted mean decrements in FEV_1 vs. time, with intermittent moderate exertion during a 6.6 hr exposure; they found no response plateaus at 0.08, 0.10, or 0.12 ppm O_3 during the first 3 hr but did show plateaus developing at each concentration during the latter portion of the exposure (See Figure V-2B).

Summer camp studies have provided the most extensive and reliable data base on acute lung function responses to ambient O_3 and other pollutants in children and adolescents living in the northeastern U.S. (Bock et al., 1985; Spektor et al., 1988a,b, 1991; Spektor and Lippmann, 1991; Lippmann et al., 1983; Lioy et al., 1985; Lioy and Dyba, 1989; Kinney et al., 1989; Berry et al., 1991; Thurston et al., 1995), southern California (Higgins et al., 1990; Avol et al., 1990, 1991), and southeastern Canada (Raizenne et al., 1987, 1989;

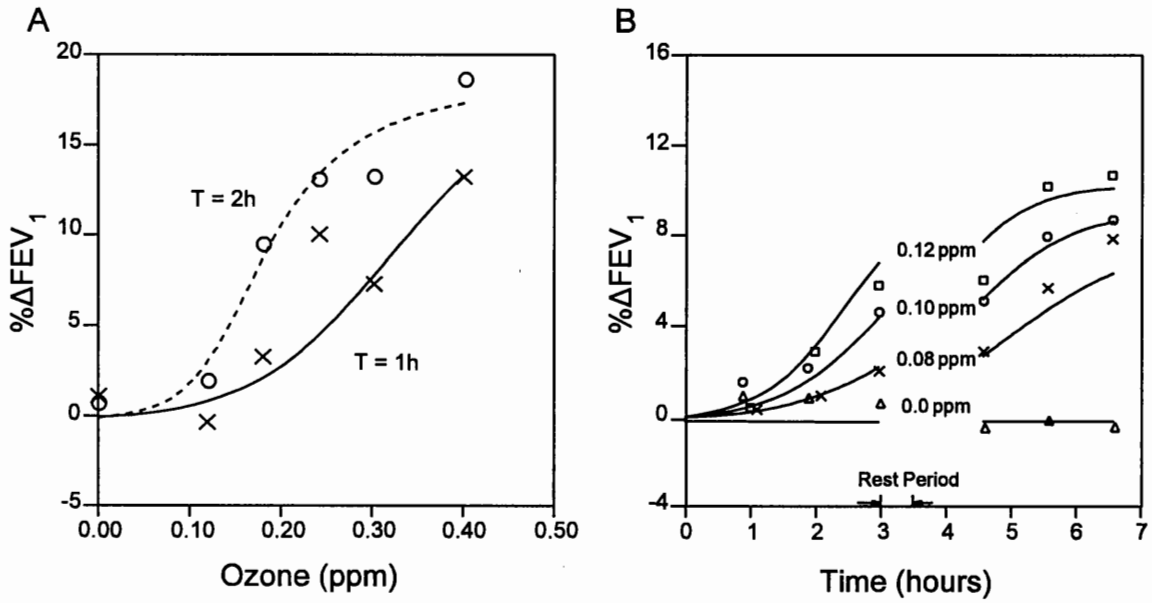


Figure V-2. Predicted mean decrements in forced expiratory volume in 1 s for 1- and 2-h exposures to ozone with intermittent heavy exercise (A) and 6.6-h exposures with moderate prolonged exercise (B).

Source: McDonnell and Smith (1994); CD, Figure 9-2, p. 9-17.

Raizenne and Spengler, 1989). Lung function changes reported at low O_3 concentrations are comparable to those reported in children and adults exposed under controlled experimental conditions, although direct comparisons are difficult to make because of differences in experimental design and analytical approach. Even though exposures at the summer camps occurred over periods of many hours to days, a key calculation made for many of the studies is the slope of the relationship between FEV_1 and the O_3 concentration measured during the previous hour, without consideration of the background levels. The average slope from six of the camp studies (Spektor et al., 1988a, 1991; Spektor and Lippmann, 1991; Raizenne et al., 1987, 1989; Higgins et al., 1990; Avol et al., 1990, 1991) was -0.50 mL/ppb O_3, within a concentration range of 0.01 to 0.16 ppm (CD, Sec. 7.4.1.2). The slope corresponds to a decrease in FEV_1 of 60 mL at 0.12 ppm from a base level of 2000 to 2500 mL or roughly a 2.4 to 3.0% decrease in FEV_1. This is comparable to the 3.4% decrease in FEV_1 reported by McDonnell et al. (1985) for boys (8 to 11 years old) exposed to 0.12 ppm O_3 during heavy exercise under controlled experimental conditions. Although outdoor studies (Spektor et al., 1988b; Selwyn et al., 1985; Brunekreef et al., 1994) of exercising adults have shown similar associations between spirometric changes and increasing O_3 concentrations (CD,

9.3.1), "daily life studies" (Kinney et al., 1989; Castillejos et al., 1992; Hoek et al., 1993b; Krzyzanowski et al., 1989) are difficult to interpret due to the role of seasonal factors (e.g., pollens, epidemics of respiratory infection, changes in activity patterns) and the preponderance of time spent indoors by subjects (CD, Sec. 7.4.1.2).

2. Respiratory Symptoms and Effects on Exercise Performance

Various human respiratory symptoms, including cough, throat irritation, chest pain on deep inspiration, nausea, and shortness of breath, have been induced by O_3 exposures of healthy and impaired individuals. As is the case for spirometric lung function decrements, O_3- exposure data do not support enhanced sensitivity to symptoms of individuals with asthma. Although eye irritation is a symptom commonly associated with exposure to ambient oxidant mixtures, which include such oxidants as O_3 and peroxyacyl nitrates, controlled human exposure studies of O_3 have demonstrated that at concentrations reported in the ambient air, O_3 alone does not induce eye irritation.

A potential linkage between changes in spirometry and at least one symptom may be explained in part by the mechanism which induces cough. The receptors responsible for cough may be unmyelinated C-fibers or rapidly adapting receptors located in the larynx and the largest conducting airways (CD, Sec. 9.3.1.1). Field and epidemiology studies (Ostro et al., 1993; Krupnick et al., 1990) which have reported spirometry changes associated with ambient O_3 levels also have indicated associations between hourly or daily ambient O_3 levels and presence of symptoms such as cough, particularly in asthmatic children.

Respiratory symptom responses to O_3 exposure follow a monotonic exposure-response relationship that has a similar form to that for spirometry responses. Increasing exposure levels elicit increasingly more severe symptoms that persist for longer periods. Symptom and spirometry responses follow a similar time course during an acute exposure and the subsequent recovery, as well as over the course of several days in a repeated exposure study. Furthermore, medication interventions that block or reduce spirometry responses have a similar effect on symptom responses. As with spirometry responses, symptom responses vary considerably among subjects, although the individual correlations between spirometry and symptom responses are relatively low.

Ozone-induced interference with exercise performance, either by reducing maximal sustainable levels of activity or reducing the duration of activity that can be tolerated at a particular work level, is likely related to symptoms. In several heavy or severe exercise studies (Schelegle and Adams, 1986; Gong et al., 1986; Adams and Schelegle, 1983) of athletes exposed to O_3, the discomfort associated with the respiratory symptoms caused by O_3 concentrations in excess of 0.18 ppm was of sufficient severity that the athletes reported that they would have been unable to perform maximally if the conditions of the exposure were present during athletic competition. In workers or active people exposed to ambient O_3, respiratory symptoms may cause reduced productivity or may curb the ability or desire to engage in normal activities.

3. Increased Airway Responsiveness

Increased airway responsiveness is an indication that the airways are predisposed to bronchoconstriction which can be induced by a wide variety of external stimuli (e.g., pollens, dust, cold air, SO_2, etc.). A high level of bronchial responsiveness is characteristic of asthma (CD, 7.2.3). Ozone exposure causes increased responsiveness of the pulmonary airways to subsequent challenge with bronchoconstrictor drugs such as histamine or methacholine. Airway responsiveness is usually measured by having an individual exhale forcefully into a spirometer designed to measure expiratory flow rates (e.g., FEV_1) or by measuring airway resistance (R_{aw}) in a body plethysmograph. Measurements of FEV_1 are taken before and after small amounts of an aerosolized bronchoconstrictor are administered, and the dose is increased until a predetermined degree of airway response has been measured. The provocative dose that produced a 20% drop in FEV_1 would be referred to as "PD_{20}" and the provocative dose that produced a 100% increase in R_{aw} would be referred to as the "PD_{100}."

Increased airway responsiveness is seen even after recovery from spirometric changes, but this effect typically disappears after 24 hrs (CD, Sec. 9.3.1.3). Although changes in airway responsiveness tend to resolve somewhat more slowly than spirometric changes and appear to be less likely to attenuate with repeated exposure, the evidence for a persistent increase in responsiveness from animal studies is inconsistent. Changes in airway responsiveness in rats and guinea pigs tend to occur at higher O_3 concentrations and, as in

humans, tend to be most pronounced shortly after the exposure and less so 24-hr postexposure. Changes in airway responsiveness appear to occur independently of changes in pulmonary function. This response does not appear to be due to airway inflammation (at least the influx of polymorphonuclear leukocytes [PMN's] into the airways) or to the release of arachidonic acid metabolites, but it may be due to epithelial damage and the consequent increased access of these chemicals to smooth muscle in the airways or to the receptors in the airways responsible for reflex bronchoconstriction. The clinical relevance of this observation is that, after O_3 exposure, human airways may be more susceptible to a variety of stimuli, including antigens, chemicals, and particles.

Healthy subjects have experienced small increases in nonspecific bronchial responsiveness, which resolve within 24 hrs, after being exposed to O_3 concentrations as low as 0.20 ppm for 1 hr (Gong et al, 1986) and 0.08 to 0.12 ppm for 6.6 hr (Horstman et al., 1990; Folinsbee et al., 1988). Asthmatic subjects typically have increased airway responsiveness at baseline, and differences in baseline bronchial responsiveness between healthy individuals and sensitive asthmatics may be as much as 100-fold. Changes induced by O_3 exposure, however, are usually only 2- to 4-fold. Only one published study (Molfino et al., 1991) suggested an O_3-induced increase in specific (i.e., allergen-induced) airway reactivity. This effect was reported after a 1-hr resting exposure of atopic asthmatics to 0.12 ppm O_3, and thus provided a plausible linkage between ambient O_3 exposure and increased hospital admissions. However, the study had experimental design flaws and has not yet been replicated. With such a limited and uncertain data base on O_3-induced airway responsiveness, it appears to be premature to draw conclusions regarding this health endpoint at this point in time.

Ongoing studies of O_3-induced increases in airway responsiveness will need to be evaluated in order to determine the exposure-response relationship for alterations in responses to inhaled antigens, especially with regard to sensitive asthmatics. Enhanced response to antigens in asthmatics could lead to increased morbidity (i.e., medical treatment, emergency room visits, hospital admissions) or to more persistent alterations in airway responsiveness (CD, 9.3.1.3).

4. Impairment of Host Defenses

As discussed below, the mammalian respiratory tract has numerous closely integrated defense mechanisms that provide protection from the adverse effects of a wide variety of inhaled particles and microbes, if they function normally. However, when these defense mechanisms break down or are impaired by O_3, there can be an increase in susceptibility to respiratory infection and related respiratory disfunction (CD, Sec. 9.3.3.2).

Mucociliary Clearance of Inhaled Particles. Impaired mucociliary clearance can result in unwanted accumulation of cellular secretions and increased numbers of particles and microorganisms in the lung, leading to increased risk of respiratory infection and bronchitis. Animal studies show that clearance of inhaled insoluble particles is slowed after acute exposure to O_3. Ozone-induced damage to cilia and increased mucus secretion likely contribute to a slowing of mucociliary transport rates. In one study investigating the effects of longer-term alveolarbronchiolar clearance, Pinkerton et al. (1993) exposed rats to an urban pattern of O_3 (continuous 0.06 ppm, 7 days/week with a slow rise to a peak of 0.25 ppm and subsequent decrease to 0.06 ppm over a 9-hr period for 5 days/week) for 6 weeks. The rats were exposed 3 days later to asbestos, which can cause pulmonary fibrosis and tumor formation. Although O_3 did not affect the deposition of asbestos at the site of maximal deposition of both O_3 and asbestos, thirty days later the lungs of the O_3-exposed animals had twice the number and mass of asbestos fibers as the air-exposed rats (CD, Sec. 6.2.3.3). In general, however, the CD (Sec. 9.3.3.2) notes that retarded mucociliary clearance is not observed in animals exposed repeatedly to O_3.

The effects of O_3 on mucociliary clearance in humans have not been well studied, and the results are somewhat conflicting. One study (Foster et al., 1987) reports an O_3-induced increase in particle clearance in subjects exposed to 0.4 ppm O_3 for 2 h, while another study (Gerrity et al., 1993) reports no O_3-induced change in particle clearance with a similar exposure regimen. The discrepancy between these two studies may be explained by differences in exposure protocol, time of particle inhalation, or time of clearance measurement, or by the presence of cough immediately following O_3 exposure, which may have accelerated clearance in the first study (CD, Sec. 7.2.4.7).

Alveolar Macrophage Function. Macrophages represent the first line of defense against inhaled microorganisms and particles that reach the lower airways and alveoli (CD, Sec. 9.3.3.2). Studies in both humans and animals have shown that there is an immediate decrease in the number of macrophages following O_3 exposure. Alveolar macrophages also have been shown to be crucial to the clearance of certain gram-positive bacteria from the lung. Several studies in both humans and laboratory animals also have shown that O_3 impairs the phagocytic capacity of alveolar macrophages, and some studies suggest that mice may be more impaired than rats (Gilmour and Selgrade, 1993; Oosting et al., 1991a). The production of superoxide anion (an oxygen radical used in bacterial killing) by alveolar macrophages also is depressed in both humans and animals (Ryer-Powder et al., 1988; Oosting et al., 1991b) exposed to O_3, and the ability of alveolar macrophages to kill bacteria directly is impaired. Decrements in alveolar macrophage function have been observed in moderately exercising humans exposed to the lowest concentration tested, 0.08 ppm O_3 for 6.6 hrs (Devlin et al., 1991).

Interaction with Infectious Agents. Concern about the effect of O_3 on susceptibility to respiratory infection derives primarily from animal studies in which O_3-exposed mice die following a subsequent challenge with aerosolized bacteria (CD, Sec. 9.3.3.2). Increased mortality of experimental laboratory animals has been shown to be concentration-dependent, and exposure to as little as 0.08 ppm O_3 for 3 hours (Coffin et al., 1967; Coffin and Gardner, 1972; Miller et al., 1978) can increase mortality of mice to a subsequent challenge with streptococcus bacteria. In addition, younger mice are more susceptible to infection than older mice (Gilmour et al., 1991, 1993a,b; Miller et al, 1978); this has been related to increased PGE_2 production in these animals, which likely decreases alveolar macrophage activity.

It has been suggested that impaired alveolar macrophage function is the mechanism likely responsible for enhanced susceptibility to bacteria. However, mortality is not observed with other rodent species, raising the question of whether this phenomenon is restricted to mice. Although both mice and rats show impaired macrophage killing of inhaled bacteria following O_3 exposure, rats mount a faster PMN response to O_3 to compensate for the deficit in alveolar macrophage function. The resulting slower clearance time in mice allows the

streptococcus strain to persist in lung tissue and, subsequently, to elaborate a number of virulence factors that evade secondary host defense and lead to bacterial multiplication and death of the host. Although increased mortality in laboratory animals is not directly relevant to humans, laboratory animals and humans share many host defense mechanisms being measured by mortality in the mouse model. Thus, this category of effect (i.e., decrement in antibacterial defenses) can be qualitatively extrapolated to humans (CD, 9.3.3.2).

With regard to antiviral defenses, a study of experimental rhinovirus infection in susceptible volunteers failed to show any effect of 5 consecutive days of O_3 exposure (0.3 ppm, 6 hrs/day) on the clinical outcome or on host response (Henderson et al., 1993). Studies in which O_3-exposed mice were challenged with influenza virus report conflicting results: some studies show increased mortality, some show decreased mortality, and still others show no change at all. However, even when increased mortality was demonstrated, there was no difference in viral titers in the lung, suggesting virus-specific immune functions were not altered. One animal study (Jakab and Bassett, 1990) reported that even though a 120-day exposure to 0.5 ppm O_3 did not affect the acute course of a viral infection from influenza virus administered immediately before O_3 exposure began, it did enhance postinfluenzal alveolitis and lung parenchymal changes.

Although there is no single experimental human or animal study or group of studies which proves that respiratory infection is worsened by exposure to O_3, taken as a whole, the data suggest that acute O_3 exposures can impair the host defense capability of both humans and animals, primarily by depressing alveolar macrophage function and perhaps also by decreasing mucociliary clearance of inhaled particles and microorganisms. This suggests that humans exposed to O_3 may be predisposed to bacterial infections in the lower respiratory tract. The seriousness of such infections may depend on how quickly bacteria develop virulence factors and how rapidly neutrophils are mobilized to compensate for the deficit in alveolar macrophage function (CD, Sec. 9.3.3.2).

5. Hospital Admissions and Emergency Room Visits

People with preexisting pulmonary disease may be at increased risk to responses associated with short-term O_3 exposures leading to increased hospital admissions and emergency room visits. Furthermore, some individuals with pulmonary disease may have an

inherently greater sensitivity to O_3 (CD, Sec. 9.3.2). Asthmatics characteristically have greater baseline bronchial responsiveness, but, depending on the severity of their disease and clinical status, their FEV_1 can be within the normal range (100 $\pm$ 20% predicted) or may be less than 50% predicted. Patients with chronic obstructive pulmonary disease (COPD) can have FEVs ranging from 30 to 80% of predicted, again depending on disease severity.

Because of their depressed functional state, small absolute changes in lung function of individuals with preexisting pulmonary disease have a larger relative impact than for healthy individuals. For example, a 500-mL FEV_1 decrease in a healthy young man with an FEV_1 of 4,000 mL causes only a 12% decline. In a 55-year-old COPD patient with an FEV_1 that is 50% of predicted, or about 1,670 mL, a 500-mL decline in FEV_1 would result in a 30% decline in FEV_1. Asthmatics with depressed baseline function would have similarly magnified relative responses and, because of increased bronchial responsiveness, may also experience larger changes in airway resistance. Evaluating the intersection of risk factors and exposures is more complex. However, an individual with more severe lung disease is unlikely to engage in heavy exertion and, thus, would be less likely to encounter an effective exposure to O_3.

About 12 million people in the United States (approximately 5% of the population) are estimated to have asthma (National Institutes of Health, 1991). The prevalence is higher among African Americans, older (8- to 11-year-old) children, and urban residents. The annual incidence of hospitalization for all asthmatic individuals is estimated to be about 45 per 1000 (National Institutes of Health, 1991). Although death due to asthma is a relatively infrequent event (i.e., on an annual basis, about one death occurs per 10,000 asthmatic individuals), over 4000 deaths are attributed to asthma each year. Mortality rates are higher among males and are at least 100% higher among nonwhites. In two large urban centers (New York and Chicago), mortality rates from asthma among nonwhites may exceed the city average by up to fivefold (Sly, 1988; National Institutes of Health, 1991; Weiss and Wagener, 1990; Carr et al., 1992). Although some innercity areas may have lower O_3 concentrations than some suburban areas, O_3 concentrations are much higher than those in most rural areas. The impact of ambient O_3 on asthma morbidity and mortality in this apparently susceptible population is not well understood. Those epidemiological studies

which have been conducted to date are subject to confounding factors and have rarely focused on innercity nonwhite asthmatics. Furthermore, controlled human exposure studies of asthmatics typically include mild to moderate asthmatics and also have not dealt specifically with nonwhite asthmatics.

A number of epidemiological studies have shown a consistent relationship between ambient oxidant exposure and acute respiratory morbidity in the population. Decreased lung function and increased respiratory symptoms, including exacerbation of asthma, occur with increasing ambient O_3, especially in children. Modifying factors, such as ambient temperature, aeroallergens, and other copollutants (e.g., particles) also can contribute to this relationship. Ozone air pollution can account for a portion of summertime hospital admissions and emergency room visits for respiratory causes. Studies conducted in various locations in the eastern United States (Cody et al., 1992; Thurston et al., 1992, 1994; White et al., 1994; Schwartz 1994a,b,c) and Canada (Bates et al., 1990; Lipfert and Hammerstrom, 1992; Burnett et al., 1994; Delfino et al., 1994a,b) consistently have shown a relationship with increased incidence of visits and admissions, even after controlling for modifying factors, as well as when considering only concentrations <0.12 ppm O_3. It has been estimated from these studies that O_3 may account for roughly one to three excess summertime respiratory hospital admissions per hundred parts per billion O_3, per million persons. In Section V-H on ozone health risk assessment, Figure V-17 summarizes the excess annual hospital admissions of asthmatics attributable to O_3 exposure for alternative air quality scenarios and provides "effect size" and "relative risk" estimates.

The association between elevated ambient O_3 concentrations during the summer months and increased hospital visits and admissions for respiratory causes has a plausible biologic basis in the physiologic, symptomatic, and field study evidence discussed earlier. Specifically, increased airway resistance, bronchial responsiveness, susceptibility to respiratory infection, airway permeability, and incidence of asthma attacks and airway inflammation suggest that ambient O_3 exposure could be a cause of the increased hospital admissions, particularly for asthmatics (CD, Sec. 9.3.2).

6. Daily Mortality

Several studies published during the 1950's (California Dept. of Public Health, 1955, 1956, 1957; Mills, 1957a,b), the 1960's (Tucker, 1962; Massey et al., 1961; Mills 1960; Hechter and Goldsmith, 1961), and the 1970's (Biersteker and Evendijk, 1976) have suggested a possible association of O_3 or oxidants with human mortality. Most of these studies were conducted using data from Los Angeles, CA, and all were flawed in some way, which prevented drawing any definitive conclusions in earlier criteria documents.

Several daily mortality studies published more recently have provided additional, though limited, evidence of the association between O_3 and daily mortality. The Shumway et al. (1988) analysis of 1970 to 1979 LA County mortality data indicated that disease factors and other pollutants dominate the seasonal cycles in mortality in LA. However, the Kinney and Ozkaynak (1991) reanalysis of the Shumway et al. (1988) data concluded that O_3 explained a small, but statistically significant, portion of day-to-day variations in total mortality in that city over a 10-year period. The authors of the reanalysis did recognize that the possible mechanism linking O_3 with mortality is speculation based on known acute pulmonary effects. They further emphasize that, although statistically significant associations have been detected among mortality and environmental variables, one can not conclude with complete confidence that such associations are causal based on results from an observational study.

Total daily human mortality data in Detroit, MI during the period from 1973 to 1982 were analyzed by Schwartz (1991) to investigate the effects of particulate matter on mortality and concluded that O_3 was "highly insignificant as a predictor of daily mortality." The CD (Sec. 7.4.1.3) concluded that the poor documentation of the mortality-O_3 modeling, especially regarding the lack of model specification details or model coefficient intercorrelations, makes the author's statement very difficult to evaluate. Finally, Dockery et al. (1992) conducted an analysis of total daily mortality in St. Louis, MO and Kingston-Harriman, TN during the period September 1985 to August 1986, also with the intention of assessing the effects of particulate matter on mortality. Although the Dockery et al. (1992) study showed no association between O_3 and mortality, this may have been in part a result of the particular methodological and exposure characteristics of the study vis-a-vis identification

of O_3 health effects. Therefore, the CD (Sec. 9.6) concludes that although an association between ambient O_3 exposure in areas with very high O_3 levels and daily mortality has been suggested, the strength of any such association remains unclear at this time.

7. Acute Inflammation and Respiratory Cell Damage

Ozone has the potential to induce inflammatory responses throughout the respiratory tract, including the nasopharyngeal region and the lungs. Humans and laboratory animals exposed to O_3 can develop inflammation and increased permeability in the nasal passages. A positive correlation was reported between nasal inflammation in children and measured ambient O_3 concentrations. Experimental studies of rats suggest a potential competing mechanism between the nose and lung, with inflammation occurring preferentially in the nose at lower O_3 concentrations and shifting to the lung at higher concentrations. It is unclear if this represents a peculiarity of rats or is a more general phenomenon (CD, Sec. 9.3.3.1).

In general, respiratory inflammation can be considered to be a host response to injury and indicators of inflammation as evidence that respiratory cell damage has occurred. Inflammation induced by exposure of humans to O_3 can have several potential outcomes: (1) inflammation induced by a single exposure (or even several exposures over the course of a season) can resolve entirely; (2) repeated acute inflammation can develop into a chronic inflammatory state; (3) continued inflammation can alter the structure and function of other pulmonary tissue, leading to disease processes such as fibrosis; (4) inflammation can interfere with the body's host defense response to particles and inhaled microorganisms, particularly in potentially vulnerable populations such as children and older individuals; and (5) inflammation can interfere with the lung's response to other agents such as allergens or toxins. Except for outcome (1), the possible chronic responses have not been demonstrated with inflammation induced by exposure of humans to O_3. It is also possible that the profile of response can be altered in persons with preexisting pulmonary disease (e.g., asthma, COPD) or smokers.

The recent use of bronchoalveolar lavage (BAL) as a research tool in humans has afforded the opportunity to sample cells and fluid from the lung and lower airways of humans exposed to O_3 and to ascertain the extent and course of inflammation and its constitutive elements. Several studies (Aris et al., 1993a,b; Schelegle et al., 1991; Koren et

al., 1989a,b; Devlin and Koren, 1990; McGee et al., 1990; Koren et al., 1991; Devlin et al., 1995; Hazucha et al., 1995; Seltzer et al., 1986) have shown that humans exposed for short-term periods (1- to 3- hr) to 0.2 to 0.6 ppm O_3 had O_3-induced inflammation, cell damage, and altered permeability of epithelial cells lining the respiratory tract (allowing components from plasma to enter the lung). The lowest concentration of O_3 tested, 0.08 ppm for 6.6 hours with moderate exercise, also induced small but statistically significant increases in these endpoints (Devlin et al., 1990, 1991; Koren et al., 1991).

Polymorphonuclear leukocytes (PMNs), generally considered to be the hallmark of inflammation, make up 8 to 10% of recovered BAL cells in individuals exposed for 2 hrs to 0.4 to 0.6 ppm O_3 (Seltzer et al., 1986). This is a 5- to 8-fold increase in PMNs compared to similar individuals exposed to clean air, who typically have 1 to 2% PMNs in their BAL fluid. Asthmatic individuals generally have baseline levels of PMNs which do not differ significantly from those of healthy individuals, but PMN levels can increase following allergen bronchoprovocation (CD, Sec. 9.3.3.1).

Exposures of animals to O_3 for periods $\leq$ 8hr also result in cell damage, inflammation, and altered permeability, although, in general, higher O_3 concentrations are required to elicit a response equivalent to that of humans. Because humans were exposed to O_3 while exercising and most animal studies were done at rest, differences in ventilation likely play a significant role in the different response of humans and rodents to the same O_3 concentration. Studies in which laboratory animals were exposed at night (during their active period) or in which ventilation was increased with CO_2 tend to support this idea (CD, Sec. 9.3.3.1).

Studies utilizing BAL techniques sample only free or loosely adherent cells in the lung; thus, it is possible that cellular changes have occurred in the interstitium that are not reflected in BAL studies, or that BAL changes exist in the absence of interstitial changes. However, morphometric analyses of inflammatory cells present in lung and airway tissue sections of animals exposed to O_3 are in general agreement with BAL studies. Ozone exposures of $\leq$ 8 hrs cause similar types of alterations in lung morphology in all laboratory animal species studied. The most affected cells are the ciliated epithelial cells of the airways and Type 1 cells in the alveolar region. The centriacinar region (CAR), the junction of the

conducting airways and gas exchange region, is a primary target in all species studied, possibly because it receives the greatest dose of O_3 delivered to the lower respiratory tract. Sloughing of ciliated epithelial and Type 1 cells occurs within 2 to 4 hrs of exposure of rats to 0.5 ppm O_3.

Findings from human and animal studies show that the O_3-induced inflammatory response occurs rapidly and persists for at least 24 hrs. Increased levels of neutrophils and protein are observed in the BAL fluid within 1 hr following a 2-hr exposure of humans to O_3 and continue for at least 20 hrs. The kinetics of response during this time have not been well studied in humans, although a single study shows that neutrophil levels are higher at 6 hours postexposure than at 1 or 20 hrs in different individuals. Several animal studies suggest that neutrophil and BAL protein levels peak 12 to 16 hrs after an acute O_3 exposure and begin to decline by 24 hrs, although some studies report detectable BAL neutrophils even 36 hrs after exposure. It is also clear that in humans the pattern of response differs for different inflammatory mediators. Mediators of acute inflammation, such as interleukin-6 (IL-6) and prostaglandin-E_2 (PGE_2), are more elevated immediately after exposure; whereas mediators that potentially could play a role in resolving inflammation, such as fibronectin and plasminogen activator, are preferentially elevated 18 hours after exposure. The rapidity with which cellular and biochemical mediators are induced by O_3 makes it conceivable that some of them may play a role in O_3-induced changes in lung function. Indeed, there is some evidence that BAL PGE_2 levels are correlated with decrements in FEV_1, and anti-inflammatory medications that block PGE_2 production also reduce or block the spirometric responses to O_3. Although earlier studies suggested that O_3-induced PMN influx might contribute to the observed increase in airway hyperreactivity, animal studies show that when neutrophils are prevented from entering the lung, O_3-induced hyperreactivity or increases in many inflammatory mediators still occur. In addition, studies in which anti-inflammatory drugs are used to block O_3-induced lung function decrements still show increases in neutrophils and most other inflammatory mediators (although PGE_2 is not increased) (CD, Sec. 9.3.3.1).

It is the view of staff and of medical experts consulted that the repeated acute inflammatory response and morphological changes discussed above is potentially a matter of

public health concern; however, it is also recognized that most, if not all, of these effects have begun to resolve in most individuals within 24 hrs if the exposure to O_3 is not repeated. Of possibly greater public health concern is the potential for chronic respiratory damage which could be the result of repeated O_3 exposures occurring over a season or a lifetime. The evidence for these chronic effects is discussed in the following section.

8. Chronic Respiratory Damage

To evaluate the impact on the human respiratory system of long-term exposures to O_3, it has been necessary for researchers to utilize the results from both epidemiology and animal toxicology studies. There are clear limitations in using these approaches which cannot be fully overcome when compared to controlled-exposure human experimental studies. Epidemiology studies do not provide clear causal relationships due to the presence of confounding variables (e.g., heat, humidity, other pollutants); however, the results can provide associations which may suggest causal relationships. Animal toxicology studies, though limited by species sensitivity and dosimetry differences between humans and experimental animals, can offer controlled experimental conditions for chronic exposures and thereby provide evidence of causal relationships. Dosimetric extrapolation techniques have improved dose-target tissue relationships, but lack of a full understanding of species sensitivity differences between humans and animals limits the extent to which results of toxicology data can be extrapolated to human health effects.

Epidemiologic studies (Abbey et al., 1993; Detels et al., 1991; Euler et al., 1988; Hodgkin et al., 1984; Schwartz, 1989; Stern et al., 1989, 1994; Portney and Mullahy, 1990; Schmitzberger et al., 1993) that have investigated potential associations between long-term O_3 exposures and chronic respiratory effects in humans thus far have provided only suggestive evidence that such a relationship exists. Most studies investigating this association have been cross-sectional in design and have been compromised by incomplete control of confounding variables and inadequate exposure information. Other studies have attempted to follow variably exposed groups prospectively. Studies have been conducted in Southern California (Detels et al., 1991) and in Canada (Stern et al., 1989, 1994) designed to compare lung function changes over several years between populations living in communities with high and low oxidant ambient air levels. While recognizing that pollution levels have improved markedly in Southern California during the past several decades, the findings still suggest

small, but consistent, decrements in lung function among inhabitants of the more communities which have been highly polluted; however, associations between O_3 and other copollutants and problems with study population loss have reduced the level of confidence in these conclusions. Another study (Abbey et al., 1993), reporting associations between O_3 and the incidence and severity of asthma in Seventh Day Adventists over a decade, had similar results, but were even less suggestive due to the colinearity of O_3 with other air pollutants. This is largely due to the difficulty of partitioning effects between O_3 and particles. Nevertheless, in all of the studies assessing lung function, the pattern of dysfunction associated with the long-term O_3 exposure has been consistent with the functional and structural abnormalities seen in laboratory animals, as discussed in the CD (Sec. 9.4.2 and Sec. 6.5.3.3).

The advantage of laboratory animal studies is the ability to examine closely the distribution and intensity of the O_3-induced morphologic changes that have been identified throughout the respiratory tract (CD, Sec. 6.2.4 and Sec. 9.4.2). Indeed, cells of the nose, like the distal lung, clearly are affected by O_3. Perhaps of greater health concern are the "lesions[3]" that occur in the small airways and in the centriacinar regions (CAR) of the lung where the alveoli meet the distal airways, as pictured in Figure V-3 (CD, Figure 9-12). Altered function of the distal airways, the proximal conduits of air to the gas-exchange regions, can result in reduced communication of fresh air with the alveoli and air-trapping. In fact, "lesions" found in animals following chronic O_3 exposures are reminiscent of the earliest "lesions" found in respiratory bronchiolitis, some of which may progress to fibrotic lung disease (Kuhn et al., 1989; King, 1993).

"Lesions" in the CAR are one of the hallmarks of O_3 toxicity, having been well established. The study of Chang et al. (1992) exposed rats to an urban pattern of O_3 (13 hr 0.06 ppm background, 7 days/week, on which were superimposed 9-hr peaks, 5 days/week, slowly rising to 0.25 ppm) for 78 weeks and made periodic examinations of the CAR

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[3] During the March 1995 CASAC meeting, and in subsequent written comments, substantial disagreement was expressed among Panel members regarding the use of the term "lesion." Some believe use of the term implies more serious damage than has been observed for O_3 exposures, while other Panel members believe "lesion" is an appropriate term to describe O_3-induced morphological abnormalities. The CD (Sec. 6.2.4.1) describes and discusses these degenerative changes, referred to as "lesions" for purposes of the CD and this Staff Paper.

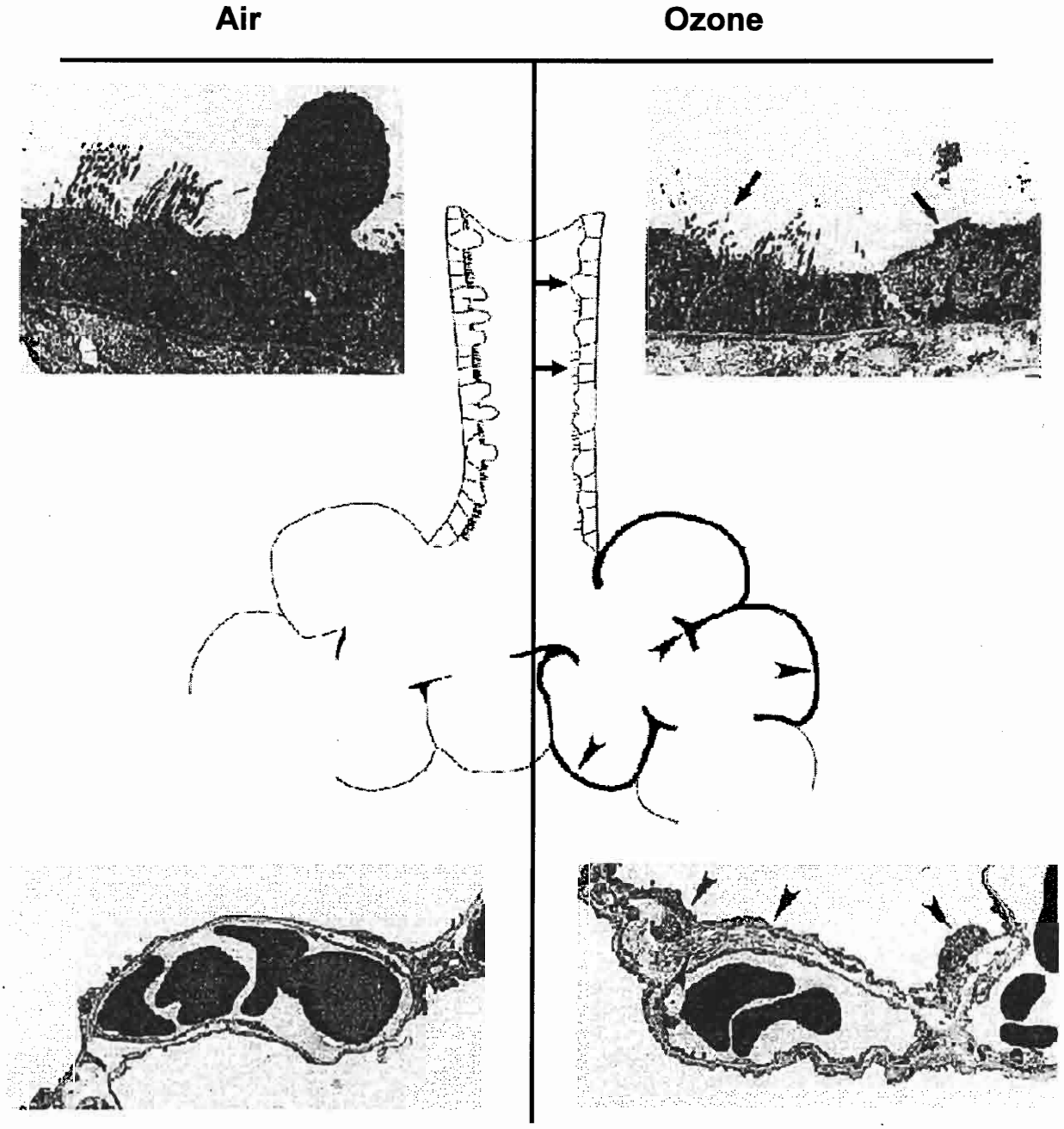


FIGURE V-3. A SUMMARY OF MORPHOLOGIC LESIONS FOUND IN THE TERMINAL BRONCHIOLES AND THE CENTRIACINAR REGION (CAR) OF THE LUNG FOLLOWING EXPOSURE OF LABORATORY RATS TO FILTERED AIR OR A SIMULATED AMBIENT PATTERN OF O_3 FOR UP TO 78 WEEKS. IN THE TERMINAL BRONCHIOLE, SIZES OF THE DOME OF CLARA CELLS BECAME SMALLER WITH O_3 EXPOSURE, AND THE NUMBER OF CILIA IS REDUCED (ARROWS). IN THE CAR, THE EPITHELIUM BECOMES THICKER, AND ACCUMULATION OF COLLAGEN FIBERS OCCURS (ARROW HEADS).
Source: Chang et al. (1992); CD, Figure 9-12.

tracheobronchial and proximal alveoli by TEM morphometry during and after exposure. In general, Chang et al. (1992) found (1) changes in Type 1 and Type 2 cell volume which returned to normal 17 weeks post exposure, (2) epithelial and endothelial basement membrane were thickened and accompanied by increased collagen fibers at 17 weeks post exposure, and (3) in the tracheobronchial regions, surface areas of ciliated and nonciliated cells decreased during exposure. In a related study of identically exposed groups of rats, Tepper et al. (1994) reported (1) increases in expiratory resistance suggesting central airway narrowing after 78 weeks exposure, (2) tidal volumes reduced at all evaluation times during the exposure, and (3) breathing frequency reduced though no single evaluation time was significant. In another related study with similar protocol, Costa et al. (1995) reported reduced lung volume, which is consistent with a "stiffer" lung (i.e., restrictive lung disease).

As shown in Figure V-4 (CD, Figure 9-13), the temporal pattern of effects during and after a chronic exposure is complex. During the early days of exposure, the end-

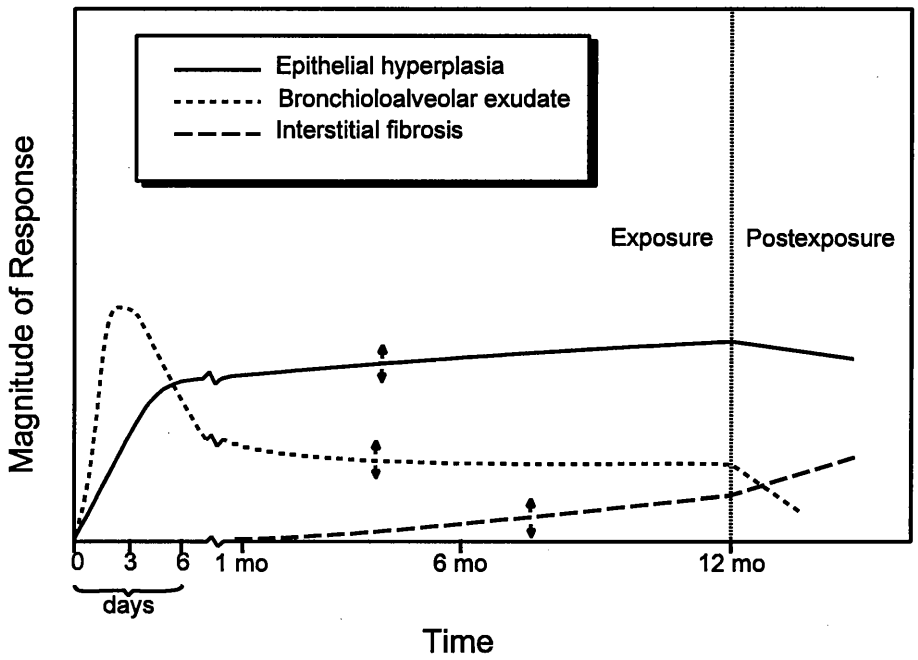


FIGURE V-4. SCHEMATIC COMPARISON OF THE DURATION-RESPONSE PROFILES FOR EPITHELIAL HYPERPLASIA, BRONCHIOLOALVEOLAR EXUDATION, AND INTERSTITIAL FIBROSIS IN THE CENTRIACINAR REGION OF LUNG EXPOSED TO A CONSTANT LOW CONCENTRATION OF OZONE.

Source: Dungworth (1989); CD, Figure 9-13.

airway lumenal and interstitial inflammation peaks, and, thereafter, appears to subside at a lower plateau of activity sometimes referred to as a "smoldering lesion". Several cytokines remain elevated beyond the apparent adaptation phase of the response and may be linked conceptually to the development of chronic "lesions" in the distal lung. However, a clear association of these BAL-derived mediators and cells with long-term toxicity has yet to be demonstrated (CD, Sec. 9.4.2).

A multicenter chronic study, supported by the National Toxicology Program (NTP) and the Health Effects Institute (HEI), involved numerous researchers and laboratories (Last et al., 1994; Szarek, 1994; Radharkrishnamurthy, 1994; Parks and Roby, 1994; Harkema and Mauderly, 1994; Harkema et al., 1994; Chang et al., 1995; Pinkerton et al., 1995; Catalano et al., 1995a,b). This NTP (1995) study further illustrates some of the complex interrelationships among the structural, functional, and biochemical effects. These three health endpoints were evaluated in a collaborative project using rats exposed 6 hrs/day, 5 days/week for 20 months to 0.12, 0.50, 1.00 ppm O_3. Although lung biochemistry and structure were affected at 0.5 ppm and 1.00 ppm but not at 0.12 ppm O_3, there were no observed effects on pulmonary function at any exposure level.

Combined analyses of the NTP (1995) collaborative studies showed that 0.5 ppm and 1.00 ppm O_3 caused a variety of structural and biochemical effects. Exposures to 0.12 ppm O_3 caused no major effects, although a few specific endpoints were altered. Hallmarks of chronic rhinitis (e.g., inflammation, mucous cell hyperplasia, decreased mucous flow) were observed in focal regions of the nasal cavity. Structural and biochemical changes included some, but not many, hallmarks of airway disease. Typical O_3-induced changes (e.g., bronchiolarization, increased interstitial matrix) observed in the tracheobronchial region and in the CAR were characteristic of centriacinar fibrosis; however, diffuse pulmonary fibrosis was not observed.

Trends for centriacinar fibrosis, airway disease, and chronic rhinitis were examined by Catalano et al. (1995a) for 10, 18, and 3 endpoints, respectively, from the individual NTP (1995) studies. A statistically significant trend was noted for the association between chronic rhinitis and increasing O_3 concentration. The differences between control and exposed rats were statistically significant at 0.50 ppm and 1.00 ppm O_3. Marginally significant and

significant trends were found for the association between centriacinar fibrosis or airway disease and increasing O_3 concentration; however, no statistically significant differences were found between control and O_3-exposed rats (CD, Sec. 6.5.3.3).

Studies of prolonged O_3 exposures in monkeys and rats reveal generally similar morphologic responses, although it appears that the monkey exhibits somewhat more tissue injury than does the rat under roughly similar exposure conditions (CD, Chapter 8). Interspecies comparisons of dosimetric data indicate that the monkey, with its similarity to the human in distal airway structure, provides data that may best reflect the potential effects of O_3 in humans exercising out of doors. As such, monkeys exposed to O_3 at 0.15 ppm for 8 h each day for 6 to 90 days exhibit significant distal airway remodeling. Rats show similar but more modest changes at 0.25 ppm O_3 after exposures of longer duration, up to 18 mo and beyond (near-lifetime). The chronic distal lung and airway alterations appear consistent with incipient peribronchiolar fibrogenesis within the interstitium. Attempts to correlate functional deficits have been variable, perhaps due in part to the degree and distribution of the "lesions" and the general insensitivity of most measures of the distal lung function. The interstitial changes may progress, however. Moreover, one recent primate study revealed evidence that intermittent challenge with a pattern of O_3 exposure more reflective of seasonal episodes, with extended periods of clean air in between extended periods of O_3, actually leads to greater injury. The reasons for this are unclear but may relate to the known loss of tolerance that occurs in both humans and animal test species with removal of the oxidant burden.

Probably the most provocative, albeit preliminary, evidence of possible pollutant effects in the population is offered by Sherwin (1991) and Sherwin and Richters (1991). They performed a pathological evaluation of the lungs from 107 Los Angeles County residents (15 to 25 years of age), who had a sudden death without disease or lung trauma. Sherwin (1991) reported that the odds ratio for severe CAR disease (defined as the extension of a respiratory bronchiolitis into the proximal acinar structures) in subjects living in metropolitan Los Angeles versus those living in other cities in Los Angeles County was 4.0 (95% confidence limit, 1.4 to 11.3). Unfortunately, no exposure data or lifetime residence data, no smoking histories, no cotinine results, nor occupational histories were available.

The lack of a smoking history on subjects is of critical importance because respiratory bronchiolotis has been shown to be an early pathologic change found in the pulmonary airways of young smokers (CD, Sec. 7.4.2.2.) Furthermore, the subjects were mainly of low socioeconomic status and only 10 were female, and the observation is limited by a lack of quantitative morphometry of lung specimens and by the lack of a control group from an ambient environment with low oxidant pollution. (Many of these limitations should be addressed in research which is currently being planned by the USEPA National Health and Environmental Effects Research Laboratory.) Therefore, although the Sherwin (1991) observation is of great interest, particularly with regard to other primate data which show O_3-associated effects in the CAR, the results are not of particular value in determining human exposure levels for O_3 which might induce chronic respiratory disease, nor do they establish a causal relationship between the oxidant environment found in metropolitan Los Angeles and the pathologic effects observed by Sherwin (1991) (CD, Sec. 7.4.2.2).

In summary, the collective data on chronic exposure to O_3 garnered in animal exposure and human population studies have many ambiguities. It is clear that the distribution of the O_3 "lesions" is roughly similar across species (e.g., monkeys, rats, mice). These responses are concentration dependent (and perhaps time or exposure-pattern dependent). Under certain conditions, some of these structural changes may become irreversible. It is unclear whether ambient exposure scenarios encountered by humans result in similar "lesions." Furthermore, it is highly uncertain whether there are resultant functional or impaired health outcomes in humans chronically exposed to O_3, particularly because the human exposure scenario involves much longer-term exposures than can be investigated in the laboratory. The epidemiology studies of lung function change generally parallel those of the animal studies, but they lack good information on individual O_3 exposure and are frequently confounded by personal or copollutant variables (CD, Sec. 9.4.2). In summary, the animal toxicology data discussed above, and in the CD (Chapter 6), provides a biologically plausible basis for considering the possibility that repeated inflammation associated with exposure to O_3 over a lifetime may result in sufficient damage to respiratory tissue such that individuals later in life may experience a reduced quality of life, although such relationships remain highly uncertain.

9. Genotoxicity and Carcinogenicity

Numerous in vitro exposure studies suggest that O_3 has either weak or no potential to cause mutagenic, cytogenetic, or cellular transformation effects. Most of these experiments utilized high concentrations of O_3 (>5.0 ppm). Because of the exposure systems used, there are unknowns about uncertainties regarding the formation of artifacts and the dose of O_3. Therefore, these studies are not very useful in health assessment. Cytogenetic effects have been observed in some, but not all, laboratory animal and human studies of short-term O_3 exposure. However, well-designed human clinical cytogenetic studies were negative.

Until recently, in vivo exposure studies of carcinogenicity, with and without co-exposure to known carcinogens, were either negative or ambiguous. A well-designed cancer bioassay study has recently been completed by the National Toxicology Program (NTP, 1995) using male and female Fischer 344/N rats and B6C3F_1 mice. Animals were exposed for 2 years to 0.12, 0.5, and 1.0 ppm O_3 (6 h/day, 5 days/week). A similar lifetime exposure was conducted, but 0.12 ppm was not used. The NTP (1995) evaluated the weight-of-evidence for this study; they found "no evidence" of carcinogenicity in rats but reported "equivocal evidence" of carcinogenicity in O_3-exposed male mice and "some evidence" of carcinogenic activity in one strain of O_3-exposed female mice. The increases in adenomas and carcinomas were observed only in the lungs. There was no concentration response. In the male mice, the incidence of neoplasms in the 2-year study was not elevated significantly by O_3 and was within the range of historical controls. Also, the lifetime exposure did not increase significantly the incidence of neoplasms, even though the incidence of carcinomas was increased. In the female mice, a 2-year (but not lifetime) exposure to 1.0 ppm O_3 only increased the incidence of animals with neoplasms. When the female mouse data from the two exposure regimens (at 1.0 ppm) were combined, there was a statistically significant increase (almost double) in neoplasms. In a companion study, male rats were treated with a tobacco carcinogen and exposed for 2 years to 0.5 ppm O_3. Ozone did not affect the response and, therefore, had no tumor promoting activity.

In summary, only long-term exposure to a high concentration of O_3 (1.0 ppm) has been shown to evoke a limited degree of carcinogenic activity in B6C3F_1 mice. Rats were unaffected. Furthermore, there was no concentration response, and there is inadequate

information from other research to provide mechanistic support for the finding in mice. For these reasons, the staff believes it is inappropriate to extrapolate these mouse data to humans.

D. Factors Modifying Acute Human Response to Ozone

There are several factors which have been identified as potentially affecting human susceptibility to O_3 exposure by altering acute physiological susceptibility. The more significant of these factors are exertion (e.g., exercise, manual labor), preexisting disease, age, gender, ethnicity/race, smoking status, environmental factors. Although most of these factors have not been addressed adequately in clinical studies in order to draw definitive conclusions, preliminary observations have been made regarding each of these potential modifiers of response. A thorough discussion is presented in Section 7.2 of the CD.

1. Exertion and Ventilation

Exertion resulting in an increased minute ventilation ($\dot{V}_E$) is a factor which increases O_3 sensitivity of most humans at any elevated O_3 concentration. This is in part due to the fact that at higher $\dot{V}_E$ there is an increase in O_3 dose received by the lungs. It is also due to the deeper penetration of O_3 into more peripheral regions of the lungs, which are more sensitive to acute O_3 response and injury. This provides general support for the hypothesis that increasing the level of exertion for most individuals increases the impact of a given concentration of O_3. Furthermore, research has shown that respiratory effects are observed at lower O_3 concentrations if the level of exertion is increased and/or the duration of exertion is extended. An increased level of exertion can cause an individual, who has a respiratory system which is highly responsive to O_3, to experience lung function impairment and symptoms sufficient to curtail activity, even though the individual is otherwise healthy.

Representative activities and associated ventilation rates are summarized in Table V-3 for varying levels of exertion. While the table identifies only a few of the many activities in which individuals engage, it is intended to provide the reader a sense of the relationship between level of exertion, ventilation rate, and type of activity.

TABLE V-3. ESTIMATED MINUTE VENTILATION RATES AND REPRESENTATIVE ACTIVITIES ASSOCIATED WITH VARYING LEVELS OF EXERTION[a]

Level of Exertion	Minute Ventilation L/min	Representative Activities[b]
Light	12-16	Level walking at 2 mph; washing clothes
Light	17-23	Level walking at 3 mph; bowling; scrubbing floors
Moderate	23-30	Dancing; pushing wheelbarrow with 15-kg load; simple construction; stacking firewood
Moderate	29-38	Easy cycling; pushing wheelbarrow with 75-kg load; using sledgehammer
Moderate	35-46	Climbing stairs; playing tennis; digging with spade
Heavy	42-55	Cycling at 13 mph; walking on snow; digging trenches
Heavy Very Heavy Very Heavy	52-57 62-79 73-93	Cross-country skiing; rock climbing; stair climbing with load; playing squash and handball; chopping with axe
Very Heavy	89-110	Level running at 10 mph; competitive cycling
Severe	107-132	Competitive long distance running; cross-country skiing

[a]See text of Criteria Document (U.S. EPA, 1986, pp. 10-13 to 10-15) for discussion.
[b]Adapted from Astrand and Rodahl (1977).

2. Preexisting Disease

Controlled studies on mild asthmatics suggest that they have similar lung volume responses but greater airway resistance changes to O_3 than nonasthmatics. Furthermore, limited data from studies of moderate asthmatics suggest that this group may have greater lung volume responses than nonasthmatics. Daily life studies reporting an exacerbation of asthma and decrease in peak expiratory flow rates, particularly in asthmatic children, appear to support the controlled studies; however, those studies are confounded by temperature, particle or aeroallergen exposure, and asthma severity of the subjects or their medication use. In addition, field studies of summertime daily hospital admissions for respiratory causes show a consistent relationship between hospital admissions for asthmatics and ambient levels of O_3 in various locations in the Northeastern United States, even after controlling for independent contributing factors.

Other population groups with preexisting limitations in pulmonary function and exercise capacity (e.g., chronic obstructive pulmonary disease, ischemic heart disease) would be of primary concern in evaluating the health effects of O_3. Unfortunately, not enough is known about the responses of these individuals to make definitive conclusions regarding their relative sensitivity to O_3. Indeed, functional effects in these individuals with reduced lung function may have greater clinical significance than comparable changes in healthy persons.

3. Age, Gender, Ethnic, and Tobacco Smoke Factors

Age Factors. Age differences as a factor influencing response to O_3 are not yet fully understood. This is in part due to the fact that most of the O_3 controlled-exposure studies have been conducted with young adults rather than with children or older subjects. However, there is a growing body of evidence, including clinical, field, and epidemiology studies, which suggests that age plays a role in determining sensitivity to O_3. Based on the available data, it appears that children respond to low-level O_3 exposures in a manner comparable to that of young adults, albeit without symptoms, while older persons exhibit a decreased sensitivity relative to young adults (CD, Sec. 9.6). The lack of symptoms in children and reduced sensitivity in the elderly could lead to an increased risk of an individual receiving a higher O_3 dose. This increased risk of O_3 exposure and dose is a direct result of children and the elderly not taking mitigating behavior to avoid exposure because they do not

experience respiratory symptoms; however, this hypothesis has not been tested and has not been demonstrated at this time.

Gender Factors. During the previous review of O_3 NAAQS, so few human or animal studies had been conducted using any female subjects in controlled-exposure studies of O_3 that few substantive conclusions could be drawn regarding gender differences. Although there are more data on female subjects than was the case previously, these new data have not yet provided conclusive evidence that men and women respond differently to O_3. Thus, the question as to whether there is a difference between males and females in respiratory susceptibility to O_3 remains unresolved. Furthermore, it can be stated that if gender differences do exist for respiratory susceptibility to O_3, they are not based on hormonal changes, differences in lung volume, or ratio of forced vital capacity (FVC) to $\dot{V}_E$ (CD, Sec. 1.7).

Ethnicity Factors. In studies thus far conducted, the lung function decrements in African-Americans were not statistically significantly greater than in other groups at all concentrations tested. Even though these results can be considered suggestive of ethnic differences, further research, particularly on non-white asthmatics, must be conducted before ethnicity can be established as a clear factor in determining pulmonary responsiveness to O_3 (CD, Sec. 7.2.1.3).

Tobacco Smoke. Results of several early studies, which compare sensitivity of individuals voluntarily exposed to tobacco smoke (i.e, smokers) versus sensitivity of those who have not been exposed to tobacco smoke, suggest that smokers are less responsive to O_3 than nonsmokers. Although data on O_3 susceptibility of both active and passive smokers remains limited, recent studies indicate that cessation of exposure to tobacco smoke leads to improved baseline pulmonary function and possibly a return to O_3 susceptibility (CD, Sec. 7.2.1.3).

4. Interactions with Other Pollutants

In general, controlled human studies of O_3 mixed with other pollutants show no more than an additive response with symptoms or spirometry as an endpoint. This applies to O_3 in combination with nitrogen dioxide (NO_2), SO_2, sulfuric acid (H_2SO_4), nitric acid (HNO_3), or carbon monoxide (CO). Indeed, at the levels of copollutants used in human exposure

studies, the responses can be attributed primarily to O_3. In one study, exposure to O_3 increased airway responsiveness to SO_2 in asthmatics. Similarly, other pollutants (e.g., particulate matter) that may increase airway responsiveness could augment the effect of O_3 on airway responsiveness.

The relatively large number of animal studies of O_3 in mixture with NO_2 and H_2SO_4 shows that additivity, synergism, and antagonism can result, depending on the exposure regimen and the endpoint studied. The numerous observations of synergism are of concern, but the interpretation of most of these studies relative to the real world is confounded by unrealistic exposure designs. For example, ambient concentrations of O_3 often were combined with levels of copollutants substantially higher than ambient, creating the possibility that mechanisms of toxicity unlikely in the real world contributed to the experimental outcome. Nevertheless, the data support a hypothesis that coexposure to pollutants, each at innocuous or low-effect levels, may result in effects of significance.

E. Sensitive Population Groups

Several characteristics which influence the extent to which an individual or population group may show increased sensitivity to O_3 have been discussed in the CD (p. 9-41). These individual or group characteristics are based on: 1) biological responses to O_3; 2) physiological status; 3) activity patterns; 4) exposure history; and 5) personal factors such as age, gender, social, ethnic, cultural, and nutritional status.

1. Active ("Exercising") Individuals

One large group of individuals at risk to O_3 exposure consists of those healthy children, adolescents, and adults who engage in outdoor activities involving exertion (i.e., "exercising" individuals) during summer daylight hours. This conclusion is based on a large number of controlled O_3-exposure human experimental studies, which have been conducted on healthy, non-smoking, exercising adults and children (ages 8 to 45). These studies also have demonstrated wide variability among subjects in sensitivity to O_3, although factors contributing to this variability are not well understood.

2. Individuals with Preexisting Respiratory Disease

There is limited evidence from human controlled exposure studies to suggest that mild asthmatics have greater changes in airway resistance following O_3 exposure than nonasthmatics but have similar lung volume responses; however, moderate asthmatics appear to have greater lung volume responses than nonasthmatics. Support for considering asthmatics to be at increased risk to O_3 exposure also comes from studies of hospital admissions for respiratory causes which show a consistent relationship between asthma and ambient O_3 levels in the northeastern U.S., even after controlling for independent contributing factors. Studies of asthmatic children which report exacerbation of asthma and decreased peak expiratory flow rates seem to provide some further evidence of asthmatics being at risk, but these studies are confounded by variables such as temperature, particle or aero allergen exposure, severity of asthma, and medication use (CD, Sec. 9.6).

Although there are limited data on individuals with preexisting respiratory disease or other limitations on their pulmonary function and exercise capacity (e.g., those with chronic obstructive pulmonary disease, ischemic heart disease), there is insufficient information at this time to draw any clear conclusions about their susceptibility to O_3 relative to other individuals. The major reason individuals with preexisting respiratory disease may be of concern is the likelihood that decrements in lung function or exercise capacity may have greater clinical importance to the individual than similar changes in healthy persons.

3. Other Population Groups

Several population groups identified in the CD (Sec. 9.6) as not providing compelling evidence to suggest that they are more responsive than the normal, healthy population include: the young and elderly, males and females, ethnic and racial groups, and individuals with vitamin E deficiency or other nutritional deficiencies. Thus, in addition to the more speculative at-risk status of those individuals with respiratory disease or pulmonary deficiency, the CD (Sec. 9.6) identifies only "exercising" or active healthy and asthmatic individuals, including children, adolescents, and adults as having demonstrated susceptibility to O_3.

F. Adverse Respiratory Effects of Ozone Exposures

As discussed in Chapter II of this Staff Paper, setting a primary O_3 NAAQS involves assessing protection of public health based on consideration of sensitive populations at risk, including factors such as the nature, severity, and frequency of O_3-induced health effects involved. This section focuses on the nature, severity, and frequency of specific O_3-induced health effects in order to provide a basis for judgments regarding physiological changes that become sufficiently severe to adversely affect the health status of those individuals experiencing such effects. In considering populations at risk, staff recognizes that there is wide variability in the severity of response to O_3 among both healthy individuals and those with impaired respiratory systems. Individual sensitivity of healthy persons to O_3 and the extent to which impaired respiratory systems amplify the impact of various effects in individuals with asthma and chronic obstructive pulmonary disease (COPD) should be taken into account in making judgments about the adversity of O_3 effects. These judgments about individual adverse effects are put into broader context in the following sections on exposure and risk analysis. This broader context includes consideration, to the extent possible, of size of the sensitive populations potentially at risk for various effects, and the kind and degree of uncertainties inherent in assessing such risks in order to form judgments about the various levels of risk and adequacy of public health protection afforded by alternative NAAQS.

In this section, staff has attempted to identify and characterize the current understanding as well as the divergence of opinion within the scientific community as to what effects and degrees of response might be regarded as adverse health effects associated with exposure to O_3. This section presents staff's views on this issue, taking into account information in the CD, opinions that have been expressed by members of the CASAC Ozone Review Panel (Panel) at its meetings and in written comments, and opinions of other health and medical respiratory experts provided during the current and previous O_3 NAAQS reviews.

In 1985, the American Thoracic Society (ATS) published general guidelines describing what constitutes an adverse respiratory health effect. While recognizing that perceptions of "medical significance" and "normal activity" may differ among physicians, lung physiologists, and experimental subjects, the ATS (1985) defined 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." Staff believes this definition provides a reasonable framework for present purposes. As discussed above in section V.C., human health effects for which clear, causal relationships with exposure to O_3 have been demonstrated fall into the first category listed in the ATS definition. Human health effects for which statistically significant associations have been reported in epidemiology studies fall into the second and third categories. These effects include respiratory illness that may require medication (e.g., asthma), but not necessarily hospitalization, as well as emergency room visits and hospital admissions for acute occurrences of respiratory morbidity. Human health effects for which associations have been suggested but not conclusively demonstrated fall primarily into the last two categories. Those health endpoints are based on studies of effects in laboratory animals and to a lesser extent on human epidemiological studies and can be extrapolated to human health effects only with great uncertainty.

1. Permanent Respiratory Injury and/or Progressive Dysfunction

An increase in daily mortality associated with O_3 exposure is unquestionably the most adverse health effect for which only limited, suggestive evidence exists. As discussed in section V.C., causal relationships have been reported in animal infectivity studies of O_3 (Coffin et al., 1967; Coffin and Gardner, 1972; Miller et al., 1978). However, only one published epidemiological study (Kinney and Ozkaynak, 1991) has provided statistically significant evidence of an association with daily mortality even at the very high levels of O_3 found in Los Angeles. In that study, the authors state that although statistically significant associations were found between daily mortality and environmental variables, one can not conclude with complete confidence that such associations are causal. Also, other pollutants (e.g., particulate matter) have been found to be significant contributors to daily mortality, but it is hard to determine the relative contributions of various pollutants (U.S. EPA, 1996b). No other human studies cited in the CD have reported statistically significant associations between O_3 and mortality.

Other adverse effects, such as scarring of lung tissue, reduced elasticity of the lungs, and accelerated reductions in lung function have been clearly demonstrated only in laboratory animal studies. These effects may be the result of repeated pulmonary inflammation. Although any association between ambient O_3 exposures and permanent structural change in the lung tissue in humans remains largely hypothetical at this time, indicators of acute pulmonary inflammation following short-term O_3 exposures have been reported in several human experimental studies. There appears to be general agreement that a single exposure to O_3 that induces an inflammatory response has little or no health significance, just as a single, short-term exposure to the sun, sufficient to result in sunburn, would have little health significance for most individuals. However, it is well documented that long-term, repeated exposures to the sun can damage the skin irreversibly. Analogously, some health scientists have cautioned that if O_3 exposures are repeated over many months or years, the highly irritating nature of O_3 could induce chronic inflammatory responses in humans, which may culminate in irreversible lung tissue damage.

Morphological abnormalities in the centriacinar region of the lungs, also referred to as "lesions" by some researchers (as discussed in Sec. V.C.8 of this Staff Paper), are among the most investigated chronic O_3 effects in laboratory animal studies. If these repeated acute responses do in fact lead to similar chronic effects in humans as have been observed in laboratory animals, it is possible that such effects could accelerate the loss of lung function and the ability of elderly individuals to engage in activities which require exertion later in life. This could impair their quality of life and could shorten longevity of affected individuals. Several efforts have been made to find associations between long-term O_3 exposure and chronic respiratory dysfunction and disease (Detels et al., 1991; Abbey et al., 1993; Schwartz, 1989). Taken as a whole, these studies suggest that it is not possible to conclude if there is an effect of O_3 on the health effects studied, in part due to limitations introduced by loss of subjects during the studies and by confounding variables such as coexposure to particulate matter. Thus, the appropriate conclusion to be drawn at this time is that associations between O_3 exposure and chronic health impacts have not been sufficiently demonstrated in humans. Some Panel members expressed views at the March 1995 and September 1995 CASAC meetings, and in subsequent written comments, that the

chronic health effects discussed above pose a sufficiently important public health threat as to warrant serious consideration in this review of the O_3 NAAQS. Other Panel members expressed the opinion that such health outcomes are too uncertain to be considered at this time. In consideration of the potential seriousness to public health of possible chronic health effects of O_3, staff agrees with the position taken in the CASAC closure letter (Wolff, 1995b) recommending that research efforts continue on the chronic health effects of O_3 to reduce the uncertainties before the next review of the O_3 NAAQS.

2. Episodic and Incapacitating Illness in Persons with Impaired Respiratory Systems

The most significant episodic health effects that have been associated with short-term O_3 exposures are increased hospital admissions and emergency room visits due to respiratory causes. Health effects related to increased respiratory hospital admissions and emergency room visits include respiratory infections (e.g., pneumonia), asthma attacks, and exacerbation of other respiratory diseases (e.g., COPD). There exists a substantial, and growing, data base which suggests an association between O_3 and increased respiratory hospital admissions for individuals with asthma and other impaired respiratory systems. By analogy, while it is plausible that healthy individuals, particularly the individuals who have lost a significant amount of their lung reserve capacity, could be adversely affected at very high O_3 levels sufficient to require hospitalization, there is no evidence to show this is occurring with ambient O_3.

Although little controversy exists regarding the adversity to the individual for responses that lead to being admitted to the hospital or to visiting an emergency room, there is still debate over the extent to which exposure to O_3 is directly responsible for these adverse responses relative to other environmental factors (e.g., exposure to other air pollutants, heat, humidity, allergens), which could confound the association with O_3. In assessing the significance of other effects of short-term O_3 exposures that have been demonstrated in controlled human exposure studies (e.g., decreased lung function, respiratory symptoms), it is important to consider the magnitude of such individual changes in persons with impaired respiratory systems (e.g., asthmatics) who already have reduced lung function. A comparable change in lung function could have greater impact on the health status, whether illness or interference with normal activity, of an individual with a preexisting

respiratory disease, such as asthma, chronic bronchitis, emphysema, or serious allergies, than on a healthy individual with normal lung function and reserve capacity. Any change in lung function that causes these individuals with impaired respiratory systems to drop below 40 to 50 percent of predicted values would be considered clinically adverse. For example, O_3-induced changes in SR_{aw}, a measure of airway narrowing, are small and of minimal clinical significance in nonasthmatic individuals. Asthmatics, however, often have baseline airway narrowing and experience larger changes in SR_{aw} on exposure to O_3 than do nonasthmatics. Because of these baseline differences, the clinical significance of increases in SR_{aw} depends both on percent change from baseline and on absolute increases in SR_{aw} (CD, p. 9-23).

Individuals with asthma represent a population subgroup which has been examined extensively in experimental and epidemiological studies of O_3. Asthmatic individuals have been found to exhibit O_3-induced airway responses that are slightly more pronounced than those found in non-asthmatic persons. It is important to understand asthma as a disease and place the effects reported in controlled human exposure studies into proper context. This involves careful definition of asthma, classification of asthma by severity of disease, discussion of medication use, and description of the nature and time course of response. These considerations of asthma have been addressed previously by the EPA and are presented in the SO_2 Staff Paper Addendum (USEPA, 1994a, pp. 11-33) and Criteria Document Addendum (USEPA, 1994b). The definition of asthma contained in those documents and taken from the Expert Panel Report from the National Asthma Education Program of the National Heart, Lung, and Blood Institute (NIH, 1991) is:

> Asthma is a lung disease with the following characteristics: (1) airway obstruction that is reversible (but not completely so in some patients) either spontaneously or with treatment, (2) airway inflammation, and (3) increased airway responsiveness to a variety of stimuli.

Working with scientists in EPA's ORD, staff developed Tables V-4a, V-4b, and V-4c (Table 9-2 in the CD), which categorize acute respiratory responses to O_3 in individuals with impaired respiratory systems according to type and severity of response. These tables are based on a similar categorization for healthy individuals developed by staff as Table VII-5

Tables V-4a, V-4b, and V-4c. Gradation of Individual Responses to Short-Term Ozone Exposure in Persons with Impaired Respiratory Systems[a]

Table V-4a

Functional Response	None	Small	Moderate	Large
FEV_1 change	Decrements of <3%	Decrements of 3% to ≤10%	Decrements of >10% but <20%	Decrements of ≥20%
Nonspecific bronchial responsiveness[b]	Within normal range	Increases of <100%	Increases of ≤300%	Increases of >300%
Airway resistance (SR_{aw})	Within normal range (±20%)	SR_{aw} increased <100%	SR_{aw} increased up to 200% or up to 15cm H_2O/s	SR_{aw} increased >200% or more than 15 cm H_2O/s
Duration of response	None	<4 hr	>4 but ≤24 hr	>24 hr

Table V-4b

Symptomatic Response	Normal	Mild	Moderate	Severe
Wheeze	None	With otherwise normal breathing	With shortness of breath	Persistent with shortness of breath
Cough	Infrequent cough	Cough with deep breath	Frequent spontaneous cough	Persistent uncontrollable cough
Chest pain	None	Discomfort just noticeable on exercise or deep breath	Marked discomfort on exercise or deep breath	Severe discomfort on exercise or deep breath
Duration of response	None	<4 hr	>4 but ≤24 hr	>24 hr

Table V-4c

Impact of Various Functional and/or Symptomatic Responses	Normal Functional and/or Symptomatic Responses	Small Functional and/or Mild Symptomatic Responses	Moderate Functional and/or Symptomatic Responses	Large Functional and/or Severe Symptomatic Responses
Interference with normal activity	None	Few individuals likely to limit activity	Many individuals likely to limit activity	Most individuals likely to limit activity
Medical treatment/Self Medication	No change	Normal medication as needed	Increased frequency or additional medication use	Increased likelihood of physician or ER visit

[a]See text for discussion, abbreviations and acronyms.
[b]An increase in nonspecific bronchial responsiveness of 100% is equivalent to a 50% decrease in PD_{20} or PD_{100} (see Chapter 7, Section 7.2.3 of the CD for a more complete discussion).

in the previous Ozone Staff Paper (U.S. EPA, 1989) produced during the last O_3 NAAQS review.

In addition to the health status of the individual, the clinical significance of individual responses to O_3 depends on the magnitude of changes in pulmonary function, the severity of respiratory symptoms, and the duration of response. Tables V-4a and V-4b categorize individual functional and symptomatic responses to O_3 exposure as either normal (i.e., none) or with graded levels of increasing severity in individuals with impaired respiratory systems, similar to Tables V-5a and V-5b for healthy individuals discussed later in this section. Pulmonary function responses are represented in these tables by changes in spirometry (e.g., FEV_1), SR_{aw}, and non-specific bronchial responsiveness. Respiratory symptom responses include cough, pain on deep inspiration, and wheeze. The predominant changes in spirometry discussed in this Staff Paper are O_3-induced decrements in FEV_1 because they are more easily quantified, have a continuous distribution, and have been used to provide most of the exposure-response relationships described in the CD and in the exposure and risk analyses. The combined impacts of both functional and symptomatic responses are presented for individuals with impaired respiratory systems in Tables V-4c and for healthy individuals in Tables V-5c as interference with normal activity and as changes in medical treatment and/or self medication. (See Tables 9-1 and 9-2 and the discussion in CD starting on p. 9-23).

It is staff's judgment that responses of individuals with impaired respiratory systems, categorized in Table V-4a as "large" for functional responses or categorized in Table V-4b as "severe" for symptomatic responses, would result in the potential for episodic or incapacitating illness. Those responses would include the more quantifiable responses such as a significant increase in nonspecific bronchial responsiveness (i.e., dose is $<25\%$ of baseline), an increase in nonspecific airway resistance (SR_{aw}) of $>200\%$ or more than 15 cm H_2O/s, and a decrease in FEV_1 of $\geq$ 20% from baseline. The less quantifiable, but potentially incapacitating effects, to the individual with impaired respiratory systems include persistent wheeze, uncontrollable cough, severe discomfort on exercise or deep breath, and multiple bronchodilator usage giving only partial relief. Since these "severe" symptomatic and "large" functional responses for individuals with impaired respiratory systems could limit activity and increase the likelihood of physician or emergency room (ER) visits as well as

hospital admissions for some affected individuals, staff recommends that they be characterized as adverse. Because "small" and "moderate" functional response and "mild" and "moderate" symptomatic responses would not be likely to result in impacts comparable to episodic or incapacitating illness, they are discussed in the following section on interference with normal activity.

3. Interference with Normal Activity

For both healthy individuals and for individuals with impaired respiratory systems, there has been a great deal of controversy regarding the extent to which other acute responses that have been associated with short-term and prolonged O_3 exposures should be considered adverse. The previous section contains a discussion of functional effects categorized as "large" and symptomatic effects as "severe" for individuals with impaired respiratory systems. Those effects would be more likely to lead to episodic or incapacitating illness in asthmatic individuals as discussed above, whereas the "small" and "moderate" functional effects and "mild" or "moderate" symptomatic effects discussed below are more likely to be limited at most to interference with normal activity of either asthmatic or healthy individuals. "Moderate" functional and/or symptomatic responses in either healthy or asthmatic individuals are most problematic with regard to judging adversity because they are not serious enough to be clearly described as adverse but may still interfere with the ability of some individuals to perform normal activity and, therefore, have the potential for adversity in some sensitive individuals.

Asthmatic Individuals. The response of asthmatic individuals to O_3 and other irritants can be highly variable depending on the severity of disease in the individual. A normal range of change in specific airway resistance (SR_{aw}) is within $\pm 20\%$ with little or no change in nonspecific bronchial responsiveness. Ozone-induced decrements in FEV_1 of $<3\%$, increases in SR_{aw} and nonspecific bronchial responsiveness approaching 100%, which last less than 4 hr, are categorized as "small" in Table V-4a. Even in conjunction with symptomatic effects categorized as "mild" (i.e., wheeze with otherwise normal breathing, cough with deep breath, and discomfort just noticeable on exercise or deep breath) lasting less than 4 hr, staff believes that these effects should not be considered adverse for asthmatic individuals. Although a few individuals are likely to limit activity, these responses are not

likely to significantly interfere with the ability of most asthmatics to conduct normal activity or change normal medication usage.

Ozone-induced decrements in FEV_1 of $>10\%$ but $<20\%$, increases in SR_{aw} of up to 200%, and increases in nonspecific bronchial responsiveness of up to 300%, which last for >4 hr but <24 hr, have been categorized for asthmatic individuals as "moderate" functional responses in Table V-4a. These responses are typically accompanied by "moderate" symptomatic responses, including wheeze with shortness of breath, frequent spontaneous cough, and marked discomfort on exercise or deep breath, which last for >4 hr but <24 hr. Based on discussions with medical experts who have worked with asthmatics, staff concluded that single O_3 exposure events which result in these responses are not likely to interfere with the normal activity of many asthmatic individuals nor to result in the increased frequency of medication use or the use of additional medications. Complete recovery could result from a single use of a bronchodilator. However, because repeated exposure of asthmatics to O_3 over periods of several days could result in exacerbation of the underlying inflammation and a buildup of mucus in the respiratory system, medical experts who were consulted expressed concern that the small airways, including the bronchioles and alveoli, may be more adversely affected than effects induced by a single, acute exposure. Staff believes that multiple exposures to O_3 that induce repeated "moderate" responses in asthmatics could result in increased frequency or additional medication usage, mucus buildup, exacerbation of inflammation, and an increased likelihood of many asthmatic individuals to limit normal activity. Therefore, staff recommends that "moderate" functional and/or symptomatic responses, when repeated, should be considered to be adverse health effects. These health endpoints are a matter of public health concern in light of the increasing asthma morbidity and mortality which has been occurring in the U.S. during the past decade.

Healthy Individuals. During the previous O_3 NAAQS review, a wide range of opinion was expressed regarding the adversity of lung function decrements, increases in the severity of respiratory symptoms and increases in nonspecific bronchial responsiveness in healthy individuals. In particular, the focus of debate was on the degree of response for acute respiratory effects that should be considered adverse for purposes of setting NAAQS. A table was presented in the previous O_3 Staff Paper (Table VII-5, p. VII-55, USEPA, 1989)

which categorized these acute respiratory responses of healthy individuals to O_3 according to type and severity of response. Several specific aspects of such responses were characterized including: (1) the magnitude of lung function decrements on a test-specific basis (e.g., FEV_1); (2) the presence of respiratory symptoms (e.g., cough, pain on deep inspiration, shortness of breath); (3) the duration of individual response; and (4) the extent to which activity is curtailed due to O_3 exposures.

At the December 1987 CASAC meeting, some members of the CASAC Ozone Review Panel expressed the belief that either limitation of activity or increased respiratory symptoms could be considered the primary determinant of adversity, while others believed that the more objective spirometry measurements were most appropriate. Some Panel members felt that healthy individuals would experience adverse effects when O_3 exposure induced any of the responses categorized in the 1989 table as "moderate" (i.e., FEV_1 decrement of 10-20%; mild to moderate cough, pain on deep inspiration, shortness of breath; complete recovery in <6 hours; and few sensitive individuals likely to discontinue activity). Other Panel members believed that adverse effects would not result unless a healthy individual encountered O_3-induced effects categorized as severe (i.e., FEV_1 decrement of 20-40%; repeated cough, moderate to severe pain on deep inspiration and breathing distress; complete recovery in 24 hours, and some sensitive individuals likely to discontinue activity).

One of the Panel members pointed out at the December 1988 CASAC meeting that because children report few, if any, symptoms when exposed to O_3 concentrations likely to induce symptoms in adults, it may be inappropriate to recommend that all categories of response be experienced by children before describing the effects as adverse. This is due to concern that by not experiencing the "early warning signals" (i.e., respiratory symptoms) children would be more likely to continue high levels of exertion during periods of exposure to O_3 levels that could potentially induce substantial pulmonary function changes and repeated acute inflammatory responses. In written comments following the March 1995 CASAC meeting, another Panel member expressed the opinion that the lack of a symptom response should not be considered a risk factor for children. This divergence of opinion by the Panel members regarding lack of a symptom response in children possibly introducing increased

risk of exposure to O_3 and resultant adverse consequences was discussed at the September 1995 CASAC meeting.

Taking into account both previous and current expert opinion, staff worked with scientists in EPA's ORD to develop Tables V-5a, V-5b, and V-5c (Table 9-1 in the CD) showing a gradation of responses to short-term O_3 exposures for healthy individuals. Consistent with ATS guidelines, current and past CASAC views, and the judgments of two EPA Administrators in the previous O_3 NAAQS rulemaking, staff recommends that functional responses categorized as "small" in Table V-5a not be considered adverse respiratory effects for healthy individuals. Individual "small" responses to O_3 exposures are characterized by 3% to $\leq$ 10% decrements in spirometry and $<$ 100% increases in nonspecific bronchial responsiveness, which last less than 4 hours. These are often accompanied by respiratory symptoms categorized in Table V-5b as "mild," such as cough only during deep inspiration or during lung function tests. "Small" functional responses and "mild" symptomatic responses would not generally be considered medically significant and would not be expected to interfere with normal activity of healthy individuals.

Staff also recommends that any of the individual functional responses categorized as "large" in Table V-5a or symptomatic responses categorized as "severe" in Table V-5b should be considered adverse respiratory effects, in and of themselves, for healthy individuals. Staff believes that such responses (e.g., FEV_1 decrements $>$ 20%, increases in nonspecific bronchial responsiveness $>$ 300%, and uncontrollable, persistent cough, and/or chest pain lasting 24 hours and longer) are medically significant under the ATS guidelines. Such responses would likely cause many individuals to halt normal activities involving physical exertion. Furthermore, individuals experiencing such effects would most likely judge that they were being adversely affected at least for the duration of the response. As discussed by Panel members at the March 1995 CASAC meeting, such effects might be similar to those experienced by an individual with acute bronchitis. Staff believes that it is more likely that responses of this degree could be associated with exposures that may be linked to more serious, but not subjectively noticeable, responses (e.g., respiratory inflammation, lung tissue damage) that individuals would not perceive were occurring.

Tables V-5a, V-5b, and V5-c. Gradation of Individual Responses to Short-Term Ozone Exposure in Healthy Persons[a]

Table V-5a

Functional Response	None	Small	Moderate	Large
FEV_1	Within normal range (±3%)	Decrements of 3% to ≤10%	Decrements of >10% but <20%	Decrements of ≥20%
Nonspecific bronchial responsiveness[b]	Within normal range	Increases of <100%	Increases of ≤300%	Increases of >300%
Duration of response	None	<4 hr	>4 but ≤24 hr	>24 hr

Table V-5b

Symptomatic Response	Normal	Mild	Moderate	Severe
Cough	Infrequent cough	Cough with deep breath	Frequent spontaneous cough	Persistent uncontrollable cough
Chest pain	None	Discomfort just noticeable on exercise or deep breath	Marked discomfort on exercise or deep breath	Severe discomfort on exercise or deep breath
Duration of response	None	<4 hr	>4 but ≤24 hr	>24 hr

Table V-5c

Impact of Various Functional and/or Symptomatic Responses	Normal Functional and/or Symptomatic Responses	Small Functional and/or Mild Symptomatic Responses	Moderate Functional and/or Symptomatic Responses	Large Functional and/or Severe Symptomatic Responses
Interference with normal activity	None	None	A few sensitive individuals likely to limit activity	Many sensitive individuals likely to limit activity

[a]See text for discussion, abbreviations and acronyms.
[b]An increase in nonspecific bronchial responsiveness of 100% is equivalent to a 50% decrease in PD_{20} or PD_{100} (see Chapter 7, Section 7.2.3 of the CD for a more complete discussion).

Establishing specific staff recommendations with regard to effects in the "moderate" categories in Tables V-5a, V-5b, and V-5c is more problematic. The effects in this category could interfere with the activities of a few sensitive, healthy individuals, particularly for the symptom responses alone or when symptoms are accompanied by lung function decrements. Those sensitive individuals who experience a combination of "moderate" functional responses (i.e., lung function loss of $>10\%$ but $<20\%$ and increased nonspecific bronchial responsiveness of $\leq 300\%$ lasting from 4 to 24 hr) accompanied by "moderate" respiratory symptoms (i.e., marked discomfort and frequent cough persisting from 4 up to 24 hr) are likely to limit activity and may perceive that they had been affected adversely. It is unlikely, however, that these individuals would seek medical treatment or use self medication.

Lung function decrements at the "moderate" level, which may be a more likely response in children, may not be noticed by the individuals affected due to a lack of respiratory symptoms. In such cases, the extent to which such responses should be judged as adverse may depend on the likelihood that exposures causing such moderate decreases in lung function are associated with more serious effects, as to which there is substantial uncertainty as discussed above. A further complication is that the likelihood of such exposures is related to the attenuation of effects that is typically observed after repeated exposures. For example, it is well established that lung function and symptom responses in individuals exposed to O_3 on consecutive days will attenuate until absent (CD, Sec. 7.2.1.4). Most individuals initially experience larger decrements in FEV_1 on the second day but by the third or fourth day will experience disappearance of FEV_1 decrements when exposed to O_3. This attenuation of response can last for as much as 1 to 2 weeks, thus reducing the self-protective behavior that might otherwise tend to limit ongoing exposure. Without respiratory symptoms or altered lung function as "early warning signals," some individuals may be more likely to expose their lungs repeatedly to O_3 levels potentially associated with more serious effects, such as pulmonary inflammation, although, as discussed above, the extent to which these more serious effects are linked to "moderate" lung function changes is uncertain.

During the March 1995 CASAC meeting, there was considerable discussion regarding the adverse nature of these acute "moderate" health effects of O_3. One of the Panel members indicated that experiencing these effects on a single occasion might be considered by the

individual to be a "nuisance." There also would be little likelihood of attenuation of response following a single O_3 exposure. However, if these same exposures were repeated on multiple occasions they might become a matter of public health concern, particularly if large segments of the population experienced "moderate" effects repeatedly. In written comments by one of the Panel members following the March 1995 CASAC meeting, it was suggested that "moderate" symptoms (e.g., frequent spontaneous cough) represent significant inflammation of airways which is an important indicator of simple chronic bronchitis, and therefore should be considered adverse only if they occur repeatedly. Similarly, with regard to marked discomfort on exercise or FVC test, this category should be considered adverse if repeated but not for a single event. These comments underscore the consistency of CASAC opinion that single, acute, O_3-induced health effects described above as "moderate" for healthy individuals should not be considered adverse. However, as one Panel member stated in written comments, "a series of peaks could well set the stage for serious illness." Because there appears to be a greater consensus of opinion regarding the adverse nature of repeated health effects of multiple O_3 exposure, it is the recommendation of staff that the number of O_3 exposures resulting in "moderate" health effects should be considered as a factor in characterizing adversity for healthy individuals. EPA staff are concerned that multiple exposures to O_3 could induce adverse effects in healthy individuals if they are particularly sensitive and could result in limitation activity or self medication due to O_3 exposure. In summary, the degree of adversity of repeated "moderate" responses in healthy individuals is likely to increase with the increasing number of occurrences and with the combination of different responses.

G. Ozone Exposure Analysis

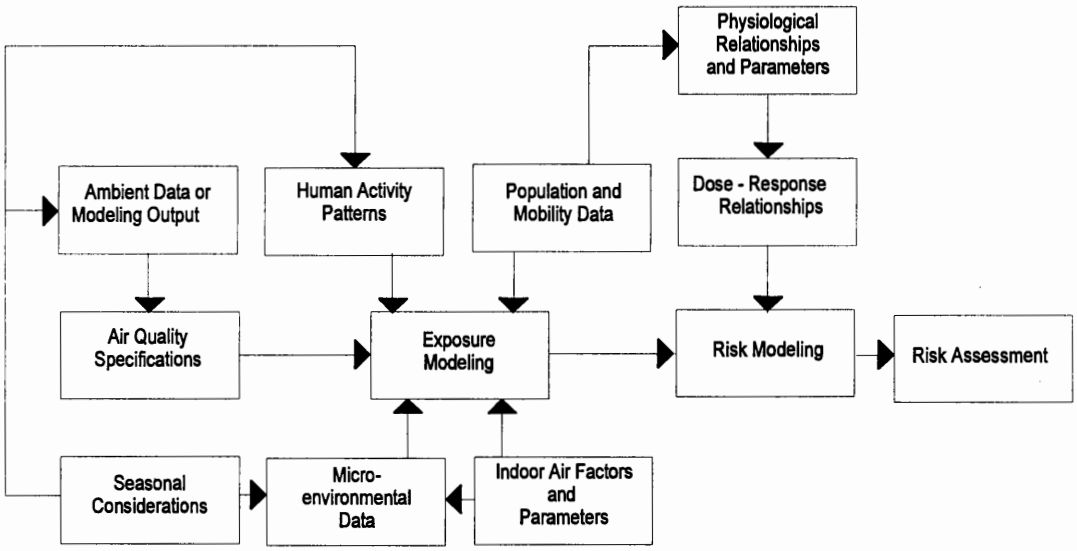
1. Overview

This section discusses a series of analyses designed to provide estimates of O_3 exposure for the general population and two subpopulations (i.e., "outdoor workers" and "outdoor children") living in 9 U.S. urban areas under the conditions that various alternative 1- and 8-hour, 1- and 5-expected exceedance NAAQS are just attained. To provide some perspective, exposure estimates are also provided for a recent year (either 1990 or 1991) for each of the 9 urban areas. The exposure estimates summarized in this section also are an important input to the "headcount" health risk assessment described in Section V.H.

The regulatory scenarios examined in the exposure analysis are limited to 1- and 5-expected exceedance alternative standards and are based on use of a single year of data. However, one can use these estimates to roughly bound the exposure and health risks for other forms of the standard under consideration (e.g., average of the 2nd daily maximum 8-hr average over a 3-year period) by using air quality analyses that compare alternative forms of the primary standard. In analyzing the exposures and health risks for any of the forms of the standard that are based on an average concentration or expected number of exceedances over a multiple year period, including the current 1-expected exceedance, 1-hr standard, the exposure and risk estimates reflect what would be expected in a typical or average year in an area just attaining a given standard. An area just attaining a standard might have annual exposures and health risks somewhat lower or higher than the average estimates over the multiple year period used to define attainment of the standard.

Figure V-5 illustrates the various components of the exposure model and how the exposure assessment relates to risk assessment. Four versions of the probabilistic NAAQS exposure model for O_3 (pNEM/O_3) were used to estimate population exposure under alternative 1- and 8-hr standards. The pNEM/O_3 exposure model builds on earlier deterministic versions of NEM by modeling random processes within the exposure simulation. A brief summary of the pNEM/O_3 model is provided below. A more detailed description of pNEM/O_3 and its application to the general population, outdoor workers, outdoor children, and a single summer camp in California can be found elsewhere in a

FIGURE V-5. MAJOR COMPONENTS OF PNEM/O_3 MODEL AND ASSOCIATED HEALTH RISK ASSESSMENT PROCEDURES



collection of exposure support documents (Johnson, 1994; Johnson et al., 1996a,b,c; McCurdy, 1994a). The pNEM/O_3 model has been designed to take into account the most significant factors contributing to total human O_3 exposure. These factors include the temporal and spatial distribution of people and O_3 concentrations throughout an urban area, the variation of O_3 levels within each microenvironment, and the effects of exertion (increased ventilation) on O_3 uptake in exposed individuals.

Three versions of the pNEM/O_3 model have been run (general population, outdoor workers, and outdoor children) and applied to the same nine major urban areas. The fourth version was applied to a specific summer camp in Pine Springs, California (see Johnson, 1994). The nine urban areas used in the general population, outdoor worker, and outdoor children versions of the model vary greatly in geographical location, O_3 "design value",[4] population size (both modeled and total MSA), and number of exposure districts included. The areas were selected to obtain as widely representative a modeling domain as possible given the overall need for monitoring data completeness in an area.

For instance, urban study area populations modeled vary from Denver, with a population of 1.5 million, to the New York area, with a population of about 10.7 million people. Information about the study area population, number of exposure districts, year and O_3 season modeled, and summary air quality statistics for the nine study areas are presented in Table V-6.

The total population included in the 9 urban study areas covered by the exposure analysis is 41.7 million people. Given the considerable additional uncertainty that would be introduced, OAQPS has chosen not to extrapolate the exposure estimates from the 9 urban areas to obtain national exposure estimates. The 9 urban areas represent a significant fraction of the U.S. urban population and include the largest areas with major O_3 nonattainment problems (e.g., Los Angeles, Chicago, New York, and Houston).

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[4] A design value is that measured air quality concentration value in a MSA that must be reduced to the O3 standard level to ensure that the area meets the current O_3 NAAQS formulation of $\leq$ 1 expected exceedances of 0.12 ppm daily maximum 1-hour average. The design 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.

TABLE V-6. CHARACTERISTICS OF OZONE STUDY AREAS USED IN PNEM/O_3 ANALYSES

Study Area	1990 Population in Study Area (millions)	1990 MSA or CMSA Population (millions)	Number of Exposure Districts	Exposure Period		Daily Max. Hourly Design Value (ppm)[a]
				Year	Months	
Chicago	6.2	8.1	12	1991	Apr-Oct	0.129
Denver	1.5	3.8	7	1990	Mar-Sep	0.115
Houston	2.4	3.7	11	1990	Jan-Dec	0.23
Los Angeles	10.4	13.8	16	1991	Jan-Dec	0.31
Miami	1.9	1.9	6	1991	Jan-Dec	0.123
New York City	10.7	18	12	1991	Apr-Oct	0.175
Philadelphia	3.8	6	10	1991	Apr-Oct	0.156
St. Louis	1.7	2.4	11	1990	Apr-Oct	0.13
Washington, D.C.	3.1	3.9	11	1991	Apr-Oct	0.174

[a]The design value listed here is the second-highest 1-hour daily maximum concentration in the O3 air quality data base for the year modeled.

2. Exposure Modeling Methodology

The pNEM/O_3 model consists of two principal parts, the cohort exposure program and the exposure extrapolation program. The cohort exposure program estimates the sequence of O_3 exposures experienced by defined population groups. The exposure extrapolation program estimates the number of persons within a particular study that are represented by each cohort and then combines this information with cohort exposure sequences to estimate the distribution of exposures over a defined population of interest.

The pNEM/O_3 methodology consists of the following five steps:

(1) define a study area, a population of interest, appropriate subdivisions of the study area, and an exposure period,

(2) divide the population of interest into an exhaustive set of cohorts,

(3) develop an exposure event sequence for each cohort for the exposure period,

(4) estimate the pollutant concentration and ventilation rate associated with each exposure event, and

(5) extrapolate cohort exposures to the population of interest.

Each of these steps is described in more detail in the following discussion.

Define the Study Area, Subdivisions of the Study Area, the Exposure Period and the Population of Interest. The study area is defined as an aggregation of exposure districts. Each exposure district is defined as a contiguous set of geographical census units (GCU). Each GCU consists of one or more census tracts as defined by the 1990 census. All GCUs assigned to a particular exposure district are located within a specified radius (15 km) of a fixed-site O_3 monitor.

As indicated previously, the nine urban areas used in the general population, outdoor worker, and outdoor children versions of the model vary greatly in geographical location, O_3 "design value", population size (both modeled and total MSA), and number of exposure districts included. Each urban area was divided into large exposure districts, varying from 6 to 16 in the nine areas modeled, corresponding to the number of air quality monitors having valid air quality data in a study area. Most of the urban areas had 10 or more districts

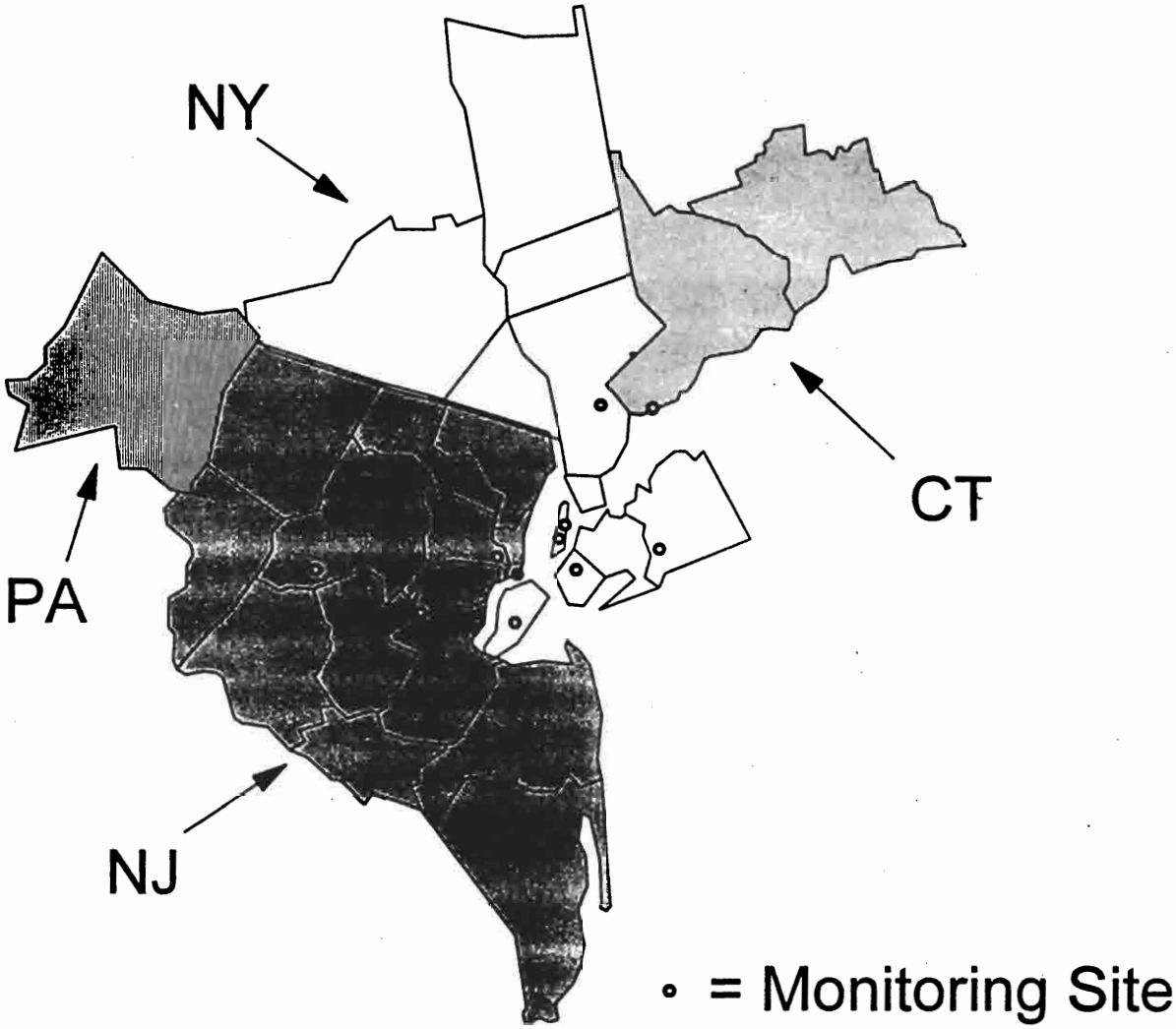
within their boundaries. The study areas included urban and suburban counties next to the central city for which the study areas are named. To illustrate this point, Figure V-6 shows the New York consolidated metropolitan statistical area and the monitors used in the New York study area for purposes of the pNEM/O_3 analysis are indicated by the circles on the map.

From 3 to 16 monitoring sites were selected to represent the spatial variation of O_3 levels in each of the 9 study areas. The number of monitors chosen for each area depends upon a data completeness criterion (i.e., data are at least 75% complete) for each monitor and the availability of home-to-work commuting data for a district. For Los Angeles, 30 possible monitors were pared down to 16 because of limitations on computational resources. This paring down was accomplished by removing one of nearby pairs of monitors that has similar O_3 air quality distributions. In the New York study area, one site, the World Trade Center, was removed because the monitor is placed on the top of the building and is not considered representative for population exposure. The nearest New York City monitor is used to represent the World Trade Center district. Otherwise, all available monitors meeting the above criteria were used in each of the study areas.

The exposure period is defined as a series of months within a particular calendar year corresponding to the designated O_3 monitoring season specified for the urban area by the U.S. EPA. For six of the nine urban areas the season is nine months long, while three areas (Los Angeles, Houston, and Miami) have a 12 month season.

The CD identifies outdoor workers and children as two population groups particularly at risk for experiencing O_3-related health effects. These two groups were identified based on the increased time they spend outdoors engaged in moderate and heavy exertion which increases the likelihood of experiencing O_3-induced health effects. While children and workers were included in the general population version of pNEM/O_3, EPA analysts felt that the procedures used did not adequately represent exposures for workers or children that spend considerable time outdoors on a regular basis. Therefore, special versions of pNEM/O_3 were developed to estimate population exposures for outdoor workers and outdoor children. Table V-7 lists the 1990 population estimates for the general population, outdoor workers, and outdoor children in each of the nine urban areas.

FIGURE V-6. New York Urban Area Monitoring Sites Used in pNEM Analyses.



Population in 1990
pNEM Study Area
= 10,660,000

NY CMSA
= 17,950,000

TABLE V-7. POPULATION ESTIMATES FOR OZONE STUDY AREAS USED IN pNEM/O_3 ANALYSES

Study Area	1990 General Population in Study Area (millions)	1990 Outdoor Worker Population in Study Area (thousands)	1990 Outdoor Children Population in Study Area (thousands)
Chicago	6.2	141	473
Denver	1.5	36	107
Houston	2.4	72	201
Los Angeles	10.4	294	798
Miami	1.9	47	133
New York City	10.7	196	783
Philadelphia	3.8	99	275
St. Louis	1.7	41	128
Washington, D.C.	3.1	76	199

Divide the Population-of-Interest Into an Exhaustive Set of Cohorts. The population of interest, in each version of the model, is divided into a set of cohorts such that each person is assigned to only one cohort. Each cohort is assumed to contain persons with identical exposures during the specified exposure period. Cohort exposure is typically assumed to be a function of (1) demographic group, (2) location of residence, and (3) location of work place. Specifying the home and work district of each cohort provides a means of linking cohort exposure to ambient pollutant concentrations. Specifying the demographic groups provides a means of linking cohort exposure to activity patterns that vary with age, work status, and other demographic variables.

Because both the intake dose received and susceptibility to O_3 health effects may vary with age, occupation, and intensity of exertion, the total population of each study area was divided into 9 age-occupation (A-O) groups. Each A-O group was further subdivided into cohorts depending upon (1) the type of air conditioning system present in the home, if any, (2) home district, and (3) work district.

Develop an Exposure Event Sequence for Each Cohort for the Exposure Period. The exposure of each cohort is determined by an exposure event sequence (EES) specific to the cohort. Each EES consists of a series of events with durations from 1 to 60 minutes. To determine average exposures for specific clock hours, exposure events are defined such that no event falls within more than one clock hour. Each exposure event assigns the cohort to a particular combination of geographic area and microenvironment. Each event also provides an indication of breathing rate. The breathing rates are classified as sleeping, slow, medium, and fast.

EESs are determined by assembling activity diary records relating to individual 24-hour periods into a series of records spanning the O_3 season of the study area. Because each subject of the activity pattern diary studies provides data for one to three days, the construction of a multi-month EES requires either repetition of data from one subject or the use of data from multiple subjects. The latter approach is used in all three PNEM versions discussed here.

The use of activity data from multiple persons to construct a multi-month EES for each cohort is believed to better represent the variability of exposure that is expected to

occur among persons included in each cohort. However, the fact that a multi-month EES is constructed by using data from multiple subjects means there is more uncertainty in the persons exposure measure and, in particular, in the estimates of how many times any individual is exposed to a given concentration. The PNEM/O_3 model probably underestimates the frequency of exposures for those individuals in the population that engage in moderate or heavy exertion on a regular basis.

For the general population version of the model, activity diary data were obtained from the Cincinnati Activity Diary Study (CADS) (Johnson, 1987). The CADS data base includes over 900 subjects who completed three-day activity diaries.

For the outdoor worker version of PNEM/O_3, additional data from six other time/activity studies were combined with the CADS database and processed to provide a unified time/activity database representative of outdoor workers. These studies are summarized in Table V-8. The activity data selected to represent outdoor workers were based on selecting data from subjects that spent at least four hours at work and spent at least 50 percent of their work time outdoors. The final pool contained 89 outdoor workers with 136 person-days of diary data. City-specific outdoor worker estimates were derived based on city-specific 1990 Census data and judgments by a panel of researchers about the percentage of outdoor workers in each of 37 Census occupation groups. Section 6 of Johnson et al. (1996c) provides a detailed description of the procedures used to develop the outdoor worker time/activity data base and population extrapolation.

For the outdoor children version of PNEM/O_3, additional data from six other time/activity studies were combined with the CADS database and processed to provide a unified time/activity database representative of outdoor children. These studies are summarized in Table V-9. The pool of activity patterns used to represent outdoor children was based on selecting children that met the following conditions:

(1) during a "non-summer" weekday the child had at least one diary day where he/she spent two hours or more outdoors, or

(2) during a "non-summer" weekend the child had at least one diary day where he/she spent three hours or more outdoors, or

TABLE V-8. CHARACTERISTICS OF HUMAN ACTIVITY STUDIES USED IN OUTDOOR WORKER EXPOSURE ANALYSIS.

Database name	Reference	Characteristics of subjects	Number of subject-days in original study	Number of subject-days used in outdoor worker PNEM analysis	Study calendar periods	Diary type	Diary time period	Breathing rates reported?
California - 12 and over	Wiley et al., 1991b	Ages 12 to 94	1762	156	Oct. 1987 - July 1988	Retrospective	Midnight to midnight	No
Cincinnati	Johnson, 1987	Ages 0 to 86	2800	105	March and August 1985	Real-time[a]	Midnight to midnight	Yes
Denver	Johnson, 1984	Ages 18 to 70	859	41	Nov. 1982 - Feb. 1983	Real-time	7 p.m to 7 p.m. (nominal)	No
Los Angeles - construction	Linn et al., 1993	Construction workers (ages 23 to 42)	19	19	July - Nov. 1991	Real-time[a]	Subject wakeup to subject returns home from work	Yes
Los Angeles - outdoor worker	Shamoo et al., 1991	Adult outdoor workers (ages 19 to 50)	60	29	Summer 1989	Real-time[a]	Midnight to midnight	Yes
Valdez	Goldstein et al., 1992	Ages 10 to 72	405	25	Nov. 1990 - Oct. 1991	Retrospective	Varying 24-h period	No
Washington	Hartwell et al., 1984	Ages 18 to 70	705	33	Nov. 1982 - Feb. 1983	Real-time	7 p.m. to 7 p.m. (nominal)	No
Total				408				

[a]Study employed the Cincinnati diary format.

TABLE V-9. CHARACTERISTICS OF HUMAN ACTIVITY STUDIES PROVIDING DATA FOR OUTDOOR CHILDREN EXPOSURE ANALYSIS.

Database name	Reference	Characteristics of subjects	Number of subject-days in original study	Number of subject-days used in outdoor children pNEM analysis	Study calendar periods	Diary type	Diary time period	Breathing rates reported?
California - 11 and under	Wiley et al., 1991a	Children ages 1 to 11	1200	257	April 1989 - Feb. 1990	Retrospective	Midnight to midnight	No
California - 12 and over	Wiley et al., 1991b	Ages 12 to 94	1762	54	Oct. 1987 - July 1988	Retrospective	Midnight to midnight	No
Cincinnati	Johnson, 1987	Ages 0 to 86	2800	384	March and August 1985	Real-time	Midnight to midnight	Yes
Los Angeles - elementary school	Spier et al., 1992; Linn et al., 1992	Elementary school students, 10 to 12 years	58	38	Oct. 1989	Real-time[a]	Midnight to midnight	Yes
Los Angeles - high school	Spier et al., 1992; Linn et al., 1992	High school students, 13 to 17 years	66	47	Sept. and Oct. 1990	Real-time[a]	Midnight to midnight	Yes
Valdez	Goldstein, et al., 1992	Ages 10 to 72	405	9	Nov. 1990 - Oct. 1991	Retrospective	Retrospecti ve	No
Washington	Hartwell et al., 1984	Ages 18 to 70	705	3	Nov. 1982 - Feb. 1983	Real-time	7 p.m. to 7 p.m. (nominal)	No
Total				792				

[a]Study employed the Cincinnati diary format.

(3) during a "summer" weekday or weekend the child had at least one diary day where he/she spent 4 ½ hours or more outdoors.

For this analysis "summer" was defined as June through August and "nonsummer" as all other months. This procedure produced a pool containing 479 outdoor children with 792 person-days of activity diary data. Outdoor children included in the analysis were in 2 demographic groups: children ages 6 to 13 ("preteenagers") and children ages 14 to 18 ("teenagers"). The city-specific percentages of outdoor children were derived based on city-specific 1990 Census data for the two demographic groups and the percentages of outdoor pre-teenager and teenager subjects in three of the time/activity studies conducted in Cincinnati and California (Johnson, 1987; Wiley et al., 1991a,b) that employed a random selection procedure to enroll subjects. About 47 percent of preteens and 31 percent of teenagers were judged to meet the selection criteria for outdoor children.

A distinct EES is developed for each cohort. The exposure event within an EES is defined by the district, the microenvironment, and the breathing rate associated with the activity being undertaken by the sampled individual.

The district is defined as being either the home or work district associated with the cohort. For children, it is assumed that their school district is the same as their home district. Population movement in pNEM/O_3 is based upon information gathered by the U.S. Census Bureau regarding householders' home-work commuting patterns (Bureau of the Census, 1990). 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 pNEM/O_3 analysis to obtain district-to-district trip information for those cohorts that work. Otherwise, cohorts are assumed to stay in their home districts.

