
Health and Smog

No Cause for Alarm

**Kenneth Chilton and
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The regulatory reform mood in Congress may provide a window of opportunity to reexamine what is arguably the "flagship" of environmental regulation: the Clean Air Act. In particular, public discontent over centralized inspection of automobiles and other programs that could restrict driving freedoms or raise the cost of commuting is focusing the reformers' spotlight on the ozone provisions of the act.

Sensing the seriousness of the reform sentiment, environmentalists and their allies, such as the American Lung Association, have launched a counterattack, calling for a more restrictive air quality standard for ozone. To be fair, the debate over the need for a tighter standard, one that measures lower exposures over a longer period, has been taking place for some time. The last time the issue was visited by the EPA, in March 1993, the agency opted to leave the standard for ozone at 0.12 parts per million measured over a one-hour period.

This article summarizes the medical evidence to date on the health effects of elevated levels of ozone. It reviews the physiological responses to short-term (one- to three-hour) exposures consistent with current air quality standards and those responses produced by prolonged exposures (six

to eight hours). A concluding section provides an estimate of the relationship between benefits and costs of current efforts to reduce ozone levels and conjectures about the costs of meeting a revised, tighter, longer-exposure standard.

Health Effects of Ozone

For over two decades medical researchers have used clinical, field, and epidemiological studies to examine the human health effects of exposure to ozone (commonly called "smog"). Clinical studies are conducted in settings wherein the environment experienced by the human volunteers is under the researchers' control. Field and epidemiological research relies on a variety of sources—hospital and doctors' records, patient diaries, and interview reports—to determine if there is a relationship between a given health effect and elevated ozone levels.

Laboratory Exposures. Clinical studies conducted in the United States and elsewhere have examined both short-term (acute) effects and long-term (chronic) effects of elevated levels of ozone, but by far the greatest amount of research has been on acute responses. Until recently, studies of the health effects of short-term exposures to ozone have focused almost exclusively on one- to three-hour exposures.

Table 1 classifies the types of physiological responses associated with short-term exposures to elevated levels of ozone. Mild responses indicate lung function reductions of less than 10 per-

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Table 1

Classes of Individual Physiological Response to Acute Ozone Exposure

	Gradation of Response			
	Mild	Moderate	Severe	Incapacitating
Decreased lung function	5-10%	10-20%	20-40%	Greater than 40%
Duration of effect	Complete recovery in less than 30 minutes	Complete recovery in less than 6 hours	Complete recovery in 24 hours	Recovery in more than 24 hours
Symptoms	Mild to moderate cough	Mild to moderate cough, pain on deep inhaling, and shortness of breath	Repeated cough, moderate to severe pain on deep inhaling, and shortness of breath; breathing distress	Severe cough, pain on deep inhaling, and shortness of breath; obvious distress
Limitation of activity	None	Few individuals choose to discontinue activity	Some individuals choose to discontinue activity	Many individuals choose to discontinue activity

Source: Review of the National Air Quality Standard for Ozone Assessment of Scientific and Technical Information. Washington, D.C.: U.S. EPA, Office of Air Quality Planning and Standards, June 1989, pp. VII-53.

cent and a mild to moderate cough, for instance. Reductions of lung function of 10 to 20 percent are termed "moderate," and those affected may experience pain when inhaling deeply. Few individuals reduce activity due to such symptoms, however.

