Chapter 3

Health Effects of Ozone

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INTRODUCTION

Ozone has been shown to cause immediate, short-term changes in lung function and increased respiratory symptoms among healthy adults and children who exercise moderately or heavily during periods of elevated ozone concentrations. Decreases in lung function and pronounced symptoms such as coughing and pain when breathing deeply have been experienced by people exposed to ozone for 1 to 2 hours at ozone levels comparable to peak levels found in many nonattainment cities. Short-term effects have also been observed at concentrations lower than the l-hour ozone standard (0.12 parts per million (ppm)) when exposures last for longer periods (about 6 hours). The implications of these effects are unclear at this time.

In addition to short-ten-n effects, ozone has been suspected of playing a role in the development of chronic lung diseases and in increasing the rate at which the adult lung ages. While not dismissing the short-term effects of ozone, many health professionals appear more concerned that repeated exposure to ozone over a lifetime may result in permanent impairment of the lungs. Some studies suggest that there may be some persistent effects associated with long-term exposure to ozone, although our understanding of such effects is currently limited. Some new research provides evidence that exposure to ozone for several hours at concentrations equal to or below 0.12 ppm is associated with inflammation of the lungs, a suspected intermediary step in the progression from acute to chronic health effects.

In this chapter, we present four different perspectives on the effects of ozone on human health. First, we present a descriptive summary of the acute and chronic effects that ozone is known or suspected to cause. The second section presents nationwide estimates of population exposure to ozone at concentrations that exceed the standard. About 35 million people-one-quarter of the people who live in nonattainment areas-are exposed to ozone concentrations above the standard, on average, about **9** hours per year. About 13 million people are exposed to concentrations above the standard while exercising at moderate levels of exertion.

Next, we present an assessment of the lung function effects that may be occurring in exercising populations exposed for several hours at concentrations common on days when the ozone standard is exceeded. For example, on a summer day when the ozone level averages 0.14 ppm, a construction worker on an 8-hour shift or a child who plays outdoors for about 4 hours would be at risk of adverse effects on lung function. People exercising more vigorously—e.g., athletes engaged in competitive sports-could expect to experience potentially adverse effects after about 2 hours.

Finally, we attempt to quantify some of the health improvements that would result from lowering ozone concentrations. If ozone concentrations were lowered enough to meet the standard in all areas, several hundred million incidents of respiratory symptoms, such as coughing or pain on deep breathing, might be avoided each year. Some people living in the worst nonattainment areas would experience dozens fewer incidents of respiratory symptoms each year, while many people living in other nonattainment areas would experience no change. Also eliminated would be about 8 million to 50 million days each year when someone's activities are restricted because they are feeling ill from exposure to ozone. By asking people what they would be willing to pay to avoid a day of coughing or restricted activity, for example, it is possible to get a rough feel for the economic value of the health improvements listed above. The uncertainties are quite large due to the many assumptions that must be made, but about $0.5 billion to $4 billion per year is a reasonable range for the portion of health benefits that we were able to evaluate. We could not estimate benefits associated with changes in lung function, or the effect of repeated exposure to ozone over a lifetime (e.g., possible premature aging of the lungs or permanent lung impairment).

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A PRIMER ON THE HEALTH EFFECTS OF OZONE[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 are 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, it has been hypothesized that ozone exposure could contribute to the accelerated aging of the lung, retardation of lung development in children, or the development of pulmonary fibrosis, a chronic lung disease. However, research is 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.

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. what are the effects of exposure to ozone over a long period of time? and
4. who appears to be most susceptible to ozone’s ill effects?

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. Studies conducted both in the laboratory and in the ambient environment generate data which 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 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 helps policymakers define an upper bound on this margin; information on sensitive populations assists in defining a lower bound. Finally, 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.

ISSUE 1: What are the lowest ozone concentrations at which health effects are observed?

The lowest concentration at which effects from ozone have been observed has been revised downward during the last 15 years, as more information has become available. In the early 1970s the threshold for responses to oxidants[3] was presumed to be 0.25 ppm. This was based on limited data, however [87]. In 1977, new ozone studies showed lung function effects to heavily exercising people at concentrations as low as 0.15 ppm [16]. During the last 5 years or so, the health effects database for ozone has greatly expanded. Scientists now believe that the *duration of exposure* to ozone and the *intensity of exercise* during exposure play a major role in determining responses at lower levels of ozone. A number of new human studies show that temporary loss of some lung function occurs in moderately to heavily exercising children and young adults exposed for 1 to 2 hours to ozone concentrations between 0.12 and 0.16 ppm [60,5,70,71]. Significant acute effects have been observed during prolonged periods of exposure (6.6 hours) at moderate exercise levels, at concentrations as low as 0.08

[1]The following summary of the health effects of ozone is based on a report prepared by Lawrence J. Folinsbee for the Office of Technology Assessment (see ref. [25a]).

[2]The air quality standard for ozone is currently under review by the Environmenta Protection Agency.

[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.

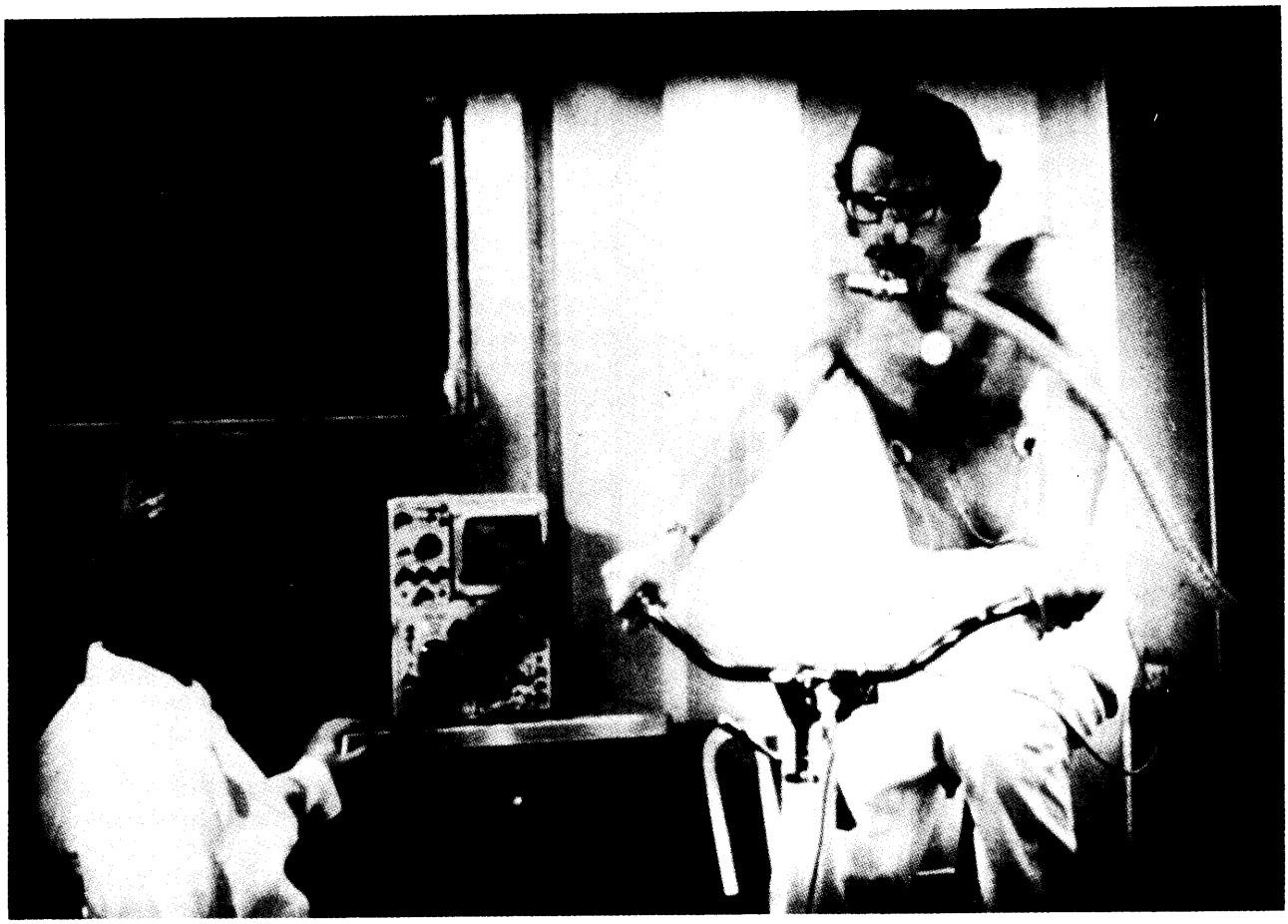


Photo credit: South Coast Air Quality Management District

Much of our understanding of the short-term effects of ozone comes from laboratory studies such as the one shown here. Volunteers breathe filtered air with known concentrations of ozone added, typically for an hour or two while exercising. Both before the experiment begins and after it is over, this volunteer's lung function was measured by having him exhale as rapidly as possible into a test device. Some healthy adults experience some temporary loss of lung function after an hour or two of heavy exercise at ozone concentrations about equal to the standard.

ppm [28,39]. This information is of crucial importance as EPA considers revising the ozone standard from its current level of 0.12 ppm for a *l-hour* averaging time. Consideration of both the *concentration* and *averaging time are* considered by EPA as it reviews the standard. Some argue that the averaging time of the standard should be extended to more accurately reflect atmospheric evidence that ozone concentrations may remain elevated for up to 8 *hours,* not just rise and fall rapidly around a sharp peak concentration. In addition, others argue that lowering the concentration level of the l-hour standard to *below* 0.12 ppm should provide some protection from prolonged exposure effects observed below that level.

ISSUE 2: 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 on lung function of inhaling

ozone at levels equal to or below the National Ambient Air Quality Standard. There is general agreement that permanent respiratory injury or episodes of pollutant-induced respiratory illness that interfere with normal activity would be considered "adverse" [23]. However, it is less clear that acute, reversible changes in lung function or increases in the incidence of respiratory symptoms, neither of which may be associated with disability, 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. Moreover, perceptions of what is a medically significant health effect can vary greatly among physicians and patients.

The EPA staff recommends, and most members of EPA's Clean Air Science Advisory Committee (CASAC) agree, that the threshold for an *individual's* adverse respiratory response to acute ozone exposure should include *all* of the following "moderate" responses: (See also table 3-l.)

- 10 to 20 percent decrement in FEV_1 (i.e., loss of lung function) in individuals (with complete recovery after 6 hours);
- mild-moderate cough, shortness of breath, pain when inhaling deeply; and
- a few individuals (i.e., some with preexisting respiratory disease or heavily exercising healthy individuals) choose to discontinue activity.

Most members of the medical community would consider a 10 percent 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 decrements in lung function. In addition, lung function losses which may not be harmful 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.

ISSUE 3: 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. 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 the following section in this chapter.)

Ozone can cause temporary loss of some lung function and increased respiratory symptoms in healthy individuals exercising heavily (e.g., competitive sports) at concentrations as low as 0.12 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 results in extended or, possibly, permanent changes in lung function, structure, state of growth or aging of the lung.

Both animal and human repeated-exposure studies as well as some epidemiologic studies have attempted to address concerns about the implications of long-term ("chronic") exposure to these low concentrations of ozone. Together, these studies have yielded preliminary evidence that there may, in fact, be some persistent effects associated with chronic exposure. To date, the most compelling evidence suggesting that ozone plays a role in the initiation or triggering of respiratory disease processes has come primarily from animal toxicology studies and human epidemiology studies. This research has also provided scientists with some initial clues about the possible link between acute reversible effects and chronic irreversible effects.

ISSUE 4: Are there any subpopulations which 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

[4] FEV_1—or the volume of air exhaled in the first second of a forced expiration—is one measure of pulmonary function that may indicate airway obstruction in the lungs.

Table 3-I-Gradation of Individual Physiological Response to Acute Ozone Exposure

Gradation of response	Mild	Moderate	Severe	Incapaciting
Change in lung function (FEV_1, FVC)	5-10°10	10-20"/0	20-40%	>40%
Duration of effect	Complete recovery in <30 min	Complete recovery in <6 hr	Complete recovery in 24 hr	Recovery in >24 hr
Symptoms	Mild to moderate	Mild to moderate cough, pain on deep inspiration, shortness of breath	Repeated cough, moderate to severe pain on deep inspiration and shortness of breath; breathing distress	Severe cough, pain on deep inspiration, and shortness of breath; obvious distress
Limitation of activity	None	Few individuals choose to discontinue activity	Some individuals choose to discontinue activity	1 Many individuals choose to discontinue activity

NOTE: EPA staff recommend that the moderate, severe, and incapacitating categories should be considered "adverse" respiratory health effects. All four types of effects within a category must be present for a response to be called "adverse."

SOURCE: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, "Review of the National Ambient Air Quality Standards for Ozone-Assessment of Scientific and Technical Information," draft staff paper, November 1968, p. VII-46.

public health," the EPA has sought to identify those subpopulations, if any, which are shown to be more sensitive to ozone exposure than the general population.