The seven microenvironments used in all three versions of pNEM are: 1) indoors-residence with a central air conditioning system, 2) indoors-residence with window air conditioning units, 3) indoors-residence with no air conditioning system, 4) indoors-nonresidential locations, 5) outdoors near a road, 6) outdoors - other locations, and 7) in-vehicle.

Estimate the Pollutant Concentration and Ventilation Rate Associated With Each Exposure Event. Pollutant concentrations associated with each exposure event depend on the O_3 air quality within each exposure district, which is estimated using ambient data from monitoring sites in each exposure district, and the microenvironment where the event occurred. The general population, outdoor worker, and outdoor children versions of pNEM/O_3 examine nine air quality scenarios. All of the regulatory scenarios are on a daily maximum basis with either 1 or 5 expected exceedances allowed per year. The scenarios (and a short-hand label for each listed in parentheses) are:

(1) 1990 or 1991 air quality--the "as is" or baseline scenario (As Is);

(2) Just attaining a 1-hr, 0.12 ppm, 1 expected exceedance standard--the current standard (1H1EX-0.12);

(3) Just attaining a 1-hr, 0.10 ppm, 1 expected exceedance standard (1H1EX-0.10);

(4) Just attaining an 8-hr, 0.10 ppm, 1 expected exceedance standard (8H1EX-0.10);

(5) Just attaining an 8-hr, 0.09 ppm, 1 expected exceedance standard (8H1EX-0.09);

(6) Just attaining an 8-hr, 0.08 ppm, 1 expected exceedance standard (8H1EX-0.08);

(7) Just attaining an 8-hr, 0.07 ppm, 1 expected exceedance standard (8H1EX-0.07);

(8) Just attaining an 8-hr, 0.09 ppm, 5 expected exceedances standard (8H5EX-0.09); and

(9) Just attaining an 8-hr, 0.08 ppm, 5 expected exceedances standard (8H5EX-0.08).

For all of the indoor and in-vehicle microenvironments the season-long sequence of hourly O_3 values is estimated using a mass balance algorithm. The mass-balance model used in pNEM/O_3 is a simplified version of the generalized Nagda, Rector, and Koontz model (Nagda et al., 1987). This model was revised to incorporate the assumption that indoor decay rate is proportional to indoor O_3 concentration. This algorithm estimates the hourly

average indoor O_3 concentrations during hour h as a function of: indoor O_3 concentration at the end of the preceding hour, the outdoor O_3 concentration during hour h, air exchange rate during hour h, and an O_3 decay rate. Values for the air exchange rate and the O_3 decay factor are sampled from an appropriate distribution, based on the available scientific literature on these parameters, on a daily basis. Air exchange rate is permitted to change hourly in the three residential microenvironments depending on whether windows are assigned a status of open or closed. This assignment is determined through use of a probabilistic model in which the status during each clock hour is assumed to be a function of air conditioning system temperature range and window status during the previous clock hour. In each of the pNEM/O_3 simulations, the O_3 concentration in a particular microenvironment during a particular clock hour is assumed to be constant.

For the two outdoor microenvironments and as an input to the mass balance algorithm for the indoor and in-vehicle microenvironments, representative ambient air quality data is required for each district in the form of a time series of hourly values for the specified O_3 season. The outdoor O_3 concentration associated with microenvironment m in district d during hour h was determined by an expression having the general form

$$C_{out}(m,d,t,s) = 1.056 \times C_{mon}(d,t,s) + e(t), \qquad \text{(equation V-1)}$$

where $C_{out}(m,d,t,s)$ is the outdoor (or ambient) O_3 concentration in microenvironment m in exposure district d at time t under regulatory scenario s, $C_{mon}(d,t,s)$ is the O_3 concentration estimated to occur at the monitor representing district d at time t under regulatory scenario s, and e (t) is a random normal variable with mean = 0 and standard deviation = 0.0053 ppm. The factor of 1.056 and the value of the standard deviation for e (t) were derived based on regression analyses relating personal exposure data and fixed site monitors obtained from the Houston Asthmatic Oxidant Study (Stock et al., 1985). The derivation of these parameters is described in more detail in Chapter 2 of Johnson et al. (1996b,c).

To represent ambient O_3 air quality concentrations in the nine urban areas, monitored values are adjusted mathematically to represent a future regulatory scenario (s) when air quality in the study area just meets the O_3 NAAQS being analyzed. 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 the O_3 exposure analyses conducted to support decisions on the NAAQS; it is sufficient to simulate the just-attaining situation without being concerned about how, when, or even if that situation will occur.

By definition, a NAAQS is attained when all monitors in an area have less than one (or five for some of the alternative 8-hr standards analyzed) expected exceedance of the standard concentration value (e.g., 0.12 ppm for the current 1-hr standard) 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. The adjustment procedure used for six of the nine urban areas 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.

The adjustment procedures were developed by comparing the O_3 data reported by a site in a high year with O_3 data reported by the same site in a low O_3 year. Therefore, these procedures are expected to perform best when used to simulate a significant reduction in the O_3 levels at a site. These procedures may produce unrealistic data sets for areas that involve either a small reduction or an increase in O_3 levels to simulate just attaining certain regulatory scenarios. Therefore, for three of the urban areas (all regulatory scenarios for Miami, Denver, and Chicago) a simpler adjustment procedure involving proportional rollback (or rollup in some cases) was used because the design values for the baseline year (i.e., 1990 or 1991) were relatively close to or, in some cases, even below the levels required to just attain the alternative standards being examined. For more information regarding the air quality adjustment procedure used to simulate a just-attaining situation see Chapter 5 in Johnson et al. (1996b,c).

An analysis evaluating the air quality adjustment procedure used to simulate attainment conditions was recently completed (Johnson, 1995). This analysis examined six of the nine urban areas (Chicago, Washington, D.C., Houston, Los Angeles, New York, and Philadelphia). O_3 levels for the baseline ("as is") year used in the pNEM analyses (either

1990 or 1991) were adjusted using the same procedures as in the pNEM analyses to just meet the levels in a "lower" year (using data from the 1992 to 1994 time period). Comparisons were then made between the adjusted data set and the observed data set for the "lower" year for each site in these six urban areas at various cutpoints of the 1-hr and 8-hr air quality distributions (50, 90, 95, 99, 99.5, 99.75 percentiles, and sixth largest value, second largest value, and largest value). The conclusion was that "the air quality adjustment procedures perform adequately in the upper-tail region (90th percentile and above) of the distribution, the region that determines the O_3 exposures of most concern in pNEM/ analyses" (Johnson, 1995). The adjustment procedures tended to overestimate 1-hr O_3 concentrations around the 50th percentile, however, because O_3 data follows a skewed distribution, the midrange value is typically closer to the 90th percentile than the 50th percentile. This limited evaluation indicates that the air quality adjustment procedures used in pNEM/O_3 do reasonably mimic changes in O_3 levels that have occurred in the past in the six urban areas evaluated. This does not guarantee that the adjustment procedure will accurately reflect future changes which depend on several variables including how controls influence the VOC/NO_x ratio, spatial patterns of growth, and transport of pollutants from other urban areas.

Because dose received by a person exposed to an air pollutant is highly dependent upon ventilation rate, exertion level is an important consideration in exposure modeling. The exposure model includes an algorithm that assigns the equivalent ventilation rate (EVR) associated with each exposure event. Clinical research by EPA suggests that there is less variability in EVR than in ventilation rate for a given level of exertion. The outdoor children version of pNEM/O_3 employs an EVR-generator module that uses one of four Monte Carlo models to generate an EVR value for each exposure event associated with a given cohort. The Monte Carlo models were developed through an analysis of data from two studies that measured heart rate of elementary and high school students while engaged in various typical daily activities (Spier et al., 1992; Linn et al., 1992). These studies then measured heart rate and ventilation rates simultaneously in a clinical setting to obtain a "calibration curve" for each subject relating heart rate to ventilation rate on a minute-by-minute basis. A regression analysis was applied to the Spier and Linn data bases to relate

various predictor variables contained in the diary data (e.g., daytime activities, day of week, breathing rate) that were considered likely to influence the EVR values.

The EVR-generator module also contained an algorithm which established an upper limit for the EVR value assigned to each exposure event. This limit was set at a level estimated to be achievable by members of the cohort who (1) exercised regularly, (2) were motivated to attain high exertion levels, and (3) were not professional athletes. Individuals engaged in amateur sports (e.g., jogging, tennis) would be included, but professional athletes would not be included. The EVR limit was derived based on a subset of the specified cohort (e.g., data from males aged 11 were used for the preteen group aged 6-13 and data from males aged 15 were used for the teenage group aged 14-18) and applied to the all members of the demographic group (either preteens or teenagers). Because the selected subset used in each case is likely to be higher than the average EVR limit for each demographic group, the pNEM/O_3 simulation will tend to overpredict the occurrence of high EVR values within each demographic group.

Extrapolate the Cohort Exposures to the Population of Interest. The cohort-specific exposure estimates were extrapolated to the general population, outdoor children, and outdoor workers by estimating the size of each cohort based on 1990 Bureau of Census (Bureau of Census, 1990) data files that list population data for age groups by census unit. The population in each census unit was multiplied by an air conditioning fraction (based on 1980 Bureau of Census data) in the specific census unit to provide an estimate of the number of outdoor children (or outdoor workers) in each air conditioning category. These air conditioning specific values were then summed over each exposure district to derive estimates for the entire study area.

3. Population Exposure Estimates Upon Attainment of Alternative Ozone Standards

The pNEM/O_3 contains a large number of stochastic variables and, therefore, exposure estimates will vary from run to run. For the general population, outdoor worker, and outdoor children exposure analyses, 10 simulations of pNEM were done for each regulatory scenario in each of the 9 urban areas to better characterize the uncertainty in the exposure estimates. Based on a previous analysis of sets of 10-run results versus a 108-run

result, McCurdy (1994b) has shown that results from only 10 runs of the model adequately predict the mean and variance observed in 100 or more runs of pNEM/O_3. Additional runs of the model would, however, increase the range of possible outcomes, but limited resources preclude undertaking more runs.

In any pNEM analysis, several different indicators are used to estimate exposure of people to various levels of air pollution. One 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. Another indicator is "occurrences of exposure:" the number of times a given level of pollution is experienced by the population of interest.

The exposure model provides exposure estimates in terms of both highest concentrations (exposures) or highest dose. The exposure estimates summarized here pertain to "daily maximum dose (MAXD)," where dose is defined as the product of O_3 concentration and ventilation rate over a defined period. The daily maximum dose does not necessarily occur during the time period of maximum O_3 concentration in a given urban area. The daily maximum dose indicator was selected because it is a better surrogate for the number of O_3 molecules that enter the oral-nasal cavities per unit time period, and therefore, is more likely to be more relevant from a health risk viewpoint than maximum exposure.

It should be stressed that the exposure model produces exposure estimates for the entire range of concentrations and that the health risk assessment, described in the next section, makes use of all exposures at a given exertion level that exceed an estimated background level of 0.04 ppm. Figure V-7 shows the exposure distributions for outdoor children living in the Philadelphia area experiencing daily maximum dose 8-hr exposures on one or more days while engaged in moderate exertion (EVR in the range 13-27 l/min-m^2). Attaining any of the alternative standards reduces the number of children experiencing daily maximum dose 8-hr exposures exceeding 0.1 ppm to less than 2,000 persons. At the 0.08 ppm level the number of children estimated to be exposed ranges from near 0 for the 0.07 ppm, 1 expected exceedance, 8-hr standard (8H1EX-0.07) to about 69,000 children for the 0.10 ppm, 1 expected exceedance, 1-hr standard (8H1EX-0.10). And at the 0.06 ppm level the number of children estimated to be exposed under moderate exertion for an 8-hr average

FIGURE V-7. EIGHT-HOUR MAXIMUM DOSE EXPOSURE DISTRIBUTIONS FOR OUTDOOR CHILDREN EXPOSED ON ONE OR MORE DAYS UNDER MODERATE EXERTION (EVR 13-27 LITERS/MIN-M2) IN PHILADELPHIA, PA

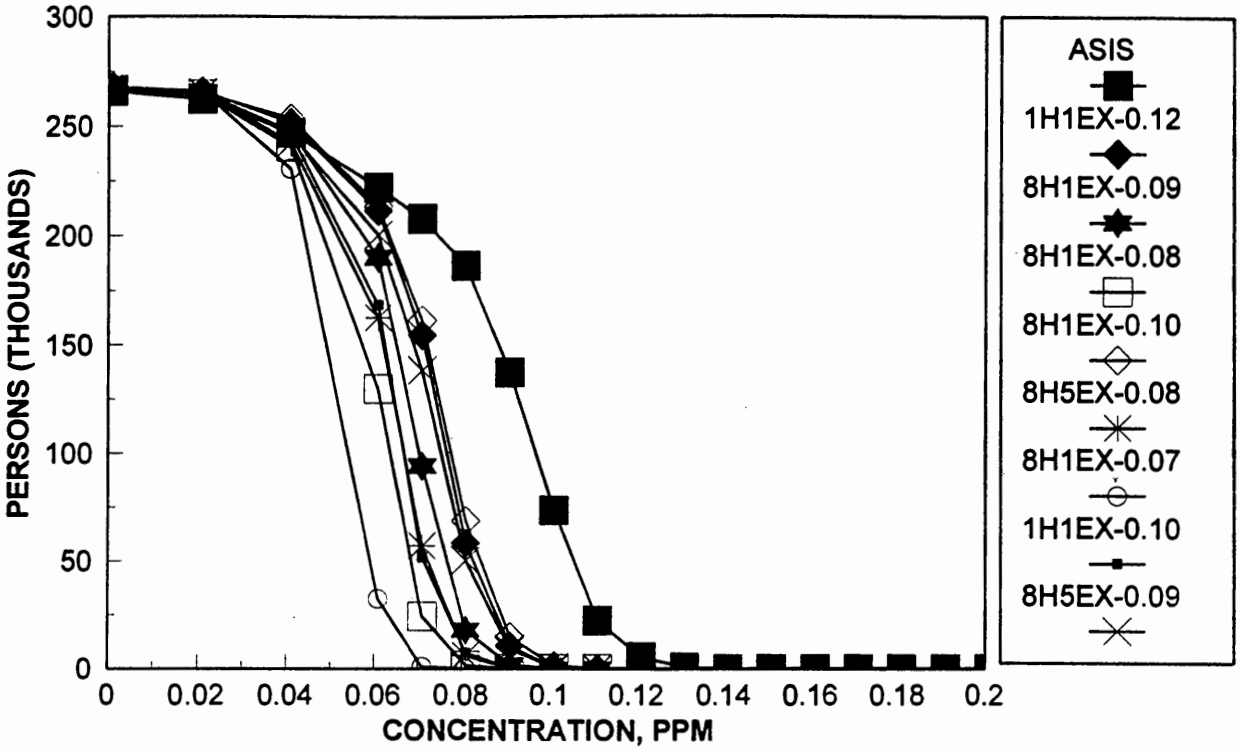
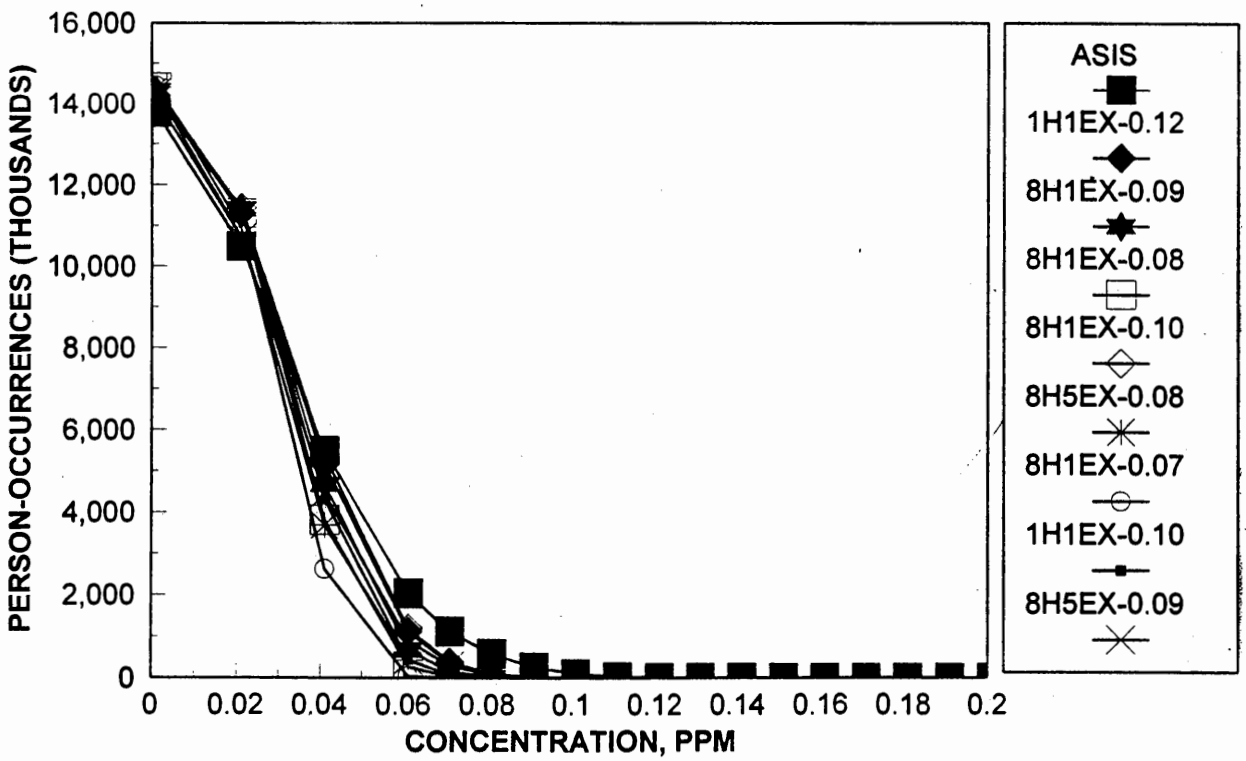


FIGURE V-8. EIGHT-HOUR MAXIMUM DOSE EXPOSURE DISTRIBUTIONS OF TOTAL OCCURRENCES FOR OUTDOOR CHILDREN EXPOSURE UNDER MODERATE EXERTION (EVR 13-27 LITERS/MIN-M2) IN PHILADELPHIA, PA



ranges from about 32,000 under the 8H1EX-0.07 standard to about 210,000 for the 8H1EX-0-.10 standard. Another exposure indicator of interest is the total number of person-occurrences at various concentration levels. Figure V-8 displays the person-occurrences for outdoor children experiencing daily maximum dose 8-hr exposures while engaged in moderate exertion in Philadelphia. It is estimated that there are roughly 14 million person occurrences of outdoor children engaged in moderate exertion for an 8-hr averaging time in Philadelphia over the 7-month O_3 season. Attaining any of the alternative standards reduces total person occurrences exceeding 0.06 ppm to less than 1.5 million. One can calculate an average number of exposures per person to a given level by dividing the total person occurrences at a given O_3 level by the number of children experiencing a given level one or more times per season. For exposures exceeding 0.06 ppm, the average number of occurrences per person for the alternative standards analyzed ranges from about 1.5 to 5. For exposures at or above 0.08 ppm the average number of occurrences per person drops to a range of 1.0 to 1.5. Similar figures showing persons and person-occurrences outdoor children living in three other urban areas (Houston, Washington, D.C., and New York) are included in Appendix B.

Tables V-10 and V-11 provide summary exposure estimates for the Philadelphia study area for outdoor children for 2 particular exposure indicators. Similar tables are available for outdoor children, outdoor workers, and the general population for the 9 urban areas in the exposure support documents cited at the beginning of this section. Table V-10 is for 1-hr MAXD exposure estimates where the O_3 concentration exceeded 0.12 ppm and EVR equaled or exceeded 30 l/min-m^{-2}, while Table V-11 is for exposures where the O_3 concentration exceeded 0.08 ppm and EVR in the range of 13-27 l/min-m^{-2}. These indicators were selected because they correspond to the lowest concentration levels at which effects were observed in the 1 to 2 hour clinical studies at heavy exertion ($\geq$ 30 l/min-m^2) and 6 to 8 hour studies at moderate exertion (13-27 l/min-m^2). Use of any single cutpoint in the exposure distribution to compare alternative standards must be done with caution. Using any single cutpoint does not adequately represent the differences in the entire exposure distribution between alternative standards.

TABLE V-10. ESTIMATES OF ONE-HOUR MAXIMUM DOSAGE EXPOSURES EXPERIENCED BY OUTDOOR CHILDREN IN PHILADELPHIA DURING WHICH OZONE CONCENTRATION EXCEEDED 0.12 ppm AND EVR[a] EQUALED OR EXCEEDED 30 LITERS· MIN^{-1}· M^{-2}

Statistic[b]	Regulatory scenarios - 1-Hour Standards		
	Baseline[c]	1H1EX-0.12 ppm	1H1EX-0.10 ppm
Mean Estimate of the Number of Outdoor Children	9,700	81	0
Percent of Total Outdoor Children Population	3.5	0.03	0.00
Range in this percentage for 10 runs	1.3-5.8	0.00-0.2	-
Mean Estimate of Person-Occurrences	10,100	81	0
Percent of Total Person-Occurrences	0.02	[d]	0.00
Range in this percentage for 10 runs	0.01-0.03	[d]	-

Statistic[b]	Regulatory scenarios - 8-Hour Standards						
	Baseline[c]	8H1EX-0.10 ppm	8H1EX-0.09 ppm	8H1EX-0.08 ppm	8H1EX-0.07 ppm	8H5EX-0.09 PPM	8H5EX-0.08 ppm
Mean Estimate of the Number of Outdoor Children	9,700	140	0	0	0	0	0
Percent of Total Outdoor Children Population	3.5	0.05	0.00	0.00	0.00	0.00	0.00
Range in this percentage for 10 runs	1.3-5.8	0.00-.21	-	-	-	-	-
Mean Estimate of Person-Occurrences	10,100	140	0	0	0	0	0
Percent of Total Person-Occurrences	0.02	[d]	0.00	0.00	0.00	0.00	0.00
Range in this percentage for 10 runs	0.01-0.03	[d]	-	-	-	-	-

[a]Equivalent ventilation rate = (ventilation rate)/(body surface area).
[b]Mean or range for 10 runs of pNEM/O_3.
[c]Baseline scenario is based on 1991 ambient air quality levels.
[d]Less than 0.01 percent.

TABLE V-11. ESTIMATES OF EIGHT-HOUR MAXIMUM DOSAGE EXPOSURES EXPERIENCED BY OUTDOOR CHILDREN IN PHILADELPHIA DURING WHICH OZONE CONCENTRATION EXCEEDED 0.08 ppm AND EVR[a] WAS IN THE RANGE 13-27 LITERS· MIN^{-1}· M^{-2}

Statistic[b]	Regulatory scenarios - 1-Hour Standards		
	Baseline[c]	1H1EX-0.12 ppm	1H1EX-0.10 ppm
Mean Estimate of the Number of Outdoor Children	186,000	58,400	7,500
Percent of Total Outdoor Children Population	67.7	21.2	2.7
Range in this percentage for 10 runs	65.6-70.2	18.7-24.3	1.1-4.4
Mean Estimate of Person-Occurrences	580,000	79,300	7,700
Percent of Total Person-Occurrences	0.98	0.13	0.01
Range in this percentage for 10 runs	0.89-1.1	0.12-0.17	0.01-0.02

Statistic[b]	Regulatory scenarios - 8-Hour Standards						
	Baseline[c]	8H1EX-0.10 ppm	8H1EX-0.09 ppm	8H1EX-0.08 ppm	8H1EX-0.07 ppm	8H5EX-0.09 PPM	8H5EX-0.08 ppm
Mean Estimate of the Number of Outdoor Children	186,000	68,800	18,000	1,630	0	50,300	6,200
Percent of Total Outdoor Children Population	67.7	25.0	6.5	0.6	0.0	18.3	2.3
Range in this percentage for 10 runs	65.6-70.2	20.9-30.2	4.3-10.7	0.1-2.3	-	15.1-21.8	0.9-4.8
Mean Estimate of Person-Occurrences	580,000	98,100	20,300	1,630	0	65,900	7,100
Percent of Total Person-Occurrences	1.0	0.17	0.03	e	0.0	0.11	0.01
Range in this percentage for 10 runs	0.89-1.1	0.14-0.20	0.02-0.06	0.00-0.01	-	0.08-0.14	0.0-0.02

[a]Equivalent ventilation rate = (ventilation rate)/(body surface area).
[b]Mean or range for 10 runs of $pNEM/O_3$.
[c]Baseline scenario is based on 1991 ambient air quality levels.
[d]Less than 0.01 percent.

In order to get a feel for how exposure estimates vary across the 9 urban study areas, Figures V-9, V-10, and V-11 show the mean percent of outdoor children exposed on 1 or more days to the 8-hr MAXD indicators of interest for the various regulatory scenarios analyzed. Figure V-9 presents these estimates for the "as is" situation, the current 1-hr standard, and a 0.10 ppm, 1-hr, 1-expected exceedance standard. The considerable variability in baseline O_3 levels across the urban areas analyzed results in large variation in "as is" exposure estimates. Except for Miami and Denver, which were either in or near attainment of the current 1-hr standard, exposures $\geq$ 0.08 ppm at moderate exertion would generally be significantly reduced upon attainment of the current 1-hr standard. Attaining the 0.10 ppm, 1 expected exceedance, 1-hr standard would further reduce exposures for this indicator. Figure V-10 presents similar estimates for the alternative 8-hr, 1-expected exceedance standards. Finally, Figure V-11 shows estimates for the 1- and 5-expected exceedance standards set at 0.08 and 0.09 ppm.

Some summary observations from the exposure analyses of the general population, outdoor workers, and outdoor children are listed below:

(1) For 8-hr exposures at moderate exertion (EVR in the range 13-27 l/min-m^2), outdoor children appear to have the highest percentage and number of individuals exposed to levels exceeding 0.08 ppm.

(2) For 1-hr exposures at heavy exertion ($\geq$ 30 l/min-m^2), it depends on the urban area whether outdoor children or outdoor workers have the highest O_3 exposures exceeding 0.12 ppm.

(3) While not shown in this Section, the exposure estimates for exceeding 0.12 ppm at any exertion level are considerably higher than the numbers and percentages presented in Tables V-10 and V-11. For example, the model predicts 269,000 (97.8 percent) outdoor children exceeded 0.12 ppm under baseline (1991 air quality) compared to only 9,700 or 3.5 percent of outdoor children living in the Philadelphia study area for this same baseline air quality when EVR was $\geq$ 30 l/min-m^2. Thus, exertion level and its associated ventilation rate at maximum dose significantly affects MAXD exposure estimates.

FIGURE V-9. MEAN PERCENT OF "OUTDOOR CHILDREN"
EXPOSED ON ONE OR MORE DAYS TO OZONE LEVELS >= 0.08 PPM
AT MODERATE EXERTION (EVR in the range 13-27 liters/min-m^2)

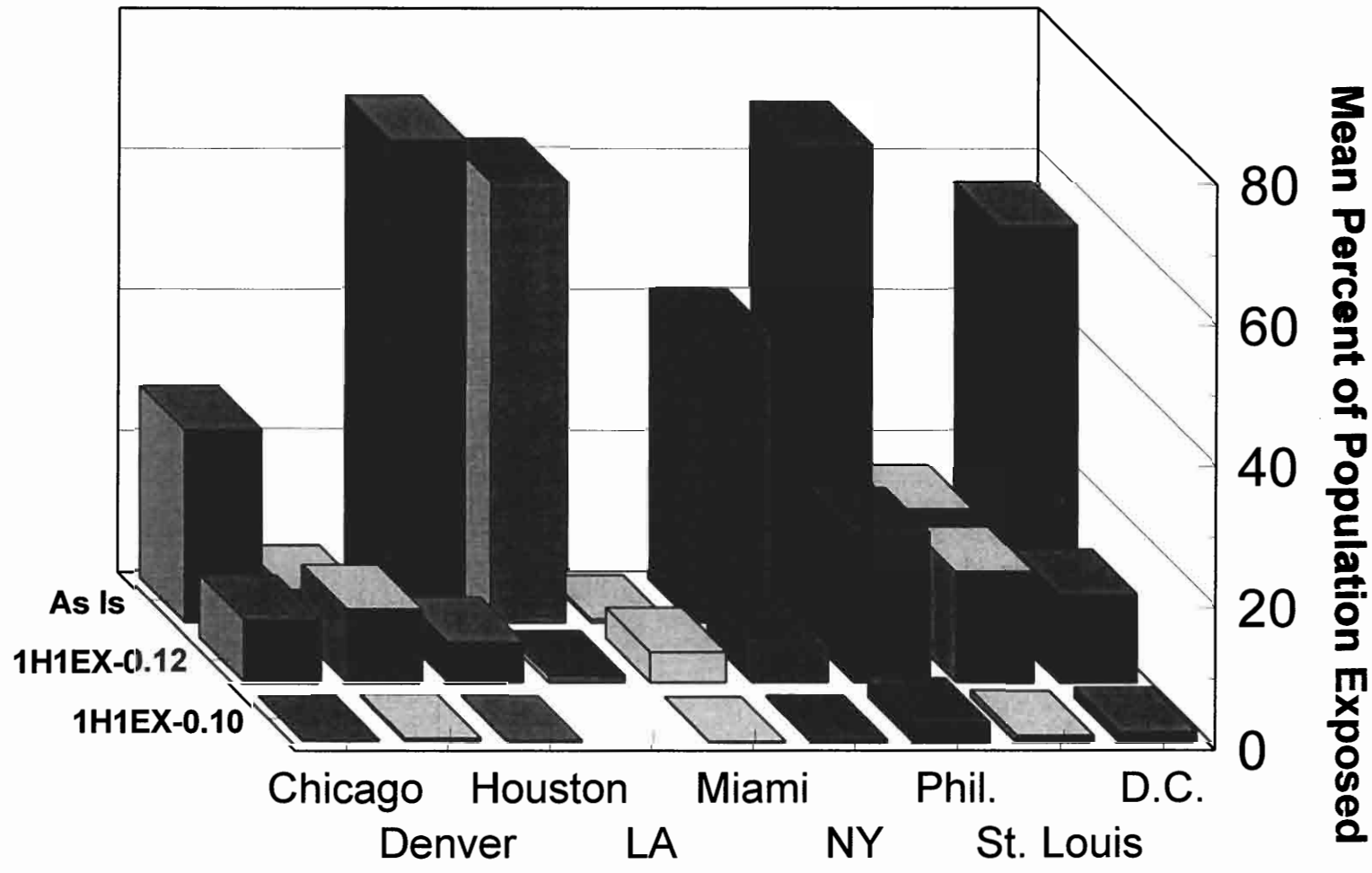


FIGURE V-10. MEAN PERCENT OF "OUTDOOR CHILDREN" EXPOSED ON ONE OR MORE DAYS TO OZONE LEVELS >= 0.08 PPM AT MODERATE EXERTION (EVR in the range 13-27 liters/min-m^2)

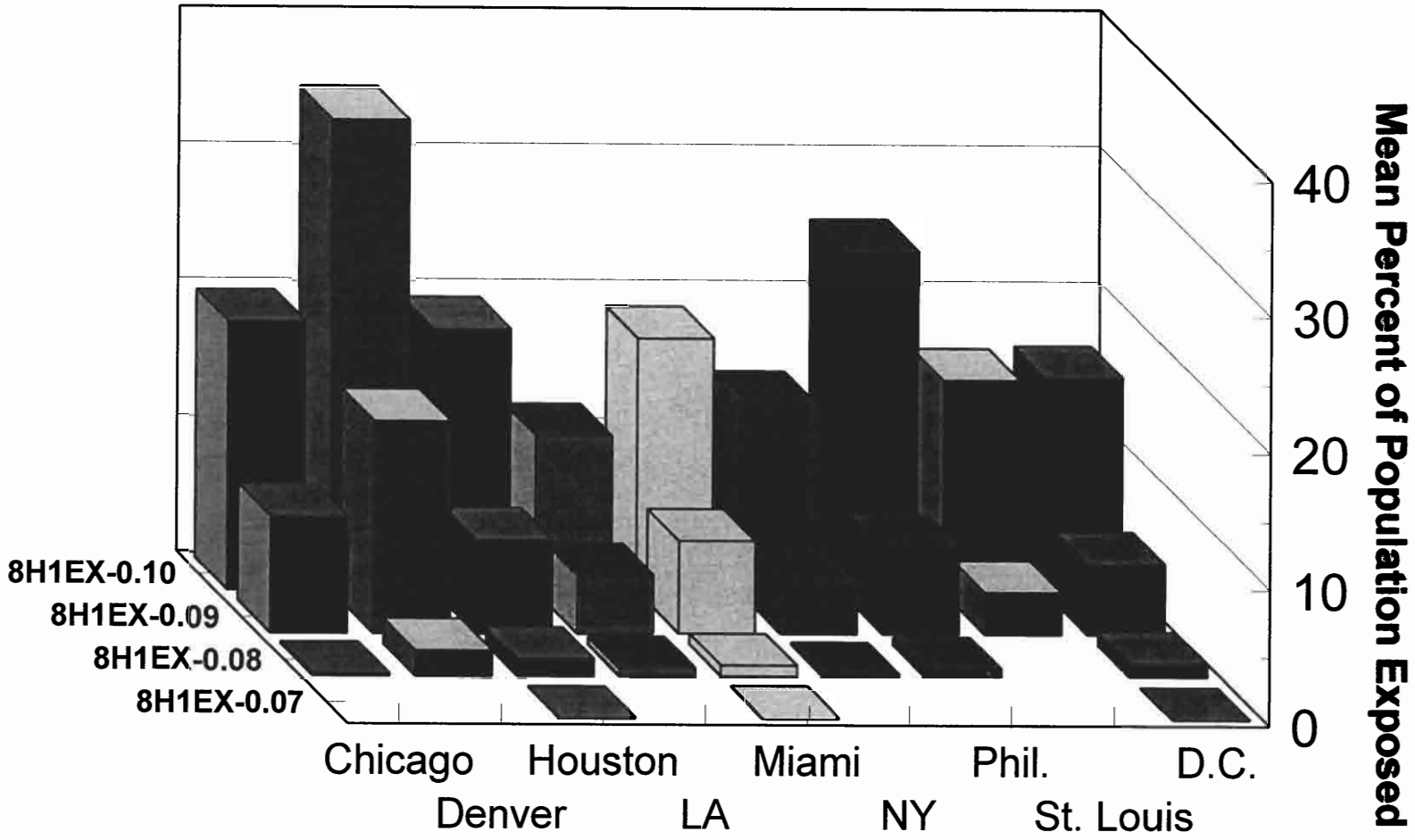
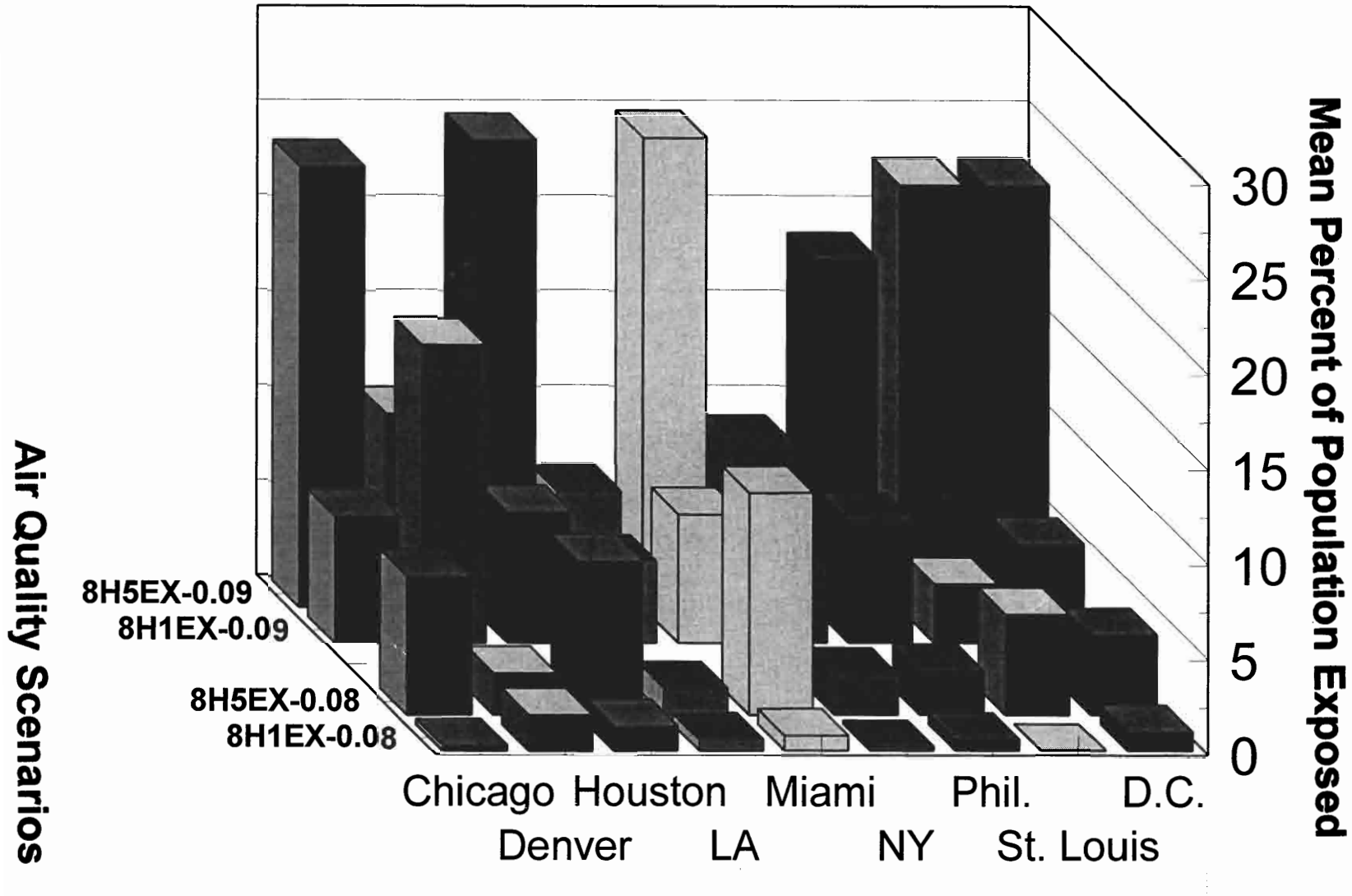


FIGURE V-11. MEAN PERCENT OF "OUTDOOR CHILDREN" EXPOSED ON ONE OR MORE DAYS TO OZONE LEVELS >= 0.08 PPM AT MODERATE EXERTION (EVR in the range 13-27 liters/min-m^2)



(4) On both an absolute number and percentage basis, exposure estimates are greater for the 8-hr, $\geq$ 0.08 ppm indicator at moderate exertion than the 1-hr, $\geq$ 0.12 ppm indicator at heavy exertion. This is not surprising since more people normally engage in moderate exertion than in heavy exertion activities.

(5) Based on the general population, outdoor worker, and outdoor children estimates, the 0.10 ppm, 1 exceedance, 8-hr regulatory option usually provides the least protection when judged by either 1- or 8-hr MAXD indicators and in most study areas results in greater exposures than the current 1-hr standard.

(6) There are relatively small differences in comparing the distributions of daily maximum dose 8-hr exposure estimates for outdoor children associated with 1- and 5-expected exceedance standards set at the same concentration level. However, if one selects a particular cutpoint on the distribution, such as exposures exceeding 0.08 ppm under moderate exertion, the differences between these two forms of the standard can appear to be more significant in some urban areas. In the vast majority of the nine study areas the 5-expected exceedance form of the standard results in greater exposures. However, in at least two study areas (i.e., Denver for the 8-hr daily max dose and St. Louis for the 1-hr daily max dose), the estimated exposures are somewhat greater for the 1-expected exceedance standard than the 5-expected exceedance standard set at 0.09 ppm. This seemingly illogical result occurs because the design value monitor for the 1-expected exceedance standard is different than the design value monitor for the 5-expected exceedance standard. This can occur because the site with the maximum second highest 8-hr daily maximum concentration is not always the same as the site with the maximum sixth highest 8-hr daily maximum value. The combination of significant differences in population sizes assigned to different districts in an urban study area and differences in the impact of the air quality adjustment procedure due to the design monitor being in different exposure districts for these two forms of the standard can lead to exposures being higher for a 1-expected exceedance form than the 5-expected exceedance form.

4. Caveats and Limitations

A number of caveats must be acknowledged concerning the pNEM/O_3 results. Probably the most important caveat is that there is considerable uncertainty concerning a number of important inputs to the model. Listed below are the most important caveats and limitations in the current version of the exposure model.

(1) The subjects who contributed to the human activity database may not provide a balanced representation of U.S. outdoor children or outdoor workers. The majority of subjects resided in either the State of California or in Cincinnati. Although the algorithm which constructs exposure event sequences attempts to account for effects of local climate on activity, it is unlikely that this adjustment procedure corrects for all inter-city differences in children's or outdoor workers' activities. Time/activity patterns are likely to be affected by a variety of local factors, including topography, land-use, traffic patterns, mass transit systems, and recreational opportunities.

(2) As discussed previously, the average subject provided less than two days of diary data. For this reason, the construction of each season-long exposure event sequence required either the repetition of data from one subject or the use of data from multiple subjects. The latter approach was used in the outdoor children and outdoor worker pNEM/O_3 analyses to better represent the variability of exposure expected to occur among the children included in each cohort. The principal deficiency of this approach is that it may not adequately account for the day-to-day repetition of activities common to individual children. Consequently, pNEM/O_3 may tend to under-estimate the number of people who experience multiple occurrences of high exposure while engaged in moderate or heavy exertion. For example, the outdoor children analysis does not adequately reflect exposures for children attending residential summer camps because this type of activity pattern is not included in human activity pattern data base used in the outdoor children exposure analysis.

(3) Exposure estimates have been presented separately for outdoor children, outdoor workers, and the general population and have not been aggregated.

Any aggregation would have to adjust the general population exposure estimates to avoid double counting exposures for workers and children.

(4) The algorithm that assigns the EVR associated with each exposure is based on an analysis of data from several studies conducted by Dr. Hackney and his associates in Los Angeles. Because of the small sample sizes (e.g., 39 children and 36 outdoor workers) in these studies and the lack of subjects below age 10 or above the age of 50 there is uncertainty which cannot be quantified about these EVR estimates. The pNEM/O_3 model also employs an EVR limiting algorithm that determines the maximum EVR that can be maintained for a given duration by an individual that exercised regularly and was motivated to reach a high ventilation rate. In general, the EVR limiting algorithm tends to allow more high EVR values to occur than would occur in the total population of interest (i.e., outdoor children, outdoor workers, or general population).

(5) The air quality adjustment procedures used to simulate just attaining alternative NAAQS were based on statistical analyses of O_3 data from sites that experienced moderate reductions in O_3 levels during the 1980's. These procedures assume that (1) the Weibull distribution provides a good fit to most O_3 data and (2) the parameters of the Weibull distribution fitting data from a particular monitoring site will change over time in a predictable fashion. Because of the empirical basis for the adjustment procedure, there is less confidence in the predicted air quality levels for just attaining alternative standards in Los Angeles where significant reductions would have to take place to attain any of the alternative standards analyzed. The adjustment procedure was developed and tested with a focus on the tail of the 1-hr and 8-hr air quality distributions. Therefore, there is more uncertainty about how well the adjustment procedure characterizes longer averaging times (i.e., seasonal 8-hr averages) and 1-hr and 8-hr daily maximum values that are in the middle of the distribution. A limited evaluation of the adjustment procedure (Johnson, 1995) suggests that the approach does a reasonable job of estimating the upper

10% of the distribution of hourly O_3 values based on an empirical analysis of six of the nine urban areas included in the exposure analysis. Further research and analysis is needed to better characterize uncertainty about possible changes in the spatial pattern and shape of O_3 air quality distributions associated with control strategies adopted to attain the O_3 NAAQS in the future.

(6) The pNEM/O_3 model uses a mass balance model to estimate O_3 levels in residential buildings (windows open), residential buildings (windows closed, nonresidential buildings, and inside motor vehicles. For some of these microenvironments the data base on air exchange rates (AER) and O_3 decay rates, which are key inputs to the mass balance model, is rather sparse. For example, the AER and O_3 decay rate for motor vehicles is a point estimate based on data for a single vehicle. In contrast, data on AER values for residential buildings with closed windows are based on a lognormal distribution fit to AER data from 312 residences across the U.S. It should be noted that the uncertainties about O_3 levels in these "indoor" microenvironments should not have a significant effect on exposure estimates at moderate and high exertion where exposure levels exceed 0.08 ppm, since these are likely to be due to outdoor exposures.

H. Ozone Health Risk Assessment

1. Overview

This section summarizes an assessment of risks for several categories of respiratory effects associated with attainment of alternative 1-and 8-hr O_3 NAAQS. This risk assessment builds upon the earlier O_3 NAAQS health risk assessment described in detail in Hayes et al. (1987) and summarized in the previous O_3 OAQPS Staff Paper (U.S. EPA, 1989). The O_3 health risk assessment considers the same alternative air quality scenarios examined in the exposure analysis described in Section V.G.

The objective of the risk assessment is to estimate the magnitude of risk to the most susceptible populations (i.e., outdoor workers and outdoor children) 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 estimate as to the possible 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 in the CD, aid the Administrator in judging which alternative O_3 NAAQS provides an adequate margin of safety. Risk estimates for nine urban areas and the methodology used to generate these estimates are described in detail in Whitfield et al. (1996).

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

(1) concentration-response or exposure-response relationships used to characterize various respiratory effects of O_3 exposure;

(2) distributions of O_3 1-hr and 8-hr daily maximum concentrations upon attainment of alternative NAAQS obtained from the pNEM/O_3 analyses described in Section V.G.; and

(3) distributions of population exposure, in terms of the general population, outdoor workers, and outdoor children exposed and occurrences of exposure, upon attainment of alternative O_3 NAAQS obtained from the O_3 exposure analyses.

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 activities at different exertion levels).

More specifically, benchmark risk is the probability that a time-averaged O_3 concentration will exceed a given benchmark concentration k or more times in a given period at some location within a geographic area. The benchmark concentration is the time-averaged O_3 concentration that will cause the occurrence of a specific health effect or response in up to a given percentage of a sensitive population (e.g., outdoor children) under given conditions of exposure. Benchmark risk, which is calculated assuming that all members of the sensitive population are exposed outdoors under identical exposure conditions, is a measure of the hazard posed by elevated ambient O_3 levels.

The second measure, headcount risk, is a population risk measure that assesses number of people or percent of the sensitive population that would be adversely affected given normal movement and activity patterns of the population of interest. Headcount risk also provides estimates of the number of occurrences 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 standard that provides an adequate margin of safety.

2. Exposure-Response Relationships

Risk estimates have been developed for a variety of respiratory effects reported to be associated with O_3 exposure. Table V-12 summarizes the effect categories covered by the risk assessment that are summarized in this section of the Staff Paper. Each of the effects is associated with a particular averaging time and for most of the acute (1 to 8-hr) responses effects also are estimated separately for specific EVR ranges that correspond to the EVR ranges measured in the health studies used to derive exposure-response relationships. An 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. Risk estimates are summarized

TABLE V-12. BASIS FOR ACUTE HEALTH ENDPOINTS ADDRESSED BY RISK ASSESSMENT

Health Endpoint	Exposure Time (Exertion Level)		
	1-hr (heavy)[a]	1-hr (moderate)[b]	8-hr (moderate)[c]
FEV1 decrement ≥ 10%, 15%, 20%	Avol et al., 1984 Kulle et al., 1983 McDonnell et al., 1985	Seal et al., 1993	Folinsbee et al., 1988 Horstman et al., 1989 McDonnell et al., 1991
Lower respiratory (moderate/severe) symptoms	Avol et al., 1984		
Cough (moderate/severe)	Kulle et al., 1985 McDonnell et al., 1983	Seal et al., 1993	Folinsbee et al., 1988 Horstman et al., 1989 McDonnell et al., 1991
Chest pain on deep inspiration (moderate/severe)	Kulle et al., 1985 McDonnell et al., 1983	Seal et al., 1993	Folinsbee et al., 1988 Horstman et al., 1989 McDonnell et al., 1991
Acute excess respiratory-related hospital admissions for asthmatics		Thurston et al., 1992[d]	

[a]Equivalent Ventilation Rate (EVR) ≥ 30 l min^{-1} m^{-2}

[b]EVR ≥ 16 and ≤ 30 l min^{-1} m^{-2}

[c]EVR ≥ 13 and ≤ 27 l min^{-1} m^{-2}

[d]There is no exertion level associated with the concentration-response relationship for this endpoint because it is based on epidemiological data that does not provide this information.

in this section for a variety of acute health endpoints. For lung function decrements estimates are provided for the lower end, midpoint, and upper end of the range of response that might be considered an adverse health effect (i.e., $\geq$ 10, 15, or 20% FEV_1 decrements). For acute symptomatic effects, this section focuses on responses that the staff recommends be considered as adverse effects (i.e., moderate or severe cough, moderate or severe pain on deep inspiration (PDI)).

Risk estimates have been calculated for each acute effect separately. Consequently, no risk estimates are available for multiple effects such as the joint probability of having moderate or severe cough and a FEV_1 decrement $\geq$ 15%. Preparing such joint effects risk estimates would be very difficult given the existing data base. The basis for these staff recommendations was discussed in Section V-F of this Staff Paper.

For the 1-hr, heavy exertion cases, exposure-response relationships were derived separately based on three controlled chamber studies (Avol et al., 1984; Kulle et al., 1985; and McDonnell et al., 1983) and used to develop independent risk estimates. Table V-13 summarizes the studies used to estimate 1-hr exposure-response relationships for populations engaged in heavy exertion. 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.

For the 1-hr, moderate exertion cases, exposure-response relationships were derived based on a single, relatively large controlled human chamber study (Seal et al., 1993). This study is summarized in Table V-14.

A pooled data set based on three controlled human exposure studies (Folinsbee et al., 1988; Horstman et al., 1989, McDonnell et al., 1991) served as the basis for developing exposure-response relationships for the 8-hr, moderate exertion cases. These studies are summarized in Table V-15. It was felt that these data sets could be pooled because the studies were performed in the same location using essentially identical experimental protocols

TABLE V-13. SUMMARY OF STUDIES USED IN DEVELOPING 1-HOUR EXPOSURE-RESPONSE RELATIONSHIPS FOR POPULATIONS ENGAGED IN HEAVY EXERTION

Study Protocol	Avol et al. (1984)	Kulle et al. (1985)	McDonnell et al. (1983)
Number of subjects	50 bicyclists: 42 male and 8 female; complete data were available for 48 of the subjects	20 healthy males; 8 of 20 subjects exposed to 0.30 ppm[a]	135 healthy males; complete data were available for 132 of the subjects
Exposure concentrations (ppm)	0.00, 0.08, 0.16, 0.24, and 0.32	0.00, 0.10, 0.15, 0.20, 0.25, and 0.30	0.00, 0.12, 0.18, 0.24, 0.30, and 0.40
Ventilation rate[b] (L/min)	57.6 ± 12.5	67.8 ± 8.2	65.6 ± 7.4
EVR[c] ($L/min/m^2$)	30.3	35.7	34.3 ± 3.1
Exercise pattern	Continuous (10-min warm-up, 60 min of continuous exercise, 10-min cooldown and measurement)	Intermittent (4 cycles of 14 min of exercise alternated with 16 min of rest)	Intermittent (4 cycles of 15 min of exercise and 15 min of rest)
Exercise duration (heavy) (min)	60	56	60
Exposure duration (h)	1.33	2	2.5[d]
Subject exposures	Exposed to all concentrations	Exposed to all concentrations	Divided about equally into 6 groups, each exposed to a single concentration

[a] Data for exposures of 0.30 ppm not reported in Kulle et al. (1985) were taken from Hayes et al. (1987).

[b] Mean ± standard deviation; averages of group (based on ozone concentration) means.

[c] Estimated for Avol et al. and Kulle et al. by dividing ventilation rate by 1.9 m^2, the approximate human body surface area, to obtain equivalent liters per minute; calculated for McDonnell et al. from available data.

[d] Includes a final 30-min period during which subjects rested, and spirometric and symptoms measurements were made.

TABLE V-14. SUMMARY OF THE STUDY USED TO DEVELOP 1-HOUR EXPOSURE-RESPONSE RELATIONSHIPS FOR POPULATIONS ENGAGED IN MODERATE EXERTION

Study Protocol	Seal et al. (1993)
Number of subjects	372 African-American and White males and females
Exposure concentration (ppm)	0.00, 0.12, 0.18, 0.24, 0.30, or 0.40
Mean ventilation rate[a] (L/min)	45
EVR[b] (L/min/m^2)	23.8 ± 2.8
Exercise pattern	Intermittent (4 periods of 15-min exercise, 15-min rest)
Exercise duration (h)	1
Exposure duration (h)	2.33[c]
Subject exposures	About 60 subjects exposed at each level; each subject exposed to only 1 concentration level

[a] Calculated from mean EVR by multiplying by 1.9 m^2, the approximate body surface area.

[b] Mean ± standard deviation; averages of group means.

[c] Includes a final 20-min period during which subjects rested, and spirometric and symptom measurements were made.

TABLE V-15. SUMMARY OF STUDIES USED TO DEVELOP 8-HOUR EXPOSURE RESPONSE RELATIONSHIPS

Study Protocol	Folinsbee et al. (1988)	Horstman et al. (1990)	McDonnell et al. (1991)
Number of subjects	10 nonsmoking males	22 nonsmoking males	38 nonsmoking males
Exposure concentration (ppm)	0.00 or 0.12	0.00, 0.08, 0.10, or 0.12	0.00, 0.08, or 0.10
Ventilation rate (L/min)	39-42[a]	37-41[a]	(40.3, 40.5, 39.6) ± (4.3, 4.3, 6.3)[b]
EVR (L/min/m^2)	20.5-22.1[c]	19.5-21.6[c]	(20.1, 20.2, 19.9) ± (1.8, 1.8, 2.3)[b]
Exercise pattern	50 min of exercise, 10 min of rest for each hour, and 35 min of rest after third hour	50 min of exercise, 10 min of rest for each hour and 35 min of rest after third hour	50 min of exercise, 10 min of rest for each hour and 35 min of rest after third hour
Exercise duration (h)	5	5	5
Exposure duration (h)	6.6	6.6	6.6
Subject exposures	Exposed to all concentrations	Exposed to all concentrations, except for 1 subject who experienced respiratory problems at 0.10 ppm	28 subjects exposed to 0.00 and 0.08 ppm; 10 subjects exposed to 0.00, 0.08, and 0.10 ppm

[a] Range of group means.

[b] Means ± standard deviation for 0, 0.08, and 0.10 ppm, respectively.

[c] Range of group means estimated by dividing the ventilation rate by 1.9 m^2, the approximate human body surface area, to obtain equivalent liters per minute.

and population groups. Several other controlled human exposure studies (summarized in Table 7-9 of the CD) have reported lung function decrements and symptoms in healthy and asthmatic subjects due to O_3 exposures lasting 6.6 to 8.0 hours (Horvath et al., 1991; Hazucha et al., 1992; and Linn et al., 1994; Horstman et al., 1995). These additional studies were not included in developing the estimated exposure-response relationships because they each involved a single exposure level and differences in study protocols precluded pooling the data from these studies with the Chapel Hill studies. The magnitude of the responses was somewhat lower in some of these studies, specifically the Linn et al. (1994) and Horvath et al. (1991), compared to the three Chapel Hill studies used in the risk assessment. However, this may have been due to use of a lower ventilation rate and attenuation due to previous exposures in Los Angeles for the Linn et al. (1994) study and the use of older, less sensitive subjects in the case of the Horvath et al. (1991) study. The responses of asthmatics in the Linn et al. (1994) and the Horstman et al. (1995) studies is more comparable to the level of responses seen in the three Chapel Hill studies used in the risk assessment.

The acute exposure-response relationships developed based on the chamber studies referenced in Table V-12 were applied to "outdoor children," "outdoor workers," and the general population. While these specific chamber studies only included adults aged 18-35, findings from other chamber studies (McDonnell et al., 1985) and summer camp field studies in at least six different locations in the northeast United States, Canada, and Southern California indicate changes in lung function in healthy children similar to those observed in healthy adults exposed to O_3 under controlled chamber conditions (CD, Section 9.3.1.2). As stated in the CD, "although direct comparisons cannot be made because of incompatible differences in experimental design and analytical approach," the range of response in the summer camp studies "is comparable to the range of response seen in chamber studies at low O_3 concentrations."

As discussed earlier in this SP, there is a growing data base of epidemiological studies reporting associations between increased acute respiratory-related hospital admissions and elevated O_3 levels during the summertime. In reviewing the studies reporting increased hospital admissions associated with elevated O_3 levels, only those studies which adequately

addressed statistical confounding by long-wave cycles in respiratory hospital admissions were considered due to concern that hospital admissions are clearly dominated by other causes (e.g., spring pollen, fall respiratory infection). Table V-16 (Table 7-23 from the CD) summarizes those studies that met this criterion. The concentration-response relationships for acute respiratory-related hospital admissions for asthmatics were derived based on one of these epidemiological studies (Thurston et al., 1992) that examined several New York cities. Only the data for the New York City population has been analyzed for the risk assessment. The choice of New York City was driven by the availability of O_3 hourly values for the entire O_3 season upon attainment of alternative standards which was produced for each of the nine urban areas as part of effort to develop exposure estimates. As shown in Table V-16, the effect size observed in other cities and studies that used the same O_3 indicator and general approach ranged from 1.4 to 3.1 admissions/100 ppb O_3/day/10^6 persons.

Concentration-response relationships are available for total excess respiratory-related admissions or excess respiratory-related admissions for asthmatics only. In this section, excess hospital admissions for asthmatics are summarized. Additional estimates, including total respiratory hospital admissions, are included in Appendix C and Whitfield et al. (1996). The Schwartz et al. (1994a,b,c) studies focus on only a subset of the total respiratory-related hospital admissions and were not analyzed in this risk assessment.

Given the lack of experimental human data, EPA sponsored an effort in 1990-1991 to develop a chronic lung injury risk assessment based on experts' judgments (Winkler et al., 1995). In the 1990-1991 assessment, the experts were explicitly told that their judgments were not being used as part of the NAAQS review process, but rather to gain a more general insight into the potential for chronic effects in areas with significantly elevated O_3 levels. Based on the age of this analysis and advice from the CASAC O_3 Review Panel, OAQPS is not considering the results from this chronic risk assessment in the current NAAQS review.

Table V-16. Summary of Effect Estimates for Ozone in Recent Studies of Respiratory Hospital Admissions[a]

Location	Reference	Respiratory Admission Category	Effect Size (± SE) [Admissions/100 ppb O_3/day/10^6 persons]	Relative Risk (95% CI)[b] [RR of 100 ppb O_3, 1-h max]
New York City, NY[c]	Thurston et al. (1992)	All	1.4 (± 0.5)	1.14 (1.06 to 1.22)
Buffalo, NY[c]	Thurston et al. (1992)	All	3.1 (± 1.6)	1.25 (1.04 to 1.46)
Ontario, Canada[c]	Burnett et al. (1994)	All	1.4 (± 0.3)	1.10 (1.06 to 1.14)
Toronto, Canada[c]	Thurston et al. (1994)	All	2.1 (± 0.8)	1.36 (1.13 to 1.59)
Montreal, Canada[d]	Delfino et al. (1994a)	All	1.4 (± 0.5)	1.22 (1.09 to 1.35)
Birmingham, AL[e]	Schwartz (1994a)	Pneumonia in elderly	0.73 (± 0.54)	1.11 (0.97 to 1.26)
Birmingham, AL[e]	Schwartz (1994a)	COPD in elderly	0.83 (± 0.33)	1.13 (0.92 to 1.39)
Detroit, MI[e]	Schwartz (1994b)	Pneumonia in elderly	0.82 (± 0.26)	1.22 (1.12 to 1.35)
Detroit, MI[e]	Schwartz (1994b)	COPD in elderly	0.90 (± 0.41)	1.25 (1.07 to 1.45)
Minneapolis, MN[e]	Schwartz (1994c)	Pneumonia in elderly	0.41 (± 0.19)	1.117 (1.03 to 1.39)
Minneapolis, MN[e]	Schwartz (1994c)	COPD in elderly	[f]	[f]

[a]See Appendix A in the Ozone Criteria Document for abbreviations and acronyms.
[b]One-way ($\beta \pm 1.65$ SE).
[c]1-h daily maximum ozone data employed in analysis.
[d]8-h daily maximum ozone data employed in analysis.
[e]24-h daily average ozone data employed in analysis. (1 h/24 h avg ratio = 2.5 assumed to compute effects and RR estimates).
[f]Not reported (nonsignificant).

Methodology for Developing Probabilistic Exposure-Response Relationships. A brief summary of the methods used to derive probabilistic exposure- and concentration-response relationships is described below. A more detailed description of the methodology can be found in Whitfield et al. (1996).

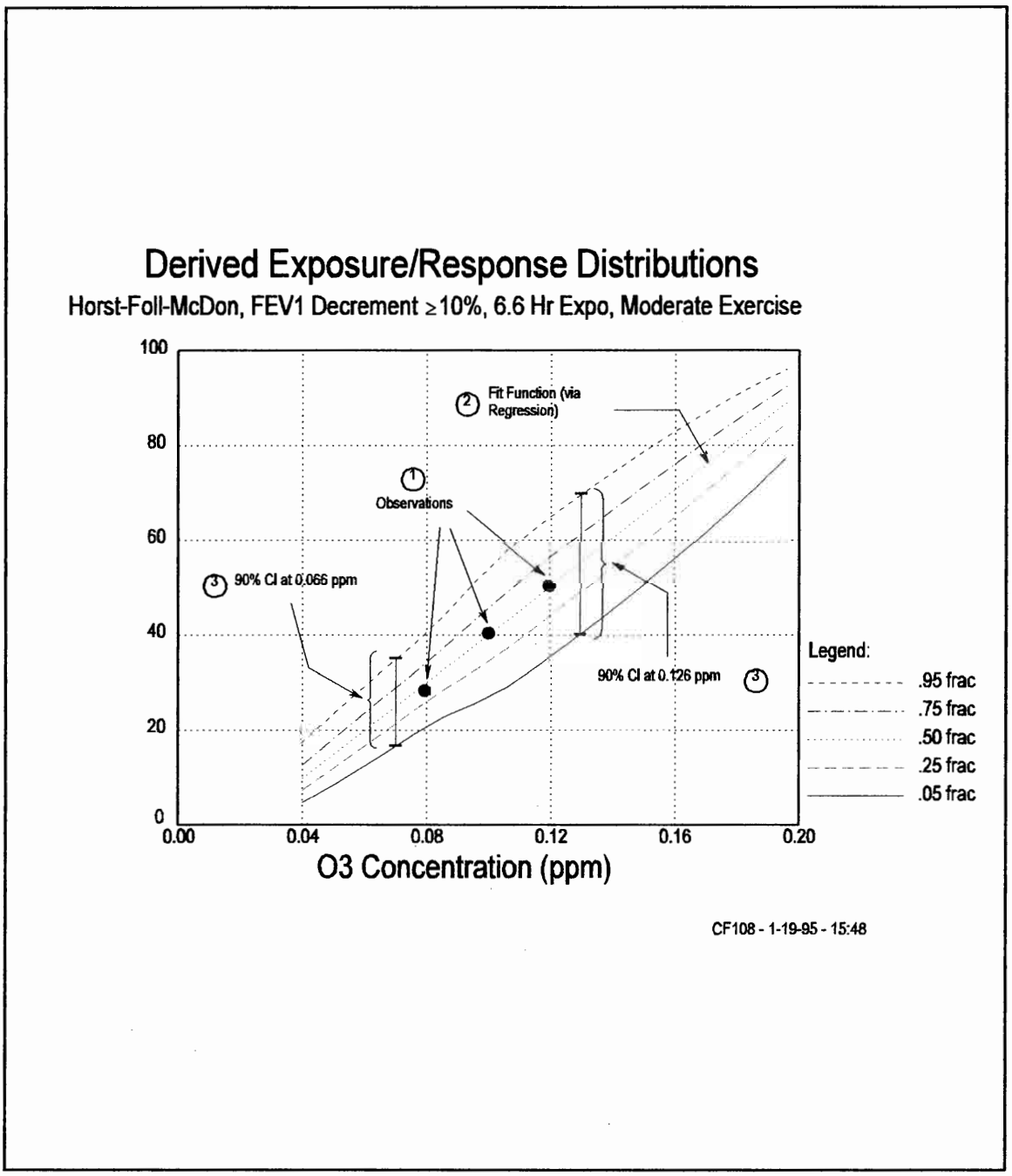
The development of exposure-response relationships for acute endpoints is a 3-step process. The starting point is data from the laboratory experiments described above. Before developing the needed probabilistic exposure-response relationships. The data were corrected for exercise in clean air in an effort to remove any systematic bias that might be present in the data attributable to an exercise effect. Generally, this correction for exercise in clean air was small relative to the total effects measured in the O_3-exposed cases. These data become the "observations" shown in Fig. V-12 and indicated by a 1 inside a circle to denote step 1.

Step 2 is to fit a function to the data via regression techniques. This step is necessary because of the need to estimate response rates at O_3 concentrations that differ from those at which laboratory data are available.

Step 3 is to develop, for example, the 90% credible interval about the fitted (predicted) response rate at O_3 concentrations needed for the risk assessment calculations (i.e., those used in pNEM). This step characterizes uncertainty attributed to sampling error based on sample size considerations. This uncertainty was estimated using a Bayesian approach involving the application of the inverse beta function with parameters X and N - X, where X is the predicted response rate at a particular O_3 concentration, and N is the number of subjects associated with the chosen O_3 concentration. The 90% CI is defined by the 0.05 and 0.95 fractiles.

For the risk assessment, response rates were calculated for 21 fractiles (for cumulative probabilities from 0.05 to 0.95 in steps of 0.05, plus probabilities of 0.01 and 0.99) at a number of O_3 concentrations that depended on the health endpoint. A function that "best fit" the data was chosen subject to the constraint that linear functions were favored, especially when the number of observation points (i.e.,O_3 concentrations at which laboratory data are available) was small. There are as few as 2 useable observation points and as many as 6 observation points for the endpoints examined.

FIGURE V-12. STEPS USED TO DEVELOP PROBABILISTIC EXPOSURE-RESPONSE RELATIONSHIPS.



Some illustrative concentration- and exposure-response relationships for some of the effects examined in the risk assessment are displayed in Appendix C. A table listing the functional form and parameters for all of the concentration-response and exposure-response relationships included in the risk assessment also is contained in Appendix C and discussed in more detail in Whitfield et al. (1996).

Other sources of uncertainty due to differences in experimental protocol, subject population, measurement error, etc. have not been quantitatively addressed for these acute health endpoints. The calculation and presentation of separate risk estimates for each of the three heavy exertion data sets provides a rough picture of the degree of uncertainty due to these other factors because this health endpoint was examined in more than one study.

3. Benchmark Risk Results

For the O_3 health risk assessment the benchmark risk is defined as the probability that, upon just attaining a given O_3 NAAQS, the daily maximum 1-hr (or 8-hr) concentration will equal or exceed the level that would cause 5 or 10% of the population of interest (e.g.,outdoor workers, outdoor children, or the general population) to exhibit particular health endpoints 1 or more times per year. The benchmark risk is estimated assuming the entire sensitive population is exposed under exertion levels associated with a particular effect.

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 population engaged in moderate or heavy exertion 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 used as a reasonable estimate of the background level for both 1- and 8-hr daily maximum concentrations experienced on a typical O_3 season day.

Benchmark risk is calculated by combining exposure-response relationships and probability distributions of daily maximum 1- or 8-hr O_3 ambient concentrations, based on conditions of exact attainment of alternative NAAQS. The benchmark risk model and more detailed discussion of the inputs to the model are contained in Whitfield et al. (1996).

Benchmark risk estimates are calculated for the 9 urban areas shown in Table V-7. Figure V-13 shows the benchmark risk estimates for Philadelphia for the 8-hr, moderate exertion health endpoint defined as FEV_1 decrements $\geq$ 20%. The solid vertical bars indicate the probability that upon attaining a given alternative standard O_3 levels will be exceeded five or more times in a season that would result in 5% of the population experiencing this endpoint if they were exposed while engaged in moderate exertion. The dashed vertical bars represent a similar measure, but for 10% of the population experiencing the specified endpoint. For example, attaining the 8H1EX-0.09 ppm standard results in a benchmark risk probability of around 0.95 that 5% of the population would experience an FEV_1 decrement $\geq$ 20% and the probability is about 0.7 that 10% of the population would experience this same health response, if the population were exposed to these O_3 levels while engaged in moderate exertion.

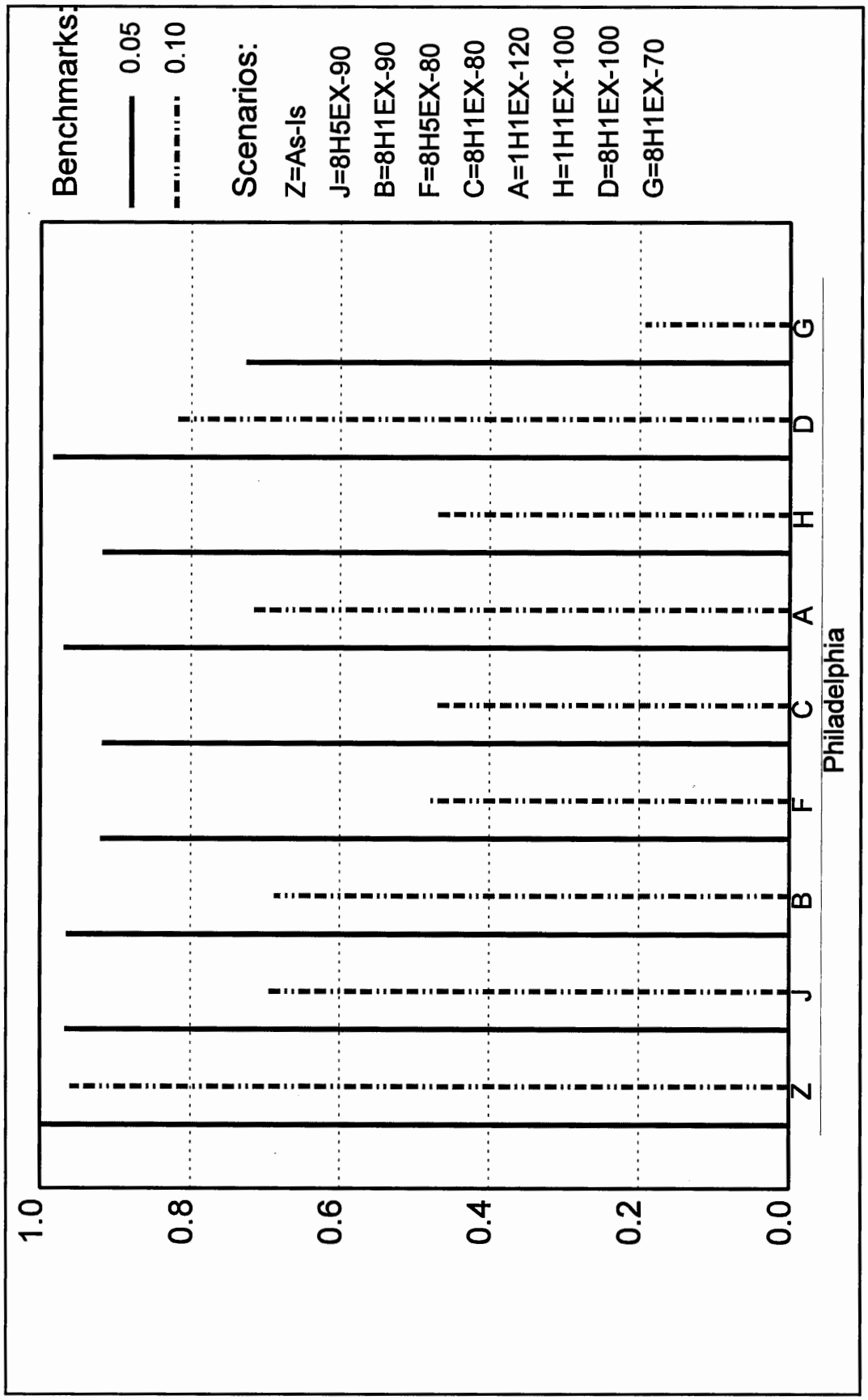
Benchmark risk estimates for selected health endpoints are presented in Appendix C. Additional health endpoints are included in Whitfield et al. (1996).

4. Population ("Headcount") Risk Results

Population, or "headcount," risk is characterized by calculating the 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. Risk estimates have been developed for the general population and for two groups expected to be at greater risk due to their increased activity or exertion level outdoors during the O_3 season: outdoor workers and outdoor children.

A major input to the headcount risk model is the series of population exposure distributions for the alternative NAAQS analyzed by EPA. Using available exposure estimates, risk estimates were calculated for the nine urban areas listed in Table V-7. For 8-hr exposures under moderate exertion, outdoor children represent the population group experiencing the greatest exposure, and, therefore, this population also has the highest risk estimates in terms of the percent of the population estimated to respond. Therefore, the remainder of this section focuses on the risk estimates for outdoor children. Whitfield et al. (1996) presents a more complete summary of the headcount risk estimates for each of the nine urban areas for outdoor children and outdoor workers. To illustrate the type of risk

FIGURE V-13. PROBABILITY THAT THE BENCHMARK RESPONSE FOR THE EIGHT-HOUR, MODERATE EXERTION HEALTH ENDPOINT FEV_1 DECREMENT ≥ 20% WILL BE EXCEEDED FIVE OR MORE TIMES IN AN OZONE SEASON.



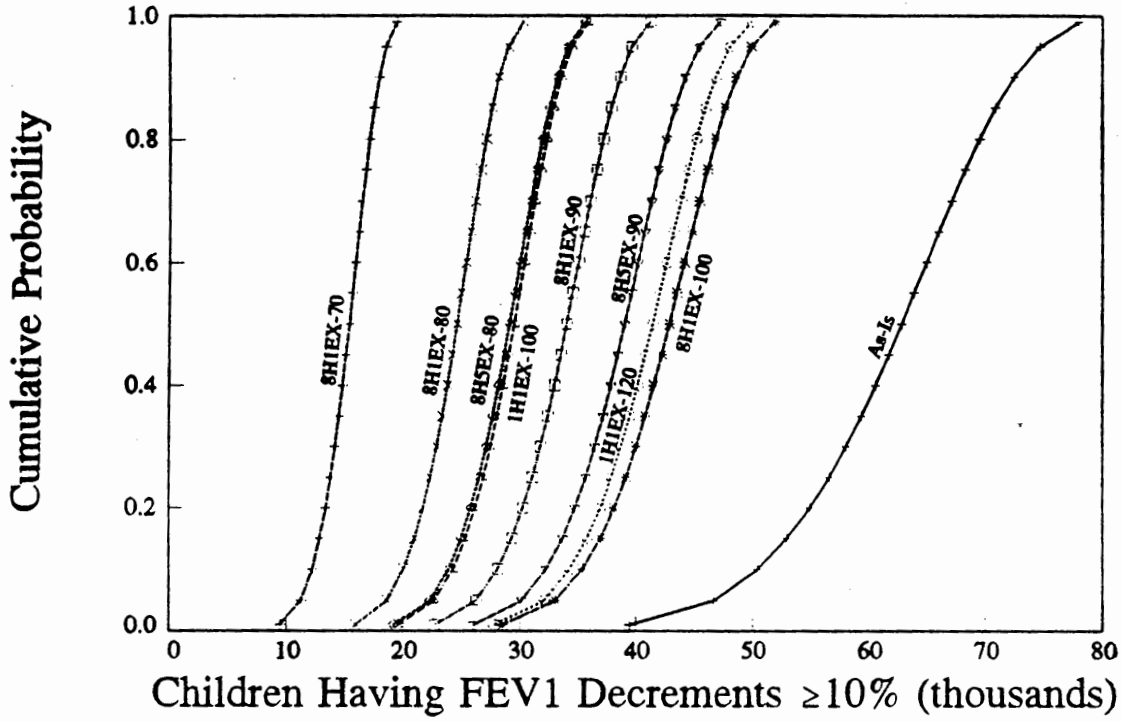
assessment output that is available, Figure V-14 shows cumulative probability distributions corresponding to just attaining alternative O_3 standards for two of the 8-hr, moderate exertion cases: percent of outdoor children estimated to have 1 or more occurrences of FEV_1 decrement $\geq$ 10% and percent of outdoor children estimated to have 1 or more occurrences of FEV_1 decrement $\geq$ 20% in Philadelphia. This figure shows, for example, that just attaining the 8H1EX-0.09 ppm standard in Philadelphia results in a median (0.5) probability that about 34,000 outdoor children would experience FEV_1 decrements $\geq$ 10%. When the health endpoint of interest is defined as FEV_1 decrement $\geq$ 20%, the median probability is that about 12,000 outdoor children would experience this effect. These risk estimates are for the number of children experiencing O_3-induced occurrences in excess of estimated background levels during a single O_3 season for the Philadelphia urban area. The variation due to the 10 different pNEM/O_3 runs for each alternative standard has been collapsed into a single representative distribution in order to better examine the differences between alternative standards.[5]

The top diagram in Figure V-15 shows outdoor children living in Philadelphia estimated to experience lung function decrements $\geq$ 15% one or more times in an O_3 season under moderate exertion for an 8-hr averaging time. Since any individual may experience multiple occurrences of an effect, risk estimates also have been developed for total occurrences of a specified effect. The bottom diagram in Figure V-15 displays total occurrences of this same response among outdoor children living in Philadelphia. As an example, Figure V-15 shows for that just attaining the 8H1EX-0.09 ppm standard in Philadelphia results in a median (0.5) probability that about 30,000 outdoor children would experience FEV_1 decrements $\geq$ 15% and a median probability that there would be 240,000 total occurrences of this effect. This results in an estimated average number of occurrences of eight per outdoor child for this endpoint.

The ratios for the mean number of occurrences and mean numbers of outdoor children responding have been calculated for three health endpoints (FEV_1 decrements $\geq$ 15% and $\geq$ 20% for 8-hour exposures under moderate exertion and moderate or severe PDI for 1-hour

[5] The representative distributions are obtained by effectively integrating across all 10 distributions for a given standard simultaneously and normalizing the result by dividing by 10. This calculation assumes that the distributions are perfectly correlated.

FIGURE V-14 REPRESENTATIVE RISK DISTRIBUTIONS FOR ALTERNATIVE AIR QUALITY SCENARIOS (FEV_1 DECREMENTS ≥ 10% AND ≥ 20%, PHILADELPHIA, OUTDOOR CHILDREN, 8 HR EXPOSURES, MODERATE EXERTION)



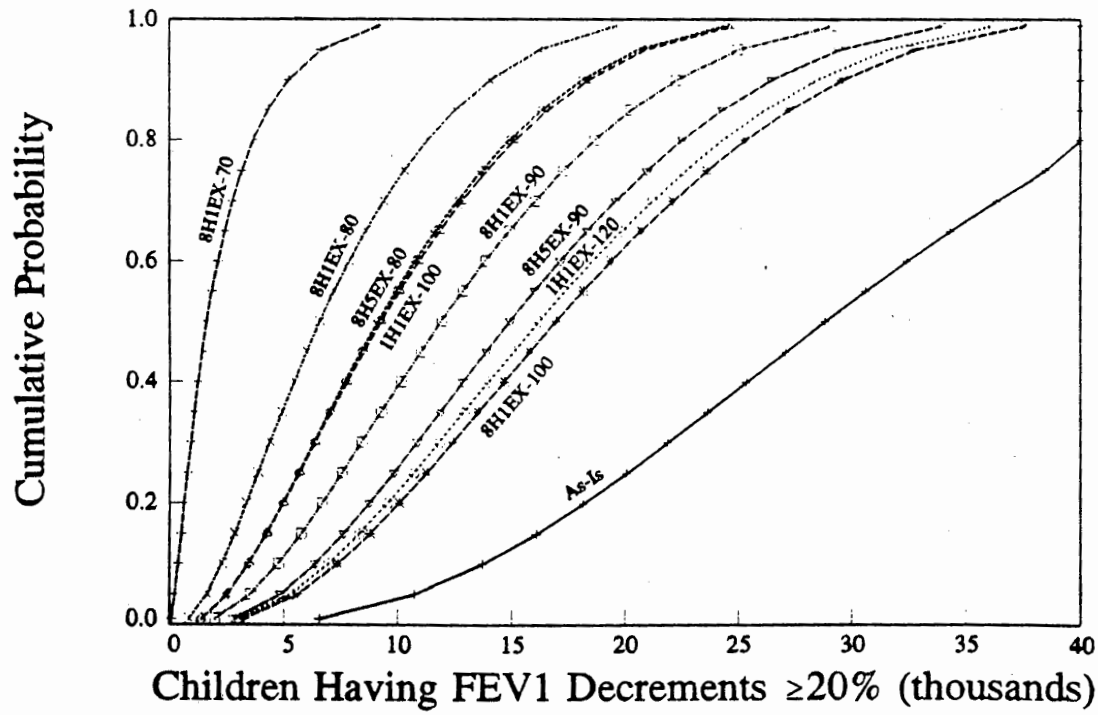
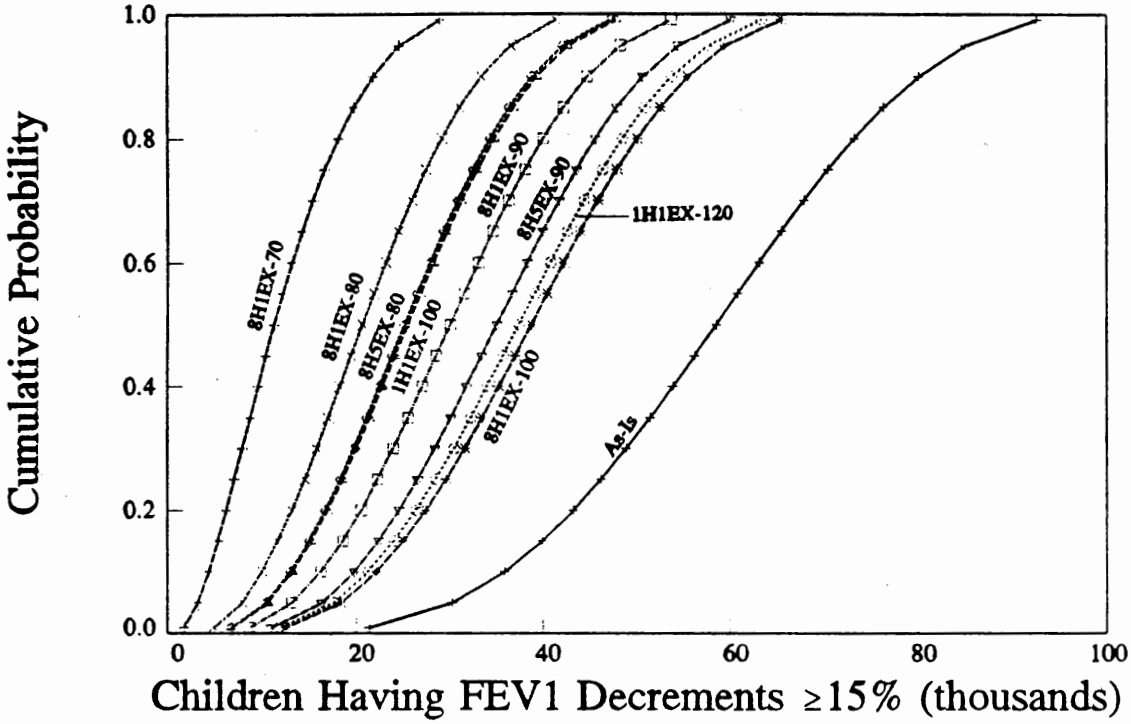
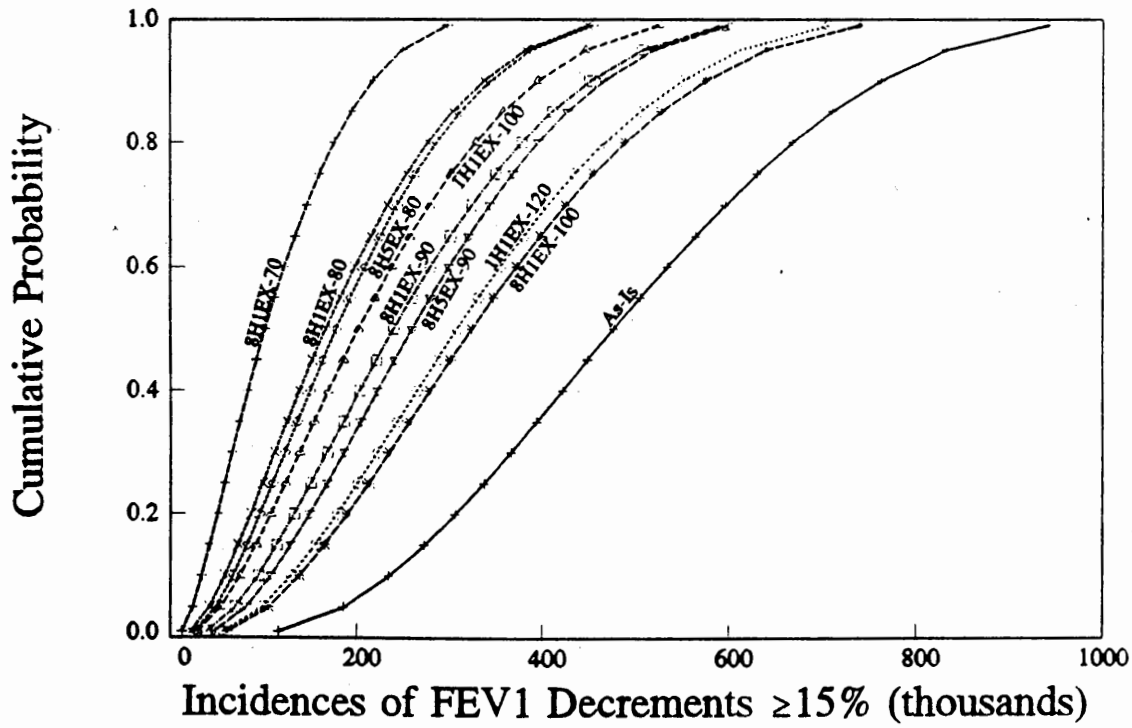


FIGURE V-15 REPRESENTATIVE RISK DISTRIBUTIONS FOR ALTERNATIVE AIR QUALITY SCENARIOS (FEV_1 DECREMENTS ≥ 15%, PHILADELPHIA, OUTDOOR CHILDREN, 8 HR EXPOSURES, MODERATE EXERTION)





exposures under moderate exertion) across the nine urban areas for five air quality scenarios (8H1EX-0.08 ppm, 8H5EX-0.08 ppm, 8H1EX-0.09 ppm, 8H5EX-0.09 ppm, 1H1EX-0.12 ppm). Figures C.24 through C.26 in Appendix C display these ratios. These ratios provide an estimate of the average number of times that a responder would experience the specified effect during an O_3 season. For the two 8-hr moderate exertion endpoints, the ratio ranges from about 4 to 8.7 for FEV_1 decrements $\geq$ 15% and from about 2 to 4.7 for FEV_1 decrements $\geq$ 20% across the nine urban areas. For the 1-hr moderate exertion endpoint defined as moderate-to-severe PDI, the ratio ranges from about 8 to 20 occurrences per responder across the nine urban areas. There is no consistent ordering or pattern in the ratios as one compares alternative scenarios across the different urban areas. In addition, there is no consistent pattern to the ratios among the alternative scenarios examined.

In order to facilitate comparison of risk estimates across the 9 urban areas, a central tendency risk estimate (the median values) for outdoor children for the acute 1- and 8-hr moderate exertion and 1-hr heavy exertion[6] health endpoints have been included in Tables V-17, V-18, and V-19 respectively. The range of risk estimates in each cell in these tables indicates the variability in risk that occurs as one compares the nine different urban areas. The range of risk estimates may be due to differences in the shape of the O_3 air quality distributions among the nine urban areas, differences in exposure due to different levels of air conditioning use in each urban area, or other differences in the exposure estimates such as the spatial pattern of population residences.

5. Excess Respiratory-Related Hospital Admissions.

As discussed earlier in this section, several epidemiology studies, mainly conducted in northeastern U.S. and southeastern Canada, have reported excess daily respiratory-related hospital admissions being associated with elevated O_3 levels during the O_3 season (see Table V-16). To gain insight into the possible impact of just attaining alternative 1- and 8-hr O_3 standards, OAQPS and ANL have developed a risk model for this endpoint for the New York City population. The model is based on the regression coefficients (and the corresponding standard errors) developed by Thurston et al. (1992) for New York City and

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[6] For the 1-hr, heavy exertion case, only the risk estimates based on the exposure-response relationships derived from McDonnell et al. (1983) are presented. Risk estimates based on Kulle et al. (1985) and Avol. (1984) are presented in Whitfield et al. (1995).

TABLE V-17. RANGE OF MEDIAN PERCENT OF OUTDOOR CHILDREN RESPONDING ACROSS NINE U.S. URBAN AREAS UPON ATTAINING ALTERNATIVE AIR QUALITY STANDARDS

1-hr Health Endpoints Under Moderate Exertion[a]	As-Is	Alternative 1- Hr Daily Maximum NAAQS		Alternative 8-Hr Daily Maximum NAAQS			
		1H1EX-0.12 ppm[b]	1H1EX-0.10 ppm	8H1EX-0.10 ppm	8H1EX-0.09 ppm	8H1EX-0.08 ppm	8H1EX-0.07 ppm
FEV_1 decrement $\geq$ 10%	2.9-22.0	2.4-6.1	0.5-1.9	4.0-8.8	2.7-6.3	1.8-6.3	1.1-3.1
FEV_1 decrement $\geq$ 15%	1.2-12.9	0.9-2.8	0.1-0.7	1.8-4.3	1.1-2.9	0.7-2.9	0.4-1.3
FEV_1 decrement $\geq$ 20%	0.6-8.1	0.5-1.5	0.1-0.3	1.0-2.4	0.6-1.6	0.4-1.6	0.2-0.6
Moderate or Severe Cough	0.0-7.6	0.0-0.4	0.0	0.1-1.2	0.0-0.5	0.0-0.5	0.0
Moderate or Severe Pain on Deep Inspiration	0.6-8.2	0.5-1.6	0.1-0.4	1.0-2.6	0.6-1.7	0.4-1.7	0.2-0.7

[a]Equivalent ventilation rate $\geq$ 16 l min^{-1} m^{-2} and $\leq$ 30 l min^{-1} m^{-2}.

[b]Current O_3 NAAQS.

TABLE V-18. RANGE OF MEDIAN PERCENT OF OUTDOOR CHILDREN RESPONDING ACROSS NINE U.S. URBAN AREAS UPON ATTAINING ALTERNATIVE AIR QUALITY

8-hr Health Endpoints Under Moderate Exertion[a]	As-Is	Alternative 1-Hr Daily Maximum NAAQS		Alternative 8-Hr Daily Maximum NAAQS			
		1H1EX-0.12 ppm[b]	1H1EX-0.10 ppm	8H1EX-0.10 ppm	8H1EX-0.09 ppm	8H1EX-0.08 ppm	8H1EX-0.07 ppm
FEV_1 decrement $\geq$ 10%	5.7-29.6	6.0-15.7	3.6-11.4	8.4-18.2	6.2-13.9	4.3-10.2	2.4-6.8
FEV_1 decrement $\geq$ 15%	4.2-27.1	4.6-13.7	2.6-9.4	6.9-16.1	4.9-11.9	3.3-8.3	1.7-5.1
FEV_1 decrement $\geq$ 20%	0.7-13.7	1.1-5.9	0.4-3.5	2.4-7.3	1.5-4.8	0.9-2.8	0.2-1.2
Moderate or Severe Cough	0.0-5.6	0.0-0.6	0.0-0.1	0.2-1.3	0.0-0.5	0.0-0.1	0.0
Moderate or Severe Pain on Deep Inspiration	0.0-7.8	0.0-0.3	0.0	0.1-1.1	0.0-0.2	0.0	0.0

[a]Equivalent ventilation rate $\geq$ 13 and $\leq$ 27 l min^{-1} m^{-2}.

[b]Current O_3 NAAQS.

TABLE V-19. RANGE OF MEDIAN PERCENT OF OUTDOOR CHILDREN RESPONDING ACROSS NINE U.S. URBAN AREAS UPON ATTAINING ALTERNATIVE AIR QUALITY STANDARDS[a]

1-hr Health Endpoints Under Heavy Exertion[b]	As-Is	Alternative 1-Hr Daily Maximum NAAQS		Alternative 8-Hr Daily Maximum NAAQS			
		1H1EX-0.12 ppm[c]	1H1EX-0.10 ppm	8H1EX-0.10 ppm	8H1EX-0.09 ppm	8H1EX-0.08 ppm	8H1EX-0.07 ppm
FEV_1 decrement $\geq$ 10%	0.1-4.4	0.1-0.5	0.0-0.2	0.2-0.7	0.1-0.5	0.1-0.2	0.0-0.1
FEV_1 decrement $\geq$ 15%	0.0-1.8	0.0	0.0	0.0	0.0	0.0	0.0
FEV_1 decrement $\geq$ 20%	0.0-1.3	0.0	0.0	0.0	0.0	0.0	0.0
Moderate or Severe Cough	1.0-7.0	1.4-3.7	0.9-2.6	1.9-3.9	1.5-3.3	1.0-2.5	0.7-1.9
Moderate or Severe Pain on Deep Inspiration	0.5-5.4	0.7-2.4	0.4-1.5	1.1-2.7	0.8-2.2	0.6-1.4	0.3-1.0

[a]Based on exposure-response relationships derived from McDonnell et al. (1983).

[b]Equivalent ventilation rate $\geq$ 30 l min^{-1} m^{-2}.

[c]Current O_3 NAAQS.

estimated daily maximum hourly average O_3 levels over an entire season at various monitors in New York City upon attainment of alternative standards developed by IT-AQS for the pNEM/O_3 analyses. Since the original published analysis only examined the relationship of excess hospital admissions and daily maximum 1-hr O_3 concentrations, we are unable to address at this time the relationship between 8-hr daily maximum O_3 concentrations and excess hospital admissions for the New York area. However, Delfino et al. (1994) report a similar effect size in their study involving excess hospital admissions where they used the 8-hr daily maximum O_3 concentration on the day prior to admission. Thurston et al. (1992) developed regression coefficients for two types of respiratory admissions: (1) for asthmatics only and (2) for total respiratory-related admissions. Since the results are fairly similar, only the risk estimates for asthma admissions are presented here. The regression coefficient (11.7 admissions/ppm $O_3/10^6$ people) for excess hospital admissions for asthmatics and its standard error (4.7 admissions/ppm $O_3/10^6$ people) were used to define a probabilistic concentration-response relationship. A 1-day lag is associated with O_3 exposure and the subsequent admissions of asthmatics. The model is described in more detail in Section 6 of Whitfield et al. (1996).

One hour daily maximum O_3 concentrations for one O_3 season under various alternative air quality standards were used to estimate the number of excess (i.e., attributable to O_3 concentrations higher than background) respiratory-related admissions of asthmatics. Risk estimates have been prepared using 4 different monitors in the New York City area (Queens-monitor 9, Greenwich-monitor 1, White Plains-monitor 11, and Babylon-monitor 12). The O_3 concentration-response relationship developed by Thurston et al. (1992) was based on air quality data from the Queens monitor. Therefore, the risk estimates based on the Queens County monitor most closely represent the air quality index used in the original study. In each analysis, the air quality was adjusted to just attaining a particular standard at the monitor with the highest O_3 levels for the New York area (i.e., the Babylon monitor) and the O_3 levels were adjusted at the other monitors using the procedures described in Johnson et al. (1996b).

The median estimate of the concentration-response relationship and the 0.05 and 0.95 fractiles estimates for O_3-induced, excess respiratory admissions for asthmatics in New

York City are shown in Figure V-16. Figure V-17 displays the cumulative probability function for excess annual hospital admissions attributable to O_3 for asthmatics, corrected for background O_3 of 0.04 ppm, for each of nine air quality scenarios.[4] Similar estimates also are available for excess total respiratory-related admissions attributable to O_3 exposure and are included in Whitfield et al. (1996).

The hospital admissions risk model produces a median estimate of nearly 390 excess annual admissions (corrected for background) for the 1991 "As Is" scenario using the Queens monitor and 214 day O_3 season. Thurston et al. (1992) examined unscheduled admissions during a 3-month period in 1988. When the hospital admissions risk model is limited to the same time period used in the original study and with background set at 0 ppm (the approach used in Thurston et al. (1992)), nearly identical excess daily admissions estimates are obtained (5.9 per day, s.d. 2.5).

To examine the impact of using alternative monitors to serve as the basis for an O_3 index for the New York City area population, the risk estimates for asthmatics and total respiratory hospital admissions also have been calculated using other monitors and are included in Whitfield et al. (1996).

Focusing on the estimates based on the Queens County monitoring site, the median estimate for O_3-induced hospital admissions for asthmatics in the New York City area is about 210 (90% credible interval (C.I.) = 70-344) upon attaining the current 1H1EX-0.12 ppm standard. This represents a nearly 50% decrease in O_3-induced admissions due to concentrations in excess of an estimated 0.04 ppm background compared to the As Is scenario. It is estimated that attaining an 8H5EX-0.08 ppm standard would reduce O_3-induced excess hospital admissions to about 120 (90% C.I. = 41-199) which represents a 70% decrease in O_3-induced admissions from the As Is scenario due to concentrations in excess of the estimated 0.04 ppm background.

There is very little difference in the risk estimates for excess hospital admissions between the 1- and 5-expected exceedance standards set at the same concentration level.

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[7] The population used in the hospital admissions analysis is smaller (i.e., 7.3 million people) than the New York urban area population used in the pNEM/O_3 analysis and risk estimates for the other health endpoints (i.e., 10.7 million people) and represents the same geographic area as in the Thurston et al. (1992) study.

FIGURE V-16. CONCENTRATION-RESPONSE RELATIONSHIP FOR DAILY HOSPITAL ADMISSIONS OF ASTHMATICS IN NEW YORK CITY AREA.

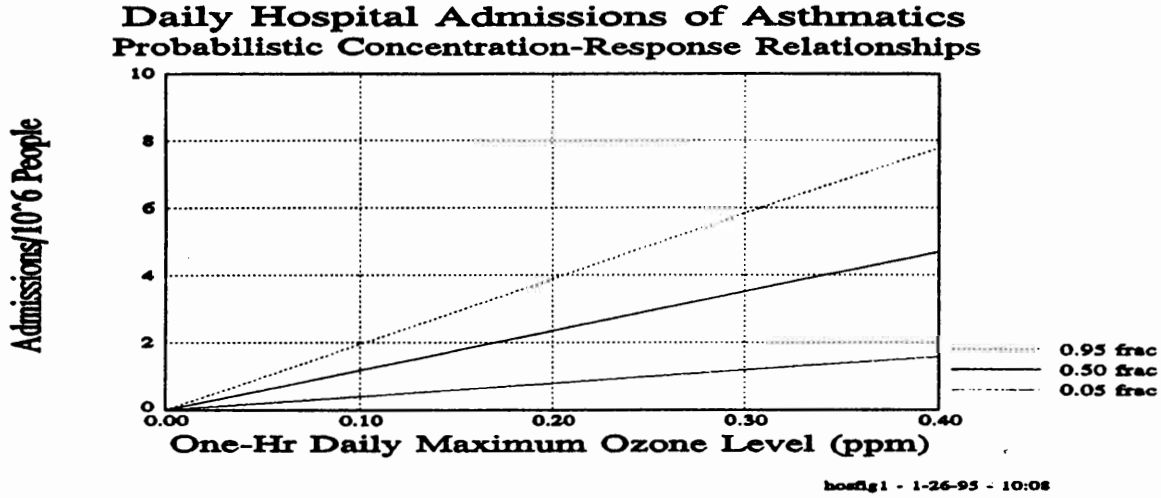
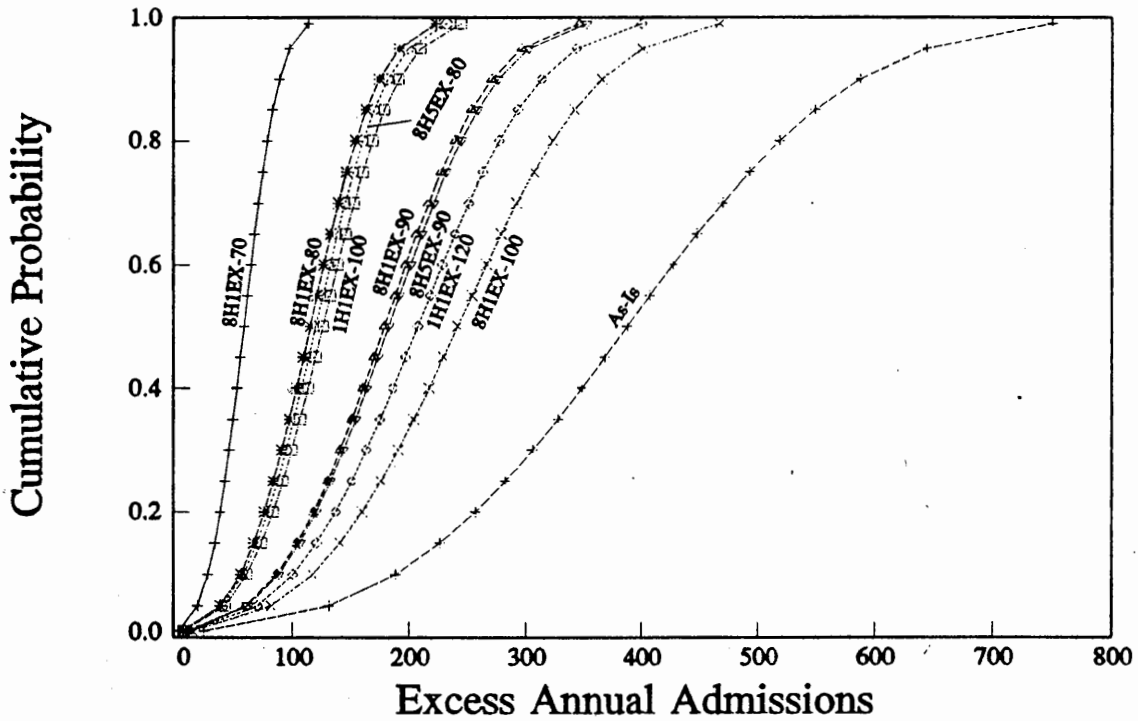


FIGURE V-17. EXCESS ANNUAL HOSPITAL ADMISSIONS OF ASTHMATICS ATTRIBUTABLE TO OZONE EXPOSURE FOR ALTERNATIVE AIR QUALITY SCENARIOS.



For example, the median risk estimate is 115 (90% C.I. = 39-191) for the 8H1EX-0.08 ppm standard and 120 (90% C.I. = 41-199) for the 8H5EX-0.08 ppm standard.

It should be recognized that estimated O_3-induced hospital admissions represents only a small portion of the overall respiratory-related hospital admissions for asthmatics from all causes. Another way to examine the risk results which highlights this point is presented in Table V-20. The excess admissions come from the hospital admissions risk model. The estimates for asthmatic respiratory-related hospital admissions due to all causes are based on (1) the 14-16 thousand admissions per O_3 season estimates provided by Thurston (1995) and (2) excess admissions attributable to exposures at O_3 levels > 0.04 ppm. As expected as the population base for comparison increases the percentage change relative to admissions associated with the current 1-hr standard decreases substantially. For example, the excess admissions associated with concentrations exceeding a 0.04 background results in a 42% reduction in admissions for the 8H5EX-0.08 ppm standard relative to just attaining the current 1H1EX-0.12 ppm standard. However, this represents only a 12% reduction in O_3-induced excess hospital admissions when the contribution of all O_3 concentrations are considered (i.e., background is set equal to 0 ppm). Finally, if the comparison is made in terms of all respiratory related admissions during the O_3 season, the reduction associated with attaining the 8H5EX-0.08 ppm standard relative to the current 1H1EX-0.12 ppm standard is only 0.6 percent.

6. Assumptions and Limitations Associated with the Health Risk Assessment

This section briefly summarizes a number of assumptions and limitations should be kept in mind in interpreting results of the O_3 health risk assessment. A fuller discussion of the assumptions and limitations is contained in Whitfield et al. (1996). These assumptions and limitations include the following:

(1) Extrapolation of Exposure-Response Relationships. In developing the probabilistic exposure-response relationships for the 1- and 8-hr health endpoints based on controlled human exposure studies it was necessary to extrapolate below the lowest exposure level used in these studies (i.e., 0.08 ppm for the moderate exertion studies used to represent 8-hr exposure-response relationships, 0.08 or 0.12 ppm for the heavy and moderate exertion

TABLE V-20. Admissions of New York City Asthmatics — With a Comparison Relative to Meeting the Current Standard (1 h, 1 expected exceedance, 0.12 ppm)

Issue	Air Quality Scenarios			
	1H1EX-0.12 ppm (Scenario A)	8H1EX-0.08 ppm (Scenario C)	8H5EX-0.08 ppm (Scenario F)	As-Is (Scenario Z)
Excess Admissions[b] (background = 0.04 ppm)	207[c] (70, 344)[d]	115 (39, 191)	120 (41, 199)	388 (132, 644)
% Change from Current Standard[e]	0	??	??	??
Excess Admissions[b] (background = 0 ppm)	909 (308, 1,509)	804 (273, 1,336)	797 (270, 1,324)	1,065 (361, 1,768)
% Change from Current Standard[e]	0	-11.6	-12.3	17.2
All Admissions[f] (thousands)	14,819.00	14,727.0	14,732.0	15[g] (14-16)[g]
% Change from Current Standard[h]	0	-0.6	-0.6	-99.9

[a] Expected exceedance.

[b] Admissions of asthmatics attributable to exposure to ozone.

[c] Median estimate.

[d] 90% credible interval (about the median).

[e] Because of the necessary assumption that results across scenarios are highly correlated (i.e., if admissions are high for one scenario, they are high for all scenarios), there is very little variation in the percentage change from the current standard.

[f] Admissions of asthmatics for any respiratory-related reason; for scenario*i*, based on estimates of all admissions and excess admissions attributable to ozone levels >0.04 ppm for As-Is scenario, and estimate of excess admissions attributable to ozone levels >0.04 ppm for scenario*i* (e.g., for scenario 1H1EX-0.12 ppm: 14,800 ≈ 15,000 - 388 + 207).

[g] Admissions of New York City asthmatics for any respiratory-related reason during the 1988-90 ozone seasons (Thurston, 1995).

[h] Variation in these results is attributable to the different numbers of admissions of New York City asthmatics for any respiratory-related reason during the 1988-90 ozone seasons (Thurston, 1995).

studies used to develop 1-hr exposure-response relationships). Based on an initial sensitivity analysis, a significant portion of the estimated risks are due to exposures between the estimated background value of 0.04 ppm and the lowest measured values in the various controlled human exposure studies relied upon in this risk analysis. The CASAC O_3 Exposure and Risk Subcommittee generally supported the extrapolation of modeled exposure-response relationships when they reviewed the proposed risk assessment methodology in March 1994.

(2) Exposure and air quality estimates. A major input to the headcount risk estimates for the general population, outdoor workers, and outdoor children is the O_3 exposure analysis estimates for these populations. 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. There are uncertainties about the appropriate O_3 monitor to use in applying the excess respiratory-related hospital admissions risk model to the New York City area and in the procedures used to adjust O_3 levels to just attaining alternative air quality standards in the New York area. Benchmark risk estimates for the other acute health endpoints are affected by uncertainty in projecting O_3 concentrations upon attainment of alternative NAAQS at the design value monitor. In addition, the values selected as representing reasonable estimates of background concentrations for 1- and 8-hr daily maximum levels are subject to uncertainty. Since all of the risk estimates presented here are calculated as O_3-induced risk in excess of background, alternative values for background could potentially alter the risk estimates. Argonne National Labs has conducted some limited sensitivity analyses in two of the urban areas to examine the influence of different background assumptions on selected headcount risk estimates. Generally, the results indicate that O_3 exposures in the range of 0.04-0.06 ppm contribute little to the total headcount risk

estimates. Therefore, alternative values for background in the range 0.03-0.06 ppm are likely to have little impact on the overall risk estimates. The results of this limited sensitivity analysis is described in more detail in Whitfield et al. (1996).

(3) Age. The risk assessment has been applied to the general population, outdoor workers, and outdoor children. 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.

(4) Attenuation or enhancement of response. For the acute health endpoints, 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 O_3-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.

(5) Interaction between O_3 and other pollutants. The controlled human exposure 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. Analysis of lung function decrement data from several field studies of children at a variety of summer camps in the northeast has found similar O_3-induced lung function changes as observed in the controlled human exposure studies (see pp.9-7 and 9-8 of the CD).

(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 the general population, outdoor workers, and outdoor children regardless of smoking status. To the extent that smokers are less responsive than nonsmokers, risk estimates may be overstated.

(7) Selection of Averaging Time. In developing risk estimates for 1-hr exposures at moderate and heavy exertion, data from 1- to 2 ½-hr controlled human exposure studies were used and matched with 1-hr exposures at moderate or heavy exertion. The studies that ran longer than an hour were conducted with intermittent exercise periods. McKittrick and Adams (1994) has reported that lung function responses were very similar for subjects exposed either continuously exercising for 1 hour or exposed for 2 hours with intermittent exercise. In matching the 1-hr exposure-relationships to 1-hr exposure estimates, the EVR range selected for the exposure estimates was selected to match the hourly average EVR in the health effects studies. The 8-hr, moderate exertion risk estimates were developed based on three controlled human exposure studies that were conducted using a 6.6-hr exposure period. Since the lung function response appears to level off after 4-6 hours of exposure, it is unlikely that the exposure-response relationships would have been appreciably different, even if the studies had been conducted for 8 hours.

