2. EFFECTS OF OZONE

In this chapter we present a summary of the effects of ozone on human health, a description of population exposure to ozone, and a discussion of ozone's effects on crops and forests. Four major health effects issues are presented, along with a discussion of the impact of ozone on the development of respiratory disease, lung function, symptoms, and susceptible populations. In the exposure section, an assessment of the magnitude and frequency of violations of the ozone standard in nonattainment areas is made, and factors influencing ozone exposure in these areas are described as a prelude to estimates of nationwide human exposure to ozone. Finally, in reviewing the effects of ozone on crops and forests, we discuss ozone concentrations that occur in rural areas; crop yield reductions associated with exposure to ozone at these levels; estimates of the agricultural benefits expected to result from reducing ozone; and potential injury to different tree species due to ozone exposure.

2.1 Effects on Human Health[1]

Human exposure to ozone primarily affects the lungs. Ozone has been *shown* to cause immediate, short- term changes in lung function and increased respiratory symptoms, and has been *suspected* of playing a role in the long-term development of chronic lung diseases. The immediate or "acute" effects may include some breathing difficulty and coughing, but such effects appear to be reversible, usually disappearing after a few hours. Ozone has also been suspected of playing a role in initiating asthma attacks.

Although the short-term effects are important, many health professionals appear to be more concerned that repeated exposure to ozone over a lifetime may result in permanent impairment of the lung. Since ozone damages the tissues lining the airways of the lung, ozone exposure could play a role in accelerated aging of the lung, retardation of lung development in children, or the development of pulmonary fibrosis, a chronic lung disease. However, existing data are just beginning to shed light on questions about the possible long-term effects of ozone exposure. We are not yet able to confirm or dismiss many of the concerns about these effects,

[1]The following summary of the health effects of ozone is derived largely from a draft report prepared by Lawrence J. Folinsbee for the Office of Technology Assessment.

Major Issues

The debate over health effects from ozone has centered around four major issues: 1) what are the lowest ozone concentrations at which health effects are observed; 2) what constitutes an "adverse health effect" from ozone exposure; 3) who appears to be most susceptible to ozone's ill effects; and 4) what are the effects of exposure to ozone over a long period of time? All of these issues play an important role in the standard-setting process.[2] Determining the lowest level at which health effects are observed is a crucial first step in this process. Studies conducted both in the laboratory and in the ambient environment generate data that help scientists define the lowest observable effects level. Once this level has been determined, a margin of safety is built into the standard to protect the groups most sensitive to the pollutant. The margin of safety is designed to protect these populations against health effects that research has not yet been identified. Deciding which effects are to be considered "adverse" and determining which populations may be most sensitive to ozone are essential to setting an "adequate" margin of safety. Information about adverse effects help policy makers define an upper bound on this margin; information on sensitive populations assist in defining a lower bound. Studies of the long-term effects of exposure to a pollutant also provide input to the standard-setting process. These four major issues are discussed briefly below.

What are the lowest ozone concentrations at which health effects are observed?

The lowest level at which effects from ozone can be observed has been revised downward during the last 15 years, as more information has become available. In the early 1970's the threshold for responses to oxidants[3] was presumed to be 0.25 parts per million or "ppm." This was based on limited data, however.[4] In 1977, new ozone studies showed lung function effects to exercising persons at concentrations as low as 0.15 ppm.[5] During the last five years or so, the health effects data base for ozone has greatly expanded. Scientists now believe that the duration of exposure to ozone and the intensity of exercise during exposure play the greatest role in determining responses at lower levels of ozone. Some of the most significant acute effects have been observed during prolonged periods of exposure (6.6 hours) to ozone and at heavy exercise levels, at concentrations as low as the current standard level of 0.12 ppm.[6] A number of new human studies show that lung function decrements occur in

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*The air quality standard for ozone is currently under review by EPA.

[3]Photochemical oxidants are a group of chemically-related pollutants. From the standPoint of health and welfare effects, ozone is the most important photochemical oxidant. Ozone typically comprises over 90 percent of the total mass of photochemical oxidants measured in urban air.

[4]Schoettlin and Landau, 1961.

[5]Delucia and Adams, 1977.

[6]Folinsbee et al., 1988.

moderate to heavily exercising children and young adults exposed for 1 to 2 hours to ozone concentrations between 0.12 and 0.16 ppm.[7 8 9 10] The prevalence and significance of effects at levels between 0.08 and 0.12 ppm are less clear, and are currently under investigation.

What is an adverse health effect?

The Clean Air Act directs EPA to set air quality standards for pollutants that may produce "an adverse effect on public health or welfare." A great deal of discussion has been conducted within the scientific and medical community as to what constitutes an "adverse health effect," especially with regard to the effect of ozone inhalation on human lung function at or below the National Ambient Air Quality Standard. While there is general agreement that permanent respiratory injury or episodes of pollutant-induced respiratory illness that interfere with normal activity[11] would be considered "adverse," it is less clear that small changes in lung function indicators or minor increases in the incidence of respiratory symptoms constitute an adverse health effect.

The broad continuum of effects and the diversity of scientific opinion make it difficult to precisely define what is and is not an adverse health effect. The EPA staff recommends that the threshold for an *individual's* adverse respiratory response to acute ozone exposure include *any* of the following responses: (See also Table 2- 1.)

- 10 to **200/0** decrement in FEV_1 in individuals[12] (w/complete recovery after 6 hrs.);

  mild- moderate cough, shortness of breath, pain when inhaling deeply; and

  individual decision to discontinue activity (due to lung function losses and respiratory discomfort).

Most members of the medical community would consider a 10% or greater *group* mean loss in lung function to be sufficient to warrant concern about damage to the lung, especially if one considers that some individuals in these groups are likely to experience greater than average lung function decrements. In addition, lung function losses that may not be harmful

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[7] Linn et al., 1986.
[8] Avol et al., 1987.
[9] McDonnell et al., 1983.
I O Mc Donnell et al., 1985.
[11] Ferris et al., 1985.
[12] FEV_1 -or the volume of air exhaled in the first second of a forced expiration--is one measure of pulmonary funciton that may indicate obstruction in the lungs.

TABLE 2-1. Grades of Individual Response to Acute Ozone Exposure.

Note: EPA staff recommends that the moderate, severe and incapacitating categories should be considered "adverse" respiratory health effects. All effects in each category are associated with each other.

GRADATION OF RESPONSE	MILD	MODERATE	SEVERE	INCAPACITING
CHANGE IN SPIROMETRY FEV_1, FVC	5-10%	10-20%	20-40%	>40%
DURATION OF EFFECT	COMPLETE RECOVERY IN <30 MIN	COMPLETE RECOVERY IN <6 HR	COMPLETE RECOVERY N 24 HOURS	RECOVERY IN >24 HOURS
SYMPTOMS	M D TO MODERATE COU H	MILD TO MODERATE COUGH, PAIN ON DEEP INSPIRATION, SHORTNESS OF BREATH	REPEATED COUGH, MODERATE TO SEVERE PAIN ON DEEP INSPIRATION AND SHORTNESS OF BREATH; BREATHING DISTRESS	SEVERE COUGH, PAIN ON DEEP INSPIRATION, AND SHORTNESS OF BREATH; OBVIOIS DISTRESS
LIMITAT ON OF ACTIVITY	N ONE	FEW ND V DUAL S CHOOSE TO D SCONTINUE ACT V TY	SOME INDIVID JALS CHOOSE TO DISCONTINUE ACTIVITY	MANY INDIVIDUA S CHOOSE TO DISCONTIMIE ACTIVITY

Source: Review of the National Ambient Air Quality Standards for Ozone - Preliminary Assessment of Scientific and Technical Information, Draft Staff Paper. U.S. Environmental Protection Agency. Office of Air Quality Planning and Standards. November 1987. p. VII-45.

for people with normal, healthy lungs may be more significant for individuals with preexisting lung disease. Certainly effects that could be incapacitating and could interfere with normal activity (e.g. asthma attacks) should be considered adverse. However, perceptions of what is a medically significant health effect will vary greatly among physicians and patients.

Are there any subpopulations that are particularly susceptible to ozone's ill effects?

In response to the Clean Air Act's mandate that EPA set air quality standards for pollutants, "allowing an adequate margin of safety ... to protect the public health," the EPA has sought to identify those subpopulations, if any, that are shown to be more sensitive to ozone exposure than the general population.

Two major groups have been identified by EPA as being potentially at increased risk of developing adverse health effects from exposure to ozone:
1) a subgroup of the general population with preexisting disease (e.g., asthma, chronic obstructive pulmonary disease); and 2) those individuals who exercise or work outdoors. [13] The first group is of concern because their already compromised respiratory systems may be at greater risk than individuals without preexisting disease exposed to the same ozone dose. The second group is at risk because by exercising or working in an outdoor environment, they are increasing the dose of ozone to their lungs. To date, neither of these groups *as a whole* has been clearly shown to be more sensitive to ozone than the rest of the population, although some *individuals* with in these groups appear to be more sensitive. The strongest evidence for a population "at-risk" exists for healthy, heavily exercising individuals.

In addition to the above-mentioned groups, studies have shown that there is a subpopulation of otherwise healthy individuals who consistently respond more significantly to the same dose of ozone than do their cohorts. These ozone-sensitive individuals are called "responder s." The EPA estimates that from 5-20°A of the healthy population may represent a subgroup of responders who are at abnormally high risk to ozone exposure .[14] The factors that would account for such individual variability in sensitivity are unknown at this time.

What are the implications of long- term human exposure to ambient ozone levels?

Perhaps the most important health concern with respect to ozone is the *potential* for irreversible damage to the lung from repeated exposure to ozone over a long period of time.

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[13] EPA OAQPS Draft Staff paper, November 1987. While EPA mentions preexisting reSp iratory disease' asa characteristic in the second at-risk group, perhaps the more relevant aspect of this group is that they are exercising, because this will increase the dose of ozone being inhaled into the lungs.

[14] Ibid., p. VI- 13.

This is especially critical when one considers that a significant percentage of the U.S. population is living in areas that may experience recurrent episodes of ozone concentrations at or near the national standard. (For further discussion of population exposure to ozone in nonattainment areas, see Section 2.2, which follows.)

Ozone can cause acute decrements in lung function and increased respiratory symptoms in healthy individuals exercising heavily (e.g. competitive running) at concentrations as low as O.12 ppm. However, while the effects of short-term exposure to this level of ozone appear to be reversible, it is not known if repeated exposure to ozone levels in the range of 0.08 to 0.20 ppm would result in extended or, possibly, permanent changes in lung function or structure. In other words, it is not clear if repetitive exposure to ozone would cause permanent, chronic health effects.

Both animal and human repeated-exposure studies as well as many epidemiological studies have attempted to address concerns about the implications of long-term ("chronic") exposure to Ozone. Together, these studies have yielded preliminary evidence that there may, in fact, be some persistent effects associated with chronic exposure. However, estimates of the risks associated with chronic exposures cannot be made with this limited data base.

The Development of Respiratory Disease

Ozone is suspected of playing a role in the initiation or triggering of respiratory disease processes. The evidence that suggests that such an effect is plausible comes primarily from two types of investigations -- animal toxicology studies and human epidemiology studies -- although human chamber studies may also contribute valuable information.

Animal studies

Animal studies have shown that ozone exposure can cause biochemical and structural changes in the lung. Some of these changes are suspected of playing a role in the development of chronic lung diseases. Studies of animals exposed to relatively high levels of ozone (0.50 ppm) have revealed that it may be responsible for at least temporarily reducing the ability of the lungs to clear foreign material and, therefore, to ward off infection.[15] Several studies have shown an increased response to *bacterial* infection in animals exposed to ozone levels as low as 0.08-0.10 ppm for several hours.[16] [17] Continuous exposure to ozone (at 0.50 ppm) has also been shown to alter the course of *viral* infection in mice by leading to

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15 Foster et al., [1987]"

[16] Miller et al., 1978.