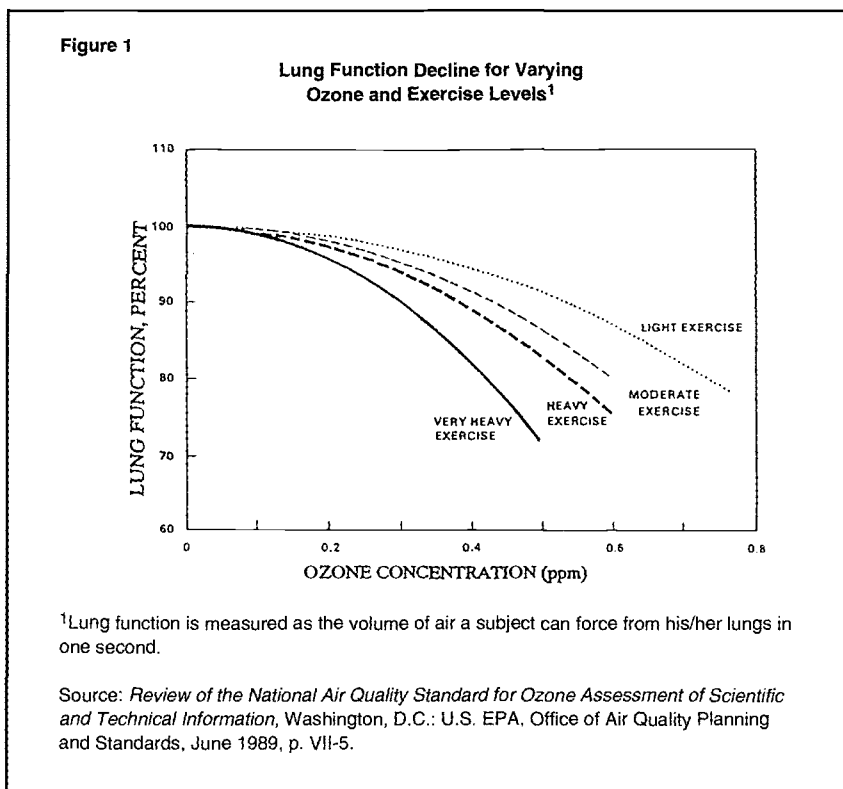
Not surprisingly, decreases in lung function and the severity of respiratory symptoms generally increase as individuals are exposed to higher concentrations of ozone. Such physiological changes are reversible, however. Individuals who experience even severe losses in breathing capacity (20 to 40 percent) when exposed to elevated ozone levels for one to three hours typically recover completely within 24 hours. The lung function of subjects experiencing "mild" responses returns to normal after less than 30 minutes.

Physiological responses to ozone are especially influenced by the frequency and depth of breathing, which increases as exercise workload increases. Based on results from 25 clinical studies, the Environmental Protection Agency (EPA) has estimated the relationship between reduced

breathing capacity and various combinations of exercise levels and ozone concentrations. Figure 1 (page 52) illustrates the EPA's findings for healthy, adult subjects (18 to 45 years old) after one to three hours of ozone exposure.

The composite results shown in Figure 1 demonstrate that during light exercise, typical subjects experience less than a 10 percent loss in lung function at ozone levels more than *four times* the current standard. Even during very heavy exercise, lung function is usually reduced by less than 10 percent at 0.24 parts per million (ppm)—twice the Clean Air Act standard.

The Clean Air Scientific Advisory Committee suggests that "responses identified as 'mild' for an individual's response to ozone [should] not be considered an adverse respiratory health effect." The committee concludes that such mild physiological responses probably would not be considered medically significant and should not interfere with the normal activity of most individuals. Indeed, mild decreases in pulmonary function may go unnoticed by the typical person, due to



the lungs' substantial reserve capacity.

There are considerable differences in the magnitude of individual responses to ozone exposure, however. Studies have indicated that as much as 20 percent of the population does not experience significant loss of lung function even at relatively high ozone concentrations. On the other hand, approximately 5 to 20 percent of the populations studied in clinical tests pooled by the EPA show a higher level of reactivity to ozone exposure and have therefore been dubbed "responders." Unless engaged in heavy exercise, however, even the most sensitive individuals normally do not experience statistically significant declines in lung function after one to three hours of exposure to around 0.12 ppm.

Clinical studies also show that younger individuals, especially children, are more responsive to elevated ozone levels. Their increased sensitivity is in part the result of the deep breathing patterns that accompany more physically active lifestyles. However, one must take care in interpreting the results of human exposure studies on children. A 1994 epidemiological study that examined the health diaries of 1,844 schoolchildren engaged in normal daily activities over a one-year period found no clear evidence that ozone

exposure led to greater incidence of lower respiratory symptoms. While ozone exposure was associated with cough incidence, other pollutants—most notably, inhalable particles—appeared to have greater effects on the children. Indeed, particulate matter concentration was associated with the incidence of all the respiratory symptoms examined in the study. If that study's results are confirmed by future research, efforts to safeguard children's health may be better focused on pollutants other than ozone.