(8) 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 Avol and Kulle data sets by Hayes et al. (1987b) also supports the reproducibility of individual responses.

I. Alternative Forms of the Primary NAAQS

1. Form of the Current Standard

The current primary O_3 NAAQS has a level of 0.12 ppm, an averaging time of 1 hour, and is expressed in a "1 expected exceedance" form. That is, the standard is formulated on the basis of the expected number of days on which the level is exceeded. More specifically, the attainment test specifies that the expected number of days per year on which the level is exceeded be equal to or less than 1.0 (values less than 1.05 are rounded down), averaged over a three-year period, and that specific adjustments be made for missing monitoring data. The standard is applied on a site-by-site basis; data from multiple sites are not combined. These procedures have remained unchanged since the original promulgation in 1979.

2. Issues Associated with Consideration of Alternative Forms

As the above description of the current standard illustrates, the "standard" is defined by more than just its level. The following elements have been used in the formulation of air quality standards:

- the level, e.g., 0.12 ppm,
- the averaging time, e.g., 1 hour,
- the NAAQS statistic, e.g., the number of exceedances,
- the attainment test criteria, e.g., expected number of exceedances equal to or less than 1.0,
- the length of the compliance period, e.g., 3 years, and
- data handling conventions, e.g., adjustments for missing monitoring data.

The staff is considering alternatives for the NAAQS statistic, the attainment test criteria, and the data handling conventions which address some of the concerns about the stability of the attainment test and the missing data adjustment procedure which were raised during public review of the Clean Air Act O_3 Design Value Study (EPA, 1994). State agency and industry representatives expressed concern about:

(1) the probability of misclassification between attainment and nonattainment,

(2) the possibility of areas moving in and out of attainment ("flip/flops") with each additional year of data,

(3) the significant impact of year to year variability in meteorological conditions conducive to O_3 formation,

(4) the need for a more "robust" test statistic (e.g., more exceedances or a lower percentile statistic)

(5) the impact of using only a single monitor within a large network of monitoring stations,

(6) the lack of consideration of population exposure,

(7) the impact of the adjustment for missing data (e.g., areas with only three observed exceedances can fail to meet the standard if they have less than 95 percent data completeness), and

(8) the impact of O_3 transport on downwind areas.

Some of these concerns will be addressed in this standard review, while others will be considered in the development of associated new implementation strategies.

As part of this standard review, the staff is evaluating alternative approaches to specifying the NAAQS statistic and attainment test criteria that are designed to 1) better reflect the relationship between air quality and human exposure and risk; 2) increase the stability and, hence, reduce the likelihood of attainment/nonattainment flip-flops; and 3) address missing data issues. The approaches currently under consideration by the staff include:

- alternative, less variable NAAQS statistics,
- alternative attainment test criteria, including the use of a range, rather than a bright-line standard, and
- alternatives for the treatment of missing data.

Of foremost consideration in evaluating alternative forms for the primary standard is an assessment of the adequacy of the health protection provided. The staff is considering whether alternative forms address concerns raised with regard to the current standard without introducing other problems of equal, or greater concern. The staff is also giving major consideration to the feasibility of implementation and the infrastructure needed to implement alternative forms, such as the adequacy of the current ambient monitoring network. Public policy issues associated with these various alternative approaches that the staff is considering include consistency with Clean Air Act requirements, environmental equity

considerations, and the ability to effectively communicate any change in the standard to the public at large.

The staff has determined that it is more appropriate to consider misclassification and transport issues within a new implementation strategy framework. For example, the California Air Resources Board (CARB) has already implemented an approach to address misclassification by defining a nonattainment "transitional" category. Although this new category helps to reduce the probability of misclassifying borderline sites, there are important implementation considerations if such an approach were to be introduced. These include how long an area can remain in this category, and what emissions reductions, if any, would be required. The level of health protection intended by the NAAQS is not altered by this approach as long as the area eventually comes into attainment with the NAAQS.

3. Alternative NAAQS Statistics

The NAAQS statistic for the current standard is the annual expected exceedance rate. The staff has considered the use of the design value, which is a concentration-based statistic, as an alternative NAAQS statistic. Use of a concentration-based statistic is consistent with health concerns, and is also consistent with the form of the current standard, in that the level of the standard necessary to provide a given degree of health protection would be the same for a design value NAAQS statistic standard as an annual expected exceedance form. The primary reasons for considering such a change are that (1) the design value has greater temporal stability than expected exceedances, (2) it is more directly related to the database characterizing health and welfare effects, and (3) the dichotomy with currently exists between the expected exceedances attainment test and the design values is eliminated because with a concentration based standard the attainment test and the design value are the same statistic.

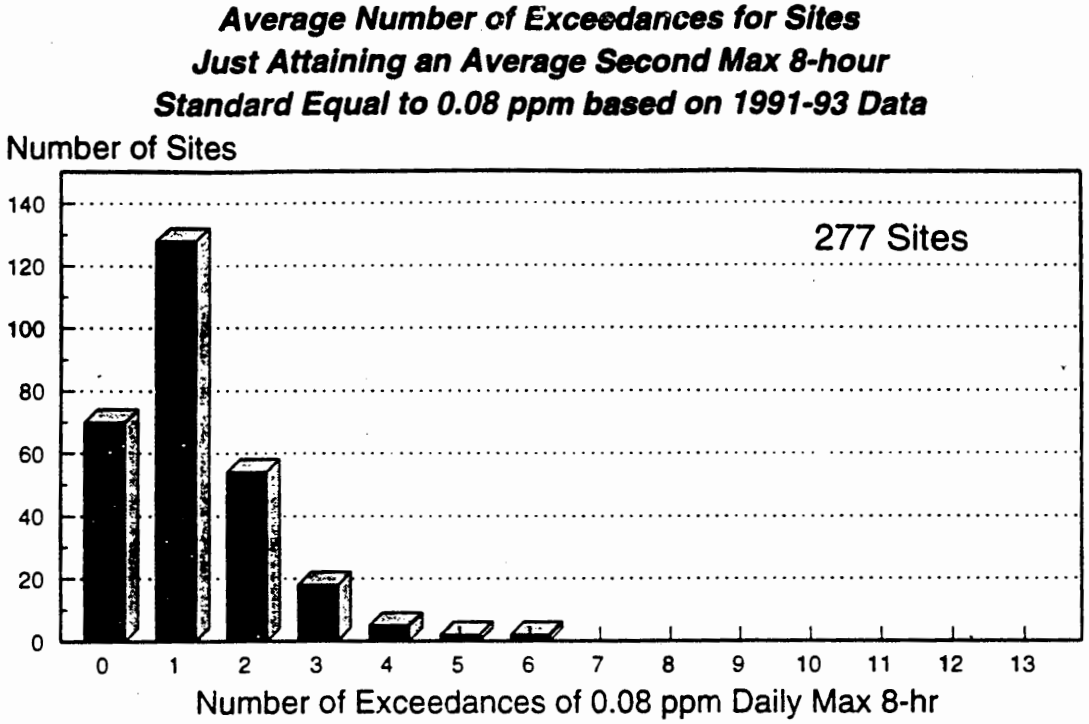
One approach to increasing stability in the air quality management process is to specify a less variable NAAQS statistic. Use of a less variable NAAQS statistic would result in a reduction in the number of borderline sites with relatively high misclassification probabilities, and thus there will be fewer reversals in compliance status ("flip-flops"), at least until some of the other nonattainment sites are brought close to attainment through emission reductions. However, use of a less variable statistic cannot reduce misclassification probabilities for sites on the borderline between attainment and nonattainment, and for those

sites there will likely be frequent classification changes if the emissions remain fairly constant from year to year.

Chock and Nance (Chock et al., 1993) suggest the use of the mean or median of the three annual second daily maxima (AvgMax2) as the NAAQS statistic. The staff has examined the relationship between the AvgMax2 statistic and a one exceedance NAAQS. The AvgMax2 statistic is about 6 percent lower, on average, than the design value concentration for a 1-hour 1 exceedance NAAQS (i.e., the fourth highest daily maximum concentration over three years), and 10 percent lower for an 8-hour 1 exceedance alternative based on ambient monitoring data from the last ten years. On the basis of exceedances, the AvgMax2 lies between the one exceedance and 5 exceedances alternatives. The top-half of Figure V-18 presents a histogram of the average number of daily maximum exceedances for 277 sites just attaining an AvgMax2 standard of 0.08 ppm based on 1991-93 data. Thirty percent of the sites had average exceedance rates greater than 1, with two sites having an average of 5 exceedances per year. The bottom half of Figure V-18 presents the maximum number of exceedances in the worst year during 1991-93 for the same comparison. Half of the sites have two or fewer exceedances in the peak year. Four sites had 10 or more exceedances in the worst year. Figure V-19 repeats this presentation for a fifth highest daily maximum 8-hour standard formulation (AvgMax5). To summarize in terms of exceedances, on average, sites meeting an average annual 2nd highest daily maximum 0.08 ppm standard have 1.2 exceedances per year, and 2.3 exceedances in the worst year of three, while sites meeting an average annual 5th highest daily maximum 0.08 ppm standard have 3.0 exceedances per year, and 5.4 exceedances in the worst year of three. Also, in the worst year of three, 95 % of sites meeting the average annual 2nd highest daily maximum 0.08 ppm standard have 7 or fewer exceedances, while 95 % of sites meeting an average annual 5th highest daily maximum 0.08 ppm standard have 12 or fewer exceedances.

Chock has also suggested the use of the 95th, or other percentiles as the test statistic. Various parametric approaches can be used to estimate the concentration percentiles by fitting a distribution to the daily maxima. Of particular importance is the tail exponential distribution, both because of certain statistical asymptotic properties of that distribution, and because it often fits the observed concentrations. One approach (Breiman et al., 1978) fits

Figure V-18. Frequency distribution of the average and maximum number of exceedances of 0.08 ppm 8-hour daily maximum concentrations for sites just attaining an average annual second highest daily maximum standard of 0.08 ppm.



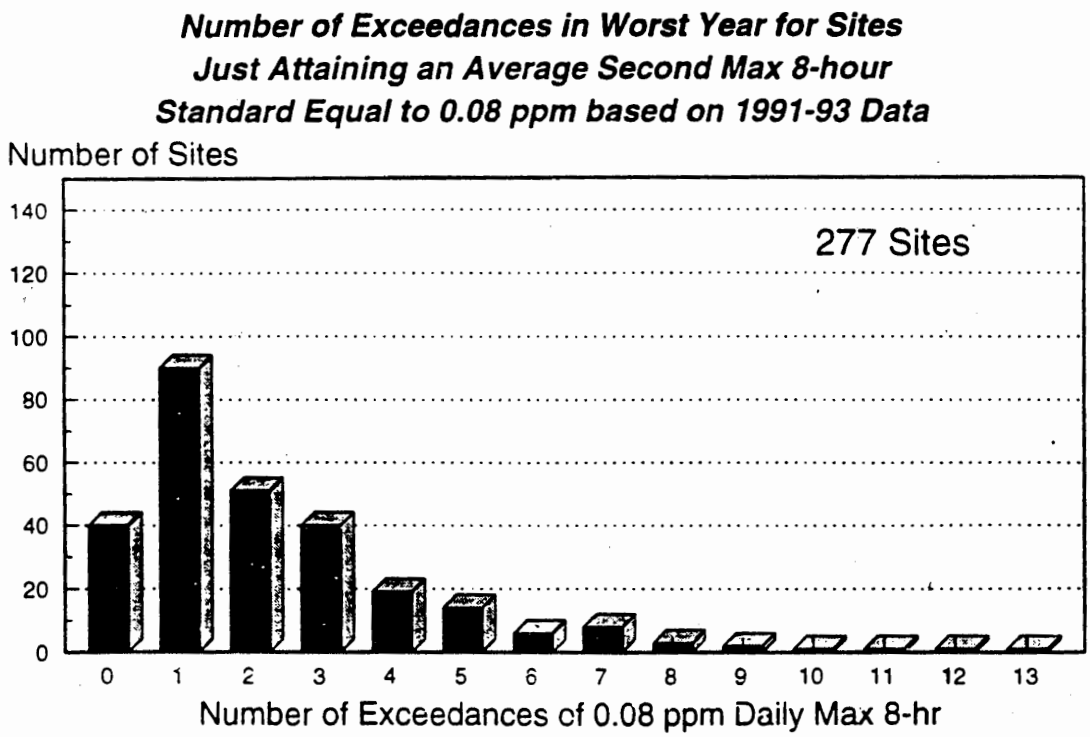
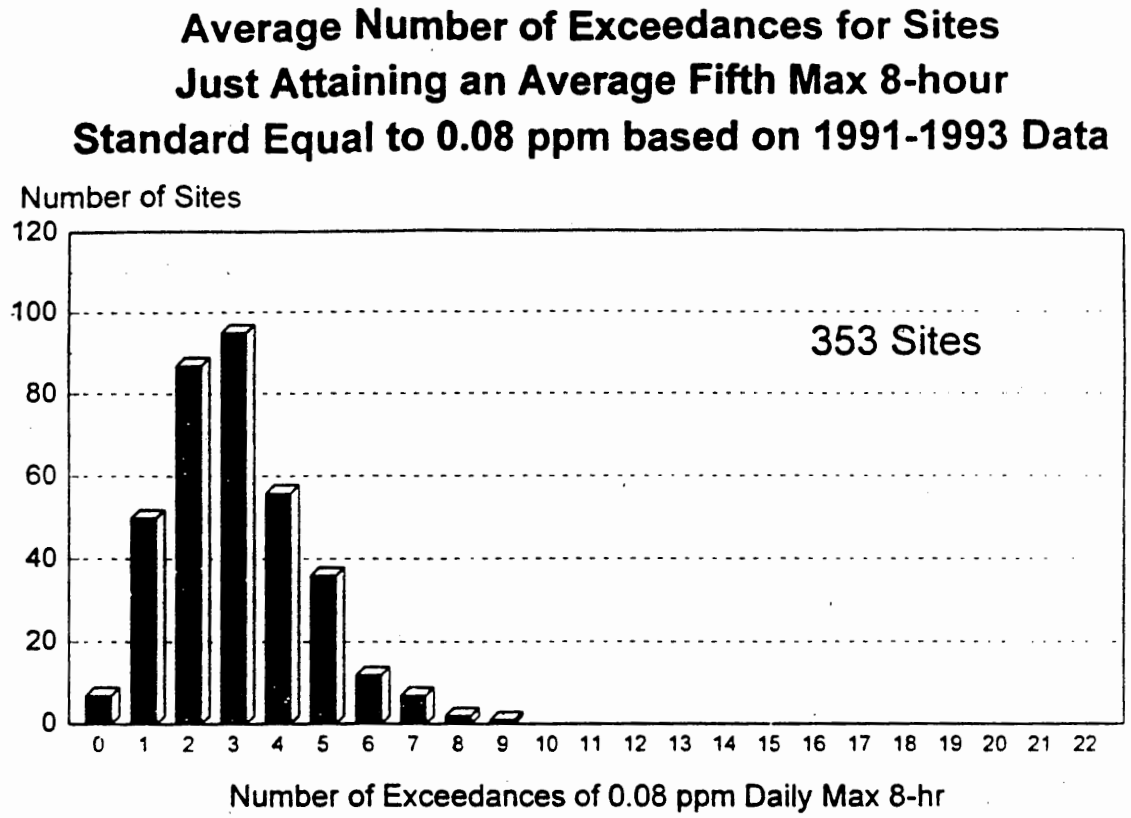
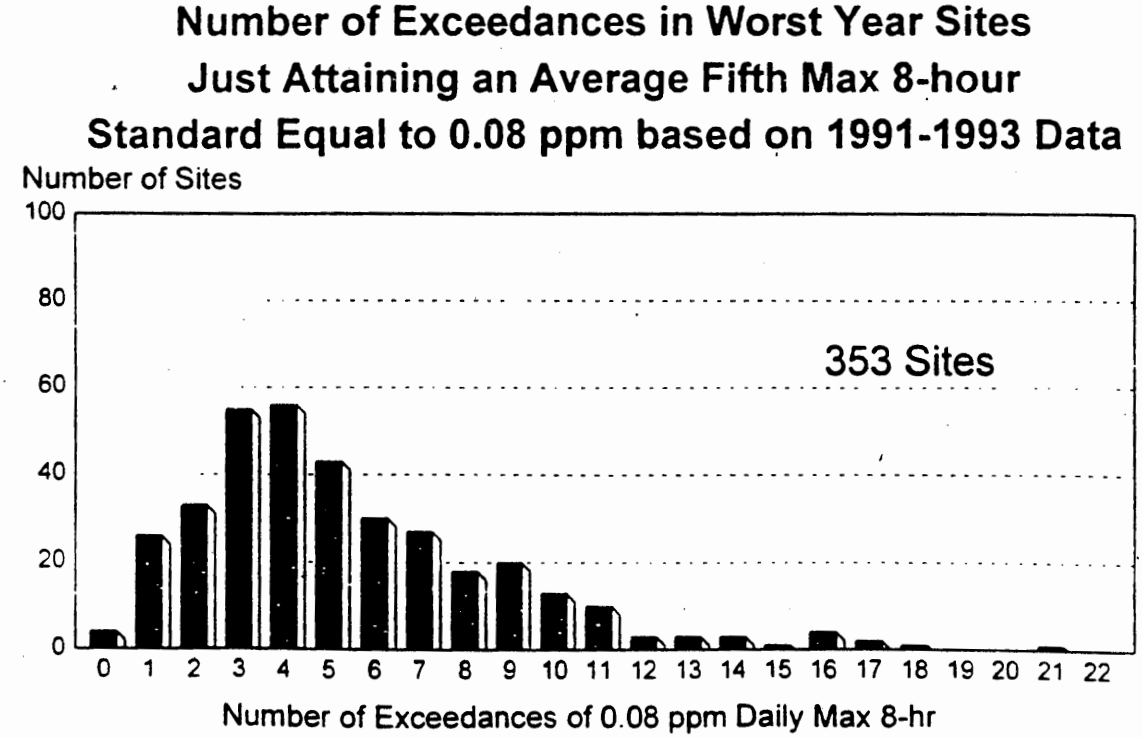


Figure V-19. Frequency distribution of the average and maximum number of exceedances of 0.08 ppm 8-hour daily maximum concentrations for sites just attaining an average annual fifth highest daily maximum standard of 0.08 ppm.





the exponential distribution to the top 5 or 10 percent of the data. Use of the "tail-exponential" approach can yield a more stable NAAQS statistic than the current expected exceedance statistic provided that the exponential assumption is applicable. For example, Larsen used a version of Breiman's approach (CARB, 1992) to derive estimates of the one in one year percentile based on three years of data, and the California Air Resources Board (CARB) uses this approach when determining attainment of the California O_3 standard.

Fairley and Blanchard have proposed several other alternatives (Fairley et al., 1991) including the use of (1) averages of annual high percentiles (such as the mean of three annual 95th percentiles), (2) averages of a certain number of the highest O_3 concentrations, and (3) spatial averages (across sites). Using averages of a certain number of the highest O_3 concentrations is very similar to using the tail exponential approach since the tail exponential design value is a high percentile plus a multiple of the average excess above that percentile. Most of these forms can be viewed as multiple exceedance standards. For the typical O_3 monitoring season, the 95th percentile translates into an average of 10 exceedances per year of the specified standard level. As noted previously, the staff is currently not considering alternatives with an average of more than 5 exceedances per year.

The staff is also addressing how concerns about the spatial representativeness of monitoring sites and population exposure might be incorporated into the form of the standard. However, any consideration of some form of spatial averaging or population weighting across monitoring sites raises issues about environmental equity, the adequacy of the current monitoring network, and the specificity of monitor siting requirements. On the other hand, such a conceptual approach may better reflect population exposure and risk. The staff is also considering whether concerns about population exposure might also be addressed by monitor siting guidance and control strategy assessments as part of the implementation process.

For alternative NAAQS statistics other than the design value, such as those discussed above, the level of the standard would need to be established as a function of the NAAQS statistic. Comparisons of the level of health protection can not be specified exactly for alternative NAAQS statistics, since health protection is a function of the entire distribution of ambient concentrations that would exist when a standard is attained, and the resultant impacts

on that distribution will be different for different forms. Alternative forms can, however, be compared on average, although the spatial distribution of protection (in terms of reduced risk of adverse health effects) would vary from one form of the standard to another.

4. Alternative Attainment Test Criteria

The current NAAQS is a "bright-line" standard. This means that a site is either attainment or nonattainment. Specifying a standard in this way conceptually relates best to pollutants that exhibit health effects thresholds. Staff is now considering specification of a range, rather than a bright-line standard. Such an approach is intended to recognize the absence of discernible health effects thresholds and the projections that population risk varies little with small changes in air quality. Use of a range for the specification of a standard is conceptually a way to recognize the continuum of risk associated with varying levels of O_3 exposure and, thus, O_3 air quality. Within this context, the staff is considering whether the specification of a range rather than a bright-line standard would help to facilitate individual and/or regulatory agency efforts to provide additional safeguards against responses that may, in a small number of particularly sensitive individuals, occur at levels even below the level of a standard that protects public health with an adequate margin of safety.

5. Alternatives for Treatment of Missing Values

The formulation of the current standard includes procedures for dealing with incomplete monitoring data. These missing data procedures assume that missing O_3 values during the O_3 season follow the same patterns as the non-missing values. A missing day during the O_3 season is assumed to be less than the level of the NAAQS only if it is a single missing value that occurs between two valid daily maximum O_3 measurements that are less than 75 percent of the level of the NAAQS. Because of the rounding convention established for estimated exceedances for the current standard, the missing data adjustment only becomes a factor when more than 5 percent of the days are missing, i.e., more than 10 days for the typical O_3 season.

The staff is exploring two different conceptual approaches to the treatment of missing data: (1) requiring a greater degree of data completeness to demonstrate compliance with the standard than noncompliance and (2) the use of simple statistical procedures to account or adjust for missing data.

In the first case, the staff is considering an average data completeness requirement that monitoring sites would have to meet to demonstrate attainment of the standard. Based on a review of the current monitoring network, more than 80 to 90 percent of all sites achieve better than 90 percent data completeness.

Secondly, the staff is evaluating whether information on meteorological conditions could be used to provide an objective procedure for judging if meteorological conditions on a missing day were not conducive to exceedances of the O_3 NAAQS, then that day could be assumed to be less than the level of the NAAQS. The literature contains numerous references to the use of meteorological data to define O_3 conducive days for peak 1-hour concentrations (Jones, 1985; Kolaz et al., 1990; Jones, 1992). Table V-21 adapted from Chu (Chu, 1995) presents a set of criteria for O_3 conducive conditions for the eastern United States. These criteria represent, to a great extent, the necessary conditions for daily maximum 1-hour O_3 concentrations to exceed 0.12 ppm. While these are conditions necessary for high O_3, they are not necessarily sufficient conditions. Other factors may also be important. Additional analyses would be needed to define the necessary conditions for peak 8-hour concentrations in the ranges of concern. The first step of such an approach might also include a review of ambient concentrations recorded at other monitoring sites in the area for possible exceedances of the NAAQS and the historical relationships among nearby monitoring sites. The meteorological data would not be viewed as a surrogate for O_3 monitoring data. Such an approach presumes the availability of meteorological data, such as a nearby National Weather Service Station. It also means that the attainment status of sites near the level of the standard with a large number of missing days could not be determined directly from the ambient data base, but rather would require an analysis of the meteorological data as well.

TABLE V-21. CRITERIA FOR O_3 CONDUCIVE CONDITIONS FOR THE EASTERN U.S.

1. $T \geq 26.5°C$ for cities north of 40°N,
   $T \geq 29°C$ for cities between 35°N and 40°N,
   $T \geq 32°C$ for cities south of 35°N.

2. Wa.m. $\leq$ 5 m/s for cities in transport regions (i.e., Midwest and Northeast),
   $W_{a.m.} \leq 4$ m/s for cities outside transport regions.

3. $W_{p.m.} \leq 7.5$ m/s for cities in transport regions,
   $W_{p.m.} \leq 6$ m/s for cities outside transport regions,
   $W_{p.m.} \leq 5$ m/s for Gulf Coast cities and Florida.

4. RH $\leq$ 75% for coastal cities north of 40°N,
   RH $\leq$ 65% for inland cities between 30°N and 40°N,
   RH $\leq$ 70% for all cities south of 30°N.

Adapted from Chu (1995).

VI. STAFF CONCLUSIONS AND RECOMMENDATIONS ON PRIMARY NAAQS

This section presents staff conclusions and recommendations for consideration by the Administrator in selecting a pollutant indicator, averaging time, form, and level of the primary O_3 NAAQS. In developing these conclusions and recommendations, the staff has drawn upon the scientific and technical information contained in the CD and summarized in Section V of this Staff Paper, the exposure and risk analyses presented in Section V, and comments provided by the CASAC and the public on drafts of this Staff Paper.

The staff has attempted to integrate information on acute and chronic health effects of O_3, the expert judgments on the adversity of such effects, and, when possible, quantitative assessments of the risk of experiencing such effects into a basis for conclusions and recommendations on the primary O_3 NAAQS. This approach recognizes that for most of the health effects associated with O_3, no population threshold can be clearly identified. Thus, the approach taken here uses assessments of exposure and risk, when possible, to provide additional insight and to inform judgments about the protection of public health with an adequate margin of safety.

As discussed in Section V.H, quantitative risk assessments have been premised on extrapolating exposure-response functions from lowest observed effect levels down to background levels. Thus, these assessments reflect a continuum consisting of levels at which health effects are certain through levels at which scientists generally agree that health effects may occur but the likelihood and magnitude of the response is more uncertain. The integrated approach taken in this Staff Paper links risk assessments, which provide estimates of how many people are likely to experience various effects, with consideration of the degree of severity of the effects as bases for judgments about the point at which risks have been reduced sufficiently to achieve protection of public health with an adequate margin of safety.

In recommending a range of options for the Administrator to consider, the staff notes that the final decision is largely a public health policy judgment. A final decision must draw upon scientific information about health effects and risks, as well as a series of judgments: 1) about when physiological effects become adverse from a public health perspective, as discussed in Section V.F of this Staff Paper, 2) the relative severity of various effects with estimates of the expected incidence of those effects, and 3) how to deal with the range of

uncertainties that are inherent in the evidence and assessments. This approach is consistent with the requirements of the NAAQS provisions of the Clean Air Act (Act) and with how the EPA and the courts have historically interpreted the Act. These provisions do not require the Administrator to establish a NAAQS at a zero-risk level but rather at a level that avoids unacceptable risks and, thus, protects public health with an adequate margin of safety.

The following staff conclusions and recommendations are based primarily upon those analyses discussed above and in Section V of the Staff Paper, staff judgment regarding those analyses, and the comments provided by the CASAC and the public.

A. Pollutant Indicator

The staff believes that the conclusions on the appropriate indicator for the primary O_3 NAAQS that were presented in the previous Staff Paper (USEPA, 1989) remain valid today. As indicated in the previous Staff Paper, it is generally recognized that control of ambient O_3 levels provides the best means of controlling photochemical oxidants of potential health concern. Further, among the photochemical oxidants, the acute-exposure chamber, field, and epidemiological human health data base raises concern only for O_3 at levels of photochemical oxidants commonly reported in ambient air. Thus, the staff recommends that O_3 remain as the pollutant indicator for protection of public health from exposure to all photochemical oxidants found in the ambient air.

B. Averaging Times

1. Short-Term and Prolonged (1 to 8 hours)

The current primary O_3 NAAQS was set in 1979 with a 1-hr averaging time. This was intended to protect the public against the health effects associated with 1- to 3-hr exposures to O_3 in addition to the health effects potentially associated with longer-term O_3 exposures which were not as well documented at that time.

Since 1979, a numerous researchers have investigated the health effects associated with short-term (1- to 3-hr) and prolonged acute (6- to 8-hr) exposures to O_3. Numerous controlled-exposure studies of human subjects, who engaged in activities involving heavy and moderate exertion, provide a basis for quantitative concentration-response relationships between 1- to 3-hr O_3 exposures and a variety of lung function parameters and respiratory symptoms. Also, field and epidemiological studies now provide additional evidence of

associations between 1-hr ambient O_3 levels and health effects ranging from respiratory symptoms and lung function decrements reported at summer camp studies to increased hospital admissions for respiratory causes. However, the field and epidemiological studies have not been analyzed sufficiently as yet to determine whether the observed effects correlate as well or better with 6- to 8-hr exposures as with the 1- to 3-hr exposures. More recent controlled-exposure studies have been conducted providing evidence that the same respiratory effects (i.e., lung function decrements and respiratory symptoms) occur when human subjects are exposed to O_3 while engaging in activities involving intermittent, moderate exertion for prolonged exposure periods of 6 to 8 hrs. These effects occur at lower concentrations of O_3 and at less severe exertion levels than for 1- to 3-hr exposures. Other effects, such as the presence of biochemical indicators of inflammation and reductions in pulmonary defense mechanisms leading to increased susceptibility to infection, have also been reported for prolonged exposures and, in some cases, for short-term exposures.

This brief summary of the averaging times associated with various acute health effects highlights that averaging times of 1 to 3 hrs and of 6 to 8 hrs have both been associated with a wide range of observed respiratory effects caused by O_3 exposure. The current 1-hr averaging time is judged to be most appropriate to address acute health effects associated with 1- to 3-hr exposures because these effects typically occur within the first hour of exposure, during moderate and heavy exertion. On the other hand, an 8-hr averaging time is judged to be more appropriate for addressing similar health effects associated with 6- to 8-hr exposures, since health effects typically build up over time in moderately exercising subjects, approaching a plateau somewhat beyond the 6.6 hr exposure periods for which most of the prolonged exposure studies have been conducted. Furthermore, it is generally convenient to assess air quality and exposure patterns in 8-hr time periods.

In selecting an averaging time or times for the primary O_3 NAAQS, questions arise as to whether both a 1-hr NAAQS and an 8-hr NAAQS are necessary and appropriate to protect public health, and, if both are not needed, which averaging time is more appropriate.

The primary way in which these questions have been addressed in this draft Staff Paper is through the quantitative risk analyses presented in section V-H. These analyses produce estimates of the reduction in the estimated risks of both 1- and 8-hr effects

associated with attaining the current 1-hr standard and several alternative 8-hr standards. Attainment of any particular 1-hr or 8-hr standard was modeled by projecting a change in the entire air quality distribution sufficient to just attain the standard. The resulting air quality distribution is then analyzed in terms of both 1-hr and 8-hr average concentrations to develop risk estimates for certain health endpoints associated with 1-hr and 8-hr exposures. These results show that attaining either the current 1-hr standard or a 0.10 ppm, 1-hr standard reduces the risk of experiencing health effects associated with either 1-hr or 8-hr O_3 exposures in areas that do not currently attain the 0.12 ppm, 1-hr standard. Likewise, attaining most of the alternative 8-hr standards examined reduces the risk of experiencing health effects associated with either 8-hr or 1-hr O_3 exposures in areas currently exceeding the 0.12 ppm, 1-hr standard. Based on these analyses, the staff believes that adequate reductions in risks from both 1-hr and 8-hr effects can be achieved through a primary standard with an averaging time of either 1 or 8 hrs. Staff judges that the 8-hr averaging time is most directly associated with health effects of concern at the lowest concentration of O_3. As a result, the staff concludes that the establishment of both 1-hr and 8-hr standards is not necessary to reduce the risks associated with the range of acute effects considered in these analyses.

The staff also has given consideration to the question of whether both a 1- and 8-hr standard are appropriate. In the staff's judgment, two short-term standards could be appropriate if the combination of two such standards were determined to be a more efficient way to provide public health protection than through a single standard with an averaging time of either 1 or 8 hours. A combination of standards may be more effective if, for example, a 1-hr standard would need to be set at a significantly lower level to provide adequate protection from 8-hr effects than would otherwise be necessary to provide adequate protection from the 1-hr effects, and if such a standard would, in effect, represent overcontrol in some geographic areas as a result of varying air quality patterns. In looking at risks associated with changes in lung function and the areas that would be impacted by alternative standards, staff judges that neither the 1-hr nor 8-hr averaging times appear to

have any advantage in efficiency. Based on the above, relative to the 1-hr alternatives, staff concludes that establishment of an 8-hr standard would likely be more directly related to providing public health protection, increased stability, and, by taking into account more air quality data, would be a more robust standard.

2. Long-Term

There is a very large animal toxicology data base providing clear evidence of lung tissue damage, with additional evidence of reduced lung elasticity and loss of lung function, caused by exposures to higher levels of simulated ambient O_3, though not at or below 0.12 ppm O_3, lasting from a few months to years. Although there have been substantial recent advancements in dosimetry extrapolation from animals to humans (see CD, Chapter 8), further research in the area of species sensitivity must be conducted before quantitative linkages to specific health effects in human could be established with known uncertainty.

Further, since there is considerable uncertainty regarding the temporal patterns and levels of exposure that might be most directly associated with any such chronic effects, should they occur, in humans (i.e., the importance of the occurrence and pattern of repeated short-term and/or prolonged peaks relative to cumulative total exposure), it is not possible to evaluate the extent to which either a 1- or 8-hr standard would contribute to protecting against any such effects. On the other hand, it is likely that the alternative 1- and 8-hr standards under consideration would directionally provide protection against such effects should they occur, in that such alternatives would result in both lower short-term and prolonged peaks as well as lower overall O_3 concentration distributions which would reduce cumulative long-term exposures. Thus, until additional research and related analyses have been conducted, the staff believes that consideration of a separate long-term O_3 NAAQS is not appropriate.

C. Form of the Standard

Based upon information contained in the CD and sections IV and V.I of this Staff Paper, and on discussions and comments received during the review of the Clean Air Act Ozone Design Value Study, the staff has reached the following conclusions on the form of the standard. Staff concludes that several test statistics should be considered in specifying the form of any new or revised primary standards. Such test statistics include the expected

exceedance rate, including both the 1-expected exceedance (the basis for the current standard) and multiple exceedances (up to 5 expected exceedances) per year averaged over three years, as well as concentration-based test statistics, including in particular the average second to the fifth highest daily maximum 8-hr concentration averaged over three years. In addition, specifying the standard in terms of a range of air quality values (e.g., the second to the fifth highest daily maximum 8-hr average concentration, averaged over a 3-year period) should also be considered. In conjunction with such alternative statistics, some form of spatial averaging or population weighting across monitors may also warrant consideration.

D. Level of the Standard

As discussed at the beginning of this section, the staff's approach to formulating recommendations with regard to an appropriate range of standard levels focuses on general conclusions regarding lowest observed effect levels and a qualitative assessment of evidence regarding health effects for which no quantitative estimates of risks were developed, together with quantitative risk assessments for selected health effects to provide additional input into consideration of an adequate margin of safety. The staff's conclusions presented in this Staff Paper are informed by qualitative evidence discussed in section V.D, judgments about adversity discussed in section V.F, and exposure and risk estimates for selected health endpoints for sensitive population groups summarized in sections V.G and V.H and Appendices B and C. Consistent with the above conclusions on averaging times, the following discussions and conclusions are primarily directed toward identifying a range of levels associated with alternative 8-hr standards for consideration by the Administrator in selecting a standard(s) that, in her judgment, would reduce risks to public health sufficiently to protect public health with an adequate margin of safety.

1. General Conclusions

Taking into account information on health effects, sensitive and at-risk populations, and adversity of effects contained in the CD and in section V of this Staff Paper, the staff has drawn the following conclusions with regard to effects that the staff judges are of particular importance in considering the need for new or revised primary O_3 NAAQS.

- In controlled-exposure human studies, the lowest range within which 1- to 3-hr exposures to O_3 at heavy exertion have induced group mean statistically significant

lung function decrements is 0.12 to 0.16 ppm, and the lowest range within which 6- to 8-hr exposures to O_3 at moderate exertion have induced group mean statistically significant lung function decrements is 0.08 to 0.12 ppm. In epidemiology studies, similar effects have been associated with short-term ambient O_3 exposures below 0.12 ppm when subjects were engaged in physical activity.

- In controlled-exposure human studies, the lowest range within which 1- to 3-hr exposures to O_3 at heavy exertion have induced group mean statistically significant respiratory symptoms, including cough and pain on deep inspiration, is 0.16 to 0.18 ppm, and the lowest range within which 6- to 8-hr exposures to O_3 at moderate exertion have induced group mean statistically significant respiratory symptoms is 0.08 to 0.12 ppm.

These lung function and symptoms effects are based on numerous controlled human exposure and field studies of both healthy and respiratory-impaired (e.g., asthmatic) subjects. As discussed in section V.F, the staff concludes that these effects are adverse to healthy individuals and those with impaired respiratory systems experiencing these effects at levels characterized as severe in Table V-5, and that the adversity of effects levels categorized as moderate for healthy individuals is a function of the number of times an affected individual would experience such effects, and is a matter for the Administrator's judgment. The staff also concludes that the population group at greatest risk for experiencing lung function effects is active outdoor children (i.e., children who typically play outdoors during summer when O_3 levels are highest) engaged in physical activity, with outdoor workers engaged in physical labor also being at increased risk relative to the entire population. Both outdoor workers and outdoor children are at increased risk for experiencing symptoms, based on exposure estimates, although children do not typically report symptoms to the same degree as adults. These groups engage in activities requiring exertion at levels that are associated with significant lung function decrements and symptoms.

In making judgments about the level at which public health protection from these effects incorporates an adequate margin of safety, the staff believes that it is important, when possible, to consider (1) the extent to which at-risk groups are likely to be exposed to ambient concentrations associated with such adverse effects, (2) the mechanisms by which

they occur, and (3) the resulting risk of experiencing adverse effects predicted for these at-risk groups. The exposure and risk analyses that have been done to further inform this decision take into account the significant variability in responses that have been observed in these studies, in that some individuals experience lung function decrements and symptoms both greater than and less than the group mean. Furthermore, these analyses recognize that there is no indication that a threshold exists at the lower end of these ranges. Drawing from the results of the risk analysis presented in section V.H, the next section summarizes risk information that the staff believes is relevant to the Administrator's consideration of an adequate margin of safety with regard to lung function and symptom effects associated with both 1- to 3-hr and 6- to 8-hr exposures to O_3.

- The lowest observed effects level at which 1- to 3-hr exposures to O_3 at very heavy exercise have induced increases in nonspecific bronchial responsiveness in healthy adults is 0.18 ppm, and the range at which 6- to 8-hr exposures to O_3 at moderate exertion have induced such increases in nonspecific bronchial responsiveness is 0.08 to 0.12 ppm. Exercising asthmatic individuals experience larger increases in nonspecific bronchial responsiveness at lower O_3 exposures, but evidence is too limited to draw quantitative conclusions at this time.

Nonspecific bronchial responsiveness is an indication of an individual's susceptibility to stimuli such as antigens, chemicals, and particles, has been demonstrated in both human and animal studies. Staff believes that increases in nonspecific bronchial responsiveness have the potential to aggravate asthma and other types of preexisting respiratory impairment, and, thus, staff concludes that this effect may be adverse for some exercising individuals with significantly impaired respiratory symptoms at levels characterized as moderate in Table V-5.

- The lowest level at which 1- to 3-hr exposures to O_3 of healthy adults engaged in activities involving very heavy exertion have been tested and have induced biochemical indicators of pulmonary inflammation is 0.20 ppm, and the range for which 6- to 8-hr exposures to O_3 of healthy adults engaged in moderate exertion have induced this effect is 0.08 to 0.10 ppm.

These indicators of inflammation have been observed in both controlled human exposure studies and in experimental animal studies. While there is divergent opinion as to

the clinical significance of a singular occurrence of acute pulmonary inflammation, the staff believes that based on scientific evidence repeated occurrences of acute pulmonary inflammation over periods lasting months to years have the potential to result in structural changes in the lungs for which there is suggestive evidence of an association with permanent respiratory injury and/or progressive dysfunction. This view is supported both by the linkages that have been demonstrated between the biochemical indicators of inflammation identified in the fluids extracted from the lungs of humans after short-term and prolonged exposures to O_3 and the structural damage which has been reported in laboratory animals as a result of long-term exposures to O_3.

In making judgments about the standard level at which public health is protected against indicators of acute pulmonary inflammation with an adequate margin of safety, the staff believes that the exposure analysis presented in Section V.G provides useful information. Such information is summarized in the next section under margin of safety considerations. No risk assessment has been conducted for the effect due to (1) the limited amount of data which is insufficient to derive exposure-response relationships and (2) the uncertainties which remain in the dosimetric extrapolation of animal to human data (as discussed in Chapter 8 of the CD), as well as large observed differences in species sensitivity between humans and laboratory animals that is yet to be adequately understood.

- Evidence from animal toxicology studies suggest that acute exposures to O_3 in the range of 0.08 to 0.10 ppm can induce pulmonary changes that decrease the effectiveness of the lung's defenses against bacterial lung infections.

The staff concludes that there is adequate evidence to reasonably anticipate that such reductions in the human defense mechanisms could result in increased susceptibility to pulmonary infection. This conclusion is based in large part on the existence of a substantial animal toxicology data base which indicates that O_3 increases susceptibility of experimental animals to respiratory infection. Although few controlled human exposure studies have been conducted to assess the impact of exposing human subjects to O_3 and a bacterial challenge, those that have been conducted have provided insufficient evidence of increased susceptibility to infection caused by O_3. This may well be a result of the extremely cautious manner in which these studies must be conducted when using human subjects with infectious material.

However, despite limited human data, a biologically plausible case can be made for prolonged exposures to O_3 increasing human susceptibility to respiratory infection. There exist many similarities between laboratory animals and humans with regard to many of the host defense mechanisms used to defend against infections of the lung. When the ability of the lungs of either humans or animals to destroy invading microbes or to remove inhaled particulate matter is adversely affected by inhaled O_3, it is reasonable to anticipate that there will be an increased risk of developing respiratory infection. Depending on the level of O_3 exposure, the period of time or number of times exposed, and the susceptibility of the individual exposed, the resulting respiratory infection could be relatively minor or result in the need for hospitalization.

- Exposures to O_3, as currently experienced in several cities in the eastern United States and Canada, are associated with excess hospital admissions and emergency room visits for respiratory causes, with evidence of this effect occurring to some extent even when hourly O_3 concentrations are as low as 0.08 to 0.10 ppm.

In a number of epidemiological studies, linear, nonthreshold associations have been reported between the daily maximum hourly O_3 concentration on the day prior to admissions and an increase in hospital admissions and emergency room visits for respiratory causes. These effects have been attributed primarily to O_3 exposures, since the effects of copollutants and other confounding factors were judged in the CD to be adequately accounted for in these analyses. The biological plausibility of O_3-related increases in hospital admissions is further supported by the controlled human exposure data showing O_3-induced increases in nonspecific bronchial responsiveness and the animal toxicology data noted above with respect to increased susceptibility to respiratory infection.

In order to provide for more informed judgments regarding which standards would reduce risks to public health sufficiently to protect public health with an adequate margin of safety, the staff has conducted a quantitative risk assessment, which is discussed in Section V.H. That risk assessment, which uses air quality monitoring data and hospital admissions data from New York City, is summarized in the next section under margin of safety considerations. Further, the staff believes that it is not yet possible to assess any association between either excess hospital admissions or emergency room visits and 6- to 8-hr exposures

because most of the basic data have not been analyzed in terms of 8-hr average concentrations at this time, although such an association with 8-hr exposures has been reported.

- An association between daily mortality and exposure to O_3 in an area with very high O_3 levels (i.e., Los Angeles) has been suggested, although the magnitude of such an effect remains unclear at this time.

Reanalysis of 1970's data from Los Angeles County suggests that O_3 exposures are associated with a small, but statistically significant, portion of day-to-day variations in total daily mortality in that city, where hourly O_3 concentrations >0.20 ppm occur, over a 10-year period. However, the researchers who conducted the reanalysis emphasized that since statistically significant associations have been detected among both mortality and environmental variables, one can not conclude with confidence that an association with mortality is causal based on results from their observational study. In another epidemiology study no such association was seen where hourly O_3 concentrations were <0.15 ppm. Other studies of this potential effect have been confounded by copollutants, especially particulate matter, and by inadequate methods to characterize exposure or to account for other confounding factors. Based on the available published evidence, the staff believes that protection against this potential effect would likely result from any O_3 standard that is protective of other effects discussed above, such as increased hospital admissions and susceptibility to pulmonary infection.

2. Margin of Safety Considerations Based on Quantitative Exposure and Risk Assessment

The following discussion presents summary results and observations of exposure and risk drawn from the quantitative exposure and risk assessments presented in sections V.G. and V.H. and Appendices B. and C.. This information is intended to provide additional insight about the extent to which at-risk populations may experience the specific health effects addressed in these analyses when various alternative standards are just attained. The staff believes that such information, when available, is useful to inform judgments about which standards would reduce risks to public health sufficiently to protect public health with an adequate margin of safety.

The staff believes that the exposure and risk assessment methods used in these analyses represent the state of the art at the present time, and that these analyses provide reasonable estimates for the purposes intended. The staff cautions, however, that in light of the many sources of uncertainty inherent in such analyses, the results should not be interpreted as precise measures of exposure and risk. Some important uncertainties inherent in the analyses include (1) the air quality adjustment procedures used to simulate just attaining the alternative standards, (2) the specification of activity patterns and associated exertion levels for the population groups of interest based on limited activity diary data, (3) the extrapolation of exposure-response functions below the lowest observed effects levels to an estimated background level, and (4) the inability to account for factors which are known to affect the exposure-response relationships (e.g., assigning children the same symptomatic response rate as has been observed for adults and not adjusting response rates to reflect the increase and attenuation of responses that have been observed in studies of lung function and symptoms upon repeated exposures). Sections V.G. and V.H., Appendices B. and C., and the associated support documents (Johnson et al., 1996 a,b,c; Whitfield et al., 1996) include a more complete discussion of uncertainties inherent in these analyses and 90% credible intervals are presented for all risk estimates.

Summary Results. The following information draws from key analyses discussed in Sections V.G and V.H and described in more detail in several technical support documents (Johnson et al., 1996a,b,c and Whitfield et al., 1996).

Table VI-1 presents a summary of risk estimates for 1-hr or 8-hr health endpoints for outdoor children upon attainment of alternative 8-hr, 1-expected exceedance standards and the current 0.12 ppm, 1-hr standard. The risk estimates in Table VI-1 are for effects associated with exposure under moderate exertion. These risk estimates are provided only for outdoor children and only for levels of lung function decrement and symptoms that the

TABLE VI-1. PERCENT OF OUTDOOR CHILDREN ESTIMATED TO EXPERIENCE VARIOUS HEALTH EFFECTS 1 OR MORE TIMES PER YEAR ASSOCIATED WITH 1- OR 8-HOUR OZONE EXPOSURES UPON ATTAINING ALTERNATIVE STANDARDS[a]

Alternative Standards (1 expected exceedance per year)	Pulmonary Function Decrements, $FEV_1 \geq 15\%$ Associated with 8-hr Exposures	Pulmonary Function Decrements, $FEV_1 \geq 20\%$ Associated with 8-hr Exposures	Moderate or Severe Pain on Deep Inspiration Associated with 1-hr Exposures
0.07 ppm, 8-hr	3.0 (1.0-6.6)[b]	0.4 (0.1-1.8)	0.3 (0.01-1.9)
0.08 ppm, 8-hr	5.1 (2.2-9.6)	1.4 (0.5-3.7)	0.6 (0.05-2.7)
0.09 ppm, 8-hr	7.7 (3.3-13.3)	2.7 (1.0-6.1)	0.9 (0.1-3.5)
0.12 ppm, 1-hr	8.3 (8.2-14.2)	3.0 (1.1-6.6)	1.0 (0.1-3.6)

[a]Estimates represent aggregate results for 9 urban areas examined. The total number of outdoor children residing in the 9 urban areas was 3.1 million.

[b]90% credible interval

staff believes are of most importance in addressing potentially adverse effects. Lung function decrements associated with 6- to 8-hour exposures at moderate exertion and symptoms associated with 1- to 2-hour exposures at either moderate or heavy exertion were found to be the effects of most concern among the full range of lung function and respiratory symptom effects evaluated in the risk assessment. These risk estimates represent an aggregate estimate for the nine urban areas examined; an aggregate estimate is presented since there is significant variability in this risk measure across the areas. The uncertainty in these risk estimates associated with sample size considerations is characterized by the 90 percentile credible intervals.

Since exposure estimates for outdoor children are higher for most exposure indicators and alternative standards than exposure estimates for outdoor workers, the risk estimates summarized here are likely to be among the highest for the populations being analyzed (i.e., general population, outdoor workers, and outdoor children). The staff has chosen to focus on the percentage, rather than the number, of individuals responding in order to reduce confusion which might result from the use of numbers of people given the differences in population size across the nine urban areas.

Table VI-2 summarizes estimates of excess hospital admissions for asthmatics in the New York City area associated with just attaining a range of alternative O_3 standards. These excess admissions only include those associated with O_3 levels exceeding an estimated background O_3 level of 0.04 ppm for an hourly average.

The staff believes the following observations on exposure and risk, based in part on the information summarized in Figures VI-1 and VI-2 and Tables VI-1 and VI-2 are useful in formulating recommendations about levels of alternative standards that reduce risks to public health sufficiently to protect public health with an adequate margin of safety.

Exposure Observations.

(1) Children who are active outdoors (representing approximately 7% of the population in the study areas) appear to be the at-risk population group examined with the highest percentage and number of individuals exposed to O_3 concentrations at and above which there is evidence of health effects,

TABLE VI.2 Admissions of New York City Asthmatics — With a Comparison Relative to Meeting the Current Standard (1 h, 1 expected exceedance, 0.12 ppm)

Issue	Air Quality Scenarios			
	1H1EX-0.12 ppm[a] (Scenario A)	8H1EX-0.08 ppm (Scenario C)	8H5EX-0.08 ppm (Scenario F)	As-Is (Scenario Z)
Excess Admissions[b] (background = 0.04 ppm)	207[c] (70, 344)[d]	115 (39, 191)	120 (41, 199)	388 (132, 644)
% Change from Current Standard[e]	0	??	??	??
Excess Admissions[b] (background = 0 ppm)	909 (308, 1,510)	804 (273, 1,340)	797 (270, 1,320)	1,070 (361, 1,770)
% Change from Current Standard[e]	0	-11.6	-12.3	17.7
All Admissions[f] (thousands)	14,819.00	14,727.0	14,732.0	15[g] (14-16)[g]
% Change from Current Standard[h]	0	-0.6	-0.6	-99.9

[a] EX stands for expected exceedance.

[b] Admissions of asthmatics attributable to exposure to ozone.

[c] Median estimate.

[d] 90% credible interval (about the median).

[e] Because of the necessary assumption that results across scenarios are highly correlated (i.e., if admissions are high for one scenario, they are high for all scenarios), there is very little variation in the percentage change from the current standard.

[f] Admissions of asthmatics for any respiratory-related reason; for scenario *i*, based on estimates of all admissions and excess admissions attributable to ozone levels >0.04 ppm for As-Is scenario, and estimate of excess admissions attributable to ozone levels >0.04 ppm for scenario *i* (e.g., for scenario 1112: 14,800 ≈ 15,000 - 388 + 207).

[g] Admissions of New York City asthmatics for any respiratory-related reason during the 1988-90 ozone seasons (Thurston, 1995).

[h] Variation in these results is attributable to the different numbers of admissions of New York City asthmatics for any respiratory-related reason during the 1988-90 ozone seasons (Thurston, 1995).

FIGURE VI-1. EIGHT-HOUR MAXIMUM DOSE EXPOSURE DISTRIBUTIONS FOR OUTDOOR CHILDREN EXPOSED ON ONE OR MORE DAYS UNDER MODERATE EXERTION (EVR 13-27 LITERS/MIN-M2) IN PHILADELPHIA, PA.

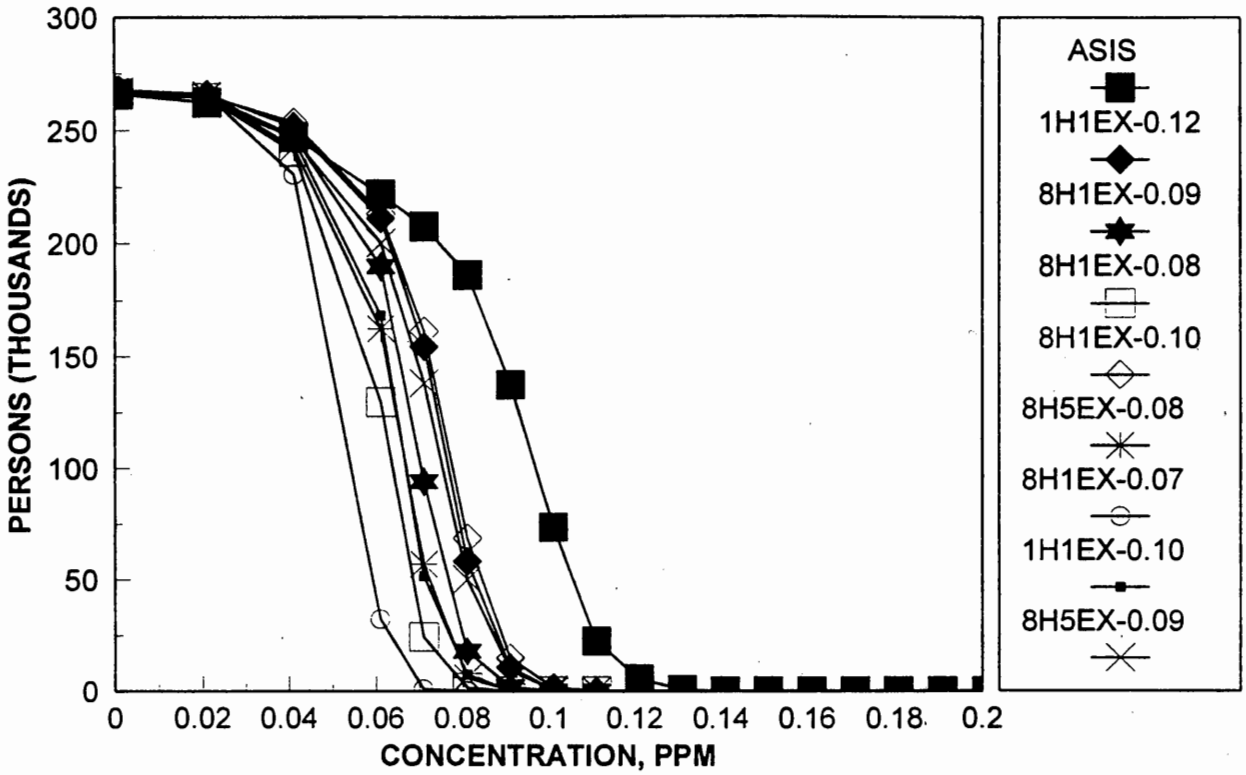
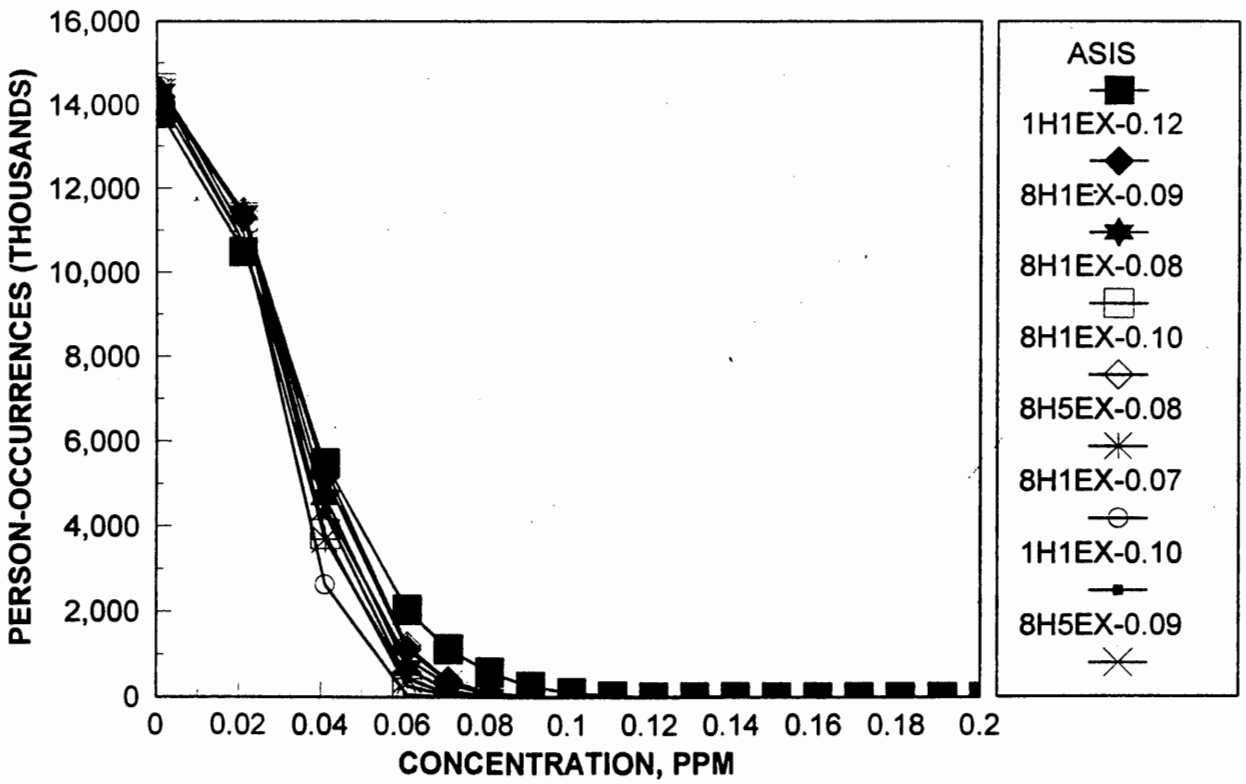


FIGURE VI-2.. EIGHT-HOUR MAXIMUM DOSE EXPOSURE DISTRIBUTIONS OF TOTAL OCCURRENCES FOR OUTDOOR CHILDREN EXPOSURE UNDER MODERATE EXERTION (EVR 13-27 LITERS/MIN-M2) IN PHILADELPHIA, PA.



particularly for 8-hr average exposures at moderate exertion to O_3 concentrations $\geq$0.08 ppm.

(2) On both an absolute number and a percentage basis, exposure estimates are higher for the 8-hr average effects level of 0.08 ppm at moderate exertion than for the 1-hr average effects level of 0.12 ppm at heavy exertion.

(3) Estimated exposures above these effects cutpoints, even on a percentage basis, vary significantly across the urban areas examined in this analysis. However, general patterns of exposure can be seen in comparing the current NAAQS and alternative standards, particularly in looking at the seven current nonattainment areas examined. For example, for estimates of the mean percent of outdoor children exposed to 8-hr average O_3 concentrations $\geq$ 0.08 ppm while at moderate exertion, the following patterns are seen: the range of estimates associated with the current 1-hr NAAQS is approximately 1-21%, dropping to approximately <3% for a 0.10 ppm 1-hr standard. For alternative 8-hr standards (of the same 1-expected-exceedance form as the current NAAQS), the estimated ranges of mean percentages of outdoor children exposed are approximately 3-7% for a 0.09 ppm standard, 0-1.3% for a 0.08 ppm standard, and from essentially 0 in most areas to <0.1% for a 0.07 ppm standard.

(4) In general, there are relatively small differences in comparing the distributions of 8-hr exposure estimates for outdoor children associated with 1- and 5-expected exceedance forms of any given alternative standard, although at particular cutpoints on the distribution, differences between these two forms can appear to be significant in some areas.

(5) Based on comparisons of air quality distributions, estimated exposures are generally comparable between 8-hr standards with 5-expected-exceedance or 5th highest daily maximum concentration forms. In either case, exposure estimates for the worst year of a 3-year compliance period would be higher than for the average or typical year, with the magnitude of the difference varying across areas. For example, for an 8-hr, 0.08 ppm standard of either

form, about 95% of current nonattainment areas would have 10 or fewer exceedances of the 0.08 ppm level in the worst year, compared to an average of less than 5 exceedances in the typical year. Exposures estimated for a year in which there were 10 exceedances would be roughly comparable to the exposures estimated to occur upon attainment in a typical year of a 0.09 ppm, 8-hr standard, with 1- to 5-expected-exceedance forms.

In taking these observations into account, the staff recognizes the uncertainties and limitations associated with such analyses, including the considerable, but unquantifiable, degree of uncertainty associated with a number of important inputs to the exposure model. A key uncertainty in model inputs results from the availability of only a limited human activity database, both with regard to the number of subjects who contributed daily activity diary data and the short time period over which each subject recorded their daily activity patterns. These limitations may not adequately account for day-to-day repetition of activities common to children, such that the number of people who experience multiple occurrences of high exposure levels may be underestimated. Small sample size also limits the extent to which ventilation rates associated with various activities may be representative of the population group to which they are applied in the model. In addition, the air quality adjustment procedure used to simulate air quality distributions associated with attaining alternative standards, while based on statistical analyses of empirical data, incorporates significant uncertainty, especially when applied to areas requiring very large reductions in air quality to attain the alternative standards examined or to areas that are now in attainment with the current NAAQS. A more complete discussion of these uncertainties and limitations is presented in Section V.G. of this Staff Paper and in the technical support documents (Johnson et al., 1996a,b,c).

Risk Observations.

(1) On both an absolute number and percentage basis, risk estimates are higher for effects associated with 8-hr exposures under moderate exertion than for effects associated with 1-hr exposures under heavy exertion.

(2) Reflecting a continuum of risk, there is a decreasing trend in the median estimates of the population estimated to experience the lung function and

symptomatic responses as one moves along the range of alternative 8-hr average, 1-expected exceedance standards under consideration. For example, based on the aggregate risk estimates summarized in Table VI-1, the median percent of outdoor children estimated to experience FEV_1 decrements greater than 15 percent is reduced from about 7.7 percent for a 0.09 ppm, 8-hr standard to about 6.8 percent for a 0.08 ppm, 8-hr standard. Attaining a 0.07 ppm, 8-hr standard results in a further reduction to about 3.0 percent of outdoor children estimated to experience this effect.

(3) In general, the differences in risk estimates for outdoor children associated with 1- and 5-expected exceedance standards set at the same standard level are relatively modest within the continuum of risk. For example, the risk estimates for lung function decrements $\geq$ 15 percent associated with a 5-expected exceedance standard set at 0.08 ppm fall between the risk estimates for the 0.08 and 0.09 ppm, 1-expected exceedance, 8-hr standards. Similarly, the risk estimates for a 5-expected exceedance standard set at 0.09 ppm fall between the risk estimates for the 0.09 and 0.10 ppm, 1-expected exceedance, 8-hr standards.

(4) Multiple occurrences of lung function decrements $\geq$ 15 percent and $\geq$ 20 percent associated with 8-hr exposures under moderate exertion are estimated to occur for outdoor children upon attainment of any of the alternative 1- or 8-hr standards analyzed. The average seasonal numbers of occurrences per responder across the urban areas included in the analysis range from four to about nine for lung function decrements $\geq$ 15 percent and from two to about five for lung function decrements $\geq$ 20 percent. Some individuals will experience more frequent occurrences of effects during the O_3 season, whereas others will experience fewer occurrences than the average in any given area.

(5) Based on comparisons of air quality distributions, risk estimates are generally comparable between 8-hr standards with 5-expected exceedances or 5th highest daily maximum concentration forms. As noted in the previous discussion of the exposure estimates, for either form the worst year of a 3-year compliance

period would be higher than for the average or typical year. For example, about 95 percent of current nonattainment areas meeting either form of an 8-hr, 0.08 ppm standard would have 10 or fewer exceedances in the worst year, compared to an average of less than five exceedances in a typical year. Risk estimates for a year in which there were 10 exceedances of 0.08 ppm, 8-hr average vary from urban area to urban area but fall between the risk estimates for a 5-expected exceedance standard of 0.08 ppm and a 5-expected exceedance standard set at 0.09 ppm.

(6) Risk estimates for excess hospital admissions for asthmatics attributable to O_3 exposures in excess of an estimated background level of 0.04 ppm are projected to be significantly reduced (44 percent) under a 0.08 ppm, 8-hr, 1-expected exceedance standard compared to the current 1-hr NAAQS (see Table VI-2).

(7) The excess hospital admissions risk estimates associated with 1- and 5-expected exceedance standards set at 0.08 ppm are very similar.

(8) When viewed from the perspective of respiratory-related admissions for asthmatics due to all causes, the excess hospital admissions attributable to O_3 exposures in excess of an estimated background concentration of 0.04 ppm constitute a relatively small portion of total admissions. For example, comparing the risk estimates associated with the current 1-hr NAAQS and a 0.08 ppm, 8-hr, 1-expected exceedance standard results in only a 0.6 percent reduction in respiratory hospital admissions for asthmatics due to all causes.

The staff believe, and the CASAC concurred, that the models selected to estimate exposure and risk are appropriate and that the methods used to conduct the health risk assessment represent the state of the art. Nevertheless, there are many sources of uncertainties inherent in such analyses. Some of the most important caveats and limitations concerning the health risk assessment for lung function and respiratory symptom endpoints include: (1) the uncertainties and limitations associated with the exposure analyses discussed above, (2) the extrapolation of exposure-response functions below the lowest observed effects levels to an estimated background level of 0.04 ppm, and (3) the inability to account for

some factors which are known to affect the exposure-response relationships (e.g., assigning children the same symptomatic response rates as observed for adults and not adjusting response rates to reflect the increase and attenuation of responses that have been observed in studies of lung function and symptoms upon repeated exposures).

Similarly, there are uncertainties and limitations associated with the hospital admission risk assessment. These include: (1) the inability at this time to quantitatively extrapolate the risk estimates for the New York City area to other urban areas, (2) uncertainty associated with the underlying epidemiological study that served as the basis for developing the concentration-response relationship used in the analysis, and (3) uncertainties associated with the air quality adjustment procedure used to simulate attainment of alternative standards for the New York City area. A more complete discussion of these uncertainties and limitations is presented in the technical support document (Whitfield et al., 1996).

E. Summary of Staff Recommendations

Drawing on the staff conclusions and observations on margin of safety considerations presented above, together with consideration of the information in the CD and section V of this Staff Paper, the staff offers the following recommendations on the primary O_3 standard.

1. Pollutant indicator

Staff recommends that O_3 remain as the indicator for controlling ambient concentrations of photochemical oxidants. This recommendation is based on the large base of health effects information attributing effects to O_3 exposure and the lack of convincing evidence demonstrating effects from exposure to ambient levels of photochemical oxidants other than O_3.

2. Averaging times

Staff recommends that further consideration be given at this time only to short-term averaging times associated with acute effects. No further consideration of a long-term standard is recommended by staff in this O_3 NAAQS review cycle. The staff offers the following additional specific recommendations:

- Staff recommends that principle consideration be given to an 8-hr averaging time be considered for a new O_3 primary standard.

This recommendation is based on 1) the health effects studies reporting a number of health effects associated with 6- to 8-hr exposures at and below the level of the current 1-hr standard; 2) the staff's judgments that the 6- to 8-hr effects at moderate exertion are of greater public health concern at lower O_3 levels than similar 1-hr effects at heavy exertion, 3) the staff's judgments that these effects are within the range that the Administrator might consider to be adverse; and 4) the exposure assessments and the quantitative risk assessments for some of the effects showing that reductions in the risks associated with these 6- to 8-hr effects can be achieved by attaining alternative 8-hr standards.

Although staff recommends that principle consideration be given to a standard with an 8-hr averaging time, staff recognizes that a standard with a 1-hr averaging time could be set at a level that would provide roughly equivalent health protection to that provided by an 8-hr standard.

3. Form of the Standard

Based on the discussion in section V.I and the conclusions presented above, staff offers the following recommendations with regard to form of the standard and attainment test issues:

- Staff recommends consideration be given to the current expected exceedance form, ranging from 1- to 5-expected exceedances, averaged over 3 years, as well as to a concentration-based form, ranging from the second to the fifth highest 8-hr daily maximum concentration, averaged over 3 years.

- Staff also recommends consideration of defining the standard in terms of a range of air quality values.

Risk analyses discussed above and in Section V.H indicate that for most of the health endpoints analyzed there is little difference in health risk, at a given level of the standard, within the ranges of 1- to 5-expected-exceedances and the second to the fifth highest 8-hr daily maximum concentration forms of the O_3 primary standard. On average, the 1-expected exceedance form provides the greatest exposure and health risk protection but only slightly greater than that provided by the second to the fifth highest 8-hr daily maximum concentration form. There is also not much difference between the fifth 8-hr daily maximum concentration form and a 5-expected exceedance form, which on average are roughly equivalent for any given level of the primary standard selected. Based on these analyses,

therefore, it is the level of the standard which mainly determines the degree of public health protection afforded by an 8-hr primary NAAQS for O_3 within those alternatives considered above.

4. Level of the Standard

In making recommendations, staff notes that the decision ultimately made by the Administrator regarding level of the primary O_3 NAAQS will be based on a policy judgment as to the degree of risk reduction that is necessary to protect public health with an adequate margin of safety. The following recommendations on level address the staff recommendation that consideration be given to a standard with an 8-hr averaging time and a form of 1- to 5-expected-exceedances or a second to the fifth highest 8-hr daily maximum concentration form.

The following staff recommendations suggest a range of levels based on considering: 1) protection against health effects directly associated with both 1- to 3-hr and 6- to 8-hr exposures (e.g., lung function decrements, respiratory symptoms, nonspecific bronchial responsiveness, acute pulmonary inflammation, and increased susceptibility to infection), as well as against the effect of increased hospital admissions; 2) quantitative risk assessments which provide insight as to the degree of protection afforded by alternative 8-hr standards for some of these effects, and 3) protection against the effects of repeated inflammatory responses that could lead over time to chronic respiratory illness.

- **Staff recommends that the upper end of the range of consideration for an 8-hour primary O_3 NAAQS be 0.09 ppm.**

As discussed in the general conclusions and margin of safety considerations presented above, the primary range of lowest effects levels relevant to all the effects of concern identified above is 0.08 to 0.10 pm. As previously discussed, the staff believes that this range of effects levels does not necessarily reflect a threshold below which effects do not occur, but rather may reflect levels at which studies finding statistically significant effects of concern have been conducted. Thus, the staff believes that in assessing the adequacy of health protection afforded by alternative standards levels it is also important to consider: (1) the severity and variability of these effects, (2) the extent to which sensitive or at-risk populations are likely to experience exposures associated with these effects, and (3)

quantitative estimates, when available, of the risk to sensitive and at-risk populations in terms of the estimated numbers or percentages of the populations groups that are likely to experience adverse levels of these effects.

Based on consideration of the above factors, the staff recommends that 0.09 pm is the highest level of an 8-hour standard that would reduce estimated exposures of the at-risk populations sufficiently to provide some margin of safety against pulmonary inflammation and increased susceptibility to pulmonary infection. Further, the staff recommends that 0.09 pm is the highest 8-hr level that would reduce the estimated risk to the at-risk populations of experiencing increased hospital admissions and emergency room visits, as well as experiencing adverse levels of lung function decrements, respiratory symptoms, and nonspecific bronchial responsiveness sufficiently to provide some margin of safety against these effects.

These staff recommendations also reflect consideration of previous advice from the CASAC during the last review of the O_3 NAAQS. In its previous review, the CASAC (McClellan, 1989) concluded that the existing 1-hr primary standard provided "little, if any, margin of safety," and that the upper end of the range of consideration for the 1-hr primary standard should be 0.12 ppm. Several members of the CASAC Ozone NAAQS Review Panel felt that consideration should be given to a 1-hr standard level of 0.10 ppm in order to provide for an adequate margin of safety and to offer some health protection against 8-hr exposures of concern. This advice provides support for considering 0.09 ppm rather than 0.10 ppm as the upper end of the range for an 8-hr standard, in that exposures associated with the 8-hr effects and risks for respiratory symptoms are greater when a 0.10 ppm 8-hr standard is just attained than when a 0.12 ppm 1-hr standard is just attained.

- **Staff recommends that the lower end of the range of consideration for the primary 8-hr O_3 NAAQS be 0.07 ppm.**

In conducting exposure and risk analyses of the 0.06 ppm level, staff concluded that the risk of health effects of concern occurring was extremely low, approaching zero in most cases. Considering both the nature of the health effects involved and the very small percentage of the population that would be affected, staff believes that a primary 8-hr standard with a level of 0.07 ppm could be judged to provide public health protection with an

adequate margin of safety for these effects of concern. A standard set at this level would be more precautionary than a standard set at the upper end of the range, in that it would provide increased protection from long-term exposures that may be associated with potentially more serious but more uncertain chronic effects.

VII. SCIENTIFIC AND TECHNICAL BASIS FOR SECONDARY NAAQS

A. Introduction

This section presents critical information for the review of the secondary NAAQS for O_3. Welfare effects addressed by a secondary NAAQS include, but are not limited to, effects on soils, water, crops, vegetation, man-made materials, animals, wildlife, weather, visibility and climate, damage to and deterioration of property, and hazards to transportation, as well as effects on economic values and on personal comfort and well-being. Of these welfare effects categories, the effects of O_3 on crops, vegetation and ecosystems are of most concern at concentrations typically occurring in the U.S. As stated in the previous CD and SP, "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" (U.S. EPA, 1989). By affecting crops and native vegetation, O_3 also directly and indirectly affects natural ecosystem components such as soils, water, animals, and wildlife and ultimately the ecosystem itself. Some of these impacts have direct, quantifiable economic value, while others are currently not quantifiable. Thus, the staff infers that increasing protection for crops and vegetation would also improve the protection afforded to these other related public welfare categories.

Ozone damages certain manmade materials (e.g., elastomers, textile fibers, dyes, paints, and pigments). The amount of damage to actual in-use materials and the economic consequences of that damage are poorly characterized, however, and the scientific literature contains very little new information to adequately quantify estimates of materials damage from photochemical oxidant exposure (CD, 1996). Effects on personal comfort and well-being have already been addressed under the section of the Staff Paper on human health. Therefore, these effects categories will not be reviewed in this portion of the Staff Paper, and the reader is referred to the last Staff Paper (U.S. EPA, 1989) for a discussion of these effects categories.

The remainder of this chapter focuses on O_3 effects on crops, native vegetation and ecosystems, drawing upon the most relevant information contained in the CD. This information includes: (1) plant response and mode of action of O_3 on vegetation; (2) environmental factors affecting plant response; (3) relevant research on O_3 effects on crops

and native vegetation; (4) considerations and criteria for selecting an appropriate measure of O_3 exposure that can meaningfully relate O_3 air quality to plant response under varying O_3 regimes; and (5) other policy relevant considerations that would assist the Administrator in judging the need for a new secondary standard, including analyses of air quality patterns, the relationships between primary and secondary standard options, national exposures, risks, and economic values.

B. Plant Response/Mode of Action

The first observation of O_3 injury to vegetation in the field (O_3 stipple on grape leaves) was reported in the 1950's (Richards et al., 1958). Since that time, a substantial amount of research has been done on the effects of O_3 on plants that has increased scientific understanding of the mechanisms of action, factors that modify plant response to O_3, and relative sensitivities of various species and cultivars to O_3 concentrations found currently in the U.S.

1. Ozone Uptake

The primary site of O_3 uptake into the plant is the leaf. The leaf is the site of gas exchange for the plant. To cause injury, O_3 must diffuse in the gas-phase from the atmosphere surrounding the leaves through the stomata into the air spaces, dissolve in the water coating the cell walls and, then, its reaction products, diffuse through or react with the membrane of the cell. Once inside the cell, they can react with cellular components and affect metabolic processes (CD, 1996).

The movement of O_3, as well as other gases, into and out of leaves is controlled primarily through the stomata. The aperture of the stomata are controlled by guard cells, which are affected by a variety of internal species-specific factors and external environmental factors such as light, humidity, CO_2 concentration, soil fertility and nutrient availability, water status of the plant and, in some cases, the presence of air pollutants, including O_3 (See U.S. EPA, 1986; Zeiger et al., 1987; Schulze and Hall, 1985; Beadle et al., 1985a and b; Kearns and Assmann, 1993). To investigate variability in diurnal gas exchange, Tenhunen et al. (1980) evaluated diurnal photosynthesis for apricot measured between July and September 1976. While there is a general pattern of increase in the morning and decrease in the evening, the path of photosynthesis (and conductance) are quite different among the days.

For example, mid-day stomatal closure is frequently observed under conditions of high temperature and low water availability (Tenhunen et al., 1980; Weber and Gates, 1990).

However, uptake measured for one leaf may not be the same for any of the other leaves on the plant. Leaves exist in a complex three dimensional environment called a canopy. Each leaf has an unique orientation within that canopy and receives a different exposure to the ambient air. In addition, a plant may be located within a stand of other plants which further modifies ambient air exchange with individual leaves. This makes it difficult to extrapolate from the uptake measured in a single leaf to that of an entire plant or canopy.

Within the canopy, due to its high reactivity, O_3 may be scavenged or absorbed by other environmental components and surfaces before it ever reaches the leaf itself. Though models of stomatal conductance for canopies and stands have been developed to account for some of this complexity, these models require the use of several assumptions that at this time, have not been adequately tested or validated by direct measurements. One particular area that needs further study is the relative importance of cumulative uptake versus the rate of uptake (CD, 1996).

Because O_3 flux incorporates environmental factors and physiological processes, several authors (Grandjean, et al., 1992a,b; Fuhrer et al., 1992) have suggested that it be the relevant measure for use in relating exposure to plant response (CD, 1996). However, measurement of flux for an entire plant or canopy is very complex. Therefore, most research has been done to try to develop appropriate surrogate measures for uptake (discussed below in section VII.E).

2. Extracellular Effects

Once within the leaf, O_3 quickly dissolves in the aqueous layer on the cells lining the air spaces. When O_3 passes into the liquid phase, it undergoes reactions that yield a variety of free radicals (e.g., superoxide and hydroxyl radicals).

Many consider membranes to be the primary site of action of O_3 (Heath, 1988; 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.

3. Intracellular Effects

Once O_3 reaction products diffuse through the cell wall and interact with or diffuse through the cell membrane they may affect cellular or organellar processes. Altered cell structure and function may result in changes in membrane permeability, carbon dioxide fixation, and many secondary metabolic processes (Tingey and Taylor, 1982).

In addition to the disruption and alteration of a number of membrane-dependent functions associated with photosynthesis, O_3 can interfere with the biochemical aspects of the photosynthetic process itself. For example, the enzyme that catalyzes CO_2 fixation during photosynthesis, RuBP, can be inhibited. Nakamura and Saka (1978) reported reduced activity of RuBP carboxylase in rice after exposure to 0.12 ppm O_3 for only 2 hours. Pell and Pearson (1983) observed 36, 68, and 80 percent decreases, respectively, in the concentration of RuBP carboxylase in the foliage of three alfalfa cultivars that had been exposed to an O_3 concentration of 0.25 ppm for 2 hours. These observations were made 48 hours after exposure on leaves that did not exhibit macroscopic injury symptoms (US EPA, 1986), showing that there is no clear connection between foliar injury symptoms and biochemical changes within the leaf.

The potential for O_3, directly or indirectly, to oxidize several other classes of biochemicals including nucleotides, proteins, some amino acids and various lipids has been demonstrated in several in vitro studies. New approaches are needed to assess the full range of in vivo biochemical changes caused by O_3 (US EPA, 1986).

Finally, changes in the in vivo concentrations of various growth regulators or hormones such as ethylene have been shown in a few studies to be correlated with O_3 sensitivity. Because ethylene production also occurs with the ripening of fruit, during periods of stress, and during leaf senescence (Abeles et al., 1992), increased levels of ethylene in the leaves could play a role in the early senescence of foliage. However, because the relationship is complex, the use of ethylene production as an index of sensitivity is still problematic (Pell, 1988).

4. Resistance and Compensation Mechanisms

Plant stress from O_3 occurs when the atmospheric concentrations exceed the limits of plant tolerance. In the leaves, O_3 injury will not occur if the rate of uptake is sufficiently small so that the plant is able to detoxify O_3 or its metabolites. Leaves may physically exclude O_3 from sensitive tissues. A few studies have documented a direct stomatal closure or restriction in response to the presence of O_3. In studies at O_3 concentrations > 0.30 ppm stomatal response was rapid (Moldau et al., 1990). In other studies, reduction in conductance in response to O_3 required hours to days of exposure (Dann and Pell, 1989; Weber et al., 1993).

Additionally, plants may have a biochemical defense in the production of antioxidants. Two general kinds of antioxidants have been reported in plants: 1) reductants and 2) enzymes. In either case excess oxidizing power is dissipated in a controlled manner, effectively protecting the tissue against damage. For detoxification to occur, oxidant and antioxidant must occur proximately and the rate of production of antioxidant must at least balance the rate of oxidant entry into the system. Because potential rates of detoxification for given tissues and the sites in which they occur are not yet known, the effectiveness of these systems in protecting plant tissue from damage to O_3 cannot be determined (CD, 1996).

Once O_3 injury has occurred in leaf tissue, some plants are able to repair or compensate for the O_3 impacts (Tingey and Taylor, 1982). In general, plants have a variety of compensatory mechanisms for low levels of environmental stress, of which O_3 is one. Since these mechanisms are genetically determined, not all plants have the same complement of defensive tools or degree of O_3 tolerance, nor are all stages in a plant's development equally sensitive to O_3.

A wide range of compensatory responses have been identified, including reallocation of resources, changes in root/shoot ratio, production of new tissue, and/or biochemical shifts, such as increased photosynthetic capacity in new foliage, and changes in respiration rates indicating possible repair or replacement of damaged membranes or enzymes. For example, replacement of injured leaf tissue has been reported for some species after exposure to O_3 (Held et al., 1991; Temple et al., 1993) and increased photosynthetic capacity of new needles in O_3 treatments compared to controls. Additionally, ponderosa pine has been shown

to recover from decreased photosynthetic rates in O_3-treated needles. In one case injured needles were able to regain the photosynthetic rate of controls after 40-50 days (Weber et al., 1993).

While these systems potentially provide protection against O_3 alteration to tissue physiology, it is not yet known to what degree or how the use of plant resources for repair processes affects the overall carbohydrate budget or subsequent plant response to O_3 or other stresses (CD, 1996).

5. Physiological Effects

The effects of O_3 injury at the cellular level in the ways described above, when they have accumulated sufficiently, will be propagated to the level of the whole leaf or plant. These larger scale effects can include: (1) visible foliar injury; (2) premature needle/leaf senescence; (3) reduced photosynthesis; (4) reduced carbohydrate production and allocation; (5) reduced plant vigor; and (6) reduced growth or reproduction or both (Miller et al., 1982; McLaughlin et al., 1982; Skelly et al., 1984; U.S. EPA, 1986).

Visible Foliar Injury. Although O_3 can significantly alter cellular and photosynthetic processes without resulting in changes in leaf appearance, cellular injury can and often does become visible. For coniferous trees, two visibly recognizable syndromes have been associated with oxidant injury. One, emergent tipburn, is noted most often on eastern white pine. This injury is characterized as a tip dieback of newly elongating needles. Silvery or chlorotic (absence or deficiency of green pigment) flecks, chlorotic mottling, and tip necrosis (tissue death) of needles may also be present. The other O_3 injury response, chlorotic decline, results in the loss of all but the current season's needles and was first noted on ponderosa pine. Yellow mottling and a reduction in the number and size of the remaining needles may also occur. In non-coniferous species, acute O_3 injury usually results in cell destruction (bifacial necrosis) due to the disruption of normal cell structure and processes and the subsequent loss of water and salts from the cell (U.S. EPA, 1978).

Acute injury usually appears within 24 hours after exposure to O_3 and, depending on the species, can occur under a range of exposures and durations. For example, a summary of limiting values for visible injury showed effects occurring across a range of exposures

from 0.04 ppm for a period of 4 h to 0.41 ppm for 0.5 h for crops, and 0.06 ppm for 4 h to 0.51 ppm for 1 h for trees and shrubs (U.S. EPA, 1986).

Chronic injury may be mild or severe, and is associated with long-term or multiple exposures to elevated O_3 levels. Under chronic exposures, disruption of normal cellular activity occurs, leading to chlorotic mottling, bleached or unpigmented lesions (flecks) or pigmentation (stippling). Though cell death may eventually result, depending on dose and environmental conditions, membrane permeability may be restored and cell recovery occur (U.S. EPA, 1978). Chronic O_3 injury patterns may be confused with symptoms resulting from normal senescence, biotic pathogens, including insects, nutritional disorders, or other environmental stresses. These patterns may appear as premature leaf senescence.

The significance of O_3 injury at the leaf level depends on how much of the total leaf area of the plant has been affected, as well as the plant's age and size, developmental stage, and degree of functional redundancy among the existing leaf area. As a result, it is not presently possible to determine with consistency across species and environments what degree of injury at the leaf level has significance to the vigor of the whole plant.

Premature Needle/Leaf Senescence. Ozone has been shown to affect needle or leaf retention in loblolly pine (Stow et al., 1992; Kress et al., 1992), slash pine (Byres et al., 1992), aspen (Keller, 1988; Matyssek et al., 1993a,b) and apple (Wiltshire et al., 1993), as well as other species. Leaf replacement may be part of the normal growth strategy employed by the plant to maintain photosynthetic production. However, leaves that have to be replaced more frequently drain energy from plant reserves and those that are irreplaceably lost represent a net loss of photosynthetic capacity that can have significant effects on plant vigor.

Impaired Photosynthesis: Changed Carbohydrate Production and Allocation. Photosynthesis, the process by which plants produce energy-rich compounds (e.g.,) for use in growth, maintenance, reproduction or storage, can be impaired by O_3. This impairment may result from the direct impact of O_3 on chloroplast function or from O_3-induced stomatal closure resulting in reduced CO_2 uptake, or both. As discussed above, this can occur without any macroscopic visible injury.

If total plant photosynthesis is sufficiently reduced, the plant will respond by reallocating the remaining carbohydrate at the level of the whole organism. Since the roots are often the largest source of stored carbohydrate, they (Andersen et al., 1991; Andersen

and Rygiewicz, 1991) and associated mycorrhizal fungi (Adams and O'Neil, 1991; Edwards and Kelly, 1992; McQuattie and Schier, 1992; Meier et al., 1990; Taylor and Davies, 1990) become especially susceptible to reduced carbohydrate availability, and quite frequently show the greatest decline in growth. Cooley and Manning (1987) reviewed the literature on carbohydrate partitioning and noted that "storage organs . . . are most affected by O_3-induced partitioning changes when O_3 concentrations are in the range commonly observed in polluted ambient air."

When less carbohydrates are present in roots, less energy will be available for root-related functions such as the acquisition of water and nutrients. Mycorrhizal fungi, which invade the roots of terrestrial plants, are of great importance for vegetational growth (U.S. EPA, 1978). These fungi increase the solubility of minerals, improve the uptake of nutrients for host plants, protect host roots against pathogens, produce plant growth hormones, and may transport carbohydrate from one plant to another (CD, 1996). Ozone has the capability of disrupting the association between the mycorrhizal fungi and host plants by inhibiting photosynthesis and the amount of sugars available for transfer to the roots. In one example, Berry (1961) examined the roots of eastern white pine injured by O_3 and observed that healthy trees had almost twice the percentage of living feeder roots as trees with O_3 injury. Primary roots of affected trees have even been shown to die after repeated needle injury (U.S. EPA, 1978).

Unlike root systems, effects on leaf and needle carbohydrate content under conditions of O_3 stress have ranged from a reduction (Barnes et al., 1990; Miller et al., 1989), to no effect (Alscher et al., 1989), to an increase (Luethy-Krause and Landolt, 1990). Friend and Tomlinson (1992) found that O_3 exposure increased retention of C_{14}-labelled photosynthate in needles of loblolly pine, and modified its distribution among starch, lipids, and organic acids (Edwards et al., 1992; Friend et al., 1992). These responses have been measured in ponderosa pine seedlings exposed to O_3 concentrations of 0.10 ppm for 6 hr/day for 20 weeks (Tingey et al., 1976).

Reduced Plant Vigor. There is no evidence to suggest that O_3 levels over most of the U.S. are high enough to kill vegetation directly. However, at current ambient levels that occur during O_3 episodes, the ability of many sensitive species or genotypes within species to

adapt to other environmental stresses, including competition for available resources, can be sufficiently compromised such that the end results prove fatal for some plants. For example, McLaughlin et al. (1982) observed that the reduced availability of carbohydrates associated with O_3 exposure resulted in enhanced susceptibility of trees to root disease and influenced the success of pest infestations (Hain, 1987; Lechowiez, 1987). Fincher (1992) and Davison et al. (1988) found that O_3 also can decrease the ability of trees to withstand winter injury caused by exposure to freezing temperatures.

Reduced Growth and/or Reproduction. As discussed above, O_3 exposure can reduce carbohydrate production or storage in plants. In annual species this affects plant growth, flowering, and seed development. Unlike annuals, deciduous perennials that must survive more than one year and develop new leaves each year after a period of dormancy depend on long-term storage of carbohydrates to get them through unfavorable growth periods. Thus, while no O_3 effects on growth may be observed above ground during a year of elevated O_3 levels, the following year may show a decrease in root growth or new biomass production.

Coniferous species also must maintain foliage from one year to the next, and may, in some spruce species, retain as many as 10 years of needles at any point in time, and continue to produce carbohydrates even during winter months. Therefore, injury to or early loss of needles can result in a greater shift in remaining carbohydrates to repair and replacement of needles, thus potentially reducing biomass production. When storage carbohydrates are limited, older needles may become the source of photosynthate for new needle growth in the spring and storage sinks in the fall (McLaughlin et al., 1982). Thus, O_3 impacts may be felt over multiple years. These "carry-over" effects have been documented in the growth of tree seedlings (Hogsett et al., 1989; Sasek et al., 1991; Temple et al., 1993) and in regrowth of roots (Andersen et al., 1991). Controlled exposures, however, have been for the most part only 2-3 years in duration so that data on the cumulative effects of multiple years of O_3 exposure are extremely limited (CD, 1996).

C. Environmental Factors Affecting Plant Response

Plant response to O_3 exposure is a function of the plant's ongoing integration of biological, physical and chemical factors both within and external to the plant. The corollary is also true: O_3 exposure can modify the plant's subsequent integrated response to other

environmental factors. Thus, there are inherent multiple sources of uncertainty which must be recognized in relating plant response under one set of growing conditions to responses under other conditions. Additionally, the numerous methodologies used in vegetation research have been designed to address particular sets of uncertainties and answer particular type of questions. When discussing the results of a study, the uncertainties introduced by the type of methodology used should also be recognized.

1. Biological Factors

Genetics. The genetic code of each plant contains a gene or genes that govern its response to O_3. Even within the genomes of a particular plant species, there is wide variability in O_3 sensitivity. This has been amply demonstrated through inter- and intraspecific comparisons which have shown that it is not uncommon to have a species with genotypes that vary by as much as 50% in the same study. These findings have significant implications for predicting plant response to O_3. First, an exposure response relationship generated for a single genotype or small group of genotypes may not adequately represent the response of the species as a whole (Temple, 1990). Further, a study that uses only the most sensitive genotypes within a species might overestimate the injury being done to the species as a whole.

Secondly, this variability in response means that O_3 can impose a selective force favoring tolerant genotypes over sensitive ones. For example, sensitive species are unable to compete for the required water and nutrients, or may not be able to reproduce (Roose et al., 1982; Treshow, 1980).

Numerous studies show that it is likely that sensitive genotypes are being lost from natural ecosystems at current ambient O_3 exposures in some parts of the U.S. Berrang et al. (1986, 1989, 1991) have presented evidence for population change in trembling aspen (Populus tremuloides L.) by showing that aspen clones from polluted areas were visibly injured to a lesser degree than those taken from unpolluted areas. Additionally, Karnosky (1981, 1989) studied the O_3 symptom expression and survival of over 1,500 eastern white pine trees growing in southern Wisconsin and found that O_3-sensitive genotypes had a ten-times-higher rate of mortality than did the O_3-resistant genotypes over a 15-year study. During the 1970's, significant numbers of sensitive white pine were lost from the

Cumberland Plateau in Tennessee. Heagle et al. (1991) found a population change in O_3 sensitivity in white clover (Trifolium repens L) after two years of O_3 exposures in open top chambers, and Gillespie and Winner (1989) found O_3 to be a strong and rapid selective force with radish cultivar "Cherry Belle."

Limited evidence also suggests that O_3 may affect the reproductive success of O_3 sensitive species. Studies on the effects of O_3 on pollen germination and tube elongation of some Scott's pine, eastern white pine, corn, petunia, and tomato generally found a negative impact of O_3 on this critical element of reproduction (CD, 1996). Reduced flowering as the result of prolonged fumigation with O_3 has been shown in Bladder campion, geranium, and carnation. This effect reduces the fitness of the affected genotypes, and may result in the eventual loss of these genetic units from O_3-stressed environments.

Plant breeders working in locations with high O_3 concentrations have developed varieties more tolerant to O_3 than those developed under low O_3 conditions for such species as alfalfa, potato, cotton, and sugar beet (CD, 1996). Likewise, nursery owners, Christmas tree growers, and seed orchard managers have all routinely discarded pollution-sensitive chlorotic dwarf and tipburned white pine trees because of their slow growth in areas with high O_3 (Umbach and Davis, 1984), and thus, contributed to the selection of more tolerant commercial forests.

In natural ecosystems, the loss of genetic diversity is considered an adverse impact by Federal Land Managers of Federal Class I areas who have been given the charge to preserve for future generations the genetic resources within their borders. In addition, such loss may have economic implications for commercially important species if the remaining populations are made up of O_3 resistant plants that are less adaptable to subsequent change, or if the O_3 tolerant trait is linked to other traits such as slower growth and productivity.

Pollutant/Plant/Pest/Pathogen Interactions. Significant research has been done on this topic since the 1986 Criteria Document. Several recent studies on the effects of O_3 on the feeding preference of herbivorous insects, and on their growth, fecundity, and survival have reported that O_3-induced changes in the host plants frequently result in increased feeding preference of a range of insect species. For example, Chappelka et al. (1988) found that O_3 enhanced the feeding preference and larval growth of the Mexican bean beetle on soybean,

leading to increased defoliation. Similarly, stimulatory responses were observed with pinworm on tomato, with aphid and weevil on European beech, with the monarch butterfly on milkweed, and with infestation by the willow leaf beetle on cottonwood (CD, 1996). These data do not provide, however, a consistent relationship between different levels or patterns of O_3 exposure and insect growth response. Additionally, the reports of O_3-insect-plant interactions only represent a small fraction of the interactions that exist, making generalization to other combinations uncertain.

With respect to the interaction between O_3 and plant disease, new information since the 1986 Criteria Document changes the earlier conclusion from it is "impossible to generalize and predict effects in particular situations" (U.S. EPA, 1986) to the conclusion in the CD that "pathogens which can benefit from injured host cells or from disordered transport mechanisms (facultive) are enhanced by pollution insult to their hosts, whereas those that require a healthy mature host for successful invasion and development (obligate) are depressed by pollutant stress to their host." In a few studies, infection of the plant with obligate bacteria or pathogens or nematodes tended to reduce the impact of O_3. The majority of these studies have been conducted in laboratories or greenhouses, which raises the question of relevance under field conditions. Much more study is needed, and with a wider range of species, to quantify the magnitude of the interactive effects to different levels of O_3 exposure.

Pollutant/Plant/Plant Interactions. While vegetation literature is replete with experimental studies associating O_3 exposure with observed effects on plants, any attempt to extrapolate these results to field conditions must recognize that other factors such as competition with other plant species for limited resources such as light, water, nutrients and space can effect the degree of injury observed. Several studies have reported that environmental and site conditions often explain the patterns of O_3 injury for a given species more than the actual O_3 concentration levels. For example, it has been reported that canopy trees can be more affected than understory, and that ponderosa pines growing at the top of ridges or on dry sites experienced greater foliar injury than those grown elsewhere (Second Progress Report of FOREST, 1994). Though very few studies have been conducted to evaluate the effects of O_3 on competition between species, it is clear that the implication of

the known effects of O_3 described in the previous section tend to impair a plant's ability to compete with other species. For example, a shift in allocation of carbohydrates away from roots to leaves and shoots results in a compromised root system which limits the plant's ability to explore the soil for water and nutrients, and injury and/or loss of leaves would limit the plant's ability to take advantage of available light. A prime example of O_3-induced shifts in species dominance is that observed in studies of the San Bernardino Forests, as discussed below in Section VII.D.

Competition may be either between plants of different (inter-) or the same (intra-) species. The planting densities and row spacings adopted for agricultural crops represent compromises between maximizing the number of plants per unit area and the adverse effects of intra-species competition. Though weeds are typical inter-species competitors, no studies appear to have been conducted on the effects of O_3 pollution on such competition. Inter-species competition also occurs in mixed plantings such as grass-clover forage and pasture plantings, and is an important feature of natural ecosystems. A consistent finding with grass-clover mixtures has been a significant shift in the mixture biomass in favor of the grass species (CD, 1996).

Recently, the development of field exposure systems have permitted some studies of crop species to be conducted in the field. Because the crops were planted at normal planting densities, inter-species competition was incorporated as an environmental factor. On the other hand, most forest tree studies have tended to be "artificial" in their use of individual seedlings or saplings or spaced trees, even when exposed to open-air systems (McLeod et al., 1992). The significance of the effects of competitive interactions on the O_3 response of the competing species is largely unknown, and leads to considerable uncertainty when extrapolating from effects on individual species to managed and natural ecosystems.

2. Physical Factors

The physical components of a plant's aerial environment are light, temperature, humidity, wind velocity and surface wetness, while the physical, edaphic components affecting the plant roots are temperature and soil moisture and salinity. Since the effects of the physical climatic factors on plant growth are major determinants of the geographic distribution of the earth's natural vegetation and of the distribution of agricultural lands and

the suitability of the crops grown on them, it is important to note when experimental conditions vary from those which are normally found in the field.

Light. In most species, light plays a major role in the opening and closing of stomata, thus dictating to some extent when O_3 can be taken up by foliage from the ambient air. Because many studies are done under reduced light intensities, it is important to note the general conclusion reported previously (U.S. EPA, 1986) that susceptibility to foliar injury is increased by low light intensities and short photoperiods. Reduced light intensities have been measured in open-top chambers in the field, resulting from the build-up of dust on the walls. However, Heagle and Letchworth (1982) could detect no significant effects on soybean growth and yield in a comparison of plants grown in unshaded open-top chambers and chambers to which shading cloth was applied.

Temperature. An important O_3-temperature interaction affecting trees and other woody perennials is winter hardiness. Several studies have shown that exposures to O_3 at realistic levels may reduce the cold- or frost-hardiness of plants, as reviewed by Davison et al. (1988). It is the temperature within the plant tissues that is important, because it affects almost all physical and chemical processes within the plant. However, in addition to air temperature, the temperature of the leaf is determined by the absorption of infra-red radiation and the loss of water vapor through transpiration. Further, temperature within the leaf has been shown to rise with the closing of stomata (Matsushima et al., 1985; Temple and Benoit, 1988). Therefore, since vapor pressure deficit and degree of stomatal closure control the rate of evapotranspiration, the effects of temperature are unavoidably confounded with these other factors.

Water Usage and Availability. Water is essential to plant survival, growth and reproduction. Because different regions of the country have different water regimes, plants growing in each of these regions are those adapted to the fluctuating water supplies from season to season. These differences among species make it difficult to draw firm conclusions about the nature of the relationship between soil moisture deficit (SMD) and O_3 effects. For example, Bytnerowicz et al. (1988) found no interaction between SMD and O_3 effects in 18 desert annual species. On the other hand, in the more mesic environment of the mid-Ohio River Valley, a field survey of milkweed revealed much less foliar injury attributable to O_3

in 1988 (a dry year), even with O_3 concentrations reaching 0.2 ppm, than in 1989 (a year with ample precipitation), and a maximum O_3 concentration of only 0.12 ppm (Showman, 1991). Though, in the latter case, SMD seemed to confer some degree of O_3 resistance, this cannot be extrapolated to other species which have not been studied. Further, the relationship between SMD and O_3 may also change throughout the life of a plant or growing season, as a plant's sensitivity to water stress varies with stage of plant development (Moser et al., 1988).

Recognizing the possibility of an interaction of drought with O_3 on the yield of agricultural crops, the National Crop Loss Assessment Network (NCLAN) studies conducted several experiments to examine this relationship. Out of eleven studies (six soybean, three cotton, and one each of alfalfa and clover-fescue), only half (three soybean, two cotton, and the alfalfa) showed significant interactions between SMD and O_3. In some cases, the lack of a significant response to O_3 reflects a decreased range of yield response under SMD within which an O_3 effect could be ascertained. Unfortunately, because different measures of SMD or SMD-induced stress were used in different studies, it is not possible to quantify the relationship between the suppression of the O_3 response and the level of drought stress. Additionally, soil conditions and the depth of the water table at different sites appear to influence the O_3 response as well (Heggestad et al., 1988).

Trees have been the subject of several recent studies on the interaction between SMD and O_3. Though there is no consistency among the studies in the treatments used or the measurements made, these studies do provide some support for the view that drought stress may reduce the impact of O_3. For example, beech, poplar, and loblolly pine seedlings have clearly demonstrated significant interactions. The CD reports that drought has reduced O_3-induced foliar injury to poplar, ponderosa pine, and loblolly pine. This work with trees, however, is not yet at the point to allow quantification of the O_3-drought interaction.

The bulk of the evidence supports the view that drought stress may reduce the impact of O_3 on plants. However, it must be emphasized that, in terms of growth and productivity, any "protective" benefit will be offset by the effects of SMD per se, as noted in the previous criteria document (U.S. EPA, 1986).

The O_3-water interaction is not confined to the effects of SMD on direct plant response to O_3. Some studies have shown that O_3 may affect various other aspects of plant water status, including water use efficiency (WUE). However, WUE is a complex resultant of both stomatal conductance and the activity of the photosynthetic system, both of which may be independently affected by O_3. Only one study has been performed on trees, and this was done at high O_3 concentrations (Johnson and Taylor, 1989). Though the foliage of loblolly pine seedlings at these higher levels adapted to a more efficient use of water, more study will be needed before it will be possible to generalize about the implications of this effect and its importance for mature trees and forest ecosystems.

Finally, the relative humidity (RH) of the ambient air can significantly influence O_3 uptake. In one study using pinto beans, O_3 uptake increased fourfold at an O_3 concentration of 0.079 ppm when the relative humidity was increased from 35% to 73% (McLaughlin and Taylor, 1981). However, stomatal responses to O_3 show considerable variability among species and even among cultivars of the same species (Elkiey, et al., 1979). The influence of RH on plant sensitivity may explain important variations in plant response under field conditions (U.S. EPA, 1986).

3. Chemical Factors

Nutritional Factors. Plants require a supply of mineral nutrients such as nitrogen, potassium, phosphorus, sulfur, magnesium and calcium for growth. For optimal growth, these supplies must be balanced. A number of studies have examined the relationship between nutrient status and plant response to O_3 exposure. Heagle (1979) found that injury and growth reductions tended to be greatest at normal levels of fertility, though the effects also were dependent on the rooting medium used. It has also been reported that increased levels of phosphorus, potassium, and sulfur have resulted in a decrease in sensitivity to O_3 (CD, 1996). On the other hand, with respect to nitrogen (the area where the most of the nutritional research has been done), the results have been mixed.

It has been suggested that the relationship between O_3 sensitivity and nutrient condition could be better characterized if studies began with a knowledge of actual plant tissue nutrient levels at the time of exposure to O_3. Cowling and Koziol (1982) indicate that

differences in sensitivity are ultimately linked to changes in the status of soluble carbohydrates in the plant tissues.

Since these nutritional studies used different combinations of nutrients, species, and experimental conditions, the results cannot be integrated to develop a general relationship between soil fertility and sensitivity to O_3. In view of the vast number of possible permutations and combination of nutrient elements and their levels that may exert effects on O_3 response, a concerted effort by researchers to use standardized protocols will have to be made if the uncertainties associated with the role of nutritional status on O_3 sensitivity is to be better understood.

Interactions with Other Pollutants. The concurrent or sequential exposure of vegetation to different gaseous air pollutants has been found to modify the magnitude and nature of the response to individual pollutants (U.S. EPA, 1986). Lefohn and Tingey (1984) and Lefohn et al. (1987) reviewed the patterns of co-occurrence of O_3, SO_2, and NO_2 in urban, rural, and remote sites in the U.S. for the years 1978 to 1982 and found that co-occurrences were usually of short duration and occurred infrequently. The most frequent types of co-occurrence were either purely sequential or a combination of sequential and overlapping exposures of short duration. This discussion will focus only on those studies which use exposure patterns or levels that are typical of ambient air.

An exception is the co-occurrence of PAN and O_3, which are both constituents of photochemical oxidant. The few studies that have been done on this combination, reviewed in the 1986 Criteria Document (U.S. EPA, 1986), show that the two gases tend to act antagonistically in both concurrent and sequential exposures. At the present, no studies have looked at the interactions between O_3 and hydrogen peroxide (H_2O_2), which is another constituent of photochemically polluted atmospheres.

Despite the fact that the photochemical formation of O_3 involves a complex series of reactions in which NO, NO_2 and HNO_3 participate as intermediate reaction products, and that in many areas daily peak O_3 levels are followed by increasing NO_2 levels, only a few studies have been done to explore possible interactive effects with O_3 and are confined to the nitrogen species, NO_2 (CD, 1996). These studies have reported both antagonistic and

synergistic or additive interactions between O_3 and NO_2 even with the same species, but, with such limited information, it is not possible to generalize the response at this time.

A large number of studies have examined the relationship between O_3 and SO_2 (CD, 1996). These studies have used a wide variety of species, exposure regimes, and experimental conditions. Because of the contradictory nature of the results from these studies, all that can be concluded is that the type of interaction, and whether or not one exists, is probably highly dependent upon species and cultivar, and possibly other environmental variables. The available evidence is insufficient to be able to decide in which way and to what extent SO_2 exposure will influence the effects of O_3 on a particular species or cultivar at a particular location.

The recognition of the damaging effects of acid rain on various terrestrial and aquatic systems has led to numerous studies of the combined effects of O_3 and simulated acid rain (SAR) or acid fog. Due to concern over the possible role of exposures to acid rain or acid fog and O_3 in the forest decline syndrome, several of the more recent studies have focused on forest tree species. Of over 80 recent reports of studies on over 30 species, more than 75% indicated no significant interactions between O_3 and SAR or acid fog (CD, 1996). However, in other studies, statistically significant interactions have been reported for several species. In most cases where significant interactions on growth or physiology have been reported, the interactions were mostly antagonistic. Overall, it appears that exposure to acidic precipitation is unlikely to result in significant enhancement of the effects of O_3 in most species. In the few cases of antagonistic interactions, the suggestion was made that these may have reflected a beneficial fertilizer effect due to the nitrate and sulfate present in the SAR applied.

Only a few studies have been published on CO_2/O_3 interactions. CO_2 alone has been found to increase leaf area and stimulate photosynthetic rates, which can have the secondary effects of inducing stomatal closure, reducing transpiration, and increasing leaf temperature. When applied together, CO_2 countered the negative effects of O_3 on photosynthesis, shoot growth rate, leaf area, and water use efficiency for radish and soybean. Because these studies were conducted in growth chambers or open-top field chambers, uncertainties due to variable environmental conditions would be introduced when applying these results to the

open field. It is further unclear whether such CO_2-induced reductions of the impact of O_3 also apply to the long term growth of trees, and how increased CO_2 will affect the impact of O_3 on ecosystems.

A limited database exists for studies involving mixtures of O_3 with two or more pollutants. Due to the small number of studies, it is difficult to draw any firm conclusions. In general, the consequences of such exposures appear to be largely dictated by the dominant individual two-way interaction (CD, 1996).

Agricultural Chemicals. Several categories of compounds (commercial fungicides, herbicides and growth regulators) that are routinely applied to agricultural plants have been found in some cases to protect against O_3 injury. Though no comprehensive and systematic studies have been reported, the existing data indicate that certain fungicides are consistent in providing protection (CD, 1996). Most of the effective fungicides have been carbamates and have also been used as antioxidants in other applications such as rubber formulations. Other compounds used as growth regulators and herbicides have also been reported to protect some plants against O_3 injury. However, these results appear to be species- or cultivar-dependent (CD, 1996). Other than noting the general efficacy of the carbamate fungicides, knowledge of the interactions of these different types of agricultural chemicals with O_3 is still too fragmentary to be able to draw any general conclusions. Thus, it is considered premature to recommend their use specifically for protecting crops from the adverse effects of O_3, rather than for their primary purpose (CD, 1996).

D. Ozone Effects on Crops and Other Vegetation

This section presents information on vegetation effects associated with exposures to O_3. Effects discussed include: 1) visible foliar injury, 2) growth reductions and yield loss in annual crops and other species, 3) growth reductions in tree seedlings and mature trees, and 4) effects that can have impacts at the forest and ecosystem levels. The section highlights results from observational and controlled studies, together with the limitations and uncertainties associated with the studies.