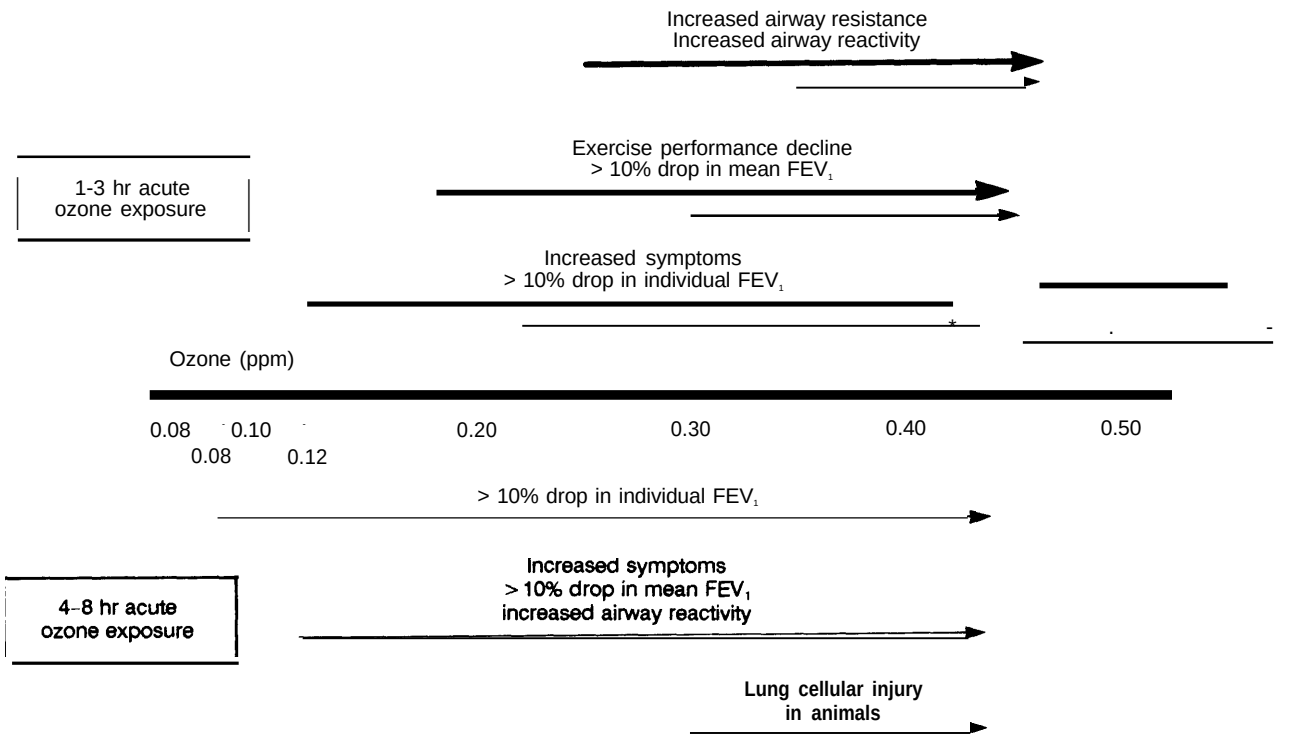
EPA has identified two major groups at increased risk of developing adverse health effects from exposure to ozone: 1) a subgroup of the general population with preexisting disease (e.g., asthma, chronic obstructive pulmonary disease); and 2) those individuals who exercise or work outdoors [98]. The first group is of concern because their respiratory systems are already compromised, placing them 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* within these groups appear to be more sensitive. In general, people with pre-existing respiratory disease have not been studied at ozone concentrations and exercise levels as high as those used for healthy subjects. The strongest evidence for a population "at-risk" exists for healthy, heavily exercising individuals.

Studies have also 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 "responders." The EPA estimates that from 5 to 20 percent of the healthy population may represent a subgroup of responders who are at abnormally high risk for the acute effects of ozone exposure [98]. The factors that would account for such individual variability in sensitivity are unknown at this time. Whether these susceptible individuals are also at increased risk for the development of chronic, irreversible effects from ozone is also unknown. (Susceptible populations are discussed at greater length towards the end of this section.)

The Acute Effects of Ozone

A great deal of research has been conducted on the acute or short-term health effects from ozone exposure. The primary acute effects investigated are: impairment of lung function, inflammation of the deep lung, respiratory symptoms, and limitations on activity. These acute effects of ozone exposure are summarized in figure 3-1, along with the ozone level at which they begin. The figure is divided into two sections: the upper section describes effects that occur with 1- to 3-hour exposures, the lower section those that occur with 4-to 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 heavy exercise for 3 hours or

Figure 3-l—Acute Effects of Ozone Exposure



Effects above the ozone concentration line are from 1 to 3 hour exposures to ozone. Effects below the line are from 4 to 8 hour exposures. FEV_1 (forced expiatory volume in 1 second) is a measure of lung function. The bolder arrows indicate the range of concentrations at **which effects** occur from exposure while exercising heavily; the lighter arrows indicate the concentrations at which effects occur while exercising moderately. Effects begin at the concentration indicated by the tail (left side) of the arrow.

SOURCE: L.J. Folinsbee, "A Summary of the Health Effects of Ozone," contractor report for OTA, June 30, 1988.

less. With moderate exercise, effects occur at low concentrations if exposures are prolonged (6 or more hours). The more adverse responses, such as cell damage shown in laboratory animal studies, occur at the higher concentrations.

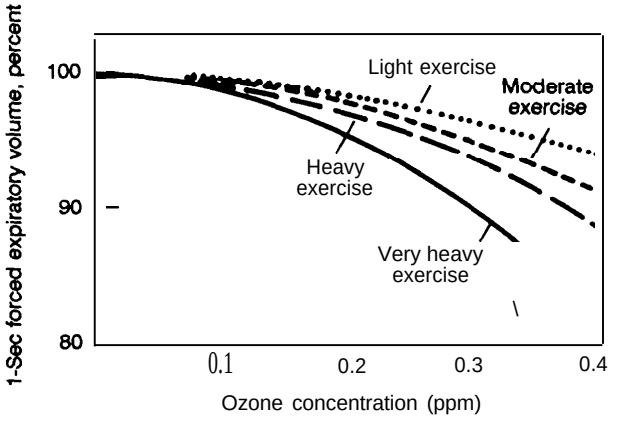
Lung Function Effects

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 "one-second forced expiatory volume" (FEV_1) and 'forced vital capacity" (FVC).[5] Ozone can cause decreases in both of these measures of lung function.

Changes in lung function depend on the dose of ozone which 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. Figure 3-2 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. It is important to point out that this figure illustrates the average effect of exercise on groups, and that a great deal of variability in response exists among *individual.* Many studies have, in fact, shown that there

[5]FEV_1 is the maximum amount of air that can be exhaled from the lungs in 1 second; FVC is the maximum amount of air that can be exhaled from the lungs after taking a full deep breath.

Figure 3-2—Percent Decreases in Group Mean Lung Function During 2-hr Ozone Exposures With Different Levels of Intermittent Exercise



An increase in exercise intensity at any given ozone concentration results in a larger group average loss of lung function (FEV_1, forced expiatory volume in 1 second). The lung function changes shown in the graph are for 1- to 2-hour exposures. Note that some individuals may experience decreases as much as three times greater than the group average.

SOURCE: U.S. Environmental Protection Agency, office of Air Quality Planning and Standards, *Review of the National Ambient Air Quality Standards for Ozone Preliminary Assessment of Scientific and Technical Information, Draft Staff Paper* (Washington, DC: November 1988).

can be a large difference between the average change in lung function for a group and changes experienced by some individuals within a group.[6]

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 observed from 11 to 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 0.12 to 0.24 ppm [70,26,3,35]. Average decreases in group mean FEV_1 ranged from 6 to 22 percent. For comparison, the range of lung function decrease due to the normal aging of the lung ranges from about 0.5 to 1 percent per year in adult males between the ages of about 30 to 70 years old [19]. While the lung function changes due to acute ozone exposure appear to be *temporary, the* changes due to normal lung aging are *permanent.* Furthermore, the mechanism initiating these permanent changes in aging lungs is quite different from that at work in lungs acutely exposed to ozone.

At ozone concentrations equal to or exceeding the current ambient air quality standard for ozone, some investigators have seen small (4 to 6 percent) but statistically significant group mean decreases in FVC and FEV_1 under conditions of heavy exercise [70,35], while others have not [85,52,60]. 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 1- or 2-hour exposure periods. The most substantial responses in this range of ozone concentration occur under conditions of moderate or heavier exercise and durations of exposure longer than 1 or 2 hours. For example, Folinsbee and coworkers recently observed 7- to 13-percent decreases in group mean FEV_1 in subjects performing moderate exercise for 6.6 hours at ozone levels of between 0.08 and 0.12 ppm [28,39]. Folinsbee, under contract to OTA, used these and other laboratory data to extrapolate the effects of multiple-hour exposures to ozone at concentrations typical of summertime conditions present in a number of U.S. cities [25 b]. A discussion of this analysis, including the lung function impacts one could expect from "typical" exercise scenarios, is presented in the third section of this chapter.

All of the lung function effects mentioned above were observed in human chamber studies. Some scientists believe, however, that chamber studies underestimate the effects from ozone exposure that may occur in populations exposed to ozone in the ambient air while engaged in normal recreational activity. The effects of ozone on lung function have also been evaluated in the ambient environment through field studies. Many of these studies have been of children in summer camp, but some have been of healthy adults engaged in outdoor exercise [89,63]. The decreases in lung function observed in these studies have been greater than those seen in

[6] In a study by Folinsbee et al. [28], the average group change in FEV_1 at 0.12 ppm of ozone was 13 percent, with individual changes *ringing* from -47.6% to +3.5 percent. Gong et al. [35] showed an average change in lung function of –5.6 percent in a group exposed to 0.12 ppm of ozone, with individual responses varying from –30 percent to +10 percent. In a study by McDonnell et al, [70], while the average group decrease in FEV_1 was <5 percent, individual responses ranged from a 17-percent decrease in FEV_1 to no change in this measure of lung function.

human chamber studies in controlled indoor environments. Some scientists have postulated that the presence of other pollutants in the ambient environment, as well as other cofactors such as temperature and humidity, have contributed to this increased effect. With regard to the more significant lung function effects observed in summer camp children, some have proposed that this is the result of their greater cumulative daily exposure to ozone. These children may be exposed to ozone outdoors practically all day long, as opposed to children in chambers who may be exposed to ozone for 1 to 2 hours, with periods in clean air both before and after ozone exposure.

A current controversy surrounding impairment of lung function from ozone exposure involves the definition of an "adverse" loss 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 *individual* within these groups experience decrements in lung function greater than the average. There is less consensus, however, as to whether or not temporary and infrequently occurring changes of less than 10 percent, in and of themselves, represent an adverse health effect for a healthy young adult. Some health professionals would consider such changes to be adverse if they restrict activity or limit performance [23]. Short-term, reversible loss of lung function could have adverse effects in individuals whose lung capacity is already reduced. However, there is no universal agreement among scientists as to the implications of such "small" changes.

Symptom Responses

Symptoms experienced by people exposed to ozone are also important markers of the effects of ozone. The major respiratory symptoms-coughing 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 1 to 3 hours at concentrations as low as 0.12 ppm have been shown to cause such symptoms in healthy young adults [70,3,52]. Pronounced symptoms such as repeated coughing or pain when taking a deep breath will almost always be associated with substantial (greater than 10 percent) lung function changes. Folinsbee and coworkers' recent study [28] demonstrated a significant correlation at 0.12 ppm between discomfort on deep breathing and changes in lung function (FVC) within *individuals.* However, most other studies that have looked for such an association have not seen it at this concentration.

Adults perceive symptoms of ozone exposure at low concentrations (0.12 ppm) [70] but children apparently do not [71,4,5]. 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 try 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.

The last section of this chapter presents the results of a health benefits study conducted for OTA [49]. This study estimates the benefits of controlling ozone with respect to symptoms *avoided* and re*stricted activity. The* benefits of reducing lung function effects and the risk of developing chronic respiratory diseases were not estimated.

Chronic Effects: The Development of Respiratory Disease

In understanding how ozone may contribute to the development of respiratory disease, information about the *mechanism* of effect is vital. Because such effects are difficult to observe in humans, however, scientists often turn to animal studies for this information. Until very recently, little information has been available on the underlying changes (e.g., biochemical and structural effects) occurring in the lungs that may mark the beginnings of respiratory disease. Because scientists cannot easily observe the changes induced by ozone exposure that may be occurring at the cellular level in the human lung, they have tended to investigate other types of responses. Both human chamber and epidemiology studies have been used to examine some of these responses, including: *symptoms* produced by exposure, the magnitude of decline in ***lung*** *function,* and the related *disability* or *peformance* decreases that may occur in exposed individuals. These responses,

while important in their own right, may also reflect changes at the cellular level that contribute to the development of chronic respiratory disease.

Animal Studies

Animal studies serve two distinct purposes: 1) providing information on the basic mechanism at work in the lungs in response to ozone exposure, and 2) providing a better understanding of the possible effects of chronic exposure to ozone.

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 (e.g., pulmonary fibrosis), although there is no scientific consensus regarding the significance of these observed effects. Studies of animals exposed repeatedly 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 thus ward off infection [29], and for causing lung inflammation [104]. Generally speaking, extended exposure to either high or low concentrations of ozone will tend to retard lung clearance. There is some evidence that acute exposure to low concentrations of ozone may actually enhance clearance.[7] Several studies, however, have shown an increased response to *bacterial* infection in animals exposed to ozone levels as low as 0.08 to 0.10 ppm for several hours [73,22]. Continuous exposure to ozone (at 0.50 ppm) has also been shown to alter the course of *viral* infection in mice by leading to structural changes in the lungs that increase the likelihood that fibrosis[8] will occur [41].