[17] Ehrlich et al., 1977.

structural changes in the lung that increase the likelihood that fibrosis[18] will occur.[19] One type of structural change in the lung which is thought to be linked to the development of lung fibrosis is the deposition of collagen-- a structural protein that contributes to "stiffening" of the lung.[20] [21] Repeated, intermittent exposure of monkeys to Ozone Concentrations as low as 0.25 ppm has been shown to result in increased lung collagen content.[22] Breathing difficulty and subsequent limitation of work performance are characteristic symptoms associated with lung stiffening. Ozone has also been shown to damage certain lung cells in animals at levels as low as 0.25 ppm.[23] However, the long-term health Consequences of this cell damage are not known. While many of these studies offer important insights about the effects of exposure to ozone, the inherent uncertainties in extrapolating from animal data make it difficult to assess risk to humans from these studies.

Epidemiologic studies

Epidemiologic studies have also been used to investigate the potential link between ozone exposure and respiratory disease.[24] One question that has received considerable attention is whether regular exposure to oxidant air pollution causes an increased rate of loss of lung function with age. Part of the normal aging process of the lung involves loss of "usable lung volume," perhaps related to the changes in elasticity of the lung known to occur with aging. (The technical term for this volume is the vital capacity, which is defined as the maximum volume of air that can be expired after taking a full deep breath.) If breathing ozone even at very low levels over a long period of time caused an acceleration of the lung aging process, we would expect to see a more rapid age-related decline in vital capacity in people who reside continuously in oxidant-polluted areas. One epidemiological investigation suggests that an accelerated rate of loss of lung function over a long period (e.g. five years) occurs among residents of high oxidant communities.[25] The evidence is far from conclusive, however, and the question of what impact ozone may have on lung function over a lifetime requires further evaluation before a definitive answer can be reached.

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[18] Pulmonary fibrosis results from the formation of excessive amounts of Protein fibers that stiffen the lung. If this stiffening is severe enough, it can produce debilitating disease.

[19] Jakab, 1988.

[20] Last et al, 1979.

[21] Bhatnagar et al, 1983.

[22] Tyler et al, in press.

[23] Crapo et al, 1984.

[24] Epidemiologic studies involve large groups of people who are exposed to oxidant air pollution (mostly ozone) in their daily life and who may experience a variety of adverse responses from this exposure. The kinds of responses that are examined include changes in lung function over many years, the rate of occurrence of asthma attacks, the rate at which people with pre-existing lung disease are admitted to the hospital, and even the death rate from lung or other diseases.

[25] Detels et al., 1987.

Human chamber studies

Prolonged acute exposure (up to 6.6 hours) of humans in controlled laboratory settings to ozone concentrations similar to those found in many nonattainment cities (0.12-0.18 ppm) have had several effects, including: progressively larger changes in respiratory function and symptoms with time[26] and increased responsiveness of individuals to inhaled substances.[27] The relationship between short-term changes in the lung and the progressive development of chronic structural and functional damage is not known. Some health professionals postulate that the link between acute and chronic effects is the lung inflammation observed in the animal and human subjects of short-term ozone studies. Before this inflammatory response disappears, some suggest that it may induce other changes in the lung that might persist over time. Airway inflammation is also a feature of the development of a number of respiratory diseases, most notably asthma and chronic bronchitis.

Issues of susceptibility and adaptation from prolonged exposure

Both animal studies and clinical chamber studies of humans have been used to investigate the effect of repeated exposure to ozone over an extended period of time (over several months in animal studies, over several days in human chamber studies). The importance of such studies is that they help us understand the longer-term effects of ozone on the lung -- i.e., whether or not prolonged exposure to ozone makes individuals more susceptible to subsequent exposure to ozone and other pollutants and whether or not lung function effects are reversible once exposure to ozone ends. Chamber studies of humans show two notable responses to repeated ozone exposure: 1) when an individual is exposed to ozone on two consecutive occasions separated by less than 48 hours, the second exposure generally causes greater lung function effects than the first one[28] 29 and 2) with continued exposure, these effects begin to diminish in intensity and after four or five days the pulmonary function effects are undetectable.[30] [31] [32] This gradual loss of functional response has been called “adaptation.”

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26 Folinsbee et al., 1988.
27 McDonnell et al., 1987.
28 Folinsbee and Horvath, 1986.
29 Bedi et al., 1986.
30 Horvath et al., 1981“
31 Kulle et al., 1982„
32 Linn et al., 1982.

The adaptive responses of individuals who live in areas with high ozone levels might be different from the responses of subjects exposed to ozone for only a few consecutive days in a laboratory setting. Recent preliminary evidence indicates that people who live in Los Angeles may become less sensitive to ozone during the entire “smog season” but regain their sensitivity during the relatively smog-free winter season.[33] In this study, "adaptation" did not disappear rapidly, as in the chamber exposures, but appeared to persist for at least 2-3 months after the end of the smog season. Although this suggests that processes other than those observed in a chamber may be involved in long-term adaptation to ozone exposure, further evaluation is needed before a definitive answer can be reached.

Though measurable lung function changes and symptom responses may lessen for a period, other changes within the lungs are ongoing. In other words, the process of lung injury and repair is a continuous one.[34] Individuals who, through adaptation, experience fewer or less severe symptoms, may be at increased risk since they may be more able to tolerate exercise outdoors during peak ozone episodes, and, hence, receive potentially greater tissue damage over the long-term. Research on animals shows that some lung injury may continue during an “adaptive” period (e.g. effects on host defense system[35] and increased susceptibility to disease[36]), even though other measures of response may be reduced.

Lung Function Effects

Ozone has well-documented short-term, reversible effects on lung function. In studies of people exposed to ozone, the most commonly measured lung function effects are changes in “forced expiatory volume” (FEV)[37] and “forced vital capacity” (FVC). Ozone can cause decreases in both of these measures of lung function.

Changes in lung function depend upon the dose of ozone that is ultimately delivered to the lung. A number of factors influence dose, including the concentration of ozone in the air, duration of exposure, and the average volume of air breathed per minute, referred to as the ventilation rate. The ventilation rate increases with exercise intensity. Figure 2-1 describes the dose-response relationship between ozone and FEV_1. As this diagram shows, an increase in exercise intensity at any given ozone concentration results in a decrease in group mean FEV_1.

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[33] Hackney and Linn, 1987.

[34] EPA, “Air Quality Criteria for Ozone and Other Photochemical Oxidants,” August 1986.

[35] Gardner et al., 1972.

[36] Gardner and Graham, 1977.

[37] More commonly, FEV1, or the volume of air which can be expired in One second, will be measured.

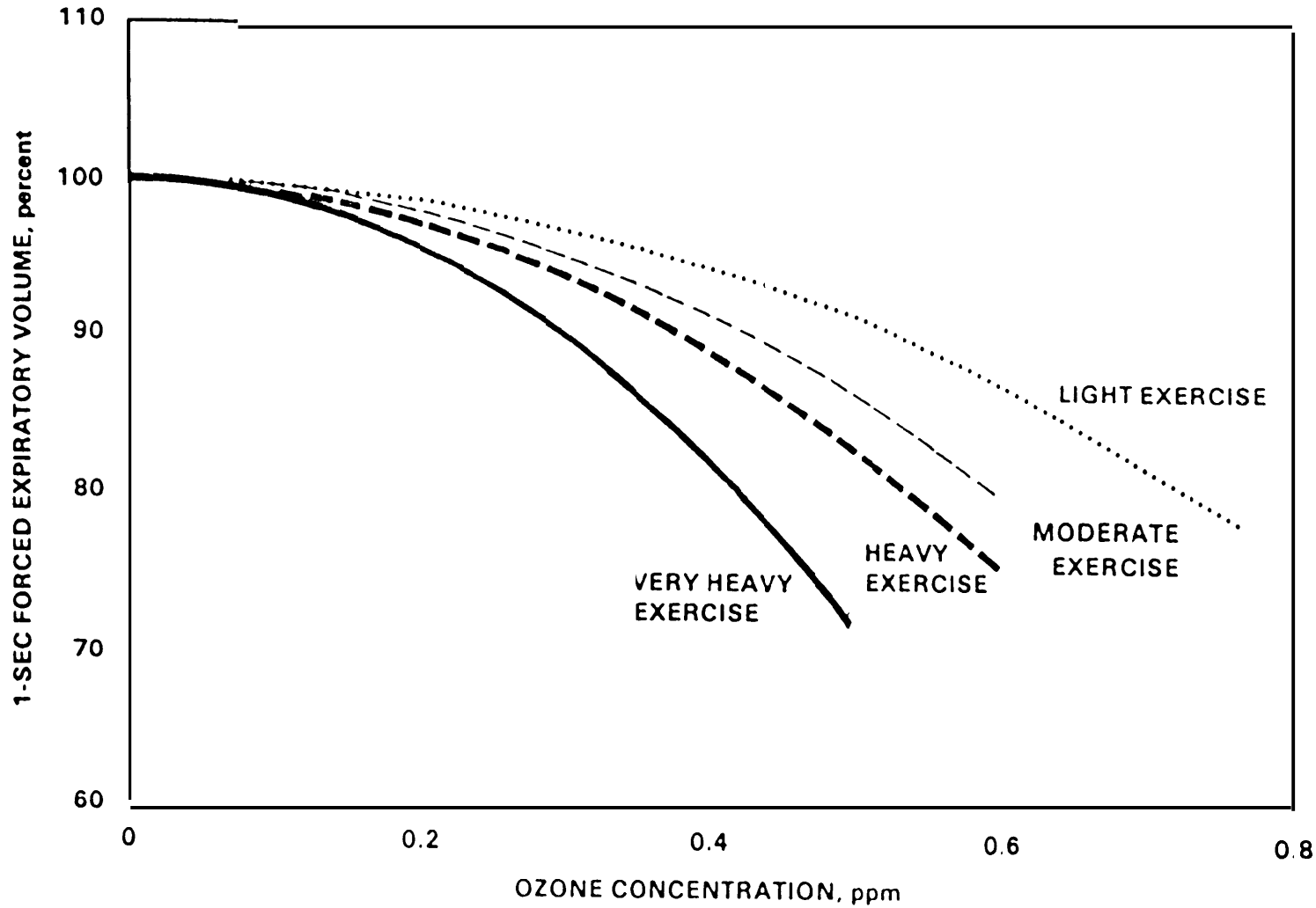

Figure 2-1. Percent decreases in lung function during 2-hr ozone exposures with different levels of exercise.

Source: Air Quality Criteria for Ozone and Other Photochemical Oxidants, Vol. I of V, U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office, August 1988, p. 1-158.

Prior to 1980, there was very little information on lung function changes from controlled exposures to ozone concentrations below 0.30 ppm. This was mainly because under the conditions of rest or mild exercise employed in most of these studies, there was little, if any effect from 1-2 hour exposures to ozone levels less than 0.30 ppm. However, a number of studies, using higher exercise levels, have since shown clear responses to ozone levels between 0.16-0.24 ppm.[38] 394041 Average decreases in group mean FEV_1 ranged from 6-22%.

At ozone concentrations approaching the current ambient air quality standard for ozone, some investigators have seen small (4-6%) but statistically significant group mean decreases in FVC and FEV_1 under conditions of heavy exercise,[42] 43 while others have not.[44] 45 46 Because of the variability in observed changes in lung function among different studies, it is difficult to draw any definite conclusions about changes in lung function in the range of 0.08 to 0.16 ppm ozone for one- or two-hour exposure periods. The most substantial responses in this range of ozone concentration occur under conditions of heavy exercise and durations of exposure longer than one hour. For example, Folinsbee and coworkers recently observed 13% group mean decreases in FEV_1 in subjects performing heavy exercise for 6.6 hours at the level of the standard, 0.12 ppm.[47]

The current controversy surrounding impairment of lung function from ozone exposure involves the definition of an "adverse" decrement in lung function. Group mean decreases in either FEV_1 or FVC of greater than 10 percent are clearly significant enough to be considered adverse, especially in light of the fact that some individuals within these groups experience decrements in lung function greater than the average. Temporary and infrequently occurring changes of less than 10 percent, in and of themselves, probably do not represent an adverse health effect for a healthy young adult. However, some health professionals would consider such changes to be adverse if they restrict activity or limit performance [48], Short-term reversible decrements in lung function could have adverse effects in individuals with already reduced lung capacity. However, there is no universal agreement

38 McDonnell et al., 1983.

39 Folinsbee et al., 1984.

40 Avol et al., 1984,

41 Gong et al., 1986.

42 McDonnell, 1983.

43 Gong, 1986.

44 Schelegle and Adams, 1986.

45 Kulle et al., 1985.

46 Linn et al., 1986.

47 Folinsbee et al., 1988.

48 Ferris et al., 1985.

among scientists as to the implications of such "small" changes. There is also little convincing evidence available at this time to indicate whether there are long-term consequences from short-term lung function changes.