The results presented in this section are in terms of a number of different air quality index forms. Table VII-1, which presents the 10-year summary of yearly average air quality monitored at U.S. sites for the years 1982 to 1991 for three selected forms, is included here

Table VII-1. SUMMARY OF OZONE EXPOSURE INDICES CALCULATED FOR 3-MONTH GROWING SEASONS FROM 1982 to 1991

Year	No. Sites	M7 ppm Mean	M7 ppm CV	SUM06 ppm·h Mean	SUM06 ppm·h CV	SIGMOID ppm·h Mean	SIGMOID ppm·h CV
1982	99	0.052	18.7%	26.8	68.8%	26.3	56.7%
1983	102	0.056	21.9%	34.5	58.1%	33.0	52.3%
1984	104	0.052	18.2%	27.7	58.4%	27.4	47.9%
1985	117	0.052	17.1%	27.4	59.6%	27.4	47.6%
1986	123	0.052	19.1%	27.7	65.0%	27.7	51.8%
1987	121	0.055	17.6%	31.2	56.4%	30.4	46.8%
1988	139	0.060	17.8%	45.2	46.8%	42.9	42.4%
1989	171	0.051	17.5%	24.8	78.7%	25.8	59.4%
1990	188	0.053	18.3%	25.8	76.2%	26.6	59.2%
1991	199	0.054	18.4%	28.3	74.2%	28.9	59.5%
Among Years		0.054	10.0%	29.5	42.1%	29.4	31.0%

Modified from Table 5-20, U.S. EPA, 1996

to provide a context in which to consider the reported vegetation effects. These forms include the M7 (seven hour seasonal mean), the SUM06 (all hourly O_3 concentrations equal to or above 0.06 ppm summed over 3 months), and an example of a sigmoidal form, SIGMOID, (all hourly concentrations weighted by a specific sigmoidal weighting function and summed over 3 months). Another sigmoidal form used in this chapter, W126, has an inflection point at 0.067 ppm and gives equal weight to values above 0.10 ppm.

1. Visible Foliar Injury

Visible foliar injury can be an effect of concern either when it directly represents loss in the intended use of the plant, ranging from reduced yield and marketability to impairment of the aesthetic value of individual plants or natural landscapes, or when it serves as an indicator of the presence of concentrations of O_3 in the ambient air which are associated with more serious effects. Because visible foliar injury was the first effect of O_3 to be observed, the database associated with it is large and covers a wide variety of species. However, much of this database is incomplete in terms of characterizing the O_3 concentrations and exposure regimes that were experienced by plants in the field, or was produced under unrealistically high or low O_3 exposure levels in artificial growing conditions. Studies conducted more recently have begun to remedy those limitations.

Reduced Yield or Marketability. Loss of use may occur when changes in quality and/or physical appearance result in reduced yield or marketability of leafy crops (e.g., spinach, lettuce, cabbage) and ornamental plants. Unfortunately, little research has been done to describe the relationship between O_3 concentrations and changes in visible responses on leafy crops and ornamentals. Heck et al. (1984b) summarize O_3 effects on a variety of vegetables. Four varieties of spinach are shown to incur 10% yield loss and 30% yield loss over the ranges of 0.043 to 0.049 ppm and 0.08 to 0.082 ppm (7 h seasonal means), respectively (U.S. EPA, 1986). Additionally, Empire lettuce was reported to experience a 10% and 30% yield loss at the 7 hr seasonal mean concentrations of 0.053 ppm and 0.075 ppm, respectively. Temple et al. (1986) reported a 35% reduction in lettuce head weight at 0.128 ppm (7 h mean over 52 days), while Olszyk et al. (1986) found no effects at a 7 week mean of 0.034 ppm. Earlier studies, cited in the 1978 CD, reported that to prevent visible foliar symptoms for crops, concentrations in the range of 0.10 to 0.25 ppm for a duration of

1 hour were identified as a limiting value, which decreased to 0.04 ppm to 0.09 ppm when duration was increased to 4 hours. For trees, the ranges of concentration were slightly higher, including 0.06 to 0.17 ppm at the 4 hour duration. These limiting values are still considered relevant today, although it is recognized that the studies available at the time often used experimental protocols that were unrealistic with respect to the natural growing environment of the plants (CD, 1996).

Foliar symptoms that can decrease the value of ornamentals including turf grasses, floral foliage, and ornamental trees and shrubs have also been reported. For example, when petunia, geranium, and poinsettia were exposed to O_3 for 6 h/day for 9 days (petunia), 8 days (geranium), and 50 days (poinsettia), flower size was significantly reduced in all three species at a concentration of 0.10 to 0.12 ppm, and flower color was reduced at the same or lower concentrations. All of these changes in flower appearance occurred without visible injury to the plant leaves. Ozone concentrations of 0.10 ppm for 3.5 h/day for 5 days or 0.20 ppm for 2 h were high enough to elicit injury in most turf grasses (U.S. EPA, 1986).

Impairment of Aesthetic Value. On a larger scale, foliar injury currently occurring on native vegetation in national parks, forests, and wilderness areas in some cases may be degrading the aesthetic quality of the natural landscape, a resource important to public welfare. The first concerted effort to relate total oxidant concentrations in national forests to injury in white pine began in 1975. Injury was observed in the Jefferson and George Washington National Forests and throughout the Blue Ridge Mountains, including areas of the Shenandoah National Park (Hayes and Skelly, 1977; Skelly et al., 1984). Taylor and Norby (1985) report that there were an average of five episodes (i.e., any day with a 1 h concentration $>$ 0.08 ppm) during the growing season in this area, with episodes lasting from 1 to 3 consecutive days.

In the Great Smoky Mountains National Park, surveys made in the summers from 1987 through 1990 found 95 plant species, including herbaceous and woody plants, exhibited foliar injury symptoms consistent with those thought to be caused by O_3 (Neufeld, et al., 1992). At the same time, O_3 monitoring data indicated that there were both elevated concentrations and prolonged exposures to O_3, especially at the higher elevation sites which could experience as much as 2 times the levels experienced at lower elevation sites. In order

to verify that O_3 produced these symptoms, 28 species that had shown foliar injury symptoms in the field were fumigated with O_3 in open-top chambers. Twenty-five of the 28 showed foliar injury symptoms like those found in the field in response to O_3 (Neufeld, et al. 1992).

In a similar survey, Chappelka et al. (1992) examined black cherry, yellow poplar, sassafras, and white ash in the Shenandoah and Great Smoky Mountains National Parks. Black cherry exhibited foliar injury symptoms in both parks, with the percentage of leaves injured in 1991 ranging from 18 to 40% and from 8 to 29% in the two parks, respectively. Black cherry also exhibited the highest percentage of symptomatic trees (97%).

The western U.S. contains the largest forested area in the world documented to have visible injury from high O_3 exposures, the Sierra Nevada Mountains, an area approximately 300 miles long (Peterson and Arbaugh, 1992). Foliar O_3 injury to ponderosa and Jeffrey pine was first documented there in the early 1970's (Miller and Millecan, 1971). Monitoring of visible injury to ponderosa pine on National Forest land in the western Sierra Nevadas, however, was not begun until 1975 (Duriscoe and Stolte, 1989). Results of the monitoring in the Sierra Nevada and Sequoia National Forests showed that there was an increase in chlorotic mottle of pines in the plots from approximately 20% in 1977 to 55% in 1988, and an increase in severity of injury as well. Sequoia National Forest and the Sequoia-Kings Canyon National Park, the southernmost federal administrative units, have the highest O_3 levels, with mean hourly averages ranging from 0.018 to 0.076 ppm, and annual hourly maxima of 0.11 to 0.17 ppm for 1987.

Since 1991, there has been an annual survey of the amount of crown injury by O_3 to the same trees in approximately 33 sample plots located in several National Parks and Forests in the Sierra Nevada Mountains. Dominant tree species in the area are ponderosa and Jeffrey pine, white fir, sugar pine, incense cedar, Douglas fir, California black oak, and the giant sequoia (Peterson and Arbaugh, 1992). Big cone Douglas fir is usually rated as less sensitive than ponderosa or Jeffrey pine; however, injury symptoms resulting from elevated O_3 have been seen (Peterson et al., 1995). Based on their study, the authors conclude that while O_3 does not have the same level of impact on this tree as on ponderosa and Jeffrey pine, reduced needle retention and lower recent growth rates could indicate increased O_3 stress (or O_3 stress mediated by climate) in big cone Douglas fir (CD, 1996).

Visible Injury as an Ozone Indicator. Though visible foliar injury cannot at present serve as a surrogate measure for other O_3-related vegetation effects, it can be a useful indicator that phytotoxic concentrations of O_3 are present in the ambient air. It can thus serve as a warning that other O_3 impacts may be taking place on the injured plant or other nearby vegetation.

Several field studies have been conducted which successfully used sensitive species as bioindicators of O_3 concentration including studies of morning glory (Nouchi and Aoki, 1979), milkweed (Douchelle and Skelly, 1981), and pinto bean (Oshima, 1975). The value of deploying networks of bioindicators for the early detection of developing regional oxidant pollution problems, identification of trends in pollutant occurrence, and the supplementation of physical monitoring networks has been demonstrated (Laurence, 1984). This use of plants as bioindicators is an important element of the Environmental Monitoring and Assessment Program (EMAP), which seeks to identify and document associations between selected indicators of natural and anthropogenic stresses and the condition of ecological resources. This information can then be used to track national trends in pollution and provide sound data on which to base environmental risk management decisions (U.S. EPA, 1993). Surveys have been made in Class I areas in New Hampshire and Vermont during the years 1988 to 1990 (Manning et al., 1991). Ozone injury was extensive on vegetation growing in open-top and ambient air experimental plots in both states in 1988 when O_3 was unusually high. The incidence and intensity of O_3 injury symptoms was considerably less in both 1989 and 1990, though some degree of symptoms were evident on all plants. Based on the studies, it was determined that black cherry, milkweed, white ash, white pine and two species of blackberry were all reliable biological indicators of ambient O_3 exposure (Manning et al., 1991).

Concurrently, a regional initiative, the Forest Health Monitoring Program (FHM), which began in 1990 as a cooperative program between the USDA, EPA, and EMAP, monitors the condition of forests in the Northeastern United States. In 1992, bioindicator evaluation was conducted on 39 of 222 forested plots. Sensitive plant species in the Northeast include blackberry, milkweed, black cherry, white ash, and white pine. Of the 39

plots, 28% included plants which showed some visible symptoms of O_3 injury (U.S.D.A., 1993, Summary report).

2. Growth/Yield Reductions in Annual Crops

As was discussed in section VII.B, O_3 can interfere with carbon gain (photosynthesis) and allocation of carbon with or without the presence of visible foliar injury. As a result, the carbohydrates that remain may be allocated to sites of injured tissue or employed in other repair or compensatory processes, thus reducing plant growth and/or yield. Growth reductions indicate that plant vigor is being compromised which can lead to yield reductions in commercial crops.

Agricultural Crop Studies. Annuals tend to be fast growing, have no need for long-term storage of carbohydrates, and, in the case of well-fertilized crops, have less need for extensive root development. Instead, most resources go toward producing seeds for the following year, making fruit or seed production the most significant of the processes sensitive to a reduction in carbohydrate production occurring as a result of O_3 exposure. Changes in susceptibility to insect damage is likely to be of greater concern than for perennials which may have the chance to recover the following year.

The largest body of research to date on the growth and yield effects of O_3 on annuals is that for agricultural crop species. The majority of this research occurred before 1986, and includes the National Crop Loss Assessment Network (NCLAN) studies which remain the largest, most uniform database for crops available today. The NCLAN project which began in 1980 was originally undertaken to quantify the relationships between O_3 exposure and yields of major agricultural crops. The project was intended to provide data necessary for a credible evaluation of the economic effects of ambient O_3 on U.S. agriculture, and for input into the review of the O_3 NAAQS (Preston and Tingey, 1988).

The NCLAN protocol was designed to produce crop exposure-response data representative of the areas in which the crops were typically grown. The United States was divided into 5 regions over which a network of field sites was established. In total, 15 species (corn, soybean, wheat, hay (alfalfa, clover, and fescue), tobacco, sorghum, cotton, barley, peanuts, dry beans, potato, lettuce, and turnip), were studied. The first 13 of these 15 listed species, accounted for greater than 85% of US agricultural acreage planted (Preston

and Tingey, 1988). These 13 species, which included 38 different cultivars, were studied under a variety of unique combinations of sites, water regimes, and exposure conditions, producing a total of 54 separate cases. These studies were a tremendous improvement over earlier studies because crops were grown 1) using typical farm practices and 2) using open-top chambers, which produce the least amount of environmental modification of any outdoor chamber (CD, 1996). Another major advantage of the NCLAN approach is that it used a wide range of exposure levels (e.g., charcoal filtered, ambient and modified ambient), allowing for the use of regression analyses to develop exposure-response functions, which could be used to predict yield loss as a function of O_3 exposure levels across the range of treatment levels, cultivars, and growing conditions used in the studies.

Some plant scientists continue to express concern that in the case of NCLAN, experiments using OTC's were designed and conducted in a way that results in overestimation of O_3 impacts. For example, the modified ambient treatments contained numerous high peaks (O_3 concentrations equal to or above 0.10 ppm), occurring more frequently than in typical ambient air quality distributions. Such exposure patterns have raised questions among some researchers as to how much of the plant's response was a result of having an excessive number of high peaks versus a cumulation of more moderate exposures. Exposure durations were species dependent but typically went from stand establishment to harvest (on average 78 days). Some crops were grown in more than one geographical region and repeated over years. In addition, the charcoal filtered chambers used to establish baseline crop yield loss were exposed to approximately 0.025 ppm O_3, which is lower than the range of 0.03 to 0.05 ppm identified in chapter 4 of the staff paper as the value for seasonal background O_3 levels. The result of using this lower level of 0.025 ppm is an overestimation of yield loss relative to that expected using 0.03 to 0.05 ppm. These aspects of the NCLAN protocols contribute to the various types of uncertainty inherent in extrapolating controlled field study results of percentage yield reductions to non-chambered ambient field conditions and crop regions having different O_3 air quality distributions.

Based on regression of the NCLAN analyses, at least 50% of the species/cultivars tested were predicted to exhibit a 10% yield loss at a 7-hour seasonal mean O_3 concentration

of 0.05 ppm or more (CD, 1996). These findings produced by the NCLAN project have also been reported in terms of various cumulative exposure indices, such as the 3-month, 12 hour SUM06 and W126, and are shown in Table VII-2 (derived from Tables 5-21 and 5-22 in the CD). Review of the NCLAN data indicates that differences in O_3 sensitivity within species may be as great as differences between species with substantial variation in sensitivity from year to year. Figure VII-1a and b show how many of the 54 NCLAN cases experience a 10 or 30% yield loss, respectively, for each 10 ppm-hr change in O_3 exposure level, expressed in terms of the 12 h, 3 month W126 index. In Figure VII-1a, 40% of the cases will experience 10% yield loss at 40 ppm-hrs. In contrast, only half that amount, 20%, will experience 30% yield loss at 40 ppm-hrs. This suggests that the variability in sensitivity increases as O_3 exposures increase. Additionally, Figure VII-2 (taken from Figure 5-29 in the CD) shows that as 24 hour SUM06 levels increase the range of variability in relative yield loss between the 50th and 75th percentiles among NCLAN cases increase, from a 2 % difference in yield loss at 10 ppm-hr to a 27 % difference at 60 ppm-hrs, thus showing a disproportional increase in impact on sensitive species as O_3 exposure levels increase.

In a re-analysis of NCLAN data, Lesser et al., (1990) predicted relative yield losses for a number of crops species or groups of species (compared to an assumed background concentration of 0.025 ppm) of 10 to 20% at 12 h seasonal means of 0.045 to 0.06 ppm, respectively. Most significantly, based on the NCLAN results, it can be seen that several economically important crop species are sensitive to O_3 levels typical of those found in the U.S.

Other studies (on beans, potatoes, tomatoes, onion, and tobacco) examined effects of O_3 under ambient conditions by using the chemical protectant, ethylene diurea (EDU). Though there has been some concern that EDU might itself alter plant growth, it is generally considered an objective method for evaluating O_3 effects (U.S. EPA, 1986). Estimates of yield loss were provided by comparing yield from plants grown in ambient air that were protected with EDU to those that were not. Studies indicated that yields were reduced by 18 to 41% relative to the chemically protected controls when ambient O_3 concentrations exceeded 0.08 ppm during the day for 5-18 days over the growing season (U.S. EPA, 1978).

Table VII-2. COMPARISON OF EXPOSURE-RESPONSE VALUES CALCULATED USING THE 3-MONTH, 12-HOUR SUM06 AND W126 EXPOSURE INDICES FOR 54 NCLAN CASES

Species	Cultivar	Moisture[a]	3 mo 12-h SUM06[c] Values for Yield Losses of 10%	3 mo 12-h SUM06[c] Values for Yield Losses of 30%	3 mo 12-h W126[d] Values for Yield Losses of 10%	3 mo 12-h W126[d] Values for Yield Losses of 30%
Barley (Linear) [b]	CM-72	DRY	173.1	250.0	117.2	250.0
Barley (Linear)	CM-72	WET	250.0	250.0	1382.7	3329.4
Corn (L) [b]	PIO		41.6	64.1	38.6	62.2
Corn (L)	PAG		55.8	74.1	55.0	73.6
Cotton (L)	ACALA	DRY	35.7	59.8	28.3	53.0
Cotton (L)	ACALA	WET	23.1	42.5	16.4	35.0
Cotton (L)	ACALA	DRY	24.8	48.0	18.8	41.6
Cotton (L)	ACALA	WET	14.0	35.5	9.1	28.4
Cotton (L, Linear)	ACALA	DRY	63.2	103.5	61.6	107.7
Cotton (L, Linear)	ACALA	WET	60.0	203.2	62.0	210.0
Cotton	STONEVILLE		30.9	56.6	22.2	48.4
Cotton	MCNAIR	DRY	73.4	114.0	68.5	113.4
Cotton	MCNAIR	WET	26.6	59.3	22.6	54.2
Kidney Bean	CAL LT RED		15.2	20.9	15.0	20.9
Kidney Bean (L)	CAL LT RED		17.7	28.6	15.6	27.1
Lettuce (T) [b]	EMPIRE		36.5	45.5	34.6	44.3
Peanut (L)	NC-6		36.2	62.7	29.4	56.1
Potato	NORCHIP		9.9	33.5	10.1	34.3
Potato	NORCHIP		20.3	42.5	20.1	51.5
Sorghum	DEKALB		67.6	114.2	65.0	121.2
Soybean	CORSOY		15.3	31.2	12.2	28.5
Soybean	CORSOY		42.0	53.0	35.5	48.2

Table VII-2. (Cont'd.). COMPARISON OF EXPOSURE-RESPONSE VALUES CALCULATED USING THE 3-MONTH, 12-HOUR SUM06 AND W126 EXPOSURE INDICES FOR 54 NCLAN CASES

Species	Cultivar	Moisture[a]	3 mo 12-h SUM06[c] Values for Yield Losses of 10%	3 mo 12-h SUM06[c] Values for Yield Losses of 30%	3 mo 12-h W126[d] Values for Yield Losses of 10%	3 mo 12-h W126[d] Values for Yield Losses of 30%
Soybean	AMSOY		32.8	51.4	24.6	46.1
Soybean	PELLA		18.2	61.6	16.9	57.2
Soybean	WILLIAMS		15.5	52.4	14.5	49.2
Soybean	CORSOY	DRY	71.2	81.2	66.8	76.7
Soybean	CORSOY	WET	70.0	154.0	63.1	188.7
Soybean	CORSOY	DRY	89.1	96.4	91.5	101.7
Soybean	CORSOY	WET	62.2	87.6	56.7	86.5
Soybean	CORSOY	DRY	10.2	34.7	10.0	34.0
Soybean	CORSOY	WET	11.8	29.9	8.9	26.3
Soybean	WILLIAMS	DRY	21.1	48.8	17.2	46.1
Soybean	WILLIAMS	WET	14.8	36.6	11.4	33.0
Soybean	HODGSON		8.4	28.4	8.0	27.0
Soybean	DAVIS		13.8	46.9	13.7	46.4
Soybean	DAVIS		23.4	47.2	19.2	43.6
Soybean	DAVIS	DRY	57.1	193.4	56.9	192.6
Soybean	DAVIS	WET	35.2	79.6	26.8	73.5
Soybean	DAVIS	DRY	45.9	66.1	40.2	62.3
Soybean	DAVIS	WET	24.1	55.7	18.9	50.8
Soybean	YOUNG	DRY	38.8	90.1	32.8	87.4
Soybean	YOUNG	WET	25.0	64.9	20.4	60.0
Tobacco (L)	MCNAIR		24.4	70.3	18.9	63.9
Turnip (T)	JUST RIGHT		7.4	14.5	5.2	12.0
Turnip (T)	PURPLE TOP		5.9	13.4	4.0	11.6
Turnip (T)	SHOGOIN		6.6	14.0	4.1	11.7

Table VII-2. (Cont'd.). COMPARISON OF EXPOSURE-RESPONSE VALUES CALCULATED USING THE 3-MONTH, 12-HOUR SUM06 AND W126 EXPOSURE INDICES FOR 54 NCLAN CASES

Species	Cultivar	Moisture[a]	3 mo 12-h SUM06[c] Values for Yield Losses of 10%	3 mo 12-h SUM06[c] Values for Yield Losses of 30%	3 mo 12-h W126[d] Values for Yield Losses of 10%	3 mo 12-h W126[d] Values for Yield Losses of 30%
Turnip (T)	TOKYO CROSS		9.3	15.9	7.2	14.7
Wheat	ABE		25.1	37.5	22.1	35.4
Wheat	ARTHUR		21.3	37.5	17.3	35.0
Wheat	ROLAND		7.4	21.3	5.4	18.1
Wheat	ABE		34.8	40.9	32.3	40.0
Wheat	ARTHUR		27.7	46.5	25.4	46.2
Wheat	VONA		2.9	9.7	2.6	8.8
Wheat	VONA		7.7	16.5	6.0	14.6

[a]Wet refers to experiments conducted under well-watered conditions while dry refers to experiment conducted under some controlled level of drought stress.

[b]For those studies whose species name is followed by "(Linear)" a linear model was fit. A Weibull model was fit to all other studies. For those studies whose species name is followed by "(L)" a log transformation was used to stabilize the variance. For those crops whose name is followed by "(T)" the yield is expressed as either g/plant or g/m.

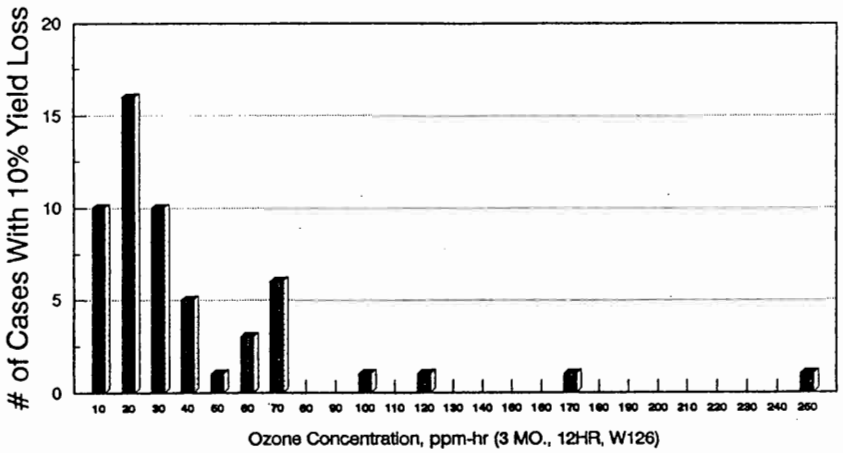
[c]The 12-h SUM06 value (ppm-h) that was predicted to cause a 10 or 30% yield loss (compared to zero SUM06).

[d]The 12-h W126 value (ppm-h) that was predicted to cause a 10 or 30% yield loss (compared to zero W126),

Modified from CD Tables 5-21 and 5-22.

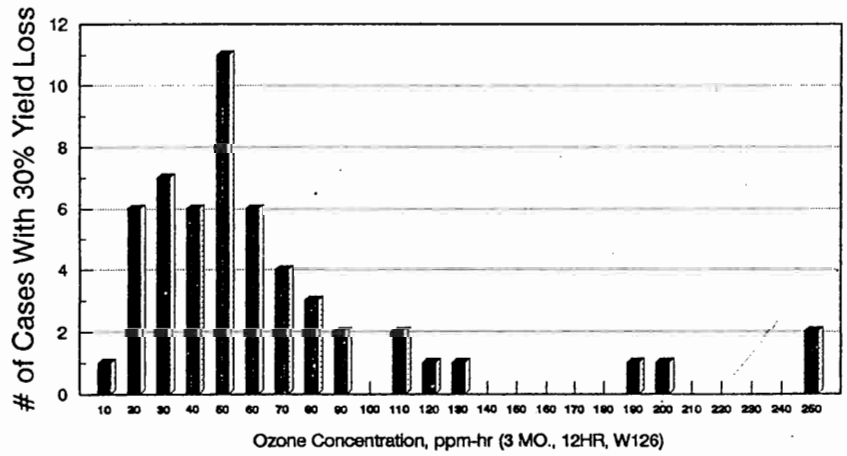
FIGURE VII-1. Variability in NCLAN Crop Yield Sensitivities

A.



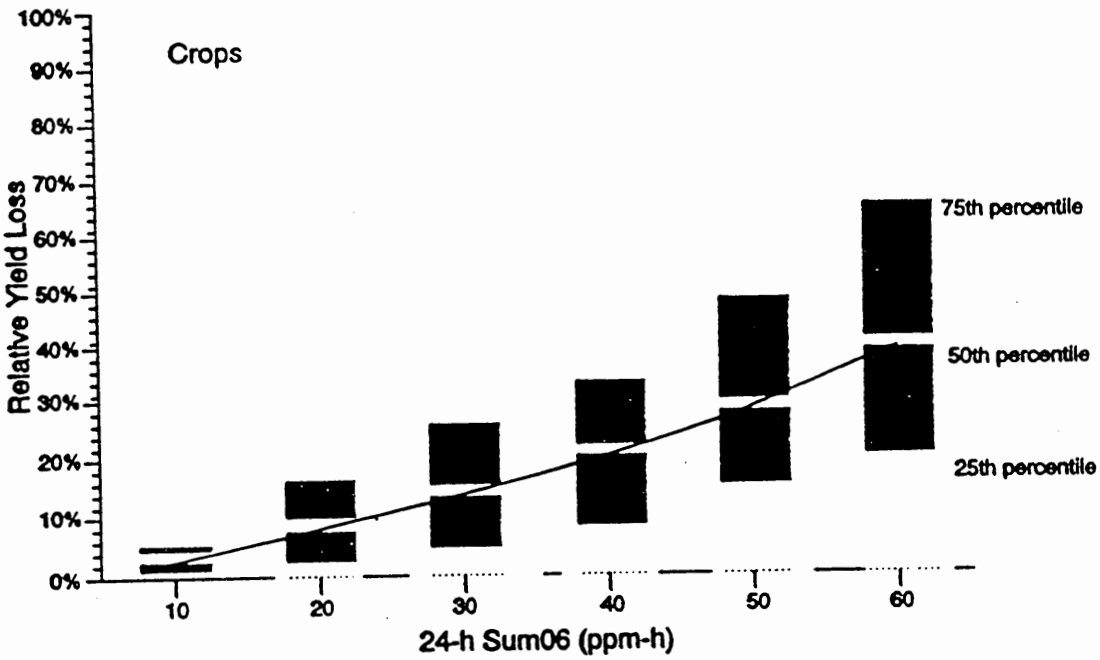
Graph Shows Different Ozone Levels Required to Produce 10% Yield Loss in 54 Different Cases

B.



Graph Shows Different Ozone Levels Required to Produce 30% Yield Loss in 54 Different Cases

Figure VII-2. Median Crop Yield Loss from NCLAN Crops



Box-plot distribution of yield loss predictions from Weibull and linear exposure-response models that relate biomass and ozone exposure as characterized by the 24-h SUM06 statistic using data from 31 crop studies from the National Crop Loss Assessment Network (NCLAN) program. Separate regressions were calculated for studies with multiple harvests and/or cultivars resulting in a total of 54 individuals equations from the 31 NCLAN studies. Each equation was used to calculate the predicted relative yield or biomass loss at 10, 20, 30, 40, 50 and 60 ppm·h and the distributions of the resulting losses plotted. The solid line is the calculated Weibull fit at the 50th percentile. From Hogsett et al. (1995).

Taken together, the studies discussed above as well as others (e.g., Heagle et al., (1988b), Miller et al., (1988), and Temple et al., (1988) continue to provide a basis for the conclusion presented in the last two Criteria Documents that O_3 concentrations at ambient levels in the U.S. are sufficiently elevated in several parts of the country to impair the growth and yield of commercial crops.

Caveats and Uncertainties. In order to isolate and measure a plant's response to O_3 from the plant's response to other environmental variables, many study designs employ some type of exposure chamber. A chamber allows the researcher to create a variety of O_3 regimes while all other variables are kept constant or their conditions well-characterized. Though there are numerous fumigation systems, the most widely utilized design has been the open-top chamber (OTC). This review of the standard relies heavily on agricultural crop exposure-response functions developed in open top chambers during the National Crop Loss Assessment Network (NCLAN) from 1980 to 1988. Other types of exposure methods are also discussed below. For a more detailed discussion of methods and uncertainties, see the discussion in the 1996 CD.

The main advantage of the OTC is that it provides the least amount of environmental modification of any outdoor chamber. However, the open-top chamber does alter ambient microclimate conditions such as light intensity, wind velocity, rainfall, dew formation and persistence, air temperature and relative humidity (CD, 1996). As a result, exhaustive comparisons between plants grown in carbon filtered chambers (CF), non-filtered chambers (NF), and similarly sized and located ambient air plots (AA) have been conducted. These comparisons demonstrate that the only consistently observed effect is that chamber plants were taller than those grown in the ambient air (Heagle et al., 1988a). Though plant yield in ambient air can be greater than, less than or equal to that in NF chambers, there is no evidence that there is a large effect of chambers on plant response to O_3 (Heck et al., 1994; Heagle et al., 1988a; Olszyk et al., 1992).

An additional concern in open-top chambers is the addition of trace pollutants (N_2O_5 and NO) in chambers receiving O_3 generated from dry air and NO_2 in chambers receiving ambient air. Generated O_3 treatments have been shown to be more phytotoxic than ambient O_3 treatments. To avoid this problem, OTC studies have used filtered vs. non-filtered ambient O_3. The drawback to this approach is that low ambient O_3 levels make detecting

differences in plant response between filtered and non-filtered chambers difficult, thus requiring a high number of replications for statistical reasons.

Several other exposure methodologies have been employed in more natural environmental conditions. One method involves using chemicals, specifically EDU, to protect plants from the effects of O_3 in the field. This technique is cheaper and easier to apply to large areas than open top chambers, eliminates the uncertainties associated with chamber effects, and reduces uncertainties associated with scaling up from small to larger areas.

However, field protocols for the use of EDU have not been well established. Frequency and rate of application that protects plants vary with species and edaphic and atmospheric conditions. Two-treatment studies of EDU and plant response to O_3 (Kostka-Rick and Manning, 1992a,b) indicate that protection is variable, suggesting that the experimental system under investigation (soil, plant, climate) would have to be extremely well characterized and understood for interpretation of results. The mechanism by which EDU protects plants, beyond being a systematic antioxidant, is unknown; understanding this mechanism has the potential to contribute to the broader understanding of the mechanisms of O_3 injury at the cellular/metabolic level of the plant.

A third method uses open air field fumigation systems, such as the zonal air pollution system. Such systems have the capability to fumigate plants with diurnally varying patterns of concentrations typical of ambient O_3 under field conditions. However, studies which use such systems are the least controllable and repeatable. Another method is the ambient gradient system. This method is structured to take into account the preexisting gradient of pollutant concentrations over a given area where a species is grown. For ambient gradient studies to be interpretable, good characterization of site parameters (rainfall, temperature, radiation, soil type, etc.) is needed, as well as knowledge of how these factors should be used to adjust apparent plant response. At this time, however, the relationships are still incompletely understood, and therefore confound interpretation of the results.

3. Growth Reductions in Tree Seedings and Mature Trees

Since the preparation of the 1986 CD, a number of new studies have been published relating O_3 exposure to effects on deciduous and evergreen seedlings and mature trees.

These studies partially address a significant gap in O_3 effects data identified by CASAC in the last review of the O_3 NAAQS.

The relationship between responses to O_3 exposure in seedlings and mature trees is still not well understood. Several studies describe a number of potential differences between seedlings and mature trees (Cregg et al., 1989; Pye, 1988) including stomata number, photosynthetic rate, water use efficiency, nutritional needs, recycling capacities, and canopy effects (e.g., sun vs. shade, wind speed, CO_2 concentrations). Limited experimental evidence shows no consistent relationship between the sensitivities of seedlings and mature trees. For example, Samuelson and Edwards (1993) found that while the canopy weight of a mature 30 year old northern red oak experienced a 41% reduction when exposed to a 7 h seasonal mean of 0.069 ppm as compared to a very low exposure level of 0.015 7 hr seasonal mean, two year old seedlings were not affected at similar exposures. Thus, because tree seedling studies can not at this time be extrapolated to quantify responses to O_3 in mature trees, they will be discussed separately below.

Deciduous And Evergreen Seedlings. Growth and productivity has been reported to be affected by O_3 for numerous species in the Blue Ridge Mountains of Virginia. In the Shenandoah National Park, Duchelle et al. (1982, 1983) compared the growth of seedlings and productivity of herbaceous vegetation grown in charcoal-filtered air in open-top chambers to that in open plots, and found that tulip poplar, green ash, sweet gum, black locust, as well as several evergreen species (e.g., Eastern hemlock, Table Mountain pine, pitch pine, and Virginia pine), common milkweed, and common blackberry all demonstrated growth suppression. Except for the last two species mentioned, almost no visible injury symptoms accompanied the growth reductions.

Between 1989 and 1992, the EPA's National Health and Environmental Effects Research Laboratory-Western Ecology Division (NHEERL-WED) in Corvallis sponsored a research program to address the effects of tropospheric O_3 on forest trees. Using the same open top chamber methodology as NCLAN, this program has developed exposure-response functions for six deciduous species, including aspen, red alder, black cherry, red maple, sugar maple, and tulip poplar and five evergreen species, including douglas fir, ponderosa pine, loblolly pine, eastern white pine, and Virginia pine. Table VII-3 presents the individual results for all cases evaluated in this study (Table 5-28 from the CD).

Table VII-3. Exposure-Response Values that Relate Total Biomass (Foliage, Stem, and Root) to 12-H SUM06 Exposures(*) Adjusted to 92 Days (ppm-h/year)

Rate of Growth	Habit	Study	Species	SUM06 for Loss of 10%	SUM06 for Loss of 30%
FAST	D	1	Aspen - wild	19.08	43.14
FAST	D	1	Aspen - wild	15.83	53.60
FAST	D	2	Aspen - wild	43.72	63.67
FAST	D	2	Apsen - wild	55.90	70.81
FAST	D	3	Apsen - wild	55.44	66.49
FAST	D	3	Aspen - wild	18.66	45.76
FAST	D	4	Aspen 216	14.70	37.78
FAST	D	4	Aspen 253	8.09	27.38
FAST	D	4	Aspen 259	4.69	15.87
FAST	D	4	Aspen 271	13.28	24.58
FAST	D	5	Aspen 216	9.52	32.21
FAST	D	5	Aspen 259	5.18	17.56
FAST	D	5	Aspen 271	29.64	37.71
FAST	D	6	Aspen - Wild	14.99	50.73
SLOW	E	7	Douglas Fir	89.31	160.46
SLOW	E	7	Douglas Fir	250.00	250.00
SLOW	E	7	Douglas Fir	90.84	109.85
SLOW	E	7	Douglas Fir	94.44	250.00
SLOW	E	8	Douglas Fir	72.03	73.89
SLOW	E	8	Douglas Fir	70.82	70.94
SLOW	E	8	Douglas Fir	63.03	69.23
SLOW	E	9	Ponderosa Pine	17.86	60.45
SLOW	E	9	Ponderosa Pine	26.30	89.03
SLOW	E	10	Ponderosa Pine	18.53	55.96
SLOW	E	10	Ponderosa Pine	27.09	85.87
SLOW	E	10	Ponderosa Pine	11.29	38.23
SLOW	E	10	Ponderosa Pine	21.64	73.25
SLOW	E	11	Ponderosa Pine	19.47	64.72
SLOW	E	11	Ponderosa Pine	14.86	50.29
SLOW	E	11	Ponderosa Pine	27.85	69.15
SLOW	E	12	Ponderosa Pine	55.18	86.23
SLOW	E	13	Ponderosa Pine	43.42	146.93
FAST	D	14	Red Alder	32.05	68.80
FAST	D	15	Red Alder	17.87	60.51
FAST	D	15	Red Alder	79.04	95.77
FAST	D	16	Red Alder	35.84	121.31

Table VII-3. (cont.) Exposure-Response Values that Relate Total Biomass (Foliage, Stem, and Root) to 12-H SUM06 Exposures(*) Adjusted to 92 Days (ppm-h/year)

Rate of Growth	Habit	Study	Species	SUM06 for Loss of 10%	SUM06 for Loss of 30%
FAST	D	16	Red Alder	250.00	250.00
FAST	D	17	Red Alder	21.81	67.19
FAST	D	18	Black Cherry	6.59	20.31
FAST	D	19	Black Cherry	4.37	12.60
SLOW	D	20	Red Maple	71.74	147.00
FAST	D	21	Tulip Poplar	23.44	28.69
FAST	D	21	Tulip Poplar	19.85	67.19
FAST	D	22	Tulip Poplar	14.66	25.25
FAST	E	23	Loblolly GAKR 15-91	71.03	240.44
FAST	E	23	Loblolly GAKR 15-23	212.08	250.00
SLOW	D	24	Sugar Maple	25.29	30.26
SLOW	D	24	Sugar Maple	23.81	29.14
SLOW	E	25	E. White Pine	21.60	28.28
SLOW	E	25	E. White Pine	31.51	106.68
SLOW	E	26	Virginia Pine	191.24	250.00

Source: Hogsett et al. (1995).

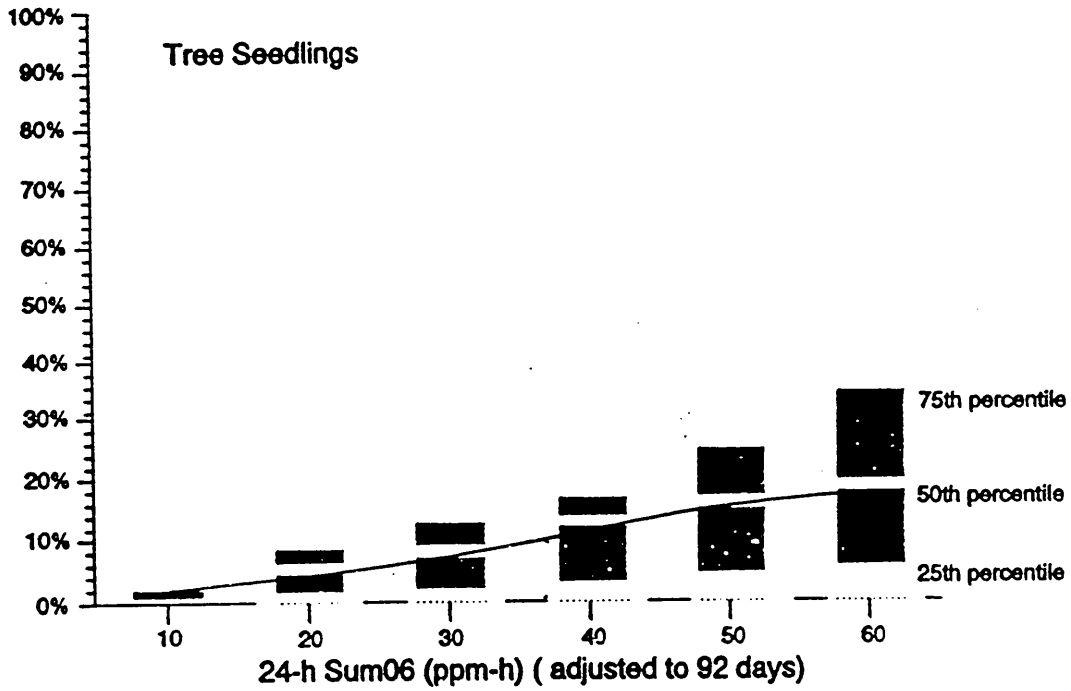
*Note: Seeding studies were not all of equal duration. To compare the results from seedling studies of varying exposure duration, the SUM06 value is calculated for an exposure of a fixed period of 92 days per year. The calculation assumes that exposures can be scaled up or down in uniform fashion.

When the distribution of the relative yield losses for various percentiles of the deciduous and evergreen seedling studies are aggregated, (Figure VII-3), a 24 hr. SUM06 exposure of 33.3 ppm-h over 92 days is associated with less than 10% biomass reduction in 50% of the seedling cases studied. When the exposure-response functions for just the deciduous seedling cases are combined, the results show that a lower 24 hr. SUM06 exposure of 31.5 ppm-h over 92 days is associated with less than 10% biomass reduction in 50% of the deciduous cases (Table 5-29, CD). For the evergreen seedlings, a 3 month, 24 hr. SUM06 exposure of 42.6 ppm-h was predicted to result in less than a 10% biomass loss in 50% of the evergreen cases studied. Thus, the evergreen seedlings studied, on average, exhibited less sensitivity to O_3 than the deciduous seedlings studied.

As with crops, these studies also showed that there was significant variability in sensitivity to O_3 among species and genotypes within species. For example, red alder seedlings showed substantial variability, with a 10% reduction in biomass observed over the range of 21.7 to 95.8 ppm-h (24 h SUM06), and a 30% biomass loss observed over the range of 73.5 to 250.0 ppm-h (24 h SUM06). This variation in growth response to O_3 exposure can also result from different climates and growing environments (e.g., drought, nutrient level), pest/pathogen interactions, exposure intensity and dynamics, and genetics (Hogsett et al., 1995). Because it is not known whether the genome(s) that were studied represent the complete range of sensitivities within a given species, the results from these studies should be used with caution.

Some studies further showed that several deciduous species as seedlings are extremely sensitive to O_3 with respect to biomass loss. For example, Davis and Skelly (1992a, b) and Simimi et al., (1992) describe black cherry seedlings as very sensitive, with 24 hour SUM06 exposures as low as 12.9 ppm-h over 92 days predicted to cause a 10% biomass loss (Hogsett et al., 1995). Two different Aspen clones showed 10% biomass loss at 24 hour SUM06 exposures of 10.96 and 9.49 ppm-h, respectively. Given the mean 3 month 24 hour SUM06 value over the 10 year period 1982-1991 of 29.5 ppm-h (from Table VII-1), the potential for biomass loss in such sensitive seedling species could be significant.

Figure VII-3. Median Biomass Loss From Seedlings



Box-plot distribution of biomass loss predictions from Weibull and linear exposure-response models that relate biomass and ozone exposure as characterized by the 24-h SUM06 statistic using data from 26 tree seedling studies conducted at the Environmental Research Laboratory in Corvallis, Oregon; Smoky Mountain National Park, Tennessee; Michigan; Ohio; and Alabama. Separate regressions were calculated for studies with multiple harvests and/or cultivars resulting in a total of 56 equations from the 26 seedling studies. Each equation was used to calculate the predicted relative yield or biomass loss at 10, 20, 30, 40, 50 and 60 ppm·h and the distributions of the resulting losses plotted. The solid line is the calculated Weibull fit at the 50th percentile. From Hogsett et al. (1995).

Other studies have been performed on Douglas fir, Jeffrey, Lodgepole, Monterey, ponderosa, shore, sugar, and western white pine and Sitka spruce by Wilhour and Neely (1977). In closed-top field chambers, O_3 was added to charcoal-filtered air at the constant exposure of 0.10 ppm for 6 h/day for 126 days. Across species, observed reductions ranged from 0 to 11% for height and 0 to 21% for stem dry weight. In another study, hybrid poplar was exposed to 0.15 ppm for 12 hrs/day for 102 days in open-top chambers, with observed reductions ranging from 3% to 58% for height and 1 to 14% for stem specific gravity (Patton, 1981). Hogsett et al. (1985a) noted growth reductions in height, diameter, and root systems in two varieties of slash pine seedlings under chronic episodic exposure regimes typical of the southeastern U.S. Both varieties of slash pine exhibited an increasing reduction in growth with increasing O3 concentration, with the most pronounced change observed in the growth of roots. The significance of these findings is not yet understood. Because trees are perennials, the effect of even a 1-2% per year loss in seedling biomass (versus 10 to 20% yield loss in crops), if compounded over multiple years under natural field conditions of competition for resources, could be severe. Furthermore, given the variability in meteorology and O_3 concentrations between years, it is not known to what degree seedlings would recover given periods of more favorable conditions. Because of these uncertainties, the staff caution against treating equal percentages of yield loss in annuals and biomass loss in perennials as representing the same degree of adversity.

Uncertainties and Limitations In Seedling Studies. In order to more accurately understand the results of the seedling studies presented above, there are several important caveats and limitations to keep in mind. The 11 species selected were grown in open top chambers (see discussion of caveats and uncertainties for OTC above). The influence of multiple environmental factors were not taken into account as the seedlings were grown under optimal growing conditions and few experiments included multiple year exposures. These facts make it problematic when trying to predict effects on perennial species growing in an ecosystem context (Hogsett, et al., 1995). The parameter used to measure O_3 effects on seedlings, total biomass loss, is measured against biomass at an O_3 concentration level of SUM06 equal to 0 ppm-hrs. However, the database shows that there is no distinguishable threshold between concentrations that produce an effect and those that do not, and biomass

loss occurring at exposure levels below 0.06 ppm may be significant for some sensitive species. Thus, the data are limited to the conditions under which the experiments were conducted.

Deciduous and Evergreen Trees. Many important field observations of mature evergreen trees occurred prior to 1986, and were discussed in the 1986 CD. One such study by Mann et al. (1980) reported a reduction in radial growth of sensitive white pine individuals of as much as 30 - 50% annually over a period of 15 to 20 years on the Cumberland Plateau in Tennessee. Field studies in the San Bernardino National Forest indicated that over a period of 30 years, O_3 may have reduced the growth in height of ponderosa pine by as much as 25%, radial growth by 37%, and total volume of wood produced by 84% (Miller et al., 1982). Because these observations were made in the field with numerous uncontrolled environmental factors, the extent to which the observed growth effects can be attributed to O_3 is uncertain. It is reported, however, that O_3 was a significant contributor that potentially exacerbated the effects of the other environmental stresses.

Several field studies indicate that injury associated with exposure to O_3 and other oxidants has been occurring in the Appalachian Mountains for many years. Benoit et al. (1982) conducted studies in the Blue Ridge Mountains of Virginia to evaluate the long term effects of oxidants on growth in eastern white pine of reproducing age. By comparing growth rates from the period 1955-1959 with those in 1974-1978, decreases of 26, 37, and 51% were reported for tree species characterized as tolerant, intermediate, and sensitive, respectively. Because no significant change in seasonal precipitation occurred over the same time period, the effects on growth were attributed to O_3, which during the latter time period reached peaks frequently in excess of 0.12 ppm and monthly averages of 0.05-0.07 ppm on a recurring basis (U.S. EPA, 1986). Duchelle et al. (1982), monitoring in the same area, reported peak hourly averages >0.08 ppm for the months of April through September in 1979 and 1980. As early as 1979, Skelly et al. (1984) concluded that the most sensitive eastern white pines were so severely injured by oxidant exposure that they were probably being removed from the population.

In 1985, to evaluate growth changes in O_3-stressed ponderosa and Jeffery pine, Peterson and his coworkers conducted the largest investigation of regional tree growth in the

western U.S. (Peterson et al., 1987; Peterson and Arbaugh, 1988, 1992; Peterson et al., 1991). Using cores to determine whether growth reductions had occurred, they randomly sampled both trees with visible O_3 injury symptoms and asymptomatic trees. Major decreases in growth occurred for both symptomatic and asymptomatic trees during the 1950's and 1960's. The percentage of trees exhibiting growth decreases at any given site never exceeded 25% in a given decade (Peterson et al., 1991). Mean annual radial increment in trees with visible symptoms of O_3 injury was 11% less than trees at sites without O_3 injury. Trees larger than 40 cm diameter and trees older than 100 years showed greater decreases in growth than smaller and younger trees. Again, the significance of these effects on the above and below ground forest ecosystem is unknown.

The response of a number of fruit and nut trees to O_3 exposure has been reported (McCool and Musselman, 1990; Retzlaff et al., 1991, 1992a, b). Almond has been identified as the most sensitive, but peach, apricot, pear, and plum have also been affected under study conditions. Net growth of almond, as well as stem diameter of peach and the stem diameter and number of shoots produced on apricot were reduced by four months of once-weekly exposure to 0.25 ppm-h O_3 for 4 h (a high level of exposure generally found with fruit and nut trees only in California). A few studies have measured O_3 effects on citrus or avocado. Valencia orange trees (during a production year) exposed to a seasonal 12 h mean of 0.04 and 0.075 ppm had 11 and 31% lower yields than trees grown in filtered air at with a very low O_3 concentration of 0.012 ppm. In contrast, growth of Ruby Red grapefruit was not affected by concentrations 3 times ambient (CD, 1996). Avocado growth was reduced by 20 or 60% by exposure to 12 h seasonal means of 0.068 and 0.096 ppm during two growing seasons.

The methodologies employed in mature tree studies often differ from those used with seedlings. In only a few cases have OTCs large enough for mature trees been used, which were built at great expense (Mandl et al., 1989). Other exposure methods that have been used with large trees include branch and leaf chambers. Though these chambers have many advantages, it is not yet known whether the branch or leaf being studied is responding the same as other parts of the plant which are experiencing different environmental conditions.

Thus, estimating total tree response from branch or leaf chamber studies introduces a high degree of uncertainty.

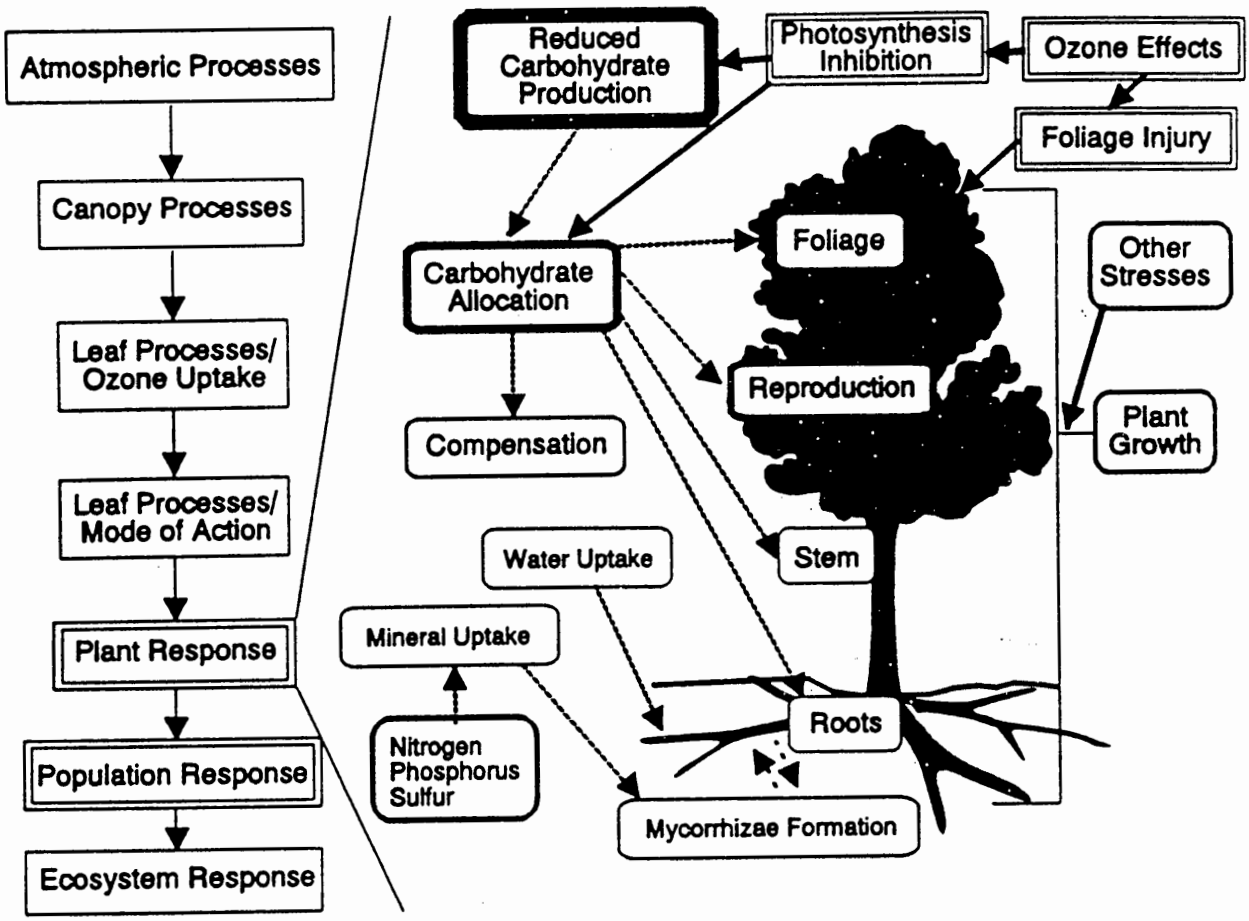
In light of the greater difficulties associated with conducting studies on mature trees, the scientific community has developed process-level models to simulate growth and stand dynamics over time under various O_3 levels. These models "utilize the large base of data in tree physiology and forest ecology, watershed chemistry, and atmosphere-forest canopy meteorology to develop models of tree physiology and growth and to subsequently scale these investigations to the levels of forest stands and landscapes" (Taylor, et al., 1994). TREGRO models whole tree or seedling growth and simulates multiyear O_3 effects to either seedlings or mature trees under selected climates or soil conditions. ZELIG considers the competition for resources that occurs between four individuals of the same or different species in a stand. Such modeling studies are expected to lead to a better understanding of O_3 effects on mature trees and forests in the future.

4. Forest and Ecosystems Effects

Plant populations can be affected by O_3 exposure particularly when they contain many sensitive individuals. Changes within sensitive populations, or stands, if they are severe enough, ultimately can change community and ecosystem structure. This progression of effects is depicted in Figure VII-4 (Figure 5-30 from the CD). Structural changes that alter the ecosystem functions of energy flow and nutrient cycling can arrest or reverse ecosystem development.

The only known example of the above sequence of events occurring in which O_3 has been a fundamental stressor, is the San Bernardino Forest ecosystem. This ecosystem has experienced chronic O_3 exposures over a period of 50 or more years. From 1968 to 1972 the average daily maximum for total oxidants for each month was measured at Rim Forest (5,640 ft.), in southern California, where the highest concentrations are usually recorded. For the months of May through August, the average daily maximum for total oxidants went from a low of 0.14 ppm in 1969 to approximately 0.28 ppm in 1971, with concentrations rarely going below 0.05 ppm at night at this elevation. For the same period the total number of hours/month exceeding 0.10 ppm varied from around 4 to 15. Ozone concentrations exhibited a cyclic diurnal pattern, with the monthly average of hourly values ranging from

Figure VII-4. Diagram of the Propagation Pathway of Ozone Effects From Plants to Ecosystems



Effects of ozone on plant function and growth. Reduced carbohydrate production decreases allocation and resources needed for plant growth processes. Individual plant responses must be propagated hierarchically through the more integrative levels of population and community to produce an ecosystem response. Solid black arrows indicate the effects of ozone absorption; stippled arrows indicate affects on plant functions. Double border indicates site of response; darkened border indicates site of impact.

0.07 to 0.10 ppm at 10:00 am and from 0.15 to 0.22 ppm at 4:00 pm. The primary effect of O_3 at these high levels was on the more susceptible members of the forest community, individuals of ponderosa and Jeffrey pine, that could no longer compete effectively for essential nutrients, water, light and space. As a consequence, there was a decline in the sensitive species, permitting the enhanced growth of more tolerant species (Miller et al., 1982; US EPA, 1978, 1986).

Follow-up studies of the San Bernardino forest ecosystem done from 1973 to 1978 reported that the major changes in the ecosystem began with injury to ponderosa and Jeffrey pine. Foliar injury, premature senescence, and needle fall decreased the photosynthetic capacity of stressed pines and reduced the production of carbohydrates needed for growth and reproduction by the trees. Decreased carbohydrate production resulted in a decrease in radial growth and in height of stressed trees. Numerous other organisms and processes were also affected either directly or indirectly, including successional patterns of fungal microflora and their relationship to the decomposer community. Nutrient availability was influenced by the carbon and mineral nutrients accumulated in the heavy litter and thick needle layer under stands with the most severe needle injury and defoliation. A comparison of species of lichens found on conifers during the years 1976 to 1979 with collections from the early 1900's indicated a 50% reduction in species in the more recent period.
The sequence of events occurring in the San Bernardino forest confirm that adequate protection of vegetation will have indirect benefits for soils, wildlife and other welfare categories.

Studies on the combined effect of O_3 and nitrogen together, typical of the conditions in the San Bernardino forest, have shown that the effect of O_3 for some species is greater at higher levels of nitrogen than at low levels of nitrogen. Because nitrogen and sulfur compounds also occur in the pollutant mixture to which the mountains downwind of Los Angeles are exposed (Bytnerowicz et al., 1987 a, b; Solomon et al., 1992), this finding suggests that plants grown with a high nitrogen supply are more sensitive to chronic O_3 stress in terms of biomass reduction (Tjoelker and Luxmoore, 1991).

Since the period when these earlier studies were conducted, air quality in the San Bernardino region has improved. For example, the total number of days/year with

concentrations greater than 0.12 ppm was as high as 159 in 1978, whereas only 105 such days occurred in 1990 (Davidson, 1993). For the period 1974 to 1988, there was an improvement shown in the injury index used to describe chronic injury to crowns of ponderosa and Jeffrey pines in 13 of 15 plots located on the gradient of decreasing O_3 exposure in the San Bernardino Mountains (Miller et al., 1989). Two exceptions were noted in plots located at the highest exposure end of the gradient, where the basal area increase of ponderosa pine was generally less than competing species. Ponderosa and Jeffery pines in plots with slight to severe crown injury lost basal area in relation to competing species that are more tolerant to O_3, namely white fir, incense cedar, sugar pine and California black oak. In effect, stand development was reversed, and the development of the normal fire climax mixture dominated by ponderosa and Jeffery pine was altered. This allowed the formation of a fuel ladder that could jeopardize the remaining overstory trees in the event of a catastrophic fire. Continued monitoring of this system is needed to determine if declining O_3 would eventually allow ponderosa and Jeffery pine to resume dominance in basal area.

An example of the consequences of losing a dominant species in eastern forests is the elimination of the American chestnut from eastern deciduous forests in North America during the first half of this century (Taylor and Norby, 1985). Before the blight, American chestnut comprised 20 to 25% of the canopy in eastern Tennessee. A full half century later recovery patterns are complex, with six distinct successional forest types occupying former American chestnut sites. Thus, it would appear that the nature of community dynamics, particularly in mixed species, uneven aged stands, indicates that subtle long-term forest responses (e.g., shifts in species composition) to elevated levels of a chronic stress like exposure to O_3 are more likely than wide-spread community degradation (Shaver et al., 1994).

Dieback of the spruce-fir forests in the Appalachian mountains has been attributed to many causes, with O_3 sometimes listed. Though this high elevation forest is exposed to a broad range of air pollution stresses, the main culprit of the dieback for Mt. Mitchell is always stated as the balsam wooly adelgid, an insect. There have been no studies done to show insect preference for O_3 damaged trees in this system or that O_3 had weakened trees attacked by the insect. However, given that O_3 can predispose some plants to insect attack,

and considering the example of such a connection in the San Bernardino Forest in California, staff believes this possibility deserves further study.

E. Biologically Relevant Measures of Ozone Exposure

The CD lists a number of exposure indices that have been used in vegetation research. These measures vary considerably in their ability to capture biologically relevant aspects of O_3 exposure that have been shown or theorized to have the greatest potential to influence plant response, thus making it difficult to compare study results that are expressed using different indices. Therefore, a discussion of the current scientific understanding of exposure dynamics and how different index forms capture these exposure features is described below. This information, in combination with the information on welfare effects discussed in the preceding section, permits the evaluation of plant response relative to O_3 exposure levels, patterns, and duration.

1. Biological Considerations

The information discussed in the previous sections describes the plant processes that can be impacted by O_3 once it enters the leaf, and the ways a plant can protect itself in some cases from O_3 injury through stomatal control, antioxidant production, or compensation. Additionally, numerous sources of variability/uncertainty (e.g., biological, chemical, physical, and experimental) were presented that must be considered when explaining study results or comparing one study scenario to other studies done under a different set of conditions. Thus, in the discussions below of the features of O_3 exposure (e.g., concentration, duration, timing, and exposure pattern) that influence plant uptake, it is important to remember that the magnitude of the plant response will be modified by the environmental and biological context in which these exposures occur.

One measure, O_3 uptake, accounts for both the biological and air quality features. Of all the available exposure measures identified, O_3 uptake most closely relates to O_3 dose which is the concentration and duration of the O_3 exposure that is taken up by the plant and actually reaches the target tissue. However, because uptake depends on so many species- and situation-specific variables, it is very difficult to measure and not particularly useful as a basis for standard setting. As a result, researchers have focused their research on identifying suitable surrogate exposure indices for plant response to O_3.

Exposure Duration. In comparisons of replicate studies of varying duration in which O_3 was determined to be the primary cause of variation in plant response, greater reductions in yield (reproduction) or growth occurred to plants exposed for the longer duration (Lee et al., 1991; Olszyk et al., 1993; Adaros et al., 1991). Likewise, with respect to foliar injury, Jacobson identified limiting values for crops and trees which showed that as time of exposure was extended, less O_3 was needed to produce the same response (Jacobson, 1977). Additionally, this relationship between duration of O_3 exposure and plant response has also been supported based on statistical analyses, in which indices that accounted for the length of exposure were better able to rank vegetation effects than those which did not take the duration of exposure into account (Lee et al., 1989). Lefohn (1988) and Lefohn et al. (1988b) conclude that duration has value in explaining variation in plant response and that a cumulative-type index is preferred over a mean or peak index based on statistical fit. Thus, an index that cumulates O_3 exposures over the period of plant sensitivity is desirable.

Despite differences in the period of plant sensitivity across species, a constant duration over which to cumulate exposure must be defined in order to set a nationally consistent standard. Under the current secondary NAAQS, the timeframe of concern (season of highest O_3 production) varies from state to state and may consist of anywhere from 4 to 12 months. Lee et al. (1989) analyzed air quality from 82 non-urban site-years of ambient O_3 data across the U.S. Exposure patterns for the 82 non-urban site-years have similar long-term averages but differ widely in how the O_3 concentrations are distributed within the O_3 season. When the same fixed consecutive three month measurement period during the O_3 season was used for every area, less than 63% of the concentrations of 0.06 ppm or higher were captured. By using instead a floating maximum consecutive three- or four-month time period at each site within the O_3 season, up to 73% and 83% of the peak concentrations were captured, respectively.

Though for most agricultural species the growing season is only approximately 3 months, it has been suggested that trees or other perennial species may require cumulation of exposure over a longer growing season (e.g., 5 months) in order to include the most relevant exposures. However, given that the importance of a longer seasonal period for trees has not yet been adequately investigated, and the maximum consecutive three month period captures

around 3/4ths of exposures to concentrations above 0.06 ppm, the staff judges that the maximum 3-month period is an appropriate surrogate for the entire O_3 season.

An additional aspect of duration involves consideration of the year-to-year meteorological variability which can produce widely varying O_3 levels. This aspect can be addressed by considering averaging seasonal exposures over multiple years. Because annuals go through their entire life cycle within a period of one year, an appropriate secondary standard for annuals must be able to provide protection on a year-by-year basis. In addition, recent seedling research has shown that some perennials also show significant growth effects from O_3 within an annual timeframe. Likewise, foliar injury to sensitive trees in Class I areas often results from short-term acute annual exposures. Additional analyses would be needed to explore the impact of meteorological variability on seasonal exposure indices before a multi-year average could be evaluated with respect to its ability to provide year-by-year protection.

Seasonal patterns of exposure. The sensitivity of annual species to O_3 can vary within the same growing season due to changes in phenology (plant developmental stage). For NCLAN crops, Lee et al. (1989) tested various phenologically weighted functions for their fit to NCLAN data. Out of two exponential and 16 gamma phenological functions, the gamma function which had the highest weight during the time period between 20-40 days before harvest resulted in the best fit. These statistics mirror the biological finding that O_3 can negatively effect different aspects of plant reproduction (Venne et al., 1989; Feder, 1986; Krause et al., 1975; Ernst et al., 1985; Houston and Dochinger, 1977). Increased sensitivity of reproductive processes has implications for yield outputs, genetic success, and aesthetics (flower production) for ornamentals. This increase in reproductive sensitivity has not been found in all tested cases, indicating that phenology is species-dependent. More information on a wider range of species would be needed before a phenologically-based index suitable for national standard setting could be established. Based on the available information, the staff judges that the maximum consecutive three month time period within existing O_3 seasons will likely include the sensitive phenological stages in annuals in most cases.

Exposure Concentrations. As has been stated earlier, there is no threshold O_3 concentration or seasonal exposure level above which effects occur and below which they do not for all species. Over the years, many studies have shown that, depending on the duration of exposures and sensitivity of the plants, injury to crops and other vegetation could occur when exposed to O_3 concentrations that ranged from 0.04 to 0.4 ppm, with higher concentrations usually causing injury in the shortest period of time (CD, 1996). This is due to the known mode of action of O_3 described in section VII-B that vegetation effects occur when the amount of pollutant absorbed exceeds the ability of the plant to detoxify O_3 or repair the initial impact (Tingey and Taylor, 1982). Because many factors influence the amount of O_3 that is absorbed by a plant at any given time, it is impossible to state with certainty that a given concentration will have a known impact on the plant unless all other factors have been accounted for.

Over the last several decades, research has continued to advance the understanding of the complexity of interaction between O_3 air quality and exposure dynamics and timing of plant sensitivities. Until the early 1980's, seasonal means were commonly used to characterize the O_3 exposure believed to be relevant to plant response. However, Larsen and Heck (1984) mathematically showed that it was possible for two air sampling sites with the same daytime arithmetic mean O_3 concentration to experience different estimated crop reductions. At about the same time, concerns about using the long-term average to summarize O_3 exposures began appearing in the literature, specifically that peak concentrations, which some believed might be the most important in determining plant response, were not adequately accounted for by a mean exposure index (CD, 1996).

Since that time several studies have attempted to relate O_3 exposure to plant response. Unfortunately, no two studies have exposed plants in the same manner or under similar conditions so that the data from each study is unique. Exposure methods, concentrations and durations used, age of plants at exposure, length of exposure, the plants exposed and the media in which they were grown all differ across experiments. Some exposures were in chambers in the greenhouse, others in open-top chambers and others in the ambient air. Though the results in general have been inconclusive, two different viewpoints have emerged.

One set of studies, including Musselman et al., (1983, 1986, 1994) and Hogsett et al. (1985b) find evidence to support the view that peak concentrations are important in defining an exposure index. Musselman et al., 1983 and Hogsett et al. (1985b) were among the first to demonstrate that variable concentrations produced greater effect on plant growth than fixed or set diurnal patterns of exposure of equal total exposure with lower peak concentrations. Subsequently, Musselman et al. (1986) and Musselman et al. (1994) found that patterns with higher peak concentrations or longer duration of higher concentrations produced significantly greater effect on top dry weight (kidney bean) than the square wave pattern. These results provide evidence that 1) total exposure (i.e. SUM00), being unable to differentiate among the exposure patterns, is a poor predictor of plant response; 2) the peak concentrations or sequence of peak concentrations (> 0.16 ppm) are important in determining plant response; and 3) greater weight should be given to higher concentrations when describing exposure. Though the Hogsett et al., 1985b study contained both "mid-range" and "peak" concentrations, Musselman, et al. (1983, 1986, and 1994), used exposure levels containing peaks that greatly exceed those in any of the other exposure studies or in ambient air, possibly influencing their findings on the importance of peaks in determining plant response.

The above findings were further investigated for tree seedlings. Hogsett and Tingey (1990) exposed ponderosa pine and aspen seedlings to three different exposure regimes and observed greater growth reductions in the episodic exposure pattern, which had the largest peak concentrations of the three patterns. The smallest growth reductions in both species were observed with the more constant high elevation pattern that had peak concentrations below 0.10 ppm. The authors concluded that temporal pattern and concentration were important in influencing long-term growth response of tree seedlings, just as in crops, and, consequently, should be considered in measures of exposure.

Other publications that have been cited as evidence supporting the importance of high concentrations in eliciting plant response are a series of retrospective studies reporting regression results using data from the NCLAN program (Lee et al., 1987, 1988; Lefohn et al., 1988a; Tingey et al., 1989). These studies were in general agreement and consistently favored the use of cumulative peak-weighted exposure indices. However, Lefohn and Foley

(1992) note that the high number of hourly O_3 concentrations above 0.10 ppm in the NCLAN protocol may prevent generalization of these findings to other types of exposure regimes.

On the other hand, Tonneijck and Bugter (1991), Tonneijck (1994), and Krupa et al., (1993, 1994, 1995) stress the view that mid-level (0.05 to 0.09 ppm) concentrations are as equally important as higher hourly concentrations in affecting vegetation. Krupa (1995) states that in their analyses of European data, "concentrations > 90 ppb (0.09 ppm) appeared to be of little importance because such concentrations in general appear to occur during atmospheric conditions which did not facilitate ... uptake." Most of the studies listed above base their conclusions on the foliar injury response of Bel W-3 tobacco, which has been noted in numerous earlier studies to have an inconsistent relationship with O_3 air quality. The authors Tonneijck and Bugter (1991) and Tonneijck (1994) also acknowledge this deficiency and state that "foliar injury on tobacco Bel W3 was poorly related to the ambient O_3 in the Netherlands." Further complicating the interpretation of their results, the papers do not cite the actual O_3 concentrations to which the plants were exposed, except as mean values. The 1996 CD concludes that based on the known inconsistency of Bel W3, the conclusions presented by Krupa et al., 1995 needed to be verified.

As stated earlier under section VII-B-1, stomatal conductance is influential in determining the dose received by the plant. The information presented above suggests that the range of concentrations of concern for any particular plant can change depending on the combination of other stresses acting on the plant, the phenological stage of the plant, the duration of the exposure and stomatal conductance.

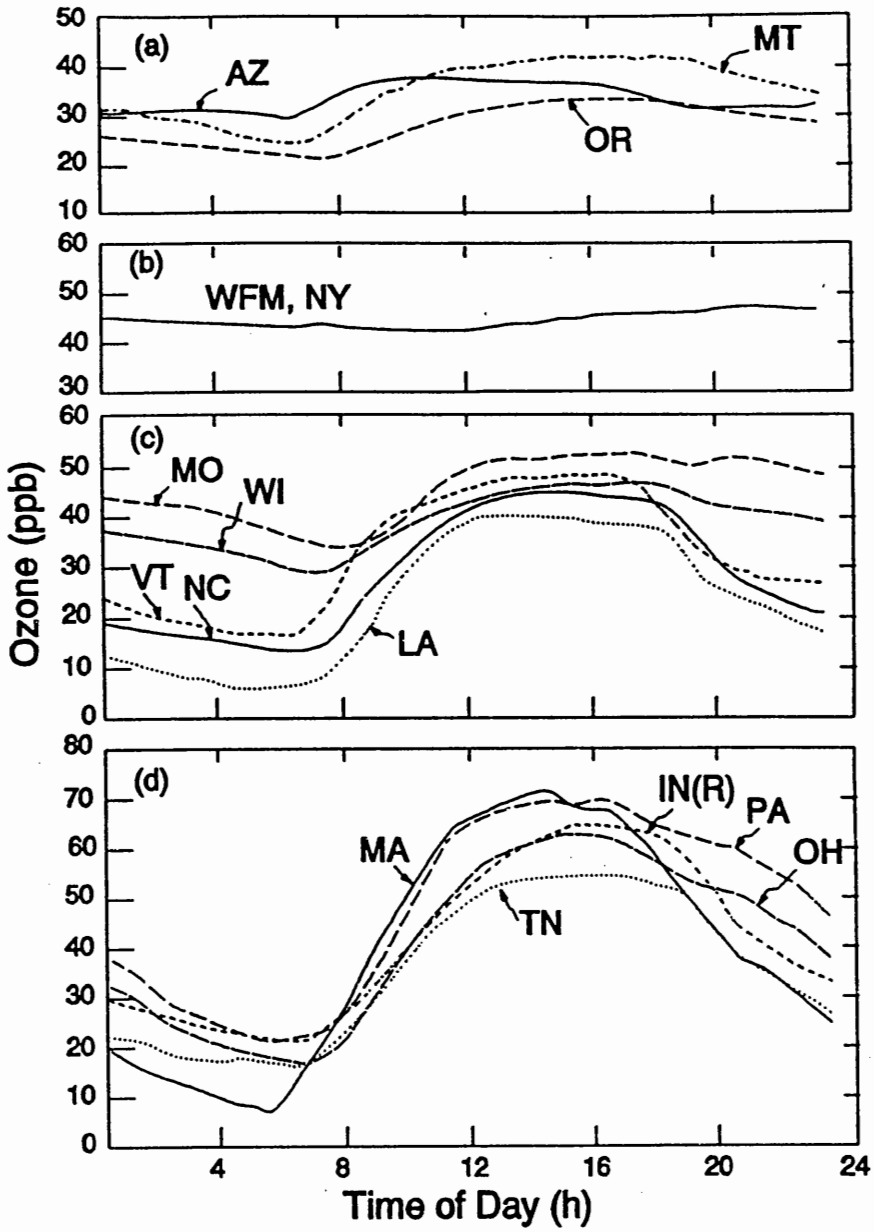
Diurnal Patterns of Exposure. Most plants are believed to open their stomata during the day and close them at night. It has been reported, however, that some plants have stomata that stay open well after the sun has set. For example, it has been reported (U.S. EPA, 1978) that white pine keeps its stomata open all night, while Tobiessen (1982) reported that many early successional trees, such as black cherry, big-toothed aspen and white ash open their stomata at or before dawn, whereas late successional species tend not to show such a pattern. This suggests that if environmental conditions favor stomatal opening, and O_3 levels are elevated, plants may take up O_3 in the predawn hours (Neufeld et al., 1992).

Air quality information has already shown that diurnal patterns in rural sites may vary significantly from the typical urban pattern of an early afternoon peak, due to long-range transport or elevational effects. Figure VII-5 shows a range of diurnal patterns of O_3 concentrations for U.S. rural sites. Long-range transport processes bringing O_3 from more distant urban areas can change the timing of rural O_3 peaks from afternoon to evening to early morning hours. Furthermore, at mountainous sites which are above the nocturnal inversion layer, more constant levels occur because there is little or no nighttime scavaging of O_3 by nitric oxide. Urban areas, on the other hand, typically experience a wide range of O_3 concentrations that build to a peak in the daytime and then fall to near negligible levels at night from scavenging. On reviewing the AIRS database for 1990 to 1992 for those agricultural and forested site types experiencing a 3-month, 24 hour SUM06 greater than or equal to 26.4 ppm-hours in the U.S., it was found that 70% of the sites experienced at least 50% of the occurrences of $\geq$ 0.10 ppm O_3 peaks during the 7 hour period 9 am to 4 pm (CD, 1996).

The published literature shows that the most major effects associated with O_3 exposure are linked to the disruption of the photosynthetic process. Since photosynthesis occurs only when certain minimum light conditions are met, the staff believe that the diurnal time period of greatest concern for vegetation, therefore, can be defined as the daylight hours. Using a daily exposure period longer than the traditional 7 hour window of 9 am to 4 pm is supported by a study done by Heagle et al. (1987) which compared the effect of increasing the time frame of exposure from 7 to 12 h/day. They found that plants receiving exposures for an additional 5 h/day (i.e, 12 h) showed 10% greater yield loss than those exposed for 7 h/day.

Though the length of daylight hours can range from close to 12 hours to as much as 16 hours in different parts of the U.S. during the summertime, maximum O_3 months, the staff feel that the percent of concentrations occurring outside the 12 hour daylight window are sufficiently small and outside the period of greatest sensitivity for most studied vegetation, as to make the 12 hour daylight (8 am to 8 pm) an appropriate timeframe over which to cumulate exposure.

Figure VII-5. Diurnal Ozone Patterns at Rural Sites



Diurnal behavior of ozone at rural sites in the United States in July. Sites are identified by the state in which they are located. (a) Western National Air Pollution Background Network (NAPBN); (b) Whiteface Mountain (WFM) located at 1.5 km above sea level; (c) eastern NAPBN sites; and (d) sites selected from the Electric Power Research Institute's Sulfate Regional Air Quality study. IN (R) refers to Rockport.

Source: Logan (1989)

Intra-episodic Patterns of Exposure. Other factors, including a predisposition effect of early O_3 exposures on plant sensitivity to later O_3 episodes (McCool et al., 1988) can contribute to variations in biological response. Consideration of this factor suggests the need for weighing O_3 exposures to account for predisposition time. However, the role of predisposition in influencing plant response varies with species and environmental conditions and is not understood well enough to allow specification of a weighing function for use in characterizing plant exposure.

Summary. In summary, in spite of the complexity inherent in vegetative systems, research developed over the last several decades and most recently since 1988, has produced information on exposure dynamics and their role in producing plant response. Data in published literature still supports the conclusion that cumulative seasonal exposure and higher concentrations are important features of exposure for both crops and trees. Ideally, an exposure index would account for all of the variation in vegetation effects that are associated with exposure to O_3. A second, more practical objective is the specification of an exposure index that is applicable in the ambient air quality standard setting process. An exposure index for a NAAQS should be easy to develop and applicable to a wide range of species and environmental/exposure conditions. The attainment of these criteria, however, necessarily represents a compromise in the features included in the formulation of the best exposure index (Lee, et al., 1989). The section that follows highlights those types of forms which meet these criteria.

2. Alternative Forms of the Secondary NAAQS

The discussion of exposure indices presented in the CD groups indices into several generalized forms including one event, mean, cumulative, concentration weighing, and multicomponent. These general forms are discussed below with regard to their biological relevance and their suitability as a basis for standard setting.

One Event, Mean, and Cumulative Forms. The one event form, which includes the current form of the NAAQS, measures only one or a very limited number of peak events out of a plant's entire growing season. If O_3 concentrations never achieve the level of the peak of concern, the assumption is that no growth or yield effects of concern are occurring. It also does not distinguish among exposures of different durations. This index form does not

account for many exposures and patterns of exposure that are associated with vegetation effects. The mean form, such as the seasonal mean of the 7-hr daily means, averages all selected concentrations equally and does not address the varying patterns of exposure. Larsen and Heck (1984) demonstrated that for two air sampling sites with the same daytime arithmetic mean O_3 concentration very different estimated crop reductions could occur. The cumulative form sums all hourly concentrations across a season (SUM00) and, thus, contains an exposure duration component, but still weights all concentrations equally. Using air quality data, Lefohn et al., 1989 showed that the magnitude of the SUM00 exposure index was largely determined by the lower hourly average concentrations. In a similar study, Lefohn et al. (1992) noted that the magnitude of the SUM00 index did not adequately account for the occurrence of the higher hourly average concentrations in the ambient treatments. The coupling of the air quality considerations as described by Lefohn et al. (1989, 1992), with the biological findings reported by Musselman et al. (1983, 1994) and Hogsett et al. (1985b), builds a consistent picture that the SUM00 index does not adequately account for the occurrence of peak hourly concentrations. The one event, mean, and cumulative forms do not take into account the many features of O_3 exposure regimes that influence plant response and are, therefore, limited in their usefulness as predictors of O_3 injury.

Multicomponent Forms. In contrast to the relatively simple forms discussed above, multicompartment forms have been developed which attempt to take into account a range of factors that have been associated with vegetation effects. For example, Lee et al. (1989) analyzed crop yield data for 17 individual NCLAN studies, using regression analysis to evaluate a total of 614 indices, including 589 variations of the Generalized Phenological Weighted Cumulative Index (GPWCI) using the Weibull model. The exposure indices were evaluated on the basis of statistical fit along three different criteria and an average score given. Though no single exposure index performed best for all 17 cases, there was a group of indices that were always at or near the top ranking scores. The 100 top performing indices were all GPWCI indices, with overall best index being the GPWCI index PWCI485, with a sigmoid weight centered at 0.062 ppm and a phenological gamma weighting scheme placing maximum weight 30 days prior to maturity. Although such multicomponent indices

may well take into account many relevant factors, including plant phenology, predisposition effects of early O_3 exposures to later ones, and relative impacts of different concentration ranges, such forms have not been advanced as being applicable for standard setting due to being species-specific and highly complex.

Concentration-Weighted Forms. In the same analysis by Lee et al. (1989), 25 general index forms were also evaluated. Several threshold (SUM06, SUM07, SUMO8, AOTO8) and sigmoidally weighted cumulative indices were nearly as optimal in fitting the NCLAN database as was the multicomponant GPWCI index. The sigmoidal weighting function that performed the best increases monotonically from 0 to 1 and assigns a weight of 0.5 at 0.062 ppm (designated SIGMOID in later documents). In a separate study, Lefohn et al. (1988) compared two sigmoidal functions, W126 (which has an inflection point at 0.067 ppm and gives equal weight to values above 0.10 ppm) and W95 (which gives greater weight to values above 0.10 ppm). Though the W126 performed better than the W95, it has not been directly compared to the SIGMOID to determine if one better represents plant response. The W126 form does, however, give less weight to the lowest range of concentrations (i.e., those that fall within typical background levels) than the SIGMOID.

Several threshold cumulative forms also attempt to take into account evidence that peaks produce a disproportionate response relative to lower concentrations by selecting a "threshold" value below which the O_3 concentrations are not counted. These forms assigned a weight of 0 to concentrations below the "threshold" and concentrations above the "threshold" are assigned a weight of 1. The hourly concentrations which fall above the "threshold" level (i.e., the SUMXX forms) or the difference between the concentration and "threshold" levels (i.e., the AOTXX forms) are then added together to give a cumulative seasonal total exposure. The establishment of a "threshold" value is somewhat arbitrary and is not based on any evidence of a discernible threshold for vegetation effects in general. The "threshold" levels identified in previous research have sometimes been set to factor in other considerations, such as the level of background concentrations or the ability of models to predict air quality concentrations below certain concentrations.

Staff notes that the results of the retrospective analyses of exposure indices have received several critiques which point out the artificiality of the O_3 exposure regimes used in

the NCLAN studies to elicit vegetation responses (Lefohn and Foley, 1992). The concern is that the use of an exposure regime which contains unrealistically large numbers of high peaks may have exaggerated the vegetation response. Thus, it is not clear at this time whether the exposure indices identified in Lee et al. (1989) as the best predictors of plant response in the NCLAN studies would have been ranked in the same order under a different exposure profile. Others have critiqued the analyses because they do not directly address biological factors, but rather reflect only statistical associations.

Additionally, this same NCLAN database has been used to calculate levels of O_3 exposure for different indices that would provide "equivalent" protection for crops from certain defined yield loss percentages. Lefohn and Foley (1992) suggest it is important that any exposure index that sets a level of protection based on the response of plants in the NCLAN experiments should recognize the peakiness of the exposure regimes used when attempting to predict biological responses over the range of ambient O_3 exposure regimes.

Though variations of three selected exposure indices (SUM06, AOT06, and W126) being considered in this review performed equally well in statistically predicting plant response from the NCLAN data in Lee, et al. (1989) which had high numbers of peak concentrations, questions have been raised as to how these indices would compare under different exposure scenarios. In attempting to address these questions, staff compared how these selected exposure indices differentially weight peak, mid-range and low O_3 concentrations for various ambient air quality scenarios from several NCLAN studies and air quality distributions produced from the AIRS database for a variety of selected monitored locations (Appendix F). These comparisons were done on the basis of the percentage of the total value of the index contributed by selected portions of the range of O_3 concentrations. For example, using the O_3 ambient air quality distribution for one NCLAN study (R85CO) conducted in Raleigh with cotton in 1985 to compare SUM06, AOT06, and W126, the following percentages were observed:

	$\leq$ 60	60-80	80-100	$\geq$ 100 (ppb)
SUM06	0	60	30	10
AOT06	0	38	42	20
W126	15	42	31	12

From such comparisons staff observed that for any given representative ambient air quality distribution, the percentage of the total index value contributed by the upper mid-range and peak concentrations was highest for the AOT06 index, indicating that AOT06 effectively weighted the upper mid-range and peak concentrations relatively more than did either the W126 or the SUM06 indices. Furthermore, unlike the AOT06 and SUM06 indices which do not include concentrations below 0.06 ppm, low concentrations (i.e., <0.06 ppm) can account for a significant percent of the total for the W126 index while concentrations between 0.06 and 0.08 ppm tend to represent the greatest percentage of the total value in the SUM06.

F. Considerations in Characterizing Adverse Welfare Effects

Though exposure-response functions of O_3 on vegetation have been developed from studies described in the sections above, several additional pieces of information (e.g., national O_3 exposure patterns; location of O_3 sensitive species) are added in this section to put this information into a national context that can more fully inform the Administrator as to the need for additional protection for vegetation against O_3-induced adverse welfare effects and to better characterize the residual risks and benefits associated with various policy decisions.

As part of this effort, the staff took into account the possibility of a new primary standard consistent with the options presented in section VI. Recognizing that attainment of any of these alternatives would generally lower O_3 exposures nationally, the staff performed several comparisons using both monitored and projected air quality to evaluate the impact of attainment of various primary options on O_3 concentrations of concern to vegetation. Benefit and risk reductions associated with reductions in O_3 exposure from the attainment of a separate secondary standard are therefore considered incremental to those achieved by attaining any of the primary standard options.

1. Exposure Characterization

National Monitoring Network. Sparse air quality monitoring has always constrained the characterization of national rural and remote air quality. The first national network of air monitoring stations designed to measure levels of O_3 in remote areas (100 or more miles from any major urban area) within the 48 contiguous states was established in 1976 and ran

through 1983 as a cooperative effort between the U.S. EPA and the U.S. Forest Service. By 1980, this National Air Pollution Background Network (NAPBN) consisted of eight sites operating in selected National Forests, although only six of these sites operated year-round (Evans et al., 1983; Evans, 1985). As of 1987, less than 2% of the O_3 monitoring sites in the AIRS database, were classified as forested. The remaining sites were divided between residential, commercial or industrial, 79%, agricultural, 17%, and desert or mobile, 3% (U.S.EPA, 1987).

Though the rural monitoring network has grown to now include approximately 80 monitors in Class I areas, the majority of AIRS sites are still located in urban or near urban areas. Even many of the monitors classified as rural occur within cities or Census Metropolitan Statistical Areas (CMSAs), and often show O_3 air quality patterns typical of urban areas (e.g., low nighttime O_3 due to scavenging (Stasiuk and Coffey, 1974), with high diurnal peaks, often including occurrences of hourly average concentrations above 0.10 ppm). The 1991 monitoring network for both urban and rural U.S. monitors still show large sections of the country with little or no monitor coverage (Figures VII-6a and 6b). Some of these non-monitored areas are significant for a variety of crops such as wheat, barley, corn, sorghum, soybean and kidney bean production, as well as for the tree species black cherry, sugar maple, red maple, aspen, red alder, white pine, Douglas fir, and ponderosa pine.

Using 1991 to 1993 AIRS monitoring data, the staff examined the question whether attaining a proposed primary standard could provide sufficient protection to vegetation with respect to cumulative seasonal exposures that have been shown to injure plants. The specific example chosen for comparison was the primary option (0.08 ppm, 8 hr., 1 or 5 expected exceedences) and the secondary option (SUM06, 12 hr., 25 to 38 ppm-hrs.). Staff examined the 8-hour daily maximum and 12-hour SUM06 design values for 581 counties (those having sufficient monitoring data in AIRS for the period 1991-1993). Figures VII-7a and b show the associations between the 8-hour values and the SUM06 values. These figures show, for example, that almost all areas that are within or above a SUM06 range of 25-38 ppm-hr would also fall above an 8-hour design value of 0.08 ppm. Thus, those monitored sites that would likely be of most concern for effects on vegetation would also be addressed by an 8-hour primary standard set at a 0.08 ppm level.

Figure VII-6b. 1991 Rural Ozone Monitoring Site Locations

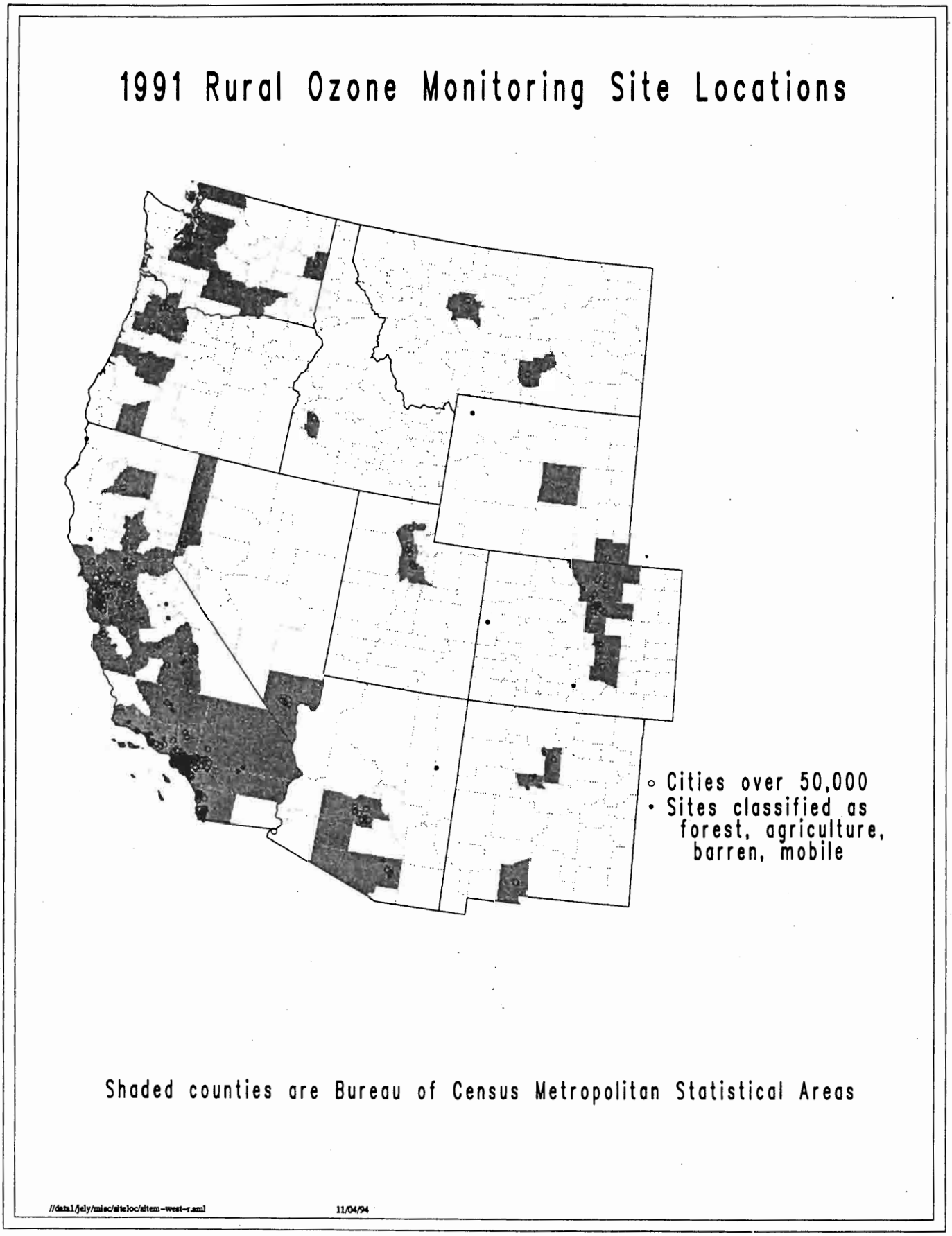


Figure VII-6a. 1991 Rural Ozone Monitoroing Site Locations

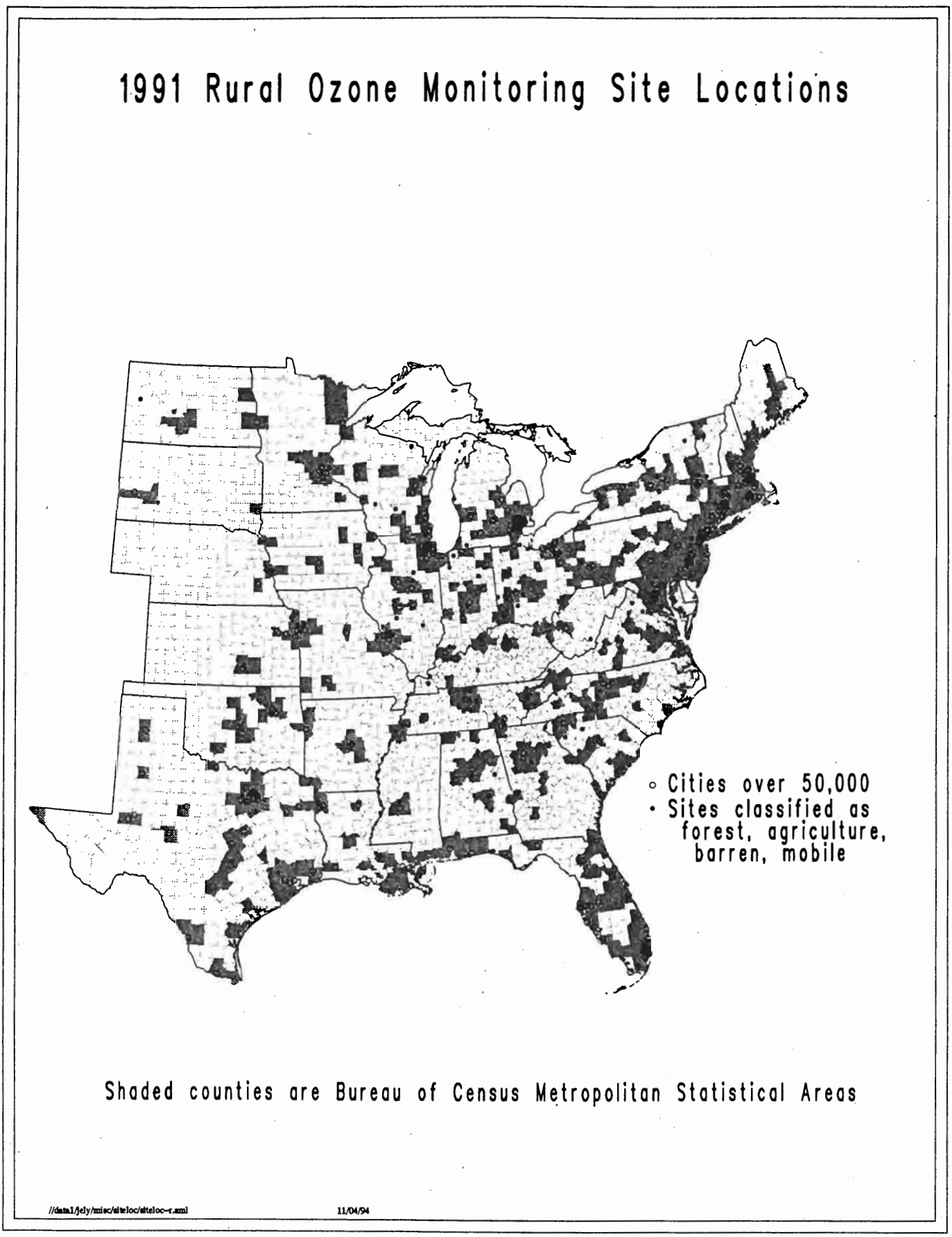


Figure VII-7a. 1991-1993 Air Quality Relationships

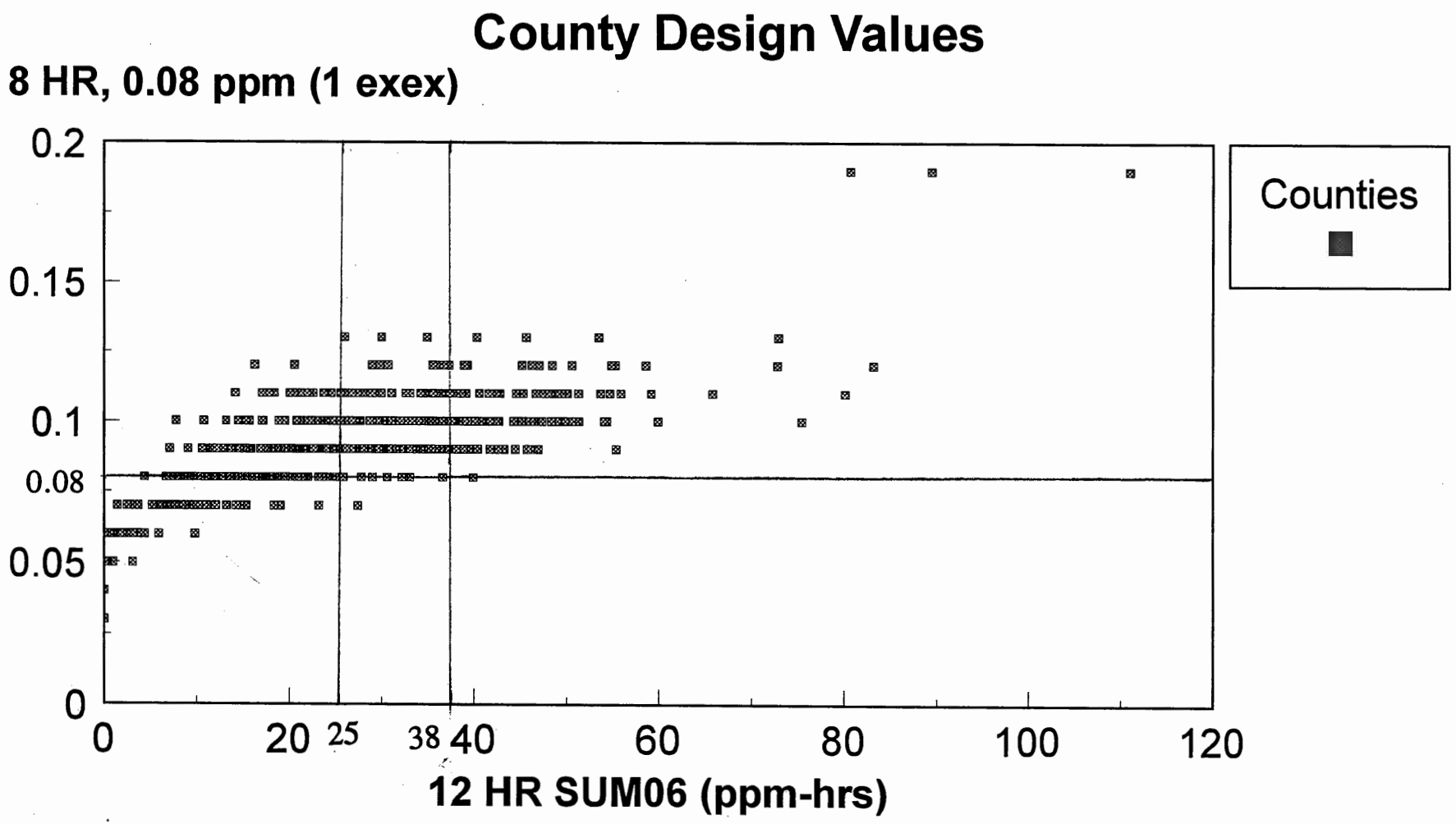
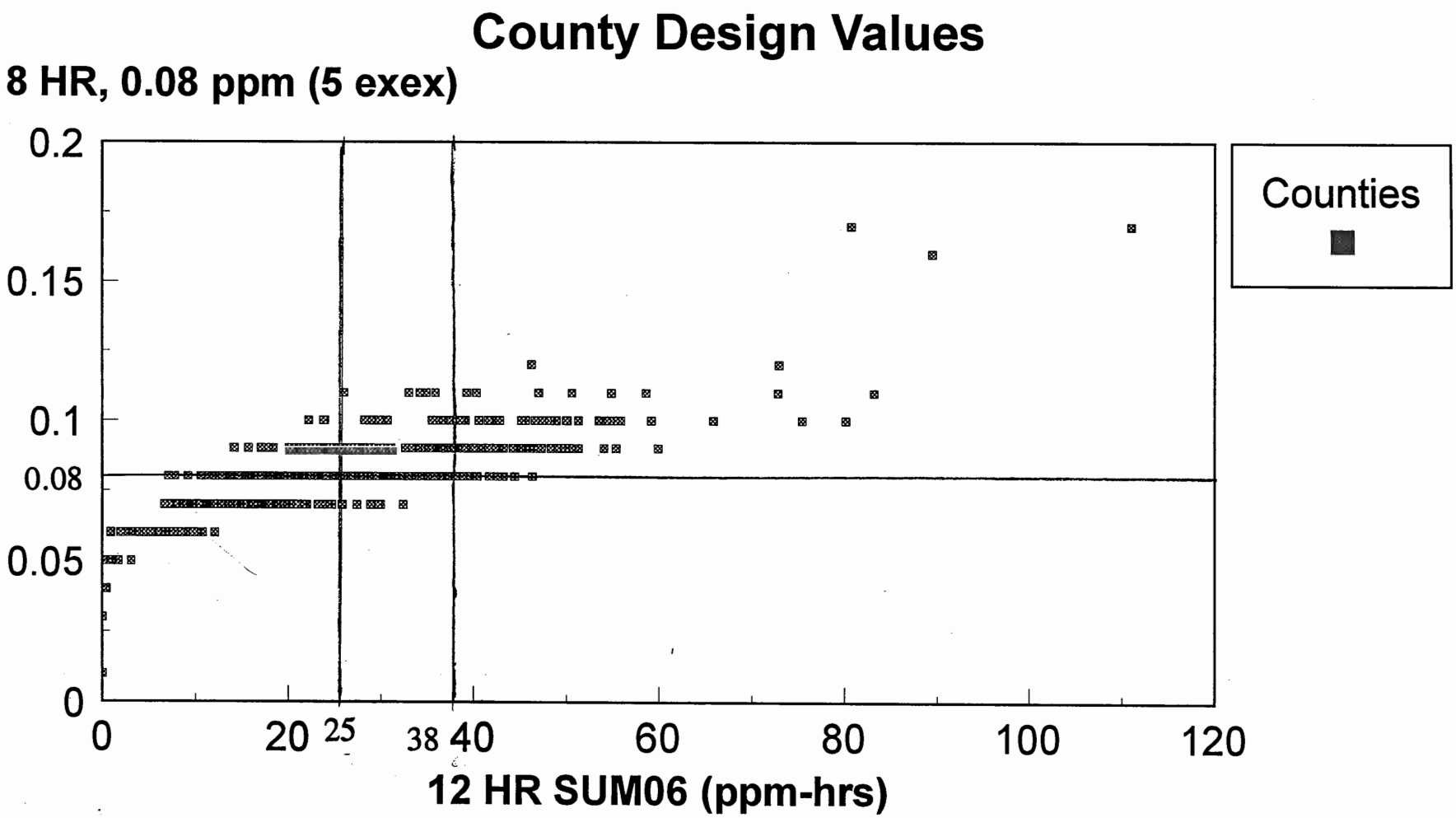


Figure VII-7b. 1991-1993 Air Quality Relationships



However, given that the greatest proportion of vegetation in the U.S. is found growing outside of urban centers where there are few or no monitors and where different environmental and elevational factors interact with O_3 precursors or O_3 transported into the site, there is considerable uncertainty as to the exact nature and strength of the relationship between urban O_3 air quality and distributions that occur in rural or remote areas.

In recent years, more and more researchers have undertaken studies to better characterize O_3 air quality in rural, remote or "clean" sites, (Lefohn and Jones, 1986; Lefohn and Foley, 1992; Logan, 1989; Bohm, 1992). Though often extremely limited in scope, these studies continue to refine and build on the work by Evans et al., 1983 and Evans, 1985 and can generally be divided between western and eastern U.S.

Western Air Quality. Logan (1989) corroborated an earlier finding from the three western sites (Apache, AZ; Ochoco, OR; Custer, MT) in the original NAPBN that O_3 hourly average concentrations above 0.08 ppm rarely are exceeded at remote western sites except in some areas in California. For example, outside of California, even near urban sites, O_3 concentrations remained low, with growing season means ranging from 0.012 to 0.022 ppm, 0.028 to 0.037 ppm, and 0.032 to 0.058 ppm in Washington, Utah and Colorado, respectively. Not unexpectedly, there is little evidence of O_3 injury at these sites. On the other hand, Bohm (1992) reports that Yosemite and Sequoia National Parks, which receive pollutants transported from highly urbanized areas, had 24 h means ranging from 0.036 to 0.085 ppm on 75% of summer days, whereas Lake Gregory had a growing season mean of 0.073 ppm. During 49% of the summer days, means of diurnal patterns ranged from 0.085 to 0.100 ppm, decreasing with altitude and distance from the source (Bohm, 1992). These levels, as mentioned in the earlier effects section, have been associated with growth decreases and foliar injury in some species.

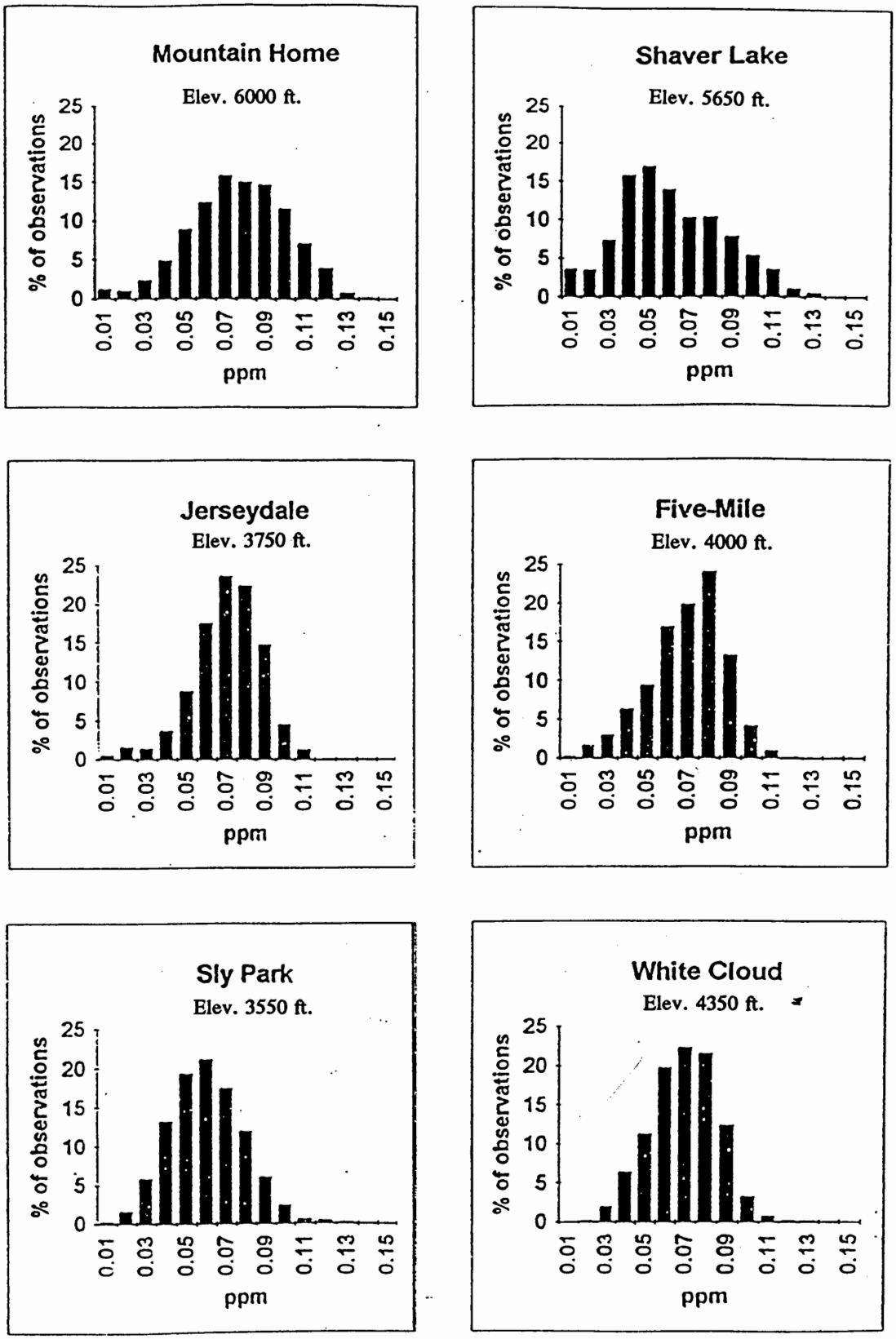
An ongoing study, the Sierra Cooperative Ozone Impact Assessment Study (SCOIAS), co-operated by the U.S. Forest Service, California Air Resources Board, and the University of California at Davis, is documenting exposure of sensitive pine species in the Sierran forests to O_3 and the amount of injury the trees exhibit. As part of this project, six sites, ranging in elevation between 3550 and 6000 feet above mean sea level, were selected to

monitor O_3 concentrations and meteorological conditions (temperature, humidity, wind speed, wind direction and solar radiation).

After completing 3 to 4 years of data collection (1990, 1991 to 1993) the researchers found that O_3 concentrations were typically highest in the afternoon, and increased as one moved toward the southern end of the network, which also was in the direction of increasing elevation. Stations located on well defined steep slopes show a very strong diurnal variation in O_3 concentrations and meteorological conditions. Air quality distributions of hourly data from 1992 for these sites are shown in Figure VII-8. Hourly peak O_3 concentrations from June through September were greater than 0.06 ppm at all sites nearly every day, in excess of 0.08 ppm at most sites more than half the days and in excess of 0.10 ppm at least a few days a month. The most impacted sites, Mountain Home (6000 ft.) and Shaver Lake (5650 ft.) were the southernmost units and had concentrations above 0.10 ppm for nearly half of the days during the monitoring period. At the two sites in the middle of the network (Jerseydale (3750 ft.) and Five-Mile Learning Center (4000 ft.)) the diurnal variations in O_3 were not very well pronounced and nighttime values remained relatively high. At White Cloud (4350 ft.), the northernmost unit in the study, the highest concentrations occurred at night, when winds were from the NNE (Sierra Cooperative Ozone Impact Assessment Study, 1993). At the time of this interim report, foliar injury information at these sites is still incompletely characterized. However, these O_3 exposures are well above those concentrations associated with vegetation injury. According to the report, "available literature indicates that needle injury occurs from exposure to O_3 concentrations of 0.06 ppm, and is significant at and above 0.08 ppm .... The recorded data suggest that serious to severe exposure ($>$ 0.08 ppm) of pines to ozone is likely" (Sierra Cooperative Ozone Impact Assessment Study, 1993).