One type of structural change in the lungs that some scientists believe may be linked to the development of lung fibrosis is the deposition of collagen-a structural protein that contributes to "stiffening" of the lungs [53,11].9 Repeated, intermittent exposure of monkeys to ozone concentrations as low as 0.25 ppm has been shown to result in increased lung collagen content [91], although it is not certain whether such increases alone are great enough to cause fibrosis. Injury to the periphery of the lungs has been demonstrated in rats exposed to ozone at the current standard level of 0.12 ppm [15]. Ozone has also been shown to damage certain lung cells in animals at levels as low as 0.25 ppm [14]. 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. For example, uncertainties about: 1) how the distribution of dose within the respiratory system compares among animals and humans, and 2) whether, for a specified dose to a target site, responses in the two species would be quantitatively and qualitatively equivalent, make dose-response comparisons a difficult task.

Epidemiologic Studies

Epidemiologic studies have also been used to investigate the potential link between ozone exposure and respiratory disease. These 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. 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. If breathing ozone over along period of time causes an acceleration of the lung aging process, we would expect to see a more rapid age-related decline in lung volume in people who reside continuously in oxidant-polluted areas. One epidemiologic study of populations living in southern California suggests that

[7]Some scientists believe that short-term exposure to ozone does not allow enough time for the cilia (a defense mechanism of the lungs against foreign material) to be damaged, which tends to occur when the lungs are exposed to ozone for a prolonged period. Cilia normally act to clear out foreign material in the lungs, and some suspect that short-term exposure to ozone may increase the liquid flow of mucus in the lungs, stimulating the cilia to react to clear the lungs.

[8]Pulmonary fibrosis results from the formation of excessive amounts of protein fibers that stiffen the *hmg.* If this Stiffening is severe enough, it can produce debilitating disease.

[9]In addition to its suspected role in the development of fibrosis, lung stiffening is associated with breathing difficulty and subsequent limitation of work performance.

respiratory function is affected by chronic exposure to ozone. The study showed an association between an accelerated loss of lung function over an extended period of time (5 years) and residing in a high oxidant community [17].[10] 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.

Susceptibility and Adaptation From Repeated Exposure to Ozone

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 [27,10]; and 2) with continued exposure, these effects begin to diminish in intensity and after 4 or 5 days the pulmonary function effects are undetectable [40,50,58]. This gradual loss of functional response has been called ''adaptation. ''

The adaptive responses of individuals who live in areas with high ozone levels, however, 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 "smog season" but regain their sensitivity during the relatively smog-free winter season [61]. In this study, "adaptation" did not disappear rapidly, as in the chamber exposures, but appeared to persist for at least 2 to 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.

Though measurable lung function changes and symptom responses may lessen for a period during repeated exposure, *other* changes within the lungs may still be ongoing. For example, research on animals shows that some lung injury, in the form of effects on host defense systems [33], increased susceptibility to disease [34], and lung inflammation [46], may continue during an "adaptive" period when lung function changes and symptom responses are reduced. Therefore, individuals who, through adaptation, experience fewer or less severe symptoms, may be at increased risk for longer-term damage because of these other, ongoing effects. Since these individuals may believe that they are able to tolerate exercise outdoors during peak ozone episodes because they experience fewer symptoms, they may receive potentially greater tissue damage over the long-term.

The Possible Link Between Acute and Chronic Effects

New research examining the effects from prolonged exposure to ozone at levels equal to or below the standard are providing scientists with preliminary information on the possible links between acute and chronic effects. Prolonged exposure to ozone at concentrations *equal to* or *below the* ozone standard can be associated with inflammation of the lungs, a suspected intermediary step in the progression from acute to chronic health effects [46,21]. However, questions about the degree of tissue injury occurring, and, if it occurs repeatedly, whether this injury leads to chronic health effects, remain unanswered. Not only has tissue injury in the lungs been demonstrated at 0.12 ppm, but the elasticity of the lungs also appears to have been affected. This latter effect is believed to accelerate the normal aging process of the lungs [13,6].

Human Chamber Studies

Prolonged acute exposures (up to 6.6 hours) of humans in controlled laboratory settings to ozone concentrations similar to those found in many nonattainment cities (0.12 to 0.18 ppm) have produced several effects, including: progressively larger changes in respiratory function and symptoms with time [28], increased responsiveness of the airways of individuals to inhaled substances [72], and increased membrane permeability [43,46]. The relationship between these 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 ob-

[10] While the Detels et al. study [17] does not provide scientists with quantitative dose-response data, its results showing an association between living in a high oxidant area and increased lung function losses, contribute to our understanding of the potential long-term effects of ozone exposure.

served in the animal and human subjects of short-term ozone studies. Before this inflammatory response disappears, some suggest that it may induce other, persistent changes in the lung or, with additional exposure or a concurrent infection, might culminate in chronic degenerative respiratory effects. Airway inflammation occurs during the development of a number of respiratory diseases, most notably asthma and chronic bronchitis.

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. This desire has been echoed more explicitly in the legislative history of the Act [94].

At present, scientists postulate that about 5 to 20 percent of the healthy population may represent a subgroup of "responders" [98] who may be significantly more responsive than the general population to the same dose of ozone. Some also consider people with pre-existing respiratory disease (e.g., asthma, chronic bronchitis), individuals who exercise heavily or work outdoors, and children as potential "at-risk" groups.

The strongest evidence for increased responsiveness exists for people who exercise intensively outdoors, since 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. However, limited data make it difficult to confirm the susceptibility of people with preexisting respiratory disease.

Athletes and Outdoor Workers

Both epidemiologic and chamber studies have indicated that athletes may be at substantial risk of experiencing decreases in work performance and temporary loss of some lung function when exercising for approximately 1 hour at ozone concentrations as low as 0.20 ppm [26,35,85]. 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 heavy manual labor while exposed to 0.12 ppm ozone experience significant loss in lung function (13 percent group mean decrease in FEV_1) and pronounced symptoms (e.g., cough, pain when inhaling deeply) [28]. This research suggests that extended periods of heavy exercise while exposed to ozone may be harmful to 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 to 0.18 ppm).

Asthmatics

Results of studies on asthmatics are mixed. A number of epidemiologic studies of asthmatics have suggested that ozone exposure may be associated with increased asthma attacks, hospital admissions for asthma, temporary loss of some lung function, and symptoms (See ref. [103,9,38,36]. 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 typically shown that the lung function and symptom responses of asthmatics to a specific low concentration level of ozone do not differ from the responses of healthy non-asthmatics [44,62,57] .11

It is unclear why asthmatics have generally failed to exhibit increased sensitivity to many of the effects of ozone in chamber studies. 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. Chamber studies of asthmatics have only recently been conducted at the higher exercise and ozone concentration 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 environ-

11 While the weight of the evidence suggests that asthmatics are no more sensitive to ozone than healthy, non-asthmatics, one recent clinical study suggests that asthmatics may be slightly more sensitive to the effects of ozone on airway narrowing, which occurs at somewhat higher ozone concentrations than the changes in FEV_1 [47].

mental factors (i.e., other pollutants, high temperatures, and humidity) in the field. Factors operating in the ambient environment may not have been replicated in clinical studies. The question of whether asthmatics may be somewhat more adversely affected by ozone inhalation is not yet resolved.

People With Chronic Obstructive Pulmonary Disease

People with chronic obstructive pulmonary disease (COPD) (e.g., 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. Like asthmatics, relatively small decrements in lung function could be adverse for them, compared to healthy individuals, who may not be affected by such changes. Several different laboratory studies have been conducted on COPD patients exposed to ozone [51,58,59,88,42], 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 to 2 hours. It would be necessary to study these individuals over longer periods of exposure and at higher exercise levels (unobtainable by many COPD patients) in order to adequately evaluate their risk from ozone exposure. Out of concern for their health, studies of patients with COPD, like those with asthma, have not been performed under such conditions to date.

Children

Concern for children as a *potentially* susceptible subgroup has been raised for several reasons:

1. their lungs are not fully developed until adulthood, increasing their risk for damage from ozone exposure;
2. they are more likely than the average adult to be exercising outdoors when ozone levels are high (summertime); and
3. their higher metabolic rates tend to lead to higher ventilation rates during exercise, which may give them a greater dose of ozone than exercising adults.

The critical question regarding children exposed to ozone is whether repeated exposure will influence their 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 [71,63]. 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 different than those experienced by adults. However, children appear to experience fewer symptoms than adults when exposed to concentrations as low as 0.12 ppm [71,3,52]. It is unclear at this time why children have weaker symptom responses. Some scientists have suggested that this lack of significant symptom response may put them at greater risk because it would fail to deter them from future ozone exposure.

The Elderly

Concern over the elderly as a possible "ozone-sensitive" subgroup has been largely because of a general belief that the most frail members of any population may be at an overall greater risk from numerous environmental stresses than the population at large. However, it is commonly accepted that these individuals are the least likely to be exercising outdoors where they might be exposed to ozone. A subgroup of healthy, older adults may be at risk because they may participate in outdoor activities where they might be exposed to ozone. The limited evidence available at this time, however, does not indicate that age plays a significant role in their response to ozone. While lung function effects have been observed in this subpopulation, several studies suggest that healthy older adults may, in fact, be less susceptible to the acute lung function effects of ozone than healthy young adults [20,82]. The extent to which pulmonary function changes reflect other events occurring in the lungs of older adults who are exposed to ozone is unknown.

Possible Synergistic Effects of Ozone and Acid Aerosols[12]

Some scientists are concerned that ozone and acid particulate and vapors may interact to affect human health. This has been prompted by research indicating that both pollutants affect a similar target in the lungs, may be enhanced by exercise, and reach peak concentrations at the same time of the year. Some laboratory findings suggest that the response of

[12]This section is based largely on EPA's "Acid Aerosols Issue paper" [99].

Photo credit: Steve Delaney

Many health scientists are worried about the effects of ozone on children at play during high ozone episodes found in many nonattainment cities. The critical unknown is whether repeated exposure will influence their lung maturation.

subjects exposed to ozone in conjunction with acid aerosols is greater than when exposed to ozone alone [75].

The two types of acid particulate that are receiving the most attention are ammonium bisulfate and sulfuric acid. There is evidence to suggest that both acids are respiratory irritants and that their ''target zone,'' owing to their small size, is the periphery of the lungs, similar to that for ozone. In addition, exercise seems to exacerbate the effects of inhaled sulfuric acid [100], as has been shown to be the case with the impact of ozone exposure. Moreover, on the east coast, airborne sulfates are most acidic in the summertime, the time of year when peak ozone levels tend to occur.[13]

Possible interaction between ozone and some acid aerosols is believed by some scientists to affect lung clearance mechanisms, lung function, and acute respiratory hospital admissions. Recent studies of animals exposed to sulfuric acid show persistent impairment of lung clearance, as does research currently underway with ozone [86].[14] Disturbance of lung clearance mechanisms is believed by some scientists to promote the inception or progression of chronic respiratory disease, but there is no proven connection at this time. Given the recent concern about chronic health effects from exposure to ozone alone, and the possibility of synergism between ozone and certain acid aerosols, this new information is of particular concern.

13011 the west coast,nitricacid, which is in vapor form under most ambient conditions, has shown a correspondence with ozone concentrations.

[14]Work on 4-, 8-, and 12-month exposures is in progress at this time.

Short-term loss of some lung function in children exposed to ozone (0.12 to 0.18 ppm) in the ambient environment has led some researchers to postulate that other pollutants, in particular, acid sulfates, may have contributed to this enhanced effect [62]. One epidemiologic study has shown a significant correlation between ozone, sulfates, temperature, and respiratory disease admissions to the hospital during the summer months [9].

Research suggests that the acidity of an aerosol is related to its toxicological potency, and is an important factor in determining whether the aerosol will interact synergistically with *ozone* [99,54]. There is some evidence that much lower concentrations of sulfuric acid—the more acidic aerosol—than ammonium sulfate (0.04 mg/m^3 v. 5.0 mg/m^3, respectively) [102,101] are needed to produce a synergistic effect. Evidence from field studies shows temporary effects on lung function of summer camp children from elevated levels of sulfuric acid (>0.04 mg/m^3) and ozone (>0.13 ppm) [79,80]. Concentrations of sulfuric acid up to 0.04 mg/m^3 have been observed in urban areas in the United States [99]. Preliminary evidence from animal studies, however, indicates effects only at much higher levels than the human studies. Effects on the rat lung do not appear until sulfuric acid concentrations reach 0.5 mg/m^3, in combination with 0.12 ppm of ozone [102].

While our understanding of the relationship between ozone and acid aerosols is limited at this time, the apparent correlation between atmospheric concentrations of ozone and acid particulate and their respective health effects, as well as the general lack of data on acids in the ambient environment, indicate a need for additional research on pollutant mixtures.