Symptom Responses

Symptoms experienced by people exposed to ozone are also important markers of ozone's effects. The major symptoms -- cough and pain when breathing deeply -- typically are observed at about the same ozone exposure levels as are changes in lung function indices; heavy exercise for one hour at 0.18 ppm will cause such symptoms in *groups* of healthy young adults.[49] [50] [51] Folinsbee and coworkers' recent study (1988) demonstrated a relationship at 0.12 ppm between discomfort on deep breathing and changes in lung function (FVC) using *individual* **data.** However, most studies have not shown an association between symptoms and lung function changes at this ozone concentration on an individual level. Pronounced symptoms such as repeated coughing or pain when taking a deep breath will almost always be associated with substantial (greater than 10%) lung function changes.

Adults perceive symptoms of ozone exposure at low concentrations (0.12 ppm)[52] but children apparently do not.[53] [54] [55] While children are certainly capable of sensing breathing discomfort, their lack of response from these low level exposures could be the result of a higher "threshold" of perception for symptoms. It has been suggested that the weak symptom responses of children may put them at greater risk from ozone exposure because they may not make efforts to avoid being exposed if they are unable to perceive the effects. Further research is needed on the sensitivity of children to the symptoms of *ozone* exposure.

Potentially Susceptible Members of the Population

Implicit in the Clean Air Act's directive that EPA set air quality standards with an "adequate margin of safety" is the desire to protect the most sensitive groups in the population. Many factors may affect susceptibility to ozone exposure, including age, sex, smoking status, nutritional status, environmental stresses, and exercise level during exposure. These six factors help EPA identify groups likely to be at increased risk to ozone. At present, scientists postulate that about 5 to 20 percent of the healthy population may

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49 McDonnell et al., 1983.
50 Avol et al., 1984.
51 Kulle et al., 1985.
52 McDonnell et al., 1983.
53 McDonnell et al., 1985.
54 Avol et al., 1985.
55 Avol et al., 1987.

represent a subgroup of "responders"[56] who may be significantly more responsive than the general population to the same dose of ozone. Also considered "at-risk" are asthmatics, people with pre-existing lung disease, children, the elderly, and individuals who exercise heavily or work outdoors. Within each of these groups some individuals have demonstrated greater-than-average sensitivity to a specified dose of ozone, although no particular group has proven to be more sensitive than the others.

The strongest evidence for increased responsiveness exists for groups who exercise intensively outdoors because the dose of ozone they receive is much higher than average due to their increased breathing rate. Because individuals with preexisting lung disease already have compromised respiratory systems, there is concern that lung function changes and other respiratory effects may be more serious for these people than for the normal, healthy population.

Asthmatics

Results of studies on asthmatics are mixed. **A** number of epidemiological studies of asthmatics have suggested that ozone exposure may be associated with increased asthma attacks, hospital admissions for asthma, decrements in lung function, and symptoms.[57] [58] [59] [60] Asthmatics have also participated in studies in which lung function and symptoms were assessed before and after breathing ozone in a controlled laboratory environment. These studies have consistently shown that the lung function and symptom responses of asthmatics to a specific level of ozone do not differ from the responses of healthy non-asthmatics.[61] [62] [63]

Because of what we know about the significant difference in response to sulfur dioxide between asthmatics and non-asthmatics, the failure of asthmatics to exhibit increased sensitivity to ozone in chamber studies is somewhat surprising. However, these have been group analyses; there may be a subpopulation of asthmatics more sensitive than a subgroup of "normals" to ozone inhalation. For example, moderate to severe asthmatics have not been studied in these controlled environments. In addition, chamber studies of asthmatics have

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[56] p. VI- 13, "Review of the National Ambient Air Quality Standards for Ozone," OAQPS Draft Staff Paper, November 1987.
[57] Whittemore and Kern, 1980.
[58] Bates and Sitzo, 1987.
59 Holguin et al., 1985
[60] Gong et al., 1987.
[61] Koenig et al., 1987.
[62] Linn et al., 1978.
[63] Linn et d., 1980.

not yet been conducted at the higher exercise levels that have yielded the most significant responses in non-asthmatics. The discrepancy between results in epidemiologic and chamber studies may also be due to interaction between ozone and other environmental factors (i.e., other pollutants, high temperatures and humidity) in the field. In other words, there may be factors operating in the ambient environment that have not been replicated in clinical studies.

Chronic obstructive pulmonary disease (COPD) patients

Patients with chronic obstructive pulmonary disease (COPD) (chronic bronchitis, emphysema), many of whom are former smokers, are also of concern as an "at-risk" subgroup because they already have poor lung function and, compared to healthy individuals, relatively small decrements in lung function could be adverse for them. Several different laboratory studies have been conducted on COPD patients exposed to ozone[64 65 66 67 68] but none have found them to experience significant reductions in lung function measures (FVC, FEV_1) even at concentrations as high as 0.30 ppm for 1-2 hours. It will be necessary to study these individuals over longer periods of exposure and at higher exercise levels in order to adequately evaluate the risk from ozone exposure faced by COPD patients. Out of concern for their health, studies of patients with COPD, like those asthma, have not been performed under such conditions to date.

Children

Children are another *potenlially* susceptible subgroup of concern. Since the lung continues to develop until adulthood, the critical question regarding children exposed to ozone is whether repeated exposure will influence lung maturation. Relatively low concentrations of ozone (at or around the standard) do appear to have an adverse impact on the lung function of active children.[69 70] On the basis of both controlled exposure studies and field studies of ambient pollutant exposure, however, children do not appear to have lung function effects that are much different than those experienced by adults.

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64 Kulle et al., 1984.
65 Linn et al., 1982.
66 Linn et al., 1983.
67 Solic et al., 1982.
68 Kehrl et al., 1985.
69 McDonnell et al., 1985.
70 Lippmann et al., 1983.

The elderly

Concern has also been expressed for elderly members of the population. A subgroup of healthy, older adults may be at risk because they may participate in outdoor activities where they might be exposed to ozone. There is not yet a consensus, however, as to whether or not this group is at higher or lower risk for pulmonary function and other ozone-related effects than younger adults. While lung function effects have been observed in this subpopulation, several studies suggest that healthy older adults may less susceptible to the acute effects of ozone than healthy young adults.[71 72] The extent to which pulmonary function changes reflect other events occurring in the lung of ozone-exposed older adults is unknown; further research is necessary to fully evaluate this group.

Athletes

Both epidemiologic and chamber studies have indicated that athletes may be at substantial risk of experiencing decreases in work performance and decrements in lung function when exercising for approximately one hour at ozone concentrations as low as 0.20 ppm. 737475 Outdoor workers exposed to ozone for prolonged periods may also be at increased risk. New research shows that volunteers performing the equivalent of a day of very heavy manual labor while exposed to 0.12 ppm ozone experience significant loss in lung function (13°/0 group mean decrease in FEV_1) and pronounced symptoms (e.g. cough, pain when inhaling deeply).[76] This research suggests that extended periods of heavy exercise may be undesirable from the point of view of respiratory health and physical performance, not only during periods of high ozone concentrations (greater than 0.20 ppm), but also at levels found in many nonattainment cities (0.12-0.18 ppm).

The acute effects of ozone exposure (e.g. decreases in lung function and symptomatic responses) are summarized in Figure 2-2, which illustrates the ozone level at which these effects begin. The figure is divided into two sections: the upper section describes effects that occur with -1-3 hour exposures, the lower section for 4-8 hour exposures. The tail of the arrow indicates the concentration at which an effect may begin. At the lowest concentrations at which effects are seen, the exposures are typically accompanied by very

71 Drechsler-Parks, 1987.

72 Reisenauer et al., 1988.

73 Folinsbee et al., 1984S

74 Gong et al., 1986.

75 Schlegle and Adams, 1986.

76 Folinsbee et al., 1988.

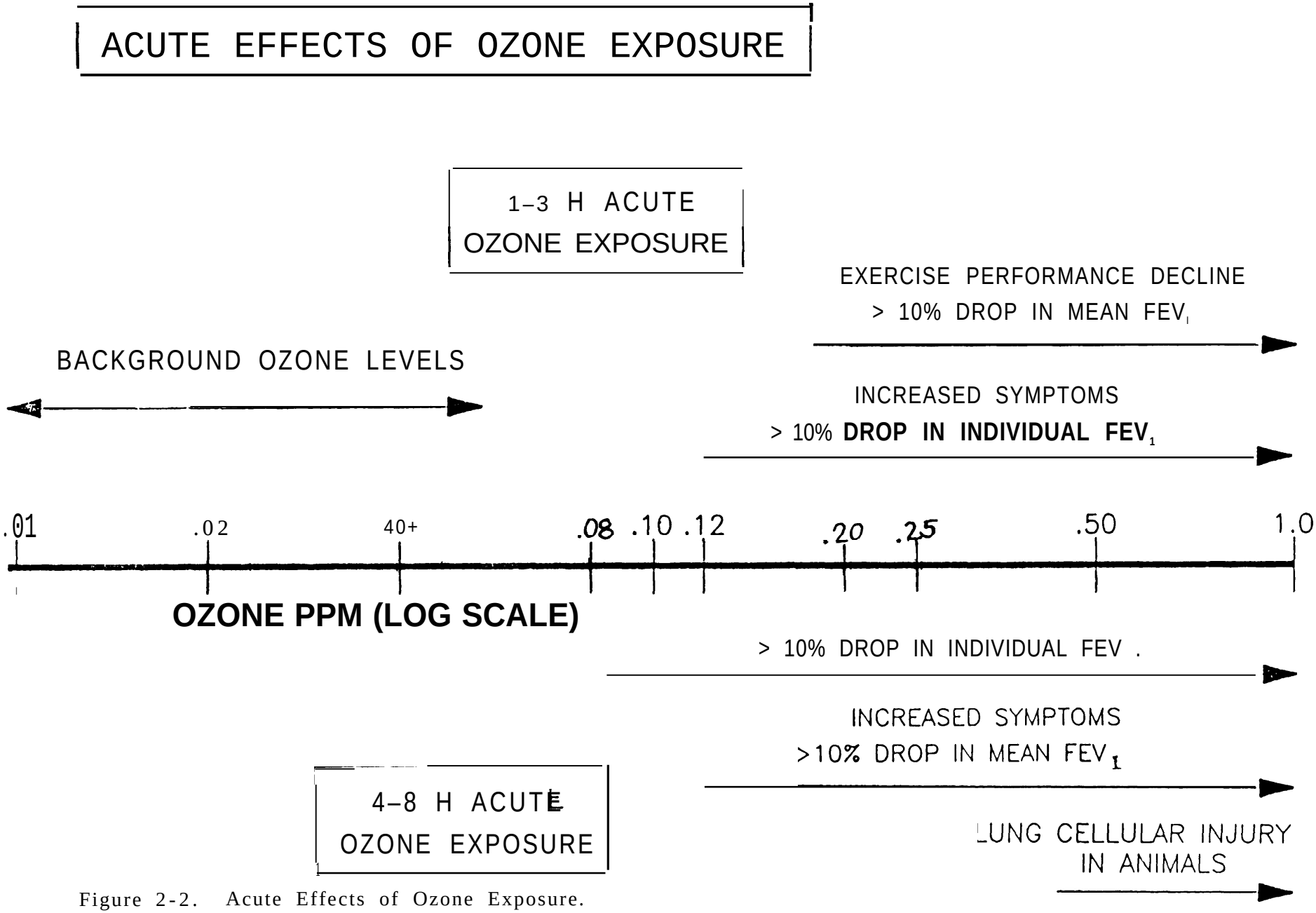

Figure 2-2. Acute Effects of Ozone Exposure.

Note: All effects above ozone scale line are associated with *1-3* h acute (short-term) exposure; all effects below line are associated with **4-8** h acute exposure. Also, FEV_1 is one measure of p ulmonary function that may indicate airway obstruction in the lungs,

Source: Draft Report for OTA by Lawrence J, Folinsbee, "A Summary of the Health Effects of Ozone," Jan. 1988.

heavy exercise. With moderate or mild exercise, effects would begin at higher ozone concentrations. Also, more adverse responses, such as cell damage shown in laboratory animal studies, tend to occur at the higher concentrations.