Eastern Monitored Air Quality. Questions have been raised as to whether O_3 air quality distributions experienced at eastern sites are representative of sites in the western United States. Differences in biogenic precursors and more gradual changes in elevation that never reach the high elevations found in the west complicate efforts to define an O_3 air quality pattern that consistently applies to all high elevation sites nationally. In eastern locations, the relatively flat air quality patterns considered typical of a high elevation site

Figure VII-8. Ozone Frequency Distributions of Hourly O_3 Concentrations from June to September 1992



(including an area being above the nocturnal inversion layer and being affected by long distance or regional transport) occur at different heights.

In a study by Winner et al. (1989) an effort was made to relate O_3 exposure patterns to elevation. Several sites were monitored in western Virginia from May to December 1982, ranging in elevation from 457 m to 1067 m. In general, the high elevation site, Big Meadows, in the Shenandoah National Park, had higher monthly O_3 concentrations than the lower elevation sites. However, the number of peak O_3 occurrences ($\geq$ 0.10 ppm) did not necessarily increase with altitude. Instead, higher monthly averages seem to be associated more with a lack of nighttime scavenging than with a large number of peak hourly concentrations. Additional comparisons with the following years 1983-1985 showed that the pattern was consistent across years.

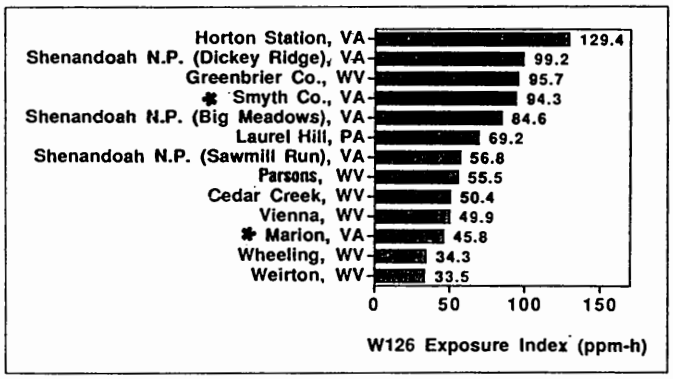
Lefohn et al. (1994) compared several sites located in West Virginia, Virginia, and Pennsylvania for the years 1988 through 1992 in terms of the seasonal (April to October) 24 h W126 exposure index, as shown in Figure VII-9. Of the 11 sites with data for all 5 years, the 6 sites with the highest exposures were also the higher elevation sites ($>$ 500 m), while those sites with the lower exposures were all below 500 m in elevation. The highest elevation sites were also observed to have a large number of O_3 episodes, with the number of hourly peaks $\geq$ 0.10 ppm ranging from only a few in 1992 to over 100 in 1988.

Though these values were collected over a 7 month period, in 1988 all 11 sites exceeded the 3 month W126 level (21.0 ppm-hr) identified by Hogsett et al. (1995) as protecting 50% of the tree seedling cases studied from greater than 10% biomass loss, while only two sites were below this level in 1991. In the other years, except for 1992, more than half the sites exceeded this level (Figure VII-9). Such patterns are consistent with results of recent monitoring in the Great Smokies National Park described by Neufeld et al. (1992). However, with only 2 permanent and 1 seasonal monitoring stations, O_3 levels are not well characterized over much of the park.

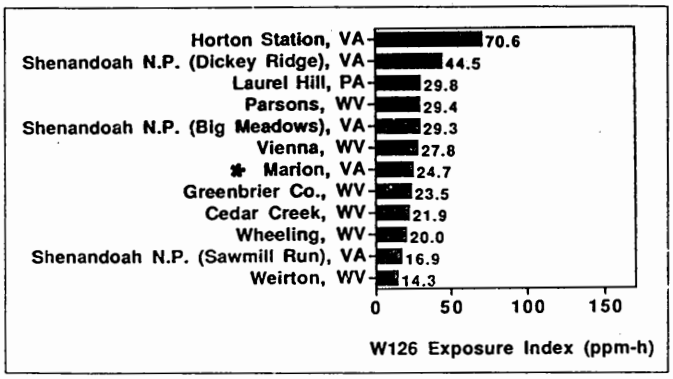
Though O_3 concentrations in the east may not reach high concentrations as frequently as in the west, the above studies indicate that O_3 air quality in some forested areas in the east contain both peak and cumulative, seasonal exposures known to have caused both foliar injury and growth reduction in some vegetation.

Figure VII-9. The Comparative Ranking of Ozone Monitoring Sites From April to October Using the 24 hr W126 Exposure Index (1988-1992)

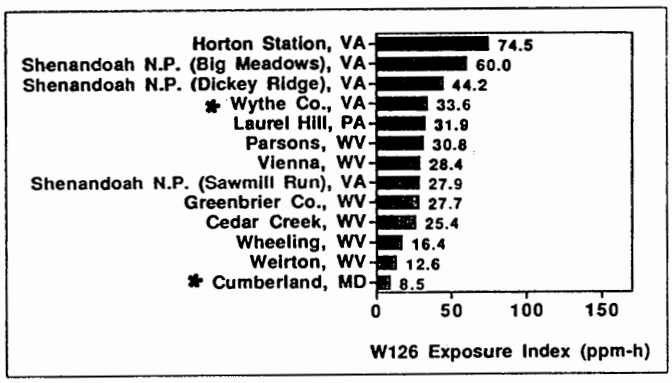
*Sites with less than 5 years of data



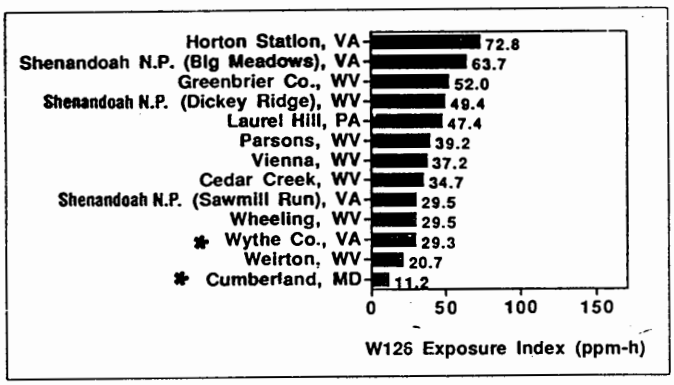
The comparative ranking of O_3 monitoring sites in April to October 1988 using the W126 exposure index.



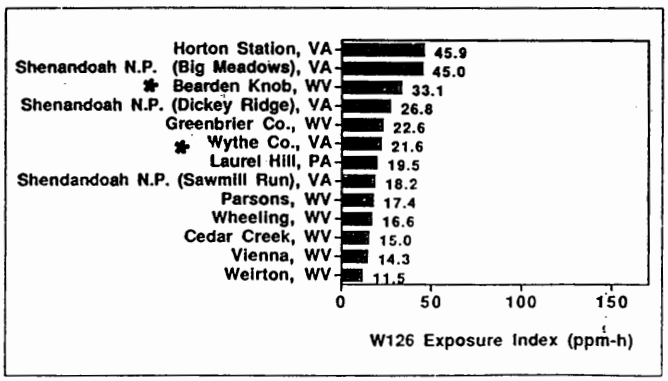
The comparative ranking of O_3 monitoring sites in April to October 1989 using the W126 exposure index.



The comparative ranking of O_3 monitoring sites in April to October 1990 using the W126 exposure index.



The comparative ranking of O_3 monitoring sites in April to October 1991 using the W126 exposure index.



The comparative ranking of O_3 monitoring sites in April to October 1992 using the W126 exposure index.

Uncertainties in Air Quality Relationships. As has been shown earlier in this document (Figure VII-5), diurnal patterns of O_3 can differ significantly between urban and rural areas. The staff's evaluation of monitoring data indicates that there is considerable overlap between areas exceeding a 0.08 ppm, 8 hr. standard and those exceeding the 25 to 38 ppm-hr. range of concern for vegetation, suggesting that improvements in national air quality from attaining an 8-hour primary standard within the recommended range of levels would also reduce levels below those of concern for vegetation in those same areas. However, there remains concern whether urban control strategies would result in attainment of either 8-hr or cumulative standards in downwind rural areas. Therefore, in order to develop a more complete understanding of rural and remote O_3 air quality and its relationship to attainment of various primary options, staff examined various techniques and methods that have been or are used to produce spatial estimations of national O_3 exposure.

Spatial Estimation of Ozone Exposure. At the time of the NCLAN research project discussed in the previous section, national air quality typical of agricultural crop growing areas was unknown. To estimate O_3 exposure over non-monitored areas across the country, NCLAN used the spatial interpolation technique of kriging. Kriging, like all spatial interpolation techniques, is based on the common observation that, on average, points close together in space are more likely to have similar values of a property than points further away. As such, spatial interpolators generate a generally smooth gradient of values from one monitored site to another. Three key issues affect the reliability and appropriateness of using spatial interpolation to estimate O_3 exposure values at non-monitored locations. These are: 1) the spatial representativeness of the monitored sites; 2) the sampling density of the monitored sites; and 3) the spatial variation of O_3. Using spatial interpolation to estimate O_3 exposure at non-monitored locations is appropriate only if the monitored sites are representative of all possible locations to which an estimate is to be made and if the monitored sites occur at a sufficient sampling density to adequately represent the non-monitored areas and capture the spatial variation in O_3 exposure in these non-monitored areas. That is, the monitored sites must be able to capture the frequency and amplitude of exposure "peaks and valleys". The existing monitoring network is inadequate to make estimates of O_3 exposure over large sections of the country (see section VII-F.1).

More recently other techniques have been used to estimate O_3 exposure over non-monitored areas. These include computer simulation or modeling approaches such as the regional oxidant model (ROM). Such modeling approaches predict O_3 air quality by simulating the many chemical and physical factors and relationships that influence O_3 formation, transport and attenuation. Obtaining the input data to run such models and identifying the mathematical relationships between all the many variables is an expensive and time consuming process. Modeling the spatial variability of O_3 air quality from these relationships can easily become a task on the same magnitude as modeling weather and climate-change, requiring massive computational power.

The NHEERL-WED is using a geographical information system (GIS) as a tool to develop an estimation technique that has characteristics of both interpolation and modeling (Hogsett et al., 1995). GIS is a formalized computer tool that allows one to integrate and manipulate spatial data. A GIS has characteristics similar to other computer programs such as computerized mapping, database managers, and spread sheets and, in fact, GIS has borrowed heavily from these technologies.

The objective of the GIS-based technique is to improve the estimation of O_3 air quality in non-monitored areas by using information on factors that influence O_3 formation and transport. These factors include sources of O_3-forming precursors, wind direction, temperature, cloud cover, elevation, and distance from emissions sources. The factors are used to identify the expected trends and patterns of O_3 air quality one could logically expect to observe between the sparse and distant monitored sites. The assumption is that areas experiencing a great number of days with elevated temperatures and low cloud cover and that are down-wind of sites having large emissions of O_3-forming precursors will have a greater potential for experiencing elevated O_3 concentrations than areas not situated in such high potential locations.

To make estimations of O_3 air quality in non-monitored areas the GIS is used to create what is referred to as a potential exposure surface (PES) using information about the influencing factors. The form of the PES is a 10 km grid superimposed across the country. Each 10 km cell of the PES receives a value representing the sum of all factors influencing O_3 formation and dispersal. Hence cells in locations with high temperatures and generally

cloud-free skies and down-wind from large amounts of O_3-forming precursors will have high values. Once the PES is created, the relationship between the PES values and monitored O_3 values at each monitored site are used to calibrate the PES so that it can be used as a surrogate for O_3 air quality in non-monitored areas. Rather than assuming a smoothly changing surface between the monitored sites as traditional interpolation techniques do, the GIS-based approach predicts more structure between monitored sites based on factors known to influence O_3 air quality. Although this integrated GIS-based approach is not as complex or sophisticated as a true computer model it has the advantage of using data that is readily available across the entire country, is relatively inexpensive to run, and allows one to quickly produce estimates of any exposure index for multiple months or years.

Uncertainties Associated with GIS-Based Ozone Air Quality Estimation Technique. As with any spatial interpolation technique that must rely on sparse data representative of urban or near-urban areas uncertainty is great and non-quantifiable. Intuitively the estimates made from the GIS-based approach make more sense than those made using a traditional spatial interpolation technique such as kriging or inverse distance weighting. This is especially true in the western U.S. where monitoring sites are many hundreds of kilometers apart. The GIS-based approach predicts large areas of the West as having low O_3 air quality due to their remoteness to sources of O_3-forming precursors whereas traditional interpolators predict a generally smooth gradient of elevated air quality between such distant monitors as San Francisco, Salt Lake City, and Seattle. The GIS-based technique makes use of the data values at each monitored site and honors the monitored value at each site. Assuming the PES is a reliable indicator of the patterns and variation of O_3 air quality between monitored sites and since the density of the 10 km grid of PES values is much greater than the density of monitored O_3 sites, which can be hundreds of kilometers apart, the ability to capture trends and variation of O_3 air quality between monitored sites is theoretically improved. However, EPA recognizes that uncertainties inherent in methods for estimating air quality and for adjusting air quality to consider different attainment scenarios remain. Moreover, while the results of applying the GIS technique correspond well with other methods for assessing and displaying air quality data from the same year, there has been no formal external peer review or performance evaluation of this national air quality extrapolation.

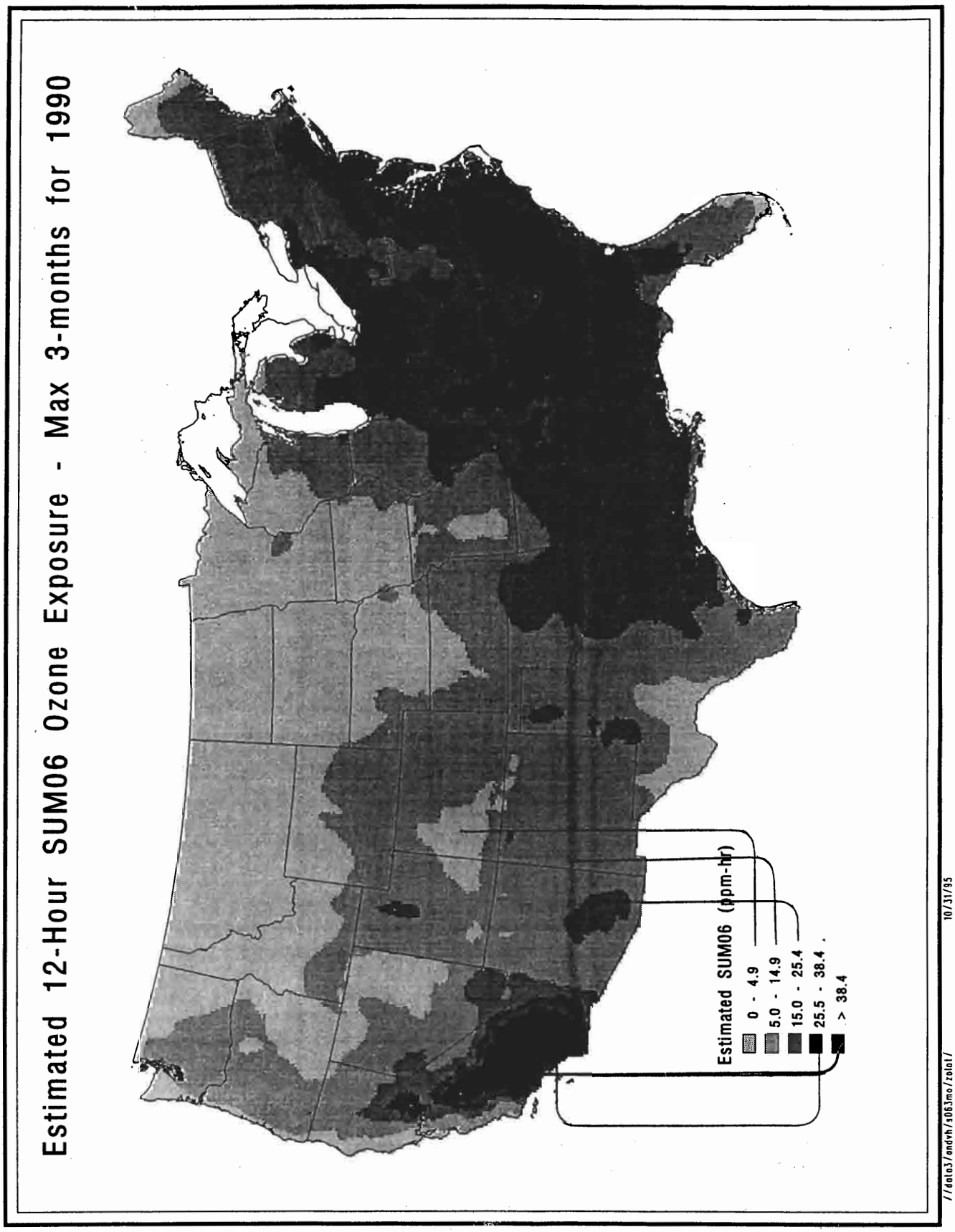
2. Assessment of Risks to Vegetation

For over twenty years EPA has been developing, defending and enforcing risk assessment-based regulation (U.S. EPA, 1995). To make its risk assessment approach more transparent to those outside EPA, however, the Agency has begun to establish Agency-wide guidance and principles for risk assessment, risk characterization and risk management. As part of this effort, the Agency's 1992 Guidance on Risk Characterization for Risk Managers and Risk Assessors was updated in February, 1995, and on March 21, 1995 the Administrator of EPA signed a "Policy for Risk Characterization". Though both of these documents were designed specifically to address human health, guidance specific to ecological risk will be available in the future. In the meantime, many of the steps involved in a human health risk assessment have parallels in environmental assessments (e.g., hazard identification; dose-response assessment; exposure assessment). Thus, uncertainties regarding human health risk assessment can also be found in techniques for assessing risks to vegetation, e.g., extrapolating from exposure-response functions established in clinical studies or, in the case of vegetation, from open-top chambers.

This section uses and builds upon effects and exposure-response information drawn from the CD with additional information on location of sensitive species and GIS-projected national O_3 air quality to identify potential areas of residual risk to vegetation for crop yield and/or tree seedling biomass loss, under various alternative primary and secondary attainment scenarios. Additionally, this section discusses potential qualitative risks to vegetation including impacts on biodiversity, long-term health of forests and forested ecosystems, aesthetic values, and on the value of vegetation as habitats for birds and other species.

In order to evaluate the present risk to vegetation under ambient air quality, the GIS was used to project seasonal O_3 air quality for a base year, selected as 1990, in terms of the 3 month, 12 h SUM06 exposure index (Figure VII-10). The map for the same scenario shown in Figure VII-10 can be found in Appendix E expressed in terms of W126. Though the uncertainty associated with these air quality projections cannot be quantified, as stated above, in the absence of more complete monitoring data it serves as a useful tool for identifying areas across the country where exposure levels would be expected to exceed those known to produce yield or biomass loss at given levels for crops and trees, respectively.

Figure VII-10



Staff judges that a visual comparison of Figure VII-10 with the 1990 map of monitored SUM06 values greater than 25 ppm-hrs by county (Figure IV-4) in chapter IV shows a good match, increasing staff's confidence in these air quality projections.

Figure VII-10 suggests that under the base year (1990) air quality, a large portion of California and a few localized areas in North Carolina and Georgia have seasonal O_3 levels above that have been reported to produce greater than 20% yield loss in 50% of NCLAN crops and greater than 17% biomass loss in 50% of studied tree seedling species. A broader multistate region in the east is estimated to have air quality sufficient to allow up to 20% yield loss in 50% of NCLAN crops and 17% biomass loss in seedlings, while at least a third of the country, again mostly in the eastern U.S. most likely has seasonal exposure levels which could allow up to 10% yield loss in 50% of NCLAN crops and studied seedlings. Thus, the staff concludes that current air quality is resulting in significant impacts to vegetation.

Maps were also generated for selected "just attain" scenarios (Figures VII-11a,b,c) by analytically adjusting air quality distributions to reflect attainment of various alternative primary standard options (see Horst, R. and M. Duff, 1995). These maps, used in estimating benefits of control, can also depict areas which might experience residual risk after attainment of the standard.

When 1990 air quality is rolled back to attaining the current 0.12 ppm, 1-hour primary and secondary NAAQS, the overall seasonal 12 hr SUM06 exposures improve, but not dramatically (Figure VII-11a). The areas in North Carolina and Georgia previously having exposure levels in the highest part of the range now drop down to the second highest level (25.5 to 38.4 ppm-hr). The area in California showing exposures in the highest part of the range shrinks, with more areas now in the second highest level. A few other areas, including areas in Texas, Louisiana, Oklahoma, Maryland, New Jersey and Delaware also dropped to the third highest range (15.0 to 25.5 ppm-hr) from the second. However, under this attainment scenario, there are still areas of the country judged to have seasonal O_3 levels sufficient to cause greater than (California) or equal to (multistate region in east) 20% and 17% yield or biomass loss in crops and tree seedlings, respectively. Thus, the staff

Figure VII-11a

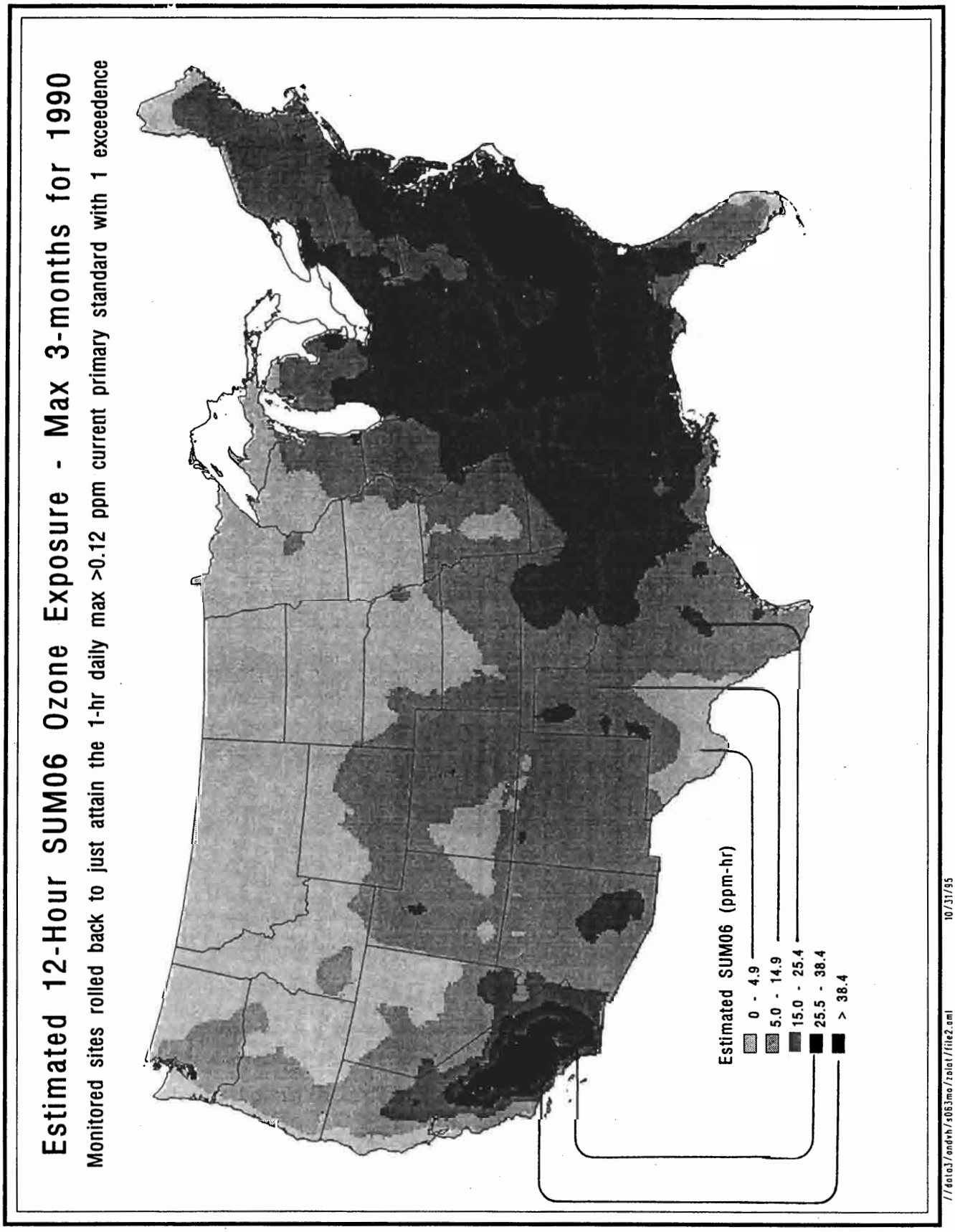


Figure VII-11b

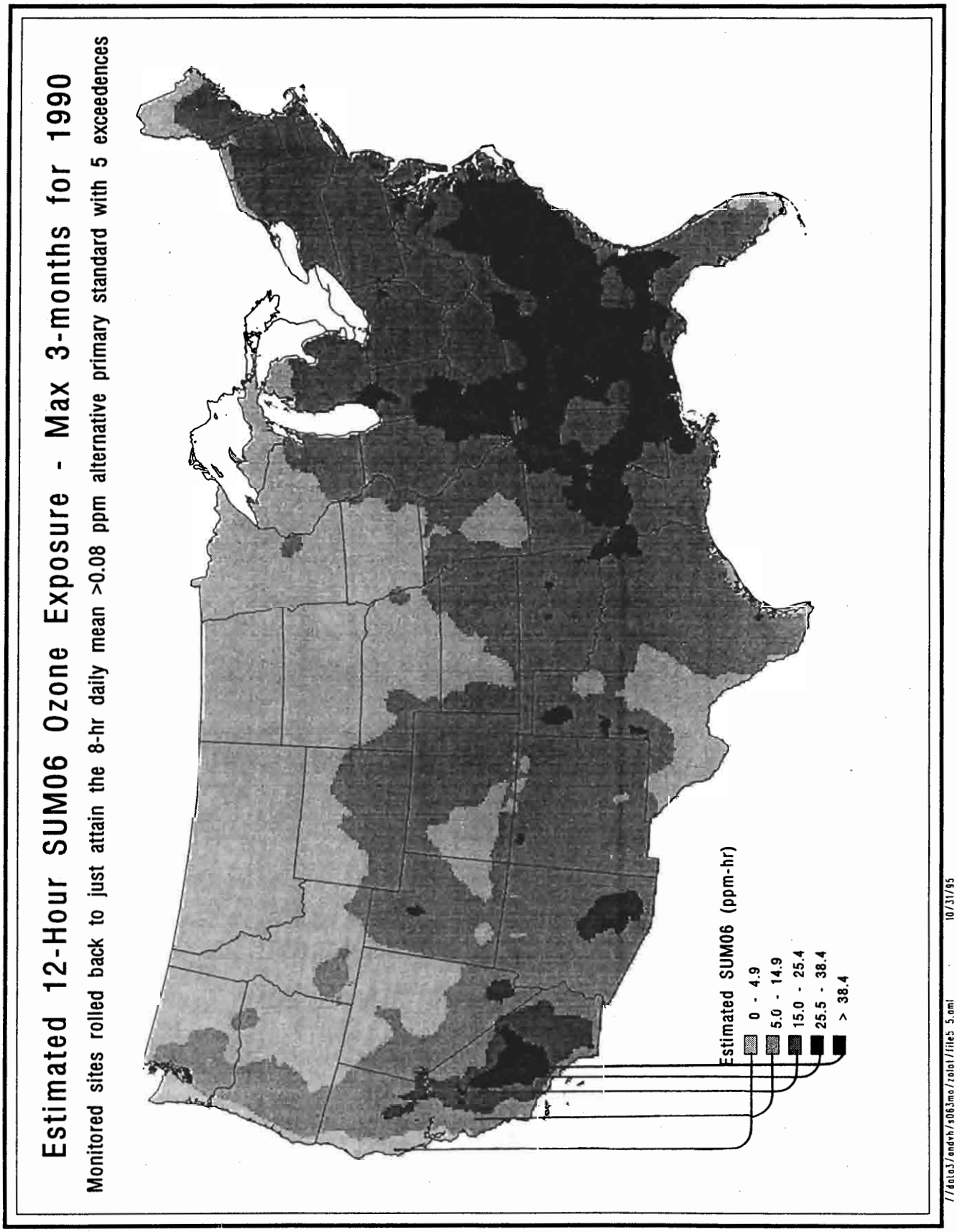
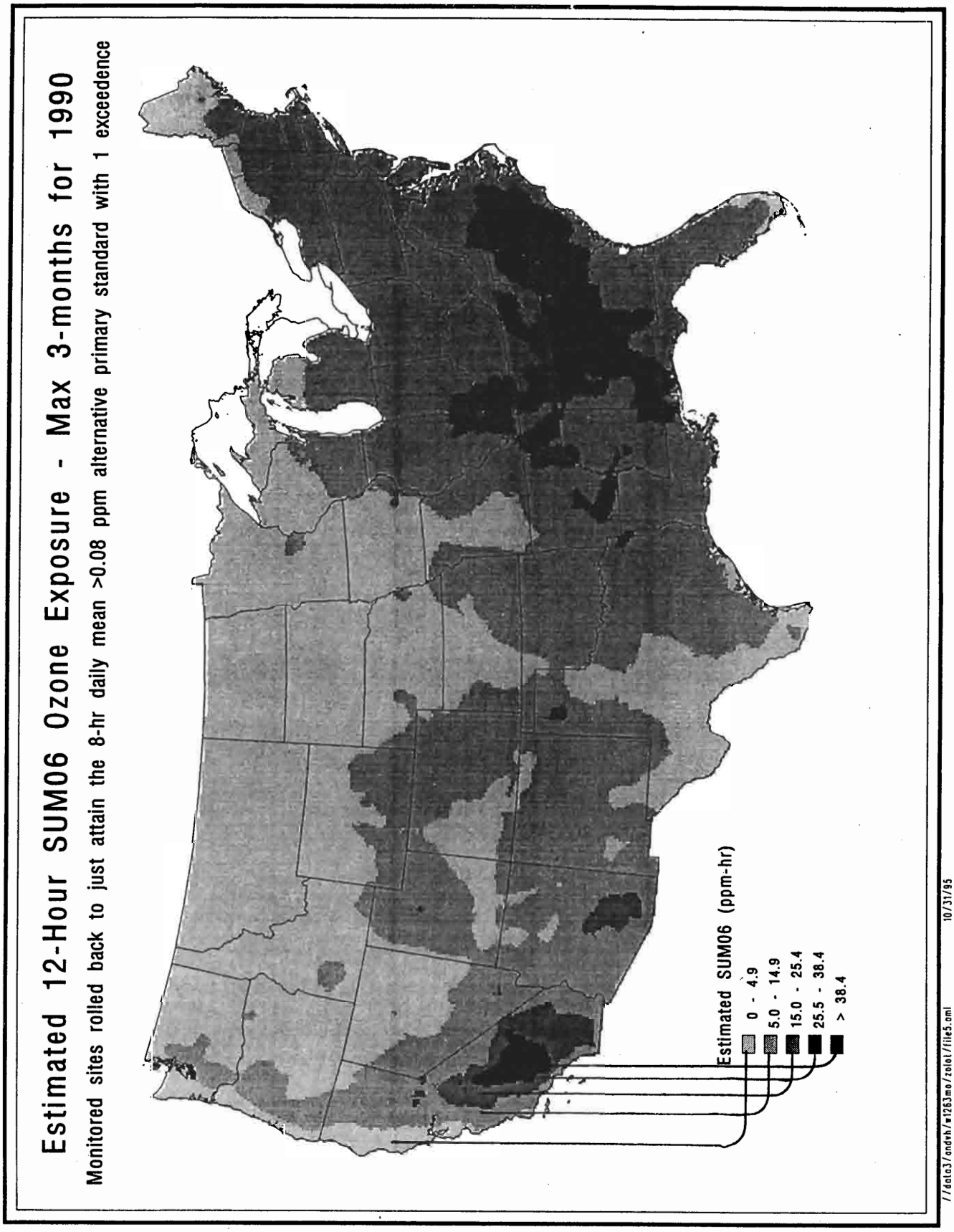


Figure VII-11c



concludes that attaining the current (primary and secondary) NAAQS does not provide adequate protection of vegetation.

To illustrate the impact of recommended alternative 8-hour primary standards, exposure maps have been generated for the 0.08 ppm, 1- and 5-expected-exceedance alternatives. These maps (Figures VII-11b,c) show a markedly improved picture of O_3 air quality compared to Figure VII-11a, with only slight improvements achieved by moving from the 5-to 1-expected-exceedance form. The only state still shown to exhibit seasonal exposures high enough to result in 20% yield loss for crops is California, while the majority of the southeast has been estimated to drop to exposure levels that could allow up to 10% yield and biomass loss in 50% of NCLAN crops and tested tree seedlings, respectively. Thus, the staff concludes that a primary standard of 0.08 ppm, 8-hr, 1- to 5-expected exceedance, when attained at all locations, would be expected to provide significantly improved protection of vegetation from seasonal O_3 exposures of concern.

It remains uncertain, however, the extent to which air quality improvements designed to reduce 8-hour O_3 concentrations would reduce all of the biologically relevant O_3 exposures measured by a SUM06 index. Thus, because 1) the biological database stresses the importance of cumulative, seasonal exposures in determining plant response, 2) plants have not been specifically tested for the importance of the daily maximum 8-hour O_3 concentrations in relation to plant response, and 3) the effects of attainment of an 8-hour standard on seasonal O_3 air quality distributions is still uncertain, these uncertainties should be considered when evaluating the vegetation benefits associated with attaining alternative primary standards.

Quantifiable Risks: Commodity Crops and Fruits/Vegetables. For eight of the NCLAN crops for which exposure-response functions were described in the CD, the 1992 U.S. Department of Agriculture national crop production statistics were used to derive the location and type of commodity crops growing in the U.S. EPA's NHEERL-WED at Corvallis, using the GIS, combined this information with projections of air quality based on 1990 monitoring data as discussed above, to produce 8 maps showing estimated yield loss

individually for each of the 8 NCLAN crops (Appendix E). Yield losses greater than 10% (relative to the baseline of yield at O_3 levels of 0.025 ppm used in the NCLAN project) are estimated to occur in a few areas for soybean, kidney bean, wheat, cotton, and peanut, with lower yield losses estimated for barley, corn, and sorghum. Building on these quantitative estimates, and also considering fruits and vegetables grown in California, section VII-F-3 estimates the economic benefits of reductions in O_3 exposure associated with alternative standards.

Quantifiable Risks: Tree Seedlings. In a process similar to that used for crops above, information on exposure-response functions for biomass loss for seedlings of eleven tree species taken from the CD and information on tree growing regions derived from the U.S. Department of Agriculture's Atlas of United States Trees (Little, 1971) was combined with projections of air quality based on 1990 monitoring data, to produce 11 maps showing estimated biomass loss individually for each of the 11 seedling tree species. These maps (Appendix E) show significant variability in projected seedling biomass loss under 1990 air quality conditions across the species studied. For example, for the most sensitive species studied, black cherry, seedling biomass loss is projected to be greater than 30% for over half of its geographic range, though it can reach as high as 44% in as much as 10% of its growing area. Tulip poplar seedlings are predicted to have a median area weighted biomass loss of 9%, though more sensitive strains growing in 10% of its geographic range may experience up to 18% biomass loss. The less sensitive white pine and aspen seedlings reach a projected biomass loss up to 10% for 10% of the growing region but only 2-3% over 50% of their mapped area. Sugar maple and ponderosa pine seedling losses range from a maximum of 3-4% in approximately 10% of their growing region though on average they experience only 1-2% biomass loss. Biomass loss estimates for the least sensitive species studied, red alder, Douglas fir, Virginia pine, and red maple seedlings, are projected to be less than 2% in all areas. These findings again show the wide variability in seedling sensitivities, both inter- and intraspecific. Though these maps show the geographical range for each species, they do not indicate that each species will be found at every point within its range. It should also be recognized that the production of these maps incorporates several separate sources of uncertainty, beginning with the exposure-response functions produced for

seedlings in open-top chambers to the uncertainties associated with the inputs to the GIS and projecting to the national level. Furthermore, percent biomass loss in tree seedlings is not intended to provide any information on expected biomass loss in mature trees of the same species, nor can it be considered comparable to percent yield loss in agricultural crops. The latter is because a 1-2% biomass loss per year in perennial species, if compounded over multiple years of exposure could become severe, while the same percentage yield loss in crops annually would not be significant.

Uncertainties in Quantitative Risk Assessment The uncertainties in exposure-response functions, experimental procedures, air quality data, and the use of GIS technology are all highlighted above. The combination of these elements in the risk assessment process itself, however, produces additional uncertainties that are discussed here. First, map interpretation can be a major source of error in perception because maps showing the geographical range for each species do not indicate that this species will be found at every point within its range. For example, maps projecting estimated biomass reduction for individual tree species may be interpreted by some as suggesting a homogeneously dense mono-culture forest, which is not the case. Furthermore, maps of species ranges do not reflect where a particular species is a key component in an ecological system, or where a particular species is economically important to the region. The AVHRR satellite data was used to mask out urban and agricultural areas, but potentially other factors influence the presence/absence of a species within the range defined by Little (Little, 1971). These include islands of high elevation which might not be conducive to that particular species or mono-culture tree plantations of different species (Hogsett, et al., 1995). However, given these uncertainties, the estimated seedling biomass losses represent potential risks that species may experience, affecting seedling establishment, reforestation or natural regeneration.

Qualitative Risks: Crops/Fruits/Vegetables/Urban Ornamentals. Beyond yield loss effects, commercial crops, including fruit and nut species, may be at risk of other indirect effects from O_3 , such as shifts in their relationships with pests and pathogens and reduced biodiversity. For example, breeding programs designed to improve yield could inadvertently be selecting plants with either greater or reduced O_3 tolerance. Several cases cited in the CD show that cultivars grown in areas with high O_3 levels are more tolerant than their

counterparts developed elsewhere (Reinert et al., 1982, Roose et al, 1982). Velissariou et al. (1992) suggested that selection for higher yields (and higher stomatal conductance) had resulted in a higher O_3 uptake for modern spring wheat cultivars, contributing to their increased O_3 sensitivity. It is therefore possible that the persistence of O_3 in crop growing regions may result in a reduction in genetic diversity of crop cultivars available, together with the loss of other beneficial traits that may be linked genetically with O_3 sensitivity. Tolerance mechanisms based on reduced stomatal conductivity would likely reduce growth of tolerant plants, while tolerance based on the production of antioxidant compounds would likely shunt plant resources away from growth to the production of the defense compounds. Such indirect effects may also occur in plants used in urban landscaping and gardens, both for aesthetics and for other purposes such as shade/cooling, animal/songbird habitat, soil retention, and commercial florists.

Qualitative Risks: Trees/Commercial Forests/Forested Ecosystems/Class I Areas. As previously discussed, methods have not at this time been adequately developed to scale biomass loss effects in seedlings to effects in mature trees. However, field observations of seedling health and mortality can provide information relevant to assessing risks to trees and forests. For example, field plot observations of seedling health and mortality in natural giant sequoia groves over a 4-year period showed that seedling numbers were reduced drastically from drought and other abiotic factors. Ozone injury symptoms were also observed in the weeks following germination. These observations suggest that O_3 could be stressing seedlings sufficiently to reduce root growth immediately after germination, thus increasing vulnerability to late summer drought.

The above observations have a counterpart in commercial tree nurseries where the importance of root development as an indicator of plant health has long been known. Toumey and Korstian (1947) stated that "in judging quality of nursery stock much greater emphasis should be placed on number, size, extent, and conditions of roots than on appearance above ground." Several more recent studies conducted at nurseries have investigated the relationship between the number of first order lateral roots/seedling (FOLR) and seedling quality, competitiveness, and survivability. For example, nursery studies of sweetgum and loblolly pine seedlings suggested that very early adverse impacts on root

growth that could result in part from O_3-related effects, can have significant impacts in growth and competition in forest environments in subsequent years (Kormanik, 1986; Kormanik et al., 1990). These more recent studies lend support to the suggestive earlier observations.

The importance of below-ground effects on trees, forests, and ecosystems has often been overlooked when evaluating responses to O_3 exposure. The below ground system is dependant on the above ground system for inputs of energy containing substrates. Mycorrhizal fungi invade the roots of the vast majority of terrestrial plants and assist the host plant in the uptake of nutrients and water, protect the roots against pathogens, produce plant growth hormones, and transport carbohydrate from one plant to another. In exchange, the roots of the host plant provide the fungi with simple sugars. This symbiotic relationship is especially beneficial to plants growing on nutrient poor soils, and contributes substantially to ecosystem function. As discussed in section VII.B, O_3 stress inhibits photosynthesis and reduces the amount of sugars available for transfer to the roots. Reduction in available sugars in the roots can alter mycorrhizal colonization and compatibility, reducing mycorrhizal formation and root growth. Berry (1961) examined the roots of eastern white pine injured by O_3 and observed that healthy trees had almost twice the percentage of living feeder roots as trees with O_3 injury. In the San Bernardino forest in California, Parmeter et al. (1962) observed that the feeder roots system of ponderosa pine exposed to O_3 showed marked deterioration (US EPA, 1986). Numerous other studies cited in the CD have documented the reallocation of carbohydrates away from roots to photosynthetically active portions of the plant as a result of O_3. For example, Spence et al. (1990) found a reduction in transport of photosynthates to roots in O_3-treated loblolly pine, and Edwards (1991) reported reduced root and soil respiration.

Beyond biomass loss and impacts on root systems, other risks to vegetation associated with O_3 include shifts in the relationship between tree species and insects or pathogens which can result in imbalances within communities that may have long-term effects. Significant risks could result, such as has been observed in the San Bernadino forest as discussed in section VII-D. Ozone effects can also reduce the ability of affected areas to provide habitats to endangered species. For example, two listed endangered plant species, the spreading aven

for commodity crops and fruits and vegetables for the 8-hour, 0.08 ppm alternatives. Additional protection from alternative secondary standards beyond that estimated for the alternative primary standards is generally estimated to be relatively small, with the degree of incremental protection decreasing as the stringency of the alternative primary standards increase. Further, of course, this incremental protection varies with the stringency of the alternative secondary standards, ranging to close to zero in many cases especially for the least stringent alternative considered. Thus, staff concludes that, when considered in conjunction with the protection provided by both primary and secondary standards, consideration of the level for a secondary standard should focus on the more stringent alternatives where some degree of incremental protection would more likely be expected.

More specifically, staff concludes that a secondary standard with a 3-month, 12-hour, SUM06 form, set at a level within the range of approximately 38 - 25 ppm-hr (corresponding to the 20% and 10% yield loss protection levels for 50% of the NCLAN crops, respectively), would provide substantial protection against vegetation effects. Though these levels would not be expected to protect the most sensitive species or individuals within a species, when considered in conjunction with the recommended alternative primary standards, such a secondary standard would be expected to provide some degree of incremental protection beyond that provided by recommended alternative primary standards. Although staff judges that this degree of incremental protection may be small at the national level, staff believes that it could be potentially significant at regional or local levels.

In addition to the more extensively studied effects associated with O_3 exposure such as visible foliar injury and decreased growth or yield in short-lived species, the available information further points to more subtle impacts of O_3 acting in synergy with other natural and man-made stressors to adversely affect individual plants, populations and whole systems. By disrupting the photosynthetic process, decreasing carbohydrate storage in roots, increasing early senescence of leaves and affecting water use efficiency in trees, O_3 exposure can disrupt or change the nutrient and water flow of an entire system. Weakened trees can become susceptible to pest and pathogen outbreaks, loss of competitive advantage and decreased reproductive (seedling survivability) success, perhaps resulting in reduced genetic variability within the species or entire ecosystem. However, staff concludes that there is

insufficient information to estimate the severity of these impacts as a function of the levels of alternative secondary standards, and, thus, no quantitative estimate has been made in the context of this review of the potential benefits associated with alternative standard levels. This information, however, should be weighed in considering the extent to which a secondary standard should be precautionary in nature in protecting against effects for which scientific studies have not adequately quantified exposure-response relationships.

E. Recommendation

Based on the conclusion that the current secondary standard does not provide adequate protection for vegetation, the staff recommends that the Administrator consider the following factors in determining appropriate revisions to the secondary standard: 1) the varying degrees of protection afforded by the alternative primary standards recommended in Section VI; 2) the incremental protection associated with alternative cumulative, seasonal secondary standards under consideration; and 3) the value of establishing a seasonal form for the secondary standard that is more representative of biologically relevant exposures. Additional consideration should be given to the possibility of ozone impacts acting in synergy with other natural and manmade stressors and the extent to which a secondary standard should be precautionary in nature against such effects, particularly given their potential significance at a regional scale and in Class I areas.

If the Administrator determines that additional protection is needed beyond that provided by the alternative primary standards recommended in Section VI (or that no revisions to the current primary standard are warranted) and/or that establishing a seasonal form for the secondary standard is justified, staff recommends consideration of a new secondary standard in the form of a 3-month, 12-hr, SUM06 exposure index, set at a level within the range of approximately 38 to 25 ppm-hrs. The upper end of this range focuses on providing additional protection against effects to a wide range of commercial crops and tree species that can be most clearly attributed to ambient O_3 concentrations above background levels. The lower end of this range would be expected to provide further incremental protection for commercial crop and tree species, while directionally providing increased protection against effects to vegetation and ecosystem resources in Class I and other areas.

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APPENDIX A

Air Quality Assessment

APPENDIX A: AIR QUALITY ASSESSMENT

This Appendix characterizes O_3 air quality status and trends for the current 1-hour O_3 National Ambient Air Quality Standard (NAAQS). Air quality patterns and data handing conventions for possible new alternative O_3 primary and secondary NAAQS are also presented. Emphasis is placed on air quality comparisons among alternative forms of O_3 standards, especially within the current range of levels of concern.

O_3 Trends

In 1993, hourly O_3 measurements made at 925 ambient air quality monitoring sites were reported to EPA's Aerometric Information Retrieval System (AIRS). Most of these sites are located within, or near, metropolitan areas. To account for the seasonal O_3 pattern and to accommodate differences in local climates, the EPA has designated specific "O_3 seasons" in each state consisting of a contiguous set of months during which minimal ambient air quality monitoring requirements must be met. In southern locales, the O_3 season spans all 12 months, while in northern states such as Montana, the monitoring season spans only the summer months June through September.

Figure A-1 displays the 10-year trend, 1984-93, in the composite average and the inter-site variability of the annual second highest daily maximum 1-hour concentration at 532 trend sites. Only those sites with at least 75 percent data completeness during the O_3 season for at least 8 of the 10 years were selected as trend sites. The 1993 composite average for these 532 sites is 12 percent lower than the 1984 level. The 1993 composite average is higher than the 1992 level, which was the lowest composite average of the past 10 years. The 1993 composite average is still the second lowest level during the past ten years. The entire concentration distribution in 1992 is also lower than any other year. The increase in the composite average between 1992 and 1993 is not statistically significant.

Figure A-1. National trend in the annual second highest daily maximum 1-hour concentration at 532 sites, 1984-93.

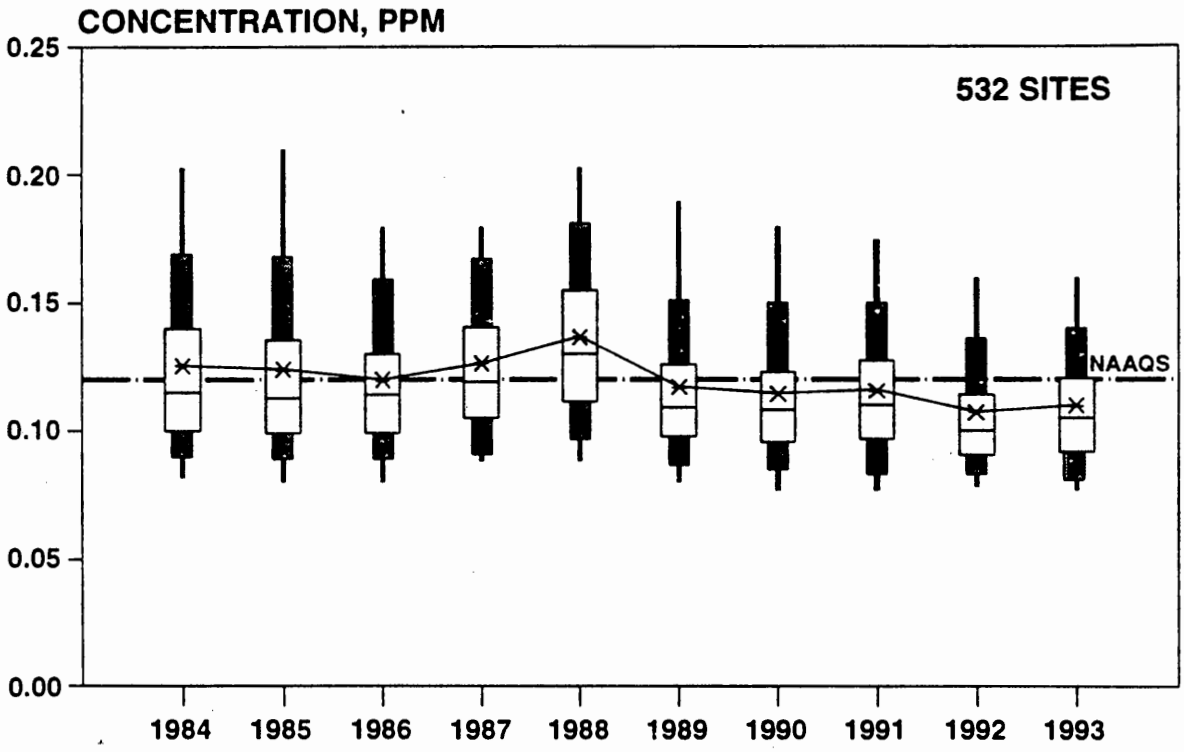


Figure A-2. National trend in the annual maximum Sum06 index value by county, 1987-93.

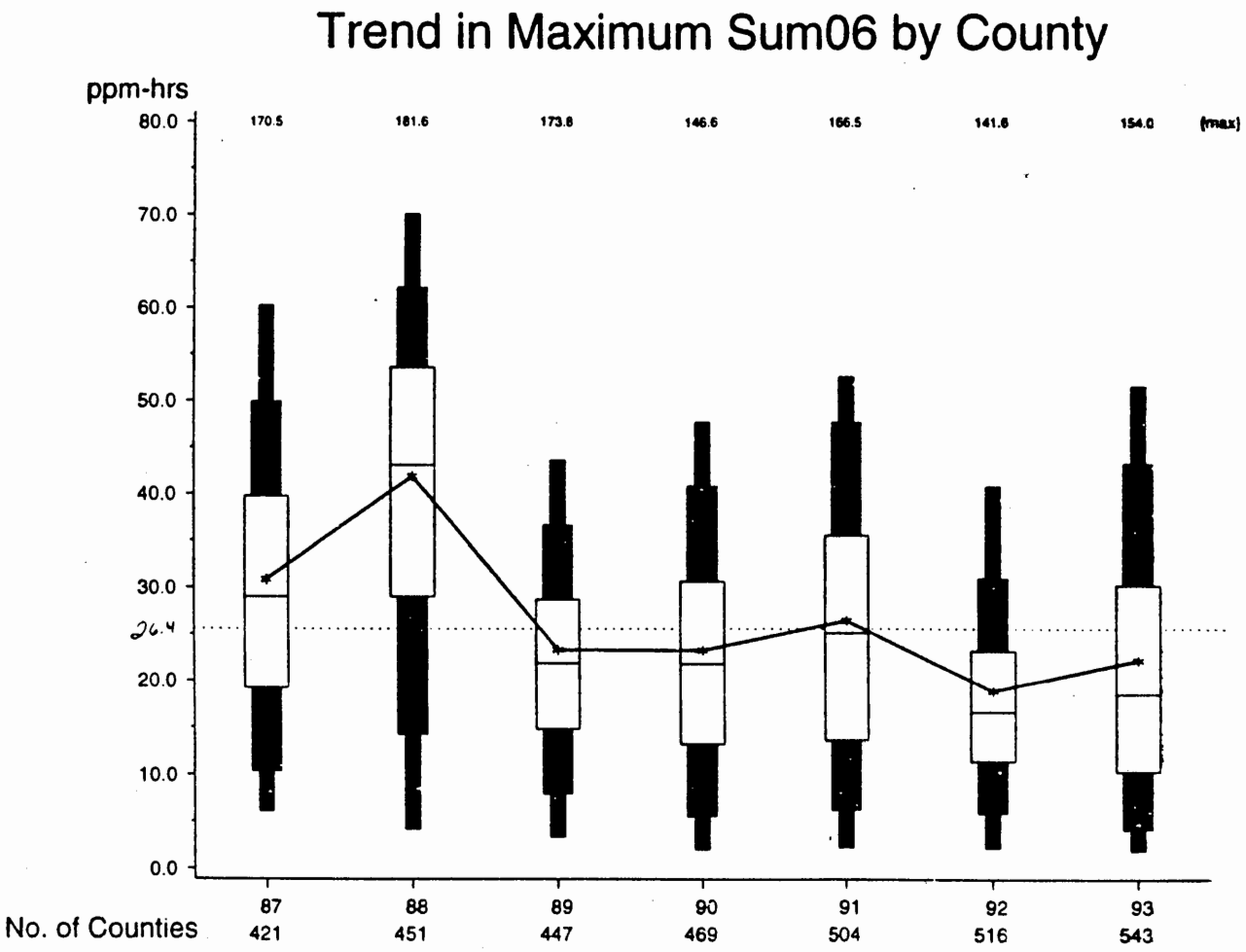


Figure A-2 presents the trend in the annual three-month maximum Sum06 value by county for the years 1987-93. Trends in exposure indices under consideration for possible secondary standards, such as the Sum06 index, exhibit temporal patterns similar to the annual second maximum hourly concentrations described above.

The interpretation of recent O_3 trends is difficult due to the confounding factors of meteorology and emission changes. Just as the increase in 1988 is attributed in part to meteorological conditions that were more conducive to O_3 formation than prior years, the 1992 decrease is due in part, to meteorological conditions being less favorable for O_3 formation in 1992 than in other recent years (Brown et. al., 1992). Meteorological conditions in 1993 were once again more favorable to O_3 formation, especially in the east and southeastern areas of the country (Brown, 1993) although the magnitude and frequency of O_3 concentrations above the NAAQS levels were significantly less than 1988. Also, since the peak year of 1988, the volatility of gasoline has been reduced by new regulations which lowered national average summertime Reid Vapor Pressure (RVP) in regular unleaded gasoline (EPA, 1989).

Year-to-year meteorological fluctuations and long-term trends in the frequency and magnitude of peak O_3 concentrations can have a significant influence on an area's compliance status. Table A-1 presents the number of areas not meeting the current O_3 NAAQS for three compliance periods. The first compliance period, 1987-89, contains the 1988 peak O_3 year and corresponds to the initial nonattainment area designations under the Clean Air Act Amendments of 1990. By 1990-1992, both as a result of more favorable meteorology (i.e., less conducive for O_3 formation) and reductions in emissions, the number of areas not meeting the O_3 NAAQS has decreased significantly.

Table A-1. Number of areas not meeting the current O_3 NAAQS.

Compliance Period	Number of Areas not meeting the O_3 NAAQS
1987-89	98
1990-92	52
1991-93	43
1992-94	33

Data Handling Conventions

If the current 1-hour standard is replaced with a standard based on an 8-hour averaging time, it will be necessary to specify some initial data handling conventions for the 8-hour averages. O_3 data are reported on an hourly basis, so that it is possible to compute a running 8-hour average O_3 concentration for each hour of the year (in the case of complete or nearly complete data). For the analyses presented in this Appendix, daily maximum 8-hour averages were considered valid if at least 18 hourly values were present during the day.

Figure A-3 shows a hypothetical example of the relationship between the 1-hour data and the 8-hour averages. The individual bars correspond to the hourly values and the line shows the corresponding 8-hour averages. There are a few technical points worth noting. When an 8-hour average is computed, it can be associated with the start hour, or the end hour of the 8-hour period, or some intermediate hour. The convention used in Figure A-3 is to have the 8-hour average identified by the start hour of the 8-hour period. For example, the 8-hour average from 4 p.m. to midnight would be plotted at 4 p.m. It should be noted that the next 8-hour period, from 5 p.m. to 1 a.m., would be plotted at 5 p.m. but it contains hourly values from two different days. If the 8-hour daily maximum is selected from

all 24 8-hour averages starting within the day, it is possible to have daily maximums from adjacent days that actually have some hourly values in common. This is unlikely for the typical urban diurnal pattern shown in Figure A-3 but it can occur for sites with less pronounced diurnal patterns, such as rural mountaintop sites.

Another point to note with 8-hour averages is that the 8-hour daily maximum for a particular day can actually be higher than the 1-hour maximum. Again, this would be uncommon but can happen because the 8-hour average could contain up to seven hourly values from an adjacent day. Figure A-4 shows 8-hour averages for three days in July 1988 at a rural mountaintop site in southeast Virginia illustrating these points. The daily maximum for July 7 is higher than any of the 1-hour values for that day because it is driven by the high hourly values in the early hours of July 8. Again, it should be noted that this particular rural site departs significantly from the classical O_3 diurnal pattern in urban areas.

Figure A-3. 1-hour O_3 data and 8-hour averages. Hypothetical urban area, three days in July.

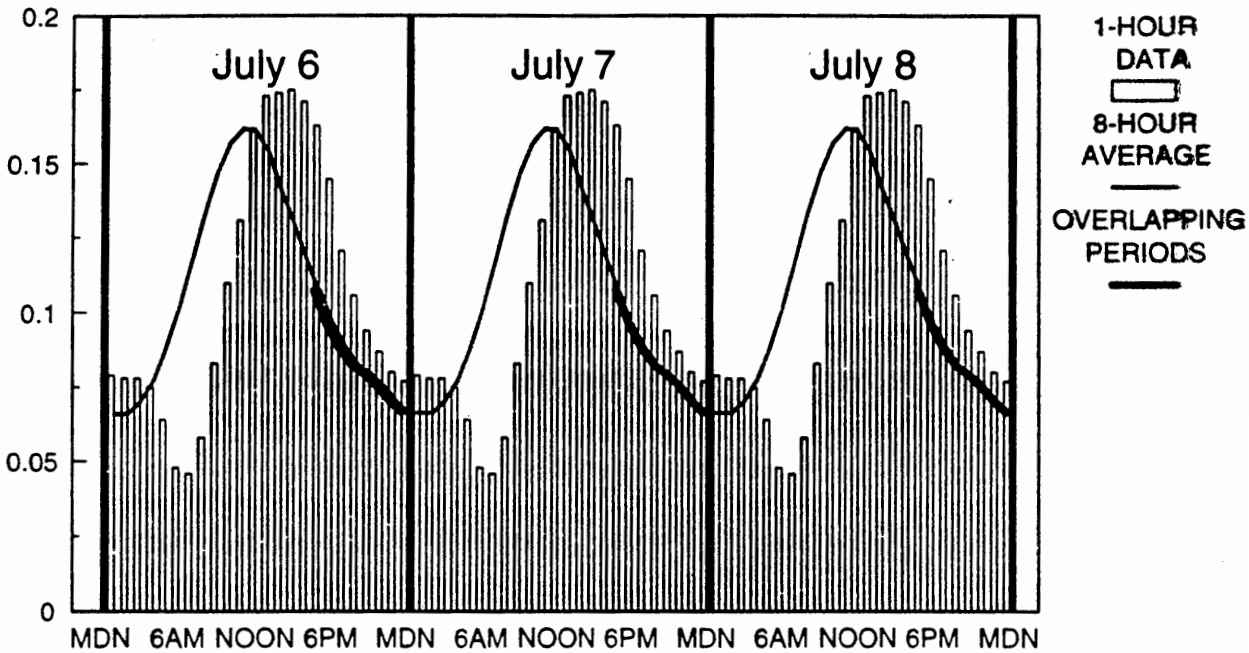
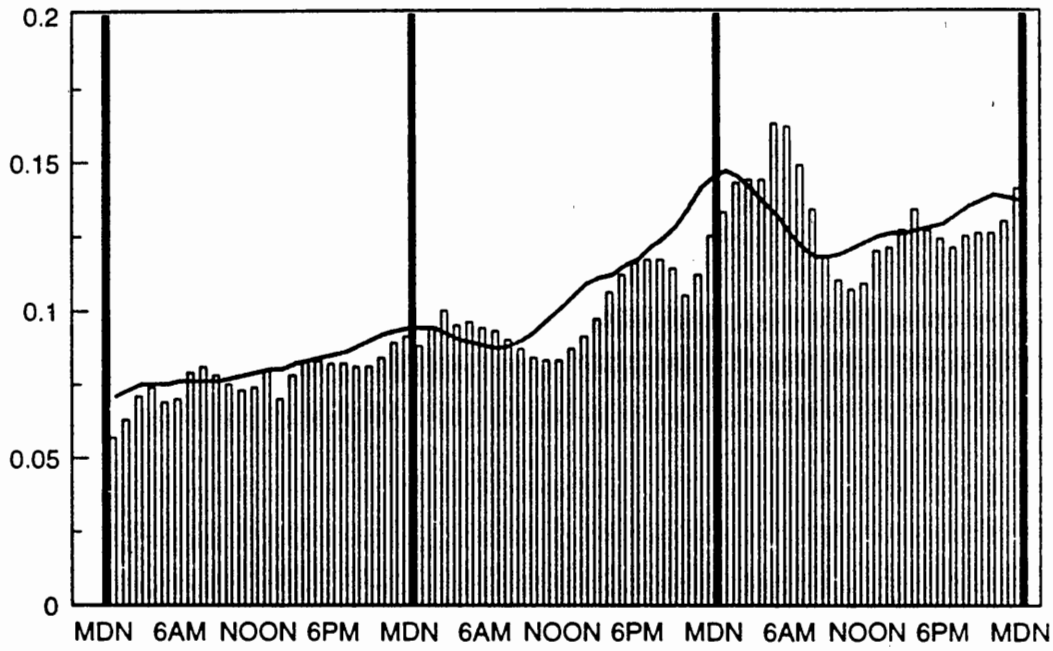


Figure A-4. 1-hour O_3 data and 8-hour averages. Smyth County, Virginia, July 6-8, 1988.



Monitoring Considerations

Implementation of an 8-hour or cumulative index standard would require the monitoring community to examine appropriate siting criteria for the new standard forms. The current O_3 monitoring network was designed to address a 1-hour standard. While extension to an 8-hour standard may require few changes, the current network may not be adequate for a cumulative index standard, particularly if there is a need for more rural monitoring stations. A preliminary analysis of 192 urban areas (Consolidated Metropolitan Statistical Areas and Metropolitan Statistical Areas, CMSAs/MSAs) for the 1990-92 period found that the 1-hour and 8-hour "design value" sites differed about 17 percent of the time. The peak SUM06 site differed from the peak 1-hour site about 28 percent of the time.

Distributions of hourly O_3 data

The distributions of hourly O_3 concentrations were examined at different monitoring environments. Sites with relatively complete data located in rural/agriculture, background, urban, and high elevation environments were selected. The data were analyzed using the MAXFIT program (Fitz-Simons et al., 1979) which fits 8 distributions: the normal, the 3-parameter log-normal, the Box-Cox distribution, the Johnson S_B, the 3-parameter gamma, the 4-parameter beta, the 3-parameter Weibull, and the extreme value distribution. Figures A-5 through A-8 display the frequency distributions of the hourly concentrations and best-fit distributions for an example site in each category.

The urban site shown in Figure A-5 is located at Taft High School in Chicago. For the hourly data, the normal and extreme value distributions gave the best fit.

The high elevation site shown in Figure A-6 is in Albuquerque, New Mexico. The normal distribution did about as

well as any for these data. The data at the lower values almost exhibited a uniform distribution.

The background environment site shown in Figure A-7 is located at Clemson University in South Carolina. The hourly data histograms resembled the hourly data for the Albuquerque site. The normal distribution produced the best approximations or fit for these data.

The Percy Priest Lake Visitor Center near Nashville Tennessee shown in Figure A-8 represents a site in a rural/agriculture-forest environment. The hourly data which are skewed, and have a long tail toward the higher concentrations, were fit best by the normal and the Box-Cox transformation.

It is clear from the above results that there are differences across monitoring environments in the distribution of hourly O_3 concentrations. Nevertheless, some interesting observations come to light from this exercise. One, the normal distribution displayed good fitting characteristics more often than one would think since the data are usually considered to be too skewed for the normal distribution. Two, the extreme value distribution did not consistently yield the best fit on the 1-hour and 8-hour daily max data. Finally, the sites selected through the process described above did not record very large O_3 concentrations during recent years. One would expect sites with high values of O_3 to exhibit data with a more pronounced coefficient of skewness.

Figure A-5. Hourly O_3 concentration distribution at an urban monitoring site.

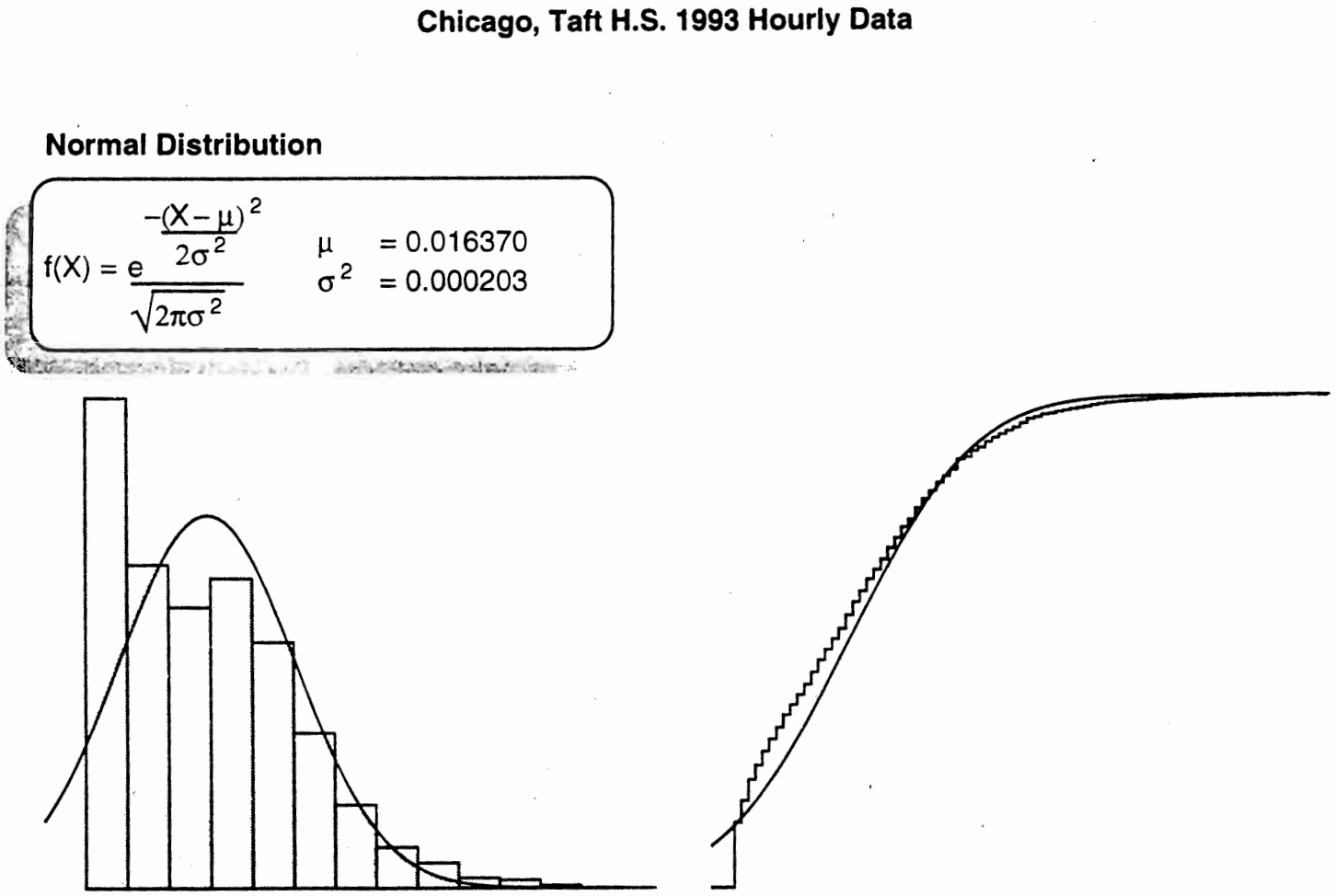


Figure A-6. Hourly O_3 concentration distribution at a high elevation monitoring site.

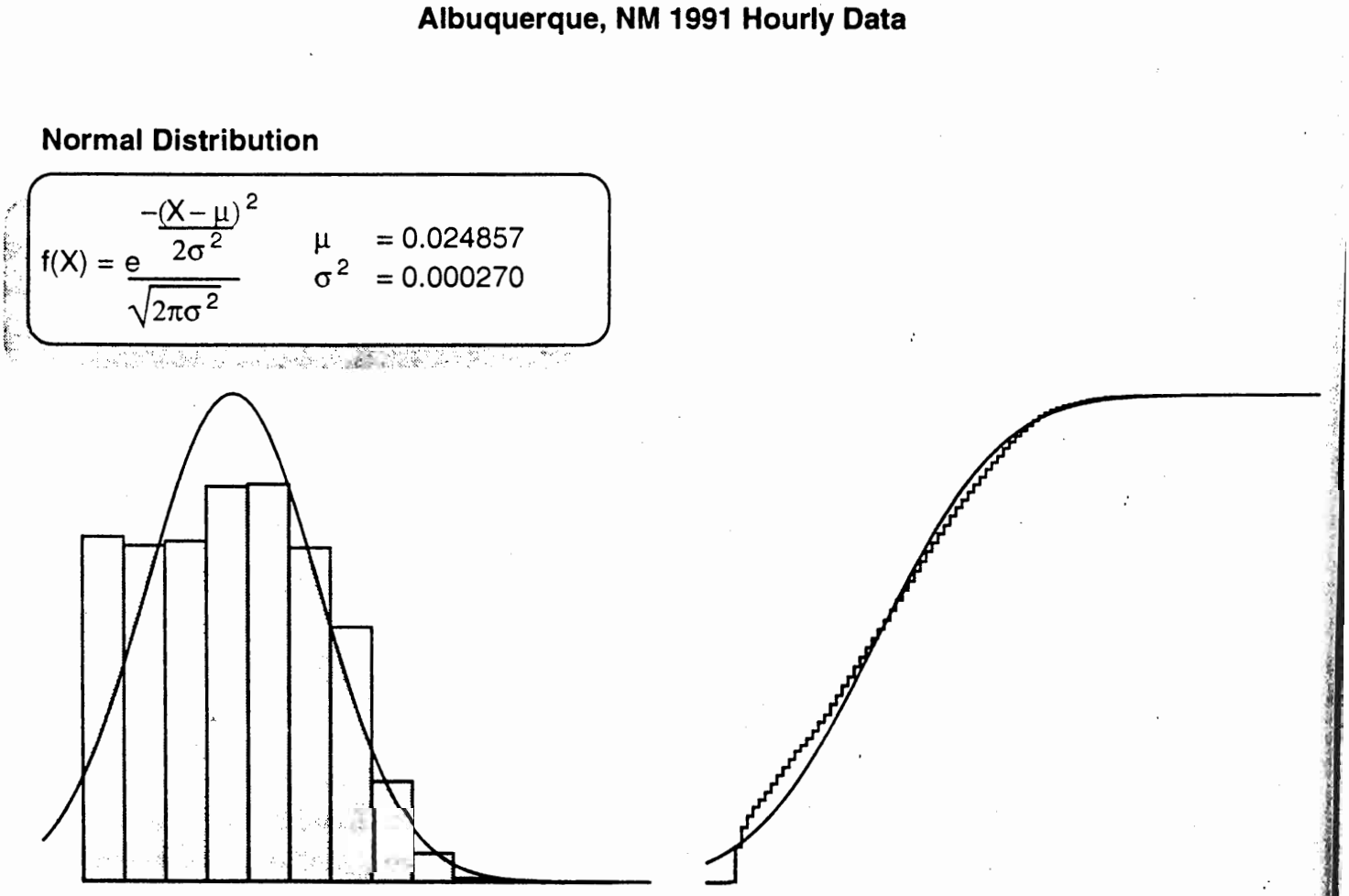


Figure A-7. Hourly O_3 concentration distribution at an urban background monitoring site.

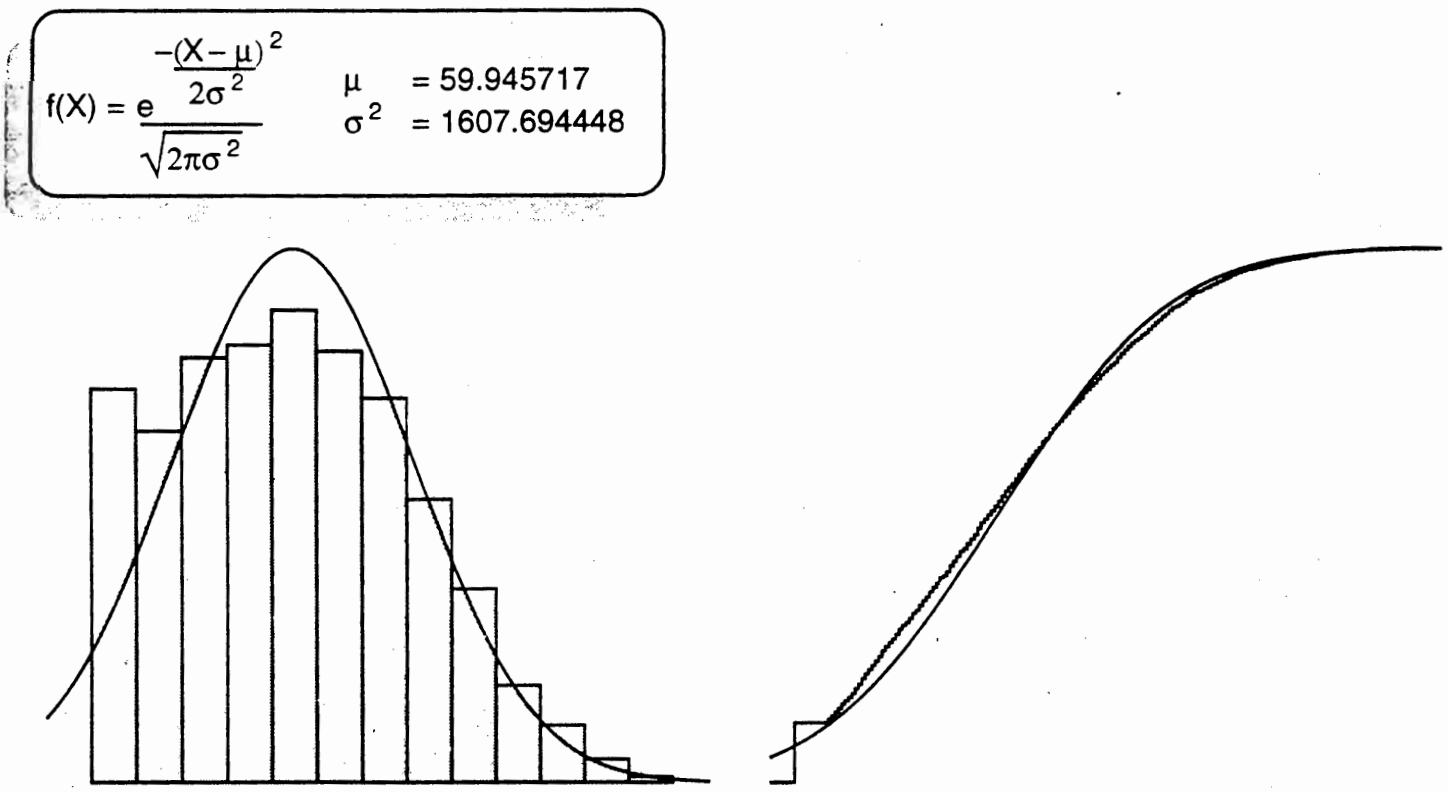
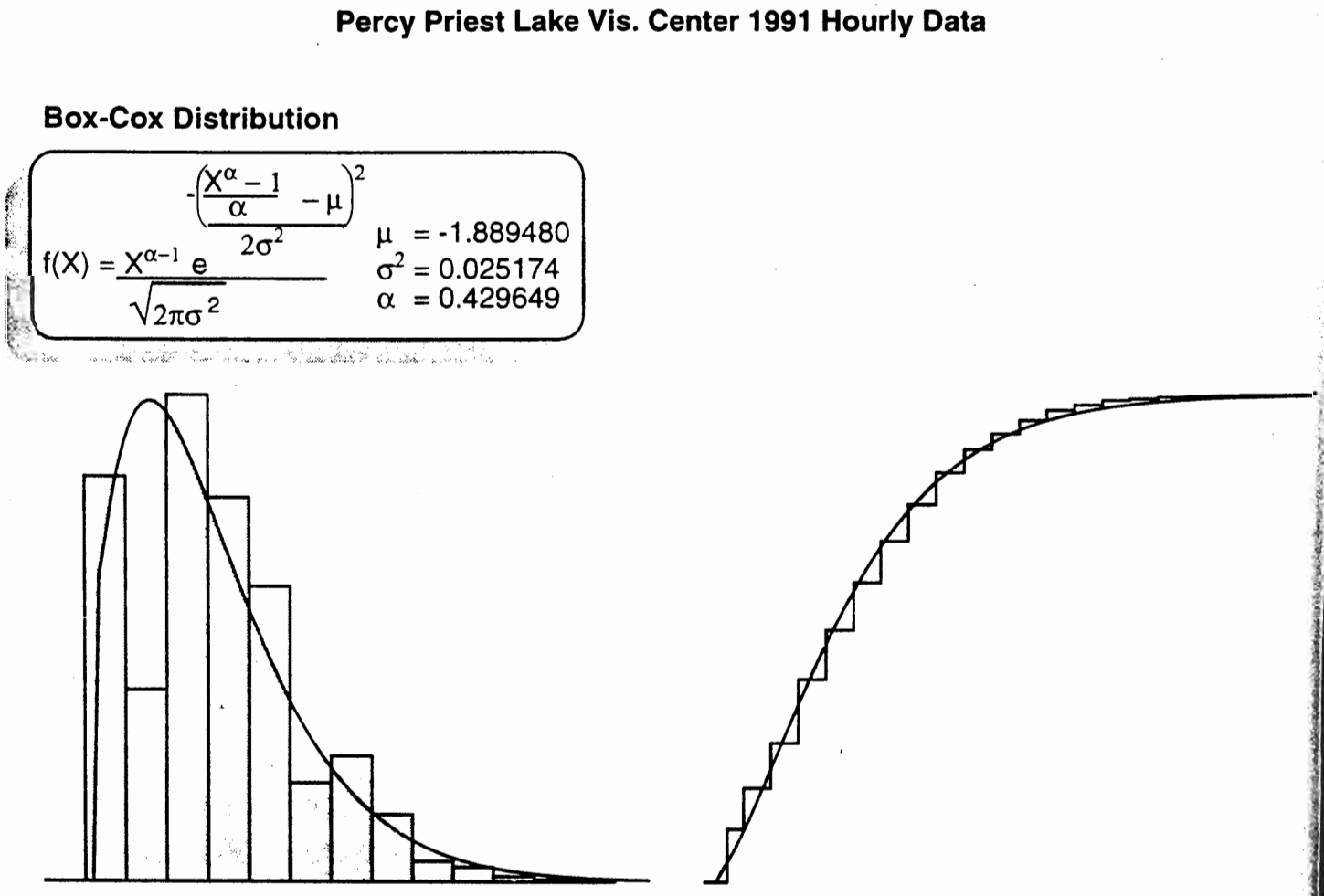


Figure A-8. Hourly O_3 concentration distribution at a rural monitoring site.



Air Quality Data Base for Standards Comparisons

Design values consistent with each of the alternative standards were calculated for each county with sufficient monitoring data. Design values are used in these comparisons since, although they are based on the form of the standard, they are independent of the level of the standard. The design value is that concentration that when reduced to the level of the standard ensures compliance with the standard. For example, the design value for a daily maximum 1 exceedance per year standard is the fourth highest daily maximum concentration given a three year compliance period. If the fourth highest day during three years is reduced to the level of the standard, then there will be exactly three days above the level of the standard, or one day per year on average. Similarly, if the standard were expressed as the average annual 2nd highest daily maximum concentration over three years, the design value is just that same average annual second highest daily maximum concentration. Thus, design values enable one to map complex standard forms into to single number for ease of comparison.

These design value estimates used all hourly O_3 concentration data available on AIRS for the years 1987-93. One-hour data were processed according to the conventions for the current O_3 standard. That is, the design value for a 1 exceedance standard is the (n^{th} +1) largest value, where "n" is the number of years meeting the annual 75 percent data completeness requirement. Eight-hour averages were computed from the 1-hour data and associated with the start hour. An 8-hour average had to have at least six hourly values. The daily maximum 8-hour average is the highest of the 24 possible 8-hour averages starting within the calendar day. These 8-hour daily maximums were then processed using the same conventions analogous to those for the 1-hour data. For the 5 exceedance option, these basic principles were extended so

that the sixth highest value would be used if only one year of data were available, the 11th if two years were available, and the 16th if all three years were valid.

The design values for the three cumulative index alternatives, Sum06, W126, and AOT06, were based upon the highest consecutive three-month value for any calendar year in the 1991-93 period. A index total was computed for each day and then totaled for the month. Daily index values were considered valid if the day had at least 18 hourly values. A month had to have at least 50 percent valid days for the month to be considered valid.

Missing Data Considerations for Secondary Standards

A missing value adjustment was used to scale up index values for a month by simply multiplying the observed index by the ratio of days in the month divided by days with valid data. A preliminary analysis of the impact of missing data on index values was conducted. Sites were selected for four groups - rural/forest and rural/agriculture, high elevation, background, and urban and city center. These sites were selected from AIRS based on the location-setting code, land use code, elevation, and monitoring objective code. Rural/forest and rural/agriculture were selected including areas classified as desert or blighted areas. However, most sites are forest and agricultural. The high elevation sites were selected by limiting the site elevation to above 1000 meters. The background sites were selected on the basis of their monitoring objective.

All subsets of sites exhibited the same patterns of missing data. All groups had a large number of single missing hours. These are mostly the missing hour each day for calibration purposes. For longer gaps, the frequency falls off exponentially as gap length grows larger. The starting hour for a series of missing data seems to have no pattern in all groups. There are some hours in the background sites that

seem to have a larger percentage, although this is likely due to chance since there a much smaller number of sites falling in this category. Site maintenance seems to be standard across different groups of sites and we can expect to see the same patterns of missing data in rural areas as in urban areas.

A site-year combination was selected that had a very high data capture (Tucson, AZ). This site then had gaps of data systematically produced and all secondary standard alternative indicators were calculated and adjusted for missing data. These were compared to the same indicators calculated without any data taken out and the relative error was calculated as:

$$RE = \frac{SA - SU}{SU}$$

where RE is the relative error, SA is the standard calculated using data with gaps and SU is the standard calculated with all data. The relative error was averaged over several data sets where gaps were shifted across the critical months used to calculate alternative indicators for the secondary standard. The results appear below.

Table A-2. Relative Errors

Gap Size	Sum06	AOT06	W126
10	0.0118	0.0030	0.0086
50	0.0201	0.0014	0.0124
100	0.0221	-0.0048	0.0120
200	0.0571	0.0360	0.0316
300	0.0842	0.0818	0.0508

The AOT06 indicator option seems to have a lower average

relative error until the gaps become larger than 100 hours in length. The SUM06 indicator seems to be the most sensitive at all levels.

Multi-year Compliance Considerations for Secondary Standards

In the air quality comparisons that follow, it is important to note that use of the maximum year in the three year compliance period interpretation for the secondary standard index options is a mixture of the old "once per year" standard and the newer "expected exceedance" standards. These maximum cumulative exposure indices do not represent a multi-year average so it differs from the form of current O_3 NAAQS. A single year compliance test is consistent with providing protection for annual species. However, if only a single year is used then, for all practical purposes, by the time the decision is made to declare an area nonattainment, the data from the next year are almost complete and might reverse the decision. To avoid this situation, earlier attainment decisions for these types of standards required eight consecutive quarters of data to show attainment (EPA, 1978). Three calendar years were selected for the compliance period for consistency with the primary standard. The 1991-93 air quality data were used to compare the difference between the maximum three-month index value and the three year average of the annual maximum three month index values. The Sum06 value in the maximum year was at least 24 percent larger than the average index value across the three years for half of the counties with monitoring data. The maximum was greater than the average by more than 22 percent for W126, and 29 percent for AOT06, for half of the counties with data.

Air Quality Comparisons

Using the ambient air quality data base and the alternative primary and secondary NAAQS, the staff estimated

the number of counties and metropolitan areas which would meet, or fail to meet, these standards based on data for 1987-89 and 1991-93. These two 3-year periods contrast the impact of 1988, and varying weather patterns with the current compliance period.

This assessment used design values, rather than expected exceedances, to facilitate the comparisons among varying standard levels. For the one exceedance standard options, the design value is simply the fourth highest concentration during the three year period. Because one exceedance is allowed for each year, if the fourth highest value is greater than the specified standard level, then the county has failed to meet the alternative NAAQS. Similarly, for the five exceedance alternative, the design value is the sixteenth largest daily maximum 1-hour concentration. Once the design value is calculated, comparisons with alternative standard levels can be made directly without having to recompute the number of exceedances. Table A-3 presents the air quality data comparisons on a county basis.

Table A-3. Number of counties not meeting selected analytic options based on design values for 1987-89 and 1991-93.

ANALYTIC OPTIONS		1987 -89	1991 -93
Primary Standard			
1-hour,	1 exceedance, 0.12 ppm	224	104
,	avg annual 2nd max, 0.12	168	72
,	1 exceedance, 0.10 ppm	384	310
,	avg annual 2nd max, 0.10	338	243
8-hour,	1 exceedance, 0.10 ppm	236	103
,	1 exceedance, 0.09 ppm	332	221
,	avg annual 2nd max, 0.09	279	160
,	3 exceedance, 0.09 ppm	263	113
,	5 exceedance, 0.09 ppm	190	67
,	1 exceedance, 0.08 ppm	419	394
,	3 exceedance, 0.08 ppm	370	274
,	5 exceedance, 0.08 ppm	321	194
,	avg annual 2nd max, 0.08 ppm	379	329
,	avg annual 3rd max, 0.08 ppm	352	274
,	avg annual 4th max, 0.08 ppm	333	227
,	avg annual 5th max, 0.08 ppm	305	187
,	1 exceedance, 0.07 ppm	467	514
Total Number of Counties with monitors		506	581
Total number of Counties in the U.S.		3142	3142

The impact of using the average annual second maximum (AvgMax2) concentration as the NAAQS statistic on the number of counties not meeting selected standard alternatives is shown in Table A-3. For each alternative, there are fewer non-compliant counties for the AvgMax2 statistic (about 20-30% less) than the current one exceedance form. Table A-4 presents a comparison of the impact of the selected analytic options on the compliance status of the original 98 areas designated nonattainment for O_3 under the Clean Air Act Amendments of 1990. A detailed listing of design values for each alternative by nonattainment area is provided in Table A-13 at the end of this Appendix.

Table A-4. Number of original nonattainment areas not meeting selected analytic options based on 1991-93 air quality monitoring design values.

ANALYTIC OPTIONS		1991-93
1-hour,	1 exceedance, 0.12 ppm	39
,	avg annual 2nd max, 0.12	34
,	1 exceedance, 0.10 ppm	87
,	avg annual 2nd max, 0.10	69
8-hour,	1 exceedance, 0.09 ppm	72
,	avg annual 2nd max, 0.09	52
,	1 exceedance, 0.08 ppm	93
,	avg annual 2nd max, 0.08	85
,	avg annual 5nd max, 0.08	64
,	5 exceedance, 0.08 ppm	63
,	1 exceedance, 0.07 ppm	97
Number of Areas with monitors.		97

Table A-5 compares the number of counties in both compliance periods with design values in excess of the current 1-hour standard with selected 8-hour one exceedance alternatives. Despite the larger number of counties with data in 1991-1993, the impact of O_3 data from the summer of 1988 is readily apparent in the earlier compliance period. The number of counties not meeting an 8-hour, one expected exceedance standard with a level of 0.10 ppm is quite close to the number under the current 1-hour 0.12 ppm standard. Tables A-6 through A-8 contrast the current 1-hour standard with the levels of concern for the Sum06, W126 and AOT06 secondary standard options. Small differences in the total number of counties with monitors among these tables and Table A-5 result from the data completeness requirements for the secondary standard alternatives. For the levels of concern, the Sum06 standard yields almost four times as many non-compliance areas as the current 1-hour primary standard. Table A-9 contrasts the one and five exceedance options for an 8-hour standard. For both compliance periods, an 8-hour five exceedance standard of 0.08 ppm is comparable to an 8-hour one exceedance standard of 0.09 ppm in terms of the number of non-complying areas. Table A-10 contrasts the alternative 8-hour one exceedance options with the three peak-weighted indices. Finally, Tables A-11 and A-12 repeat the 1-hour and 8-hour comparisons on a metropolitan area, rather than a county, basis.

Another way to view the information presented above is in terms of ratios among design values for alternative standards. For example, the average ratio of 8-hour to 1-hour design values for a 1 exceedance per year standard is 0.86 based on 1991-93 data. This ratio has been fairly stable over time increasing slightly from 0.81 in 1980 to 0.86 in 1993. The ratio is also fairly consistent across EPA Regions, with the median ratio ranging from 0.80 to 0.88. The median 8-hour/1-

hour 1 exceedance ratio for nine remote O_3 monitors in AIRS was slightly higher, 0.90, which would be expected for sites with less pronounced diurnal patterns. As noted above in Table A-5, the number of counties not meeting a 0.10 ppm 8-hour 1-exceedance standard is almost identical to the number not meeting a 0.12 ppm 1-hour standard. This is consistent with the 0.86 ratio because the ratio of the corresponding exceedance levels for the two standards (0.105 ppm/0.125 ppm) is equal to 0.84.

Design value comparisons based on 1991-93 data show that, on average, ozone design values for an 8-hour, 0.08 ppm, 1 exceedance standard are about 15% lower than for the current 1-hour standard, while design values for both an 8-hour, 0.08 ppm, 5 exceedance, and average annual 5th highest daily maximum standard are about 25% lower than the current 1-hour standard. On average, design values for an average annual 2nd highest daily maximum 8-hour standard are 18% lower than the current 1-hour standard.

In terms of exceedances, on average, sites meeting an average annual 2nd highest daily maximum 0.08 ppm standard have 1.2 exceedances per year, and 2.3 exceedances in the worst year of three, while sites meeting an average annual 5th highest daily maximum 0.08 ppm standard have 3.0 exceedances per year, and 5.4 exceedances in the worst year of three. Also, in the worst year of three, 95 % of sites meeting the average annual 2nd highest daily maximum 0.08 ppm standard have 7 or fewer exceedances, while 95 % of sites meeting an average annual 5th highest daily maximum 0.08 ppm standard have 12 or fewer exceedances.

Table A-13 lists design values based on 1991-93 monitoring data for each of the 1-hour and 8-hour standard alternatives in the original 98 areas designated nonattainment in 1991. The maximum Sum06 index value for 1991-93 is also provided in the last column.

Table A-5.

Comparison of design values for the current .12 ppm 1 hour, 1 exceedance standard vs. an 8 hour, 1 exceedance standard, by county.

1987 - 1989

NAAQS .12 ppm 1 Exc	8 Hour, 1 Exceedance NAAQS					Number of Counties
	DV <= .07	.07 < DV <=.08	.08 < DV <=.09	.09 < DV <=.10	DV > .10	
DV <= .12	39	48	84	759	36	282
DV > .12	0	0	3	21	200	224
Total	39	48	87	96	236	506

1991 - 1993

NAAQS .12 ppm 1 Exc	8 Hour, 1 Exceedance NAAQS					Number of Counties
	DV <= .07	.07 < DV <=.08	.08 < DV <=.09	.09 < DV <=.10	DV > .10	
DV <= .12	67	120	171	100	19	477
DV > .12	0	0	2	18	84	104
Total	67	120	173	118	103	581

Table A-6.

Comparison of design values for the current .12 ppm 1 hour, 1 exceedance standard vs. max Sum06 design values, by county.

1987 - 1989

NAAQS .12 ppm 1 Exc	Sum06 Max Year NAAQS			Number of Counties
	DV <= 16.5	16.5 < DV <=26.4	DV > 26.4	
DV <= .12	57	44	175	276
DV > .12	5	10	209	224
Total	62	54	384	500

1991 - 1993

NAAQS .12 ppm 1 Exc	Sum06 Max Year NAAQS			Number of Counties
	DV <= 16.5	16.5 < DV <=26.4	DV > 26.4	
DV <= .12	137	126	211	474
DV > .12	2	16	86	104
Total	139	142	297	578

Table A-7.

Comparison of design values for the current .12 ppm 1 hour, 1 exceedance standard vs. max W126 design values, by county.

1991 - 1993

NAAQS .12 ppm 1 Exc	W126 Max Year NAAQS			Number of Counties
	DV <= 13.8	13.8 < DV <=22.4	DV > 22.4	
DV <= .12	144	146	184	474
DV > .12	1	16	87	104
Total	145	162	271	578

Table A-8.

Comparison of design values for the current .12 ppm 1 hour, 1 exceedance standard vs. max AOT06 design values, by county.

1991 - 1993

NAAQS .12 ppm 1 Exc	AOT06 Max Year NAAQS			Number of Counties
	DV <= 4.8	4.9 < DV <= 7.5	DV > 7.5	
DV <= .12	285	106	83	474
DV > .12	9	21	74	104
Total	294	127	157	578

Table A-9.

Comparison of design values for an 8 hour, 1 exceedance standard vs. design values for an 8 hour, 5 exceedance standard, by county.

1987 - 1989

8 Hour 1 Exc NAAQS	8 Hour 5 Exc NAAQS		Number of Counties
	DV <=.08	DV > .08	
DV <= .07	38	0	38
.07 <= .08	48	0	48
.08 <= .09	71	16	87
.09 <= .10	25	71	96
DV > .10	2	234	236
Total	184	321	505

1991 - 1993

8 Hour 1 Exc NAAQS	8 Hour 5 Exc NAAQS		Number of Counties
	DV <=.08	DV > .08	
DV <= .07	67	0	67
.07 <= .08	120	0	120
.08 <= .09	159	14	173
.09 <= .10	39	79	118
DV > .10	2	101	103
Total	387	194	581

Table A-10.

Comparison of design values for an 8 hour, 1 exceedance standard vs. alternative secondary standard exposure indices, by county.

1991 - 1993

8 Hour 1 Exc NAAQS	No. of Counties	Alternative Secondary NAAQS Exposure Indices 10% Crop Loss		
		Sum06 > 26.4	W126 > 22.4	AOT06 > 7.5
DV <= .07	64	2	1	0
.07 <= .08	120	14	7	0
.08 <= .09	173	92	76	18
.09 <= .10	118	96	93	55
DV > .10	103	93	94	84
Total	578	297	269	157

Table A-11.

Comparison of design values for the current .12 ppm 1 hour, 1 exceedance standard vs. an 8 hour, 1 exceedance standard, by metropolitan area (CMSA/MSA).

1987 - 1989

NAAQS .12 ppm 1 Exc	8 Hour, 1 Exceedance NAAQS					Number of Areas (CMSAs & MSAs)
	DV <= .07	.07 < DV <=.08	.08 < DV <=.09	.09 < DV <=.10	DV > .10	
DV <= .12	8	19	34	30	14	106
DV > .12	0	0	1	8	72	80
Total	8	19	35	38	86	186

1991 - 1993

NAAQS .12 ppm 1 Exc	8 Hour, 1 Exceedance NAAQS					Number of Areas (CMSAs & MSAs)
	DV <= .07	.07 < DV <=.08	.08 < DV <=.09	.09 < DV <=.10	DV > .10	
DV <= .12	14	38	63	30	8	153
DV > .12	0	0	2	4	36	42
Total	14	38	65	34	44	195

Table A-12.

Comparison of design values for an 8 hour, 1 exceedance standard vs. design values for an 8 hour, 5 exceedance standard, by metropolitan area (CMSA/MSA).

1987 - 1989

8 Hour 1 Exc NAAQS	8 Hour 5 Exc NAAQS		Number of Areas (CMSAs & MSAs)
	DV <=.08	DV > .08	
DV <= .07	8	0	8
.07 <= .08	19	0	19
.08 <= .09	29	6	35
.09 <= .10	10	28	38
DV > .10	1	85	86
Total	67	119	186

1991 - 1993

8 Hour 1 Exc NAAQS	8 Hour 5 Exc NAAQS		Number of Areas (CMSAs & MSAs)
	DV <=.08	DV > .08	
DV <= .07	12	0	14
.07 <= .08	38	0	38
.08 <= .09	61	4	65
.09 <= .10	8	26	34
DV > .10	0	44	44
Total	121	74	195

Table A-13. Comparison of design values for alternative O3 air quality standards for the original 98 classified nonattainment areas.

Nonattainment Area Name	Clean Air Act Classification	D.V. 1h, 1ex	D.V. 8h, 1ex	Avg Max2 8 hr	Avg Max5 8 hr	D.V. 8h, 5ex	Sum06 8am-8pm
Albany-Schenectady-Troy NA Area	Marginal	0.10	0.09	0.09	0.08	0.08	15.8
Allentown-Bethlehem-Easton NA	Marginal	0.12	0.10	0.10	0.09	0.09	28.7
Altoona NA Area	Marginal	0.11	0.09	0.09	0.08	0.08	30.0
Atlanta NA Area	Serious	0.15	0.13	0.11	0.10	0.10	50.0
Atlantic City NA Area	Moderate	0.12	0.11	0.11	0.10	0.10	47.9
Baltimore NA Area	Severe 15	0.15	0.12	0.12	0.11	0.10	55.9
Baton Rouge NA Area	Serious	0.14	0.11	0.11	0.09	0.09	23.0
Beaumont-Port Arthur NA Area	Serious	0.13	0.11	0.11	0.10	0.09	26.8
Birmingham NA Area	Marginal	0.12	0.10	0.10	0.09	0.09	25.1
Boston-Lawrence-Worcester NA Area	Serious	0.14	0.11	0.12	0.09	0.10	29.0
Buffalo-Niagara Falls NA Area	Marginal	0.11	0.10	0.09	0.08	0.08	22.3
Canton NA Area	Attainment	0.11	0.10	0.09	0.09	0.09	45.5
Charleston NA Area	Attainment	0.11	0.09	0.07	0.07	0.07	29.7
Charlotte-Gastonia NA Area	Attainment	0.12	0.10	0.10	0.09	0.09	50.6
Cherokee Co NA Area	Attainment	0.10	0.09	0.09	0.08	0.08	32.0
Chicago-Gary-Lake County NA Area	Severe 17	0.15	0.12	0.11	0.10	0.10	38.9
Cincinnati-Hamilton NA Area	Moderate	0.13	0.11	0.10	0.09	0.09	46.1
Cleveland-Akron-Lorain NA Area	Moderate	0.14	0.12	0.13	0.11	0.10	45.6
Columbus NA Area	Attainment	0.12	0.10	0.09	0.08	0.08	31.0
Dallas-Fort Worth NA Area	Moderate	0.14	0.12	0.11	0.09	0.09	39.2
Dayton-Springfield NA Area	Attainment	0.11	0.10	0.09	0.09	0.08	35.0
Detroit-Ann Arbor NA Area	Attainment	0.12	0.11	0.10	0.09	0.09	30.1
Door Co NA Area	Marginal	0.13	0.10	0.09	0.09	0.08	19.5
Edmonson Co NA Area	Attainment	0.09	0.08	0.08	0.07	0.07	17.1
El Paso NA Area	Serious	0.14	0.09	0.09	0.08	0.08	15.7
Erie NA Area	Marginal	0.11	0.09	0.09	0.08	0.08	30.8
Essex Co NA Area	Marginal	0.12	0.10	0.10	0.09	0.09	23.0
Evansville NA Area	Marginal	0.11	0.10	0.09	0.09	0.09	40.5
Grands Rapids NA Area	Moderate	0.15	0.13	0.13	0.11	0.11	40.3
Greater Connecticut NA Area	Severe 17	0.16	0.12	0.12	0.11	0.11	35.9
Greenbrier NA Area	Attainment	0.10	0.09	0.08	0.08	0.08	22.9
Greensboro-Winston-Salem-High	Attainment	0.11	0.10	0.09	0.09	0.09	48.3
Hancock Co and Waldo Co NA Area	Marginal	0.11	0.10	0.10	0.08	0.08	13.2
Harrisburg-Lebanon-Carlisle NA	Marginal	0.11	0.10	0.10	0.09	0.09	36.5
Houston-Galveston-Brazoria NA	Severe 17	0.20	0.13	0.12	0.10	0.10	30.0
Huntington-Ashland NA Area	Attainment	0.12	0.10	0.09	0.09	0.09	35.2
Indianapolis NA Area	Attainment	0.10	0.10	0.09	0.09	0.09	38.3
Jefferson Co NA Area	Marginal	0.11	0.11	0.10	0.09	0.08	18.4
Jersey Co NA Area	Attainment	0.11	0.09	0.09	0.08	0.08	26.9
Johnstown NA Area	Marginal	0.11	0.10	0.09	0.08	0.08	29.5
Kansas City NA Area	Attainment	0.11	0.09	0.09	0.08	0.08	32.3
Kent County and Queen Anne's County	Marginal	0.13	0.11	0.11	0.10	0.10	48.9
Kewaunee Co NA Area	Moderate	0.11	0.09	0.09	0.08	0.08	18.1
Knox Co and Lincoln Co NA Area	Moderate	0.13	0.12	0.11	0.10	0.09	20.6
Knoxville NA Area	Attainment	0.12	0.10	0.09	0.09	0.09	42.7
Lake Charles NA Area	Marginal	0.12	0.09	0.09	0.09	0.09	20.4
Lancaster NA Area	Marginal	0.12	0.10	0.10	0.09	0.09	38.0
Lewiston - Auburn NA Area	Moderate	0.11	0.09	0.09	0.08	0.08	13.4
Lexington-Fayette NA Area	Attainment	0.10	0.09	0.08	0.08	0.08	29.4
Los Angeles South Coast Air Basin	Extreme	0.30	0.19	0.19	0.18	0.17	111.0

Table A-13. (cont.) Comparison of design values for alternative O3 air quality standards for the original 98 classified nonattainment areas.

Nonattainment Area Name	Clean Air Act Classification	D.V. 1h, 1ex	D.V. 8h, 1ex	Avg Max2 8 hr	Avg Max5 8 hr	D.V. 8h, 5ex	Sum06 8am-8pm
Louisville NA Area	Moderate	0.13	0.11	0.10	0.09	0.09	49.5
Manchester NA Area	Marginal	0.09	0.08	0.08	0.07	0.07	8.9
Manitowoc Co NA Area	Moderate	0.13	0.10	0.10	0.08	0.08	25.6
Memphis NA Area	Attainment	0.12	0.10	0.09	0.09	0.09	37.9
Miami-Fort Lauderdale-W. Palm Beach	Attainment	0.11	0.09	0.08	0.08	0.07	14.3
Milwaukee-Racine NA Area	Severe 17	0.15	0.12	0.11	0.10	0.10	38.9
Monterey Bay Unified NA Area	Moderate	0.11	0.09	0.09	0.08	0.08	32.9
Muskegon NA Area	Moderate	0.14	0.11	0.11	0.11	0.10	36.5
Nashville NA Area	Moderate	0.12	0.11	0.10	0.09	0.09	44.5
New York-N. New Jersey-Long Island	Severe 17	0.15	0.13	0.12	0.11	0.11	45.1
Norfolk-Virginia Beach-Newport	Marginal	0.13	0.10	0.10	0.09	0.09	46.4
Owensboro NA Area	Attainment	0.10	0.09	0.08	0.08	0.08	24.1
Paducah NA Area	Attainment	0.11	0.09	0.09	0.08	0.08	34.8
Parkersburg NA Area	Attainment	0.12	0.10	0.09	0.09	0.09	37.1
Philadelphia-Wilmington-Trenton	Severe 15	0.16	0.13	0.12	0.12	0.12	58.6
Phoenix	Moderate	0.15	0.11	0.09	0.09	0.09	39.1
Pittsburgh-Beaver Valley NA Area	Moderate	0.12	0.11	0.10	0.09	0.09	38.3
Portland NA Area	Moderate	0.13	0.11	0.11	0.10	0.09	21.2
Portland-Vancouver AQMA NA Area	Marginal	0.11	0.10	0.08	0.07	0.07	14.5
Portsmouth-Dover-Rochester, NH	Serious	0.13	0.11	0.11	0.09	0.09	17.1
Poughkeepsie NA Area	Moderate	0.13	0.10	0.10	0.10	0.09	26.0
Providence (all of RI) NA Area	Serious	0.15	0.12	0.11	0.10	0.10	29.8
Raleigh-Durham NA Area	Attainment	0.12	0.10	0.09	0.09	0.09	59.9
Reading NA Area	Moderate	0.12	0.11	0.10	0.09	0.09	34.4
Reno	Marginal	0.09	0.08	0.07	0.07	0.07	23.9
Richmond-Petersburg NA Area	Moderate	0.13	0.10	0.10	0.09	0.09	47.3
Sacramento Metro NA Area	Serious	0.15	0.12	0.12	0.11	0.11	65.8
Salt Lake City-Ogden NA Area	Moderate	0.11	0.09	0.08	0.08	0.08	25.9
San Diego NA Area	Severe 15	0.15	0.12	0.12	0.11	0.11	54.9
San Francisco-Bay NA Area	Attainment	0.12	0.09	0.09	0.08	0.08	19.9
San Joaquin Valley NA Area	Serious	0.16	0.12	0.12	0.11	0.11	83.2
Santa Barbara - Santa Maria -	Moderate	0.12	0.10	0.10	0.09	0.09	44.3
Scranton-Wilkes-Barre NA Area	Marginal	0.12	0.11	0.10	0.09	0.09	30.0
Seattle - Tacoma NA Area	Marginal	0.11	0.09	0.08	0.08	0.08	12.3
Sheboygan NA Area	Moderate	0.14	0.11	0.09	0.08	0.09	29.2
Smyth Co NA Area	Marginal	ND	ND	ND	ND	ND	ND
South Bend-Elkhart NA Area	Attainment	0.10	0.09	0.09	0.08	0.08	38.8
Southeast Desert Modified AQMD	Severe 17	0.20	0.15	0.15	0.14	0.14	103.9
Springfield (W. Mass) NA Area	Serious	0.14	0.11	0.11	0.10	0.10	24.6
St. Louis NA Area	Moderate	0.13	0.11	0.11	0.09	0.09	42.4
Sussex Co NA Area	Marginal	0.12	0.11	0.11	0.09	0.09	41.6
Tampa-St. Petersburg-Clearwater	Attainment	0.11	0.08	0.08	0.08	0.08	22.0
Toledo NA Area	Attainment	0.12	0.10	0.09	0.08	0.08	21.7
Ventura Co NA Area	Severe 15	0.15	0.13	0.13	0.11	0.12	72.9
Walworth Co NA Area	Marginal	0.12	0.10	0.09	0.08	0.09	35.8
Washington NA Area	Serious	0.14	0.11	0.11	0.10	0.10	50.1
York NA Area	Marginal	0.11	0.10	0.09	0.09	0.09	40.2
Youngstown-Warren-Sharon NA Area	Marginal	0.11	0.10	0.10	0.09	0.09	37.7

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APPENDIX B

8-Hr Daily Maximum Dose Exposure Distributions
For Outdoor Children Under Various Air Quality Scenarios

APPENDIX B

Figures B.1 through B.8 are graphs from Johnson et al. (1996b) showing 8-hour daily maximum dose exposures for outdoor children under various air quality scenarios. Two graphs are provided for each of four study areas (Houston, New York, Philadelphia, and Washington, D.C.). The graphs use two indicators to characterize O_3 exposure:

- Number of children experiencing 8-hour daily maximum dose exposures on one or more days under moderate exertion conditions,

- Number of total occurrences in which a child experiences a daily maximum dose exposure under moderate exertion conditions.

Moderate exertion conditions are defined as an EVR level between 13 and 27 l min^{-1} m^{-2}.

Figure B.1 presents results for the first indicator (number of outdoor children) based on applications of pNEM/O_3 to Houston. Nine distributions are plotted on the graph: one for baseline ("As Is") conditions; two for 1-hour, 1-exceedance standards (1H1EX-0.12 and 1H1EX-0.10); four for 8-hour, 1 exceedance standards (8H1EX-0.10, 8H1EX-0.09, 8H1EX-0.08, 8H1EX-0.07); and two for 8-hour, 5-exceedance standards (8H5EX-0.08, 8H5EX-0.09). For example, 8H5EX-0.08 indicates an 8-hour, 5-exceedances standard with O_3 concentration set at 8 pphm or 0.08 ppm.