EXPOSURE TO OZONE

As discussed above, ozone has been shown to cause short-term decreases in lung function and increased respiratory symptoms in people engaged in moderate to heavy exercise when ozone concentrations exceed the standard. There is also concern about persistent health effects associated with long-term exposure to ozone. This section presents information on the number of areas throughout the United States where the ozone standard is not met, and the population that lives in those areas. To get a sense of the frequency with which people may be exposed to elevated ozone levels and the magnitude of these exceedances, the number of times that areas fail to meet the standard is also presented. Because *living* in an area where ozone levels have been measured above the standard does not guarantee that a person will actually be *exposed* at those levels, we look at the various factors that influence exposure to ozone.

Areas Failing To Meet the Standard

An area is designated "nonattainment" for ozone if concentrations exceeding 0.125 ppm (l-hour average) are measured on more than 3 days over a 3-year period at any monitoring site in the area (i.e., the area has an "expected exceedance" rate greater than once per year, averaged over 3 years).

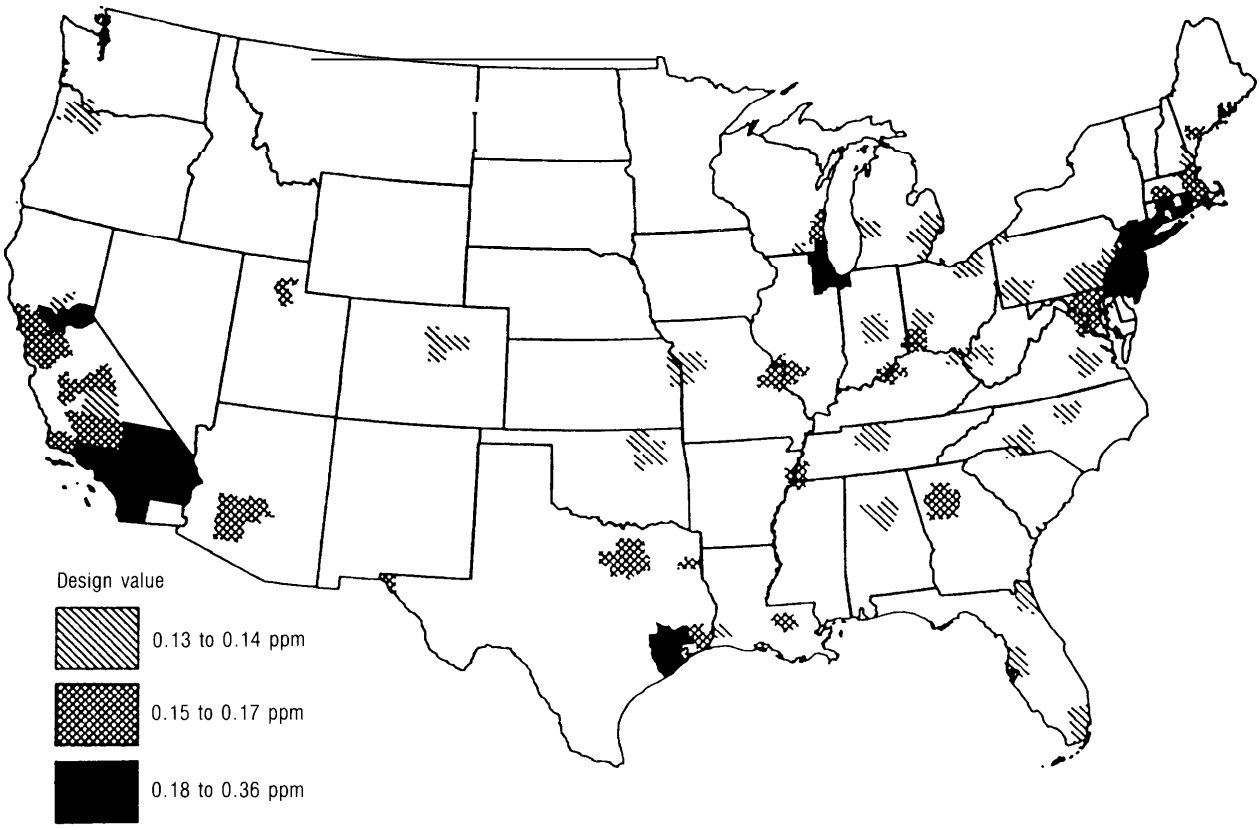
Figure 3-3 shows the metropolitan statistical areas (MSAs) and grouped or "consolidated" metropolitan statistical areas (CMSAs) that were classified as ozone nonattainment areas based on 1983-85 monitoring data. Areas that were designated nonattainment for the 1983-85 period, as well as the 1985-87 period, are listed in table 3-2. As indicated in the table, several non-MSA areas were also designated nonattainment but are not shown on the map.[15]

EPA updates the list of nonattainment areas every year as data for a new season become available. Based on the 1983-85 data, 76 urban areas (encompassing 104 individual MSAs plus the 10 non-MSA areas) were designated nonattainment. In contrast, 70 areas were designated nonattainment based on the 1985-87 period (18 areas were dropped in and 12 areas were added). The difference is partially attributable to differences in weather between the two periods. We focus on the nonattainment list from the 1983-85 period for consistency with other parts of our assessment. The list for the most recent 3-year period at the time of publication (1986-88) is not yet available.

The shading in figure 3-3 indicates the 1983-85 "design value" for each area. The design value is a

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

Figure 3-3-Areas Classified as Nonattainment for Ozons Based on 1083-85 Data



The shading indicates the fourth highest daily maximum 1-hour average ozone concentration, or "design value," for each area.

SOURCE: U.S. Environmental Protection Agency

measure of the highest l-hour average ozone concentrations in the area and is the fourth highest of all of the daily peak l-hour average ozone concentrations observed within the area over the most recent 3-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 the standard. For the 1983-85 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 Exceedances

Figures 3-4 through 3-6 show the areas throughout the contiguous United States where ozone concentrations exceeded 0.12, 0.14, and 0.18 ppm, respectively, at least 1 hour per year, averaged over the years 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.[16] The data shown were obtained from EPA [84]. 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

[16]The number of monitors in each area ranges from 1 to 18 (in Los Angeles). The average number of monitors in each area is three.

Table 3-2-Areas Classified as Nonattainment for Ozone Based on 1983-85 and 1985-87 Data

Area name	Design value (ppm) 1983-85	1985-87
1983-85 design value of 0.13 to 0.14 ppm		
Acadia National Park, ME*	0.13	----
Allentown-Bethlehem, PA	0.14	0.13
Birmingham, AL**	0.13	0.15
Charleston, WV	0.13	----
Charlotte-Gastonia-Rock Hill, NC-SC	0.13	**0.13**
Cleveland, OH	**0.14**	**0.13**
Dayton-Springfield, OH	0.13	**----**
Denver-Boulder, CO	**0.13**	**----**
Detroit, MI	**0.13**	**0.13**
Dover, DE*	0.14	----
Erie, PA	0.13	----
Gardiner, ME*	0.14	----
Grand Rapids, MI	0.13	0.13
Hancock County, ME*	0.13	0.13
Harrisburg-Lebanon-Carlisle, PA	0.13	----
Huntington-Ashland, WV-KY-OH	0.14	0.14
Iberville Parish, LA*	0.13	0.13
Indianapolis, IN	0.13	0.13
Jacksonville, FL	0.14	0.16
Janesville-Beloit, WI	0.13	----
Jefferson County, NY*	----	0.13
Kansas City, MO-KS	0.14	----
Kennebec County, ME*	----	0.12
Kent County, DE*	----	0.13
Kewaunee County, WI*	----	0.13
Kings County, CA*	----	0.13
Lake Charles, LA.	0.14	----
Lancaster, PA	0.13	----
Lexington, KY	----	0.13
Lincoln County, ME*	----	0.13
Miami-Hialeah, FL**	0.13	0.15
Montgomery, AL	----	0.14
Muskegon, MI	0.14	0.17
Nashville, TN	0.14	0.14
Norfolk, VA	----	0.13
Northampton County, VA*	0.14	----
Parkersburg, WV-OH	----	0.13
Pittsburgh, PA**	0.13	0.13
Pointe Coupee Parish, LA*	0.13	----
Portland, OR-WA**	0.13	0.15
Portsmouth-Dover-Rochester, NH-ME	0.13	0.13
Raleigh-Durham, NC	----	0.13
Reading, PA	0.13	----
Richmond-Petersburg VA	0.13	0.13
St. James Parish, LA*	0.13	----
Tampa-St. Petersburg-Clearwater, FL**	0.13	0.13
Tulsa, OK	0.13	0.12
Visalia-Tulare-Porterville, CA	0.13	0.15
York, PA	0.13	----
Yuba City, CA	0.13	----
1983-&J ***design value of 0.15 to 0.17 ppm***		
Atlanta, GA	**0.16**	0.17
Bakersfield, CA	0.16	0.16
Baltimore, MD	**0.17**	0.17
Baton Rouge, LA	**0.1 6**	0.14
Beaumont-Pod Arthur, TX	**0.16**	0.13
Boston, MA	**0.16**	0.14
Cincinnati, OH-KY-IN**	0.17	0.14
Dallas-Ft. Worth, TX**	0.16	0.16
El Paso, TX	0.16	0.16
Fresno, CA	**0.17**	0.17
Longview-Marshall, TX	**0.15**	----
Louisville, KY-IN	0.15	0.16
Knox County, ME*	----	0.15
Memphis, TN-AR-MS	0.15	0.13
Milwaukee, WI**	0.17	0.17
Modesto, CA	**0-15**	0.15
New Bedford, MA.	**0.16**	0.14
Phoenix, AZ	**0.16**	0.14
Portland, ME	0.16	0.14
Salt Lake City-Ogden, UT.	0.15	0.15
Santa Barbara-Santa Maria-Lompoc, CA.	0.16	0.14
Seaford, DE*	0.15	----
St. Louis, MO-IL**	0.16	0.16
Stockton, CA	0.15	0.14
Washington, DC-MD-VA	0.16	0.15
Worcester, MA	0.15	0.13
York County, ME*	0.15	0.15
San Francisco, CA**	0.17	0.14
1983-65 ***design value*** 0.18 to ***0.26 ppm***		
Atlantic City, NJ	0.19	0.14
Chicago, IL**	0.20	0.17
Greater Connecticut**	0.23	0.17
Houston, TX**	0.25	0.20
New **York, NY-NJ-CT***	**0.22**	0.19
Philadelphia, PA-NJ**	**0.18**	0.16
Providence, RI*	**0.18**	0.16
Sacramento, CA	**0.18**	0.17
San Diego, CA	**0.21**	0.18
1983-85design value 0.27 ppm or higher		
Los Angeles-Long Beach, CA**	0.36	0.35

*non-MSA area. **multi-MSA consolidated area. ----in attainment.

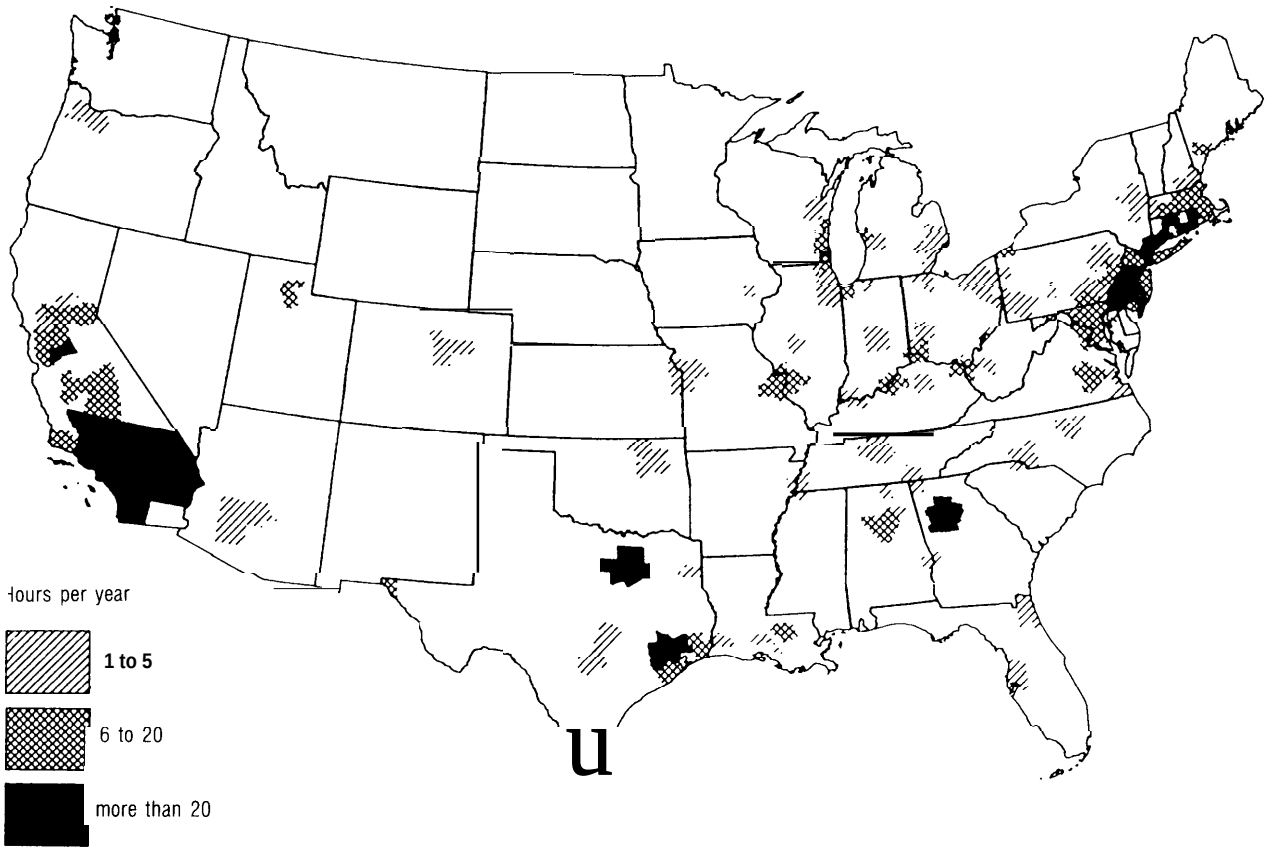
SOURCE: U.S. Environmental Protection Agency.

expected to show exceedances of the specified concentrations if data for the peak monitor in each area were used.