2.2 Exposure to Ozone

Areas Failing to Meet the Standard

An area is designated "nonattainment " for ozone if concentrations exceeding 0.12 ppm (1 -hour aver-age) are measured on more than three days over a three year period at any monitoring site in the area (i.e. the area is expected to exceed the standard more than once per year, averaged over three years).

Figure 2-3 shows the metropolitan statistical areas (MSAs) and grouped or "consolidated" metropolitan statistical areas (CMSAs) that were classified as ozone nonattainment areas based on 1983-1985 monitoring data. The areas are listed in Table 2-2. As indicated in the table, several non-MSA areas were also designated nonattainment but are not show n o n the map.[77]

EPA updates the list of nonattainment areas every year as data for a new season become available. Based on the 1983-1985 data, 76 urban areas (encompassing 94 individual MSAs plus the ten non- MSA areas) were designated nonattainment. In contrast, 62 areas were designated nonattainment based o n t h e 1984-1986 period (16 areas were dropped in 1986 and 2 areas were added), The difference is primarily attributable to differences in weather between the two periods. The nonattainment list from the 1983-1985 period has been used here for consistency with other parts of this assessment, and because the list for the most recent three-year time period -- 1985-1987, is not yet available. The list of nonattainment areas for 1985-1987 is expected to match the 1983-1985 list more closely than it matches the 1984-1986 list, because the relatively hot summers of 1983 and 1987 both saw higher numbers of violations of the ozone standard than the intervening summers d id,

The shading in Figure 2-3 indicates the 1983-1985 "design value" for each area. The design value is a measure of the highest daily maximum 1 -hour average ozone concentrations in the area and is the fourth highest of all of the daily peak 1-hour average ozone concentrations observed with in the area over the most recent three year period. Areas with design values of 0.13 ppm or higher are violating the ozone standard. On average. the higher the design value, the greater the level of emissions control required to prevent violations of

[77] The non-MSA areas are Dover, DE; Seaford, DE; Iberville Parish, LA; Pointe Coupee Pot-is h, LA; St. James Parish, LA; Acadia National Park, ME; Gardiner County, ME; Hancock County, M E; York County, ME; and Northampton County, VA.

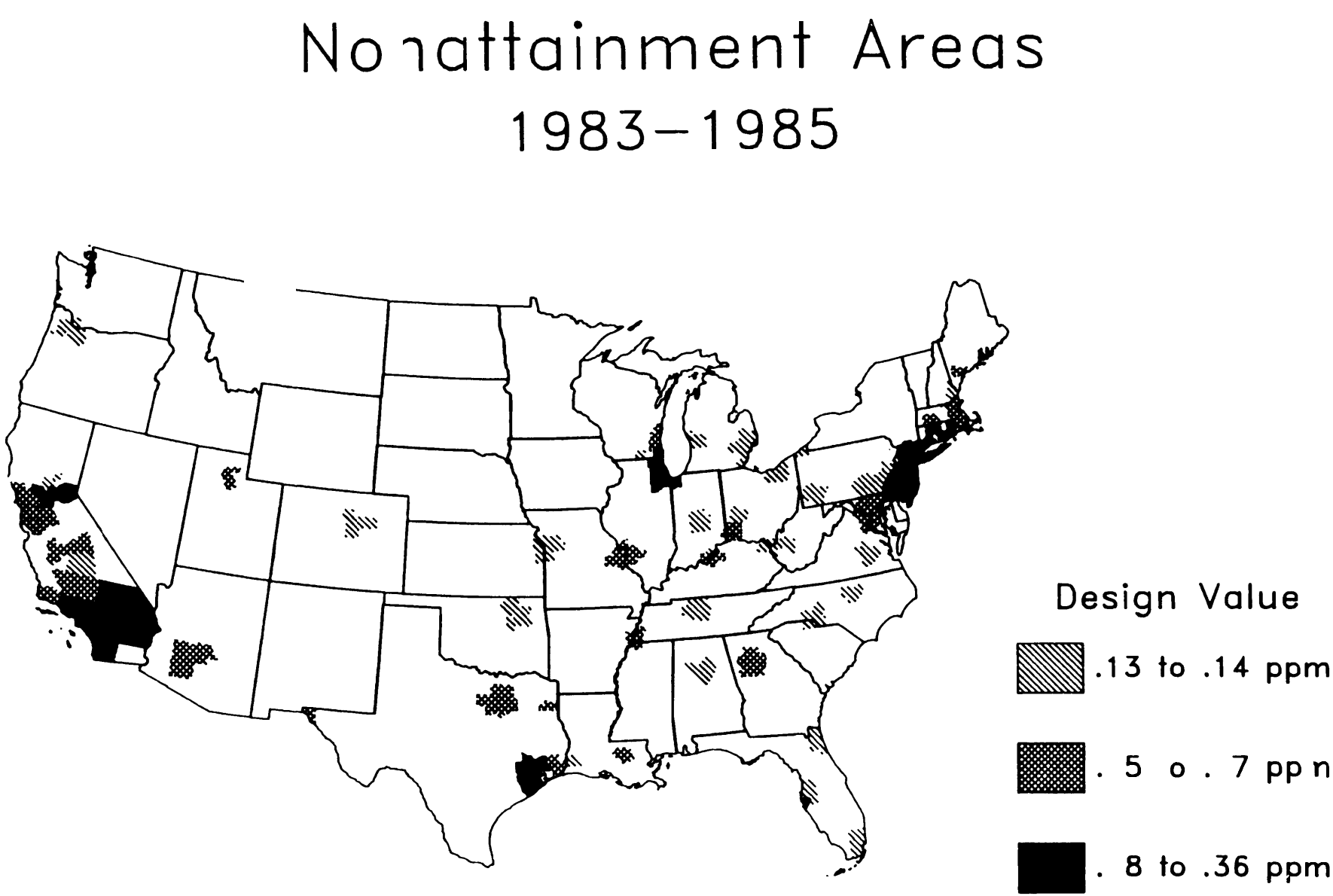

Figure 2-3. Areas classified as nonattainment for ozone based on 1983-1985 data. The areas shown are listed in Table 2-2. The shading indicates the fourth highest daily maximum 1-hour ozone concentration or "desigr value" for each area.

Table 2-2. Areas classified as nonattainment for ozone based on 1983-1985 data. * -- non-MSA area. ** -- multi-MSA consolidated area.

Area Name	Design Value (ppm)
0.13 to 0.14 ppm	
Acadia National Park, ME*	0.13
Allentown-Bethlehem, PA	0.14
Birmingham, AL**	0.13
Charleston, WV	0.13
Charlotte-Gastonia- Rock Hill, NC-SC	0.13
Cleveland, OH**	0.14
Dayton-Springfield, OH	0.13
Denver-Boulder, CO**	0.13
Detroit, MI**	0.13
Dover, DE*	0.14
Erie, PA	0.13
Gardiner, ME*	0.14
Grand Rapids, MI	0.13
Hancock Co., ME*	0.13
Harrisburg-Lebanon-Carlisle, PA	0.13
Huntington-Ashland, WV-KY-OH	0.14
Iberville Parish, LA*	0.13
Indianapolis, IN	0.13
Jacksonville, FL	0.14
Janesville-Beloit, WI	0.13
Kansas City, MO-KS	0.14
Lake Charles, LA	0.14
Lancaster, PA	0.13
Miami- Hialeah, FL**	0.13
Muskegon, MI	0.14
Nashville, TN	0.14
Northampton Co, VA*	0.14
Pittsburgh, PA**	0.13
Pointe Coupee Parish, LA*	0.13
Portland, OR-WA**	0.13
Portsmouth- Dover-Rochester, NH-ME	0.13
Reading, PA	0.13
Richmond-Petersburg, VA	0.13
St James Parish, LA*	0.13
Tampa-St Petersburg-Clearwater, FL**	0.13
Tulsa, OK	0.13
Visalia-Tulare- Porterville, CA	0.13
York, PA	0.13
Yuba City., CA	0.13

Table 2-2. (Cont.) Areas classified as nonattainment for ozone based on 1983-1985 data. * -- non-MSA area. ** -- multi-MSA consolidated area.

Area Name	Design Value (ppm)
0.15 to 0.17 ppm	
Atlanta, GA	0.16
Bakersfield, CA	0.16
Baltimore, MD	0.17
Baton Rouge, LA	0.16
Beaumont-Port Arthur, TX	0.16
Boston, MA**	0.16
Cincinnati, OH-KY-IN**	0.17
Dallas-Ft Worth, TX**	0.16
El Paso, TX	0.16
Fresno, CA	0.17
Longview-Marshall, TX	0.15
Louisville, KY-IN	0.15
Memphis, TN-AR-MS	0.15
Milwaukee, WI**	0.17
Modesto, CA	0.15
New Bedford, MA	0.16
Phoenix, AZ	0.16
Portland, ME	0.16
Salt Lake City-Ogden, UT	0.15
Santa Barbara-Santa Maria-Lompoc, CA	0.16
Seaford, DE*	0.15
St Louis, MO-IL**	0.16
Stockton, CA	0.15
Washington, DC-MD-VA	0.16
Worcester, MA	0.15
York Co, ME*	0.15
San Francisco, CA**	0.17
0.18 to 0.26 ppm	
Atlantic City, NJ	0.19
Chicago, IL**	0.20
Greater Connecticut**	0.23
Houston, TX**	0.25
New York, NY**	0.22
Philadelphia, PA-NJ**	0.18
Providence, RI**	0.18
Sacramento, CA	0.18
San Diego, CA	0.21
0.27 ppm or higher	
Los Angeles-Long Beach, CA**	0.36

the standard. For the 1983-1985 period, 39 areas had design values of 0.13 or 0.14 ppm, 27 areas had design values of 0.15 to 0.17 ppm, and 10 areas had design values of 0.18 ppm or more. The highest design value for any area was 0.36 ppm, for Los Angeles, CA.

Frequency and Magnitude of Violations

Figures 2-4 through 2-6 show the areas throughout the contiguous United States where ozone concentrations exceeded 0.12 ppm, 0.14 ppm and 0.18 ppm, respectively, at least one hour per year, averaged over the years from 1983 to 1985. By averaging data from all of the monitors in each area, the maps indicate the number of hours each concentration level was typically exceeded.[78] The data shown were obtained from EPA.[79] The all-monitor average statistics are assumed to be more representative of air quality throughout each area than data for the peak monitor (the monitor where the highest concentrations were recorded) would be. Note that more areas would be expected to exceed the specified concentrations if data for the peak monitor in each area were used.

Of the 317 (urban and nonurban) areas for which we have ozone data, Figure 2-4 shows the 130 areas where a concentration of 0.12 ppm was exceeded at least one hour per year, on average, between 1983 and 1985.[80] Sixty of those areas had concentrations equal to or greater than O. 12 ppm six or more hours per year. The Dallas, Houston and Atlanta areas and parts of California, New York, New Jersey and Connecticut all recorded concentrations greater than or equal to 0.12 ppm more than 20 hours per year. The maximum number of hours that monitored ozone concentrations exceeded 0.12 ppm in any one area was 275 hours per year.

Figure 2-5 shows the 60 areas where the all-monitor average statistics indicate that ozone concentrations reached 0.14 ppm at least one hour per year between 1983 and 1985. Twenty-four of these areas recorded ozone concentrations of at least 0.14 ppm six or more hours per year. Seven areas, namely the Houston area and parts of Connecticut and southern California, recorded concentrations of 0.14 ppm or higher more than 20 hours per year.

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[78] The number of monitors in each area ranges from one to 18 (in Los Angeles). The average number of monitors in each area is three.

[79] SAROAD, 1987.

[80] If data for the peak monitor in each area had been used instead of the all monitor average statistics, 146 areas would be indicated as having ozone concentrations greater than or equal to 0.12 ppm at least one hour per year.