The ordinate (y coordinate) of each point on the graph shows the number of children with one or more daily maximum dose exposures equal to or above the O_3 concentration indicated by the point's abscissa (x coordinate). In Figure B.1, the "As Is" curve is associated with the highest number of children exposed when the specified O_3 concentrations fall between 0.05 and 0.16 ppm. The nine curves tend to converge at lower and higher O_3 concentrations. In a similar manner, the 8H1EX-0.07 standard is associated with the lowest number of children exposed when the specified O_3 concentration falls between 0.03 and 0.08 ppm.

Appendix E of Johnson et al. (1996b) provides similar graphs for two other EVR ranges of interest: 16-30 l min^{-1} m^{-2} and $\geq$ 30 l min^{-1} m^{-2} for 1-hour exposures.

FIGURE B.3. EIGHT-HOUR MAXIMUM DOSE EXPOSURE DISTRIBUTIONS FOR OUTDOOR CHILDREN EXPOSED ON ONE OR MORE DAYS UNDER MODERATE EXERTION (EVR 13-27 LITERS/MIN-M2) IN NEW YORK, NY.

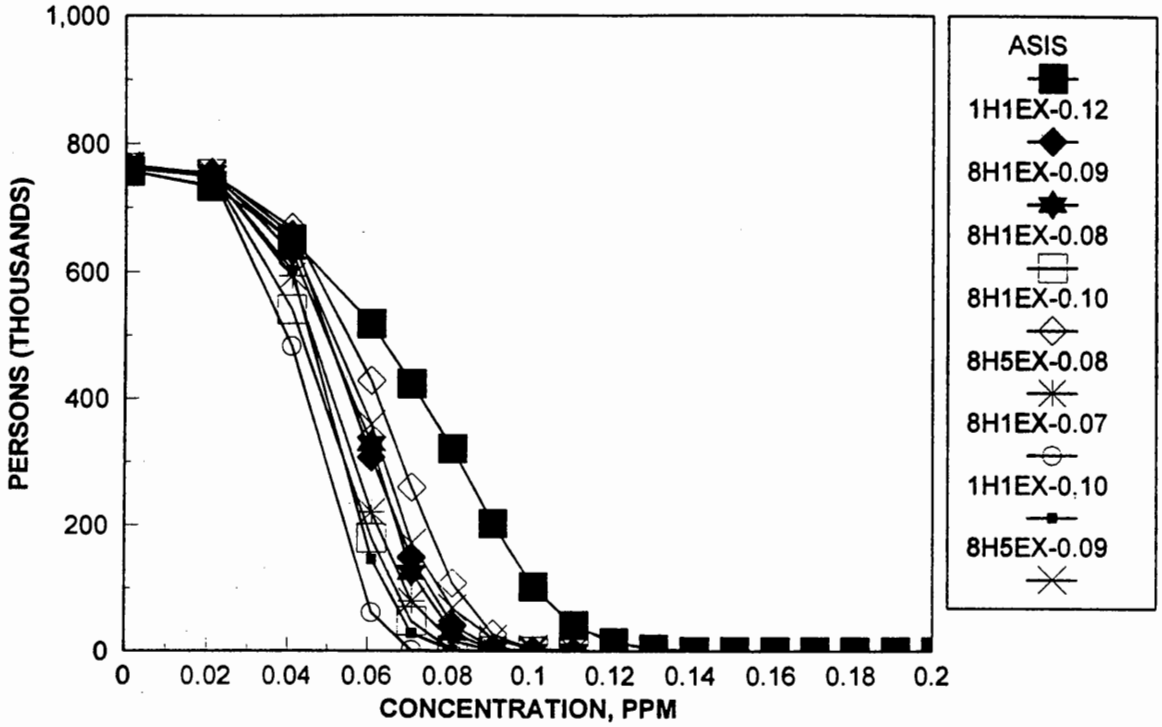


FIGURE B.4. EIGHT-HOUR MAXIMUM DOSE EXPOSURE DISTRIBUTIONS OF TOTAL OCCURRENCES FOR OUTDOOR CHILDREN EXPOSURE UNDER MODERATE EXERTION (EVR 13-27 LITERS/MIN-M2) IN NEW YORK, NY.

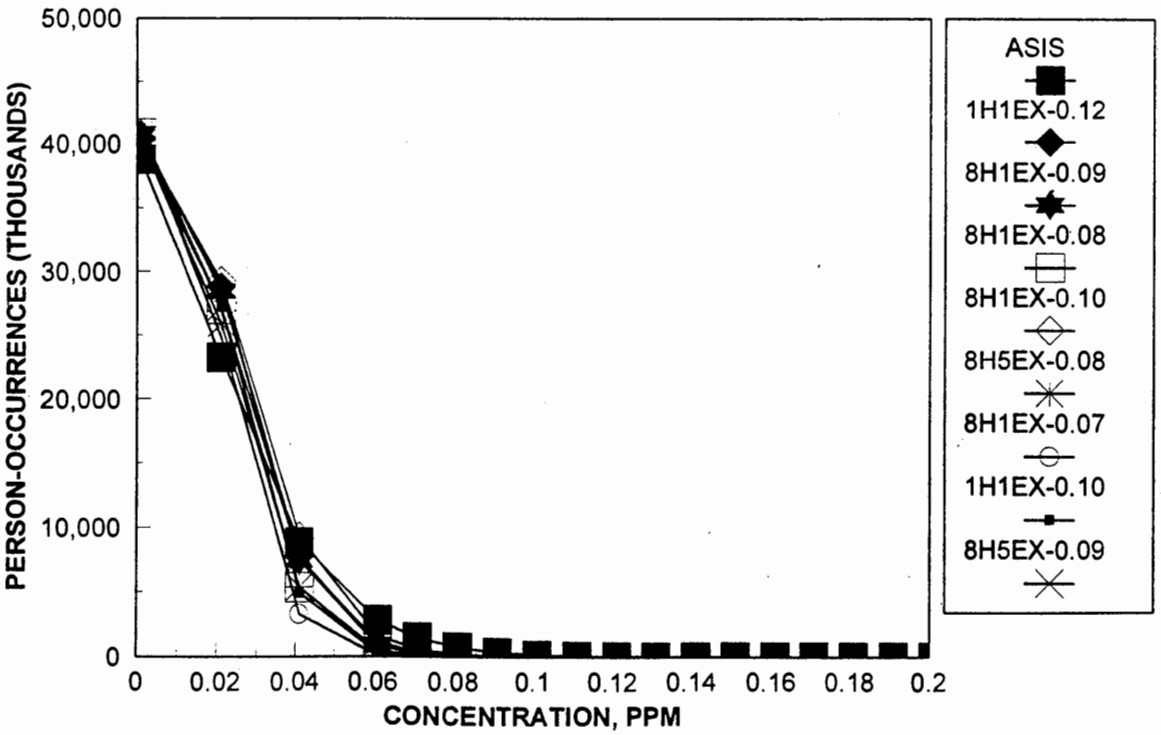


FIGURE B.1. EIGHT-HOUR MAXIMUM DOSE EXPOSURE DISTRIBUTIONS FOR OUTDOOR CHILDREN EXPOSED ON ONE OR MORE DAYS UNDER MODERATE EXERTION (EVR 13-27 LITERS/MIN-M2) IN HOUSTON, TX.

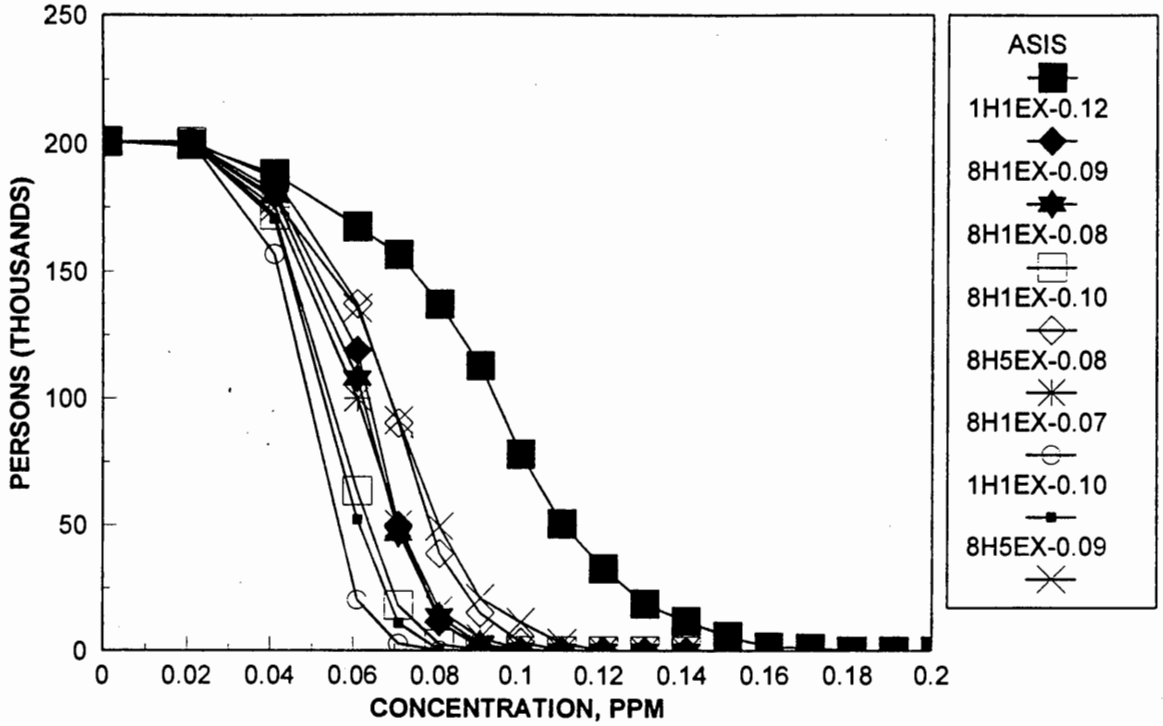


FIGURE B.2. EIGHT-HOUR MAXIMUM DOSE EXPOSURE DISTRIBUTIONS OF TOTAL OCCURRENCES FOR OUTDOOR CHILDREN EXPOSURE UNDER MODERATE EXERTION (EVR 13-27 LITERS/MIN-M2) IN HOUSTON, TX.

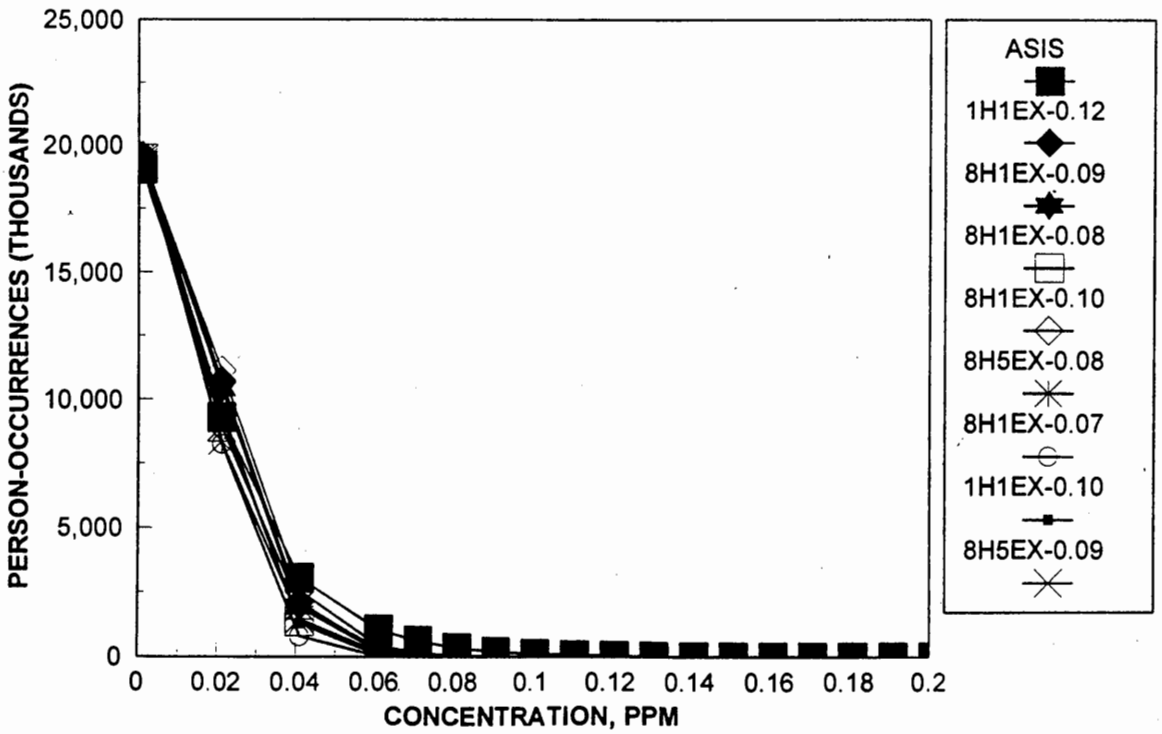


FIGURE B.5. EIGHT-HOUR MAXIMUM DOSE EXPOSURE DISTRIBUTIONS FOR OUTDOOR CHILDREN EXPOSED ON ONE OR MORE DAYS UNDER MODERATE EXERTION (EVR 13-27 LITERS/MIN-M2) IN PHILADELPHIA, PA.

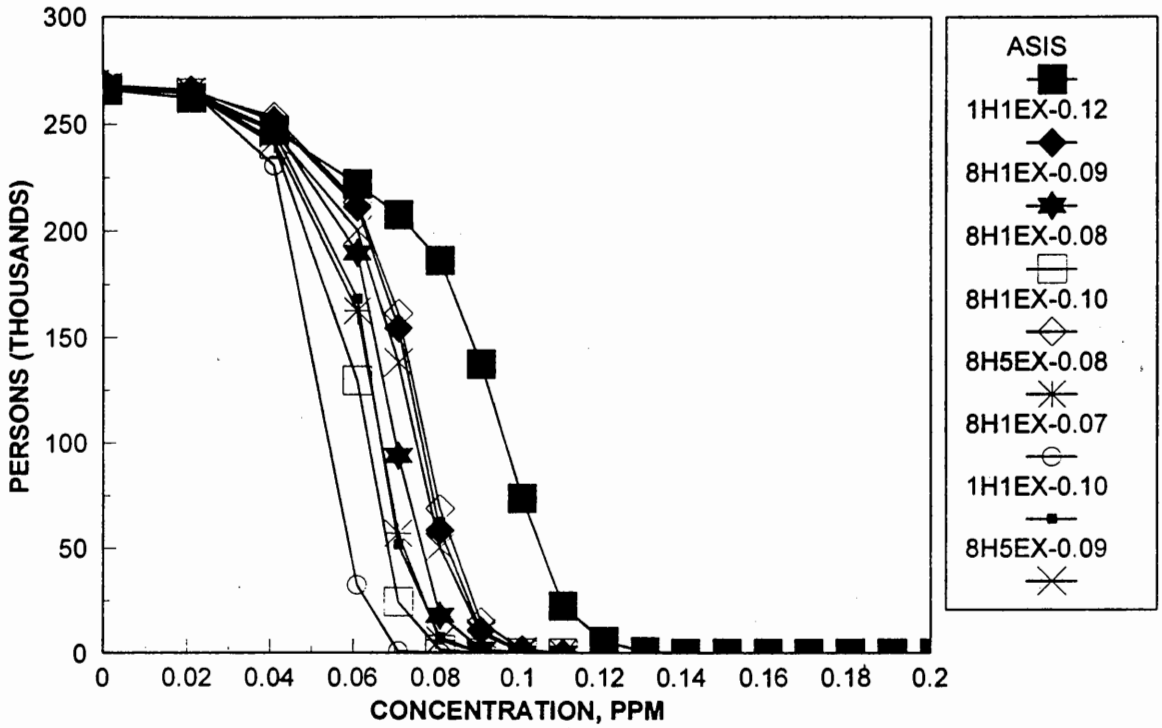


FIGURE B.6. EIGHT-HOUR MAXIMUM DOSE EXPOSURE DISTRIBUTIONS OF TOTAL OCCURRENCES FOR OUTDOOR CHILDREN EXPOSURE UNDER MODERATE EXERTION (EVR 13-27 LITERS/MIN-M2) IN PHILADELPHIA, PA.

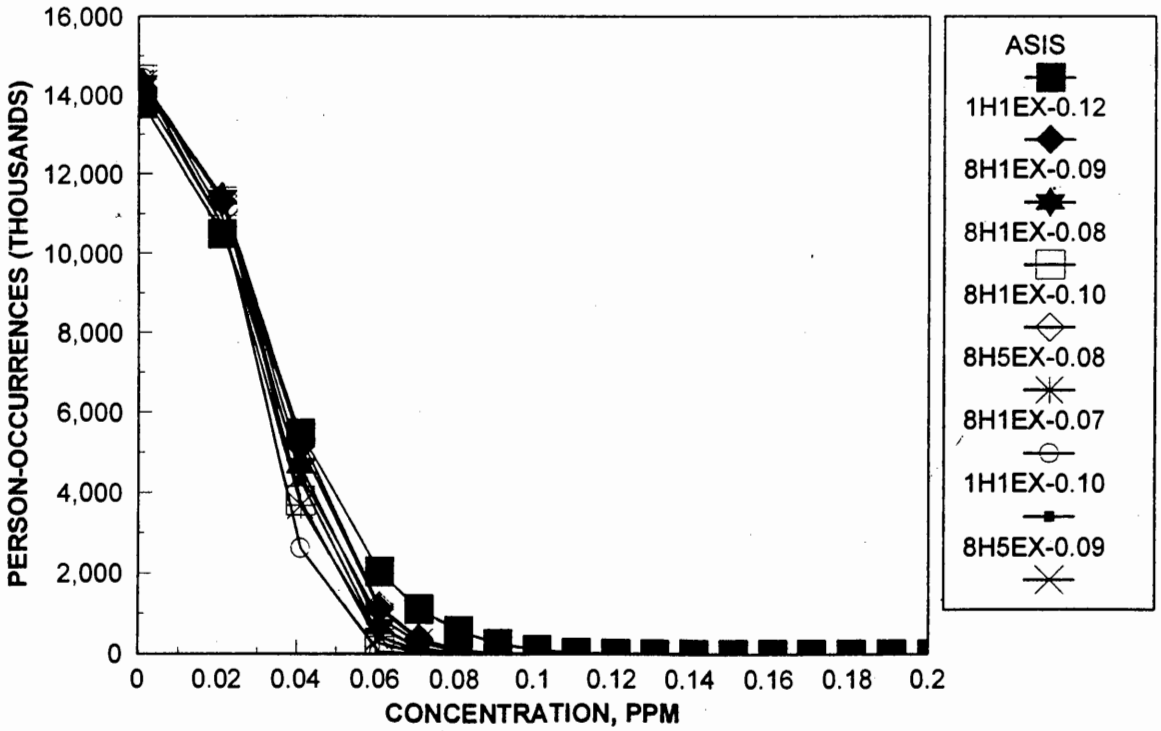


FIGURE B.7. EIGHT-HOUR MAXIMUM DOSE EXPOSURE DISTRIBUTIONS FOR OUTDOOR CHILDREN EXPOSED ON ONE OR MORE DAYS UNDER MODERATE EXERTION (EVR 13-27 LITERS/MIN-M2) IN WASHINGTON, D.C.

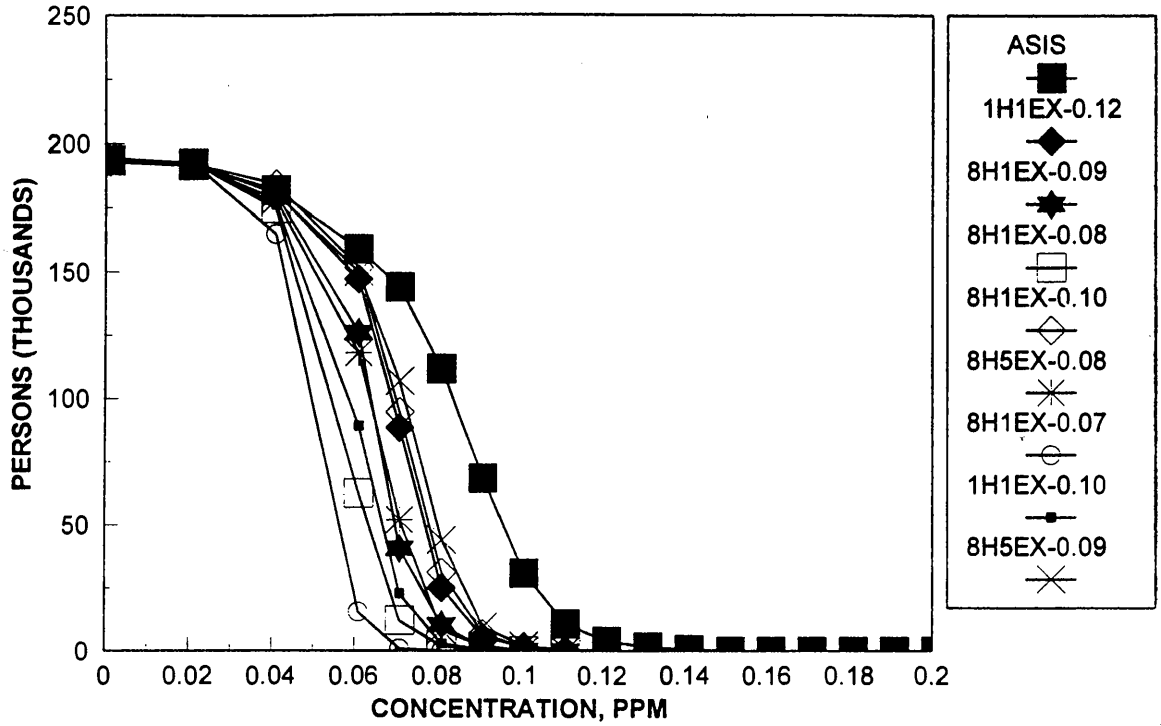
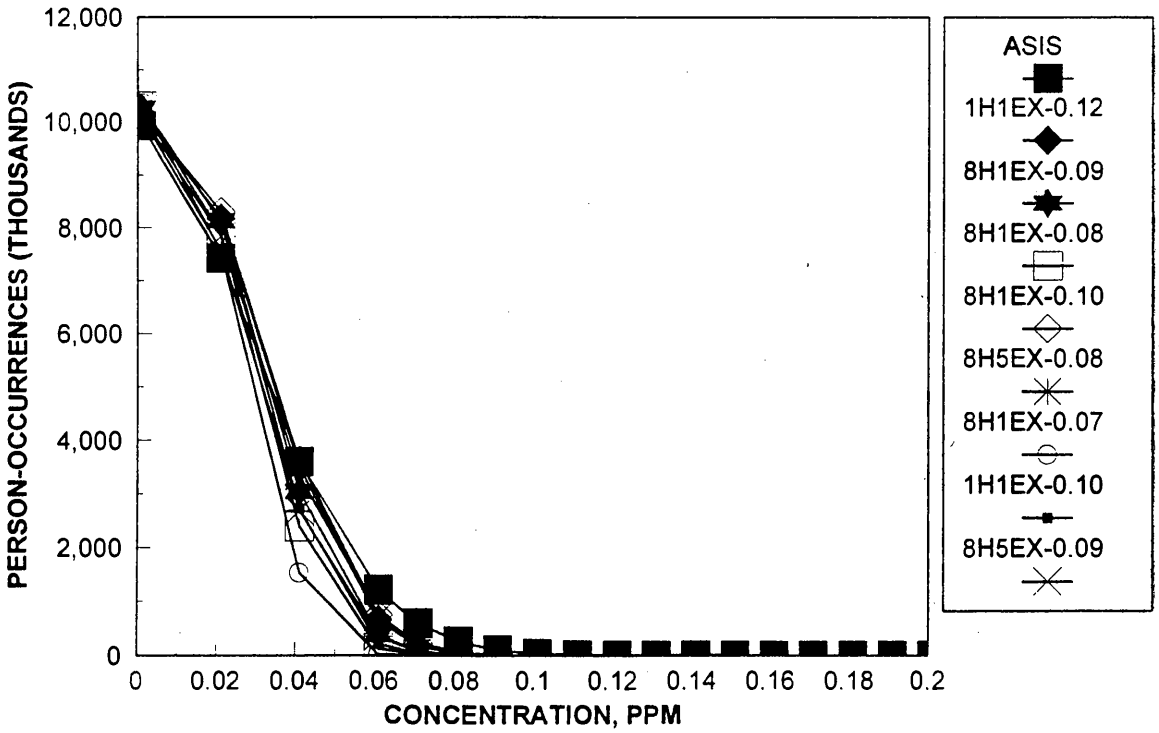


FIGURE B.8. EIGHT-HOUR MAXIMUM DOSE EXPOSURE DISTRIBUTIONS OF TOTAL OCCURRENCES FOR OUTDOOR CHILDREN EXPOSURE UNDER MODERATE EXERTION (EVR 13-27 LITERS/MIN-M2) IN WASHINGTON, D.C.



APPENDIX C

Probabilistic Exposure-Response Relationships for FEV_1 Decrements $\geq$15% and $\geq$20%

APPENDIX C

C.1 PROBABILISTIC EXPOSURE/RESPONSE RELATIONSHIPS

Figures C.1 and C.2 (which are Figures B.28 and B.29, respectively, from Whitfield et al., 1996) show the probabilistic exposure-response relationships for FEV_1 decrement ≥ 15% and ≥20%, derived from Folinsbee et al. (1988), Horstman et al. (1990), and McDonnell et al. (1991) for 8 hr exposures and individuals engaged in moderate exertion. The small squares indicate the response rates at the ozone concentrations at which subjects were exposed in these studies (0.08, 0.10, and 0.12 ppm), and the short horizontal line segments above and below the data point, which are connected by a line segment, indicate the 90% Credible Interval (CI). For example, for FEV_1 decrement ≥15%, the response rate at 0.08 ppm was about 18%, and the 90% CI about this value is about 10-28%; the derived 90% CI compares favorably to this range. The "derived" 90% CI and the "experimental" CIs compare less favorably at 0.12 ppm because the linear regression used to fit the data does not capture the nonlinear characteristics of the data. The derived and experimental CIs for other endpoints compare more favorably at all ozone concentrations for the FEV_1 ≥20% endpoint. Figures for 31 other endpoints are given in Appendix B of Whitfield et al. (1996). Characteristics (i.e., parameters of functions fit to the data, and regression r^2 values) of functions representing exposure-response relationships for 33 endpoints are listed in Table C.1 and C.2 (Tables 3.7 and 3.8, respectively, from Whitfield et al., 1996).

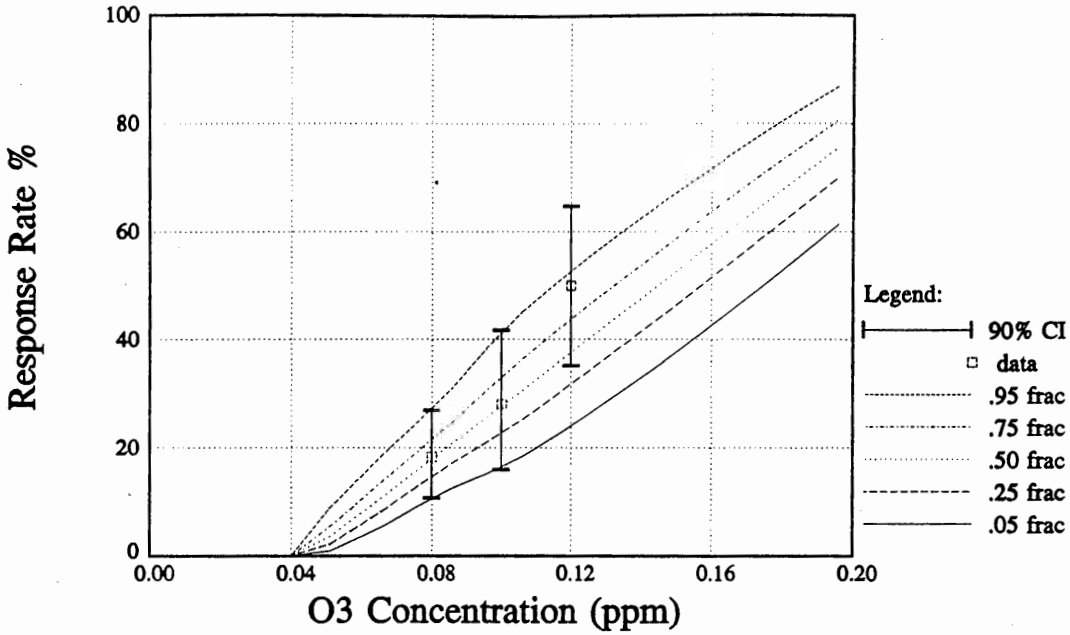


FIGURE C.1 Probabilistic Exposure-Response Relationship for FEV_1 Decrement ≥ 15% Derived from Folinsbee et al. (1988), Horstman et al. (1990), and McDonnell et al. (1991) [for 8 hr exposures, heavy exertion; includes data, medians, and 90% CIs for data and relationship]

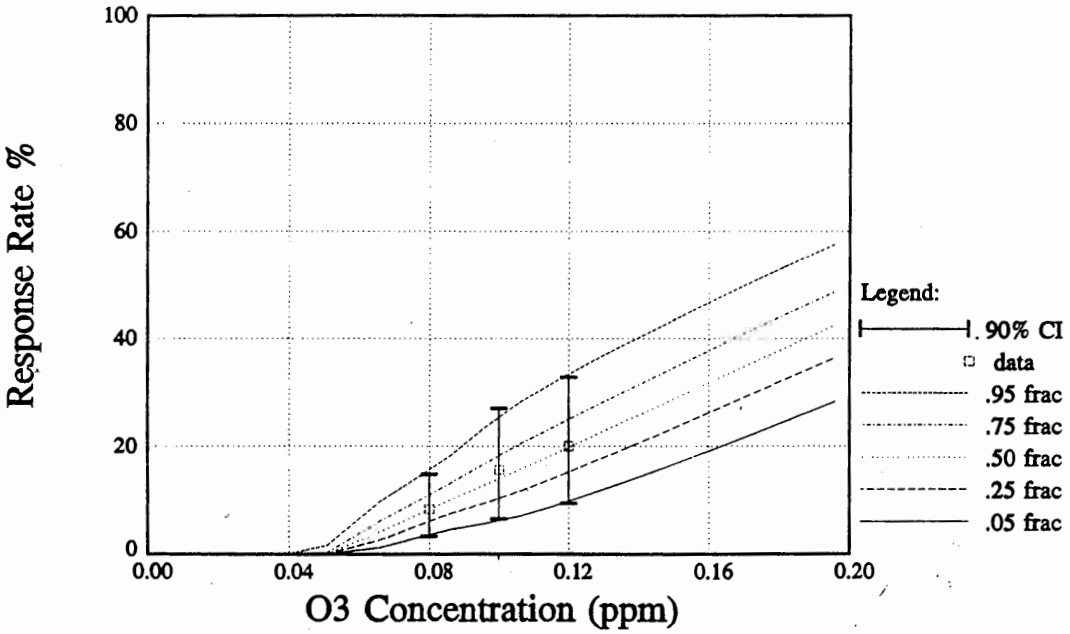


FIGURE C.2 Probabilistic Exposure-Response Relationship for FEV_1 Decrement ≥ 20% Derived from Folinsbee et al. (1988), Horstman et al. (1990), and McDonnell et al. (1991) [for 8 hr exposures, heavy exertion; includes data, medians, and 90% CIs for data and relationship]

TABLE C.1 Summary of Functions Fit to Experimental Data — FEV_1 Decrement — Corrected for Exercise in Clean Air

Study	Endpoint[a]	Function	a	b	d	e	r^2
Avol et al., 1984 (1.33 hrs)	$DFEV_1 \geq 10\%$	Linear	-0.2395	3.4388			0.98
	$DFEV_1 \geq 15\%$	Linear	-0.2400	2.9713			0.99
	$DFEV_1 \geq 20\%$	Linear	-0.2395	2.6825			0.99
Kulle et al., 1985 (2 hrs)	$DFEV_1 \geq 10\%$	Linear	-0.3225	2.3500			0.95
	$DFEV_1 \geq 15\%$	Linear	-0.2600	1.6000			0.93
	$DFEV_1 \geq 20\%$	Linear	-0.2375	1.2500			0.89
McDonnell et al., 1983 (2.5 hrs)	$DFEV_1 \geq 10\%$	Logistic		0.6420	5.5996	-27.2927	0.99
	$DFEV_1 \geq 15\%$	Logistic		0.4968	9.4948	-45.3838	1.00
	$DFEV_1 \geq 20\%$	Logistic		0.3347	12.0073	-60.4547	1.00
Seal et al., 1993 (2.33 hrs)	$DFEV_1 \geq 10\%$	Probit	-1.0276	0.7917			0.99
	$DFEV_1 \geq 15\%$	Probit	-0.6639	0.8401			0.99
	$DFEV_1 \geq 20\%$	Probit	-0.3259	0.9192			0.97
Folinsbee et al., 1988; Horstman et al., 1990; McDonnell et al., 1991 (8 hrs)	$DFEV_1 \geq 10\%$	Linear	-0.0980	5.0000			1.00
	$DFEV_1 \geq 15\%$	Linear	-0.2087	4.9000			1.00
	$DFEV_1 \geq 20\%$	Linear	-0.1462	2.9250			0.98

[a]$DFEV_1$ means forced expiratory volume (in 1 sec.) decrement.

TABLE C.2 Summary of Functions Fit to Experimental Data — Symptoms — Corrected for Exercise in Clean Air

Study	Endpoint[a]	Function	a	b	d	e	r^2
Avol et al., 1984 (1.33 hrs)	Any lower respiratory	Linear	-0.2084	2.6824			0.99
	M/S lower resp	Linear	-0.0902	0.5206			0.94
Kulle et al., 1985 (2 hrs)	Any cough	Linear	-0.2650	3.0000			0.97
	Any PDI	Linear	-0.4550	3.8000			0.79
	M/S cough	Linear	-0.1626	0.8675			-0.33[b]
	M/S PDI	Linear	-0.5250	3.0000			0.72
McDonnell et al., 1983 (2.5 hrs)	Any cough	Probit	-2.0954	1.2098			0.99
	Any PDI	Probit	-1.6071	1.5124			0.96
	M/S cough	Linear	0.0062	1.2604			0.70
	M/S PDI	Linear	-0.0427	1.1512			0.96
Seal et al., 1993 (2.33 hrs)	Any cough	Lognormal	0.2469	1.9248			0.97
	Any PDI	Lognormal	0.2464	2.3641			0.99
	M/S cough	Linear	-0.1445	1.3704			0.97
	M/S PDI	Probit	-0.3209	0.9317			0.96
Folinsbee et al., 1988; Horstman et al., 1990; McDonnell et al., 1991 (8 hrs)	Any cough	Linear	-0.2928	5.0750			0.54
	Any PDI	Linear	0.7372	10.1750			1.00
	M/S cough	Linear	-0.1747	2.3000			0.88
	M/S PDI	Linear	-0.3087	3.7000			0.93

[a]Initializations: M/S means moderate or severe, PDI means pain on deep inspiration.
[b]The data do not support a meaningful exposure-response relationship for this health endpoint. The negative r^2 value flags this situation.

C.2 HEADCOUNT RISK RESULTS

Risk results for each endpoint are available in the form of 10 probability distributions for each air quality scenario. Since there are nine scenarios, it is not practical to plot all of the distributions on one figure because the figure would be quite messy. The nine air quality scenarios include: one for baseline ("As Is") conditions; two for 1-hour, 1-exceedance standards (1112 and 1110); four for 8-hour, 1 exceedance standards (8110, 8109, 8108, 8107); and two for 8-hour, 5-exceedance standards (8508, 8509). The first digit in the code for each standard indicates the averaging time, the second digit specifies the number of exceedances, and the last two digits specify the standard expressed in pphm. For example, 8508 indicates an 8-hour, 5-exceedances standard with O_3 concentration set at 8 pphm or 0.08 ppm.

To gain insight about the risk implications of the air quality scenarios, we developed "representative distributions" and "Box plots." These are shown in Figures C.3-20 and C.21-23, respectively. Figures C.24-26 estimate the number of times that responders (i.e., outdoor children who experience a specific condition, such as having FEV_1 decrements ≥15%) respond.

C.2.1 Representative Risk Distributions

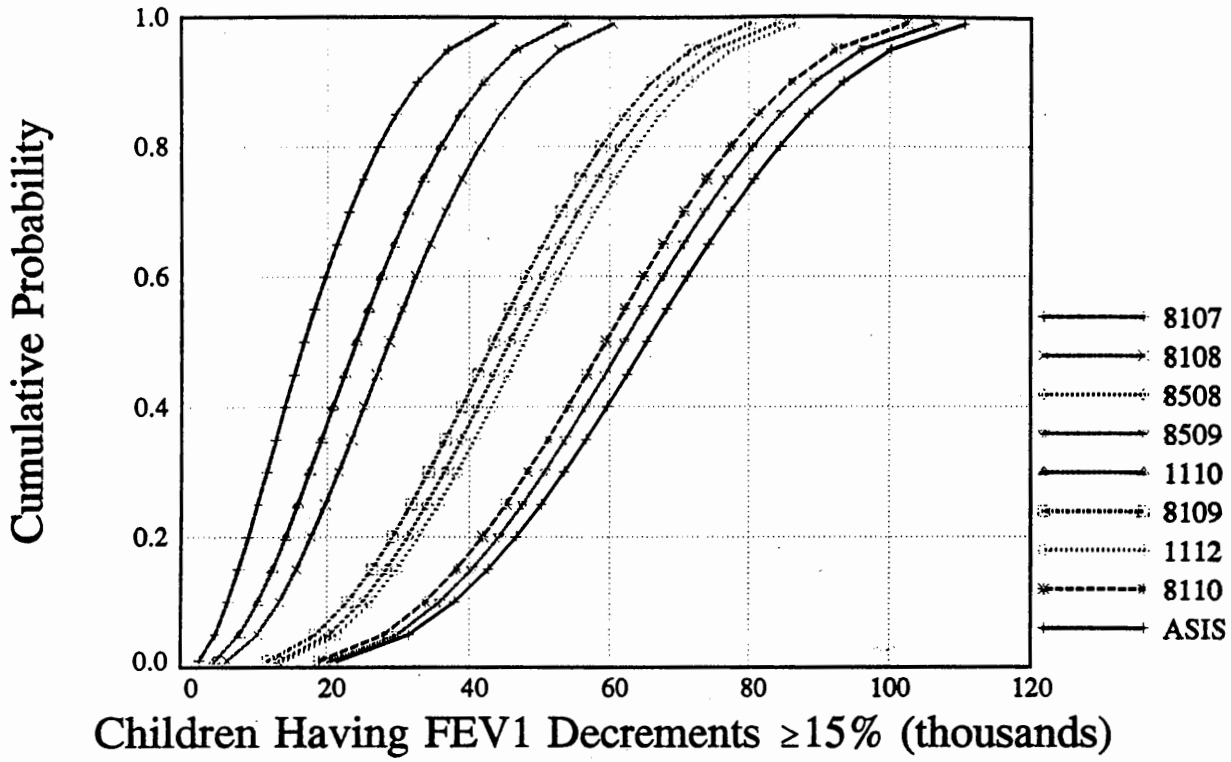
Figure C.3, which contains representative distributions, shows two sets of nine plots for FEV_1 decrement ≥15%, 8 hr exposures, moderate exertion, Chicago, and children. The top half of the figure shows representative distributions over the number of children experiencing the effect one or more times (i.e., persons basis), and the bottom half shows representative distributions over the number of times any child experiences the effect (i.e., person-occurrences basis). Each representative distribution gives some idea of the range of results among each set of 10 distributions. There is one representative distribution for each of nine air quality scenarios. Each plot is "representative" of the 10 distributions for a particular scenario. Since there are only 9 plots instead of 90, it is easier to see patterns. Each of these plots is a valid cumulative probability distribution.[1] The plot indicates, for example, that the median number of children in Chicago who may experience FEV_1 decrements ≥15% under as-is air quality is around 65 thousand. When the most stringent standard (8107) is just attained, the median estimate is about 15 thousand children.

C.2.2 Box Plots for Risk Results

Box plots provide another perspective about risk results. Each Box plot displays the ranges of the medians (or 0.5 fractile), 0.05 fractiles, and 0.95 fractiles of 10 risk distributions that result from the 10 pNEM exposure distributions that are available. These ranges are represented by rectangles in the figures (unless there is no range, in which case the rectangle

[1]The representative distribution is obtained by computing the average cumulative probability at selected points along the X-axis. This calculation, like the risk calculations described earlier, implicitly assumes that the distributions are perfectly correlated. It may be argued that perfect correlation, while not correct, is more reasonable that perfect independence, and there is no basis for choosing any other degree of correlation between these two extremes.

"collapses" into a horizontal line). There are 3 rectangles above the code letter for each standard. The top rectangle represents the range of the 0.95 fractiles, the middle rectangle represents the range of the medians, and the bottom rectangle represents the range of the 0.05 fractiles. A line connects the bottom of the 0.95-fractile rectangle and the top of the 0.05-fractile rectangle and passes through the 0.5-fractile rectangle. With this format, results for 81 scenarios (9 scenarios for each of 9 urban areas) can be displayed in one figure. For these plots, however, we switched from numbers of persons or person-occurrences to percentage of persons responding. As shown in Figure C.3, under as-is conditions in Chicago, the median risk estimates for the percentage of children having FEV_1 decrements ≥15% vary from 13-14%, the 0.95 fractiles vary from 21-22%, and the 0.05 fractiles vary from 7-8%. Box plots have the following characteristics. If the risk distributions for a particular air quality scenario are quite "similar," the rectangles will be small. If the variance of a risk distribution is small, the rectangles will be close together. If the distributions are spaced far enough apart (indicative of widely varying risk estimates for different pNEM runs), rectangles will overlap.



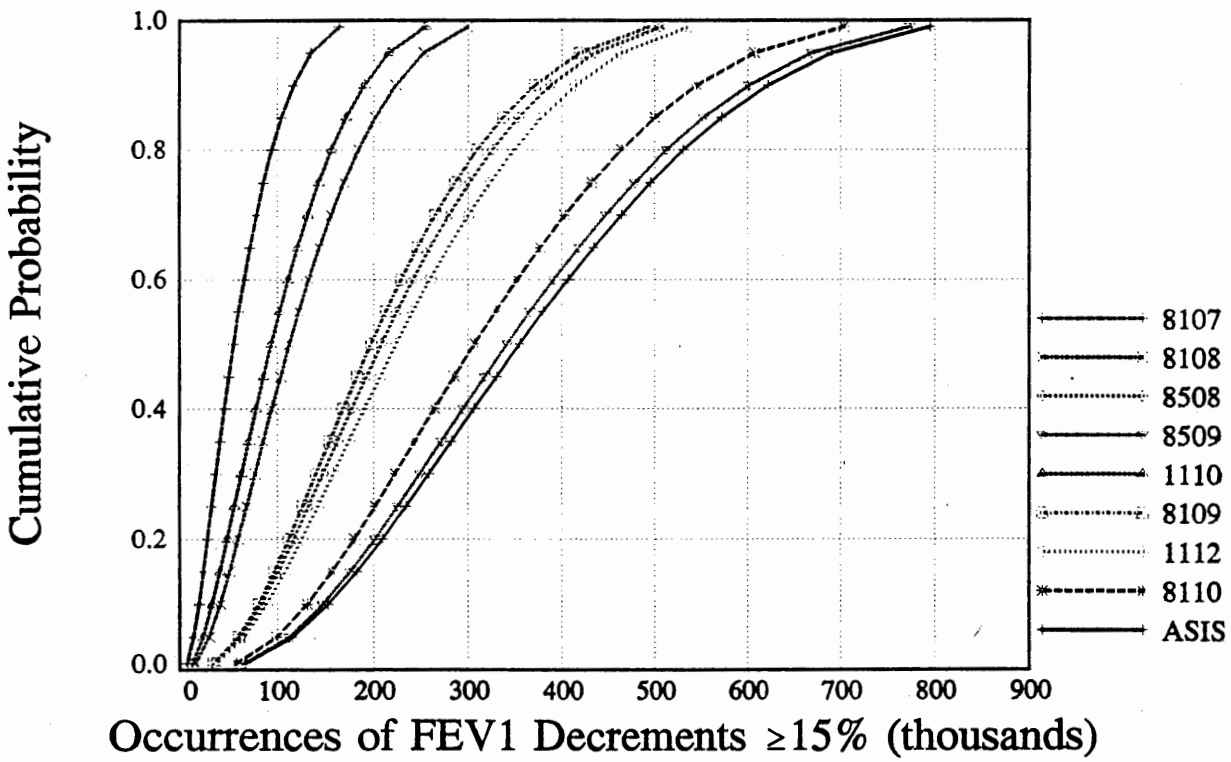
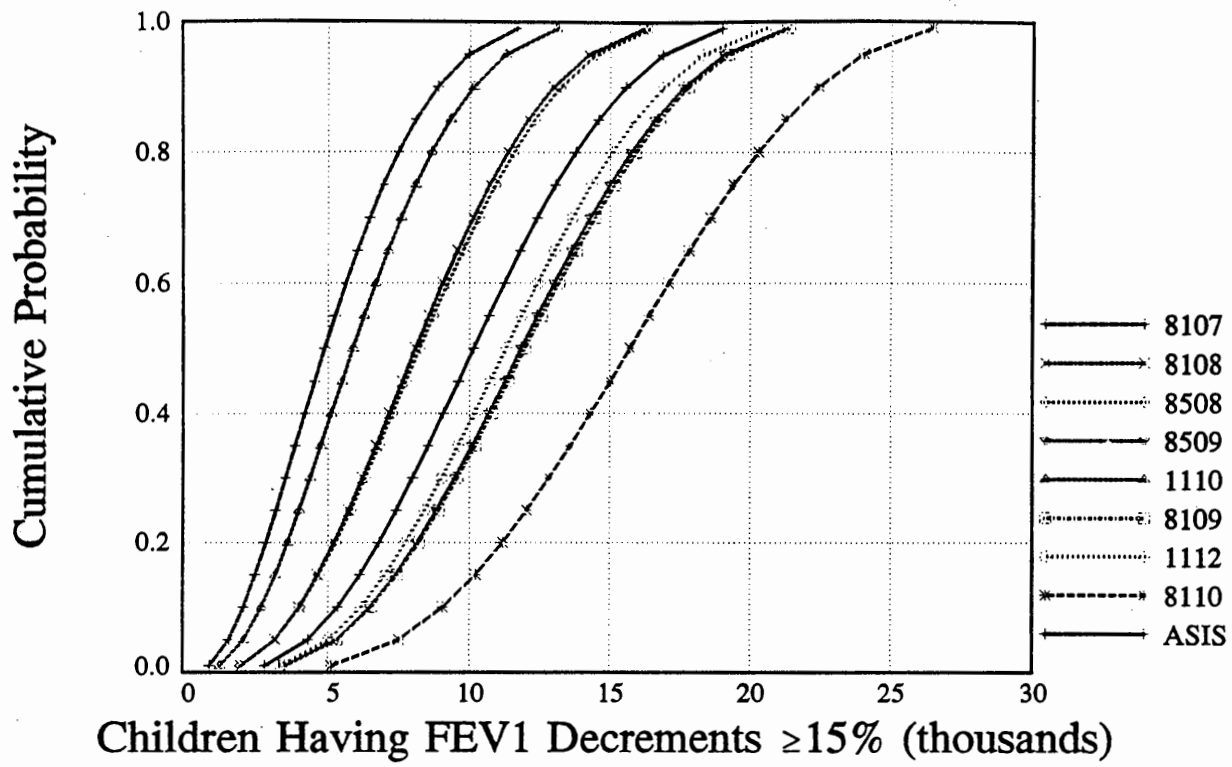


FIGURE C.3 Representative Risk Distributions for Alternative Air Quality Scenarios (FEV_1 Decrements ≥ 15%, Chicago, Outdoor Children, 8 Hr Exposures, Moderate Exertion)



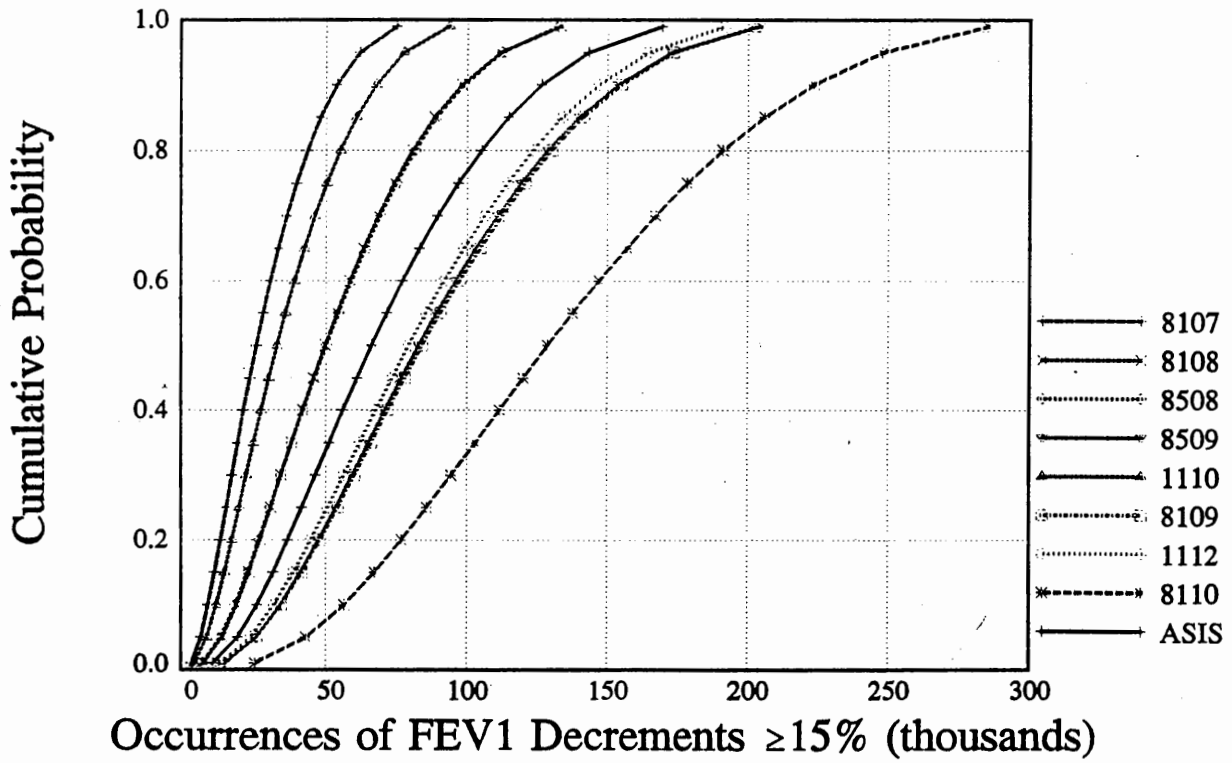
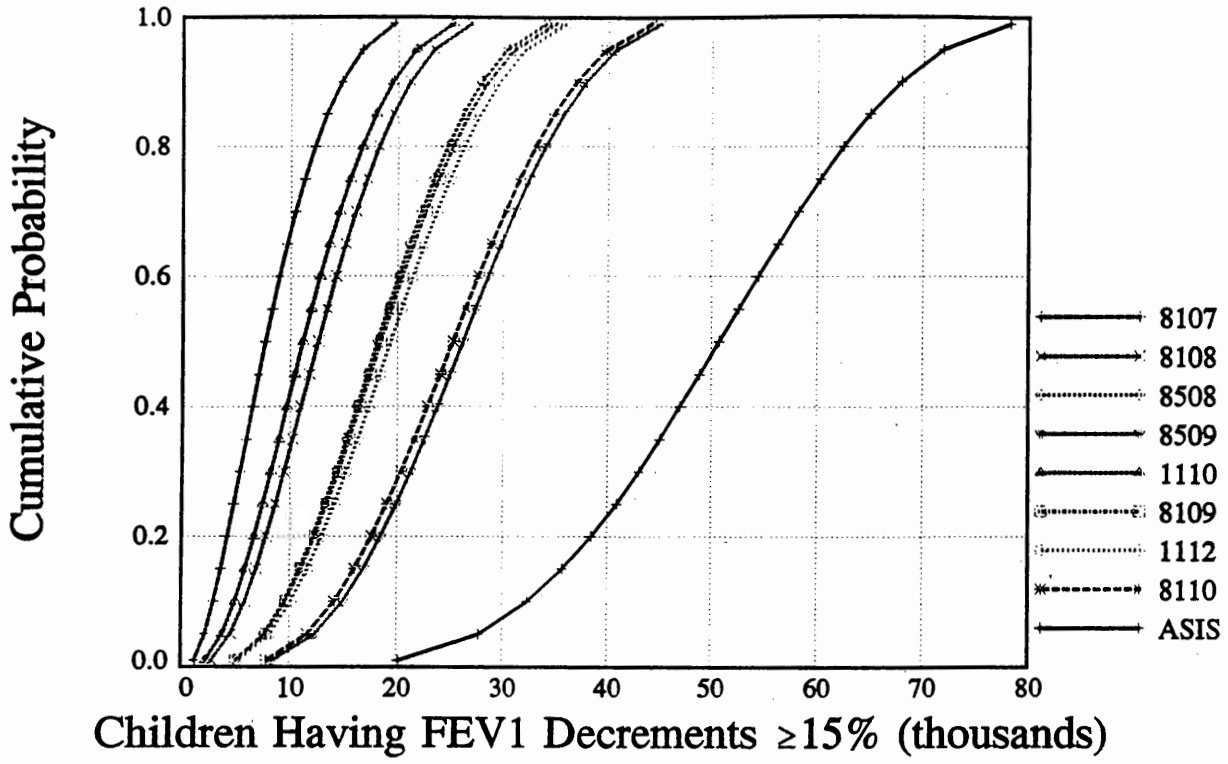


FIGURE C.4 Representative Risk Distributions for Alternative Air Quality Scenarios (FEV_1 Decrements ≥ 15%, Denver, Outdoor Children, 8 Hr Exposures, Moderate Exertion)



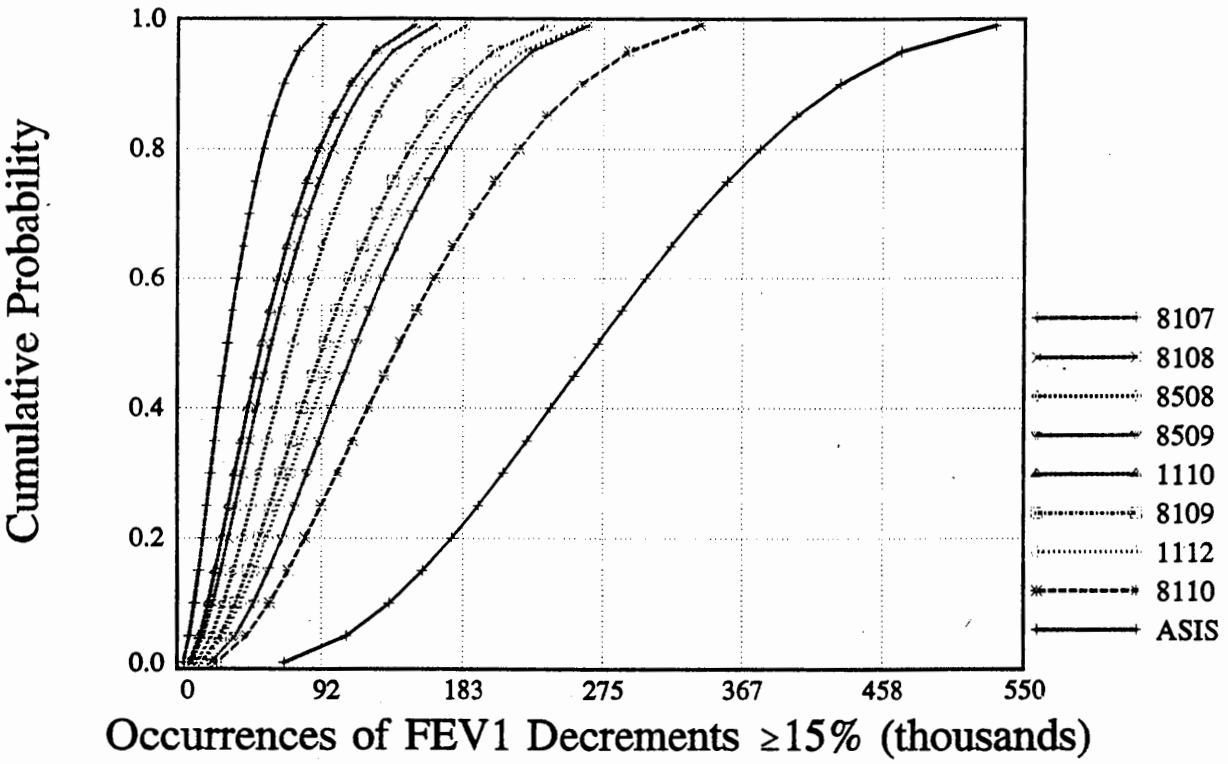
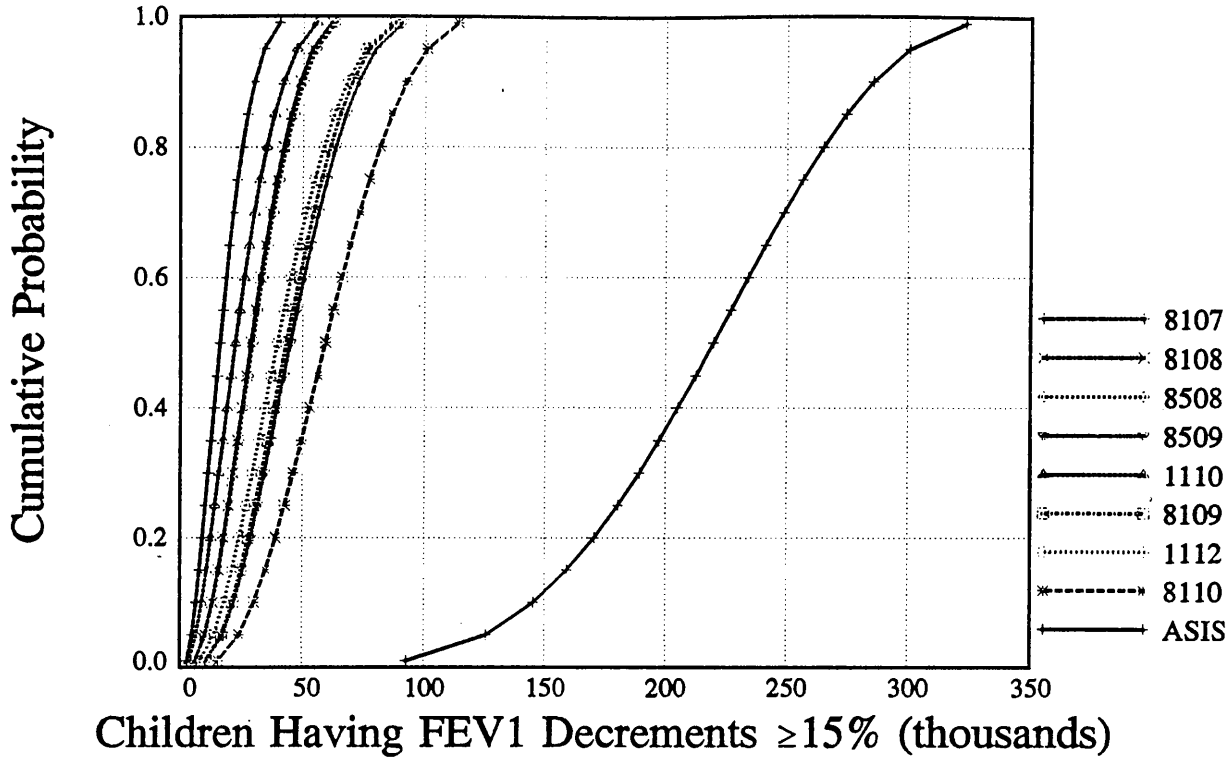


FIGURE C.5 Representative Risk Distributions for Alternative Air Quality Scenarios (FEV_1 Decrements ≥ 15%, Houston, Outdoor Children, 8 Hr Exposures, Moderate Exertion)



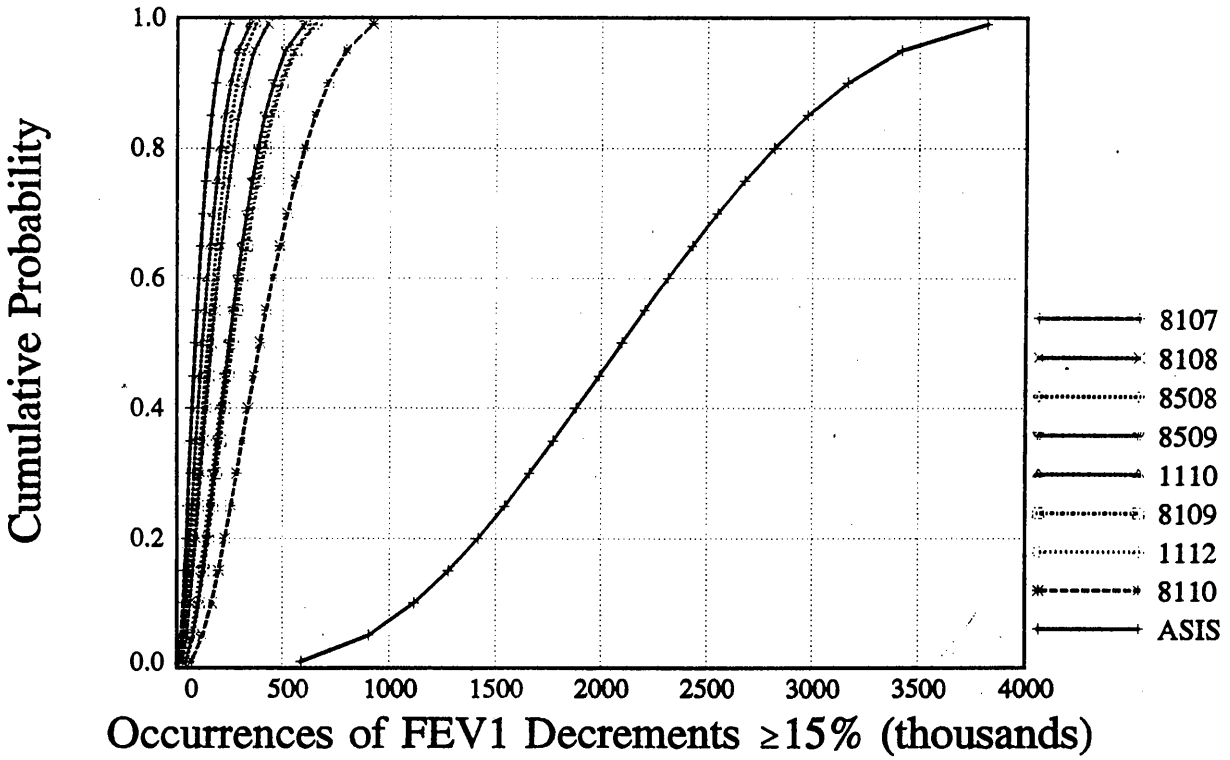


FIGURE C.6 Representative Risk Distributions for Alternative Air Quality Scenarios (FEV_1 Decrements ≥ 15%, Los Angeles, Outdoor Children, 8 Hr Exposures, Moderate Exertion)

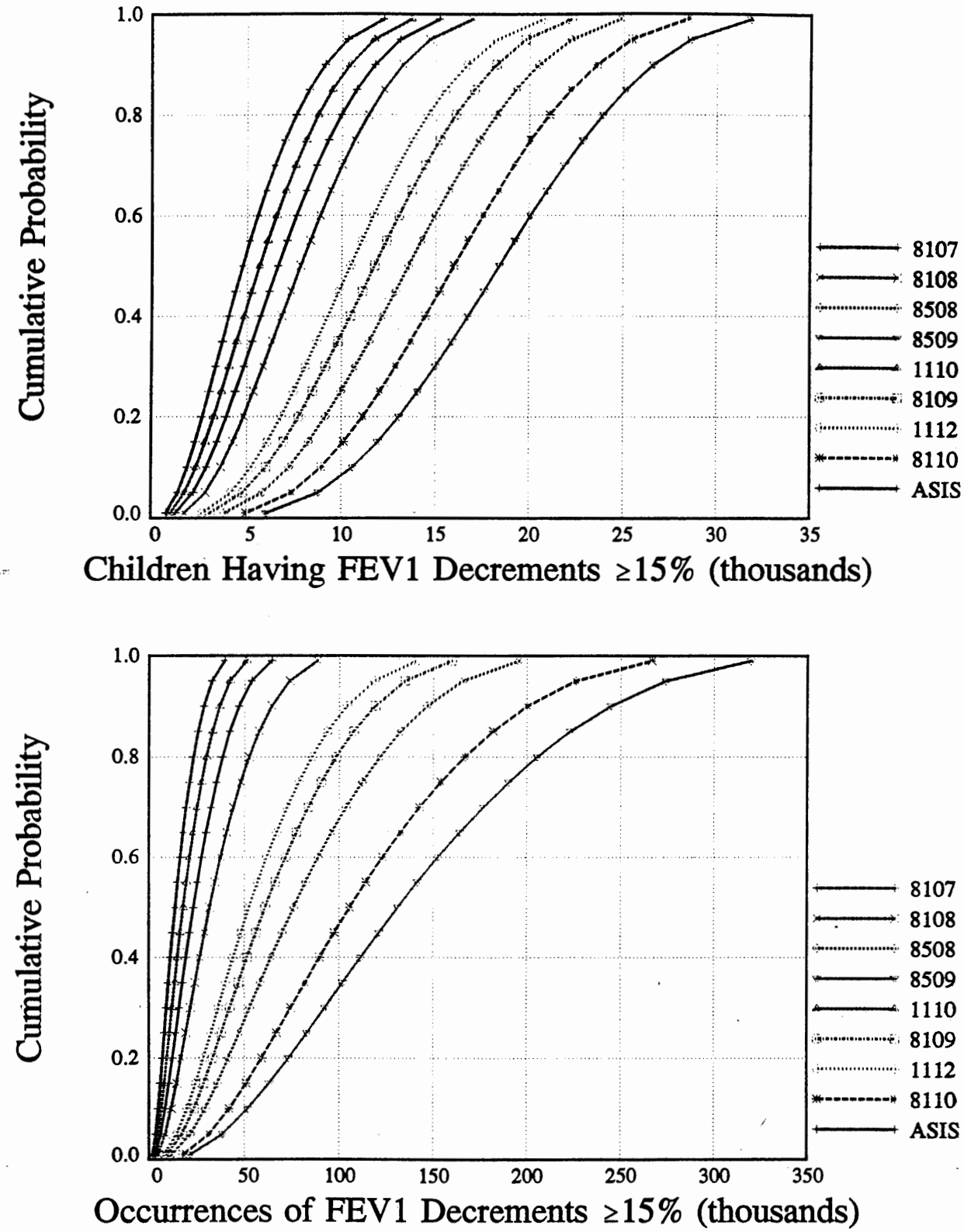
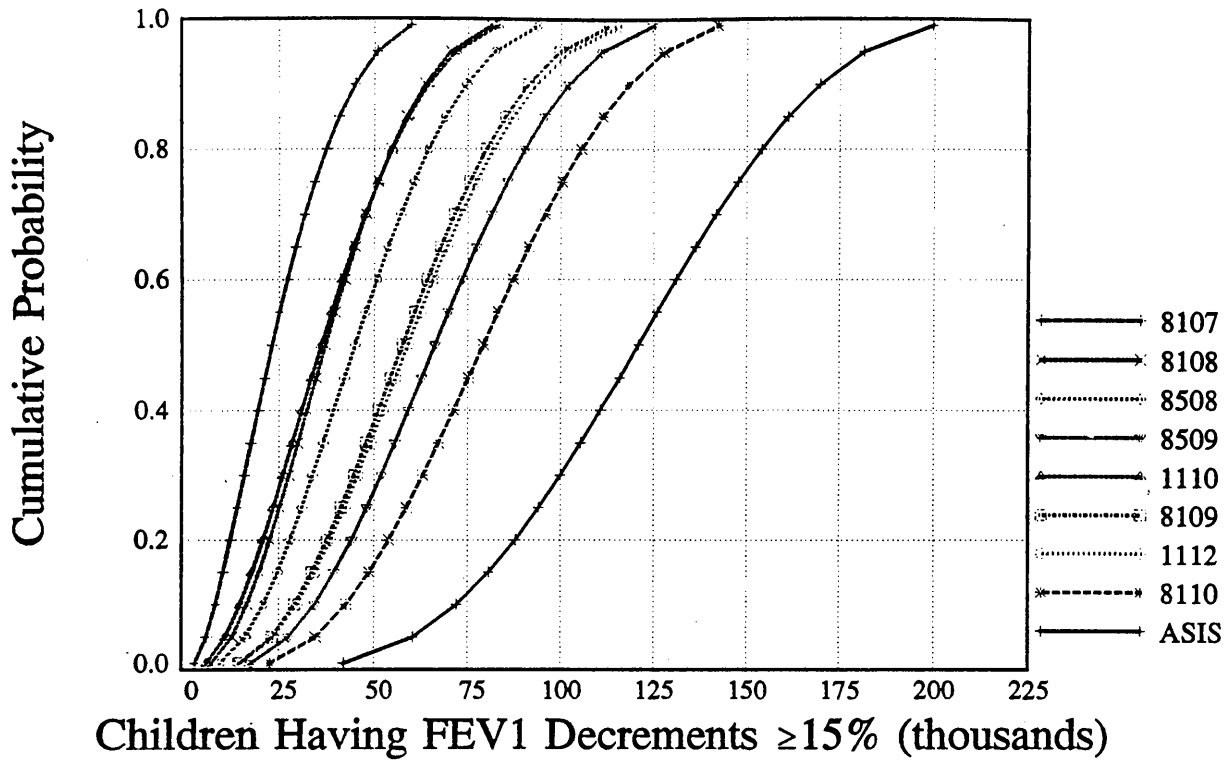


FIGURE C.7 Representative Risk Distributions for Alternative Air Quality Scenarios (FEV_1 Decrements ≥ 15%, Miami, Outdoor Children, 8 Hr Exposures, Moderate Exertion)



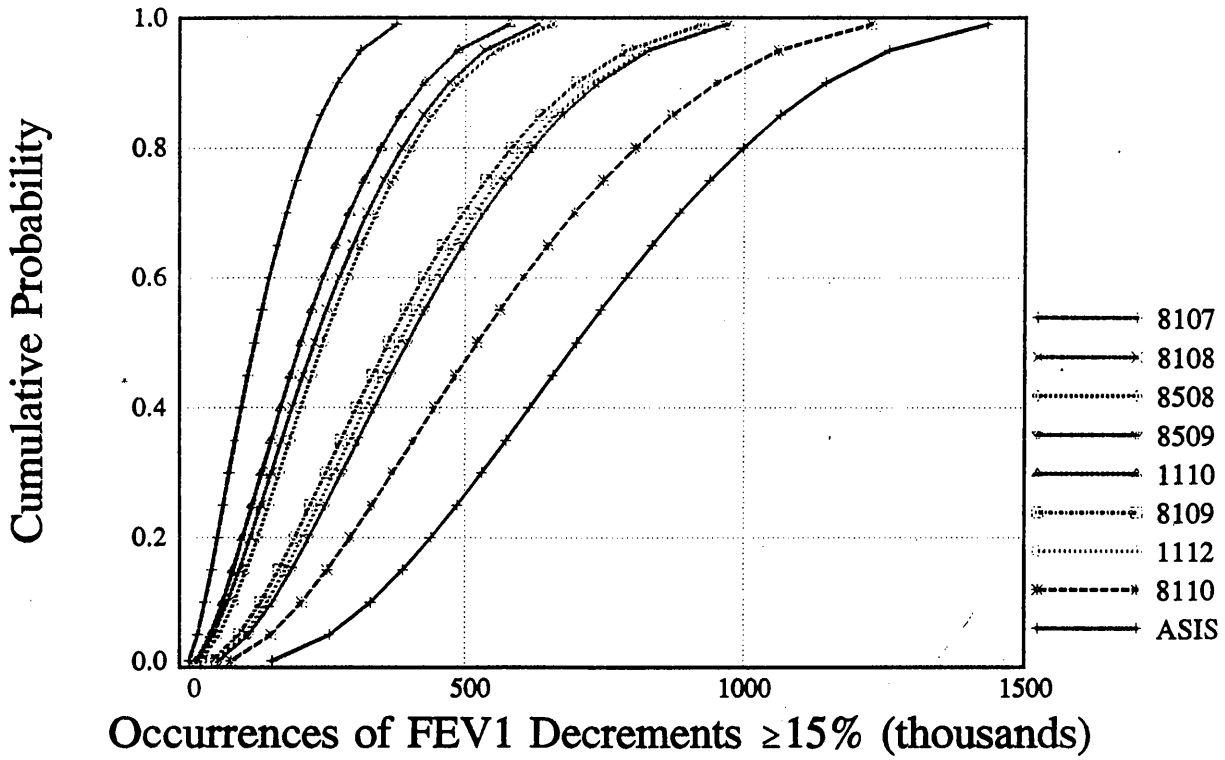
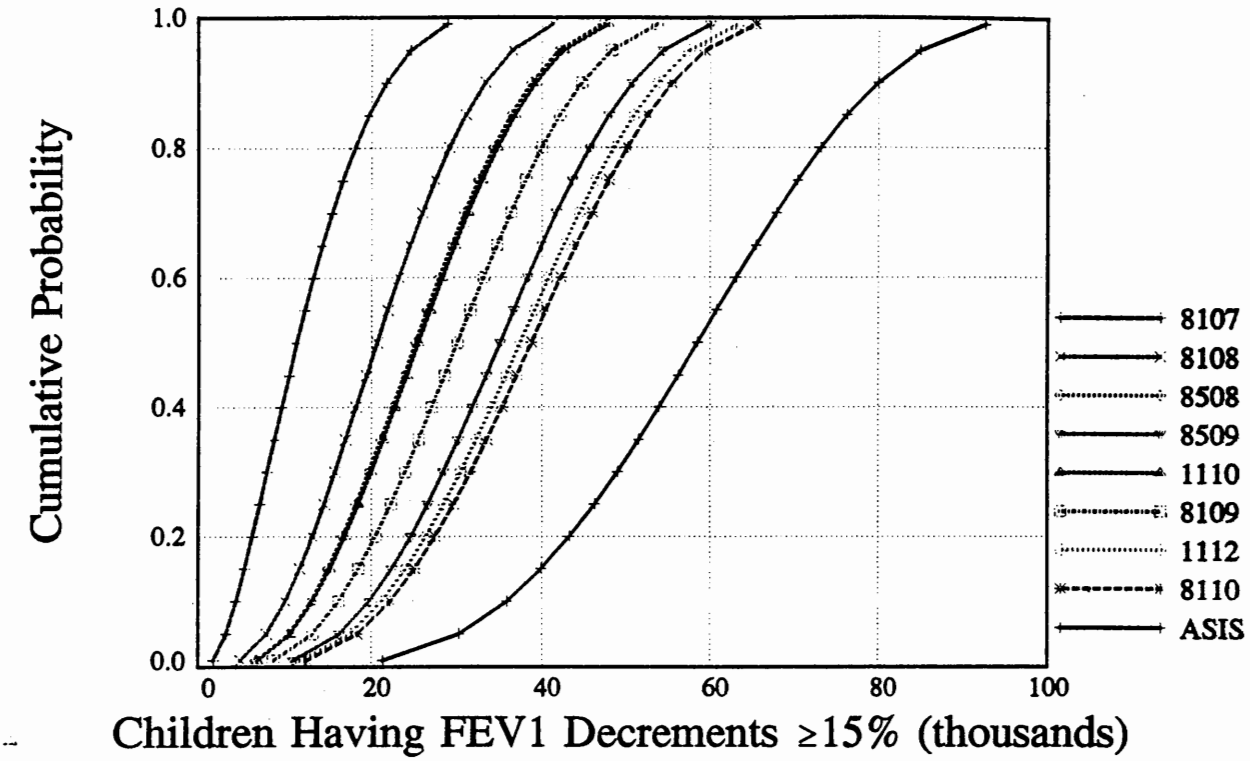


FIGURE C.8 Representative Risk Distributions for Alternative Air Quality Scenarios (FEV_1 Decrements ≥ 15%, New York, Outdoor Children, 8 Hr Exposures, Moderate Exertion)



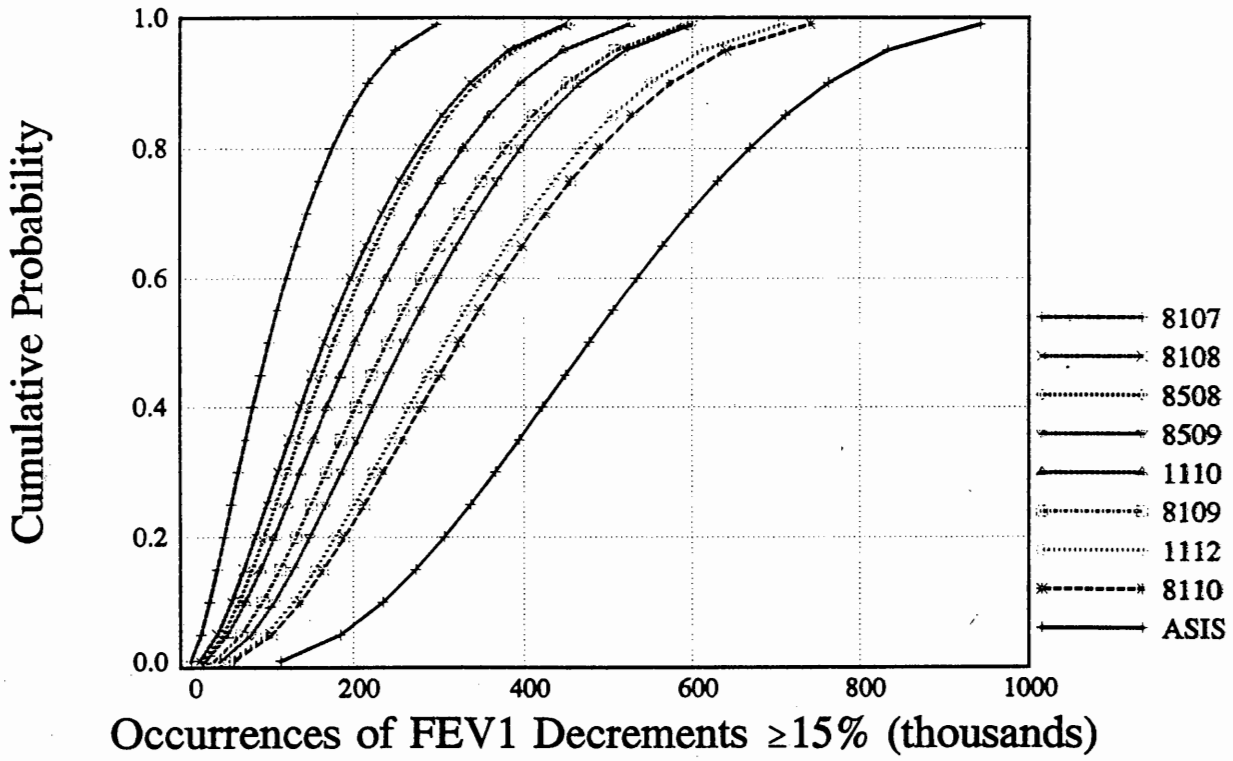
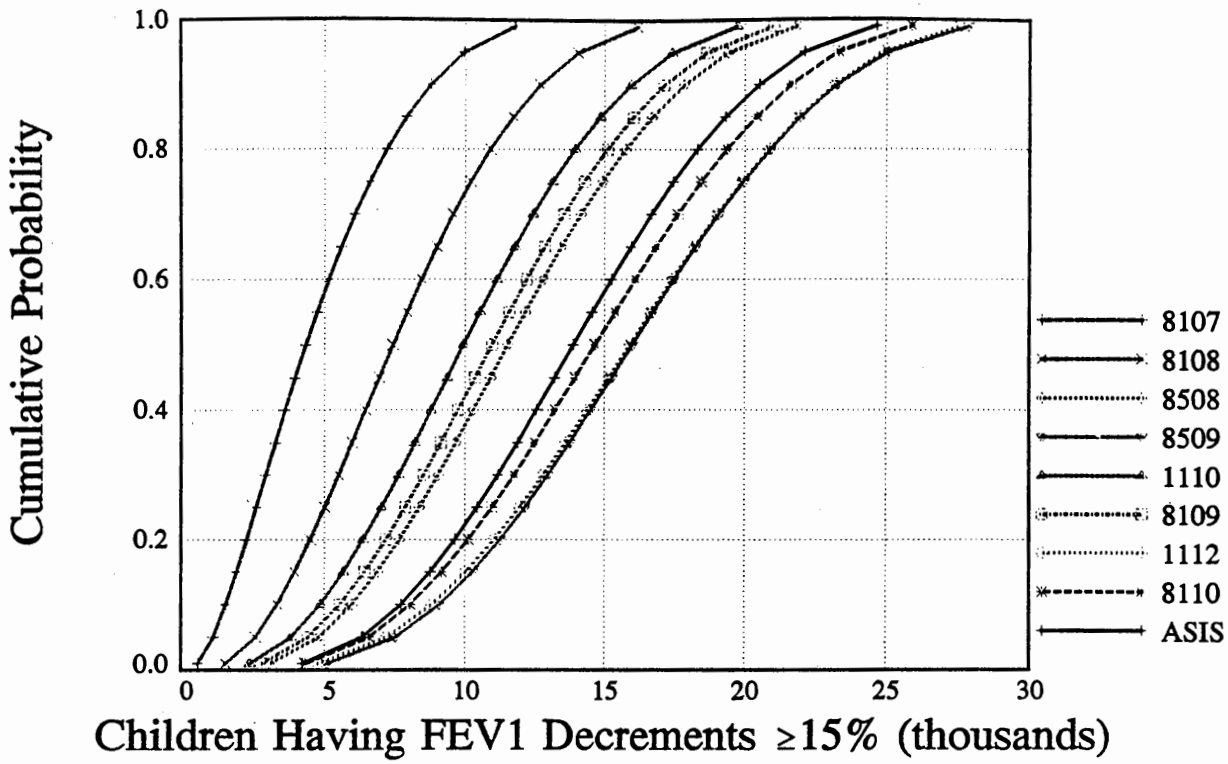


FIGURE C.9 Representative Risk Distributions for Alternative Air Quality Scenarios (FEV_1 Decrements ≥ 15%, Philadelphia, Outdoor Children, 8 Hr Exposures, Moderate Exertion)



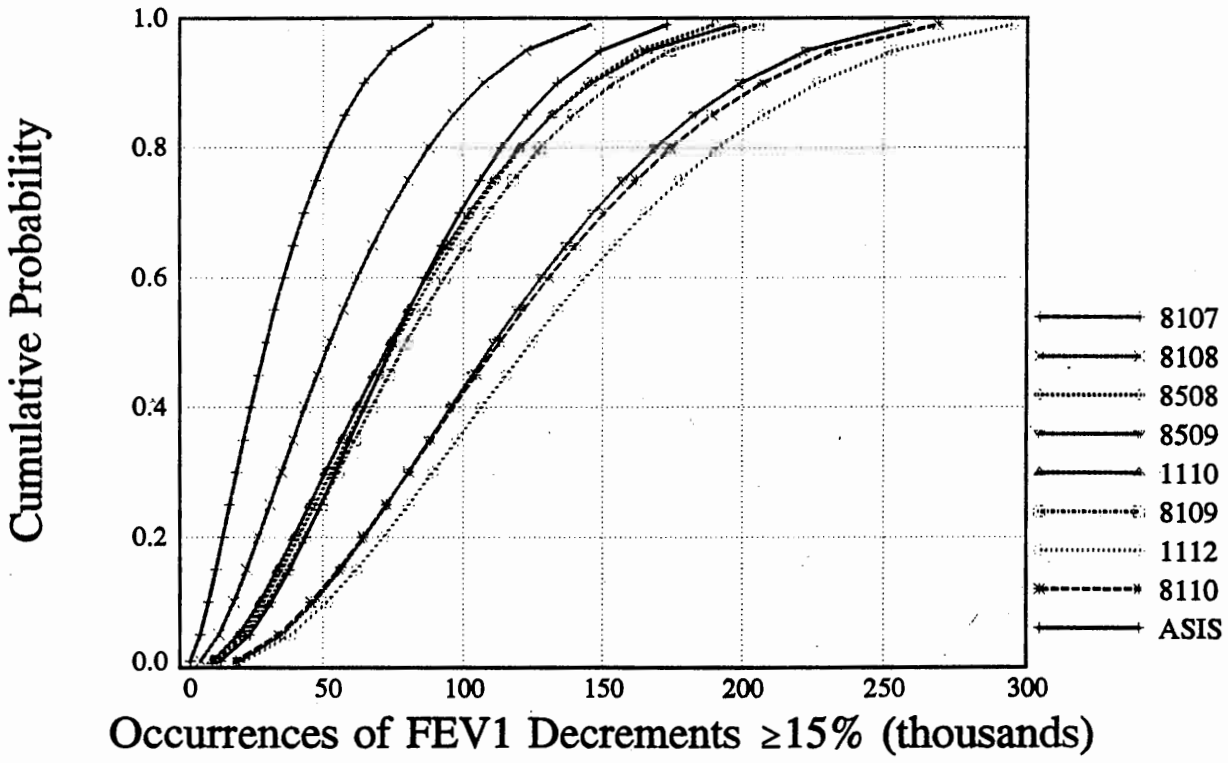
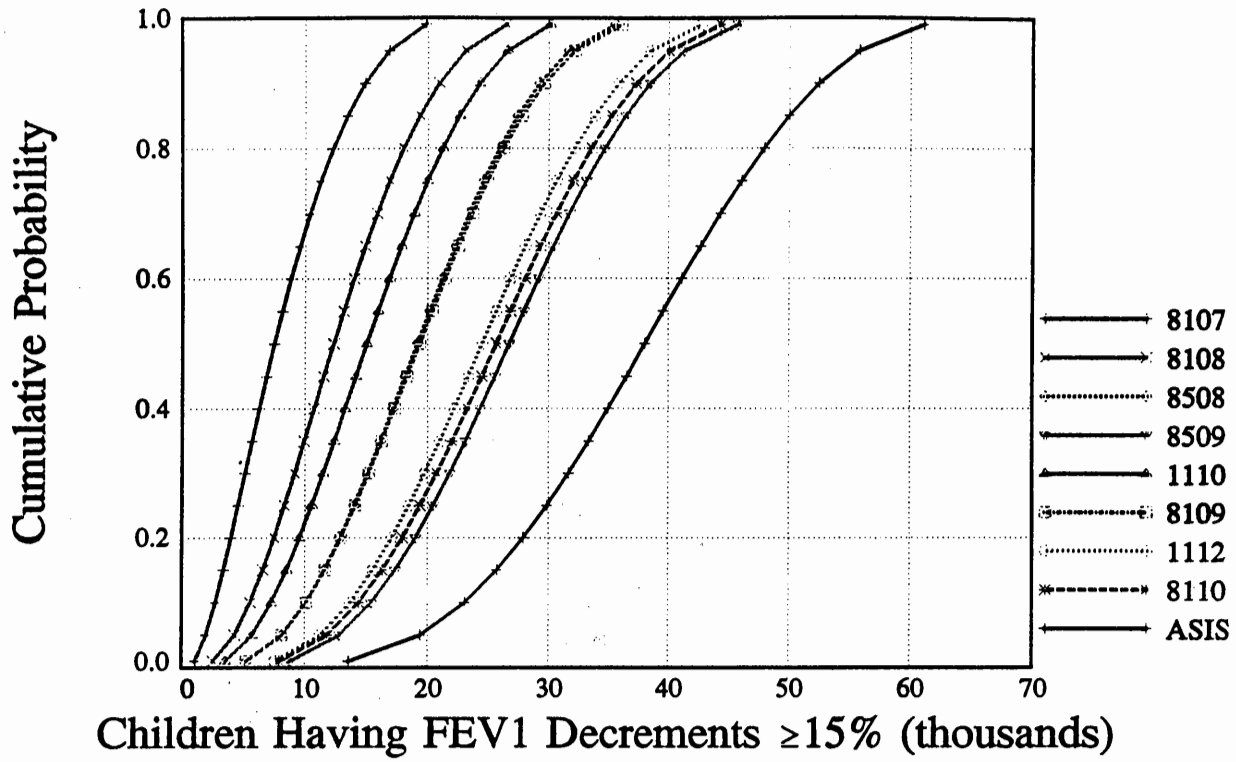


FIGURE C.10 Representative Risk Distributions for Alternative Air Quality Scenarios (FEV_1 Decrements ≥ 15%, St. Louis, Outdoor Children, 8 Hr Exposures, Moderate Exertion)



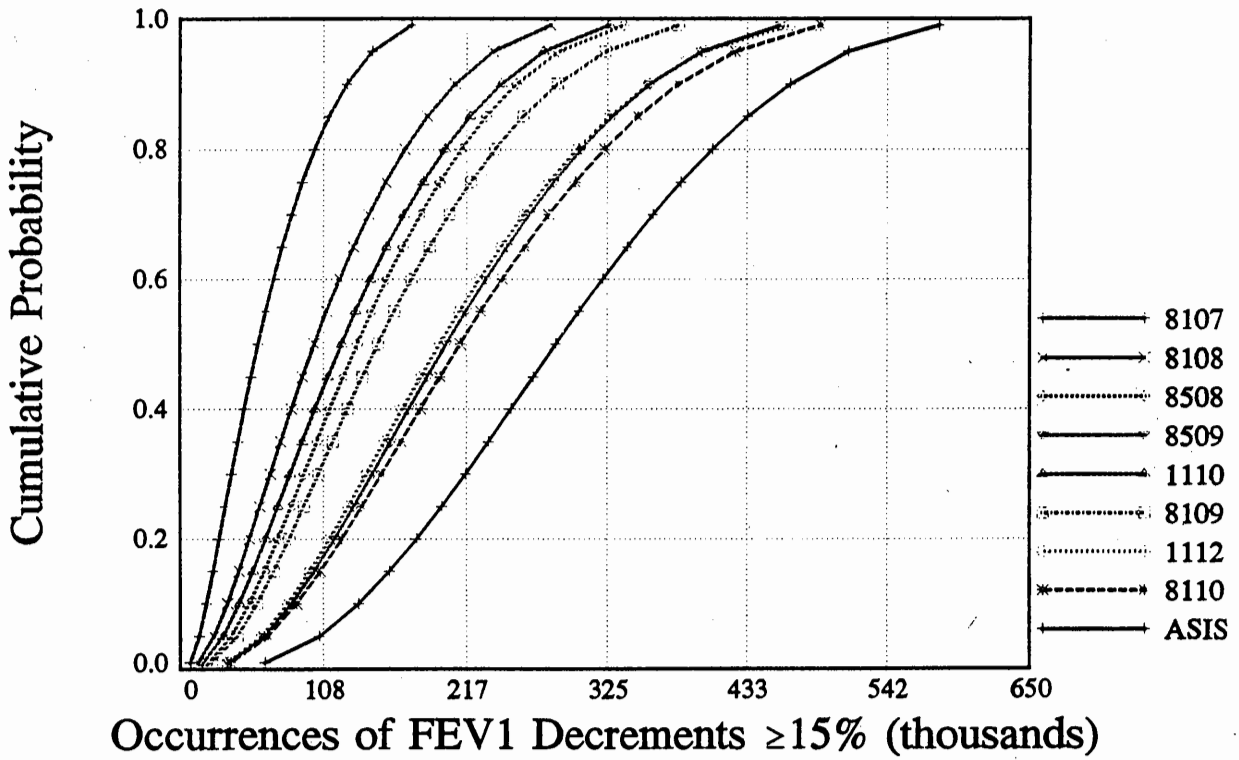


FIGURE C.11 Representative Risk Distributions for Alternative Air Quality Scenarios (FEV_1 Decrements ≥ 15%, Washington DC, Outdoor Children, 8 Hr Exposures, Moderate Exertion)

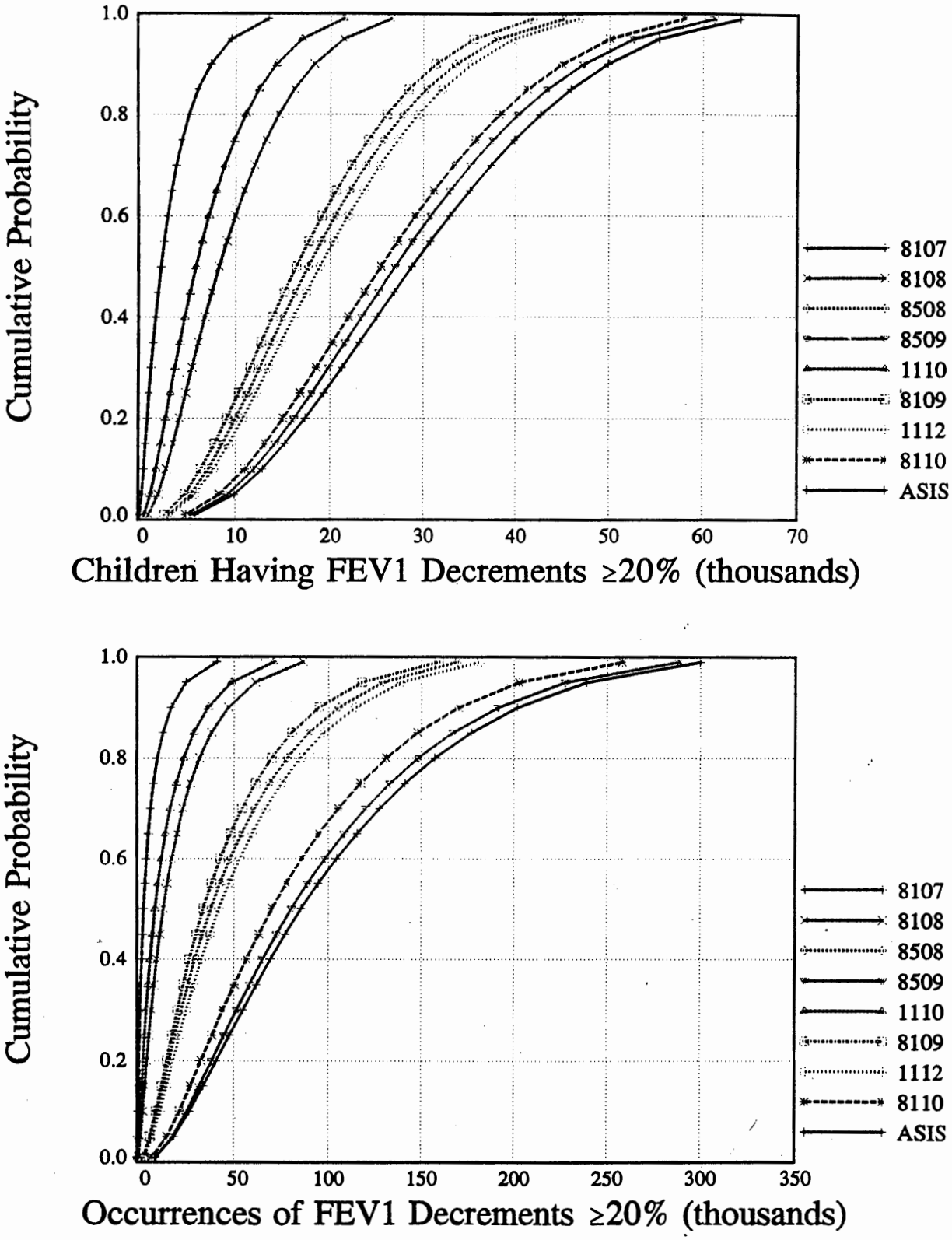
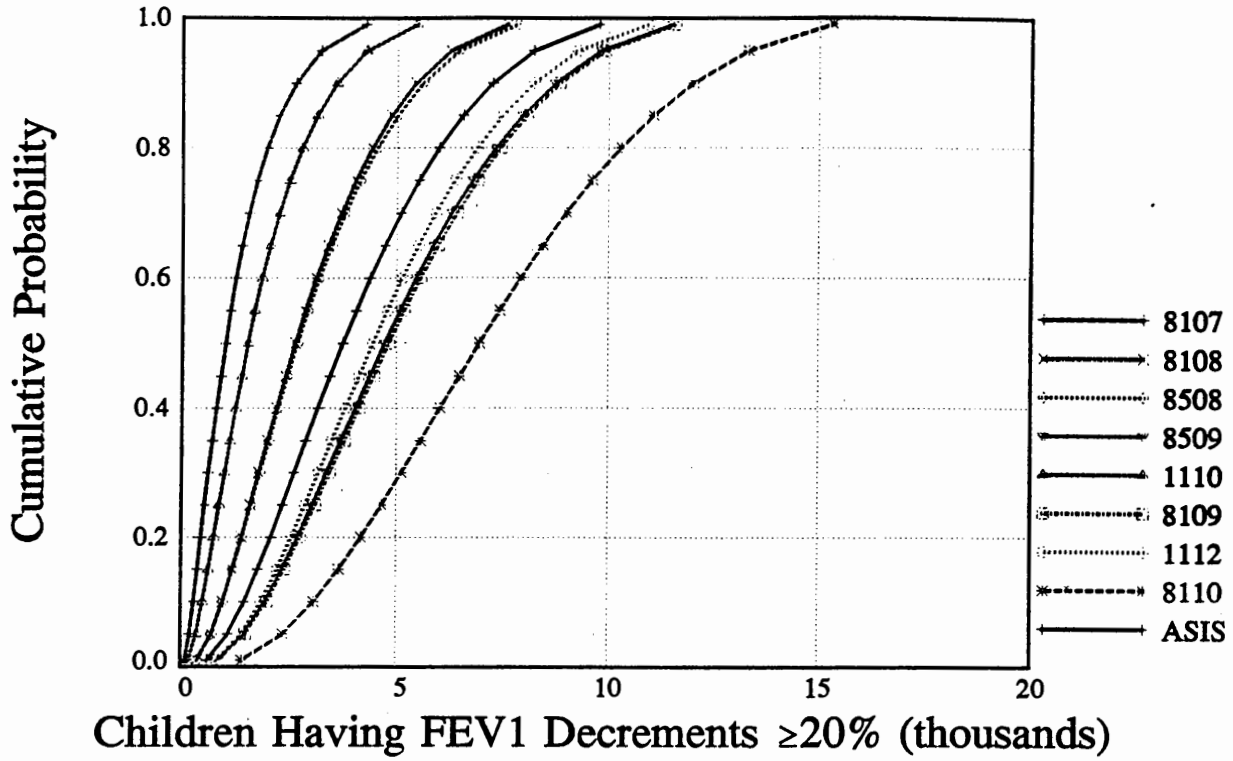


FIGURE C.12 Representative Risk Distributions for Alternative Air Quality Scenarios (FEV_1 Decrements ≥ 20%, Chicago, Outdoor Children, 8 Hr Exposures, Moderate Exertion)



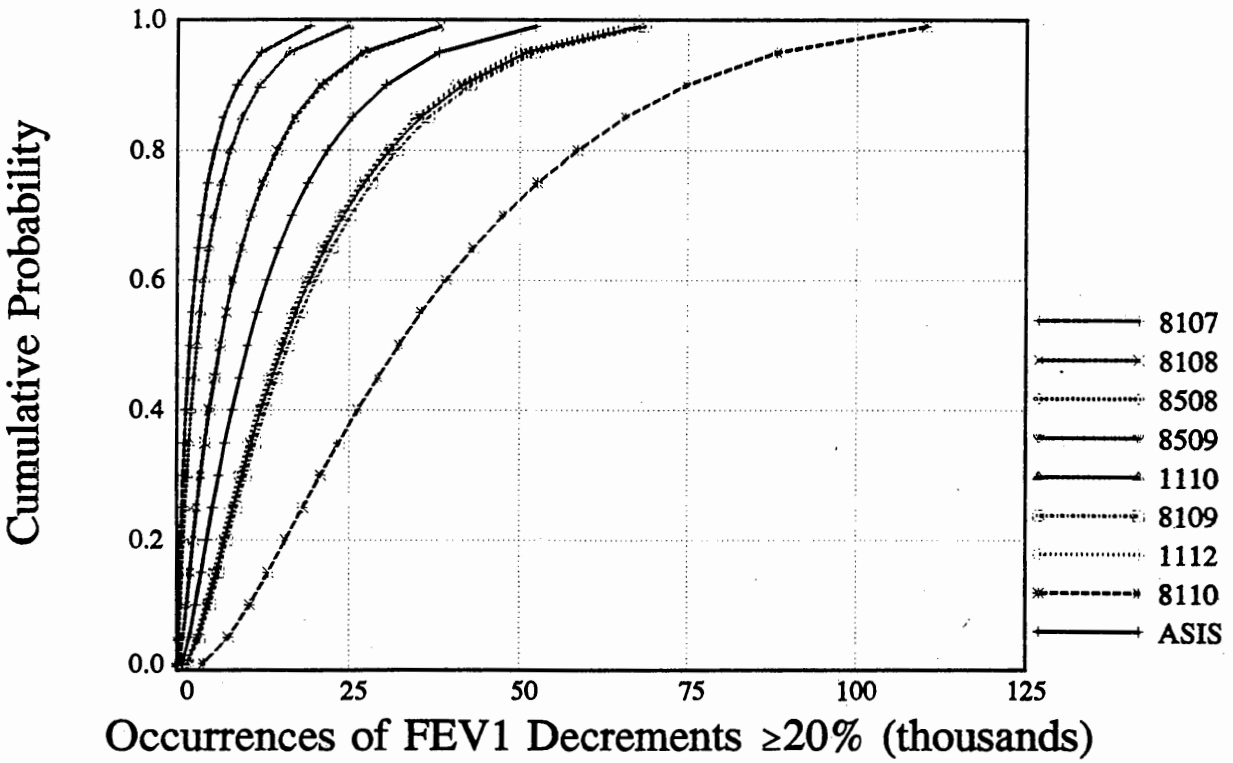


FIGURE C.13 Representative Risk Distributions for Alternative Air Quality Scenarios (FEV_1 Decrements ≥ 20%, Denver, Outdoor Children, 8 Hr Exposures, Moderate Exertion)

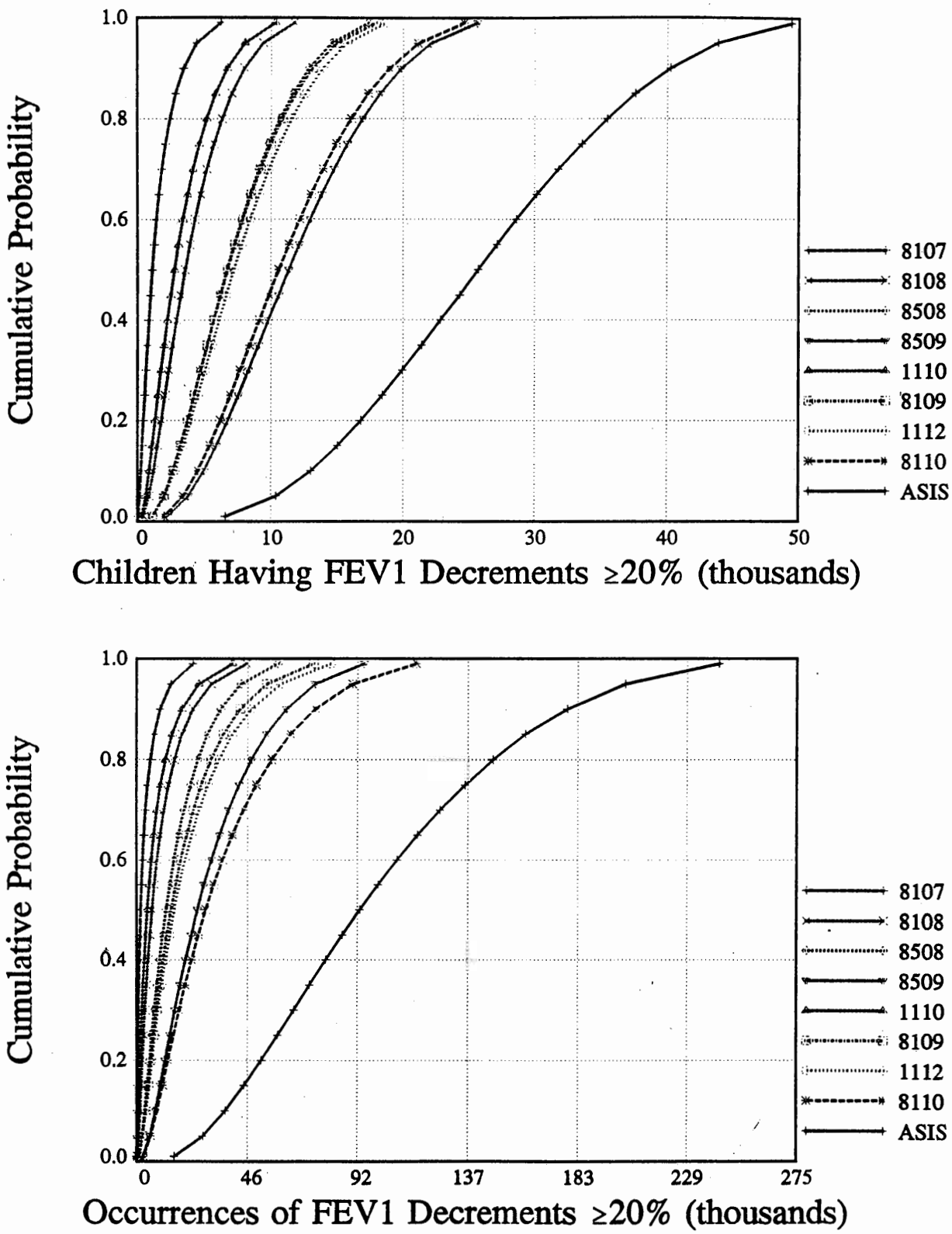
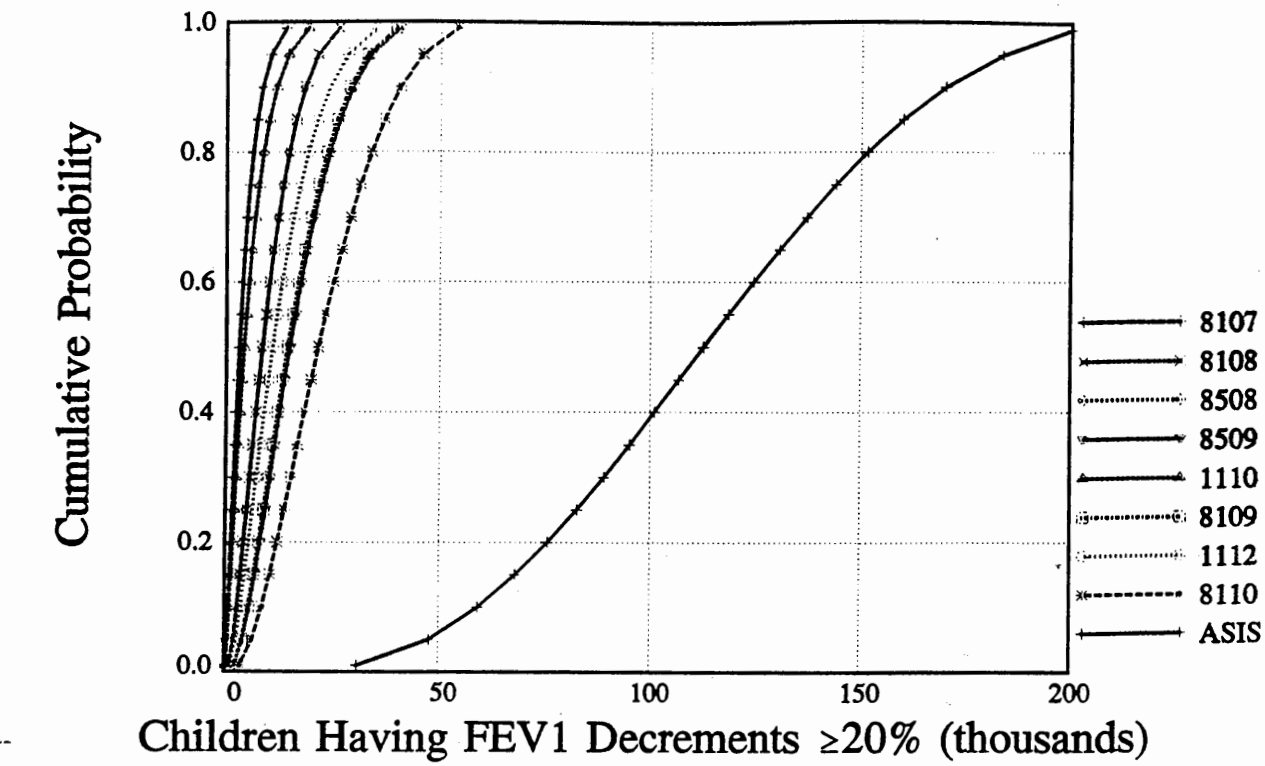


FIGURE C.14 Representative Risk Distributions for Alternative Air Quality Scenarios (FEV_1 Decrements ≥ 20%, Houston, Outdoor Children, 8 Hr Exposures, Moderate Exertion)