Of the 317(urbanandnonurban) areas for which we have ozone data, figure 3-4 shows the 130 areas where a concentration ofO.12 ppm was exceeded at least l hour per year, on average, between 1983 and 1985.[17] Sixty of those are a shad concentrations equal to or greater than 0.12 ppm for60r more hours per year, The Dallas, Houston, and Atlanta areas and

[17]If data for the peak monitor in each area had been used instead of the all monitor average statistics, 146 areas would be indicated ashavingozone concentrations greater thanor equal to 0.12 ppm at least 1 hour per year.

Figure 3-4-Areas Where Ozone Concentrations Exceeded 0.12 ppm at Least One Hour Per Year on Average, From 1983-85



Data **from all monitors located 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.

SOURCE: U.S. Environmental Protection Agency, *Storage and Retrieval of Aerometric Data,* database, processed by E.H. Pechan & Associates, 1987.

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.

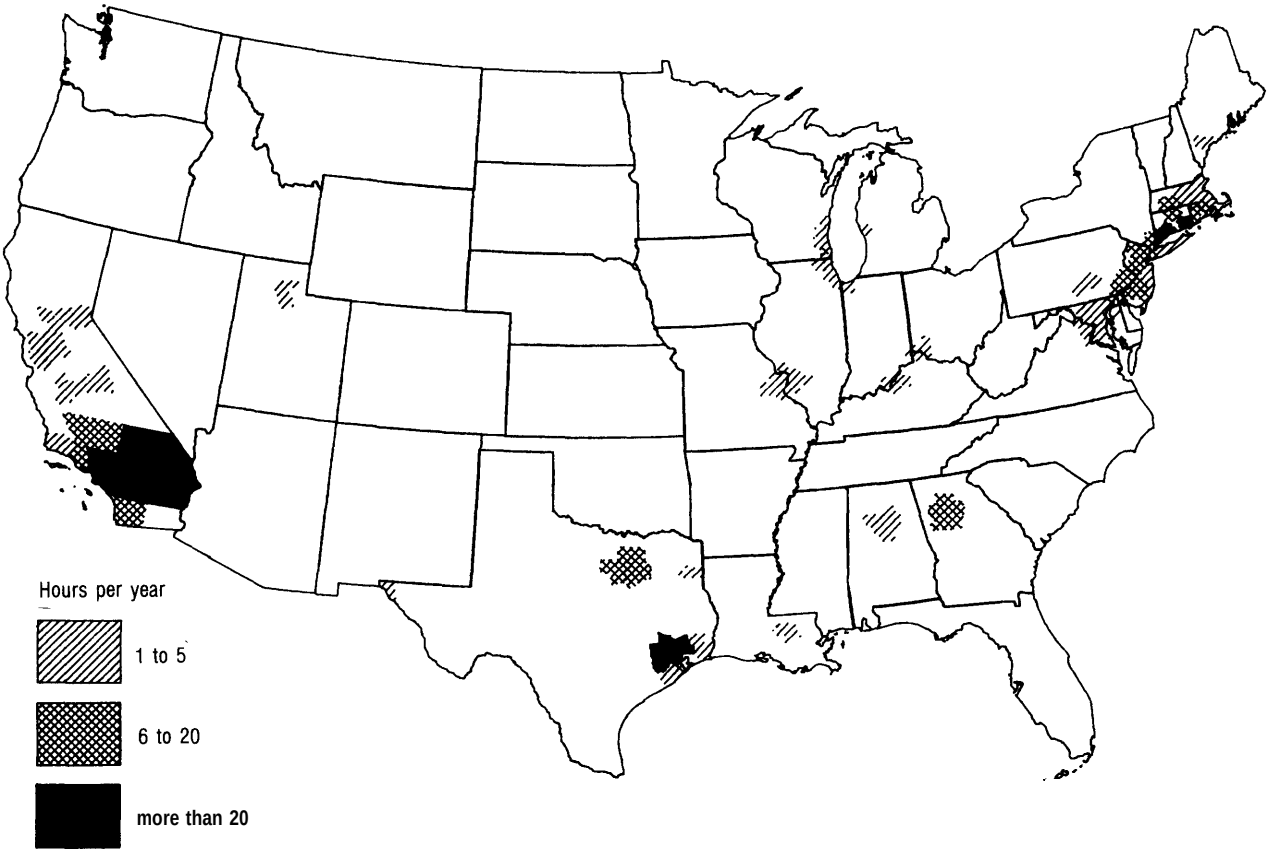
Figure 3-5 shows the 60 areas where the all-monitor average statistics indicate that ozone concentrations reached 0.14 ppm at least 1 hour per year between 1983 and 1985. Twenty-four of these areas recorded ozone concentrations of at least 0.14 ppm for 6 or more hours per year. Seven areas, namely the Houston area and parts of Connecticut and southern California, recorded concentrations of 0.14 ppm or higher more than 20 hours per year.

Figure 3-6 shows the 18 areas where concentrations were as high as 0.18 ppm for 1 or more hours per year between 1983 and 1985. The all-monitor average statistics indicate that concentrations exceeded 0.18 ppm 6 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

Figure 3-5-Areas Where Ozone Concentrations Exceeded 0.14 ppm at Least One Hour Per Year on Average, From 1983-85



Data from all monitors located 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.

SOURCE: U.S. Environmental Protection Agency, Storage and *Retrieval of Aerometric Data,* database, processed by E.H. Pechan & Associates, 1987.

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 v. 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 inhaled 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 3-7 shows a profile of ozone concentrations as they change over the day at a single monitoring site. 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 l-hour average concentration for several hours.

Figure 3-6-Areas Where Ozone Concentrations Exceeded 0.18 ppm at Least One Hour Per Year on Average, From 1983-85

Data from all monitors located 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.

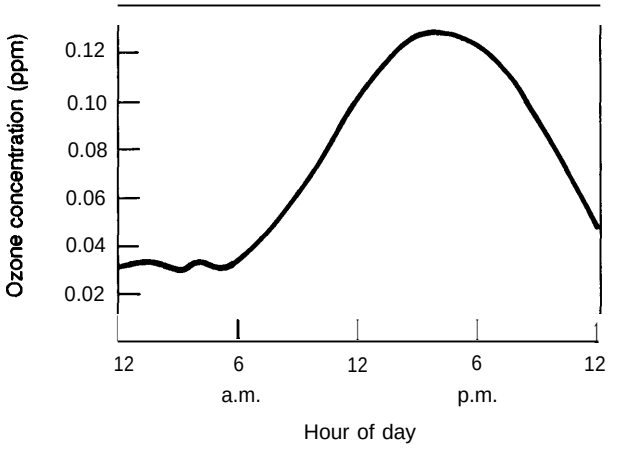
SOURCE: U.S. Environmental Protection Agency, Storage and *Retrieval of Aerometric Data, database, processed by* E.H. Pechan & Associates, 1987.

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 3-8 shows a contour map of how peak ozone concentrations on a given day vary across the New York City metropolitan area. The diagram shows 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 3-8, ozone concentrations tend to vary smoothly over large areas, and not to show sharp, localized peaks.[18]

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 [76]. Ozone concentrations inside buildings with open windows instead of air-conditioning are estimated to be about 60 percent of

[18]One exception to 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,

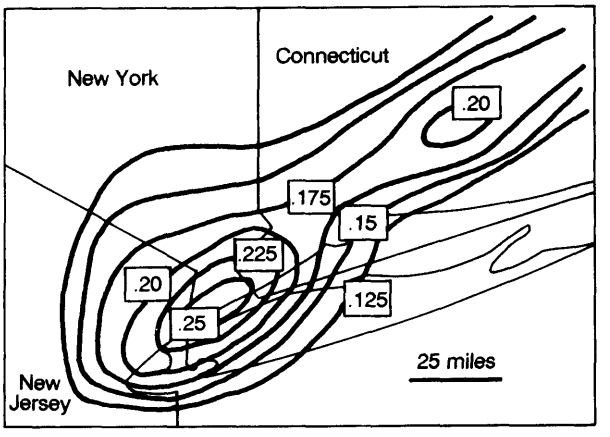
Figure 3-7-Profile of Ozone Concentrations as They Change Over the Day at a Single Monitoring Site



The profile is typical of a suburban area downwind of a strong source area or city center.

SOURCE: Adapted from U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office, *Air Quality Criteria for Ozone and Other Photochemical Oxidants, vol. I (Washington,* DC: August 1966).

Figure 3-8—Contour Map of the Variation in Daily Peak Ozone Concentrations Predicted for the New York City Area



The map was prepared with results from an urban-scale ozone model, with meteorological conditions and emissions of July 16, 1980. As shown, ozone concentrations typically vary smoothly over a large area and do not show localized peaks. Ozone concentrations in parts per million.

SOURCE: Adapted from S.T. Rae, *Application of the Urban Airshed Model to the New York Metropolitan Area,* EPA 450/4-67-011 (Research Triangle Park, NC: U.S. Environmental Protection Agency, May 1987).

outdoor concentrations [76]. 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 [77].

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.[19]

As discussed in the section on health effects, clinical and epidemiologic 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 5 to 20 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 [92] and the data presented in figures 3-4 to 3-6, approximately 130 million people live in areas where ozone concentrations are expected to equal or exceed 0.12 ppm at least 1 hour per year.[20] Eighty-six million people live in areas where concentrations reach at least 0.14 ppm at least 1 hour per year; *25* million where

[19] A 1984 Gallup survey indicated that about 18 percent of adult Americans jog at least once per week [31]. Four out of every 1,000 adults ((). 4 percent) *run* more than 6 miles at least once per week [32].

[20] EPA defines nonattainment areas as areas where ozone concentrations equal or exceed (). 125 ppm at least 1 hour per year. Over 12 million people live in areas that are included in Figure 24, with the 0.12 ppm cutoff, but are excluded with EPA's 0.125 ppm cutoff.

Table 3-3-Estimated Exposures to Ozone Concentrations Above 0.12 ppm

Exercise level	People exposed per year	Percent of people living in areas exceeding 0,12 ppm	Hours of exposure per person exposed per year
Nationwide:			
Low	34 million	26%	8.8 hr
Moderate	21 million	16%	8.6 hr
Heavy	13 million	10%	5.7 hr
Very heavy	80 thousand	<0.1%	4.1 hr
Nationwide except Los Angeles:			
Low	**24 million**	20%	3.7hr
Moderate	**16 million**	13%	**4.6 hr**
Heavy	**IO million**	8%	**3.2 hr**
Very heavy	**60 thousand**	<0.1%	**2.1** hr
LosAngeles:			
Low	9.7 miilion	97%	**22 hr**
Moderate	4.6 million	46%	24 hr
Heavy	3.0 miilion	30%	14 hr
Very heavy	20 thousand	0.2%	IO hr

These estimates are based on hourly ozone data for the period 1983-85, and take into account people's activity patterns (e.g. time commuting, time indoors at work, etc.) and location throughout the day. The estimates are broken down according to people's exercise levels. Those exercising at the higher levels are most apt to be susceptible to health impacts. The total number of people residing in areas where ozone concentrations exceeded 0.12 ppm at least 1 hour per year, on average during 1983-85, was approximately 130 million.

SOURCE: OTA, using data from T.R McCurdy, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, *National Estimates of Exposure to Ozone Under Alternative National Standards* (Research Triangle Park, NC: December 1986).

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 (62 million) live in areas where concentrations reach 0.12 ppm 6 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 10 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 3-3 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 3-3 were calculated by combining EPA's exposure estimates [69] with the number of people that we have estimated live in areas where ozone concentrations are expected to exceed 0.12 ppm more than 1 hour per year.

The numbers given in table 3-3 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 estimated to be exposed each year at low exercise levels; 21 million at moderate exercise levels; 13 million at heavy exercise levels; and approximately 80,000 during very heavy exercise.[21] In each exercise category, these numbers represent about 25 percent of the people who achieve that exercise level some time during the year. Thus, since everyone is exercising at a low level at some time (e.g., when they are walking leisurely on a flat surface), about 25 percent of the people who live in

[21] The corresponding ventilation rates for these exercise levels are: low = ≥ 25 liters/minute (I/rein); moderate = 26 to 43 l/rein; high = 44 to 63 l/min; and very high = ≥ 64 t/rein [68].