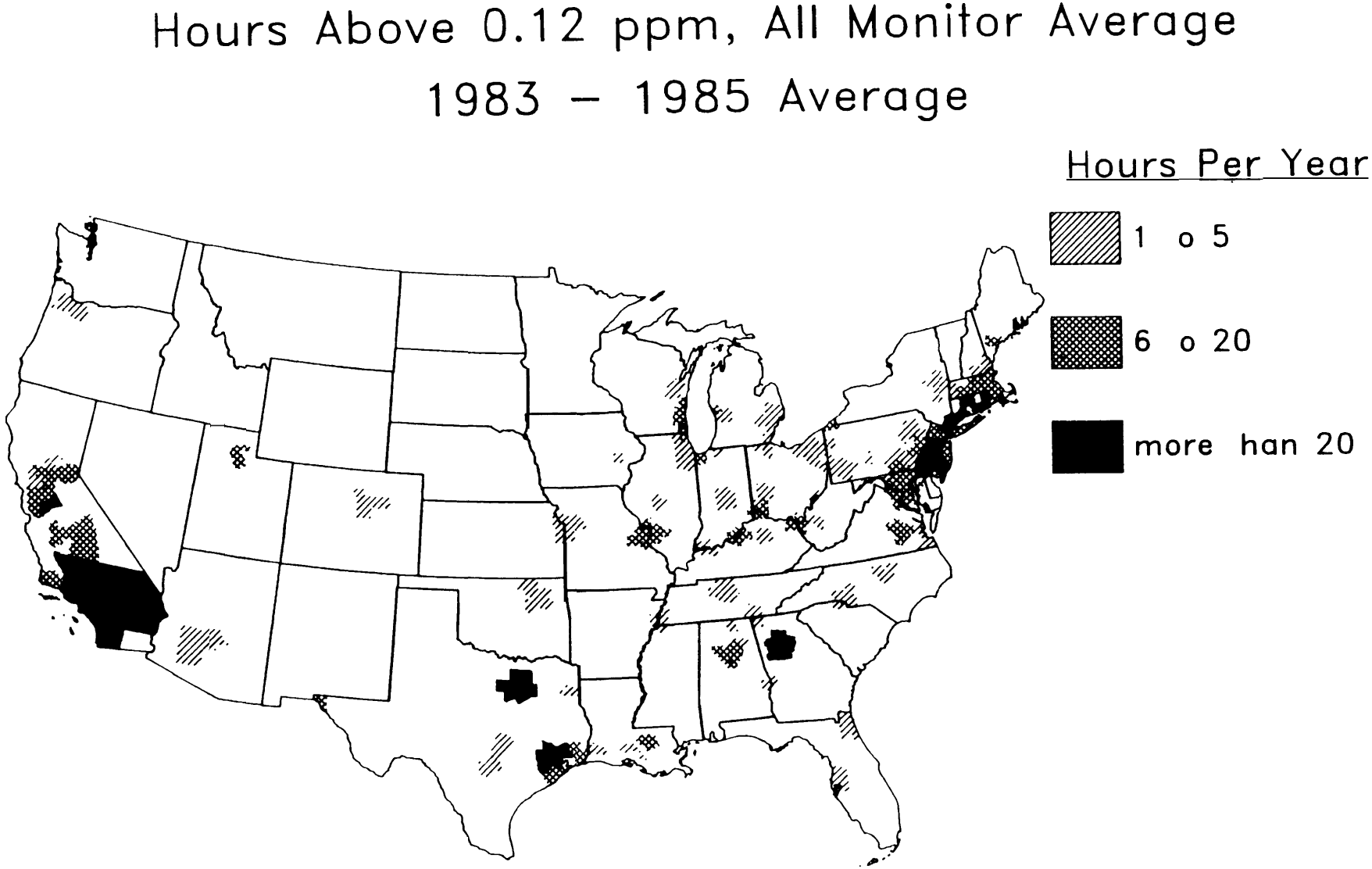

Figure 2-4. Areas where ozone concentrations exceeded 0.12 ppm at least one hour per year, on average from 1983 through 1985. Data from all of the monitors in each area were averaged in constructing the map. The shading indicates the number of hours that a concentration of 0.12 ppm was exceeded. One hundred thirty million people reside in the areas shown.

Figure 2-5. Areas where ozone concentrations exceeded 0.14 ppm at least one hour per year, on average from 1983 through 1985. Data from all of the monitors in each area were averaged in constructing the map. The shading indicates the number of hours that a concentration of 0.14 ppm was exceeded. Eighty six million people reside in the areas shown.

Figure 2-6. Areas where ozone concentrations exceeded 0.18 ppm at least one hour per year, on average from 1983 through 1985. Data from all of the monitors in each area were averaged in constructing the map. The shading indicates the number of hours that a concentration of 0.18 ppm was exceeded. Twenty five million people reside in the areas shown.

Figure 2-6 shows the eighteen areas where concentrations were as high as 0.18 ppm for one or more hours per year between 1983 and 1985. The all-monitor average statistics indicate that concentrations exceeded 0.18 ppm six or more hours per year in Houston and in two areas in Connecticut. Concentrations reached 0.18 ppm more than 20 hours per year in three areas in southern California.

Factors Influencing Exposure to Ozone

Just because an individual lives in an area where ozone concentrations of 0.14 ppm (for example) have been measured does not mean that he or she has been exposed to ozone concentrations at that level, or that if exposed, he or she would experience adverse health effects. This section discusses some of the factors that determine what a specified measured ozone concentration means for human health. The factors that need to be kept in mind include:

1) How outdoor ozone concentrations vary over time and location within a city;

2) Where people are and for how long -- especially how much time they spend outdoors versus indoors, where concentrations are lower;

3) People's activity levels -- which determine their breathing rate and the depth of the breaths they take, and thus the amount of ozone they inhale over a given period of time; and

4) Person-to-person variability in how sensitive people are to ozone.

At urban locations, ozone concentrations usually peak during the early to mid-afternoon, after building up throughout the morning. At suburban and rural locations, the peak concentrations usually occur later in the afternoon or early evening. Figure 2-7 shows a profile of ozone concentrations as they change over the day at a single monitoring site.[81] The profile is typical of a suburban area downwind of the center of a major city. Especially at suburban and rural locations, ozone concentrations often stay within 10 to 20 percent of the peak one-hour average concentration for several hours.

The first step in relating measured ozone concentrations to potential health effects is to estimate from the monitor readings the pollutant concentrations to which people have actually been exposed. Figure 2-8 shows a contour map of how peak ozone concentrations on a given day vary across the New York City metropolitan area.[82] The diagram shows

*lAd$_{ap}$t$_{e}$d from U.S. EPA, 1986.

[82]Adapted from Rae, 1987.

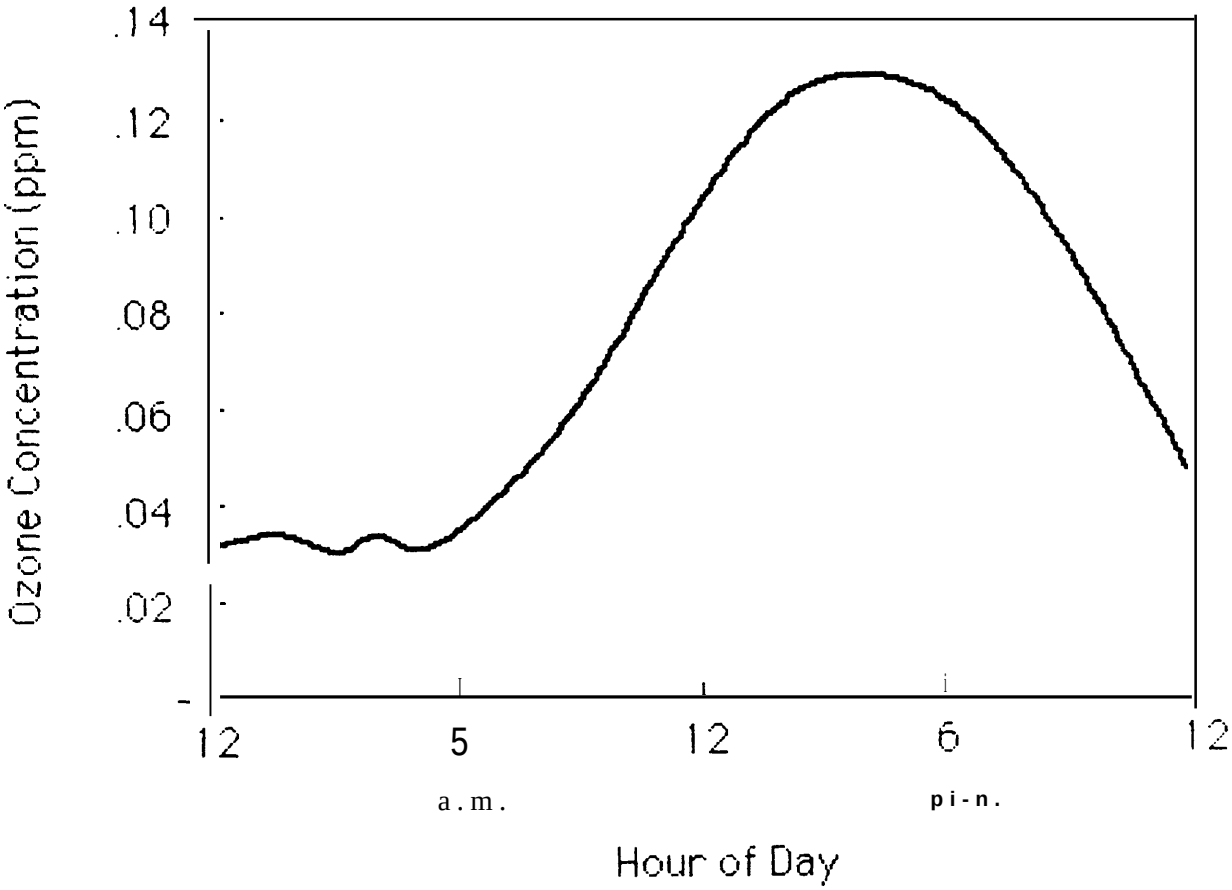

Figure 2-7. Profile of ozone concentrations as they change over the day at a single monitoring site [adapted from U.S. EPA, 1986]. The profile is typical of a suburban area downwind of a strong source area or city center.

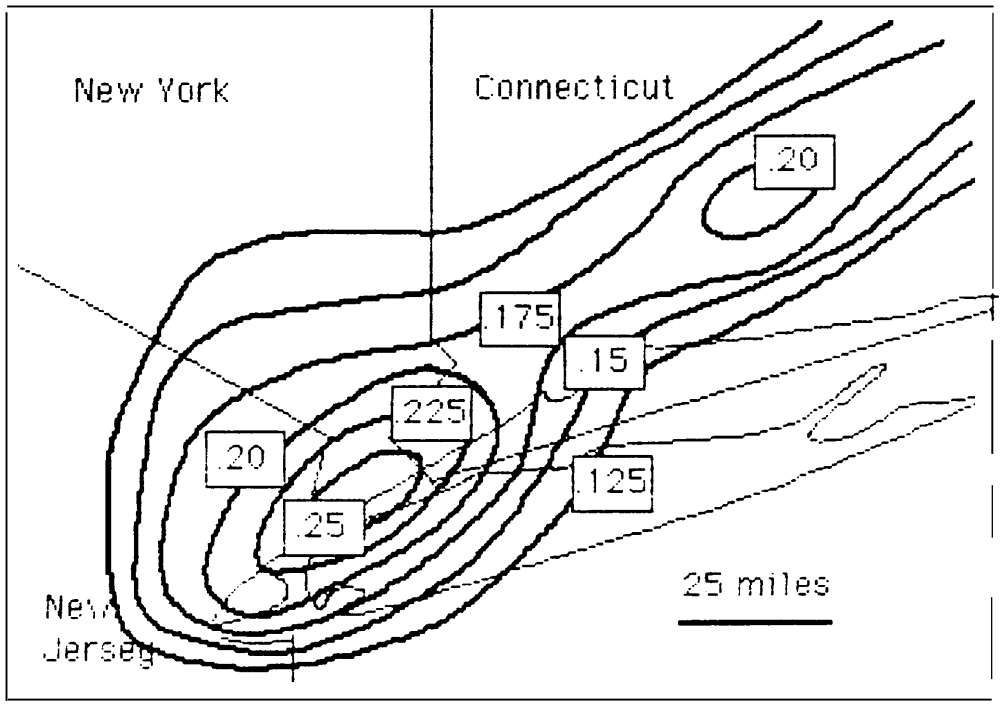

Figure 2-8. Contour map of the variation in daily peak ozone concentrations (ppm) predicted for the New York City area *using a model with meteorological* conditions and emissions of July 16, 1980 [adapted from Rae, 1987]. As shown, ozone concentrations typically vary smoothly over a large area and do not show localized peaks.