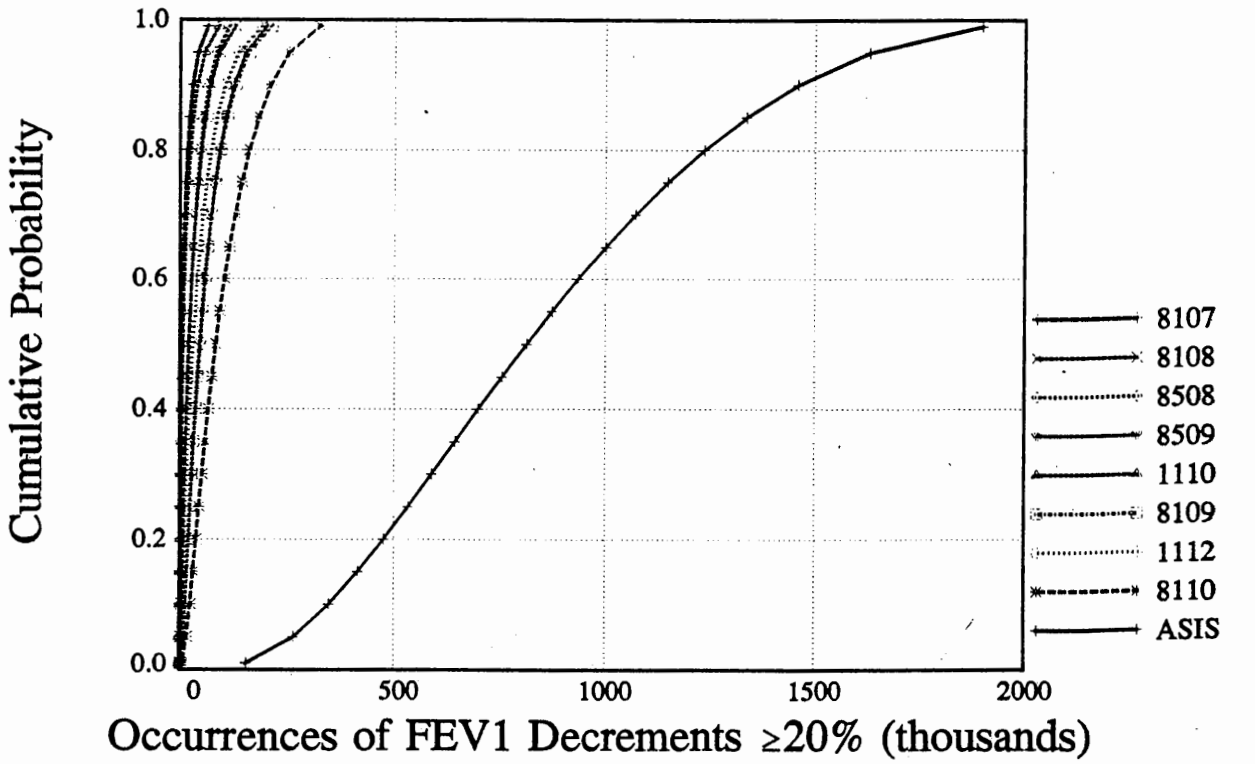


FIGURE C.15 Representative Risk Distributions for Alternative Air Quality Scenarios (FEV_1 Decrements ≥ 20%, Los Angeles, Outdoor Children, 8 Hr Exposures, Moderate Exertion)

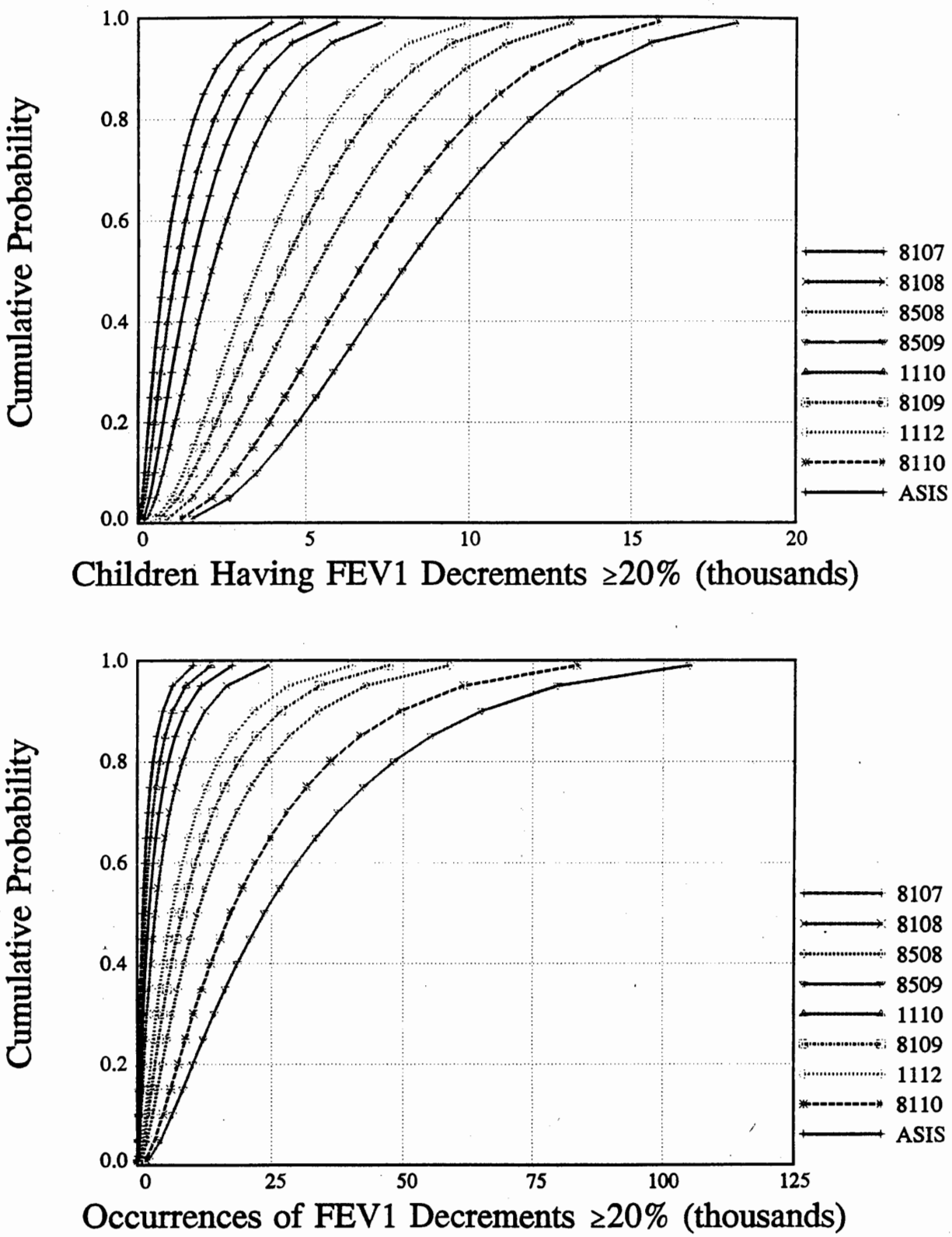


FIGURE C.16 Representative Risk Distributions for Alternative Air Quality Scenarios (FEV_1 Decrements ≥ 20%, Miami, Outdoor Children, 8 Hr Exposures, Moderate Exertion)

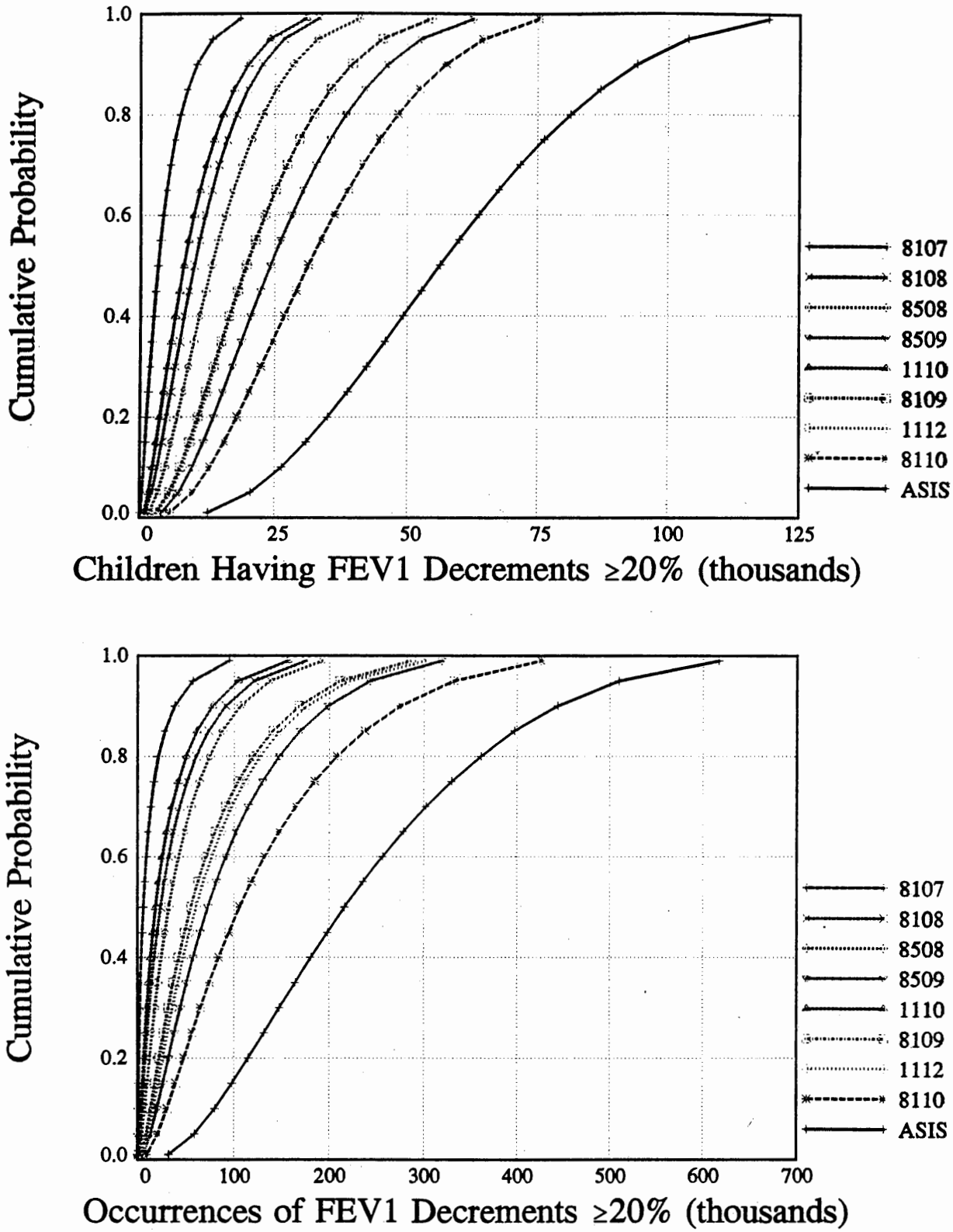


FIGURE C.17 Representative Risk Distributions for Alternative Air Quality Scenarios (FEV_1 Decrements ≥ 20%, New York, Outdoor Children, 8 Hr Exposures, Moderate Exertion)

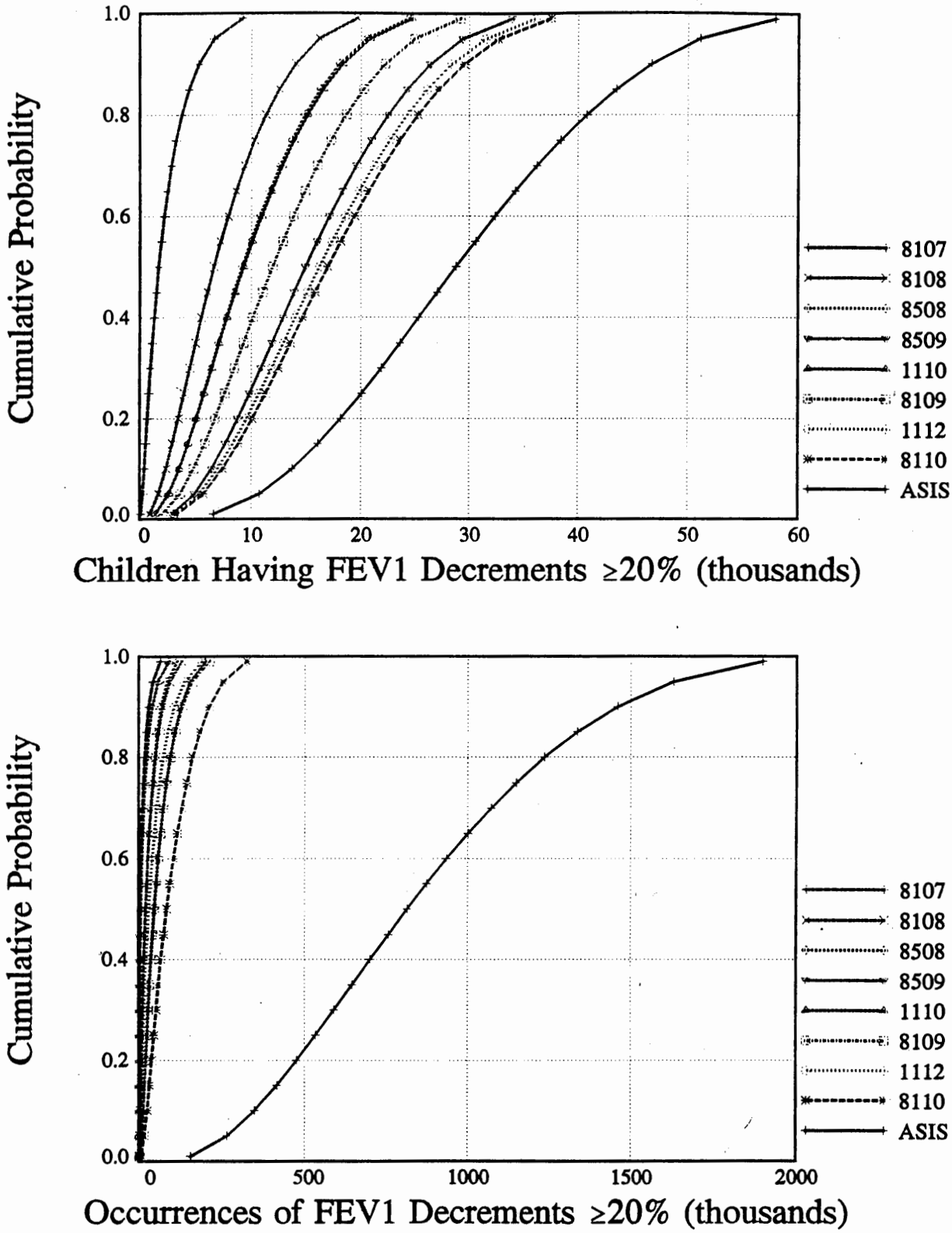
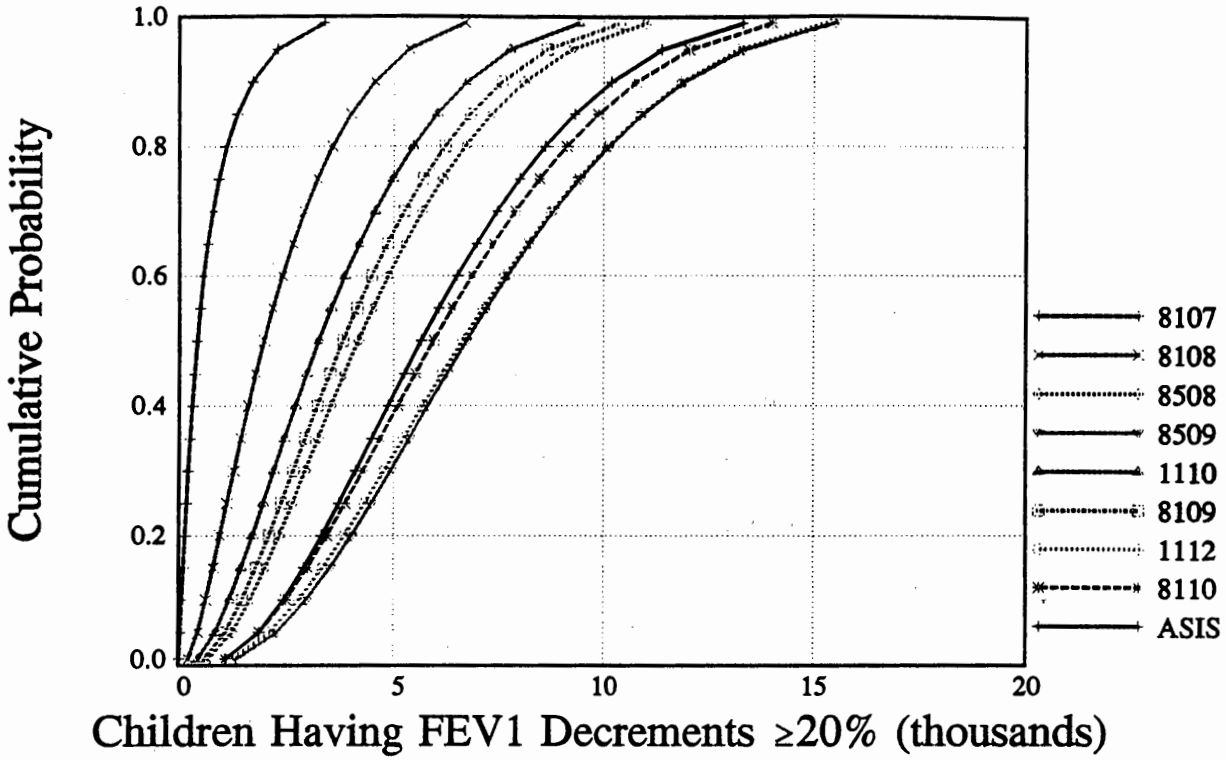


FIGURE C.17 Representative Risk Distributions for Alternative Air Quality Scenarios (FEV_1 Decrements ≥ 20%, Philadelphia, Outdoor Children, 8 Hr Exposures, Moderate Exertion)



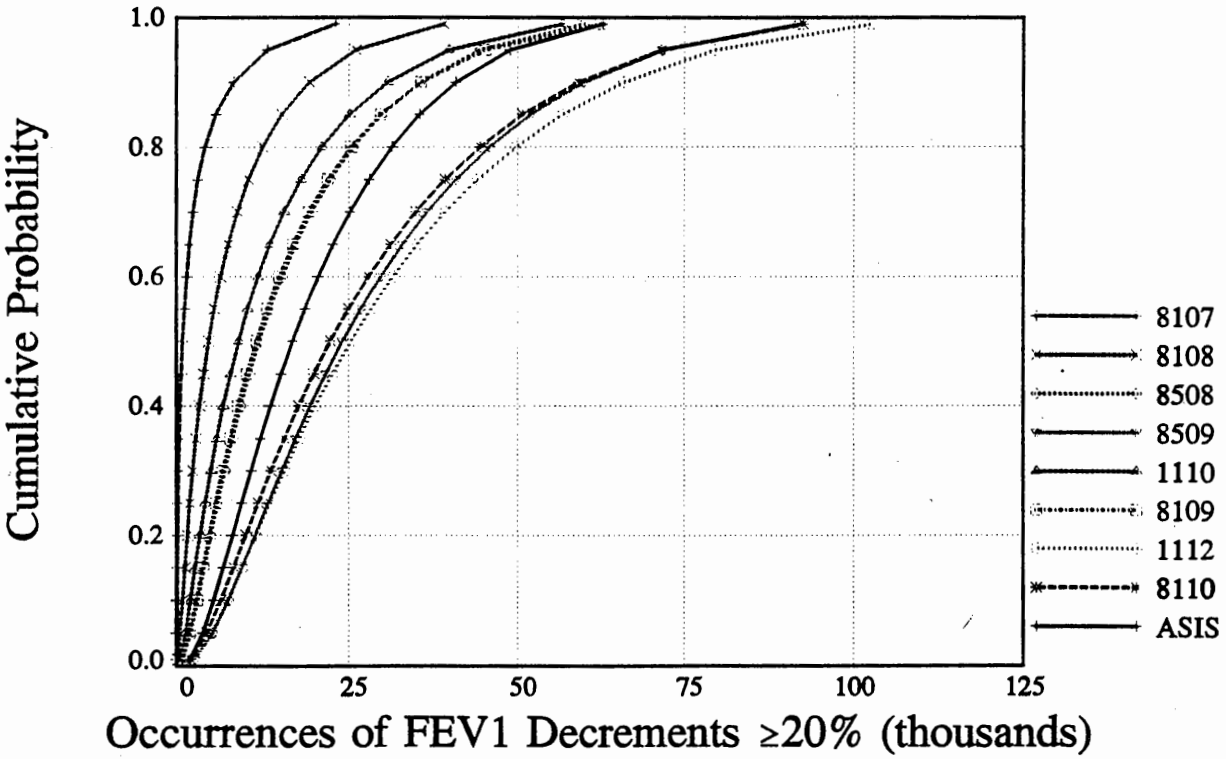


FIGURE C.19 Representative Risk Distributions for Alternative Air Quality Scenarios (FEV_1 Decrements ≥ 20%, St. Louis, Outdoor Children, 8 Hr Exposures, Moderate Exertion)

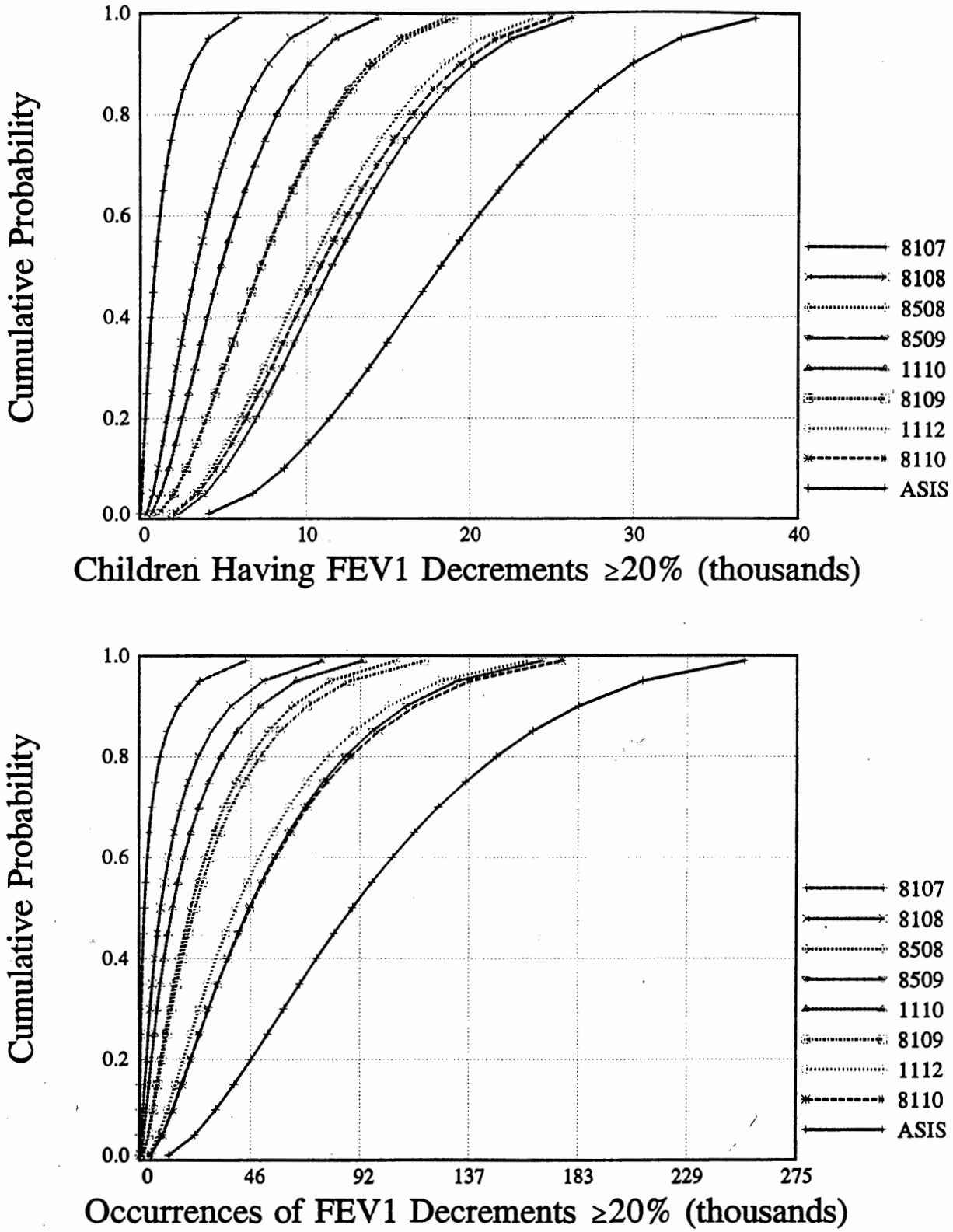


FIGURE C.20 Representative Risk Distributions for Alternative Air Quality Scenarios (FEV_1 Decrements ≥ 20%, Washington, D.C., Outdoor Children, 8 Hr Exposures, Moderate Exertion)

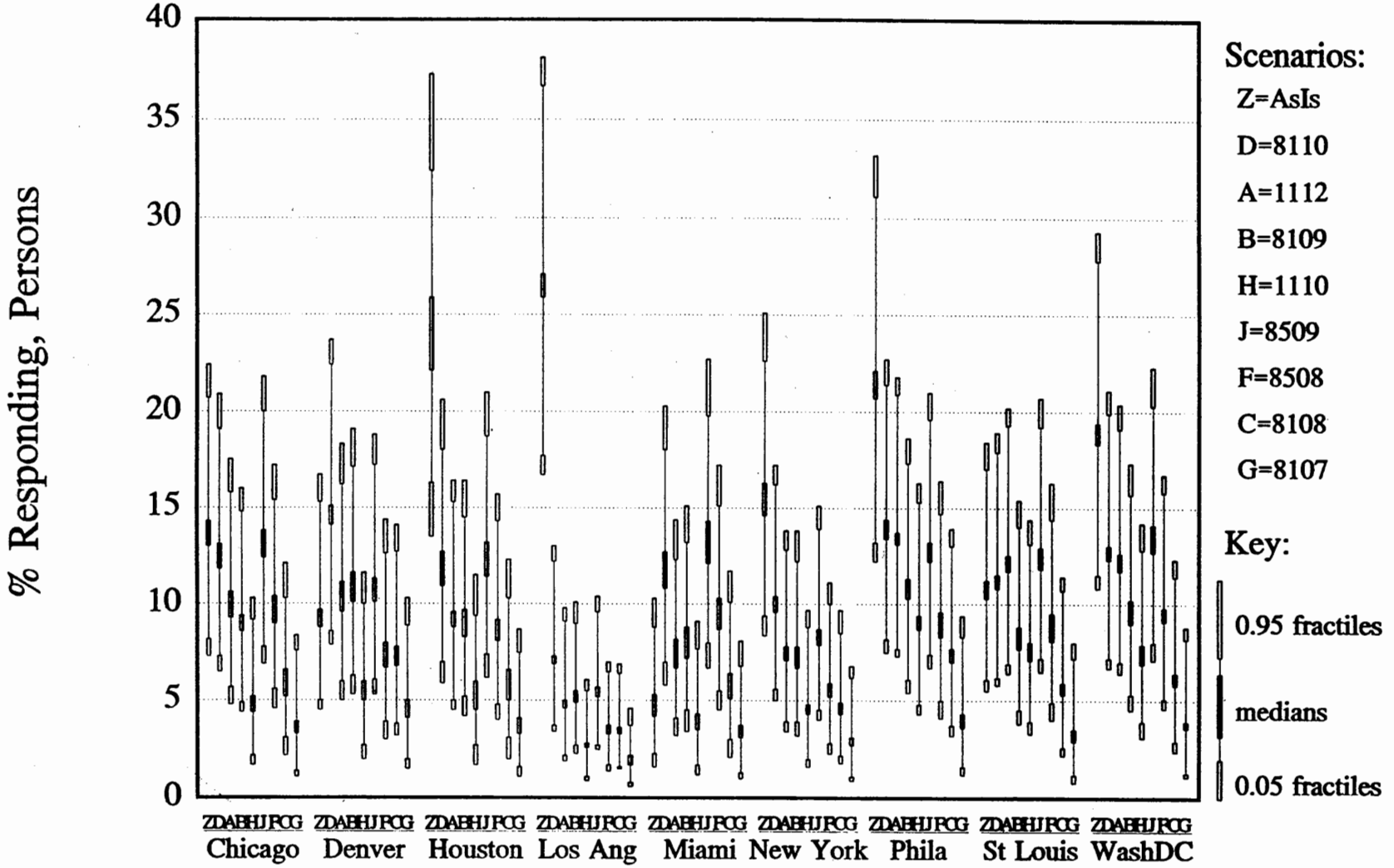


FIGURE C.21 Headcount Risk Results for the Percentage of Children Responding for the Eight-Hr, Moderate Exertion, FEV_1 Decrements ≥15% Endpoint

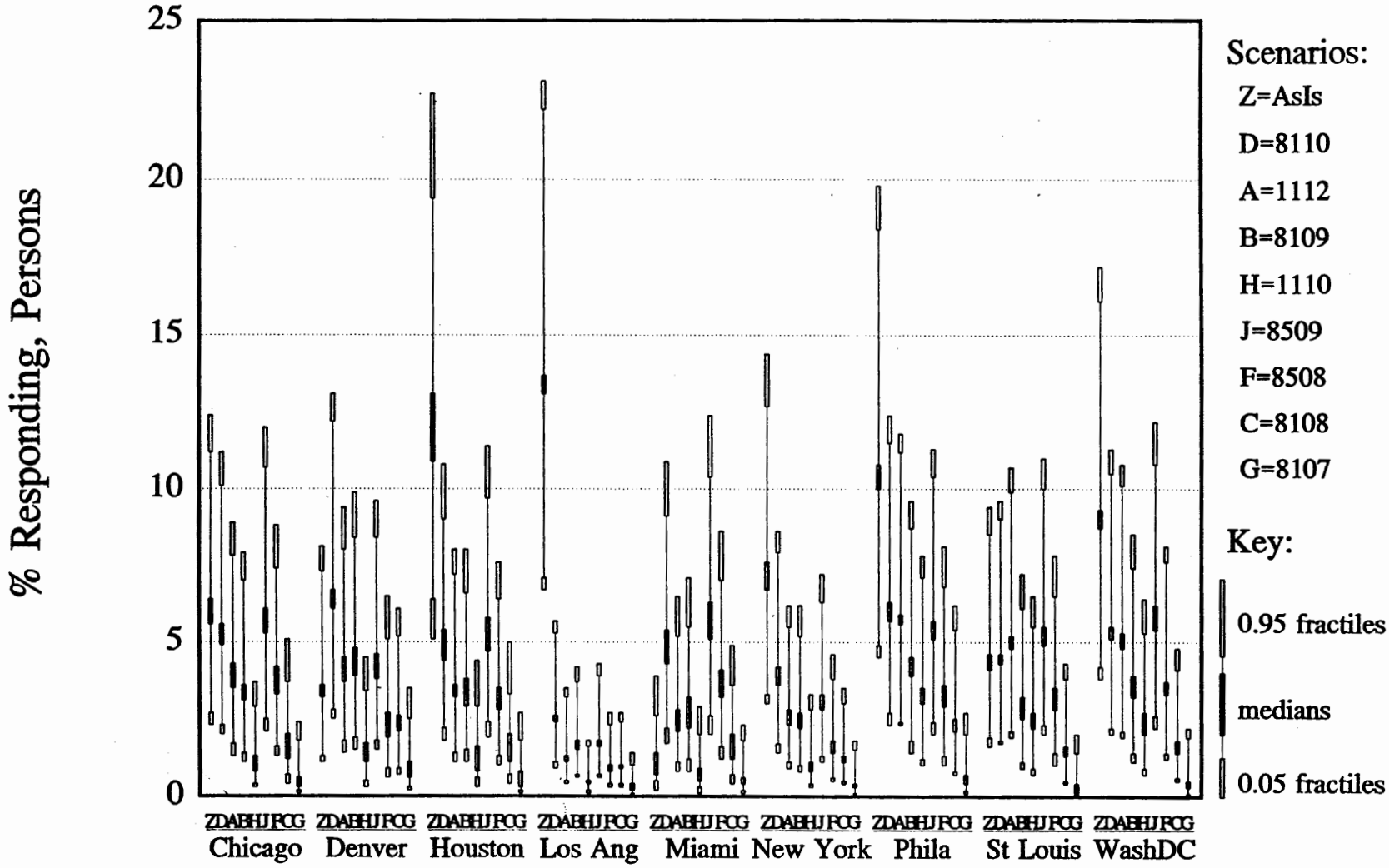


FIGURE C.22 Headcount Risk Results for the Percentage of Children Responding for the Eight-Hr, Moderate Exertion, FEV_1 Decrements ≥20% Endpoint

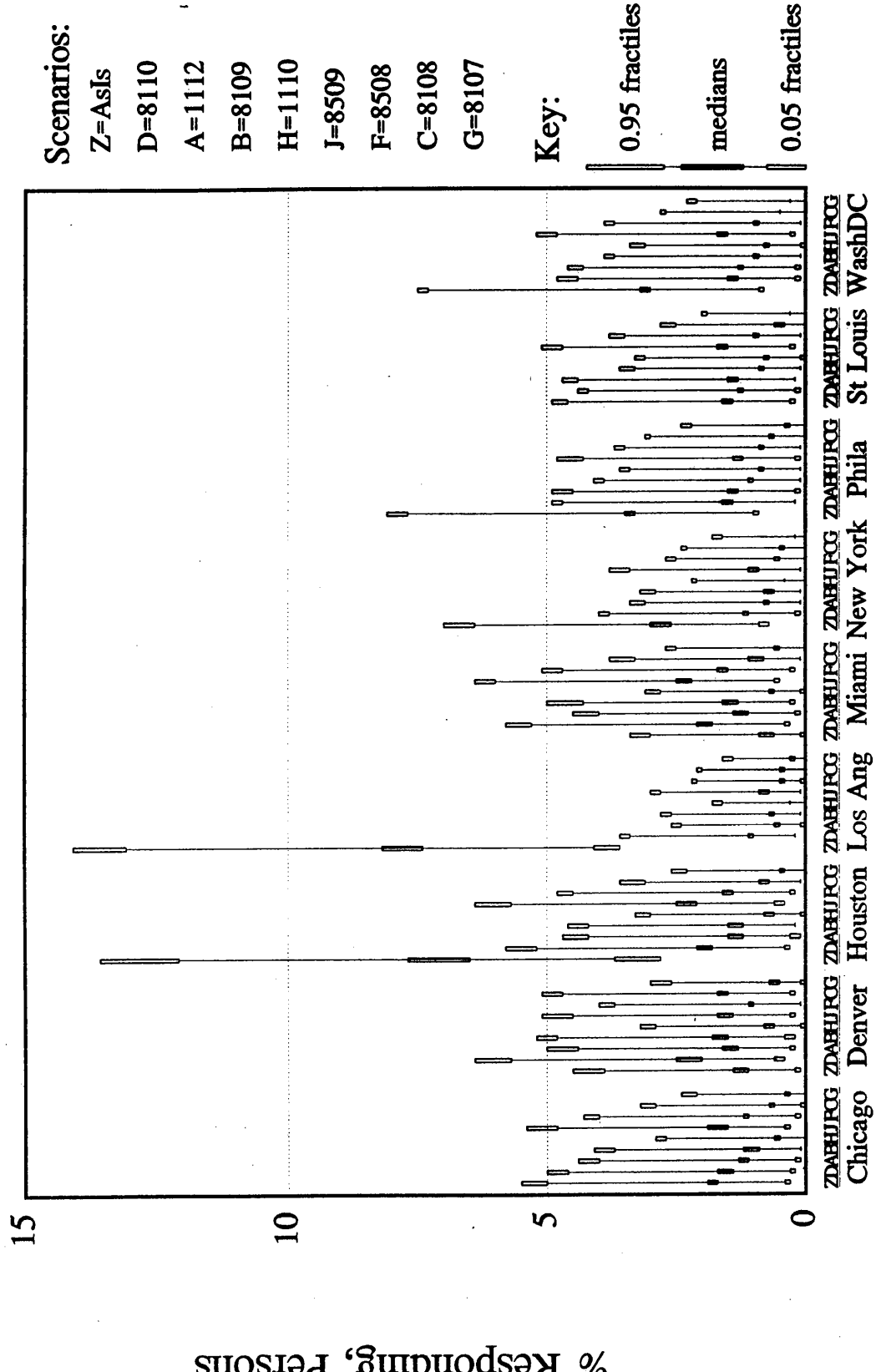


FIGURE C.23 Headcount Risk Results for the Percentage of Children Responding for the One-Hr, Moderate Exertion, Moderate-to-Severe Pain on Deep Inspiration Endpoint

C.2.3 Ratios of Mean Numbers of Occurrences and Mean Numbers of Responders

Figures C.24-26 show the ratios for the mean numbers of occurrences and mean numbers of outdoor children responding for three endpoints, nine urban areas, and five air quality scenarios. The endpoints are FEV_1 decrements ≥15% and ≥20% for 8 hr exposures at moderate exertion, and moderate-to-severe pain on deep inspiration for 1 hr exposures at moderate exertion. The following letter codes were used to identify the urban areas: CH = Chicago, DE = Denver, HO = Houston, LA = Los Angeles, MI = Miami, NY = New York City, PH = Philadelphia, SL = St. Louis, and DC = Washington, D.C.

The ratios were computed in the following way. For a specific endpoint, available risk results include 10 probability distributions (one for each of 10 pNEM runs) over the number of persons who respond one or more times, and 10 probability distributions over the number of person-occurrences (which allows for the possibility that an individual may respond more than one time). The ratio of interest here is the sum of the expected values of the person-occurrences distributions divided by the sum of the expected values of the persons distributions. The ratio is, in a sense, an estimate of the average number of times that a responder responds during an ozone season.

FIGURE C.24 RATIOS OF MEAN NUMBER OF OCCURRENCES AND MEAN NUMBER OF RESPONDERS (FEV_1 DECREMENTS ≥15%, 9 URBAN AREAS, OUTDOOR CHILDREN, 8 HR EXPOSURES, MODERATE EXERTION)

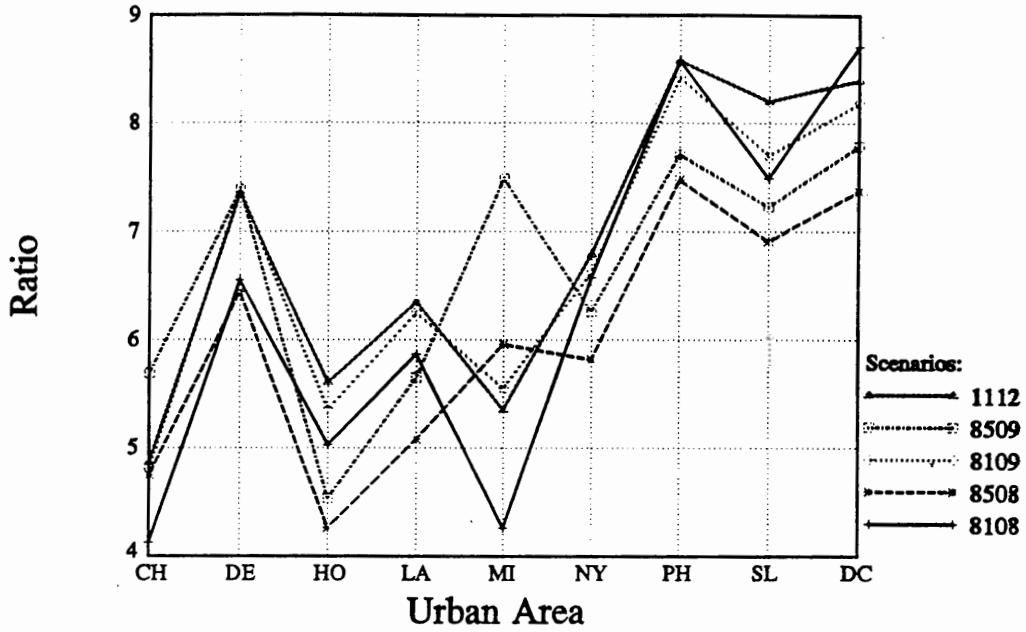


FIGURE C.25 RATIOS OF MEAN NUMBER OF OCCURRENCES AND MEAN NUMBER OF RESPONDERS (FEV_1 DECREMENTS ≥20%, 9 URBAN AREAS, OUTDOOR CHILDREN, 8 HR EXPOSURES, MODERATE EXERTION)

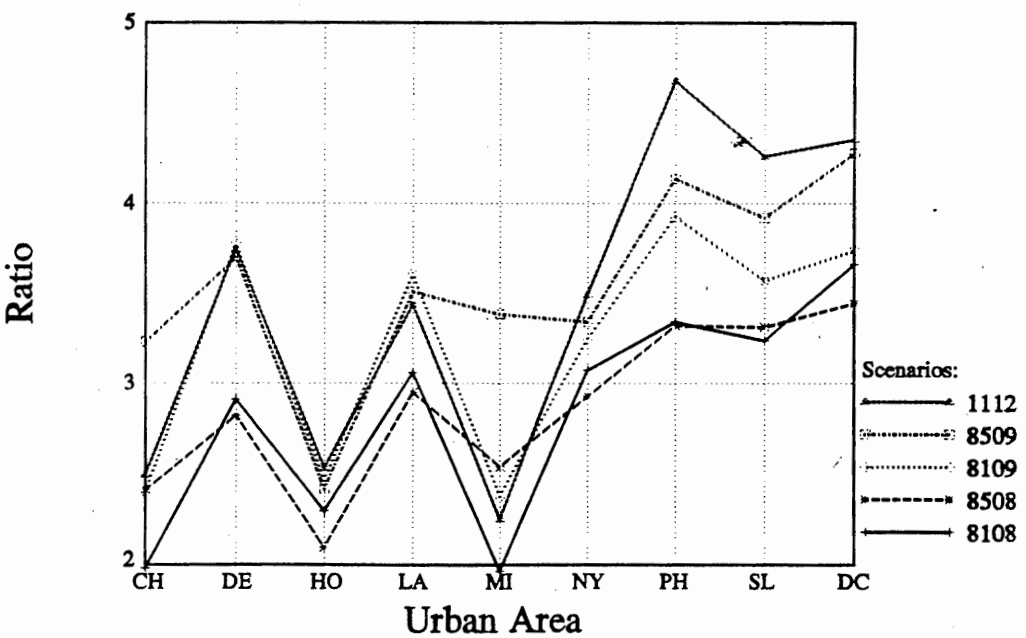
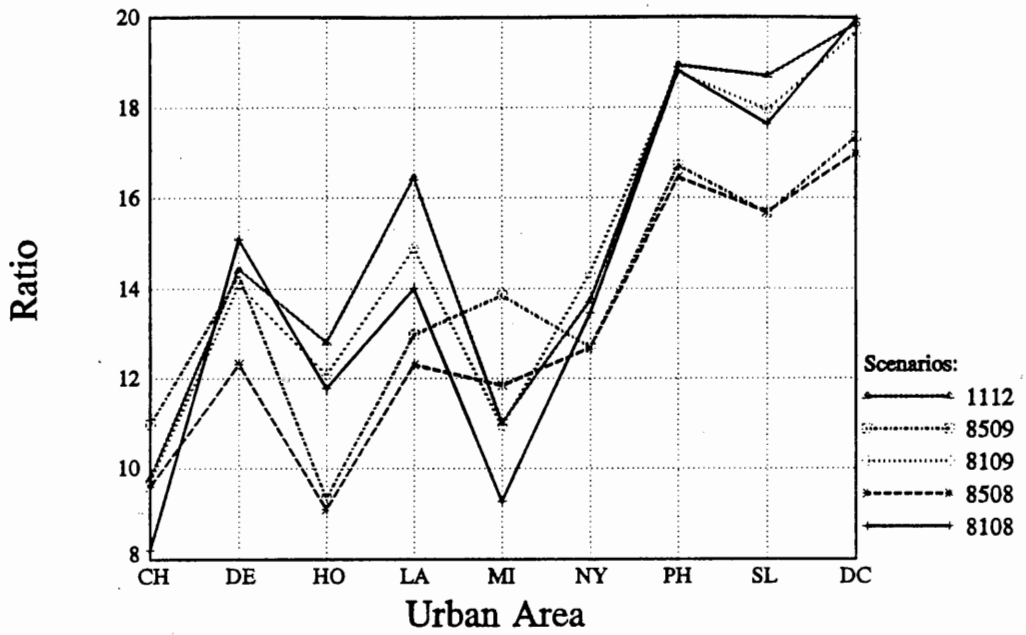


FIGURE C.26 RATIOS OF MEAN NUMBER OF OCCURRENCES AND MEAN NUMBER OF RESPONDERS (MODERATE-TO-SEVERE PAIN ON DEEP INSPIRATION, 9 URBAN AREAS, OUTDOOR CHILDREN, 1 HR EXPOSURES, MODERATE EXERTION)



C.3 BENCHMARK RISK

Figures C.27-29 illustrate benchmark risk results for the probability that the benchmark response will be exceeded 5 or more times in an ozone season. Benchmark response is r, the fraction of the population who experience the specified health effect upon exposure to ozone. Benchmark risk is defined as the probability that the benchmark response is ≥r n or more times in a given period of time (1 ozone season) at some location within a geographic region, given a specific condition of air quality (e.g., that standard 1112 is just attained). In this report, we use r values of 0.05 and 0.1 (sometimes referred to as 0.05 and 0.1 benchmarks, or 5% and 10% benchmarks).

Figure C.27 shows the graphical format used to display benchmark risk results. The figure is for the probability that the benchmark response for the FEV_1 decrement ≥15%, 8 hr exposure, moderate exertion endpoint will be exceeded 5 or more times in an ozone season for 0.05 and 0.1 benchmarks.

This figure includes results for 9 urban areas and 9 air quality scenarios for each urban area. The air quality scenarios are indicated by a letter code above the name of each area. The letter code is explained on the right side of the figure. There are 2 vertical lines for each air quality scenario: the one on the left (a solid line) is for the 0.05 benchmark, and the one on the right (a dotted line) is for the 0.1 benchmark. The height of the line indicates the benchmark risk. The benchmark risk for the 0.05 benchmark is, logically, ≤ the benchmark risk for the 0.1 benchmark.

For example, for Miami, scenario G, daily maximum 8-hr-running-average ozone concentrations, and using the distribution for the highest ozone concentration, the benchmark risk for the 0.05 benchmark is about 0.52; and the benchmark risk for the 0.1 benchmark is about 0.2. In other words, if standard 8107 is just attained in Miami, then the benchmark risk (i.e., probability) is 0.52 that ≥5% of the population will experience FEV_1 decrements ≥15% 5 or more times in an ozone season.

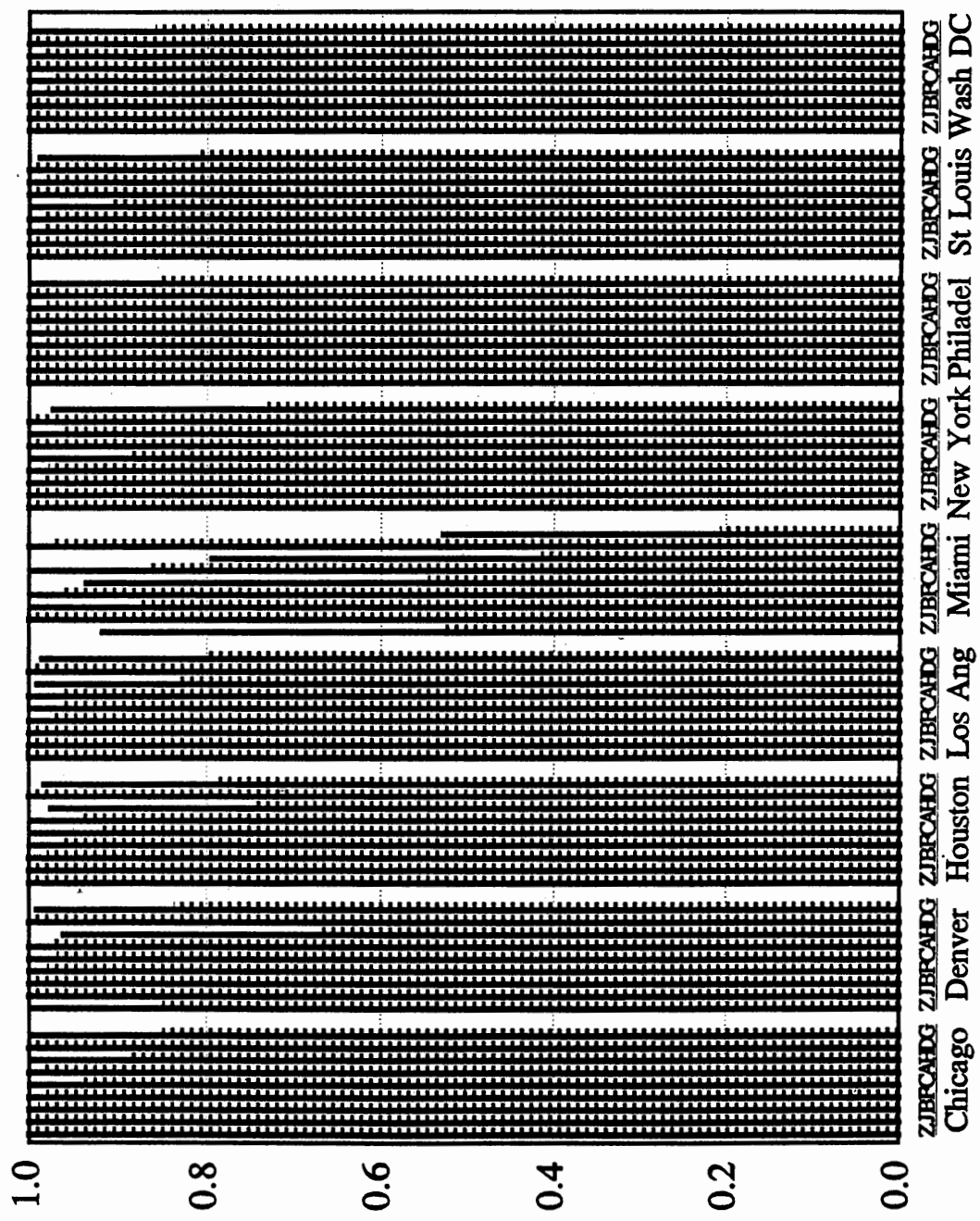


FIGURE C.27 Probability that the Benchmark Response for the Eight-Hr, Moderate Exertion, FEV_1 Decrement ≥ 15% Endpoint Will Be Exceeded 5 or More Times in an Ozone Season

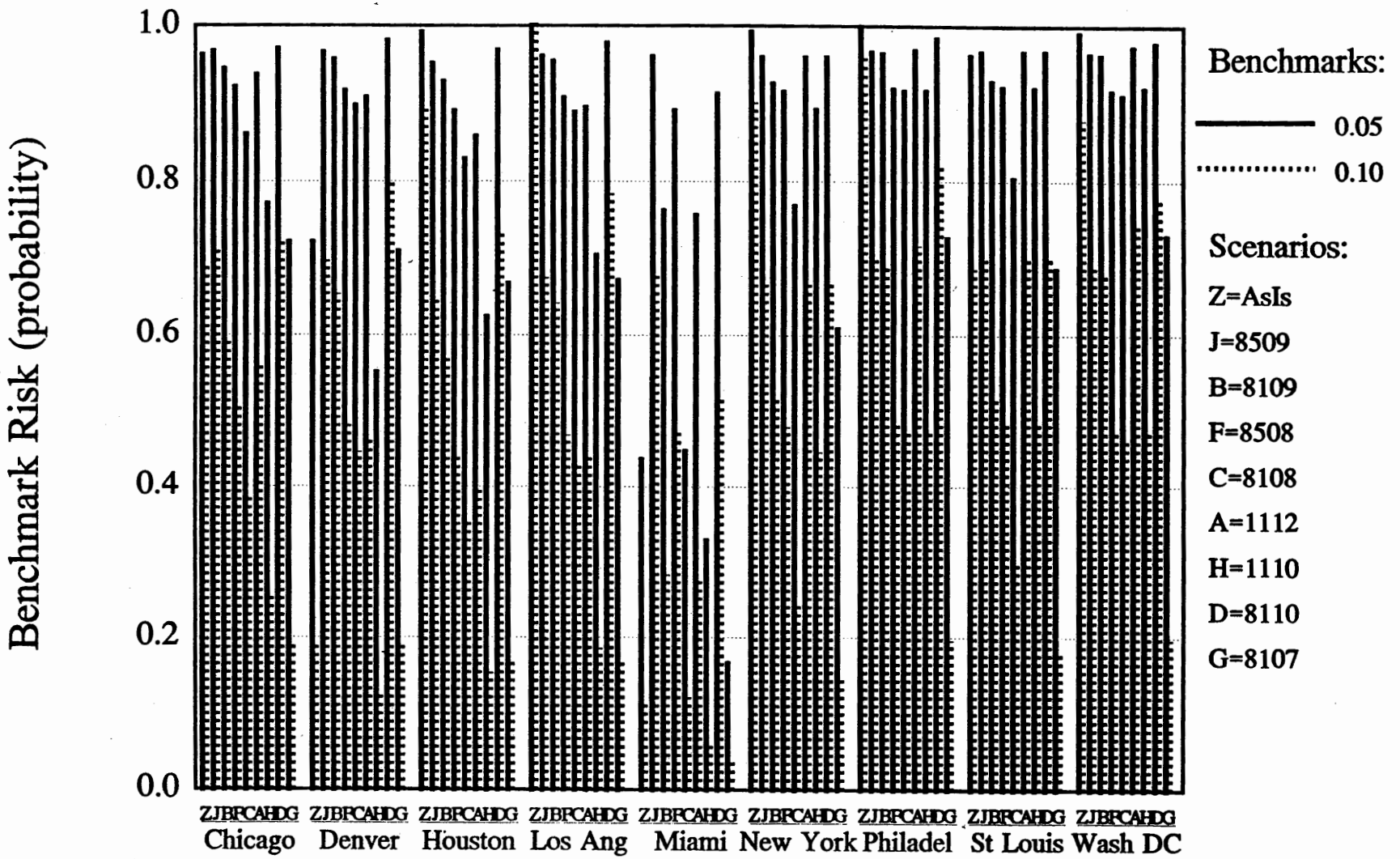


FIGURE C.28 Probability that the Benchmark Response for the Eight-Hr, Moderate Exertion, FEV_1 Decrement ≥ 20% Endpoint Will Be Exceeded 5 or More Times in an Ozone Season

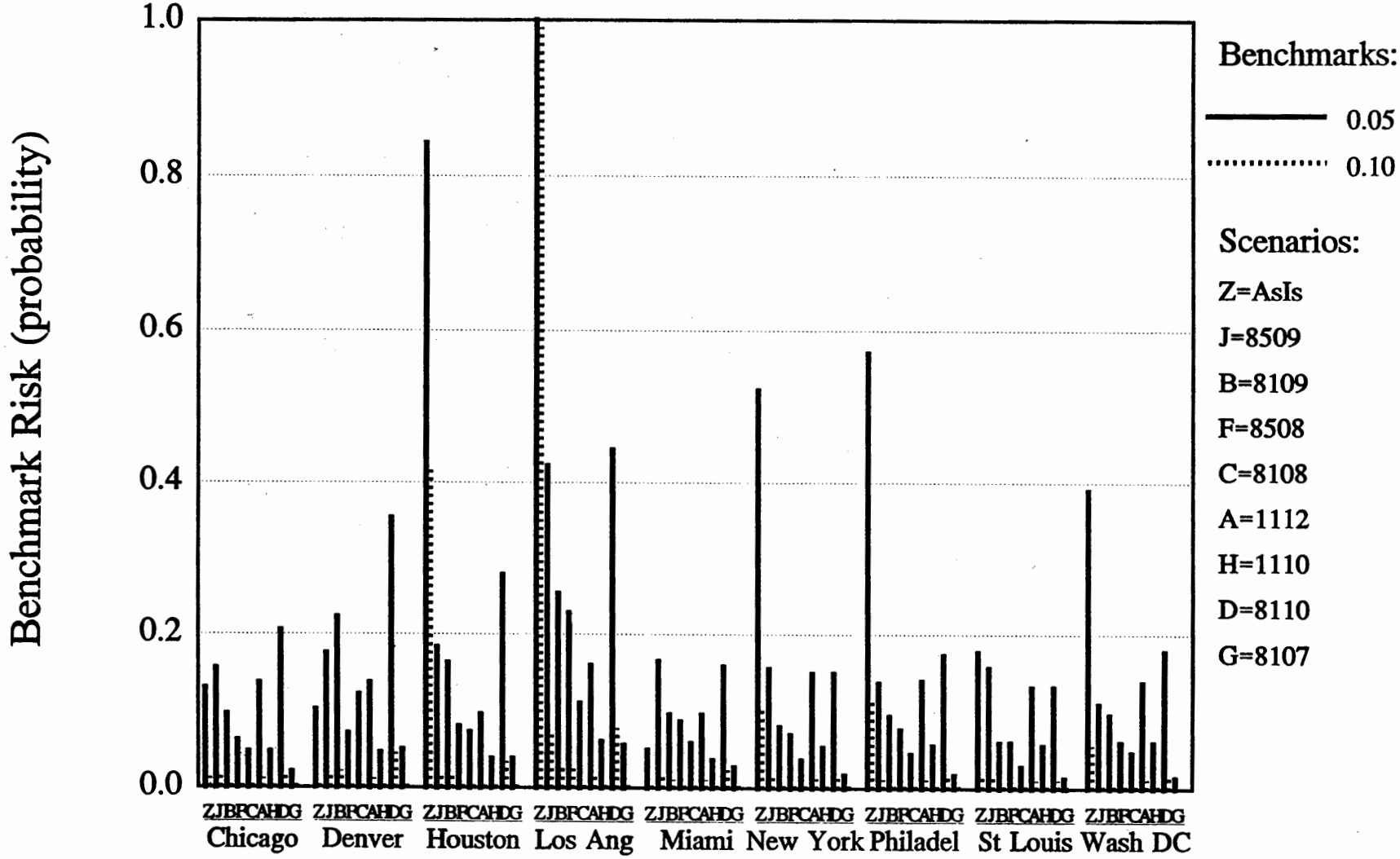


FIGURE C.29 Probability that the Benchmark Response for the One-Hr, Moderate Exertion, Moderate-to-Severe Pain on Deep Inspiration Endpoint Will Be Exceeded 5 or More Times in an Ozone Season

APPENDIX D

List of Species in Selected National Parks and Associated Ozone Response

Table D-1

Plant species exhibiting foliar injury indicative of exposure to ozone in GRSM

Species	Common Name	Lifeform	Code
Acer rubrum	Red Maple	Tree	1,2,5
Acer saccharum	Sugar Maple	Tree	1
Aconitum uncinatum	Wild Monkshood	Herb	
Aesculus octandra	Yellow Buckeye	Tree	1,2,3
Amelanchier laevis	Allegheny Serviceberry	Tree	
Anemone quinquefolia	Wood Anemone	Herb	
Anemone virginiana	Thimbleweed	Herb	
Apocynum cannibinum	Indian-hemp	Herb	
Aristolochia durior	Dutchman's Pipe	Vine	
Asclepias exaltata	Tall Milkweed	Herb	1,2,3
Asclepias incarnata	Whorled Milkweed	Herb	
Asclepias quadrifolia	Swamp Milkweed	Herb	
Asclepias syriaca	Common Milkweed	Herb	
Aster acuminatus	Whorled-wood Aster	Herb	1,3
Aster curtisii	Curtis's Aster	Herb	
Aster divaricatus	White-wood Aster	Herb	6
Aster infirmus	Entire-leaved Aster	Herb	
Aster puniceus	Purple-stemmed Aster	Herb	1,2
Betula lutea	Yellow Birch	Tree	1,2
Bidens frondosa	Beggar-ticks	Herb	
Cacalia atriplicifolia	Pale Indian-plantain	Herb	
Cacalia rugelia	Rugel's ragwort	Herb	1,2
Calycanthus florida	Sweet-shrub	Shrub	
Cephalanthus occidentalis	Buttonbush	Shrub	
Cercis canadensis	Eastern Redbud	Tree	1,2
Clematis virginiana	Virgin's-bower	Vine	
Clitoria mariana	Butterfly Pea	Herb	
Cornus florida	Flowering Dogwood	Tree	1,2
Crataegus spp.	Hawthorn	Tree	
Diervilla sessilifolia	Bush Honeysuckle	Shrub	6
Diospyros virginiana	Common Persimmon	Tree	
Eupatorium rugosum	White Snakeroot	Herb	1,2
Fraxinus americana	White Ash	Tree	
Fraxinus pennsylvanica	Green Ash	Tree	
Gaylussacia baccata	Black Huckleberry	Shrub	6
Gaylussacia ursina	Bear Huckleberry	Shrub	6
Geum radiatum	Mountain Avens	Herb	1
Gillenia trifoliata	Bowman's-root	Herb	
Glyceria nubigena	Smoky Mountain Manna Grass	Grass	1,2
Halesia carolina	Silverbell	Tree	
Hamamelis virginiana	Witch-Hazel	Shrub	
Helianthus glaucophyllus	White-leaf Sunflower	Herb	
Helianthus microcephalus	Small Wood Sunflower	Herb	
Hexastylis arifolia	Heartleaf	Herb	
Hieracium paniculatum	Panicled Hawkweed	Herb	
Impatiens capensis	Spotted Jewelweed	Herb	
Impatiens pallida	Yellow Jewelweed	Herb	
Lindera benzoin	Spicebush	Shrub	6
Liquidambar styraciflua	Sweetgum	Tree	1,2,3

Table D-1 (Continued)

Species	Common Name	Lifeform	Code
Liriodendron tulipifera	Yellow-Poplar	Tree	1,2,3,4,5,6
Lyonia ligustrina	Maleberry	Shrub	
Magnolia fraseri	Fraser Magnolia	Tree	
Menziesia pilosa	Minnie-bush	Herb	
Nyssa sylvatica	Blackgum	Tree	
Oxydendrum arboreum	Sourwood	Tree	6
Parthenocissus quinquefolia	Virginia Creeper	Vine	
Picea rubens	Red Spruce	Tree	
Pinus pungens	Table Mountain Pine	Tree	1,2,3
Pinus rigida	Pitch Pine	Tree	1,2,3
Pinus strobus	White Pine	Tree	5,6
Pinus virginiana	Virginia Pine	Tree	1,2,3,4,6
Platanus occidentalis	American Sycamore	Tree	1,2,3,6
Prenanthes altissima	Rattlesnake-root	Herb	
Prunus pennsylvanica	Pin Cherry	Tree	
Prunus serotina	Black Cherry	Tree	1,2,3,5,6
Pteridium aquilinum	Bracken Fern	Fern	
Quercus prinus	Chestnut Oak	Tree	1,6
Quercus rubra	Northern Red Oak	Tree	1,6
Rhamnus alnifolia	Alder-leaved Buckthorn	Shrub	
Rhododendron bakeri	Cumberland Azalea	Shrub	
Rhododendron calendulaceum	Flame Azalea	Shrub	
Rhododendron catawbiense	Catawba Rhododendron	Shrub	
Rhus copallina	Winged Sumac	Tree	1,2,3
Rhus radicans	Poison Ivy	Vine	
Robinia pseudoacacia	Black Locust	Tree	1,2,3
Rubus canadensis	Thornless Blackberry	Shrub	1,2,3
Rubus idaeus	Red Raspberry	Shrub	1,2,3
Rudbeckia hirta	Black-eyed Susan	Herb	1,2,3
Rudbeckia laciniata	Cutleaf Coneflower	Herb	1,2,3
Sambucus canadensis	American Elder	Shrub	
Sambucus pubens	Red-berried Elder	Shrub	
Sassafras albidum	Sassafras	Tree	1,2,3,5
Silphium asteriscus	Rosin-weed	Herb	
Smilax glauca	Glaucous Catbrier	Vine	
Smilax rotundifolia	Greenbrier	Vine	
Solidago roanensis	Roan's Goldenrod	Herb	
Stewartia ovata	Mountain Stewartia	Tree	
Stachys clingmanii	Clingman's Hedge-nettle	Herb	
Tilia heterophylla	White Basswood	Tree	
Trautvetteria caroliniensis	Tassel-rue	Herb	
Tsuga canadensis	Eastern Hemlock	Tree	1,3
Vaccinium spp.	Blueberry	Shrub	
Verbesina occidentalis	Crown-beard	Herb	1,2,3
Vitis aestivalis	Summer Grape	Vine	
Vitis vulpina	Frost Grape	Vine	

[1]Species that have been fumigated at Twin Creeks facility.
[2]Species showing foliar injury after fumigation.
[3]Fumigated species with histological samples taken.
[4]Gas exchange measured on these species at Twin Creeks facility.
[5]Species surveyed in variable trend plots at monitoring stations.
[6]Species targeted for fumigation in 1992.

Table D-2.
Vascular Plants of Acadia National Park
That Have Been Studied for O_3 Impacts

Genus Species	Common Name	Pollutant	Level	Duration	Effect	Reference
Abies balsamea	Balsam fir	O_3	25.0 pphm	8 hours	Resistant; no visible foliar injury	Davis, et al., 1972
Acer negundo	Boxelder	O_3	15.0; 25.0 30.0; 40.0 ppm	2 hours	Injury threshold over 25.0 ppm	Treshow, et al., 1973
Acer platanoides	Norway maple	O_3	0.5 ppm alone and with 1.0 ppm SO_2	single 7 1/2 hour exposure	Tolerant to all ambient and chamber exposures	Karnosky, 1981
Acer rubrum	Red maple	O_3	0.35 ppm	3 hours	Visible injury. Rated 1.8 on a 0 to 10 scale	Roberts, et al., 1985
			30.0 pphm ±	8 hrs/day for 5 months	Affected growth and leaf retention	Jensen, 1973
Acer saccharinum	Silver maple	O_3	25.0 to 35.0 pphm	8 hrs/day 5 days/wk for 5 months	Significant reductions in growth. Early season leaf loss.	Jensen, 1973
			0; 0.1; 0.2; 0.3 ppm	12 hrs/day up to 60 days	Relative growth rate, leaf area growth rate and leaf-weight all declined with increasing O_3	Jensen, 1982
		O_3 + SO_2	.05; 0.1; 0.2; ppm O_3 alone and with 0.1 ppm SO_2	12 hrs/day up to 60 days	Growth rate not affected at 0.05 and 0.1 ppm. Rates reduced by 0.2 ppm and SO_2 treatment	Jensen, 1983
Acer saccharum	Sugar maple	O_3 + Acid precip.	.03; .06; .09; .12 ul/l with pH 5.6; 4.0; 3.0 (1.25 hrs 2 times/week)	7 hrs/day 5 days/ wk for 6 to 10 wks	O_3 caused significant declines in net photosynthesis. No interaction with acid ppt	Reich, et. al., 1986
		O_3	0; 0.05; 0.1 0.15 ppm	6 hrs/day for 28 days	Significant height-growth stimulation at 0.1 ppm. Suppressed at 0.15 ppm.	Kress, et al., 1982
Achillea millefolium	Common yarrow	O_3	15.0; 25.0; 30.0; 40.0 pphm	Single 2-hr exposure	Threshold for injury was over 30.0 pphm	Treshow, et al., 1973
			5.0-7.0; 15.0; 30.0 pphm	3 hrs/day 5 days/wk through growing season	Visible injury; plus tops and root weight decreased at 15.0 pphm. Produced flowers but no seeds at all levels.	Harward, et al., 1971
Agrostis alba	Black bent	O_3	15.0 pphm	6 hrs/day x 10 days	Bleaching and necrosis of leaves. 40% leaf area injured	Hill, et al., 1974
Agrostis tenuis	Rough bent	O_3	0.5 ppm	3 hours	Moderate to severe injury	Younger, et al., 1980

Table D-2 (continued)
Vascular Plants of Acadia National Park
That Have Been Studied for O_3 Impacts

Genus Species	Common Name	Pollutant	Level	Duration	Effect	Reference
Arctostaphylos uva-ursi	Red bearberry (kinnickinick)	O_3	0.75 ul/l	4 hours	Stress ethylene production correlated with O_3 level and 3% foliar injury	Tingey, et al., 1976
Arrhenatherum elatius	Tall oat grass	O_3	70 nl/l	7 hrs/day for 2 wks	Significant increase in root:shoot ratio	Reiling, et al., 1992
Artemisia vulgaris	Common wormwood	O_3	300 ppb	4 hrs/wk for 2 yrs	1st year inhibitions of net photosynthesis; earlier senescence	Cornelius, 1982
Asclepias syriaca	Common milkweed	O_3	0.05 ppm	6 hrs/day for 7 days	Chlorosis and purple stippling on 62% of plants with average of 10% of leaf area	Duchelle, et al., 1981
Avena sativa	Oat	O_3	140 ug/m^3	2 hours	Depression of photosynthesis at or above this level maximum susceptibility 20-30 days after panicle emergence	Forberg, et al., 1987
Betula allegheniensis	Yellow birch	O_3	0; 0.075; 0.15 ul/l	6 hrs/day, 2 days/ wk for 12 wks	Dark, adaxial stipple most severe on oldest leaves. Light to moderately sensitive species	Davis, et al., 1992
			25.0 pphm mean (20 to 30 pphm range)	8 hrs/day over 110 days	Reduction in growth not significant	Jensen, et al., 1975
Betula papyrifera	Paper birch	O_3	20.0 to 30.0 pphm (25.0 pphm mean)	8 hrs/day over 110 days	Reduction in height growth, reduced average leaf length and width	Jensen, et al., 1975
Brassica rapa	Turnip	O_3	Up to 0.1 ul/l	12 hr seasonal mean	Weight reduction began at 0.055 ul/l	McCool, et al., 1987
Bromus tectorum	Cheat grass	O_3	15.0; 25.0; 30.0; 40.0 pphm	2 hours	Injury threshold 15.0 pphm	Treshow, et al., 1973
Chenopodium album	Lamb's quarters	O_3	5.0-7.0 15.0; 30.0 pphm	3 hrs/day, 5 days/wk through growing season	Moderately resistant	Harward, et al., 1971
			70.0 nl/l	7 hrs/day for 2 wks	Significantly reduced root:shoot ratio. Necrotic mottle.	Reiling, et al., 1992
Chichorium intybus	Chicory	O_3	15.0; 25.0; 30.0; 40.0 pphm	Single 2-hr exposure	Threshold for injury at 25.0 pphm	Treshow, et al., 1973
Cornus stolonifera	Redosier dogwood	O_3	0.25 ppm	8 hours	Slight to moderate injury. 33% of plants susceptible	Davis, et al., 1976

Table D-2 (continued)
Vascular Plants of Acadia National Park
That Have Been Studied for O_3 Impacts

Genus Species	Common Name	Pollutant	Level	Duration	Effect	Reference
Daucus carota	Queen Anne's-lace (wild carrot)	O_3	0.19; 0.25 ppm	Intermittent. 6-hr periods over growing season	Chlorotic spots; root dry matter decreased by 32-46%	Bennett, et al., 1976
Deschampsia flexuosa	Wavy hair grass	O_3	70.0 nl/l	7 hrs/day for 2 wks	No significant effects on growth	Reiling, et al., 1992
Epilobium angustifolium	Fireweed	O_3	15.0; 25.0; 30.0; 40.0 pphm	Single 2-hr exposure	Threshold for injury at 30.0 pphm	Treshow, et al., 1973
Festuca arundinacea	Tall fescue	O_3	0.10 ppm	6 hrs/day, 1 day/wk x 12 wks	Reduction in dry weight. Reduced nutritional value	Flagler, et al., 1982
			0.10 ppm	0 to 6 hrs/day for 3 months	Reduced root weights. Reduced mycorrhizn formation	Ho, et al., 1984
Festuca ovina	Sheep fescue	O_3	70.0 nl/l	7 hrs/day for 2 wks	No significant growth effect	Reiling, et al., 1992
Festuca rubra	Red fescue	O_3	0.5 ppm	3 hours	Moderate foliar injury	Younger, et al., 1980
Fragaria virginiana	Virginia strawberry	O_3	294 ug/m³ (0.15 ppm)	2 hours	Necrotic flecks on upper leaf surface	Lawrence, et al., 1978
Fraxinus americana	White ash	O_3	0.10 ppm	4 hrs/day x 5 days/wk x 6 wks	Visible symptoms on 22% of plants	Chappelka, et al., 1988
			0.10 ppm	6 hrs/day for 28 days	Reduced dry weight	Kress, et al., 1982
			30.0 pphm	8 hrs/day x 5 days/wk x 5 months	Significant effect on early leaf drop	Jensen, et al., 1973
Fraxinus pennsylvanica	Green ash	O_3	0.10 ppm	6 hrs/day x 28 days	Reduced height growth	Kress, et al., 1982
			0.10 ppm	4 hrs/day x 5 days/wk x 6 wks	Visible symptoms on 14% of plants	Chappelka, et al., 1988
Helianthus annus	Common sunflower	O_3	15.0; 25.0; 30.0; 40.0 pphm	2 hours	Injury threshold over 30.0 pphm	Treshow, et al., 1973
Hordeum vulgare	Common barley	O_3	0.15 mm³dm⁻³ (150 ppb)	7 hrs/day 1 to 21 days	No visible injury but there was increased arginine decarboxylase activity	Rowland-Baumford, et al., 1989
			0.05; 0.10; 0.15 ul/l	7 hrs/day x 12 days	Carbon dioxide exchange rate reduced	Rowland-Baumford, et al., 1989

Table D-2 (continued)
Vascular Plants of Acadia National Park
That Have Been Studied for O_3 Impacts

Genus Species	Common Name	Pollutant	Level	Duration	Effect	Reference
Kalmia latifolia	Mountain laurel	O_3	0.25 ppm	8 hours	Resistant at this level and duration	Davis, et al., 1974
Lepidium virginicum	Poorman's pepperwort	O_3	5.0 - 7.0; 15.0; 30.0 pphm	3 hrs/day x 5 days/ wk through growing season	Significant reduction in total plant weight even at 5.0-7.0 pphm	Harward, et al., 1971
Lolium multiflorum	Perennial rye grass	O_3	0.09 ppm	8 hrs/day x 6 wks	Reduced dry weight, leaf area, root dry weight, root:shoot ratio	Bennett, et al., 1977
			0.5 ppm	3 hours	Severe foliar injury	Younger, et al., 1980
Lolium perenne	Perennial rye grass	O_3	0.5 ppm	3 hours	Moderate to severe injury	Younger, et al., 1980
			70.0 ul/l	7 hrs/day x 2 wks	Significant root:shoot ratio increase	Reiling, et al., 1992
Medicago sativa	Alfalfa	O_3	0.05 ppm	8 hrs/day x 5 days/ wk x 12 wks	Reduced dry weight and root weight	Reinert, 1984
Mentha arvensis	American wild mint	O_3	Pretreatments: 0.02 ul/l (ppm) or filtered air. Acute dose: 0.3 ul/l	6 hrs/day x 3 days	Filtered air pretreatment had 32 ± 1.8% leaf area necrosis. 0.02 ul/l pretreatment had 39 ± 2.6%	Runeckles, et al., 1974
Mentha piperita	Mint	O_3	0.08 to 0.10 ppm	3 hours	Insignificant reduction in dry weight	Runeckles, et al., 1974
		O_3 with NO_2 Pre-treatment	0.08 to 0.10 ppm	6 hours	Insignificant reduction in dry weight	Runeckles, et al., 1974
Parthenocissus quinquefolia	Virginia creeper	O_3	0.25 ppm	8 hours	64% of exposed plants were susceptible with a large % of leaf injury	Davis, et al., 1976
Physocarpus opulifolius	Atlantic ninebark	O_3	0.25 ppm	8 hours	57% of plants susceptible; moderate injury	Davis, et al., 1976
Picea glauca	White spruce	O_3	25.0 pphm	8 hours	Resistant; no visible foliar injury	Davis, et al., 1972
			0.25 ul/l	5 days/wk for 74 days	7% reduction in height growth	Pye, 1988
Picea rubens	Red spruce	O_3 + Acid Dep.	0.5; 1.0; 1.5; 2.0 x Ambient levels + pH 3.1; 4.1; 5.1	3 months during summer	No significant effect	Lawrence, et al., 1989

Table D-2 (continued)
Vascular Plants of Acadia National Park
That Have Been Studied for O_3 Impacts

Genus Species	Common Name	Pollutant	Level	Duration	Effect	Reference
Pinus banksiana	Jack pine	O_3	25.0 pphm	8 hours	35% of plants susceptible	Davis, et al., 1972
		O_3/SO_2	Ambient levels - Indiana Dunes. No levels reported	Episodes	Tip necrosis and chlorotic mottle present in all populations; injury was low	Armentano, et al., 1987
Pinus rigida	Pitch pine	O_3	0; 0.08; 0.1; 0.15; 0.2; 0.3 ul/l	2 x 4 hours	Root starch content decreased with increased O_3	Scherzer, et al., 1989
			0.10 ppm	6 hrs/day for 28 consecutive days	Adverse growth effects; reduced height growth and dry weight	Kress, et al., 1982
			25.0 pphm	8 hours	6% of plants susceptible; chlorotic mottle	Davis, et al., 1972
Pinus sylvestris	Scotch pine	O_3	250 ug/m³	Continuously over one month	Transpiration and stomatal conductance increased at night	Skarby, 1987
Plantago lanceolata	English plantain	O_3	70.0 nl/l	7 hrs/day x 2 wks	Significant reduction in mean relative growth rate and root:shoot ratio	Reiling, 1992
Plantago major	Great plantain	O_3	70.0 nl/l	7 hrs/day x 2 wks	Significant reduction in mean relative growth rate and root:shoot ratio	Reiling, 1992
Poa annua	Annual bluegrass	O_3	0.2 ppm	2 hours	Low tolerance; visible damage	Richards, et al., 1980
			70.0 nl/l	7 hrs/day x 2 wks	Significant reduction in mean relative growth rate and root:shoot ratio	Reiling, 1992
Poa pratensis	Kentucky bluegrass	O_3	47 ppb SO_2 + 50 ppb O_3 96 ppb SO_2 + 98 ppb O_3	8 hrs/day x 5 days/ wk for 4.5 wks	0.73 ± .46% leaf injury 7.33 ± 4.04% leaf injury	Martin, et al., 1988
Polygonum aviculare	Yard knotweed	O_3	5.0-7.0; 15.0; 30.0 pphm	3 hrs/day x 5 days/ wk through growing season	Reduced top and root weight and seed production at 30.0 pphm	Harward, et al., 1971
Populus grandidentata	Big-tooth aspen	O_3	0.25 ul/l	5 days/wk for 74 days	No significant effect on height growth	Jensen, et al., 1975
Populus tremuloides	Quaking aspen	O_3	180 ppb	6 hrs/once each spring for 2 springs	Natural selection for ozone tolerance may have occurred in some subspecies	Berrang, et al., 1986

Table D-2 (continued)
Vascular Plants of Acadia National Park
That Have Been Studied for O_3 Impacts

Genus Species	Common Name	Pollutant	Level	Duration	Effect	Reference
Prunus pennsylvanica	Fire cherry	O_3	0.10; 0.20; 0.30; 0.40	1 day/wk over 9 wks	Biomass changes: +11; +1; +6; -2%	Pye, 1988
Prunus serotina	Black cherry	O_3	0.20 ppm for 5 hours	Periodically over growing season	Susceptible	Davis, et al., 1981
Quercus rubra	Northern red oak	O_3 + Acid Precip.	0.02; 0.07; 0.12 ul/l pH 5.0; 4.0; 3.0 (1.25 hrs 2 times wk)	7 hrs/day x 3 days/ wk for 4, 7, 9 wks	Significant reduction in net photosynthesis after 7 wks. No interaction with acid ppt.	Reich, et al., 1986
		O_3	0; 0.075; 0.15 ul/l	6 hrs/day x 2 days/ wk for 12 wks	No foliar stipple exhibited	Davis, et al., 1992
Rhus typhina	Stag-horn sumac	O_3	0.25 ppm	8 hours	Very susceptible to foliar injury	Davis, et al., 1976
Rumex acetoselle	Common sheep sorrel	O_3	70.0 ul/l	7 hrs/day x 2 wks	Significant growth rate reduction; slight chlorosis	Reiling, et al., 1992
Rumex crispus	Dock	O_3	25.0 pphm	2 hours	Sensitive	Treshow, et al., 1973
Rumex obtusifolius	Bitter dock	O_3	70.0 ul/l	7 hrs/day x 2 wks	Significant reduction in growth rate	Reiling, et al., 1992
Sambucus canadensis	American elder	O_3	0.25 ppm	8 hours	51% of plants susceptible to injury with moderate to high injury	Davis, et al., 1976
Silene cucubalus	Maiden's tears	O_3/ SO_2	O_3 60.0 ug/m^3 SO_2 50; 100 ug/m^3 6 wks then 4 wks	12 hrs/day 24 hrs/day	No ripened seed production	Dueck, et al., 1986
Symphosicarpus albus	Common snowberry	O_3	0.25 ppm	8 hours	11% of exposed plants were susceptible with very light foliar injury	Davis, et al., 1976
Syringa volgaris	Common lilac	O_3	0.35; 0.55; 0.75; 0.95 ppm	3 hours	Injury rating by level: 3.0; 3.5; 5.5; 7.5 on a 0 to 10 scale of injury	Cathey, 1982
Thuja occidentalis	Eastern arborvitae	O_3	None reported		Considered resistant due to slow growth rate	Harkov, et al., 1982
Toxicodendron radicans	Poison ivy	O_3	15.0; 25.0; 30.0; 40.0 pphm	2 hours	Injury threshold over 30.0 pphm	Treshow, et al., 1973
Trifolium pratense	Red clover	O_3	0.5 ug/m^3 and greater	4 weeks	Injury began at<0.5 ug/m^3	Luthy-Krams, et al., 1989
			Ambient air, 1.5x and 2.0x ambient	12 hrs/day for 2 growing seasons	Ozone reduced growth yield	Kohut, et al., 1988

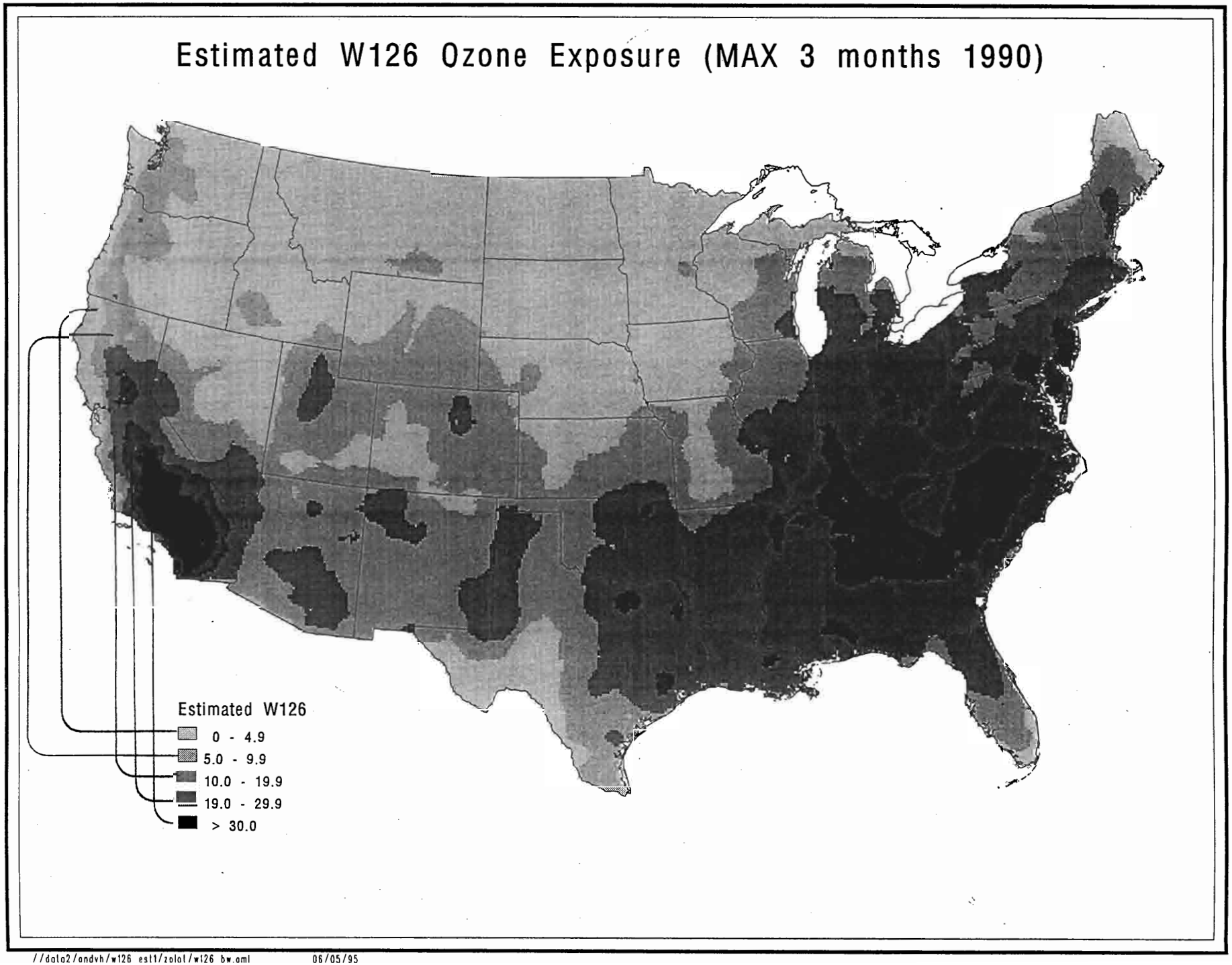
Table D-2 (continued)
Vascular Plants of Acadia National Park
That Have Been Studied for O_3 Impacts

Genus Species	Common Name	Pollutant	Level	Duration	Effect	Reference
Trifolium repens	White clover	O_3	120 to 140 ug/m³	1 hr max for several consecutive days	Necrotic flecks	Becker, et al., 1989
			0.046 ul/l	12 hrs/day for 2 yrs	Growth reduction; 19% yield reduction	Heagle, et al., 1989
			0; 0.05; 0.10; 0.15 ppm	4 hrs/day for 6 days	Reduced plant weight with increased concen.	Blum, et al., 1983
Tsuga canadensis	Eastern hemlock	O_3	0.05 ul/l 0.07 ul/l	24 hr/day - 297 days 24 hr/day - 500 days	23% reduction in height growth 40% reduction in height growth	Pye, 1988
			Not reported		Considered resistant due to intermediate growth rate	Harkov, et al., 1982
Ulmus americana	American elm	O_3	0.9 ppm	5 hours for 9 days	Visible injury within 36-48 hours	Constantinidou, et al., 1979
Urtica dioica	Stinging nettle	O_3	70.0 nl/l	7 hrs/day for 2 wks	Significant reduction in mean growth rate and root:shoot ratio	Relling, et al., 1992
Urtica gracilis	Stinging nettle	O_3	15.0; 25.0; 30.0; 40.0 pphm	2 hours	Injury threshold 30.0 pphm	Treshow, et al., 1973
Viola adunca	Hook-spur violet	O_3	15.0; 25.0; 30.0; 40.0 pphm	2 hours	Injury threshold over 30.0 pphm	Treshow, et al., 1973

Appendix E

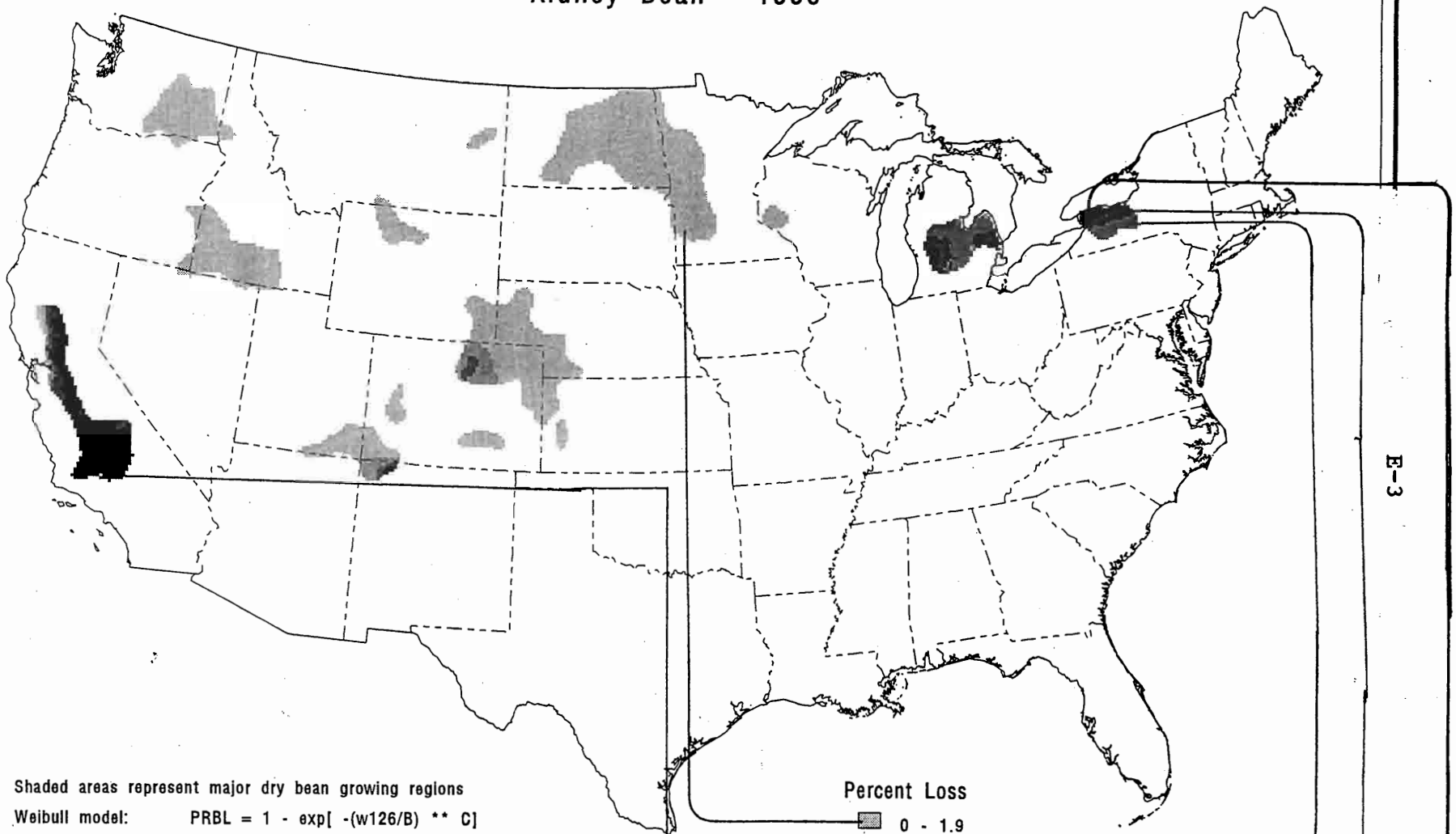
Predicted Yield and Biomass Loss for Crops and Tree Seedlings Based on Growth Regions and GIS Predictions of 1990 National Air Quality Using the W126 Index

*These maps have been generated using data which contain unquantifiable levels of uncertainties with respect to extrapolating exposure-response functions generated in open-top chambers to the field (section D.2 and D.3) and use of limited meteorological and O_3 precursor emissions data to develop national projections using the GIS (section F.1). The impact and the interaction of these uncertainties on these national projections is not known (section F.2).


Estimated W126 Ozone Exposure (MAX 3 months 1990)
Estimated W126
0 - 4.9
5.0 - 9.9
10.0 - 19.9
19.0 - 29.9
> 30.0
//data2/andyh/w126 est1/zolot/w126 bw.aml 06/05/95

Estimated Yield Loss

Kidney Bean - 1990



Shaded areas represent major dry bean growing regions

Weibull model: PRBL = 1 - exp[-(w126/B) ** C]

Weibull parameter: B = 43.1

Weibull parameter: C = 2.219

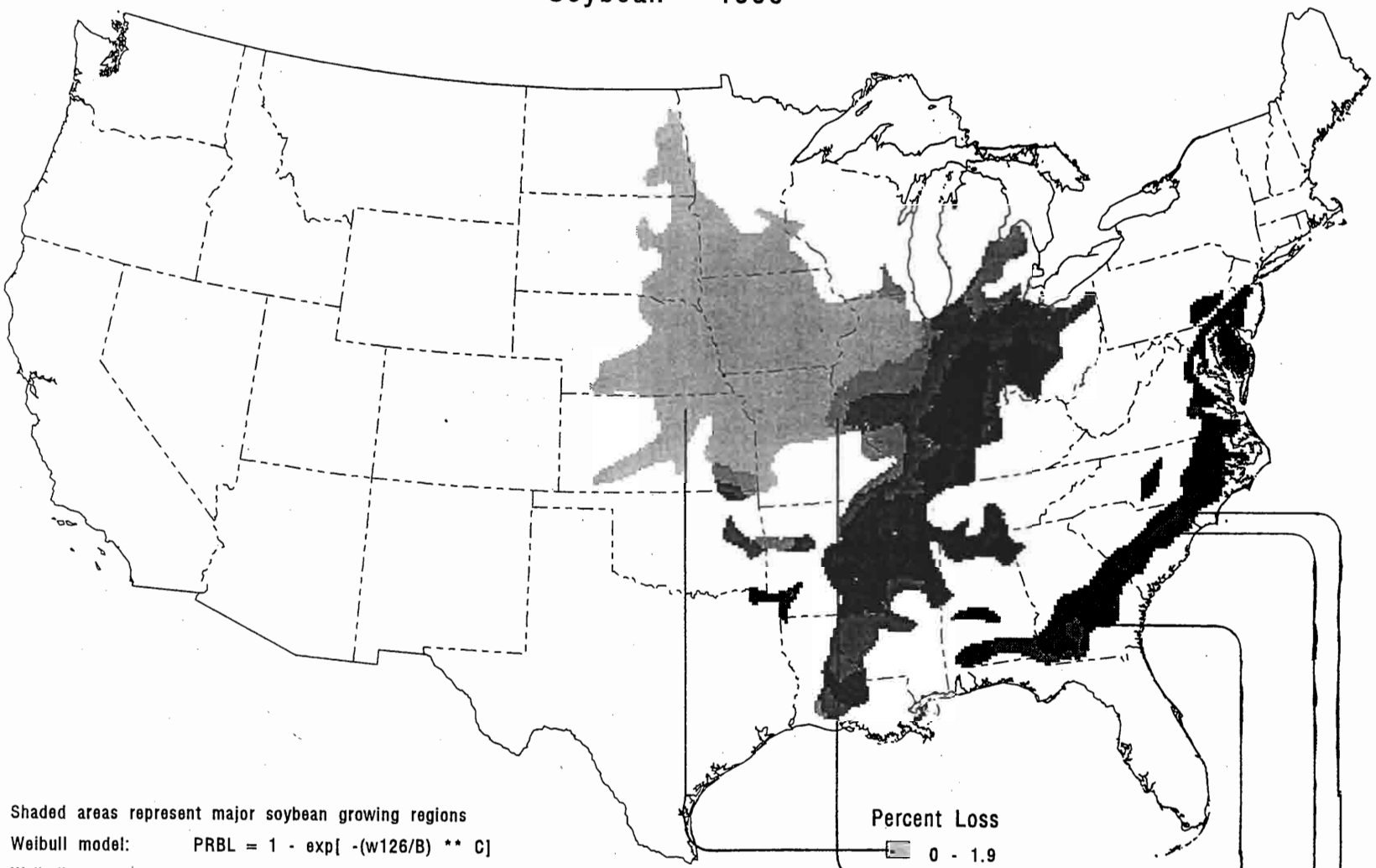
Based on estimates of 1990 maximum 3 month 12 hour W126 ozone exposure

Note: NCLAN exposure-response functions used in projections of yield loss. This map shows the geographic range for this species. It does not indicate that this species will be found at every point within its range.

//data2/andvh/bio loss3/zalat/ mao.aml 06/05/95

Estimated Yield Loss

Soybean - 1990



Shaded areas represent major soybean growing regions

Weibull model: PRBL = 1 - exp[-(w126/B) ** C]

Weibull parameter: B = 110.2

Weibull parameter: C = 1.359

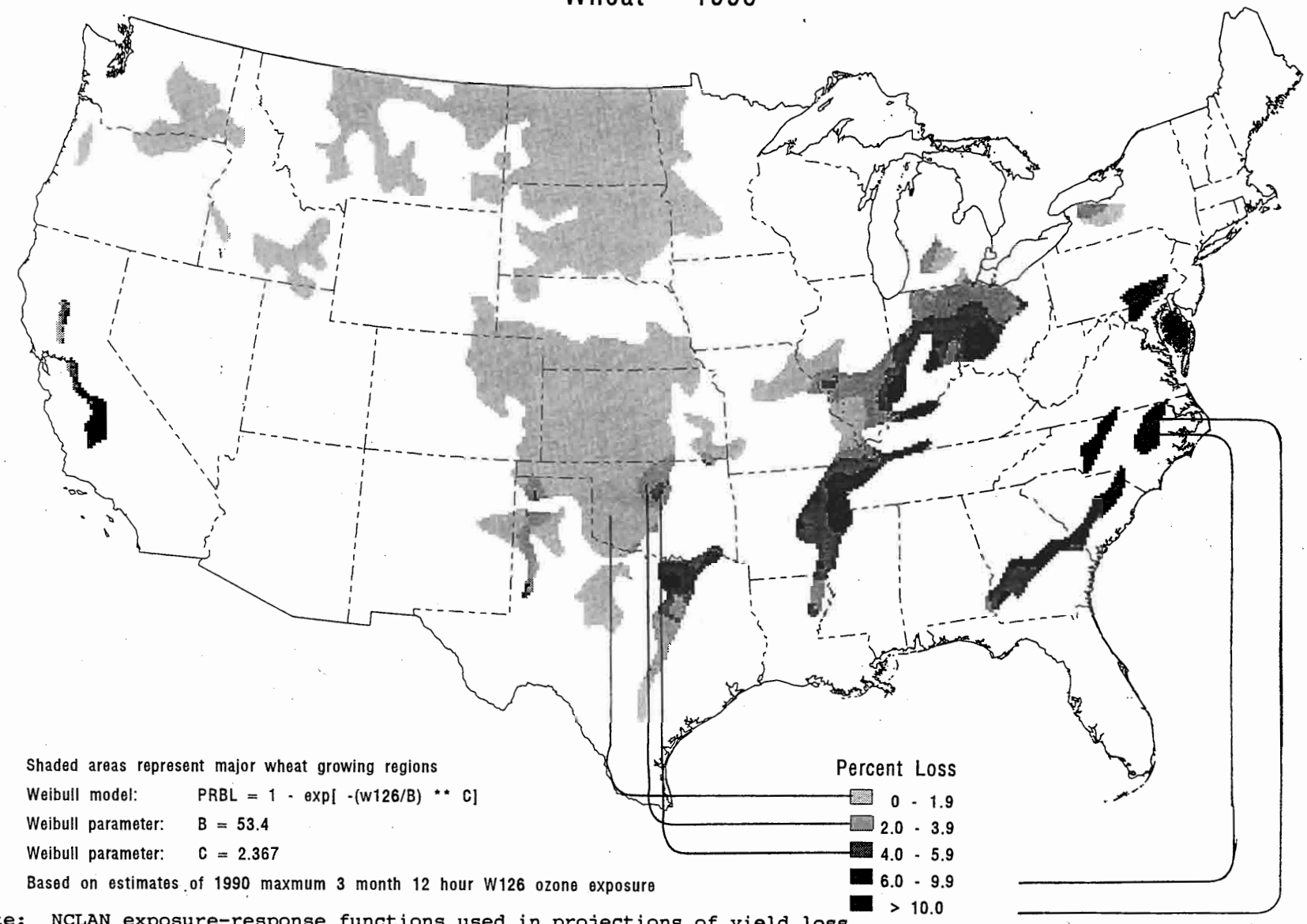
Based on estimates of 1990 maximum 3 month 12 hour W126 ozone exposure

Note: NCLAN exposure-response functions used in projections of yield loss. This map shows the geographic range for this species. It does not indicate that this species will be found at every point within its range.

//data2/ondvh/bio loss3/zolal/ map.aml 06/05/95

Estimated Yield Loss

Wheat - 1990



Shaded areas represent major wheat growing regions

Weibull model: PRBL = 1 - exp[-(w126/B) ** C]

Weibull parameter: B = 53.4

Weibull parameter: C = 2.367

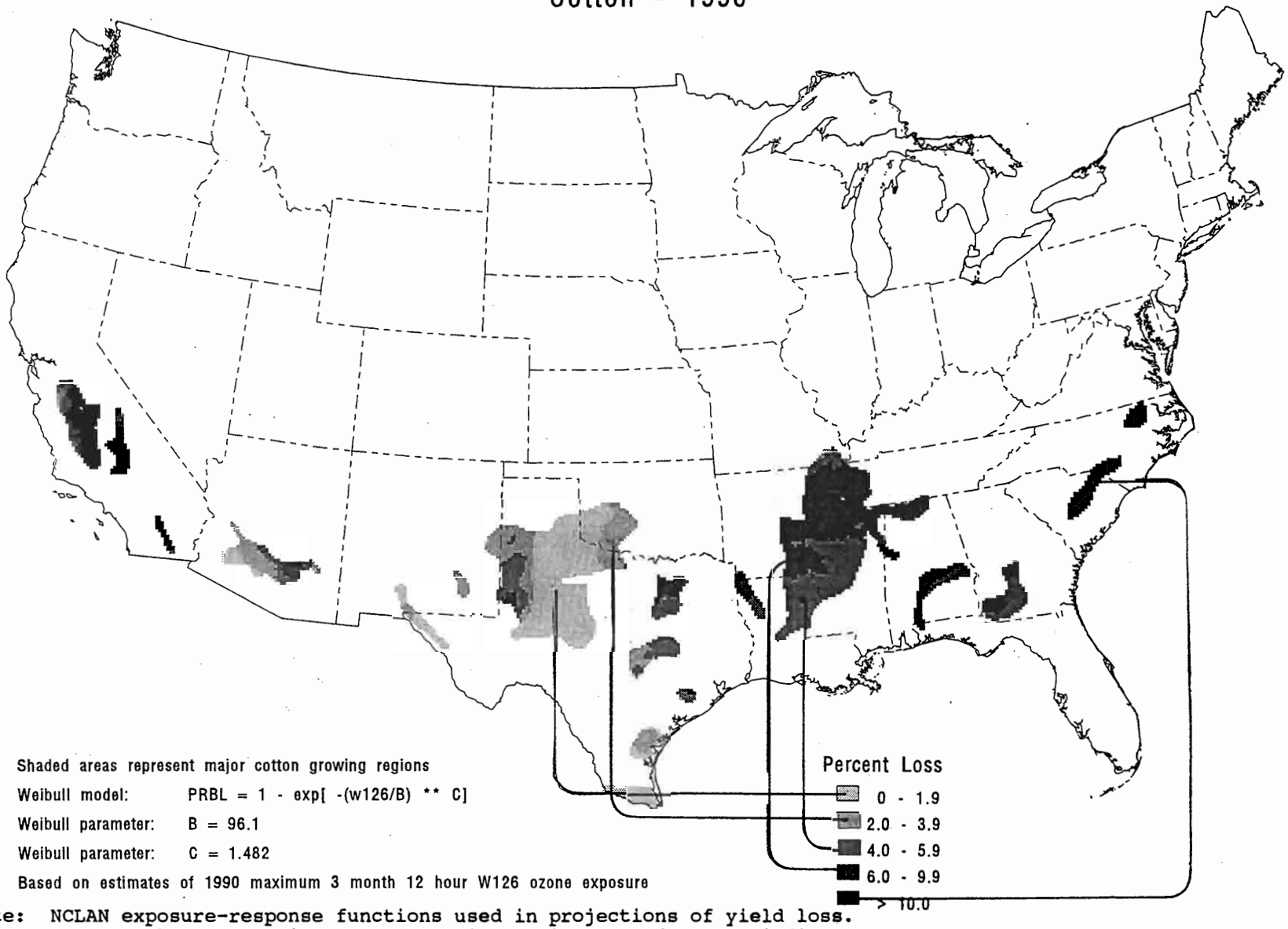
Based on estimates of 1990 maxmum 3 month 12 hour W126 ozone exposure

Note: NCLAN exposure-response functions used in projections of yield loss. This map shows the geographic range for this species. It does not indicate that this species will be found at every point within its range.

//dolp2/cndvb/bio_loss3/zplot/ map.eml 06/05/95

Estimated Yield Loss

Cotton - 1990



Shaded areas represent major cotton growing regions

Weibull model: PRBL = 1 - exp[-(w126/B) ** C]

Weibull parameter: B = 96.1

Weibull parameter: C = 1.482

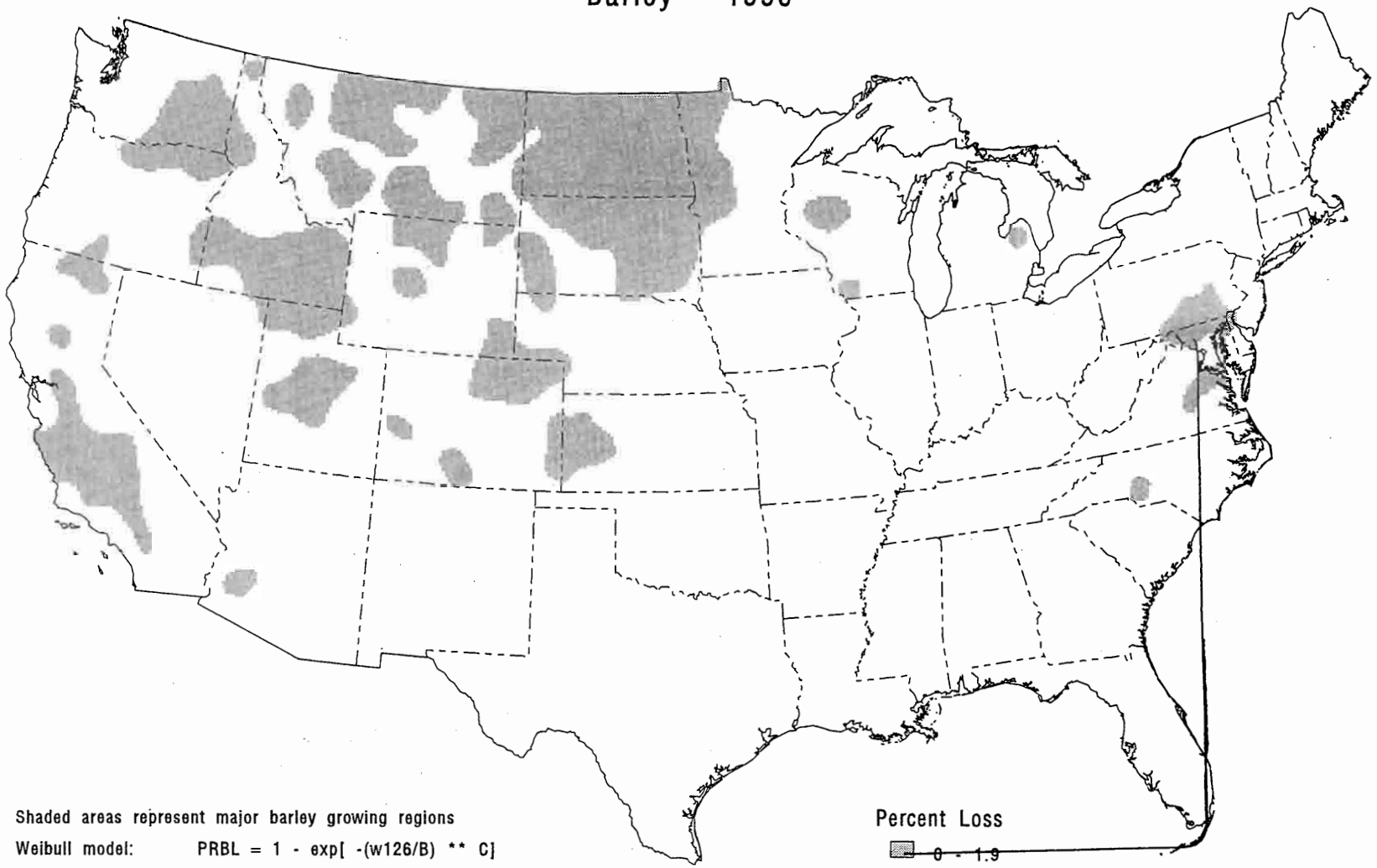
Based on estimates of 1990 maximum 3 month 12 hour W126 ozone exposure

Note: NCLAN exposure-response functions used in projections of yield loss. This map shows the geographic range for this species. It does not indicate that this species will be found at every point within its range.