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 low or moderate exercise levels. Fewer people are exposed at the highest exercise level, because relatively few people engage in very heavy exercise. Of the nationwide totals, 9.7 million, 4.6 million, 3 million, and 20,000 of the people exposed at low, moderate, heavy, and very heavy 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 moderate exercise levels are estimated to be exposed an average of about 9 hours per year; people exposed at heavy levels an average of 5.7 hours per year; and people exposed at very heavy exercise levels an average of 4.1 hours per year. However, the national averages mask considerable variability among 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 low, moderate, heavy, and very heavy 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 low, moderate, heavy, and very heavy exercise levels.

EXTRAPOLATION OF EFFECTS OF MULTIPLE-HOUR EXPOSURES TO OZONE

A carpenter spends the day hauling lumber and hammering away at the frame of a two-story house. A group of elementary school children are packed off to spend the summer at camp, where they will swim, hike, and compete in games of basketball, tennis, and the like. A high school cross-country track team begins practicing in August for their upcoming fall season, engaging in vigorous, daily routines of sprinting and long runs around the school track. Are these people at risk for adverse health effects from exposure to ozone? What conditions would make them at risk? Could their lung function be harmed by exercising outdoors when the ozone level is high? How many people like them might be harmed by working or playing in ozone contaminated environments?

In this next section we take a closer look at the effects of ozone on people performing various activities, examining the role that exercise and ozone concentration play in the time it takes for an "adverse" health effect to occur.

While data exist on the lung function effects expected from exposure to ozone above the current l-hour standard of 0.12 ppm, there is little information available on effects for longer periods of exposure and at lower ozone levels. Information about the health effects that might be experienced under such conditions is needed to assist scientists and policy makers in determining the adequacy of the current standard for protecting public health and in determining how quickly areas should be required to meet the ozone standard.

To begin to address these issues, an OTA contractor[22] developed a model to extrapolate the results of 1- to 2-hour exposure studies to conditions of multiple-hour exposures (up to 8 hours) at ozone concentrations typically measured during summertime in many U.S. cities (0.08 to 0.16 ppm). This extrapolation model predicts the average changes in lung function (measured by FEV_1 and FVC)[23] for people exercising at different intensities under these conditions. Data were selected nom a number of exposure studies[24] and applied to a regression model that expresses the *dose* of ozone that an exposed individual would receive, and then predicts a response in terms of lung function changes. Dose is assumed to be affected by: the ozone concentration in the air one is breathing, the effect of exercise intensity on one's inhalation rate, how long one is exposed to ozone, and how much of the exposure

[22]This section is based on an OTA contractor report by Lawrence J. Folinsbee [25b].

[23]Forced expiratory volume in 1 second (FEV_1) and forced vital capacity (FVC) are common measures of lung function that can be affected by exposure to ozone. FEV_1 is the maximum amount of air that can be exhaled from the lungs in 1 second; FVC is the maximum amount of air that can be exhaled from the lungs after taking a full deep breath.

[24]Model parameters were estimated from exposure studies conducted at low ozone concentrations. The primary criterion for selecting studies to include was that the exposures occurred at ozone concentrations that were within or close to the ozone concentration region of interest (i.e., <0.20 ppm).

period one is exercising. This model can be used to predict changes in lung function in exercising populations.[25] It is important to note that the predicted losses in lung function in the model are *group mean* changes, and that some *individuals* may experience FEV_1 or FVC losses greater or less than the average changes for the whole group.[26]

Given the limited time available to develop this model and the number of simplifying assumptions that had to be made,[27] the model results must be considered approximate. When more data applicable to multi-hour exposures at low ozone concentrations become available, such a model can be improved.

Two alternative activity scenarios illustrate how exercise can affect lung function, given various *ozone* concentrations. The first scenario—representing a moderate level of exercise—corresponds to activity intensities and patterns of typical construction workers, and children playing outdoors on a summer day. The second scenario corresponds to a more vigorous activity level, for example, people engaged in active sports or biking.[28] Figure 3-9 illustrates how the level of outdoor activity could affect the time it takes before an ''adverse" change in lung function might occur, given average ozone concentrations of 0.08 to 0.16 ppm.[29] With respect to "adverse" effects, we assumed that *most* scientists would not consider *group mean* decreases in FEV_1 of less than 5 percent to be an adverse effect; some scientists would call group mean decreases of 5 to 10 percent an adverse effect; and *most* scientists would call decreases of 10 percent or greater an adverse effect. The lower line on these figures represents a 5 percent cutoff; the upper line represents a 10 percent cutoff. These ranges apply to *healthy* people rather than to persons with preexisting respiratory disease. This definition of ''adverse" is consistent with the definition presented by EPA in its most recent review of the ozone standard.[30]

Figure 3-9 shows that as one undertakes more vigorous exercise, fewer hours of ozone exposure are required to produce an adverse effect on lung function, given the same ozone concentration. For example, in the graph to the *right* in figure 3-9, when average ozone concentrations are 0.14 ppm, a 5-percent loss of lung function would be anticipated

[25]For example, under Folinsbee's model, the % change in FEV_1 = 0_3 X SVE X ExpDur X ActRat X -0.0367, where O_3 is ozone concentration, SVE is specific ventilation (ratio of ventilation to vital capacity, or L/min/L of forced vital capacity), ExpDur is exposure duration, and ActRat is activity ratio (fraction of total exposure duration during which individual is exercising). The last number is the slope of the relationship between dose rate and rate of change of FEV_1. See Table 4 in [25b] for the complete range of predicted lung function changes.

[26]For example, i, a study b, Folinsbee [28], an average decrease of 13 percent in FEV_1 was experienced by a group exposed to 0.12 ppm Of OZOne over a 6.6-hour period; individual variability ranged from losses as high as 47.6 percent to positive changes of 3.5 percent.

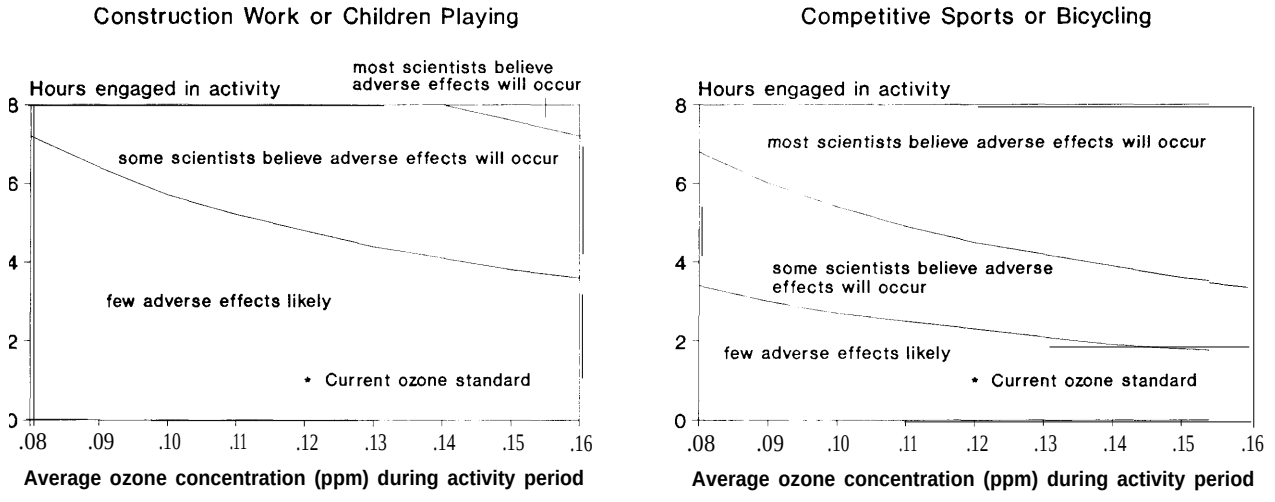
[27]In order to make generalized extrapolations across data ranges for which minimal information exists, the following assumptions were made: 1) changes in lung function area linear function of exposure duration; 2) the influence of ozone concentration on function changes over the concentration range of interest is approximately linear; 3) there is no threshold concentration for response; 4) the influence of ozone at low concentrations on people breathing at rest cannot be demonstrated and thus only the ozone exposure accompanied by exercise is relevant; 5) the effects of ozone on the lungs are a function of the size of the lungs, and more specifically, the surface area of the lungs affected by ozone; and 6) within the concentration range of interest, ozone's effects are proportional to the estimated dose of ozone breathed during exercise.

[28]For both of these exercise scenarios, we used the following formula, based on Folinsbee's analysis, to determine the hours to reach an adverse effect: number of hrs to reach adverse effect= 100/60 X FEV_1decr/(O_3 X SVE X ActRat X -0.0367). At the moderate exercise level, we assigned an SVE, or specific ventilation rate, of "6" (about 33 liters/min) to be consistent with EPA's definition of a typical ventilation rate experienced at a moderate (or "medium") level of exercise (26to434rnin). An activity ratio of 0.66 was applied, because it was assumed that a typical construction worker would be working about 40 minutes of every hour, with 20 minutes of rest time interspersed. At the heavy exercise level, the SVE was '' 10" (about 55 l/rein), which is in line with EPA's definition of heavy exercise (44 to 63 l/rein). At heavy exercise levels, we assumed an activity ratio of 0.84, or 10 minutes of rest for every 50 minutes of exercise.

[29]It is important t. point out that these ozone levels are not peak concentrations, but represent what an average ozone level would be during the period of exposure.

[30]EPA staff recommends the following definition for an ''adverse" response [98]: *Individual* lung function loss of 10 to ***20*** percent for Up to 6 hews. *with* accompanying symptoms and curtailment of some activity. We use *group mean* lung function losses of 5 to 10 percent as a surrogate for *individual* losses on the order of 10 to 20 percent, i.e., the most sensitive members of the population. Studies have shown that when groups experience losses of between about 5 percent and 10 percent, a number of individuals within these groups maybe experiencing lung function losses up to two and three times this much. Using data presented in a risk assessment prepared for EPA [37], we compared group mean lung function changes to the EPA estimates of the percent of the population with lung function losses of greater than 10 and 20 percent. A 5 percent group mean decrease in FEV_1, which in our analysis we consider a response that ''some scientists would consider adverse", is roughly equivalent to 10 to 20 percent of the population (the most sensitive individuals) experiencing equal to or greater than a 10 percent decrease in FEV_1, according to EPA's risk assesment. Furthermore, a 10 percent group mean loss in FEV_1, which we describe as a response that 'most scientists would consider adverse", is about the same as 20 percent of the population (most sensitive members) experiencing lung function losses of equal to or greater than 20 percent,

Figure 3-9—Likelihood of Adverse Effects From Ozone While Exercising



The likelihood of experiencing adverse effects depends on 1) the ozone concentration, 2) the vigorousness of the activity, and 3) the number of hours engaged in that **activity. The figure on the left shows the number of hours to reach an adverse effect under moderate exercise conditions (e.g., construction** work or children playing). The figure on the right shows that fewer hours are needed under heavy exercise (e.g., competitive sports or bicycling). The current one-hour ozone standard is shown for comparison.

SOURCE: OTA, based on work for OTA by Lawrence J. Folinsbee, Environmental Monitoring and Services.

for groups engaged in strenuous exercise-such as biking or playing tennis—for 2 hours. At the same ozone concentration (0.14 ppm), a 10 percent lung function loss would be expected to occur after about 4 hours of strenuous exercise. Looking at a more moderate level of exercise, e.g. construction work as shown in the graph to the *left* in figure 3-9, we see that it takes about twice as much time-or 4 hours-to reach lung function losses of 5 percent at 0.14 ppm than with more rigorous exercise. On a typical summer day, one might expect children to be outside playing for about 4 hours, the time it takes to experience what some scientists believe would be adverse lung function effects (5 percent average decreases). After about 8 hours-not an unlikely workday for construction workers-people exercising at a moderate level when ozone concentrations are 0.14 ppm may, on the average, have as great as 10 percent lung function losses.

Note also on these two figures that the level where adverse effects appear likely to occur (e.g., where some or most scientists become concerned about adverse effects) gets closer to the NAAQS for ozone as exercise intensity increases from moderate to heavy levels. In other words, the ozone standard is less protective of people who choose to work outdoors or exercise more vigorously than people who lead less active lifestyles.

Not only is it important to realize how one's *activity level* might affect the amount of time it would take to produce an ''adverse" health effect, but also, *within* a given activity level, how ***ozone concentration affects the*** number of hours until adverse effects occur. At a moderate level of activity-construction work or the equivalent in outdoor exercise-we see that at 0.16 ppm of ozone, it takes about 3 ½ hours to produce FEV_1 decreases of 5 percent; at 0.13 ppm it takes about 4 1/2 hours; and at 0.10 ppm, approximately 5 1/2 hours. For a group of construction workers to average 10 percent decreases in lung function-the point at which few scientists disagree that the effects are adverse-it would take about 7 hours at 0.16 ppm and 9 hours at 0.13 ppm.

Table 3-4 indicates the population *residing* in areas where ozone levels equal or exceed average concentrations ranging from 0.08 to 0.16 ppm for

between 1 to 8 hours, on at least 1 day per year.[31] Table 3-5 presents the number of cities and population living in those cities where the ozone concentration exceeds 0.14 ppm for 2-,4-, and 8-hour periods for at least 1,2,5, and 10 days a year. The estimates given in tables 3-4 and 3-5 represent "potentially" exposed people—they would not actually have been exposed unless they were outside when the recorded ozone episodes occurred. (And they would probably not be affected by their exposure unless engaged in some kind of exercise.) While we do not have precise data on the number of exercising people *actually* exposed, we can make some general statements about the percentage of individuals living in these areas who may be exposed to these ozone conditions while working or exercising outdoors.