Ozone's effects on persons suffering from lung disease (e.g., asthma or chronic obstructive lung disease) are also of particular concern. According to the available evidence, people with pre-exist-

ing lung diseases respond in a manner similar to healthy subjects when exposed to ozone concentrations typical of those found in most nonattainment areas, while exercising at moderate levels. Even when exposed to ozone concentrations of between 0.2 and 0.25 ppm, the responses of asthmatic and normal subjects are not generally different. That does not imply, however, that persons with already reduced lung function are not necessarily more at risk when experiencing the same *incremental* loss of breathing capacity as healthy subjects.

In short, the preponderance of clinical research on the effects of short-term exposures to elevated ozone levels indicates that the physiological responses experienced depend upon both ozone concentrations and exercise levels. Presently, the EPA considers an area's ambient ozone level to be safe as long as any one monitor does not register a one-hour average concentration of ozone exceeding 0.12 ppm on more than one day a year. The current standard should produce little or no discernible symptoms for the vast majority of people, even if they are heavily exercising during such a one-hour episode. A portion (5 to 20 percent) of otherwise healthy adults and children appear to be "responders,"

and could be forced to reduce their level of activity due to discomfort from exposure to ozone levels at or near the standard, if heavily exercising.

Prolonged Exposures. Most laboratory studies of human volunteers measuring the effects of prolonged exposures (six or more hours) to ozone levels at or below the current air quality standards are relatively new. The American Lung Association and numerous environmental organizations argue that the present standard is too lenient and that longer exposures should be of greater concern than the current one-hour standard. To bolster their claims, those groups point to a number of recent studies that purport to show: (1) ozone-induced inflammation in the lower respiratory tract at concentration levels as low as 0.08 ppm; and (2) significant decreases in lung function among moderately exercising individuals exposed to ozone over an extended period. Supporters of a tighter ozone standard often infer that such short-term effects are precursors of long-term, chronic lung problems.

With respect to ozone-induced airway inflammation, the scientific debate does not center on the existence of the problem, but rather on whether or not such a reaction constitutes a serious "adverse health effect." Both the common cold and mild influenza often induce airway inflammation symptoms that substantially exceed those from a typical ozone exposure. Yet there is little concern that infection-related airway inflammation produces long-term adverse consequences.

Indeed, exposure to infections such as the common cold and influenza is generally thought to be an acceptable risk inherent in normal social activity. As William S. Linn, an ozone specialist and professor of preventive medicine at the University of Southern California, notes: "The law requires children to attend school, even though school attendance probably increases their exposure to respiratory disease organisms. If other forms of inflammation have acceptable risk/benefit ratios, so might the inflammation due to ozone."

As for concern over lung function, there has been a handful of studies that show a loss of lung function at ozone concentrations well below the 0.12 ppm standard. A study in the November 1990 issue of the *American Review of Respiratory Disease* by Horstman et al. examines the effects of 6.6-hour exposures on lung function. Twenty-two healthy, nonsmoking male volunteers (ages

18-35) were exposed to ozone concentrations of 0.00, 0.08, 0.10, and 0.12 ppm while moderately exercising for six 50-minute periods, each followed by 10 minutes of rest. The researchers concluded, "Some individuals experienced substantial pulmonary distress" at low concentrations, but "others will not experience any from such exposures." Lung function "was significantly reduced after only three hours at 0.12 ppm, after 4.6 hours at 0.10 ppm, and after 5.6 hours at 0.08 ppm." At ozone levels of 0.08 and 0.10 ppm, researchers reported that breathing capacity decreased by an average of 7.0 percent. Those findings were confirmed by a nearly identical 1991 study by McDonnell et al., involving 38 young males in which lung function dropped by an average of 8 percent at 0.08 ppm of ozone. Recall, however, that the Clean Air Act Scientific Advisory Committee suggests that a "mild" response (less than 10 percent reduction in lung capacity) should not be considered an adverse health effect.

Both the common cold and mild influenza often induce airway inflammation symptoms that substantially exceed those from a typical ozone exposure.

As Table 2 (page 54) shows, averages can be somewhat deceiving. The table indicates a good deal of variation in individual responses among the 22 subjects evaluated in the Horstman study. The subjects numbered six and seven experienced the greatest loss in "forced expired volume in one second" (a common measure of lung capacity) at the 0.08 ppm level—17 and 26 percent, respectively. Oddly enough, however, the two subjects experienced a lung function loss of only 7 percent and 8 percent at the higher 0.10 ppm exposure. Subjects six and seven again exhibited "responder"-type reactions at the next level of ozone exposure, 0.12 ppm, with decreased breathing capacity of 29 percent and 39 percent, respectively. Such variability makes drawing firm conclusions from the data somewhat problematic.