ozone concentrations predicted using a model, with meteorological conditions and emissions of July 16, 1980 as inputs. As shown in the example, at any one time, outdoor ozone concentrations can vary by a factor of two or more across an urban area. However, as shown in Figure 2-8, ozone concentrations tend to vary smoothly over large areas, and not to show sharp, localized peaks.[83]

People who are outdoors during the afternoon when ozone concentrations reach their peak are apt to be exposed to higher ozone concentrations than people who are indoors. In air conditioned buildings, indoor ozone concentrations are typically about 30 percent of those measured outdoors at the same location.[84] Ozone concentrations inside buildings with open windows instead of air conditioning are estimated to be about 60 percent of outdoor concentrations.[85] Most people spend 80 to 90 percent of their time indoors. Note, however, that some people work or recreate outdoors most of the day. About 5 percent of adult men work mostly outdoors. An additional 10 percent work outside part of the time. The proportion of women who work outside is thought to be somewhat lower.[86]

Two factors determine the total amount of ozone an individual inhales over a given period of time: (1) the ozone concentrations to which the person is exposed; and (2) the depth and rate at which the individual is breathing. The depth and rate at which someone breathes is determined by the level of exercise he or she is performing. Since the amount of air and thus the amount of ozone inhaled increases with increasing physical exertion, people who are exercising or doing vigorous labor outdoors are more likely to experience health effects due to elevated ozone concentrations than people who are sitting, standing or walking at a leisurely pace. As examples, recreational jogging, swimming and bicycling can constitute heavy exercise. Those who compete in these sports are likely to be attaining very heavy exercise levels.[87]

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[83] One exception t. this general rule is that in the plumes of large NO_x sources, up to about a mile downwind of the source, ozone concentrations can be much lower than in the surrounding air. This is because extremely high concentrations of NO_x without comparably high VOC concentrations destroy ozone faster than it is produced. However, as the NO_x plume disperses, VOC and NO_x levels come into balance and net ozone production results.

[84] Paul et al., 1986.

[85] Ibid.

[86] Pope, 1986.

[87] A 1984 Gallup survey indicated that about 18 percent of adult Americans jog at least once per week [Gallup, 1984]. Four out of every 1000 adults (0.4 percent) run more than six miles at least once per week [Gallup, 1983].

As discussed in the section on health effects, clinical and epidemiological studies have shown that different people respond differently to ozone even when they are exposed to the same concentrations over the same time period and are breathing at the same rate. From five to twenty percent of the population of healthy adults are thought to be very sensitive to ozone. The reasons for their heightened sensitivity have not been established.

population Exposure Estimates

Based on 1984 census estimates[88] and the data presented in Figures 2-4 to 2-6, approximately 130 million people live in areas where ozone concentrations are expected to equal or exceed O. 12 ppm at least one hour per year. Eighty-six million people live in areas where concentrations reach at least O. 14 ppm at least one hour per year; 25 million where concentrations reach at least 0.18 ppm; and 10 million live in the Los Angeles and Anaheim, CA MSAs where ozone concentrations reach or exceed 0.25 ppm.

Of the 130 million people who live in areas where ozone concentrations reach or exceed 0.12 ppm, 43 percent (56 million) live in areas where concentrations reach 0.12 ppm six or more hours per year; 34 percent (44 million) in areas where concentrations reach 0.12 ppm at least 20 hours per year, and almost ten percent (12 million) in areas (Los Angeles, Riverside and Anaheim, CA) where ozone concentrations reach 0.12 ppm more than 100 hours each year. As with the maps presented above, it is important to note that the preceding estimates are based on the average of all of the monitors in each area, not the "peak" monitor.

The population statistics presented above might be considered the number of people "potentially" exposed to ozone -- people who, if they were outside at the "right" time and location, would be exposed to ozone concentrations above the level at which the current ozone standard is set. Table 2-3 presents estimates of actual exposures: the number of people who do happen to be in the right place at the right time to be exposed to concentrations above 0.12 ppm for at least an hour; and for each person who is exposed, the average number of times each year that exposures occur. The numbers given in Table 2-3 were calculated by combining EPA's exposure estimates[89] with the number of people we have estimated who live in areas where ozone concentrations are expected to exceed 0.12 ppm more than one hour per year.

The numbers given in Table 2-3 are broken down by the exercise levels at which the exposures were estimated to have occurred. Recall that people exercising at higher levels are expected to be more susceptible to health impacts. Nationwide, 34 million people are

88 Department of Commerce, 1986.
89 McCurdy, 1988.

Table 2-3. Estimated exposures to ozone concentrations above 0.12 ppm [adapted from McCurdy, 1988]. The estimates are based on hourly ozone data for the period 1983-1985, and take into account people's activity patterns (e.g. time commuting, time indoors at work, etc.) location throughout the day. The estimates are broken down according to people's exercise levels, as those exercising at the higher levels are most apt to be susceptible to health impacts. The total number of people residing in areas where the ozone standard was exceeded at least one hour per year, on average during 1983-1985, was approximately 130 million.

Exercise level	People Exposed	Percent of People in Areas Exceeding 0.12 ppm	Hours of Exposure Per Person Exposed
Nationwide			
sedentary	34 million	26 %	8.8 hours
low	21 million	27 %	8.6 hours
moderate	13 million	27 %	5.7 hours
heavy	80 thousand	23 %	4.1 hours
Nationwide except Los Angeles			
sedentary	24 million	20 %	3.7 hours
low	16 million	23 %	4.6 hours
moderate	10 million	23 %	3.2 hours
heavy	60 thousand	19 %	2.1 hours
Los Angeles			
sedentary	9.7 million	97 %	22 hours
low	4,6 million	77 %	24 hours
moderate	3.0 million	83 %	14 hours
heavy	20 thousand	73 %	10 hours

estimated to be exposed each year at sedentary exercise levels; 21 million at low exercise levels; 13 million at moderate exercise levels; and approximately 80 thousand during heavy exercise. In each exercise category, these numbers represent about 25 percent of the people who achieve that exercise level some time during the year. Since everyone is sedentary at some time (e.g. when they are sitting and talking), about 25 percent of the people who live in areas where ozone concentrations exceed 0.12 ppm are estimated to be exposed to concentrations at or above this level. By far the most people are exposed at sedentary or low exercise levels. Fewer people are exposed at the highest exercise level, because few people engage in heavy exercise. Of the nationwide totals, 9.7 million, 4.6 million, 3 million and 20 thousand of the people exposed at sedentary, low, moderate and high levels, respectively, are residents of the Los Angeles area.

On a nationwide basis, people who are exposed to ozone concentrations of 0.12 ppm at low exercise levels are estimated to be exposed an average of about 9 hours per year; people exposed at moderate levels an average of 5.7 hours per year; and people exposed at high exercise levels an average of 4.1 hours per year. However, the national averages mask considerable variability amongst urban areas. In particular, the national figures are skewed by the high incidence of exposures in the Los Angeles area. In Los Angeles, the average numbers of hours people are exposed at sedentary, low, moderate and high exercise levels are estimated to be 22, 24, 14 and 10 hours per year per person exposed, respectively. For the rest of the country, with the Los Angeles estimates subtracted out, the estimated numbers of hours of exposure are, respectively, 3.7, 4.6, 3.2 and 2.1 hours per year for people exposed at sedentary, low, moderate and high exercise levels.

2.3 Effects of Ozone on Crops and Forests

At concentrations that occur in rural areas throughout the southern and eastern halves of the United States, ozone reduces yields of economically important crops by from one to 20 percent, compared to yields that would be expected if ozone concentrations did not exceed natural background levels.[90] Annual agricultural benefits on the order of $2 billion per year [1985 $] would be expected to result from increased crop productivity if ozone concentrations in rural areas were reduced by 25 percent from current levels.[91] [92]

Forest damage (visible foliar injury, reduced growth rates, death of individual trees and succession of dominant species) in Southern California has been clearly linked to exposure to elevated ozone concentrations. Ozone has been shown to produce foliar injury

[90] Heck et al., 1984.
[91] Kopp et a]., 1984.
[92] Kopp et al., 1984.

and/or reduce growth rates in young trees of numerous species in controlled experiments. Ozone is suspected as being partially responsible (along with other pollutants and natural stresses) for forest declines observed in parts of the eastern United States and southern Canada. Over the last 20 to 25 years, in a variety of locations, significant fractions of the trees in stands of several species have exhibited foliar injury or decreased growth rates or both. In several cases, the location and timing of the declines suggest that air pollutants have contributed. The forest-related benefits of reducing ozone concentrations cannot currently be estimated.

This section reviews the effects of ozone on crops, indicates where elevated ozone concentrations correspond to agricultural production, and briefly discusses estimates of the agricultural benefits of reducing ozone concentrations. The section then reviews what is understood about the effects of ozone on trees and forest ecosystems, shows the location of major forested areas, and then discusses five cases in which ozone has been suggested as a cause of forest decline.

Concentrations of Ozone in Rural Areas

Fewer than 100 ozone monitors are located in agricultural areas across the United States.[93] A number of States do not have any monitors. Thus for much of the country, only rough estimates of ozone concentrations in agricultural and forested areas can be made.

For rural monitors, Figure 2-9 shows daily maximum 7-hour average ozone concentrations averaged over the 1984 growing season.[94,95] The concentrations range from 0.038 to 0.065 ppm. For comparison, the natural background value of the seasonal average daily maximum 7-hour average statistic is estimated to be between 0.025 and 0.030 ppm. The highest concentrations are seen at sites in Connecticut, New Jersey, Georgia, Texas and California. A general trend of increasing seasonal-average concentrations from north to south is expected due to the fact that sunlight intensity increases as one moves south. Note that the concentrations shown are from rural, but not necessarily remote monitoring sites, and may be affected by pollution from urban areas within a few hours upwind.

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[93] OAQPS, 1987.

94 NAPAP, 1987.

[95] Recent studies have suggested that for many crops, cumulative exposure to ozone concentrations above thresholds of 0.08 to 0.10 ppm is a somewhat better measure of exposure than the seven-hour seasonal average ozone concentration. However, the seven-hour seasonal average concentration is more widely reported.

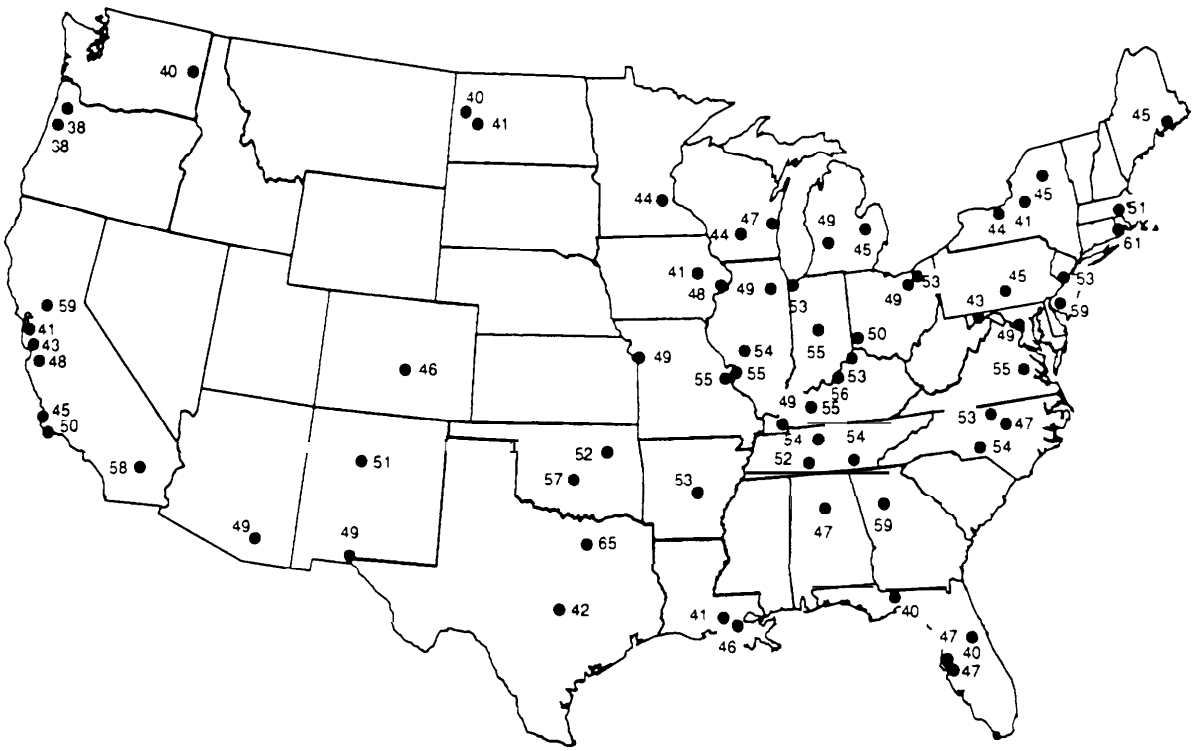

Figure 2-9. Daily maximum 7 -hour average ozone concentrations (ppb) measured at rural monitoring sites and averaged over the 1984 growing season [NAPAP, 1987].