First of all, we know that a significant portion of the U.S. population is living in areas where ozone levels are within the ranges discussed so far. For example, table 3-5 shows that 45 million people live in 24 cities where the 4-hour average ozone concentrations exceed 0.14 ppm for at least once per year. About one-third of this population lives in 14 cities where these conditions occur at least five times a year; one-quarter live in areas where these conditions occur at least 10 times a year. About 18 million people live in 10 cities in which 8-hour average ozone levels exceed 0.14 ppm at least once per year.

Second, we know that within these *potentially* exposed populations, some portion will *actually* be exposed because of their outdoor activity. One subpopulation that has been defined as potentially ''at risk" for adverse effects from exposure to ozone is peopIe who work outdoors. Looking at construction workers, we see that they could experience adverse health effects under conditions found in many areas around the United States. As mentioned above, a moderate level of exercise like construction work in areas where 7-hour average ozone concentrations exceeded 0.16 ppm would produce what most scientists would consider to be an adverse effect (10 percent group mean decrease in FEV_1). We estimate that a few percent, or about 0.4 million of the 18 million people residing in areas in which these ozone conditions occur, would be exposed at least once per year while engaged in construction work.[32] Average lung function decreases of 5 percent—a change of concern to some scientists-might be seen in people exercising at moderate levels when ozone concentrations exceed 0.14 ppm for four hours. We estimate that a few percent of the 45 million people living in the 24 cities where these conditions occur-or about one million people—would be exposed at least once per year while engaged in construction work. About one-third of this population would be exposed at least five times a year in the six cities where these conditions occur.

Table 34-Population Residing in Areas Where the Indicated Concentration is Exceeded at least One Period Per Year, for Each Averaging Time

Averaging time (hours)	Population (millions) in areas exceeding concentrations of .08 ppm	.10 ppm	.12 ppm	.14 ppm	.16 ppm	.18 ppm	.20 ppm
1	160	150	110	65	41	21	17
2	160	140	98	54	22	18	15
4	160	130	75	45	18	16	12
6	160	120	60	23	17	12	9.7
8	150	93	45	18	12	9.7	1.8

SOURCE: OTA, treed on EPA Storage and Retrieval of Aerometric Data (SARDAD) 1983-85 monitoring data.

Active children exposed to ozone under comparable conditions might also experience adverse lung function effects. For example, kids exercising moderately (at approximately the same relative intensity as construction workers) for 4 hours when the ozone concentration is 0.14 ppm would be expected to average about a 5-percent decrease in their lung function. As mentioned above, this is the point at which some scientists become concerned about adverse health effects. Since children between 5 and 14 years of age constitute about 14 percent of the total U.S. population, [93] about 6 million kids 1ive in the 24 cities where ozone concentrations exceed 0.14 ppm at least once a year. About 2 million children live in the six cities where similar ozone levels occur at least five times a year.

Finally, it is important to reemphasize that under *any* of the above-mentioned scenarios, some portion of the population will be more sensitive to ozone

[31] Data are for the period 1983-85.

[32] This estimate (''a few percent") of adversely affected construction workers is based on the following information. First, an estimated 5 percent of adult men work outdoors full-time, and another 10 percent work outdoors part of the time [77]. We assume that a smaller fraction of women work outdoors and that about 1.5 percent of the U.S. population (mostly men) are employed in nonsupervisory construction jobs [92].

Table 3-5-Number of Cities Where Ozone Concentrations Exceed 0.14 ppm for 2-,4-, and 8-hr Periods for the Specified Number of Days Per Year, on Average, and Population Residing in Those Cities

	2-hr		4-hr		8-hr	
Days per year	Population (millions)	Cities	Population (millions)	Cities	Population (millions)	Cities
>1	54	33	45	24	18	10
>2	**34**	**21**	**21**	**12**	**13**	**5**
>5	**19**	**9**	**14**		**10**	**2**
>10	**13**	**4**	**12**	**3**	**10**	**2**

SOURCE: OTA, baaed on EPA SARDAD 1983-85 monitoring data.

than indicated by the group mean responses we have considered. EPA has labeled these people as "responders," and estimates that from 5 to 20 percent of the healthy population in the United States are in this more sensitive group. Therefore, while Folinsbee's model has allowed us to predict *group* mean lung function decreases, about 5 to 20 percent of the exposed population discussed above would experience significantly larger lung function changes under the ozone exposure conditions considered here.

SELECTED NATIONWIDE HEALTH BENEFITS FROM CONTROLLING OZONE

This section looks at expected nationwide reductions in some types of health effects from reducing ozone levels in all nonattainment areas.[33] Estimates are made of the number of incidents of various respiratory symptoms and days when ozone exposure may limit a person's activity, under three scenarios: 1) current ozone levels, 2) ozone levels after all reasonably available volatile organic compound (VOC) control methods are applied, and 3) ozone levels assuming that the standard is attained in all areas. The "benefit" of control is the *difference* between the nationwide health effects from current ozone levels and the health effects after control. A rough approximation of the economic value of these health improvements is also given.

We estimate that if ozone concentrations were lowered enough to meet the standard in all areas, several hundred million incidents of respiratory symptoms, such as coughing or pain on deep breathing, might be avoided each year. Among the approximately 115 million people 1iving in nonattainment areas, some in the worst areas would experience dozens fewer incidents of respiratory symptoms each year while many in other *areas* would experience no change. About 8 million to 50 million "restricted activity days" might also be eliminated. These are days when someone feels ill enough to limit a day's worth of activities-disrupting most of the day's activities, but generally not spending the day in bed or staying home from work.

By asking people what they would be willing to pay to avoid a day of coughing or a day of restricted activity, it is also possible to get a rough feel for the economic value to individuals of the health improvements listed above. As will be discussed below, the uncertainties are quite large due to the many assumptions that must be made, but about $0.5 billion to $4 billion per year is a reasonable range for the portion of health benefits that we were able to evaluate. Under some assumptions, benefits are less than $0.1 billion per year and under others, up to about $10 billion per year.

Keep in mind, however, that we could only quantitatively estimate some of the benefits. We *did* estimate such acute health effects as the number of times per year when people experience respiratory symptoms, such as coughing or pain on deep breathing; days when someone's daily activities are restricted; and days with asthma attacks. We *did not* estimate benefits associated with changes in lung function because we had no method for assigning a value to this effect. (We did, however, include shortness of breath, a symptom of lung function changes perceptible by people without medical

[33] This section is based on results presented in an OTA contractor report by Alan J. Krupnick and Raymond J. Kopp [49].

measuring devices.) And, even though many health professionals are concerned that repeated exposure to ozone over a lifetime may result in premature aging of the lungs, along with the possibility of permanent lung impairment, current understanding does not allow us to quantity the lowered risk of chronic effects.

We also *did not* include health benefits of lowering VOC emissions that are not related to lowered ozone concentrations. Probably the most significant omission in this regard is that some VOCs are carcinogenic. A preliminary EPA assessment estimated that nationwide, about 2,000 cancers per year might result from exposure to toxic air pollutants [97]. About half of the risk from the 20 chemicals considered in the study, about 1,000 cancers per year, comes from VOCs. The specific chemicals or groups of chemicals posing the greatest aggregate risk include benzene, butadiene, formaldehyde, gasoline vapors, and emissions from hazardous waste treatment, storage, and disposal facilities. Another EPA study concluded that about 250 to 400 deaths per year might be due to exposure to three VOCs from mobile sources: benzene, butadiene, and formaldehyde [12,67,74]. These estimates of cancer risks from exposure to VOC should be regarded as rough estimates due to uncertainties about how carcinogenic these chemicals truly are, and to the simplistic method of estimating human exposure. Nonetheless, reducing VOC emissions to lower ozone concentrations will also lead to lower risks of cancer from exposure to these chemicals.

How *the Estimates Are Calculated*

To calculate the aggregate benefits from lowering ozone concentrations in nonattainment areas, several steps are followed. First "concentration-response" relationships are developed, that is, equations that describe the "response" (e.g., cough incidents or days of restricted activity days) from exposure to ozone at different concentrations. Next data are obtained on ozone levels in nonattainment areas. For this analysis we obtained 3 years of data on daily maximum hourly ozone concentrations measured at each of several hundred monitors in EPA's nationwide data base. Using the concentration-response relationships, we then calculate the effects of ozone on the population of each nonattainment county from the concentrations measured each day during the ozone seasons of 1983 through 1985.[34]

Then, using a simplified air quality model (called EKMA and discussed in chapter 4), we estimated ozone concentrations after controls have been adopted. We modeled two control scenarios: 1) air quality levels after sufficient controls have been adopted to meet the ozone standard in all areas, and 2) air quality levels after VOC emissions in nonattainment areas have been lowered by 35 percent—controls about equivalent to adopting all currently available control measures. (The emissions control aspect of this scenario is discussed in chapter 6.) We then calculate the effects of ozone—again county-by-county and day-by-day—at these lower ozone concentrations. The *diference* between the before and after estimates (either avoided episodes of respiratory symptoms or avoided days of selected adverse consequences) are displayed in a series of tables.

If desired, one can take the aggregate estimates of effects and assign dollar values to the avoided symptom incidents and restricted activity days. These values are taken from interviews where people are asked what they would be willing to pay to avoid such effects as a day of coughing or an asthma attack. As one might imagine, the range of responses is quite large, thus the dollar values must be treated as more uncertain than the estimates of adverse effects avoided.

Two types of studies are used to estimate the concentration-response relationships: clinical and epidemiologic. In clinical studies, people are exposed in laboratories to carefully monitored ozone concentrations, typically while exercising on a stationary cycle or treadmill. Researchers measure changes in lung function as well as ask the volunteers to describe any respiratory symptoms they may be experiencing. In the epidemiologic studies used in this analysis, volunteers fill out daily or biweekly diaries of their health status. These are later compared to concentrations measured at nearby ozone monitors, after controlling for many other factors such as age, sex, smoking status, occupation, daily temperatures, and concentrations of other air pollutants.

[34] For counties with more than one ozone monitor, we averaged the readings from all monitors. For counties with no ozone monitors, we averaged the readings from all monitors operating within the metropolitan area.

Each type of study has advantages and disadvantages for estimating the health benefits from lowering ozone concentrations. The clinical studies provide excellent data on how individuals respond to very specific exposure conditions (typically 1 to 2 hours of exposure while exercising vigorously). However, one is left with the difficult task of extrapolating the effects of ozone under typical daily routines-adults exercising and walking to work, children playing, and so on.

The epidemiologic studies directly produce data on effects of interest—respiratory symptoms and restricted activity—while engaged in typical day-to-day activities, but the relationship to ozone exposure is more difficult to establish. First, the effects data must be statistically compared to ozone levels that are often only rough indicators of actual exposure. Second, because several other factors that affect respiratory health must be considered simultaneously (e.g., smoking status, temperature) it is quite difficult to isolate the effect of ozone alone.

Keep in mind that none of the studies we used estimate the risks of chronic effects from longer term exposure to ozone. Whether there are chronic effects from exposure over many years and, if so, the magnitude, is still uncertain.

Selected Health Benefits of Lowered Ozone Concentrations

As mentioned above, we used two types of studies to estimate the effects from exposure to ozone: clinical studies and epidemiologic studies. Using a clinical study, we estimated three types of symptoms: the number of incidents of coughing, shortness of breath, and chest discomfort (i.e., pain on deep breathing) [70]. From the epidemiologic studies, we estimated the number of days when respiratory illness restricted normal activities [78], days with any type of respiratory-related symptom (e.g., coughing, wheezing, chest discomfort, sore throat, etc.) [49], and days of asthma attacks [38].

Table 3-6 presents our estimates of the total number of incidents of respiratory symptoms avoided from adopting the two control scenarios mentioned above. Two sets of estimates based on clinical studies are shown. The lower estimates assume that the only people who might be affected by ozone are those who engage in heavy exercise outdoors. The higher estimates assume that people exercising at light and moderate exercise levels can also be affected by ozone, but with proportionally lower effects at the lower exercise levels .[35] The time spent outdoors at each exercise level is estimated from EPA data [76].

Taking into consideration uncertainty about who will be affected, we estimate that meeting the standard in all areas would eliminate about 110 to 350 million cough incidents each year, and about 60 to 200 million incidents each of shortness of breath and chest pain. Our scenario that reduces VOC emissions by 35 percent would eliminate about 40 to 130 million coughing episodes per year and about 20 to 70 million incidents each of shortness of breath and chest pain. As shown in the table, the range is even greater when one considers possible errors due statistical estimation of the concentration-response function from clinical data.

These health benefits may be easier to conceptualize when expressed on a per-person basis, or more accurately, the type of response one might expect within a group of 100 people. Among every 100 people, averaged across all nonattainment areas, meeting the standard would eliminate about 100 to 300 cough episodes per year. The improvement, averaging about one to three fewer cough episodes per person per year, can be compared to an average of about eight cough days per person per year [24]. The number of symptom episodes avoided would vary from individual to individual, of course, for several reasons. First, not everyone is active outdoors. Second, among every 100 people, about 5 to 20 are much more sensitive than the average for unknown reasons. In addition, the average improvement varies considerably from nonattainment area to nonattainment area, depending on the severity of the ozone problem.