A 1994 study (Folinsbee et al.) examining prolonged exposures to ozone concentrations at the 0.12 ppm national standard was also inconclusive. The research explored the effects of *repeated*

Table 2

**Respiratory Response to Varying Ozone Levels
and Mild Exercise**
(Individual changes in forced expired volume
in one second [%] for 6.6-hour exposures
to 0.00, 0.08, 0.10, and 0.12 ppm ozone)

Subject	Ozone (ppm)			
	0.00	0.08	0.10	0.12
	%	%	%	%
1	1.9	-7.3	-15.6	-19.4
2	4.8	-14.4	-10.2	-10.7
3	2.1	1.4	-2.1	1.3
4	1.7	-8.2	-10.3	-7.9
5	-3.1	-5.4	-7.1	-8.1
6	0.3	-17.2	-6.8	-29.0
7	5.1	-25.9	-8.0	-38.9
8	-2.3	-4.9	1.4	-6.1
9	1.5	-16.4	-22.0	-23.8
10	2.7	0.7	2.0	2.2
11	-4.6	-1.9	0.6	-19.6
12	-0.6	-2.4	-0.7	2.8
13	-0.5	-4.7	-18.2	-16.0
14	0.3	0.5	-5.9	-2.7
15	6.4	-10.3	-7.8	-19.6
16	-0.1	7.9	0.4	-8.4
17	-0.5	-0.2	0.9	-2.8
18	2.9	-13.0	-18.8	-20.6
19	2.3	-2.2	-18.6	—
20	-0.3	-6.4	-3.0	-15.6
21	-0.3	-3.9	-8.7	-6.4
22	-1.8	-11.9	-9.5	-11.1

Source: Donald H. Horstman et al., "Ozone Concentration and Pulmonary Response Relationships for 6.6-Hour Exposures with Five Hours of Moderate Exercise to 0.08, 0.10, and 0.12 ppm," *American Review of Respiratory Disease*, Vol. 142, No. 5, November 1990, p. 1161.

prolonged low-concentration ozone exposure. Seventeen healthy, nonsmoking male subjects were exposed to 0.12 ppm ozone levels for 6.6 hours while mildly exercising on five consecutive days. While subjects did experience increased airway responsiveness during each of the five exposure days, other symptoms were fully attenuated three days into the test, suggesting an adaptation mechanism. For example, mean post-exposure-preexposure changes in lung capacity for days one to five of ozone exposure were: -11.9 percent, -6.23 percent, 1.06 percent, 1.52 percent, and 0.89 percent, respectively. Similarly, symptoms of coughing and pain on deep inhalation increased significantly on day one only. As with previous studies, there was considerable individual variability in pulmonary responses to ozone.

The results of those and other prolonged-exposure studies suggest that the observed acute effects are similar to those found in studies of shorter-term exposures. In most instances, the physiological changes are relatively minor. In all cases they are reversible and not life-threatening. Indeed, Horstman et al.'s prolonged-exposure study indicates that regardless of an individual's responsiveness to the ozone exposure, lung function returns to pre-exposure levels the very next day.

Accumulated animal studies, most often using rats subjected to long-term ozone exposures, have shown permanent damage to lung tissue at levels ranging from 0.12 ppm to 1.0 ppm. The more severe effects include decreased resistance to respiratory infection and chronic respiratory

disease.

Extrapolating the results of animal studies to human health effects is problematic, however. Congress's Office of Technology Assessment cites differences in anatomy, biochemistry, physiology, cell biology, and pathology as confounding factors in predicting health effects from animal testing.

One factor seemingly deemed irrelevant by proponents of a new standard is the ability of individuals to alter their living and working conditions to reduce their responses to exposures to elevated ozone. Unlike laboratory animals locked in cages, human beings can reduce their exercise levels and can even reduce their exposure levels by moving indoors during ozone alerts. Current Clean Air Act policy seeks to alter the environment to protect those who are presumed too foolish to protect themselves. As will be discussed later in this article, that paternalistic presumption can be extremely costly.