Effects of Ozone on Crops

Visible symptoms of injury due to ozone include light flecks, dark stipples and yellow spots or patches on plant leaves. Chronic exposures can induce premature "senescence" or maturation and loss of leaves. The minimum concentrations of ozone that produce acute foliar injury in susceptible plants exposed for four hours range from 0.04 ppm to 0.09 ppm, depending on the plant species.[96] Among other environmental factors, light conditions, temperature, relative humidity and soil water content affect how plants respond to ozone exposures.

For field and cash crops, the most important responses to ozone are reduced growth rates and yields. These effects may occur without the visible signs of injury usually associated with exposure to ozone. However, early senescence of leaves is usually found. Growth and yield reductions result primarily from reduced photosynthesis and transport of carbohydrates within plants. Table 2-4 displays the yield reductions predicted to occur for various crops exposed to seasonal average seven-hour mean ozone concentrations of 0.04 and 0.06 ppm.[97] The yield-reduction predictions are from the National Crop Loss Assessment Program (NCLAN), an eight-year study in which crops were grown in the field either in air filtered to assumed background ozone concentrations, ambient air, or air to which extra ozone had been added. The reductions shown in the table are relative to the yields obtained for crops exposed to assumed background ozone concentrations. The range of yield reductions indicated for each crop indicates differences among varieties.

Figure 2-10 shows state-level production of each of the four crops listed in Table 2-4. Figure 2-9 showed that seasonal average seven-hour mean concentrations of 0.04 ppm were widely exceeded in 1984 and that concentrations higher than 0.06 ppm were measured at a few locations. Note that due to year to year variability in weather, concentrations would be higher at some sites and lower at others, if data for a year other than 1984 were shown. Elevated ozone concentrations throughout the south impact cotton. The major soybean producing regions of the Mississippi and Ohio River valleys and corn producing regions throughout the eastern half of the United States and Texas are also impacted. High concentrations affect wheat production in most areas where it is grown, except in the northern plains states. In addition to the major crops listed in Table 2-4, yield reductions have been seen with a wide variety of other crops including alfalfa, clover, sorghum, barley, dry bean, root crops, tomatoes, spinach, lettuce and other produce.

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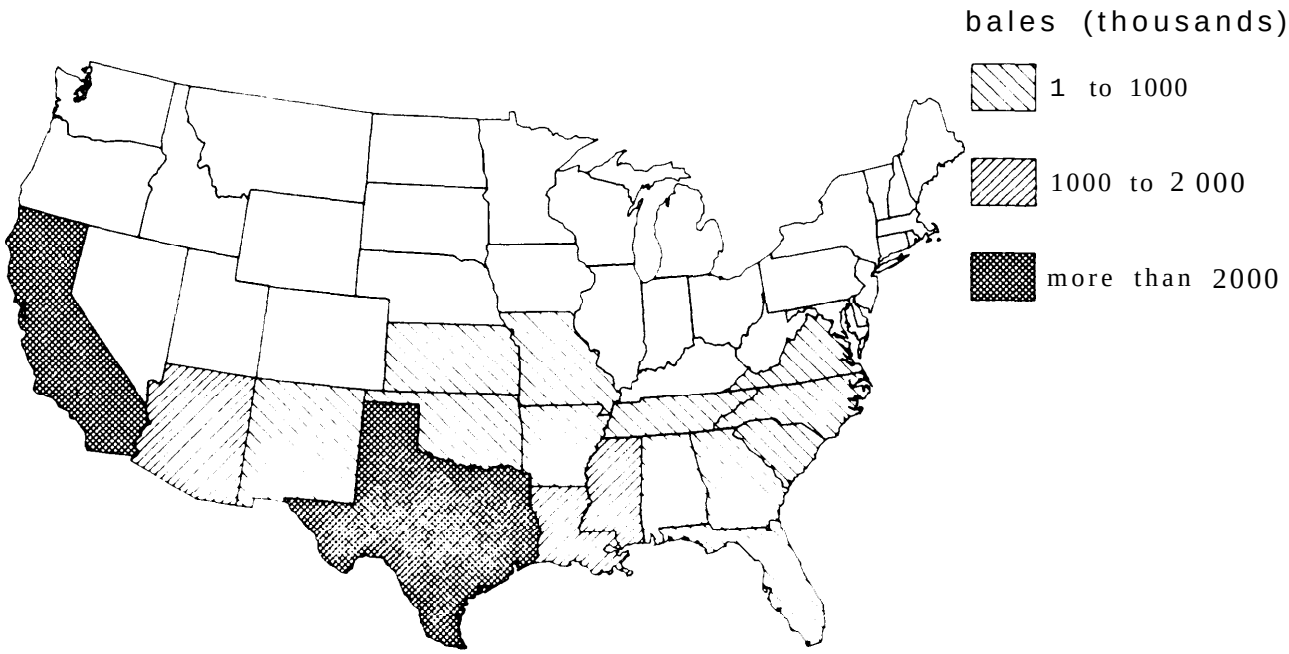
[96] Jacobson, 1977, as cited in U.S. EPA, 1987.

[97] EPA, 1987.

Table 2-4. Yield losses predicted to occur for seasonal average seven-hour mean ozone concentrations of 0.04 and 0.06 ppm [EPA, 1987]. The 0.04 ppm level is exceeded throughout the southern and eastern halves of the United States. The 0.06 ppm level is exceeded in parts of the northeast, California, Texas and Georgia. Natural background seasonal average seven-hour mean ozone concentrations are thought to be about 0.025 to 0.03 ppm.

	0.04 ppm ozone percent yield reduction	0.06 ppm ozone percent yield reduction
cotton	4.6 to 16	16 to 35
wheat	0.0 to 29	0.9 to 51
soybeans	1.7 to 15	5.3 to 24
corn	0.0 to 1.4	0.3 to 5.1



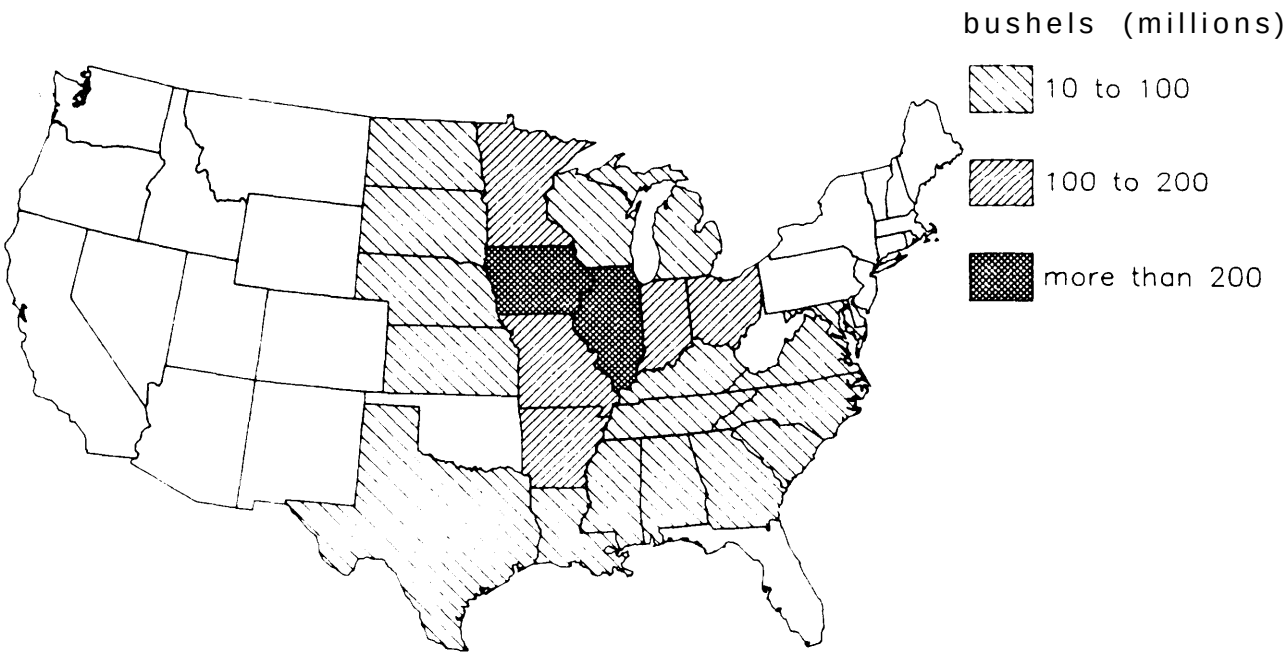
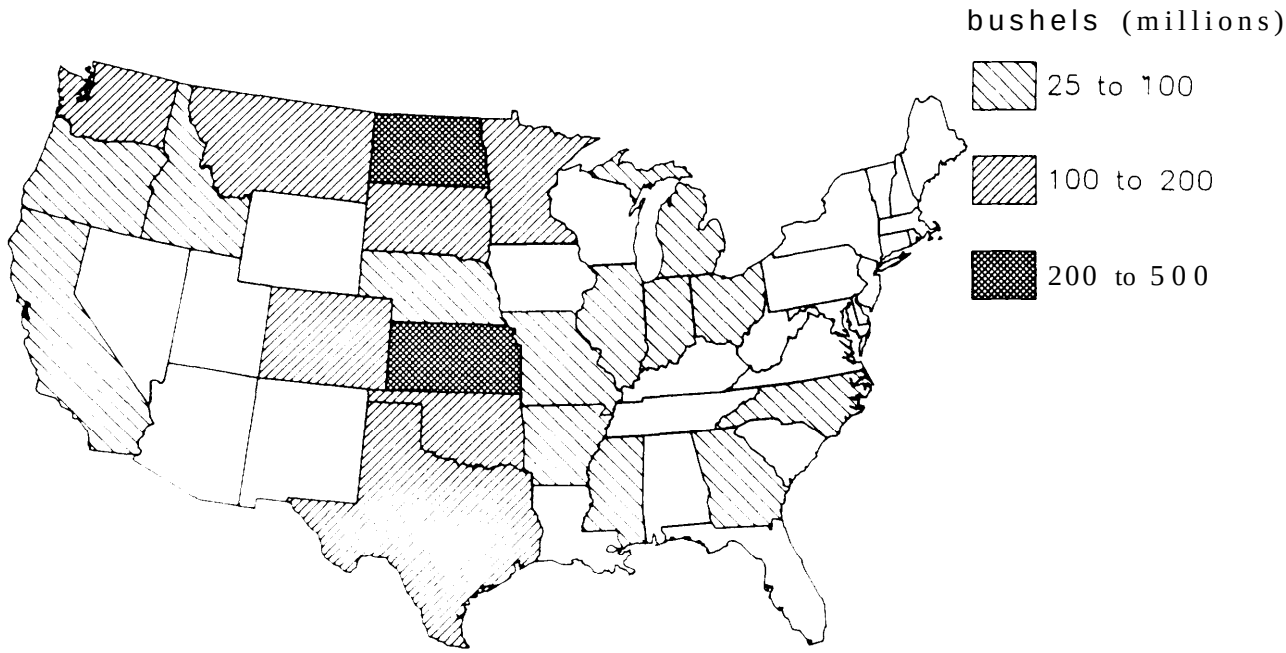


Figure 2-10. 1984 state-level (a) cotton and (b) soybean production [USDA, Agricultural Statistics, 1985].