In table 3-7, we report the per-person improvement in areas by a measure of peak ozone concentra-

[35] Many clinical studies have shown adverse effects from ozone under heavy and very heavy exercise conditions. At least one has shown effects under moderate exercise over multi-hour time periods, supporting the hypothesis that the effects of ozone are due as much to "dose' '-the total amount of ozone inhaled-as to concentration and exercise level. Limiting our analysis to only heavy exercisers is a fairly conservative assumption; extrapolating effects to all exercise levels (including light) is a reasonable extrapolation, but no clinical data exist to support it.

Table 3-6-Avoided Episodes of Respiratory Symptoms (millions of episodes per year)

	From meeting the standard		From a 350/. VOC reduction	
	Midpoint	Range	Midpoint	Range
Cough:				
Affecting heavy exercisers only	110	(78-130)	39	(29-49)
Affecting all exercisers	350	(250-440)	130	(100-160)
Shortness of breath:				
Affecting heavy exercisers only	61	(43-77)	22	(16-27)
Affecting all exercisers	200	(140-250)	72	(51-89)
Pain on deep breathing:				
Affecting heavy exercisers only	60	(42-78)	22	(15-28)
Affecting all exercisers	200	(140-260)	72	(51-93)

SOURCE: OTA, modified from A.J. Krupnick and R.J. Kopp, The Health and Agricultural *Benefits of Reductions in Ambient Ozone in the United States*, contractor report prepared for the Office of Technology Assessment, June 1988.

Table 3-7-Avoided Episodes of Respiratory Symptoms (episodes per 100 people per year)

	From meeting the standard		From a 35% VOC reduction	
	Heavy exercisers only	All exercisers	Heavy exercisers only	All exercisers
Cough:				
All area average	96	310	36	120
Peak <0.14 ppm	17	55	12	39
Peak 0.14 to 0.18	63	210	24	79
Peak 0.18 to 0.27	150	490	50	170
Peak >0.27	430	1410	140	470
Shortness of breath:				
All area average	55	180	20	65
Peak <0.14 ppm	10	33	7	24
Peak 0.14 to 0.18	38	120	14	46
Peak 0.18 to 0.27	88	290	29	95
Peak >0.27	240	780	74	240
Pain on deep breathing:				
All area average	54	189	20	65
Peak <0.14 ppm	10	32	7	23
Peak 0.14 to 0.18	36	120	14	45
Peak 0.18 to 0.27	85	280	28	93
Peak >0.27	**240**	**790**	**79**	**260**

SOURCE: OTA, modified from A.J. Krupnick and R.J. Kopp, The *Health and Agricultural Benefits of Reductions in Ambient Ozone in the Unifed States*, contractor report prepared for the Office of Technology Assessment, June 1988.

tions, We use the fourth highest concentration observed over the 3-year period in *each county (i.e.,* the equivalent of a county-level "design value") rather than characterize an entire metropolitan area by a single concentration. In those areas where peak ozone concentrations are close to the standard (between 0.12 and 0.14 ppm), meeting the standard would eliminate about 15 to 55 cough episodes per year among every 100 people. In those areas with the worst ozone problems, meeting the standard would eliminate 400 to 1,400 cough episodes per year among every 100 people.

Table 3-8 presents the benefits estimated using *the* epidemiologic studies. Meeting the standard in all areas would eliminate about 25 million days per year of restricted activity and about 50 million days with respiratory-related symptoms. About 2 million days of asthma attacks would also be eliminated. Our scenario that reduces VOC emissions by 35 percent would eliminate about 8 million restricted activity days per year, about 18 million symptom days, and about 0.6 million asthma attack days.

Note that in the tables we have disaggregate restricted activity days and symptom days into

Table 3-8-Avoided Days of Adverse **Consequences (millions of** days per year)

	From meeting the standard		From a 35% VOC reduction	
	Midpoint	Range	Midpoint	Range
Restricted activity days:				
Adults 18		(5.3-34)	5.9	(1.9-10)
Children 7.7		(2.3-15)	2.5	(0.8-4.4)
Days with any respiratory symptom:				
Adults 34		(22-46)	12.6	(8.2-17)
Children 15		(10-20)	5.4	(3.5-73)
Asthma-attack days:				
All 1.9		**(1.0-3.0)**	**0.6**	**(0.4-09)**

SOURCE: OTA. modifiedfrom A.J. Krupnick and R.J. Kopp. *The Health and Agricultural Benefits of Reductions in Ambient Ozone in the United States*, contractor report prepared for the Office of Technology Assessment, June 1988.

improvements among adults and improvements among children. The epidemiologic studies that we relied on found effects in adults only. his might be because children are less likely to perceive symptoms than adults, or might only indicate that children are less likely to report symptoms. The estimates given in the text assume that children and adults are affected in similar ways.

In table 3-9, we once again express these improvements on a per-person basis. Among every 100 people (adults and children), averaged across all nonattainment areas, meeting the standard would eliminate about 45 days with respiratory symptoms each year. About half (25 days) would also be days of restricted activity. Among every 100 asthmatics, meeting the standard would eliminate about 60 asthma-attack days each year. Improvements from lowering VOC emissions by 35 percent would average about 8 fewer restricted activity days and 15 fewer days with respiratory symptoms among every 100 people. Among every 100 asthmatics, we would expect to see 20 fewer asthma-attack days. These improvements can be compared to a current total of about 130 respiratory-related restricted activity days [95] and 800 cough days each year among every 100 people [24]. Among 100 asthmatics, one would expect about 1000 asthma-attack days each year [49]. Each of these totals is the number of days of poor respiratory health from all causes, not just air pollution.

Again, there is considerable variation from area to area. For example, in those areas where peak ozone concentrations are close to the standard (between 0.12 and 0.14 ppm), meeting the standard would eliminate about 8 days with respiratory symptoms each year, and 3 days of restricted activity, among every 100 people. In those areas with the worst ozone problems, meeting the standard would eliminate about 200 days with respiratory symptoms, and 120 days of restricted activity, among every 100 people.

Note that the epidemiologic studies predict lower benefits than the clinical studies, that is, the epidemiologic studies predict fewer health effects from exposure to ozone than the clinical studies. Several reasons are possible. First, people may be less likely to exercise outdoors on days with high ozone concentrations due to both the pollution and high temperatures. If so, one would expect that fewer people would actually be affected than the number predicted from laboratory studies. Second, there is considerable variation among similar types of studies. For example, EPA compares data from the McDonnell study (which we used) to a similar study by Kulle and concludes that the McDonnell study predicts about twice as many people would experience at least mild cough after exercising heavily for two hours at ozone concentrations in the range of 0.12 to 0.20 ppm [98]. There are too few epidemiologic studies to be able to get a feel for how variable they might be. All we can conclude is that the difference between the results predicted by the clinical and epidemiologic studies falls within the range of uncertainty of this type of analysis.

Assigning a Dollar Value to Health Improvements From Lowered Ozone Levels

Although it is extremely difficult to assign a dollar value to the health improvements described above, table 3-10 presents our best guesses, based on the limited information available in the economic literature. These are derived by simply multiplying our

Table 3-9-Avoided Days of Adverse Consequences (days per 100 people per year)

	From meeting the standard		From a 35% VOC reduction	
	Midpoint	Range	Midpoint	Range
Restricted activity days:				
All area average	23	(7-44)		(2-13)
Peak <0.14 ppm	3	(I-6)	2	
Peak 0.14 to 0.18	13	(4-23)	5	(2-8)
Peak 0.18 to 0.27	34	(11-61)	11	(4-19)
Peak >0.27	120	(32-240)	32	(lo-57)
Days with any respiratory symptom:				
All area average	44	(29-60)	16	(1 1-22)
Peak <0.14 ppm	8	(5-1 1)	6	(4-8)
Peak 0.14 to 0.18	30	(19-40)	11	(7-1 5)
Peak 0.18 to 0.27	69	(45-94)	23	(15-31)
Peak >0.27	195	(130-260)	64	(42-87)
Asthma-attack days (per 100 asthmatics):				
All area average	58	(31-89)	19	(11 -28)
Peak <0.14 ppm		(5-1 3)	6	(4-9)
Peak 0.14 to 0.18	35	(20-51)	13	(7-1 8)
Peak 0.18 to 0.27	86	(48-130)	27	(15-39)
Peak >0.27	280	(145-449)	78	(44-1 10)

SOURCE: OTA, modified from A.J. Krupnick and R.J. Kopp, The *Health and Agricultural Benefits of Reductions in Ambient Ozone in the United States*, contractor report prepared for the Office of Technology Assessment, June 1968.

Table 3-10-Dollar Value of Selected Health Benefits (millions of dollars par year)

	From meeting the standard		From a 35% VOC reduction	
	Midpoint	Range	Midpoint	Range
Based on epidemiologic studies:	**550**	**(150-1 ,500)**	**190**	**(54-500)**
Based on clinical studies:				
Heavy exercisers only affected:				
Two episodes per symptom day	570	(200-1 ,400)	210	(75-520)
One episode per symptom day	1,100	(400-2,900)	420	(150-1,000)
All exercisers affected:				
Two episodes per symptom day	1,900	(670-4,700)	680	(250-1,700)
One episode per symptom day	***3,700***	***(1*** ,300-9,500)	1,400	(500-3,400)

SOURCE: OTA, modified from A.J. Krupnick and R.J. Kopp, *The Health and Agricultural Benefits of Reductions In Ambient Ozone in the United States*, contractor report prepared for the Office of Technology Assessment, June 1988.

estimates of the number of days of improved respiratory health by a dollar value for each day of adverse health effects. A range of dollar values are available from four studies where people were directly asked how much they would be willing to pay to avoid a day of respiratory symptoms [90,18,83,66].

The estimates in table 3-10 assume the following dollar value of each health effect: People would be willing to pay $5 to avoid each day of respiratory symptoms, with a range of $2.50 to $10. People would be willing to pay $18 to avoid each day of restricted activity, ranging from $11 to $30. And people would be willing to pay $25 to avoid each day of asthma attacks, with a range of $9 to $41. These were chosen as reasonably representative estimates of the "typical" responses found in the available studies, but keep in mind that the range of *individual* responses in the studies was enormous.

For example, in one of the studies [18], the arithmetic average response for the value of a cough day was about $11. However, half the survey respondents replied $1 or lower. For shortness of breath, the average response was about $8, but over half of the respondents replied that they would be willing to pay nothing. In addition, these average values do not include very high responses (e.g., one respondent valued a cough day at $10,000).

With these limitations in mind, let us turn to table 3-10. From epidemiologic studies, we were able to quantify the following health benefits from lowering

ozone concentrations: avoided days with respiratory symptoms, avoided days of restricted activity, and avoided days of asthma attack. We estimate that the economic value of these health improvements from meeting the standard would be about $550 million per year, ranging from about $150 million to $1.5 billion per year.[36] The economic value of these health improvements from a 35-percent reduction in VOC emissions would be about $190 million per year, ranging up to about $500 million per year.

From the clinical studies, we are able to estimate the number of avoided episodes of three types of respiratory symptoms: coughing, shortness of breath, and pain on deep breathing. Dollar benefits based on these studies range from values about equal to those stated above, to several times as much.

Our "best" estimates from the clinical studies of the economic value of the respiratory symptoms avoided from meeting the standard range from about $570 million to $3.7 billion per year. Under alternative reasonable assumptions, benefits range from about $200 million per year to about $9.5 billion per year. The higher estimate assumes: 1) a somewhat higher probability of experiencing respiratory symptoms from exposure to ozone, and 2) that people would be willing to pay $10 to avoid a day of respiratory symptoms.

Our "best" estimates of the value of respiratory symptoms avoided from a 35-percent reduction in VOC emissions range from about $210 million per year to about $1.4 billion per year. Under alternative assumptions, our estimates range from $50 million to $3.4 billion per year. Again, the higher estimate assumes a somewhat higher responsiveness to ozone and that people would be willing to pay $30 to avoid a day of restricted activity.

We have no way of judging which of the estimates presented in the table are more likely. Neither approach—using epidemiologic studies or clinical studies—seems clearly superior for this type of benefits assessment. For meeting the standard, about $0.5 billion to $3.7 billion per year is the range of our "best" estimates for the portion of health benefits that we were able to evaluate. From lowering VOC emissions by 35 percent, about $0.2 billion to $1.4 billion per year is a reasonable range for the portion of health benefits that we were able to evaluate. In either case, the benefits could reasonably be lower or about 2½ times greater, depending primarily on the value one assigns to a day of respiratory illness.

Again, none of the studies we used estimate the risks of chronic effects from longer term exposure to ozone. As discussed in an earlier section, many health professionals appear to be particularly concerned over the possibility of permanent damage to the lung from exposure to ozone over many years. We were not able to quantify these risks and include them in our benefits estimates.

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[36] In our analysis, we assume that on days of restricted activity, respiratory symptoms will also be felt. We therefore assume that the estimate that people are willing to pay to avoid a day of restricted activity includes the amount they are will to pay to avoid a day of respiratory symptoms. Our estimates of economic value have been adjusted to reflect this overlap.

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