Field and Epidemiological Studies. A variety of individual-level field studies and aggregate-level time-series studies has addressed the effects of ozone on lung function and increased human morbidity and mortality. Research examining the outdoor exposures of physically active people, usually children in summer camps, provides some quantitative evidence relating lung function declines to ambient ozone exposure.

It is important to point out a key difficulty with the camp studies. It appears that ozone has a much stronger effect on lung function in comparatively low-exposure New Jersey camp studies than in the highest-exposure camp study in Southern California. If further research confirms the existence of this anomaly, it may suggest that other airborne pollutants are causing the reactions in New Jersey and/or that California campers have developed defenses to many of the effects of ozone exposure.

Other field studies that have analyzed summertime daily hospital admissions due to respiratory symptoms also show a positive relationship with ambient levels of ozone. The association has been shown to remain even after controlling for the effects of temperature and copollutants. However, those studies provide only *suggestive* epidemiological evidence for chronic health effects from ozone exposure. Lack of information on the actual levels of ozone exposures that individuals experience and other confounding variables have made it difficult to

establish more definitive results.

It does seem logical, however, that if prolonged exposure to elevated ozone does cause chronic health effects, residents of Los Angeles should exhibit those effects most dramatically. In the Los Angeles area, ozone levels exceeded 0.12 ppm for one hour or more for an average of 138 days a year during the period 1987-89. Nonetheless, evidence of chronic effects of ozone on Los Angeles residents is rare and, at this time, speculative.

A 1991 autopsy study of over 100 Los Angeles accident victims aged 14 to 25 offers some suggestive evidence of permanent lung damage. Twenty-seven percent of the subjects' lungs had severe structural abnormalities. Forty-eight percent had similar but less severe lung damage. Although the observed abnormalities were not expected in such young people, the researchers made no comparison to subjects from low-level air-pollution areas. Also, the researchers could not attribute their findings to ozone alone; smoking, drug abuse, or other airborne pollutants could have caused the observed abnormalities.

Although suspected, a link between cancer and elevated levels of smog has eluded researchers. In a 1991 study, medical researchers at Loma Linda University examined cancer incidence and mortality among 6,000 nonsmoking, California Seventh-Day Adventists. The researchers found that neither average concentration nor "any threshold levels of ozone were statistically significantly associated with cumulative incidents" of any symptom of "airway obstructive disease." They did find that the relative risk of respiratory cancer for persons exposed to ozone levels above 0.10 ppm for more than 500 hours a year was of "borderline statistical significance."

The book on ozone's health effects is certainly not closed. Population studies and clinical animal studies of ozone's chronic effects continue to raise concern among medical researchers. Nonetheless, any causal relationship between elevated levels of ozone and incapacitating or irreversible health effects in humans remains highly speculative.

Are the Health Effects Adverse? Essentially, the debate over the appropriate stringency of the ozone standard emanates from a fundamental tenet of the Clean Air Act: when setting air-quality standards, the EPA must provide the public protection against "any adverse health effect" and do so with an "adequate margin of safety."

However, the issue of whether the observed physiological responses are "adverse health effects" or not is difficult to resolve.

Unable to differentiate clearly between statistical significance and health significance, the EPA's Clean Air Science Advisory Committee states that the "resolution of the adverse effects issue represents a blending of science and policy judgments." The Office of Technology Assessment concurs with that view, concluding that air-quality regulations "can only continue to balance the costs and benefits of different regulatory levels rather than choose a regulatory level . . . that will clearly avoid adverse human health effects."

It is imprudent to require a reduction in the ambient air standard for ozone without a scientific consensus on the *health* significance of the observed physiological responses to relatively low and longer-term exposures. Nor should such a policy change be undertaken without consideration of the costs of reaching a standard of less than 0.12 ppm.