//data2/andvh/bio loss3/zalal/ mao.aml 06/05/95

Estimated Yield Loss

Barley - 1990



Shaded areas represent major barley growing regions

Weibull model: PRBL = 1 - exp[-(w126/B) ** C]

Weibull parameter: B = 6998.5

Weibull parameter: C = 1.388

Based on estimates of 1990 maximum 3 month 12 hour W126 ozone exposure

Percent Loss

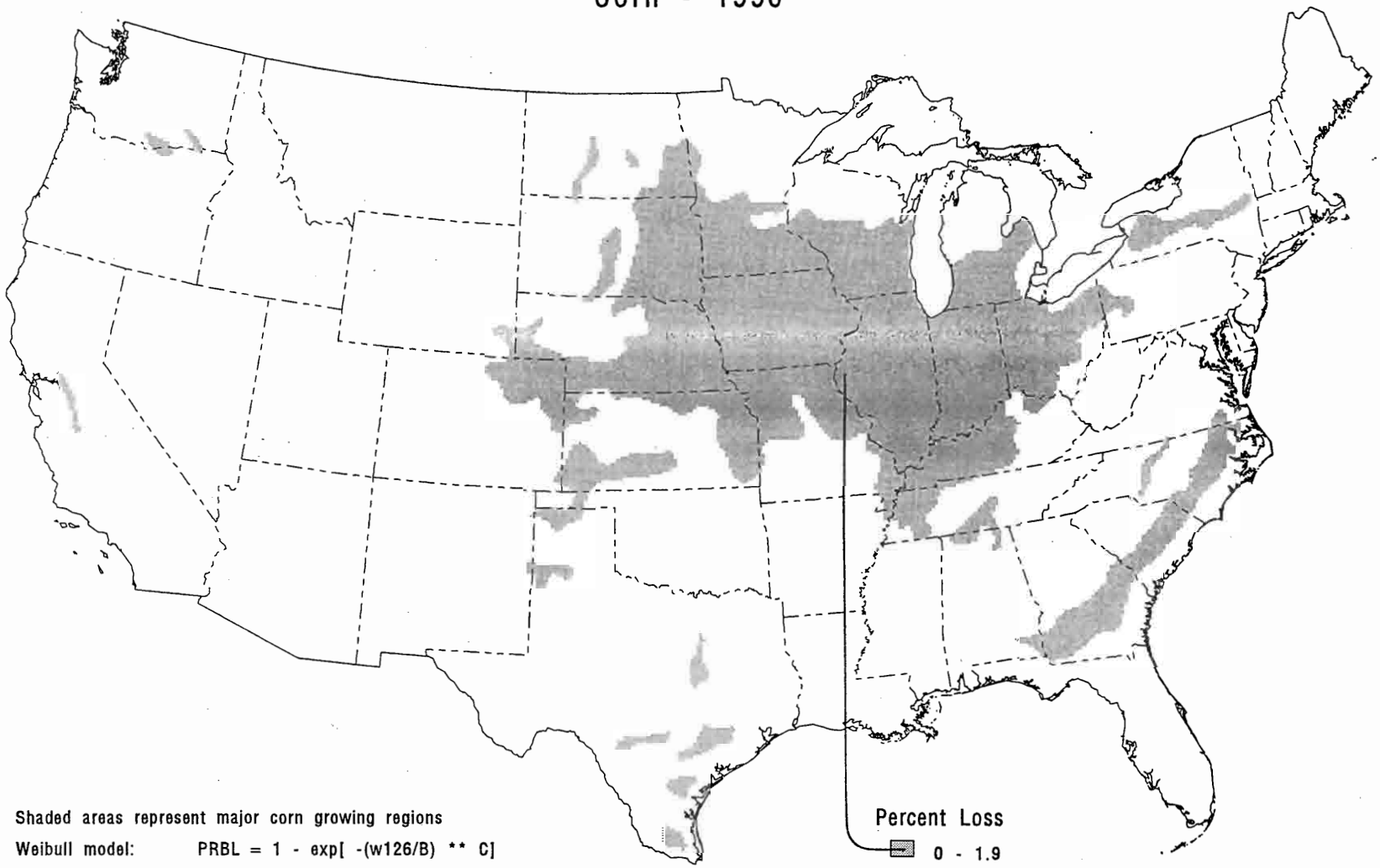
- 0 - 1.9
- 2.0 - 3.9
- 4.0 - 5.9
- 6.0 - 9.9
- > 10.0

Note: NCLAN exposure-response functions used in projections of yield loss. This map shows the geographic range for this species. It does not indicate that this species will be found at every point within its range.

//data2/ondvh/bio loss3/zplot/ map.aml 06/05/95

Estimated Yield Loss

Corn - 1990



Shaded areas represent major corn growing regions

Weibull model: PRBL = 1 - exp[-(w126/B) ** C]

Weibull parameter: B = 97.9

Weibull parameter: C = 2.966

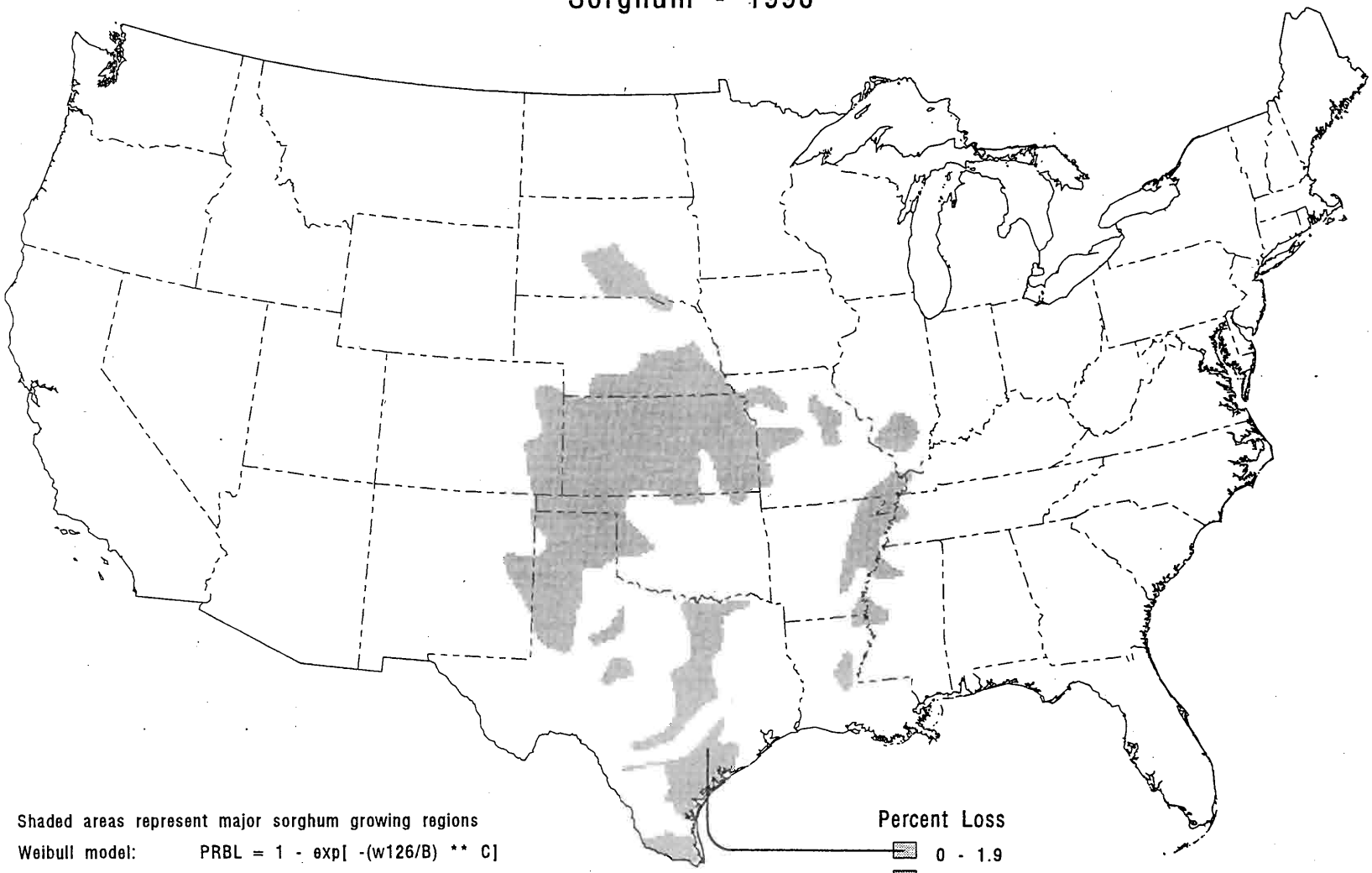
Based on estimates of 1990 maximum 3 month 12 hour W126 ozone exposure

Note: NCLAN exposure-response functions used in projections of yield loss. This map shows the geographic range for this species. It does not indicate that this species will be found at every point within its range.

//data2/andvh/bio loss3/zolat/ mao.aml 06/05/95

Estimated Yield Loss

Sorghum - 1990



Shaded areas represent major sorghum growing regions

Weibull model: PRBL = 1 - exp[-(w126/B) ** C]

Weibull parameter: B = 205.3

Weibull parameter: C = 1.957

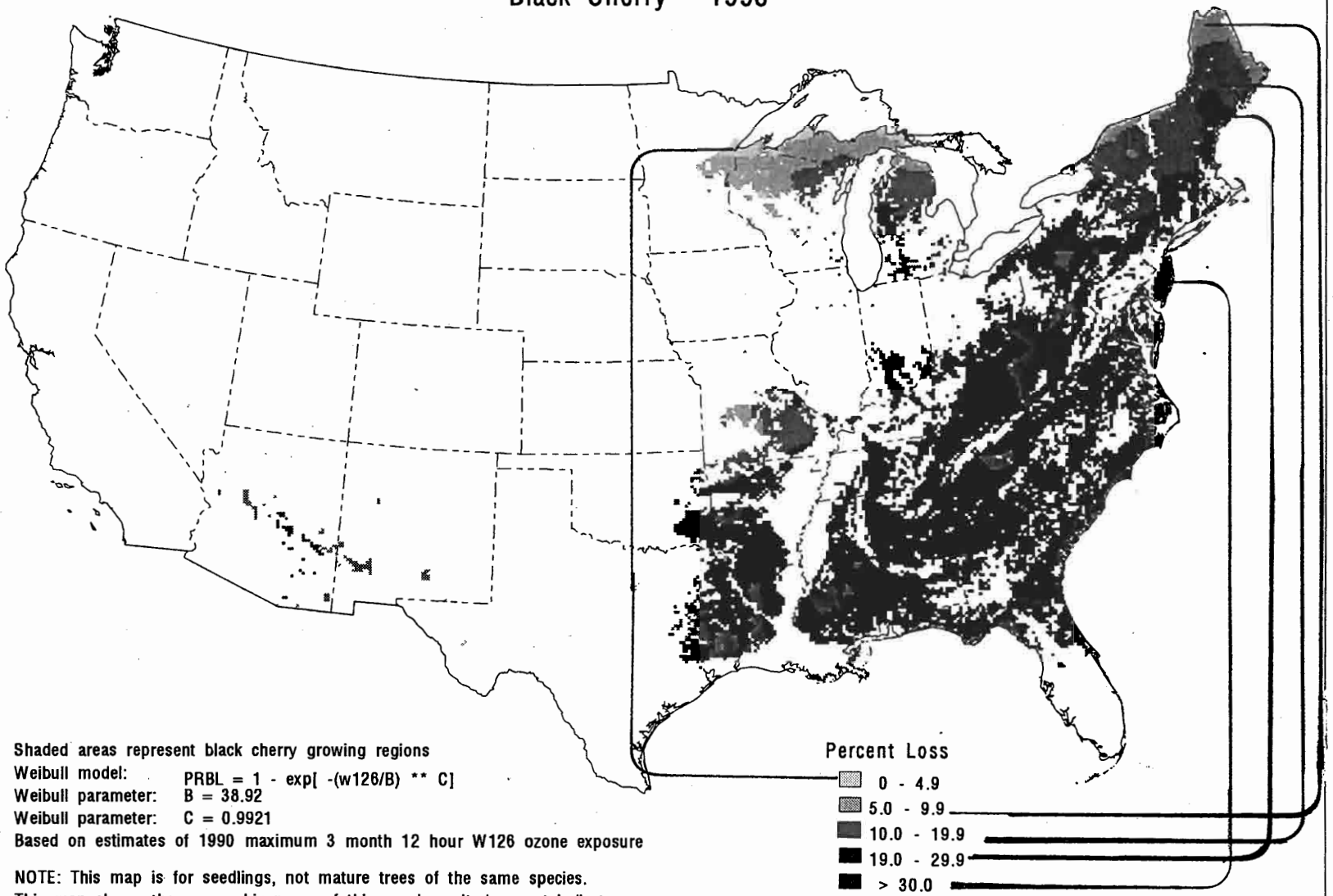
Based on estimates of 1990 maxmum 3 month 12 hour W126 ozone exposure

Note: NCLAN exposure-response functions used in projections of yield loss. This map shows the geographic range for this species. It does not indicate that this species will be found at every point within its range.

//data2/andyh/bio_loss3/zplot/ map.aml 06/05/95

Estimated Seedling Biomass Loss

Black Cherry - 1990



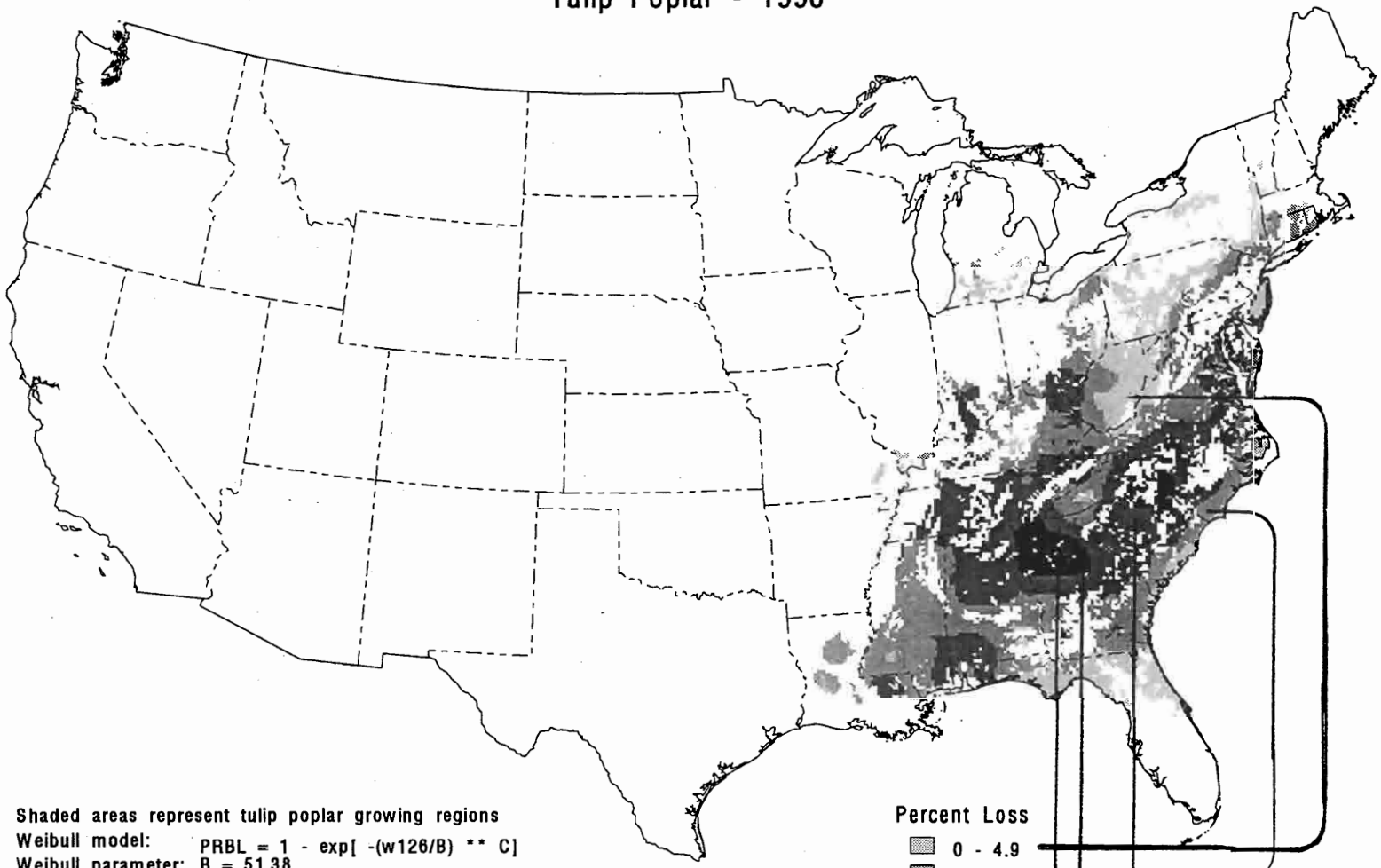
Shaded areas represent black cherry growing regions
Weibull model: PRBL = 1 - exp[-(w126/B) ** C]
Weibull parameter: B = 38.92
Weibull parameter: C = 0.9921
Based on estimates of 1990 maximum 3 month 12 hour W126 ozone exposure

NOTE: This map is for seedlings, not mature trees of the same species. This map shows the geographic range of this species. It does not indicate that this species will be found at every point within its range.

//data2/andvh/bio loss4/zalot/ map.aml 04/23/96

Estimated Seedling Biomass Loss

Tulip Poplar - 1990



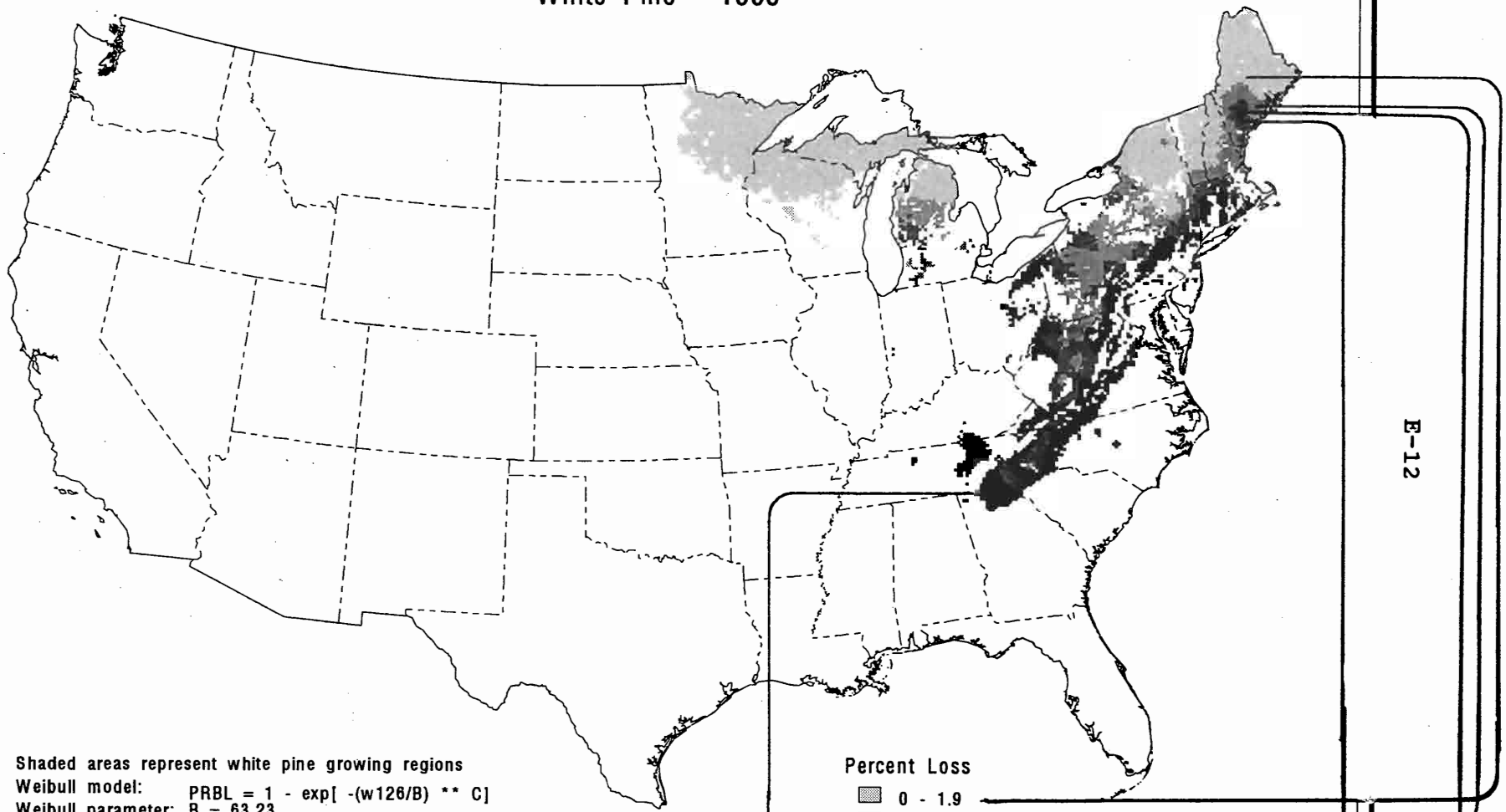
Shaded areas represent tulip poplar growing regions
Weibull model: PRBL = 1 - exp[-(w126/B) ** C]
Weibull parameter: B = 51.38
Weibull parameter: C = 2.0889
Based on estimates of 1990 maximum 3 month 12 hour W126 ozone exposure

NOTE: This map is for seedlings, not mature trees of the same species. This map shows the geographic range of this species. It does not indicate that this species will be found at every point within its range.

//data2/andvh/bio_loss4/zalot/_map.aml 04/23/96

Estimated Seedling Biomass Loss

White Pine - 1990



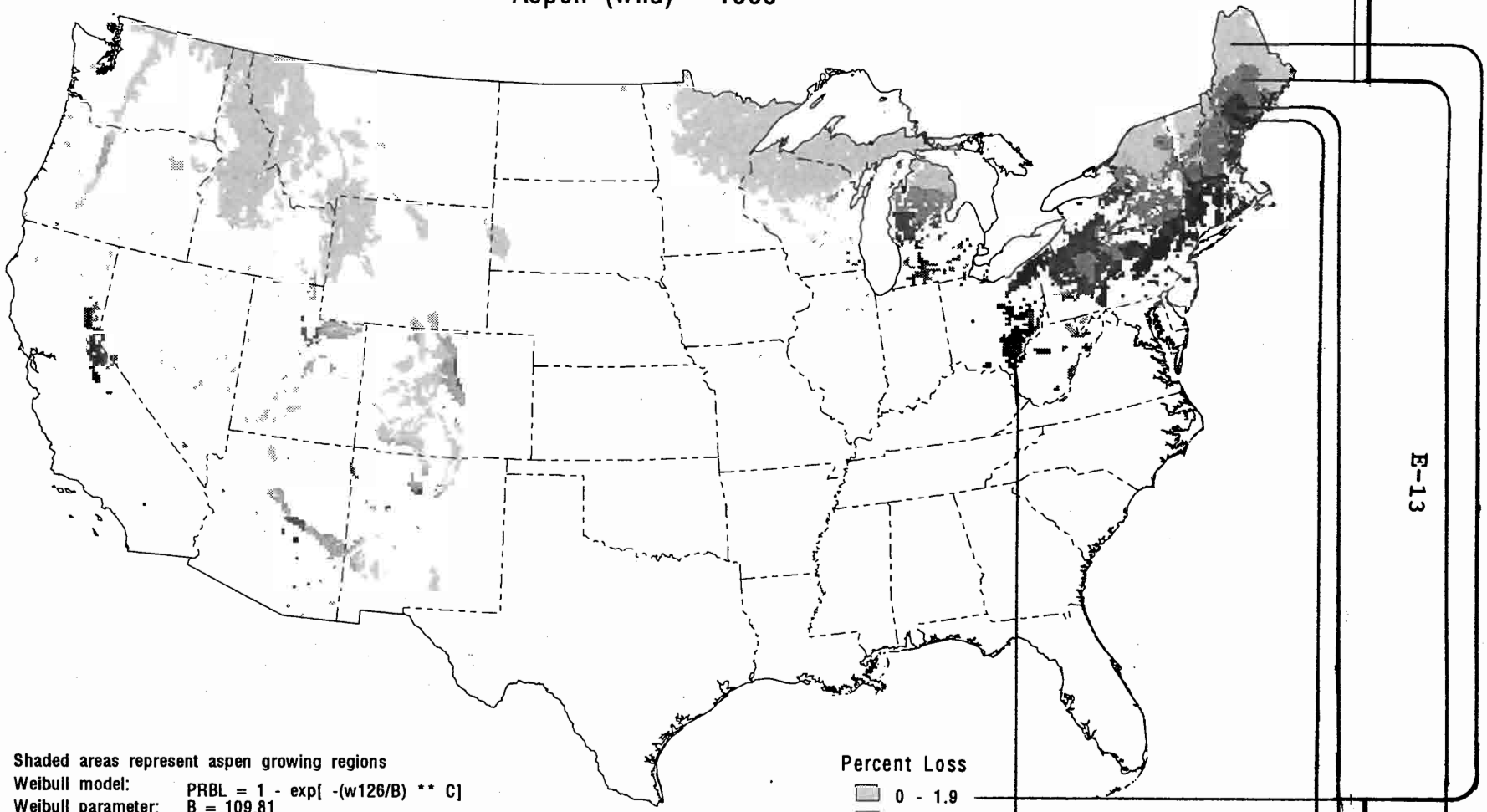
Shaded areas represent white pine growing regions
Weibull model: PRBL = 1 - exp[-(w126/B) ** C]
Weibull parameter: B = 63.23
Weibull parameter: C = 1.6582
Based on estimates of 1990 maximum 3 month 12 hour W126 ozone exposure

NOTE: This map is for seedlings, not mature trees of the same species. This map shows the geographic range of this species. It does not indicate that this species will be found at every point within its range.

//data2/andvh/bio loss4/zalat/ mao.aml 04/23/96

Estimated Seedling Biomass Loss

Aspen (wild) - 1990



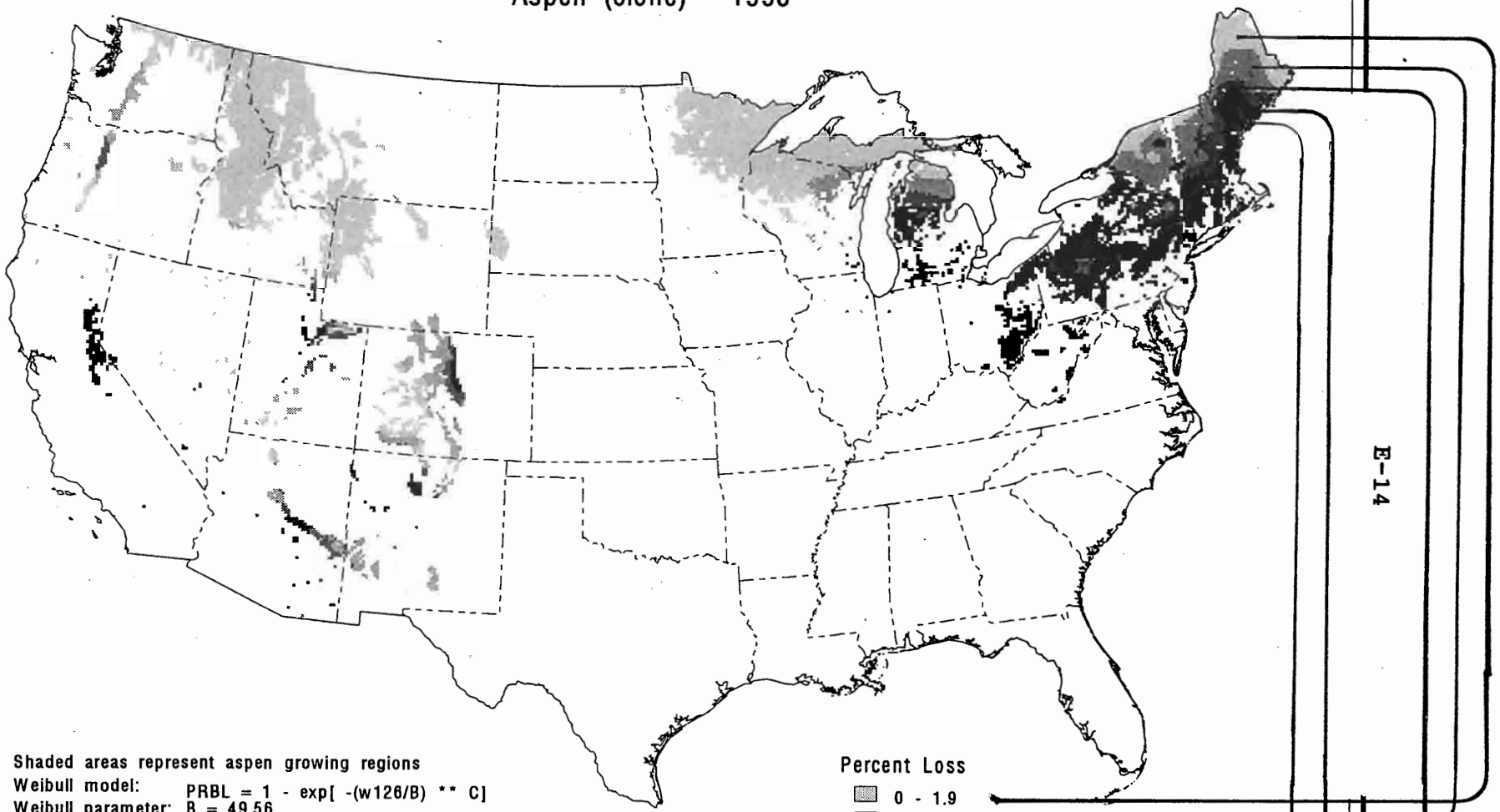
Shaded areas represent aspen growing regions
Weibull model: PRBL = 1 - exp[-(w126/B) ** C]
Weibull parameter: B = 109.81
Weibull parameter: C = 1.2198
Based on estimates of 1990 maximum 3 month 12 hour W126 ozone exposure

NOTE: This map is for seedlings, not mature trees of the same species. This map shows the geographic range of this species. It does not indicate that this species will be found at every point within its range.

//data2/andvh/bio_loss4/zalot/ map.aml 04/23/96

Estimated Seedling Biomass Loss

Aspen (clone) - 1990



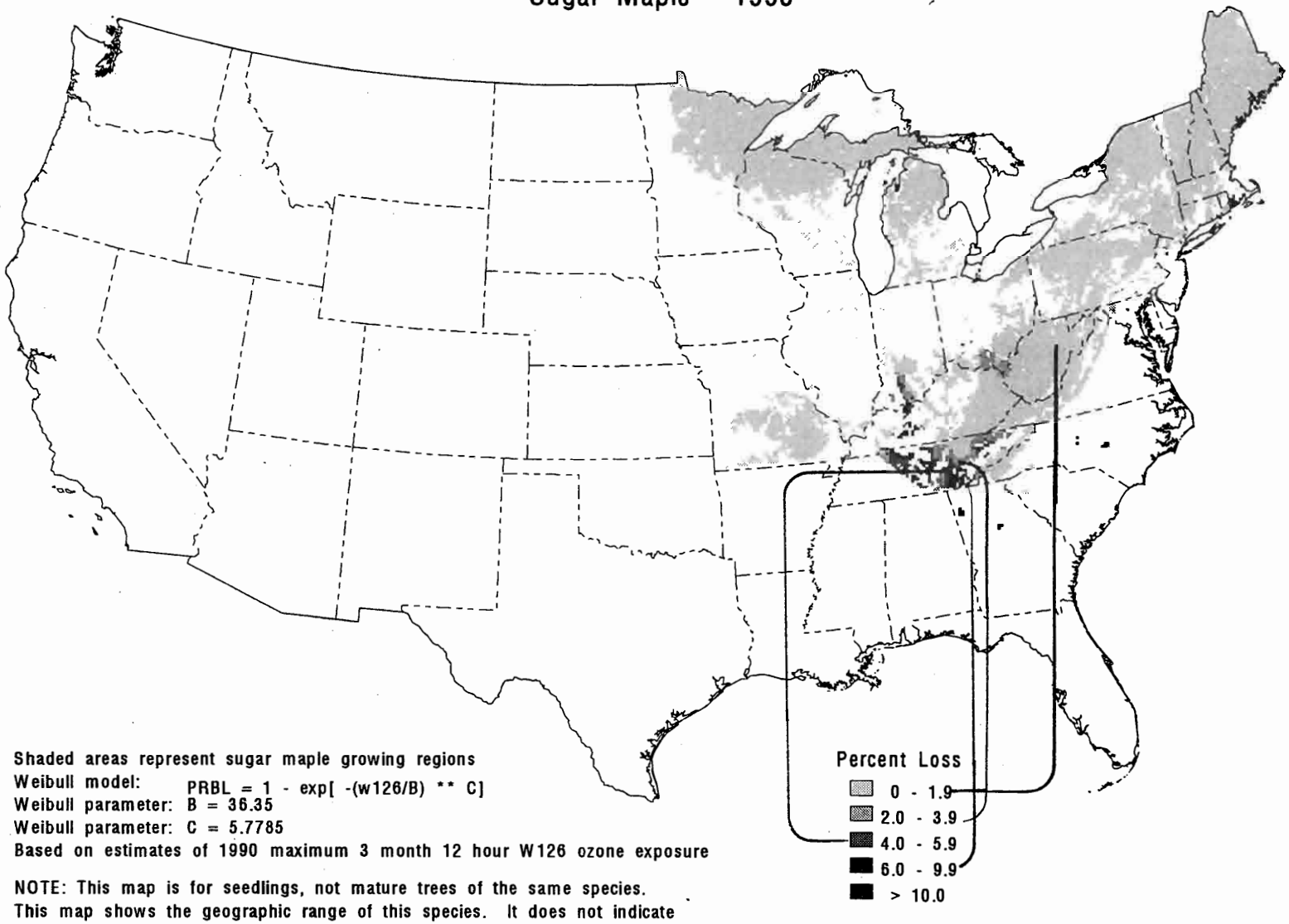
Shaded areas represent aspen growing regions
Weibull model: PRBL = 1 - exp[-(w126/B) ** C]
Weibull parameter: B = 49.56
Weibull parameter: C = 1.4967
Based on estimates of 1990 maximum 3 month 12 hour W126 ozone exposure

NOTE: This map is for seedlings, not mature trees of the same species. This map shows the geographic range of this species. It does not indicate that this species will be found at every point within its range.

//data2/ondvh/bio loss4/zalat/ map.aml 04/23/96

Estimated Seedling Biomass Loss

Sugar Maple - 1990



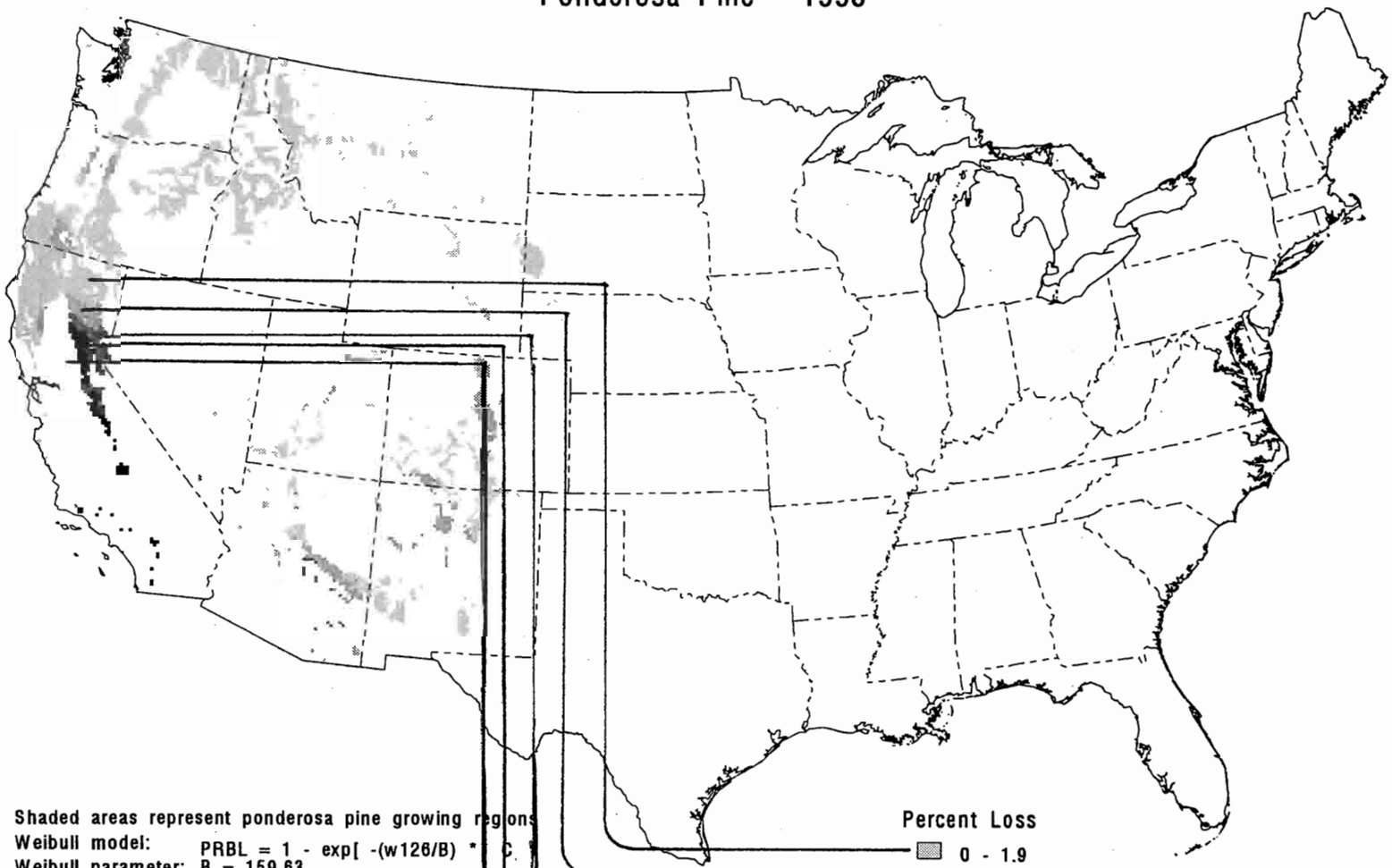
Shaded areas represent sugar maple growing regions
Weibull model: PRBL = 1 - exp[-(w126/B) ** C]
Weibull parameter: B = 36.35
Weibull parameter: C = 5.7785
Based on estimates of 1990 maximum 3 month 12 hour W126 ozone exposure

NOTE: This map is for seedlings, not mature trees of the same species. This map shows the geographic range of this species. It does not indicate that this species will be found at every point within its range.

//data2/andvh/bio_loss4/zalot/ map.aml 04/23/96

Estimated Seedling Biomass Loss

Ponderosa Pine - 1990



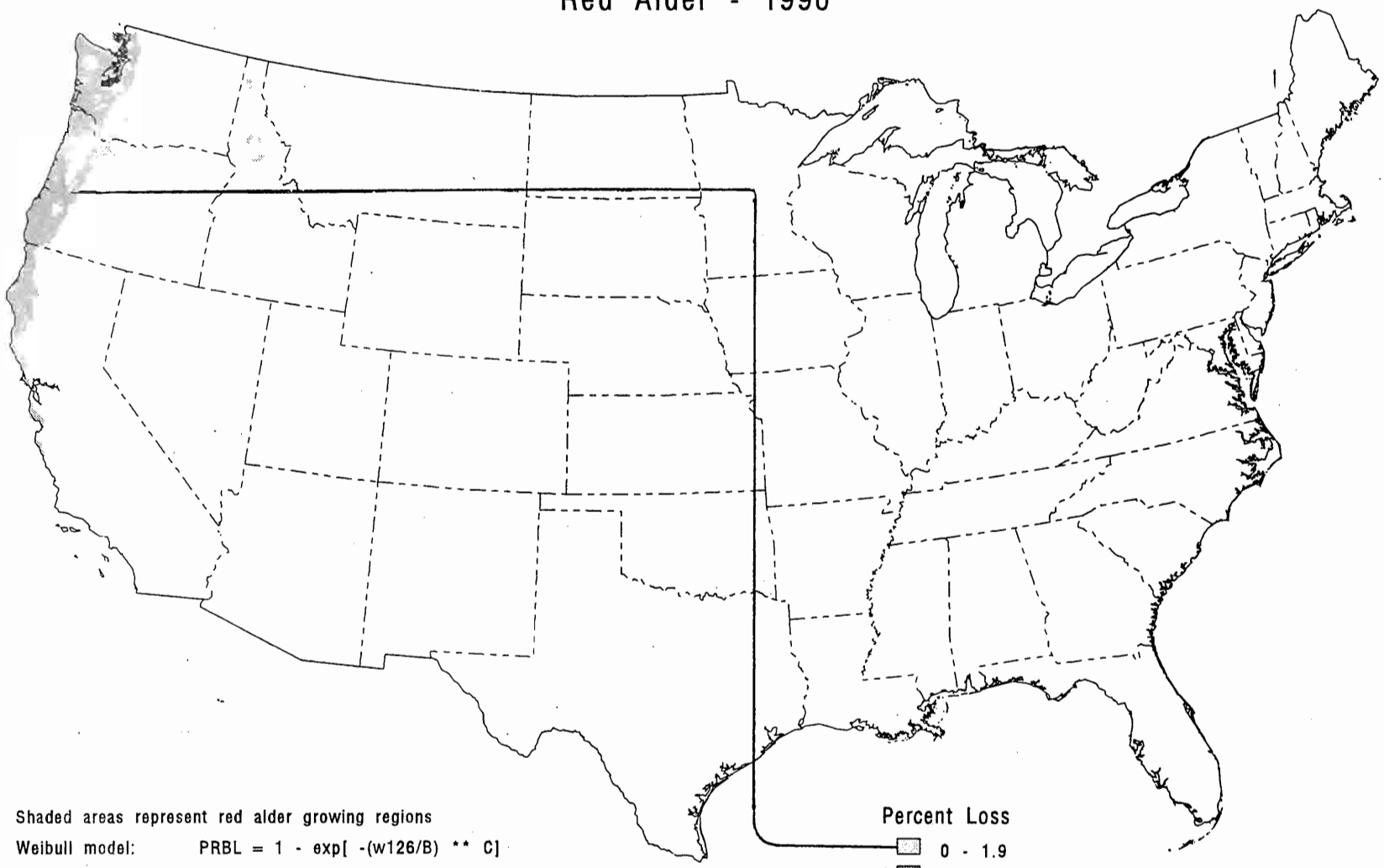
Shaded areas represent ponderosa pine growing regions
Weibull model: PRBL = 1 - exp[-(w126/B) * C
Weibull parameter: B = 159.63
Weibull parameter: C = 1.1190
Based on estimates of 1990 maximum 3 month 12 hour W126 ozone exposure

NOTE: This map is for seedlings, not mature trees of the same species.
This map shows the geographic range of this species. It does not indicate that this species will be found at every point within its range.

//data2/ondyh/bio loss4/zalot/ map.aml 04/23/96

Estimated Seedling Biomass Loss

Red Alder - 1990



Shaded areas represent red alder growing regions

Weibull model: PRBL = 1 - exp[-(w126/B) ** C]

Weibull parameter: B = 179.06

Weibull parameter: C = 1.2377

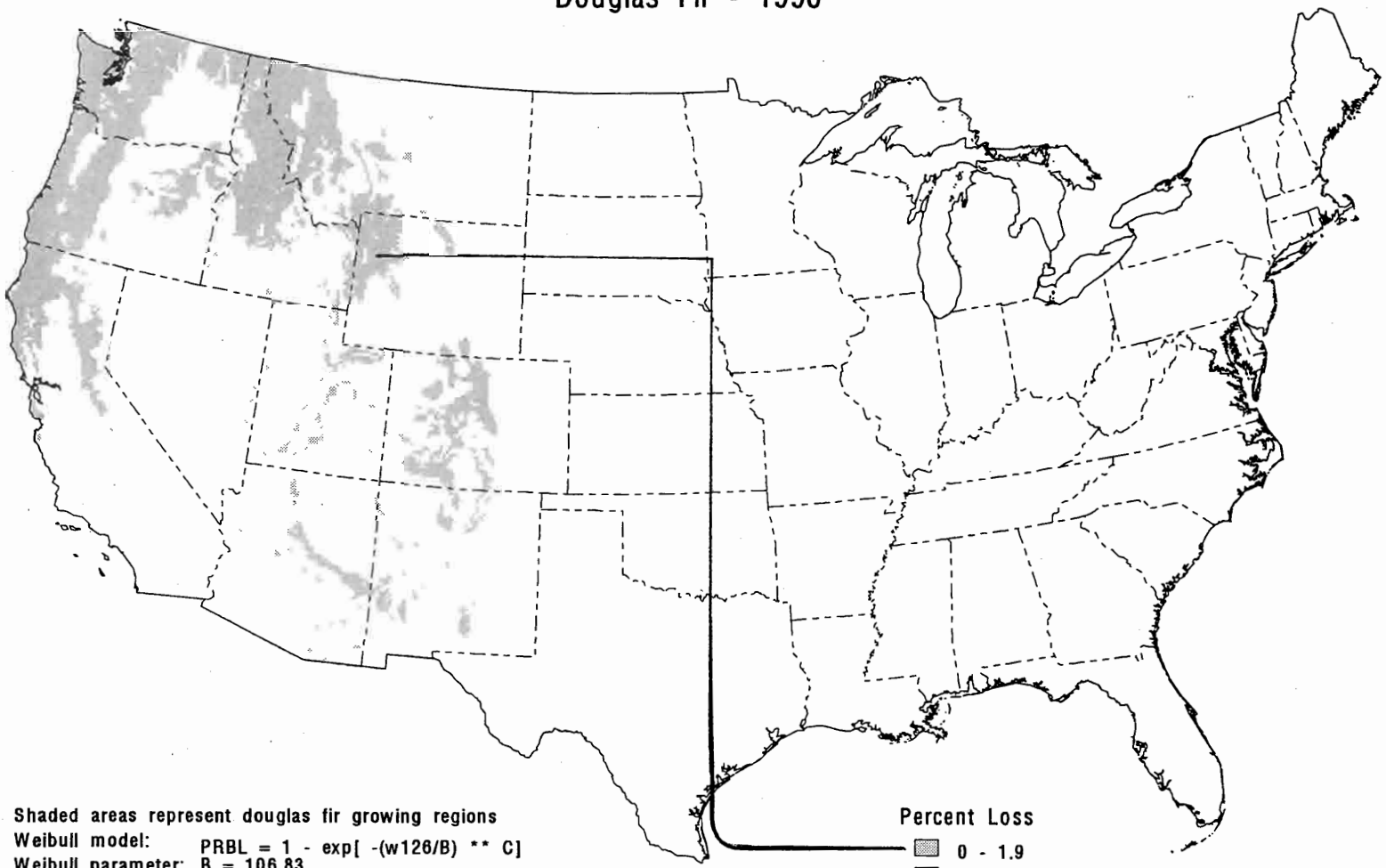
Based on estimates of 1990 maximum 3 month 12 hour W126 ozone exposure

Note: This map is for seedlings, not mature trees of the same species. This map shows the geographic range of this species. It does not indicate that this species will be found at every point within its range.

//data2/andyh/bio_loss3/zalol/ map.aml 06/06/95

Estimated Seedling Biomass Loss

Douglas Fir - 1990



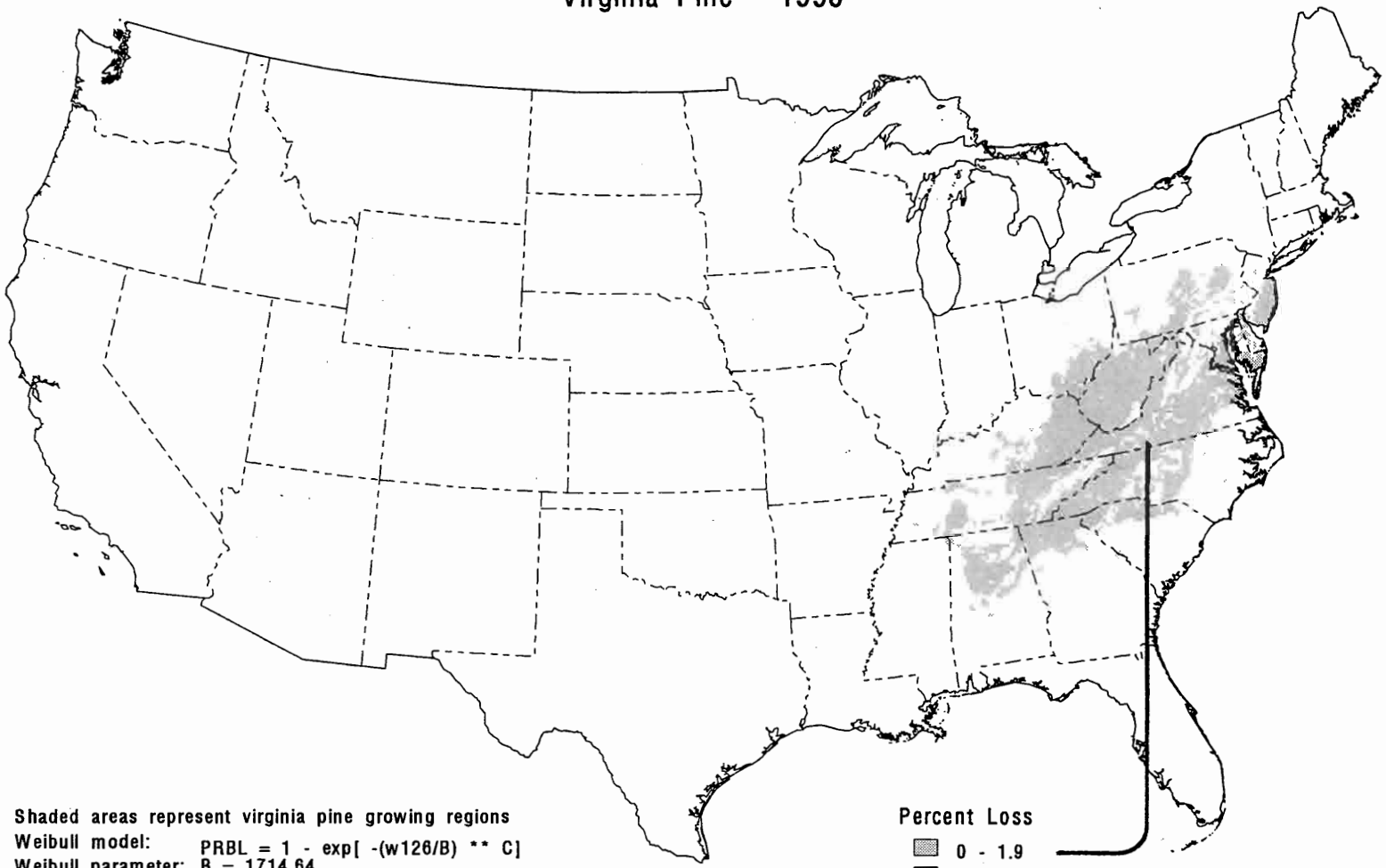
Shaded areas represent douglas fir growing regions
Weibull model: PRBL = 1 - exp[-(w126/B) ** C]
Weibull parameter: B = 106.83
Weibull parameter: C = 5.9631
Based on estimates of 1990 maximum 3 month 12 hour W126 ozone exposure

NOTE: This map is for seedlings, not mature trees of the same species. This map shows the geographic range of this species. It does not indicate that this species will be found at every point within its range.

//data2/gndvh/bio loss4/zolot/ map.aml 04/23/96

Estimated Seedling Biomass Loss

Virginia Pine - 1990



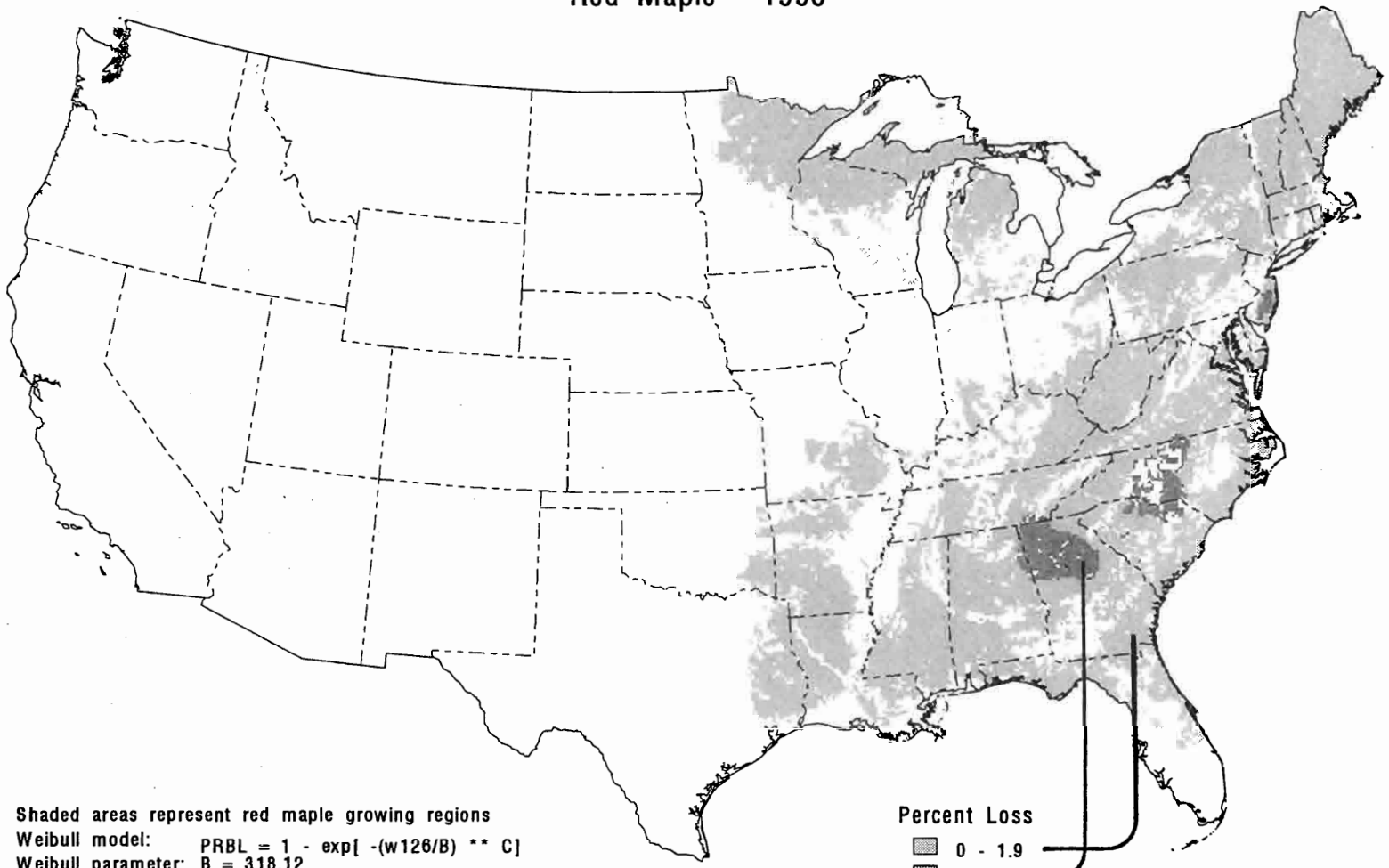
Shaded areas represent virginia pine growing regions
Weibull model: PRBL = 1 - exp[-(w126/B) ** C]
Weibull parameter: B = 1714.64
Weibull parameter: C = 1.0
Based on estimates of 1990 maximum 3 month 12 hour W126 ozone exposure

NOTE: This map is for seedlings, not mature trees of the same species. This map shows the geographic range of this species. It does not indicate that this species will be found at every point within its range.

//data2/andvh/bio loss4/zplot/ mao.aml 04/23/96

Estimated Seedling Biomass Loss

Red Maple - 1990



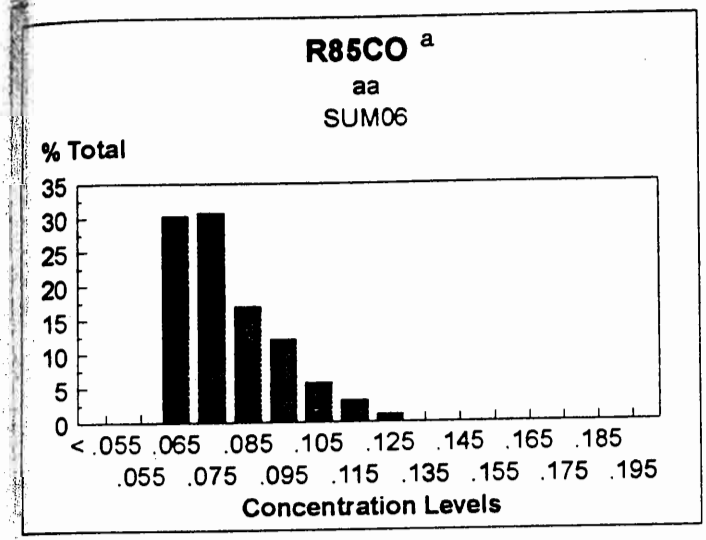
Shaded areas represent red maple growing regions
Weibull model: PRBL = 1 - exp[-(w126/B) ** C]
Weibull parameter: B = 318.12
Weibull parameter: C = 1.3756
Based on estimates of 1990 maximum 3 month 12 hour W126 ozone exposure

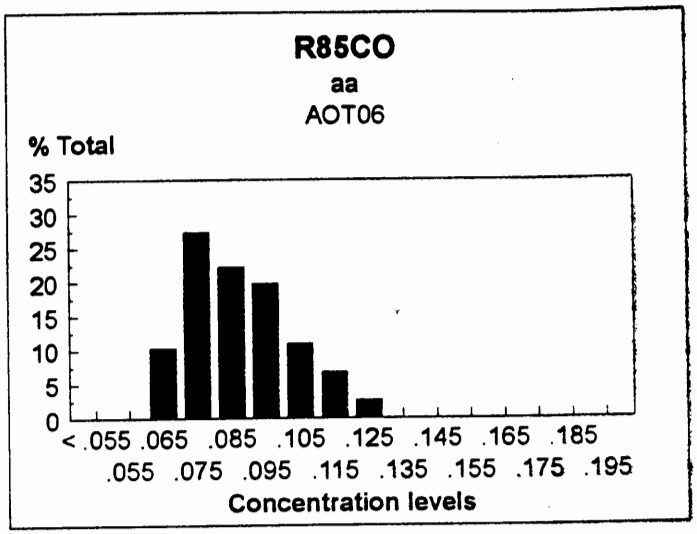
NOTE: This map is for seedlings, not mature trees of the same species. This map shows the geographic range of this species. It does not indicate that this species will be found at every point within its range.

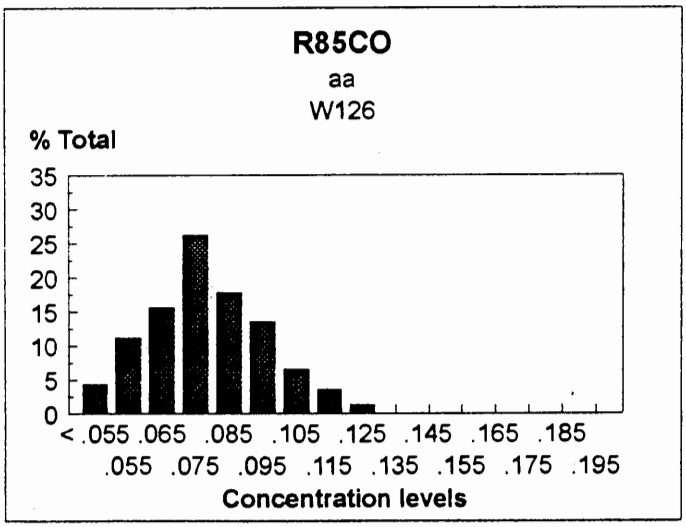
//data2/andvh/bio_loss4/zelot/ map.aml 04/23/96

APPENDIX F

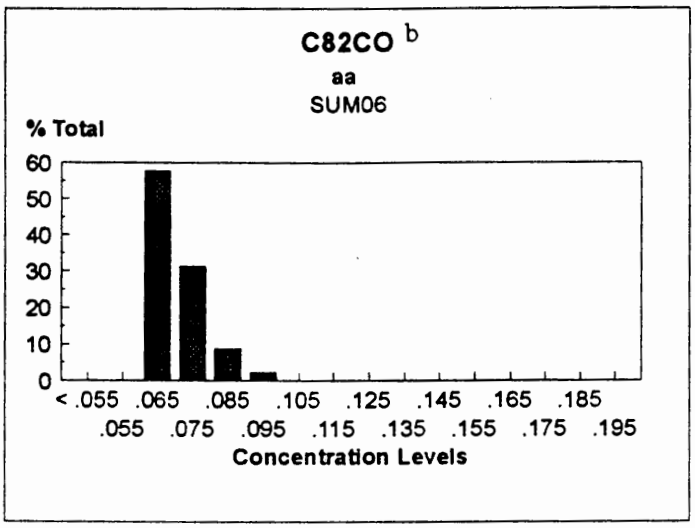
Selected Ambient Ozone Air Quality Distributions for NCLAN, Rural (Class I) and Urban Sites in Terms of Three Different Exposure Indices

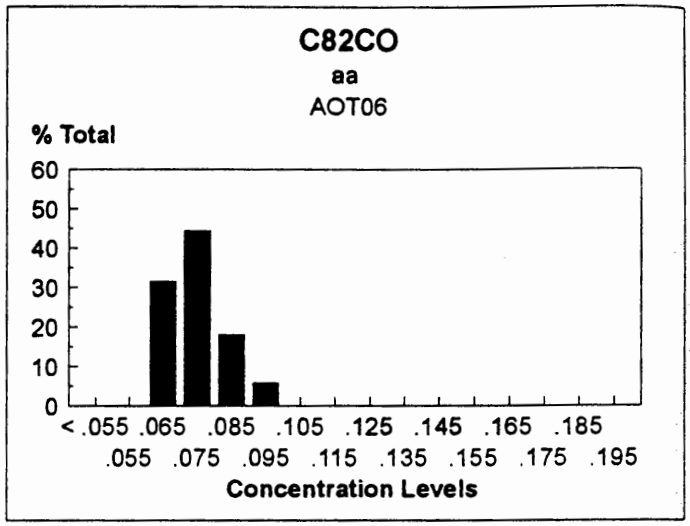


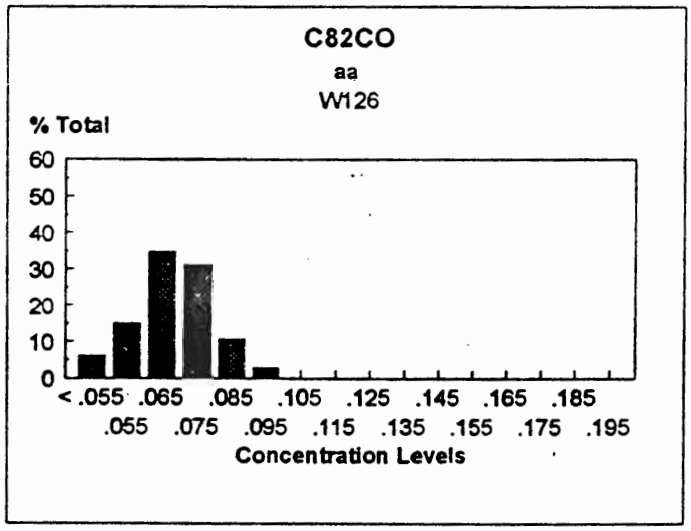




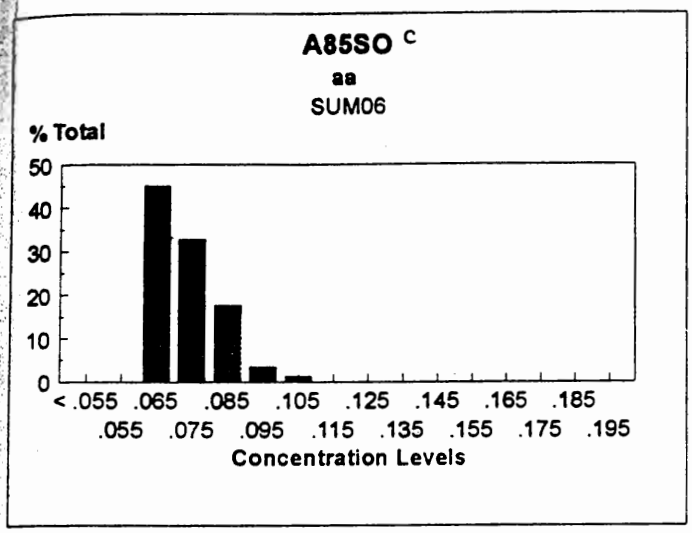
[a]R85CO: Taken from the 1985 NCLAN cotton (McNair) study in Raleigh, NC.

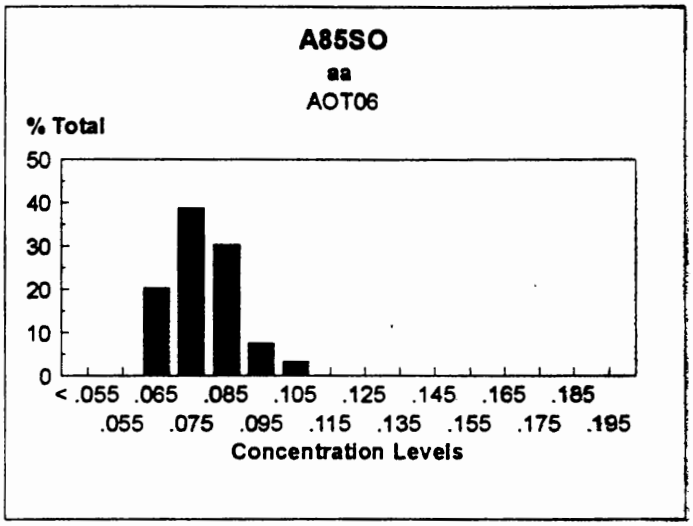


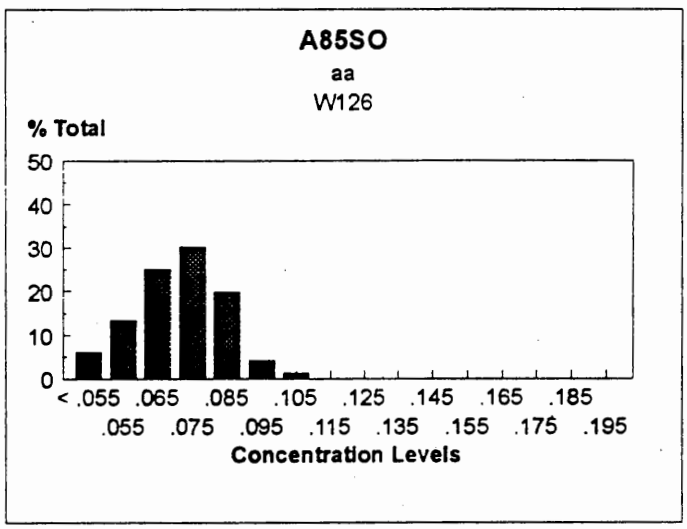




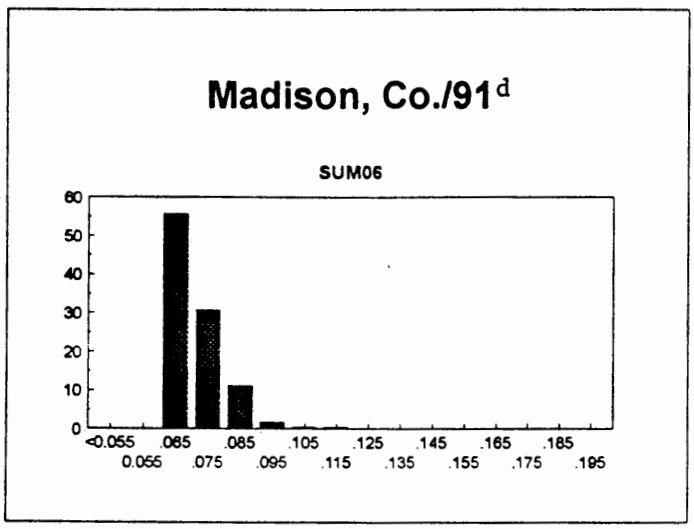
[b]C82CO: Taken from the 1982 NCLAN cotton (Shafer) study in California

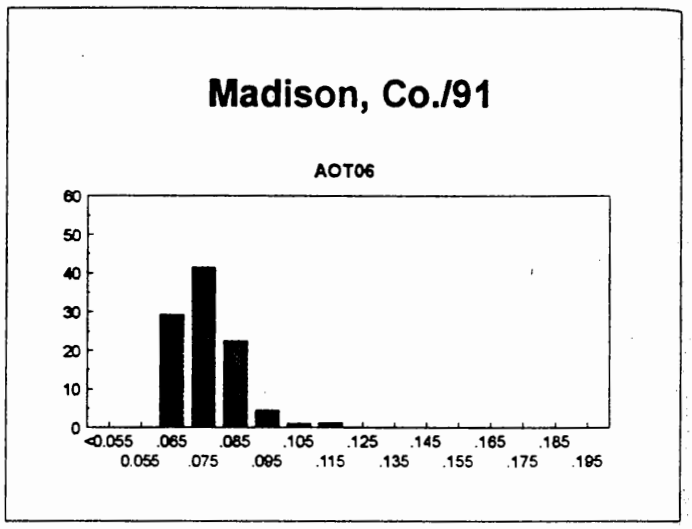


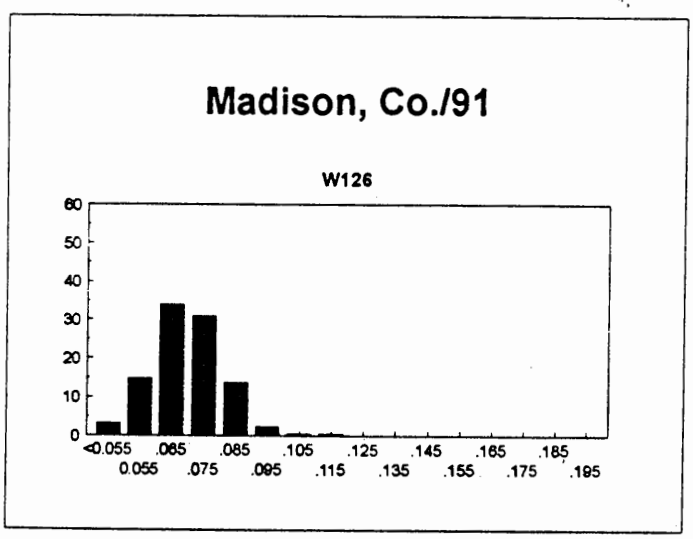




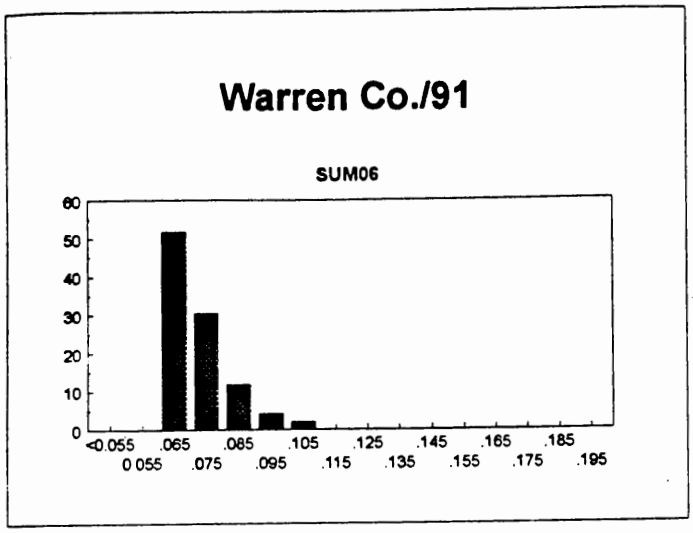
[c]A85SO: Taken from the 1985 NCLAN soybean (Corsoy) study in Argonne, Illinois.

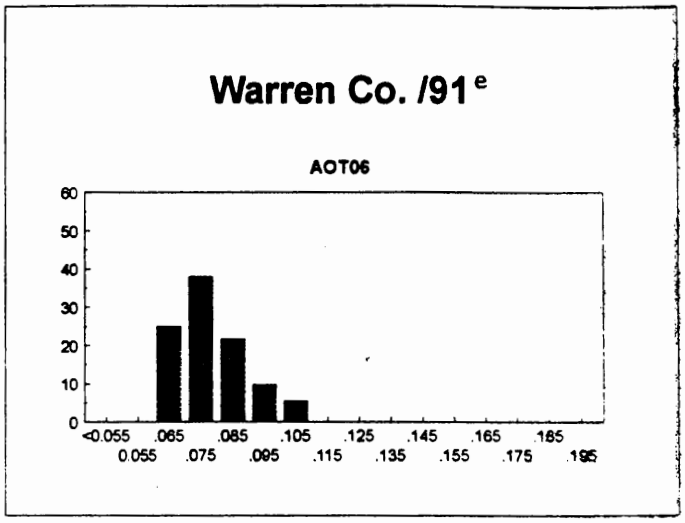


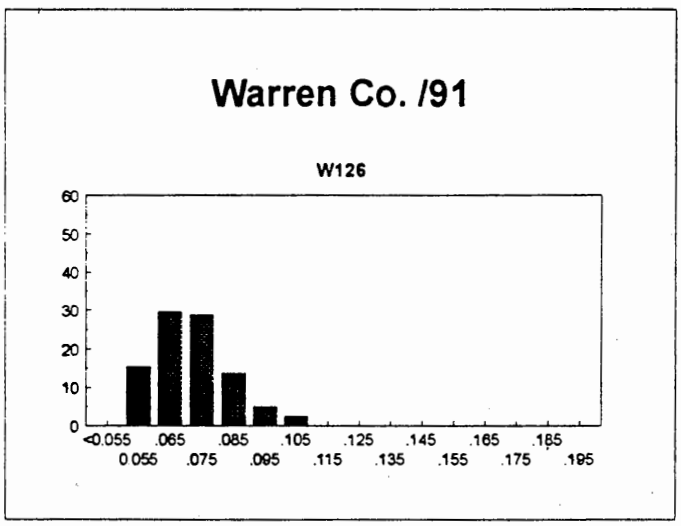




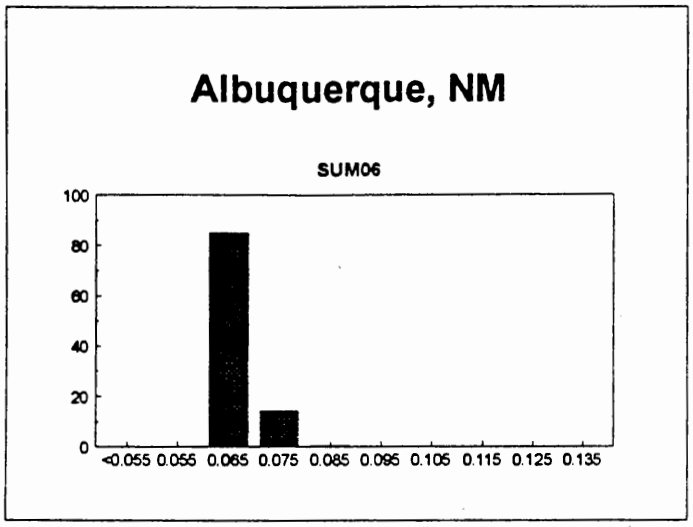
[d]Madison County, 1991: Big Meadows Site, Shenandoah National Park, Virginia. Elevation 1073m.







[e]Warren County, 1991: Dickey Ridge Site, Shenandoah National Park, Virginia. Elevation 610m.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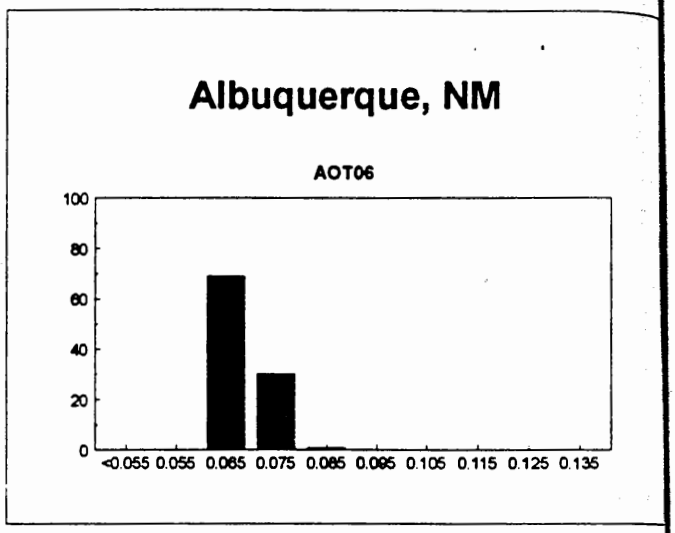
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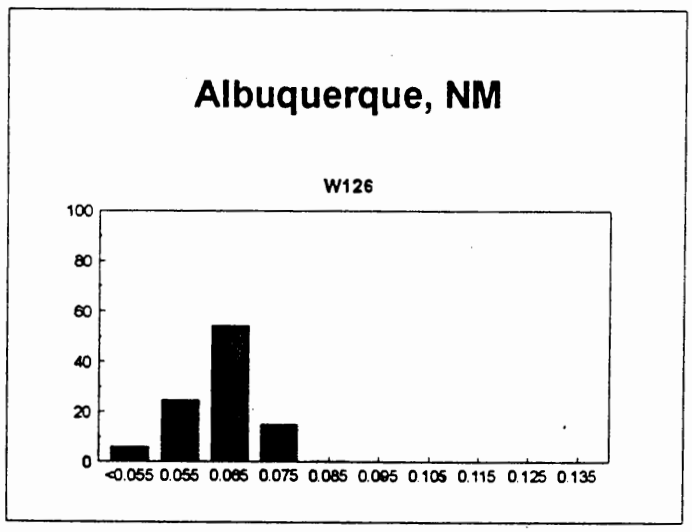
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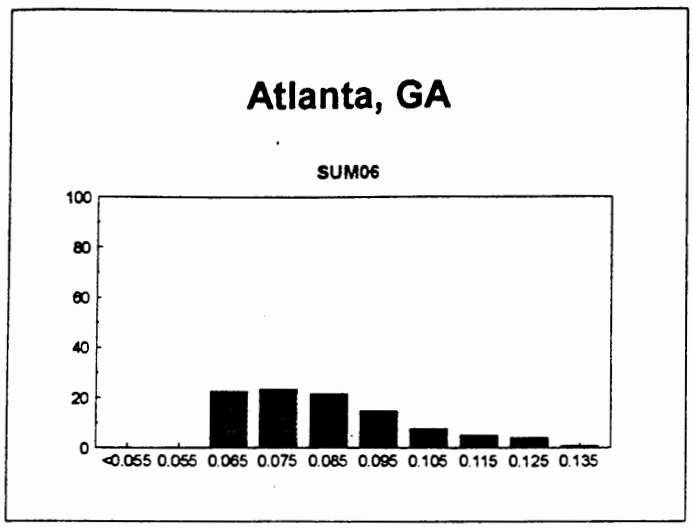
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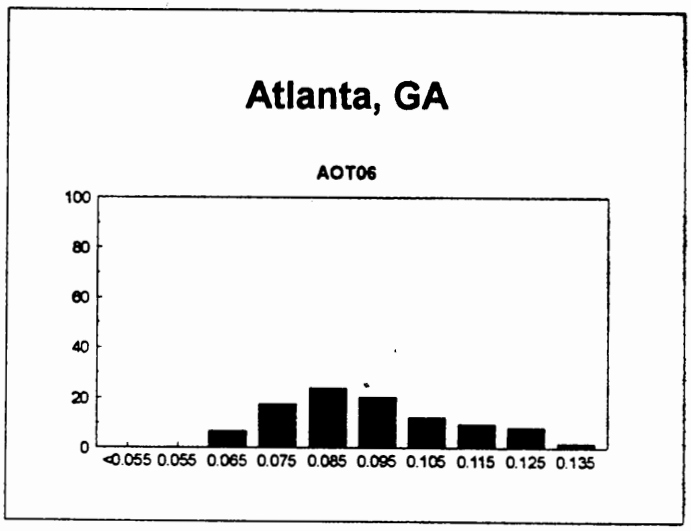
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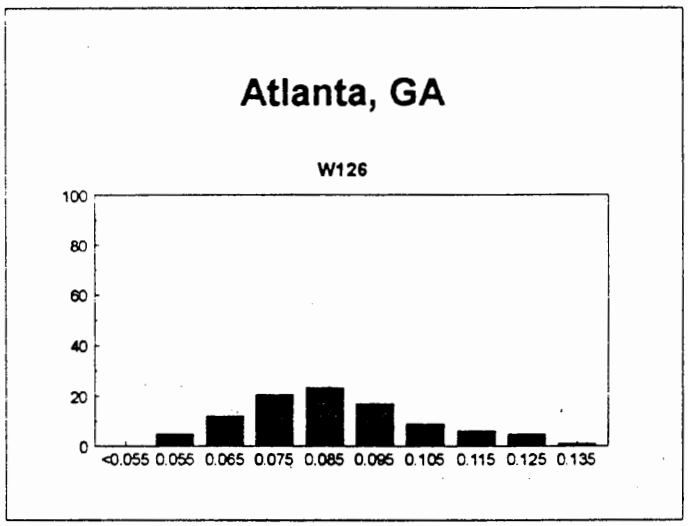
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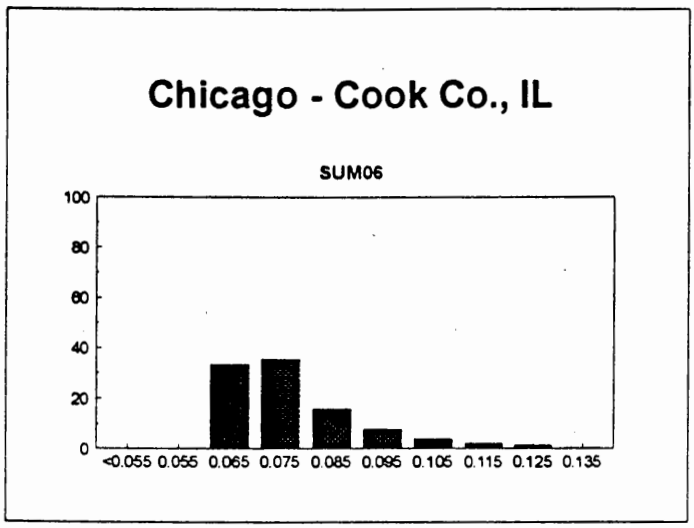
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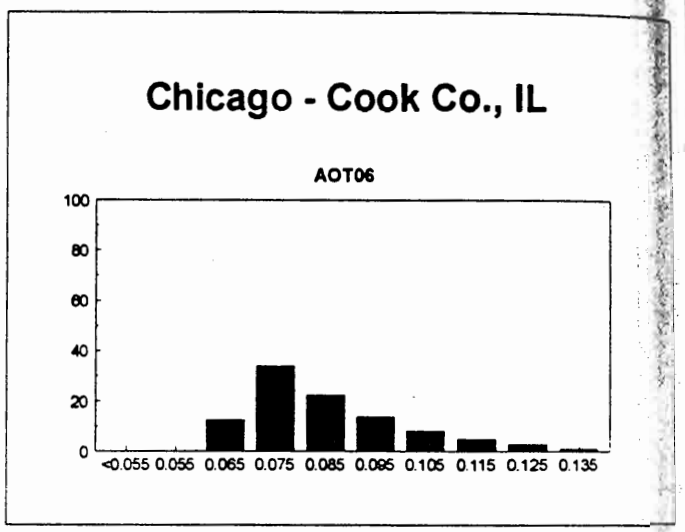
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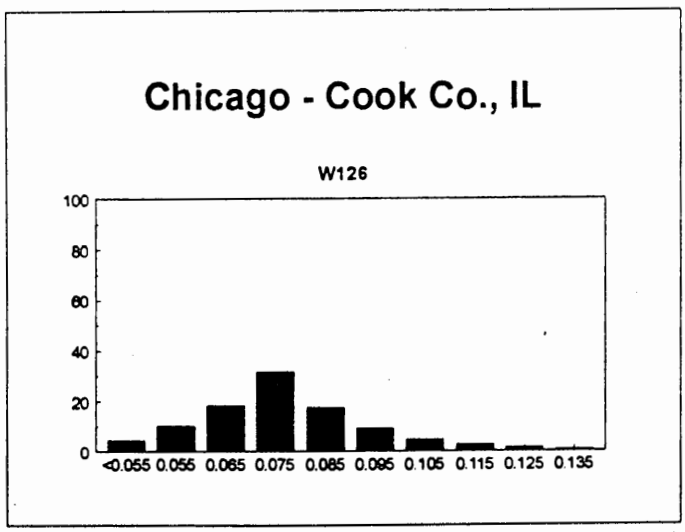
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APPENDIX G

Letters of Closure on the Criteria Document and Staff Paper from the Chairman of the Clean Air Scientific Advisory Committee to the Administrator of the EPA

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460
November 28, 1995

EPA-SAB-CASAC-LTR-96-001

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

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

RE: CASAC Closure on the Air Quality Criteria for Ozone and Related Photochemical Oxidants

Dear Ms. Browner:

A Panel of the Clean Air Scientific Advisory Committee (CASAC) of EPA's Science Advisory Board (SAB) met on July 20 and 21, 1994 and March 21 and 22, 1995, to review drafts of the document entitled Air Quality Criteria for Ozone and Related Photochemical Oxidants. At these meetings and in subsequent written comments, the Committee made extensive recommendations for strengthening the document. In August 1995, revisions to the Criteria Document were mailed to the CASAC members for review. On September 19, 1995, the Panel met to complete this review. The resulting comments by the Committee members note with satisfaction the improvements made in the scientific quality and completeness of the Criteria Document. The changes are consistent with CASAC's recommendations.

At the September 1995 meeting the Panel came to closure on the Criteria Document. It was the consensus of the Panel members that the Criteria Document provides an adequate review of the available scientific data and relevant studies of ozone and related photochemical oxidants. The document is quite comprehensive and will provide an adequate scientific basis for regulatory decisions on ozone and related photochemical oxidants based on available information. At the meeting and subsequently in writing, Panel members provided the Agency with additional comments for consideration. Most of these comments were directed at Chapter 5, the ecological effects chapter. Although the Panel would like to have these comments considered for incorporation in the Criteria Document, the Panel did not feel that it was necessary to review another revised version.

On behalf of the Panel, I would like to thank EPA staff for their efforts in preparing the Criteria Document on the accelerated schedule. Our comments on the OAQPS Staff Paper will follow shortly.

Sincerely,

George T. Wolff

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

SCIENCE ADVISORY BOARD
CLEAN AIR SCIENTIFIC ADVISORY COMMITTEE
OZONE PANEL ROSTER

Chair

Dr. George T. Wolff, General Motors Environmental & Energy Staff, Detroit, MI

Members

Dr. Stephen M. Ayres, Office of International Health Programs, Virginia Commonwealth University, Medical College of Virginia, Richmond, VA

Dr. Jay S. Jacobson, Boyce Thompson Institute, Cornell University, Ithaca, NY

Dr. Joe L. Mauderly, Inhalation Toxicology Research Institute, Lovelace Biomedical & Environmental Research Institute, Albuquerque, NM

Dr. Paulette Middleton, Science & Policy Associates, Inc.,Boulder, CO (did not participate)

Dr. James H. Price, Jr., Texas Natural Resource Conservation Commission, Austin, TX

Consultants to CASAC

Dr. Stephen D. Colome, Integrated Environmental Services, University Tower, Irvine, CA

Dr. A. Myrick Freeman, Department of Economics, Bowdoin College,, Brunswick, ME

Dr. Allan Legge, Biosphere Solutions,Calgary, Alberta, CANADA

Dr. Morton Lippmann, Institute of Environmental Medicine, New York University Medical Center, Tuxedo, NY

Dr. William Manning, Department of Plant Pathology, University of Massachusetts, Amherst, MA

Dr. Roger O. McClellan, Chemical Industry Institute of Toxicology, Research Triangle Park, NC

Dr. D. Warner North, Decision Focus, Inc., Mountain View, CA

Dr. Frank E. Speizer, Harvard Medical School, Channing Lab, Boston, MA

Dr. George Taylor, Biological Services Center, Desert Research Institute, University of Nevada, Reno, NV

Dr. Mark J. Utell, Pulmonary Disease Unit, University of Rochester Medical Center, Rochester, NY

Science Advisory Board Staff

Mr. A. Robert Flaak, Designated Federal Official, U. S. Environmental Protection Agency, Science Advisory Board (1400F), 401 M Street, SW, Washington, DC 20460

Ms. Connie Valentine, Staff Secretary, U. S. Environmental Protection Agency, Science Advisory Board (1400F), 401 M Street, SW, Washington, DC 20460

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

November 30, 1995

EPA-SAB-CASAC-LTR-96-002

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

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

RE: CASAC Closure on the Primary Standard Portion of the Staff Paper for Ozone

Dear Ms. Browner:

A Panel of the Clean Air Scientific Advisory Committee (CASAC) of EPA's Science Advisory Board (SAB) met on March 22, 1995, to review a draft of the primary standard part of the document entitled Review of National Ambient Air Quality Standards for Ozone Assessment of Scientific and Technical Information OAQPS Staff Paper. At that time, a draft of the secondary standard portion of the document was not completed. At the March meeting, the Panel made extensive recommendations for strengthening the document. In August 1995, a revised Staff Paper, which included a first draft of the secondary standard portion was sent to CASAC panel members for review. On September 19 and 20, 1995, the Panel met to compete this review. The Panel members' comments reflect their satisfaction with the improvements made in the scientific quality and completeness of the primary standard portion of the Staff Paper. The changes made in that portion of the document are consistent with CASAC's recommendations. However, the Panel Members provided additional comments to your staff at the meeting and subsequently in writing. Although the Panel would like to have these comments considered for incorporation in the Staff Paper, the Panel did not feel that it was necessary to review another revised version and came to closure on the primary standard portion. It was the consensus of the Panel that although our understanding of the health effects of ozone is far from complete, the document provides an adequate scientific basis for making regulatory decisions concerning a primary ozone standard.

The Panel could not come to closure, however, on the secondary standard portion of the Staff Paper which was a first draft. To facilitate further development of

this part of the Staff Paper, the Panel members have provided detailed comments to your staff. The Panel felt that the suggested revisions were extensive enough to warrant a review of the next draft.

I would like to summarize for you the Panel's recommendations concerning the primary standard. It was the consensus of the Panel that EPA's selection of ozone as the surrogate for controlling photochemical oxidants is correct. It was also the consensus of the Panel that an 8-hour standard was more appropriate for a human health-based standard than a 1-hour standard. The Panel was in unanimous agreement that the present 1-hour standard be eliminated and replaced with an 8-hour standard.

The Panel felt that the weight of the health effects evidence indicates that there is no threshold concentration for the onset of biological responses due to exposure to ozone above background concentrations. Based on information now available, it appears that ozone may elicit a continuum of biological responses down to background concentrations. This means that the paradigm of selecting a standard at the lowest-observable-effects-level and then providing an "adequate margin of safety" is no longer possible. It further means that EPA's risk assessments must play a central role in identifying an appropriate level.

To conduct the risk assessments, the Agency had to identify the population at risk and the physiological responses of concern, develop a model to estimate the exposure of this population to ozone, and develop a model to estimate the probability of an adverse physiological response to the exposure. The Panel agrees with EPA that the selection of "outdoor children" and "outdoor workers," particularly those with preexisting respiratory disease are the appropriate populations with the highest risks. After considerable debate, it was the consensus of the Panel that the Agency's criteria for the determination of an adverse physiological response was reasonable. Nevertheless, there was considerable concern that the criteria for grading physiological and clinical responses to ozone was confusing if not misleading. The Panel concurs with the Agency that the models selected to estimate exposure and risk are appropriate models. However, because of the myriad of assumptions that are made to estimate population exposure and risk, large uncertainties exist in these estimates.

The results of two of the risk analyses are presented in Tables VI-1 and VI-2 in the Staff Paper and are reproduced in the attached tables. The ranges of the risk estimates across nine cities for outdoor children are presented in Table VI-1. Because of the large number of stochastic variables used in the exposure models, the exposure estimates vary from run to run. However, the ranges are not reflective of all of the

uncertainties associated with the numerous assumptions that were made to develop the estimates.

The single estimates presented in Table VI-2 do not reflect any of the uncertainties associated with these estimates. (Table VI-2 contains only the estimated hospital admissions due to asthma which account for over 85% of the estimated total hospital admissions due to ozone exposure). These uncertainties need to be explicitly articulated in order to put the estimates in proper perspective. Nevertheless, based on the results presented in these and other similar tables presented in the Staff Paper, the Panel concluded that there is no "bright line" which distinguishes any of the proposed standards (either the level or the number of allowable exceedences) as being significantly more protective of public health. For example, the differences in the percent of outdoor children (Table VI-1) responding between the present standard and the most stringent proposal (8H1EX at 0.07 ppm) are small and their ranges overlap for all health endpoints. In Table VI-2, the estimates in row 1, which appeared in the draft Staff Paper, suggest considerable differences between the several options. However, when ozone-aggravated asthma admissions are compared to total asthma admissions (rows 5 and 6), the differences between the various options are small. Consequently, the selection of a specific level and number of allowable exceedences is a policy judgment. Although it was the consensus of the Panel that the ranges of concentrations and allowable exceedences proposed by the Agency were appropriate, a number of Panel members expressed "personal" preferences for the level and number of allowable exceedences. Of the ten panel members who expressed their opinions, all ten favored multiple allowable exceedences, three favored a level of 0.08 ppm, one favored the mid to upper range (0.08 - 0.09 ppm), three favored the upper range (0.09 ppm), one favored a 0.009 - 0.10 ppm range with health advisories issued when the 8-hour ozone concentration was forecasted to exceed 0.007 ppm, and two just endorsed the range presented by the Agency as appropriate and stated that the selection should be a policy decision. The members who favored the lower numbers expressed concern over the evidence for chronic deep lung inflammation from the controlled human and animal exposure studies and the observations of pain on deep inspiration in some subjects.

Because there is no apparent threshold for responses and no "bright line" in the risk assessment, a number of panel members recommended that an expanded air pollution warning system be initiated so that sensitive individuals can take appropriate "exposure avoidance" behavior Since many areas of the country already have an infrastructure in place to designate "ozone action days" when voluntary emission reduction measures are put in place, this idea may be fairly easy to implement.

It was also the consensus of the Panel that the form of the 8-hr standard be more robust than the present 1-hour standard. The present standard is based on an extreme

value statistic which is significantly dependent on stochastic processes such as extreme meteorological conditions. The result is that areas which are near attainment will randomly flip in and out of compliance. A more robust, concentration-based form will minimize the "flip-flops," and provide some insulation from the impacts of extreme meteorological events. The Panel also endorses the staff recommendation for creating a "too close to call" category.

Since the last ozone NAAQS review, the scientific community has made great strides in their understanding of the health effects of ozone exposure because of ongoing research programs. Panel members were very impressed with how much more we understand now as compared to the prior round. Nevertheless, there are still many gaps in our knowledge and large uncertainties in many of the assessments. For example, there is little information available on the frequency of human activity patterns involving outdoor physical exercise. Little is also known about the possible chronic health impacts of ozone exposure over a period of many years. In addition, there is no clear understanding of the significance of the inflammatory response inferred from the broncholavage data. Panel members stated, however, that the scientific community is now in a position to frame the questions that need to be better resolved so the uncertainties can be reduced before the next ozone review in 5 years. For this reason, it is important that research efforts on the health and ecological effects of ozone not be reduced because we have come to closure on this review.

CASAC would appreciate being kept informed of progress on establishing a revised or new ozone standard, and plans for research on ozone effects. Please do not hesitate to contact me if CASAC can be of further assistance in this matter. We look forward to receiving the revisions of the secondary standard portion of the Staff Paper.

Sincerely,

George T. Wolff

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

TABLE VI-1. RANGE OF MEDIAN PERCENT OF OUTDOOR CHILDREN RESPONDING ACROSS NINE U.S. URBAN AREAS UPON ATTAINING ALTERNATIVE AIR QUALITY STANDARDS.

Health Endpoints	Range of Median Risk Estimates Associated With Just Attaining Alternative Standards (percent of outdoor children responding)							
	Alternative 1-Hour NAAQS		Alternative 8-Hour Daily Maximum Standards					
			1 Expected Exceedance Standards				5 Expected Exceedance Standards	
	1H1EX 0.12 ppm	1H1EX 0.10 ppm	8H1EX 0.10 ppm	8H1EX 0.09 ppm	8H1EX 0.08 ppm	8H1EX 0.07 ppm	8H5EX 0.09 ppm	8H5EX 0.08 ppm
FEV_1 decrement ≥ 15%[a]	4.6-13.7	2.6-9.4	6.9-16.1	4.9-11.9	3.3-8.3	1.7-5.1	5.2-14.3	3.3-10.3
FEV_1 decrement ≥ 20%[a]	1.1-5.9	0.4-3.5	2.4-7.3	1.5-4.8	0.9-2.8	0.2-1.2	1.6-6.3	0.8-4.1
Moderate or Severe Pain on Deep Inspiration[b]	0.5-1.6	0.1-0.4	1.0-2.6	0.6-1.7	0.4-1.7	0.2-0.7	0.2-0.8	0.4-1.7
Moderate or Severe Cough[c]	1.4-3.7	0.9-2.6	1.9-3.9	1.5-3.3	1.0-2.5	0.7-1.9	1.5-3.3	1.1-2.9

[a]Risks associated with 8-hour exposures under moderate exertion (equivalent ventilation rate ≥ 15 l min^{-1} m^{-2}) based on exposure-response relationships derived from Follinsbee et al. (1988), Horstman et al. (1989), and McDonnell et al. (1991).

[b]Risks associated with 1-hr exposures under moderate exertion (equivalent ventilation rate ≥ 16 and ≤ 30 l min^{-1} m^{-2}) based on exposure-response relationships derived from Seal et al (1993).

[c]Risks associated with 1-hr exposures under heavy exertion (equivalent ventilation rate ≥ 30 l min^{-1} m^{-2}) based on exposure-response relationships derived from McDonnell et al. (1983).

Table VI-2 (revised)
ESTIMATED HOSPITAL ADMISSIONS FOR ASTHMATICS IN THE NEW YORK CITY AREA

	1H1EX 0.12	1H1EX 0.10	8H1EX 0.10	8H1EX 0.09	8H1EX 0.08	8H1EX 0.07	8H5EX 0.09	8H5EX 0.08	AS IS
Excess Admissions[a]	210	130	240	180	110	60	180	120	=385[d]
% ^ from present std	0%	-38%	+14%	-14%	-48%	-71%	-14%	-42%	+83%
Excess + background[b]	890	810	920	860	790	740	860	800	1065[e]
% ^ from present std	0%	-9%	+3%	-3%	-11%	-17%	-3%	-10%	+20%
All asthma admissions[c]	28,295	28,215	28,325	28,265	28,195	28,145	28,265	28,205	28,470[f]
% ^ from present std	0%	-0.3%	+0.1%	-0.1%	-0.4%	-0.5%	-0.1%	-0.3%	+0.6%

a - excess asthma admissions attributed to ozone levels exceeding a background concentration of 0.04 ppm; from Table VI-2, page 155 in the August 1995 OAQPS Draft Staff Paper

b - asthma admissions included in (a) plus those due to background ozone concentrations; admissions due to background = 1065[e] - 385[d] = 680

c - asthma admissions due to all causes = 28,470[f] - 385[d] - Excess Admissions from row 1

d - estimated from Figure V-15, page 125 in the August 1995 OAQPS Draft Staff Paper

e - from page 127, line 13 in the August 1995 OAQPS Draft Staff Paper

f - total admissions from asthma = total asthmatics (365,000 - from page 126, line 24) x hospitalization rate (78/1000 asthmatics - from page 126, line 29)

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

April 4, 1996

EPA-SAB-CASAC-LTR-96-006

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

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

RE: Closure by the Clean Air Scientific Advisory Committee (CASAC) on the Secondary Standard Portion of the Staff Paper for Ozone

Dear Ms. Browner:

A Panel of the Clean Air Scientific Advisory Committee (CASAC) of EPA's Science Advisory Board (SAB) met on March 22, 1995, to review a draft of the primary standard portion of the document entitled *Review of National Ambient Air Quality Standards for Ozone Assessment of Scientific and Technical Information* - OAQPS Staff Paper. At that time, a draft of the secondary standard portion of the document was not completed. In August, 1995, a revised Staff Paper, which included a first draft of the secondary standard portion was sent to the CASAC panel members for review. On September 19 and 20, 1995, the Panel met to complete this review. The Panel members' comments reflect their satisfaction with the improvements made in the scientific quality and completeness of the primary standard portion of the Staff Paper and reached closure on that part (see CASAC Letter Report: EPA-SAB-CASAC-LTR-96-002, November 30, 1995). However, the Panel could not come to closure on the secondary standard portion of the Staff Paper which was a first draft. To facilitate further development of this part of the Staff Paper, the Panel members provided detailed comments to your staff. The Panel felt that the suggested revisions were extensive enough to warrant a review of the next draft.

On March 21, 1996, a subset of the Panel, consisting of all four of the Panel members with expertise in ozone effects on vegetation plus three additional CASAC members, met in Research Triangle Park, NC to review a second draft of the secondary portion of the Staff Paper. In addition, a Panel member with expertise in economics

reviewed the Staff Paper and provided written comments. Overall, the comments from the Panel members reflected their satisfaction that the Staff Paper was much improved; however, the verbal and written comments provided to your staff indicated that important, additional modifications are still required. Nevertheless, it was the consensus of the Panel that an additional review of the document by the Panel was not necessary. Consequently, the majority of the Panel agreed to come to closure on the Staff Paper assuming that the Agency would incorporate the Panel's latest comments. It was the opinion of six of the seven members of the Panel who were present that the Staff Paper will provide an appropriate scientific basis for making regulatory decisions concerning a secondary ozone standard once the additional changes are incorporated. The additional modifications are summarized below.

It should be pointed out that the Panel members all agreed that damage is occurring to vegetation and natural resources at concentrations below the present 1-hour national ambient air quality standard (NAAQS) of 0.12 ppm. The vegetation effects experts were in agreement that plants appear to be more sensitive to ozone than humans. Further, it was agreed that a secondary NAAQS, more stringent than the present primary standard, was necessary to protect vegetation from ozone. However, agreement on the level and form of such a standard is still elusive for a number of reasons.

The first issue is the level of uncertainty associated with the crop loss risk assessment presented in Tables VII-5a-d through VII-7 of the Staff Paper. While some of the sources of uncertainty are addressed earlier in the Staff Paper, other sources of uncertainty are not addressed at all. The estimates in these Tables should only be presented as rough estimates for a number of reasons. First, the dose-response functions are based upon open-top chamber studies which have the advantage of providing the least amount of environmental modification of any outdoor chamber, but, nevertheless, they still alter ambient microclimate conditions which will introduce uncertainty. In these studies, plant response to ozone has been optimized under conditions which do not reflect the real-life ambient field conditions. Two of the plant experts said that the open-top chamber experiments by their very design and execution produced results that overestimated the effects of ozone on plant yield. The other two experts agreed that the open-top chambers do alter the environment in the chamber with respect to ambient field conditions but did not agree with there being a positive bias. Research has not yet provided methods that clearly are better than open-top chambers for establishing ozone dose-response relationships for a wide variety of crops. Second, the estimated exposures are based on a non-peer-reviewed, empirical model which has not been subjected to any performance evaluation. In addition,

insufficient details are given either in the Staff Paper or the unpublished Agency report for anyone to perform an evaluation. Third, the estimated exposures are then extrapolated to hypothetical scenarios where various secondary NAAQS are attained. Details of this extrapolation procedure are also insufficient to judge the appropriateness of the procedure. Fourth, the exposure estimates are then extrapolated to the entire coterminous U.S. using a Geographic Information System (GIS) which is based on an unpublished, non-peer-reviewed, internal EPA memorandum that contains insufficient details to adequately evaluate the GIS. The exposure estimates and the dose-response function estimates are then input into the economic models which introduce additional uncertainties. Furthermore, the losses are computed from an assumed 12-hr. background ozone concentration of 0.025 ppm which is too low and will over-inflate the crop loss estimates. A more reasonable 12-hr. daylight, summertime background is more likely closer to the 8-hr. background of 0.03-0.05 ppm. As a result, the Panel felt that the absolute values of the numbers in Tables VII-5a-VII-7 are highly uncertain estimates of crop losses and are a result of a propagation of uncertainties. They are rough estimates, and this should be explicitly stated in this discussion. The Panel believes, however, that these Tables can be of some use in identifying rough relative incremental benefits associated with a given NAAQS as long as it is recognized that small differences in benefits may have no significance because of these uncertainties.

A related issue is the estimated yield losses and seedling biomass losses displayed on the maps in Appendix E of the Staff Paper. Since these are also based on the results of open-top chamber experiments as well as the results of the GIS technology approach, the uncertainties are large. The concern here is that the maps will be used out of context and the caveats ignored. The limitations and uncertainties of the data need to be clearly stated in the legend of each map.

The SUM06 standard reflects a change in thinking over the current 1-hour standard with respect to how plants respond to ambient ozone exposure. This proposed form of the standard implicitly recognizes that vegetation response to ambient ozone is cumulative. However, there is disagreement over whether this is the best form for a cumulative standard and what the level of the standard should be to protect vegetation from damage by ozone. One of the Panel's ecology experts thinks the form and the range of between 25 to 38 ppm-hours proposed by the Agency is appropriate. A second expert thinks the form proposed by the Agency is appropriate and biologically based, but feels that a level of 20 ppm-hours is necessary to adequately protect natural resources. The other two experts are uncomfortable with a SUM06 form because they feel it lacks a biological basis. One member stated that he feels very uncomfortable with SUM06 and would not want to defend it because he feels there is too much

uncertainty associated with its derivation. The fourth expert is concerned that a SUM06 form is unnecessarily complicated, and the level proposed by the Agency would not eliminate ozone damage. Instead, he proposes that the 1-hour average ozone should not exceed 0.05 ppm for more than one hour between the hours of 0700-1500. In his written comments, the Panel's economist noted that the welfare benefits of a secondary standard depend on the decision regarding the primary standard. For example, he points out that if the primary standard remains at 0.12 ppm for 1-hour, or is changed to an 8-hour standard of 0.09 ppm with one allowable exceedence, Table VII-5a suggests potentially significant incremental benefits associated with a secondary standard based on SUM06. He further states that if the primary standard is set at 0.07 or 0.08 ppm with one exceedence, there is little to be gained by establishing a separate secondary standard.

Although the three remaining CASAC members were neither biologists or economists, they offered their opinion on the secondary standard proposals. Two think the form proposed by the Agency is appropriate. One thinks that the level proposed by the Agency is appropriate, while the other feels that the Administrator's discretion should be broader than the range presented in the Staff Paper. One of these members pointed out, however, that the Staff Paper does not make it clear enough that the SUM06 standard as proposed is a practical choice being made as to the level of effects that will be tolerated and not a level that will prevent effects from occurring. The third is uncomfortable with SUM06 and based on the estimates in Tables VII-5a-VII-7, recommends an 8-hour standard at the same level as the new primary standard. The three members also concurred that given the crudeness of the risk assessment estimates, policy decisions cannot be based firmly on science.

A number of the Panelists offered their insights as to why there are such divergent opinions on the recommended form and level of the standard. The main issues are the lack of sufficient rural ozone data, and the lack of relevant plant exposure studies. There are serious deficiencies in terms of the distribution of monitoring sites, particularly in rural areas that prevent us from accurately assessing exposure once ozone damage is observed. The Panel is in agreement that plants are being damaged by ozone and that the current secondary standard is not sufficiently protective, but there remain important limitations to our understanding of the extent of the response of vegetation to ozone under field conditions. Five years from now, if we do not have the results of research coupling ozone air quality and plant biology under conditions more representative of ambient field conditions, to avoid the shortcomings of the open-top chamber experiments, then we will continue to be hampered by our inability to come to consensus on the levels of air quality that are protective of vegetation and ecosystems

at the most reasonable cost. In addition, a number of Panelists expressed the importance of knowing the consequences of decisions concerning National Ambient Air Quality Standards. Once a decision is made to change the standard or to maintain the status quo, we must be able to determine, by appropriate monitoring and research, what the consequences will be in terms of ambient air quality and effects on vegetation and ecosystems.

In summary, a majority of the Panel has come to closure on the secondary part of the ozone Staff Paper despite the desire of the Panel for additional significant revisions. These revisions have been communicated to your staff by this letter and in written comments by individual Panel members. The Panel trusts that your staff will address these concerns.

CASAC would appreciate being kept informed of progress on establishing a revised or new ozone standard, and plans for research on ozone effects. Please do not hesitate to contact me if CASAC can be of further assistance in this matter. We look forward to seeing the final version of the secondary standard portion of the Staff Paper.

Sincerely,

George T. Wolff

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

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16. ABSTRACT

This staff paper evaluates and interprets the updated scientific and technical information that EPA staff believes is most relevant to the review of primary and secondary national ambient air quality standards for ozone. This assessment is intended to bridge the gap between the scientific review in the 1996 criteria document and the judgments required of the Administrator in setting ambient air quality standards for ozone. The major recommendations presented in the staff paper include: (1) Ozone should remain as the surrogate for controlling ambient concentrations of photochemical oxidants; (2) The one-hour primary standard should be replaced by an 8-hour standard; (3) The range of consideration for the level of the primary standard should be 0.07 to 0.09 ppm; (4) Consideration should be given to the current expected exceedance form, ranging from 1- to 5-expected exceedances, averaged over 3 years, as well as to a concentration-based form ranging from the second to the fifth highest 8-hour daily maximum concentration, averaged over 3 years; (5) Consideration should be given to defining the primary standard in terms of a range of air quality values; (6) If the Administrator determines that additional protection is needed beyond that provided by the alternative primary standards recommended, or that no revisions to the primary are warranted, and/or that establishing a seasonal form for the secondary standard is justified, consideration should be given to a new secondary standard in the form of a 3-month, 12-hour, SUM06 exposure index set at a level within the range of approximately 38 to 25 ppm-hours.

17. KEY WORDS AND DOCUMENT ANALYSIS

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