1984 Wheat Production

USDA, Agricultural Statistics, 1985



1984 Corn Production

USDA, Agricultural Statistics, 1985

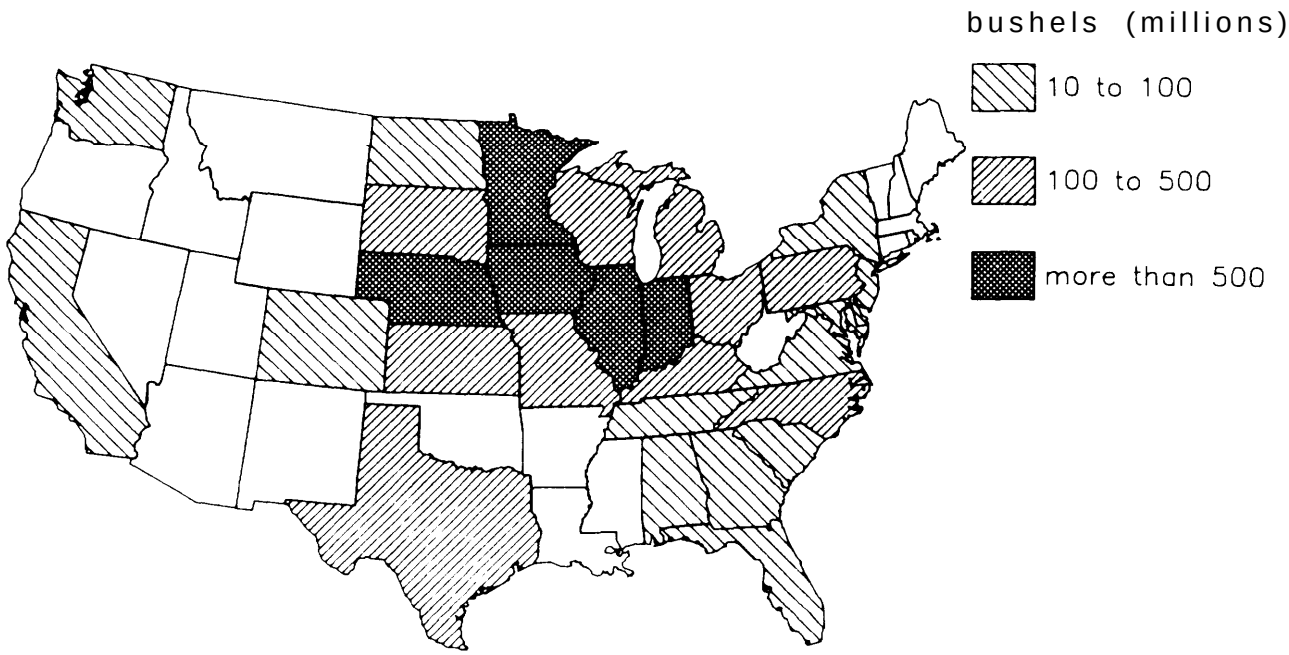


Figure 2-10. 1984 state - level (c) Wheat and (d) corn production [USDA, Agricultural Statistics, 1985].

Based on NCLAN's predicted yield responses, economic models of crop supply and demand have been developed to estimate the agricultural benefits of reducing ozone concentrations.[98 99] Reductions in ozone concentrations alter the supply of crops by increasing yields. Prices are determined by market forces as well as whatever agricultural price support policies are in place. The models use baseline ozone concentrations that are extrapolated to rural areas from both suburban and rural monitors (which generally show similar seasonal average values). While major uncertainties exist in these models, several investigators have used different models and still been fairly consistent in predicting that total annual benefits on the order of $2 billion per year [1985 $] would accrue to consumers and farmers if ozone concentrations in rural areas were reduced by 25 percent.[100] Note, however, that the benefits estimates depend heavily on assumptions about agricultural policies, base year and background ozone concentrations, and the experimental relationships between crop yields and ozone concentrations.

Potential Effects of Ozone on Forests

Ozone-induced injury in trees shows up primarily as foliar injury, including leaf or needle discoloration and premature loss. In extreme cases, leaves and then branches of injured trees die back. Ultimately individual trees can die prematurely. Effects that may not be apparent to the eye include reduced growth rates and increased susceptibility to diseases and other stresses. Reduced photosynthesis and decreased allocation of carbohydrates to tree roots are possible reasons for the increased susceptibility. Controlled experiments suggest that growth rates may be reduced by ozone even though the characteristic visible signs of ozone damage are not present. Weakening of species and premature death of individual trees can have broad ecological impacts, as species which are more resistant to ozone take over. All of these effects, including a transition in dominant species, have been observed in the San Bernardino mountains east of Los Angeles, and attributed to exposure to ozone originating from emissions in the Los Angeles basin.

Many of the effects of exposure to ozone also occur due to numerous other causes. In most cases, it is likely that multiple stresses contribute to observed declines, making it difficult to sort out primary causes or the effect of eliminating or mitigating a single stress. Effects observed in studies that have been conducted in controlled environments in order to isolate the effects of ozone do not always match those observed in natural environments. Moreover, controlled studies have been performed almost exclusively on seedlings or saplings,

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98 **Adams et al.**, 1984.

[99]Kopp et al., 1984.

[100]U.S. EPA, 1987.

rather than mature trees. So, while exposure to ozone has been suggested as an explanation for several cases of forest or individual species decline in the United States, Canada and Europe, in most of these cases no consensus exists on the role of ozone.

Figure 2-11 shows the major forested areas of the United States, and identifies the types of trees that dominate in each area. Comparing Figure 2-11 with Figure 2-9 indicates that elevated ozone concentrations are present in the western conifer region of California, and the eastern hardwood and southeastern yellow pine regions. An additional consideration is that high-elevation forests are likely to be exposed to higher long-term average concentrations than nearby low-elevation forests, due to the tendency for elevated ozone concentrations to be maintained at high altitudes overnight and into the morning, while low - elevation surface concentrations are depleted at night.

Ozone has been suggested as a factor in several confirmed and reported cases of forest or species decline in the United States.[101] Ozone has been implicated as a cause of decline in the first two cases discussed below. It has been suggested as a contributing factor in the other cases.

Ponderosa and Jeffrey pine in the San Bernardino National Forest and other locations in southern California

Ozone is generally held to be a principal cause of visible injury and accelerated mortality of ponderosa and Jeffrey pine and other species in the San Bernardino and San Gabriel Mountains of southern California. The symptoms of injury observed there have been duplicated in controlled exposure studies. At some sites in the San Bernardino National Forest east of Los Angeles, daytime (14 hour) average ozone concentrations of 0.10 ppm are typical during June, July and August.[102] The decline of ponderosa and Jeffrey pine in the national forest has been so severe that if current trends persist, incense cedar and white fir are expected to replace them as the dominant species in the forest.[103] Growth reductions in association with visible foliar injury have also been observed in Jeffrey pine at Sequoia and Kings Canyon National Parks in California.[104]

White pine in the eastern United States

Controlled exposure studies and field studies support the hypothesis that concentrations of ozone widely observed in the eastern United States injure white pine trees,

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[101] NAPAP, 1987.
[102] Ibid.
[103] McBride et al., as cited in NAPAP, 1987.
[104] Peterson et al., 1987.

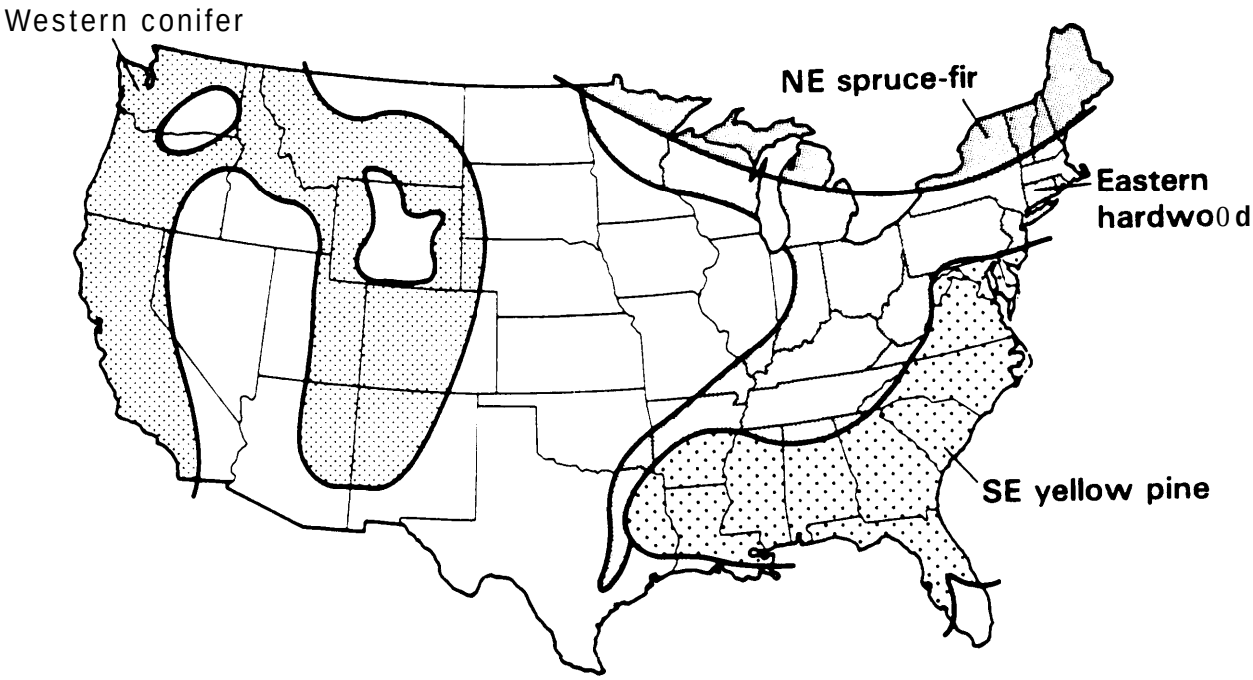

Figure 2-11. **Major** forested areas and dominant tree-types of the United States [NAPAP, 1987].

although as with other tree species, not all white pines are equally sensitive.[105] Foliar injury, reduced growth rates and increased mortality are apparent in trees throughout the eastern United States.

Red spruce at high elevation sites in the eastern United States

Since the mid 1960's, the number of live red spruce in some high elevation forests in the northeast has decreased by 40 to 70 percent.[106] Decreased radial growth, dieback, and increased mortality have been observed at high elevation sites in the Appalachians from Vermont and New Hampshire to North Carolina, with the highest mortality rates in the northeast.[107] Regionwide trends of colder winters and increasing pollutant levels since about 1960 have both been suggested as explanations.[108] At above-cloud-base sites in remote or rural parts of the eastern United States, nighttime and early morning ozone concentrations are significantly higher than concentrations measured at adjacent sites at lower elevations. The frequent presence of clouds enhances ozone uptake.[109] Scientists suggest that since conditions at high elevations are marginal for red spruce to begin with, the added stresses of colder winter temperatures and/or increased air pollution could readily push high elevation forests into decline.[110]

Yellow pine **in the southeastern United States**

Average growth rates in natural stands of yellow pine have been reduced by up to **50** percent over rates observed in the late 1950s.[111] The causes of the widespread growth reductions are unknown, but may include the natural aging of the stands, increased competition from hardwoods, drought, and exposure to air pollution. Preliminary results indicate that controlled exposure to ozone has similar effects on loblolly pine as have been observed with other species, including reduced photosynthesis and reduced growth.[112] However, the role of ozone in the yellow pine case has not been firmly established.

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[105] Woodman and Cowling, 1987.

[106] NAS, 1986.

[107] NAPAP, 1987.

[108] NAS, 1986.

[109] NAPAP, 1987.

[110] NAS, 1986.

[111] Sheffield et al., 1985, as cited in NAPAP, 1987.

[112] Heck, 1988.

Sugar maple in Pennsylvania, New York, New England and southeastern Canada

Crown dieback and elevated mortality rates became apparent in stands of sugar maple and associated hardwoods at some locations in southeastern Canada in the late 1970s. Damage has been noticed more recently in the northeastern United States. Pest infestation or disease is the apparent cause in all of the cases in the United States, although some of the cases in Canada cannot be explained.[113] Air pollution has been suggested as a contributing factor. Regionwide average growth rates in the United States have not declined.[114]

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[113] NAPAP, 1987.

114 Hornbeck et al., 1987, as cited in NAPAP, 1987.

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