Understanding the Extent of the Problem

Unlike other air pollutants, ozone is not emitted directly into the air, but is formed through complex chemical reactions between emissions of volatile organic compounds (VOCs) and nitrogen oxides (NO_x) in the presence of sunlight during periods of elevated temperatures. Since the formation of smog requires sunlight, concentrations are minimal around sunrise, rise to maximum levels in the early afternoon, and fall to minimal levels again at night. Ozone levels also follow a seasonal pattern. In general, conditions are best for the formation and accumulation of ozone in the United States during the late spring and summer, when sunlight is most intense, temperatures are elevated, and stagnant meteorological conditions are present. Without sunlight, ozone begins to break down rapidly. Thus, ozone does not accumulate from one day to the next. Ozone also moves with air masses—for more than 100 miles in some cases—and dissipates fairly rapidly. Violations of the current ozone standard are highly sensitive to meteorological fluctuations.

The Clean Air Act very precisely defines an area to be "nonattainment" if the fourth highest daily monitor reading taken during the most recent three-year period registers a one-hour average concentration of ozone above 0.12 ppm. Even if all four highest readings were to occur in

a single year, and no monitors recorded a one-hour average above 0.12 ppm in the other two years, the area would be designated as nonattainment. Thus, a single year of abnormally high temperatures and stagnant air flows can force an area that is normally in compliance with the standard into nonattainment status for a minimum of two more years.

The Long Hot Summer of 1988. Extreme weather conditions in 1988, in particular, created a distorted picture of America's smog woes. Indeed, that year had more favorable days for ozone formation in the United States than any other in the past 25 years; the summer of 1988 ranked as the third hottest summer since 1895. In 1988, 258 counties had at least one monitoring site that had a one-hour average ozone level above 0.12 ppm—at least one "exceedance." By contrast, only 115 counties recorded at least one exceedance in 1990—the 15th warmest summer in the United States since 1895.

Because the Clean Air Act Amendments of 1990 require that the 1988 data be included in determining the classification of an area's nonattainment status, the extent of the problem is being overstated. The EPA currently reports that 89 non-California areas fail to meet the national ozone standard. If the EPA were to use 1989-91 data to determine an area's attainment status, only 28 non-California areas would be nonattainment.

The extent to which ozone is more a California problem than a truly "national" problem is made clear in a 1992 study entitled "The Truth about Ozone and Urban Smog" written for the Cato Institute by K. H. Jones. According to Jones, who authored the chapter on air quality status and trends in eight of the Council on Environmental Quality's annual reports, 85 percent of human exposures to ozone levels above 0.12 ppm in the United States occur in California—82 percent of which occur in the Los Angeles Basin.

Even in areas where ozone levels are high, progress is being made. Ventura County, California, which is classified as "severe" by the EPA, has seen the number of ozone exceedances drop from 122 days in 1974 to 13 days in 1993. San Francisco, an area that violated ozone standards on 65 days in 1969, had only two violations in 1994—an achievement that effectively removes the Bay area from the federal smog-watch list.

Jones's analysis shows that if ozone monitoring data are adjusted for temperature variations,

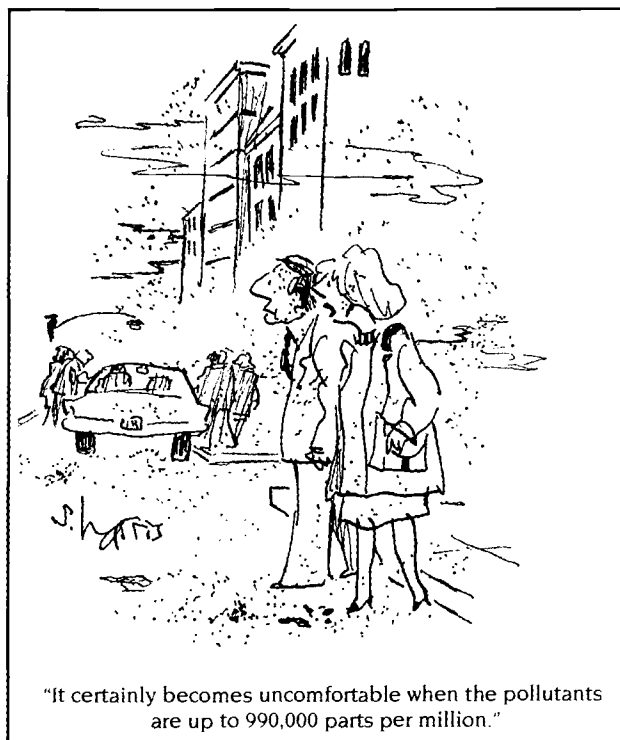
smog concentrations in non-California regions are declining as well. Since 1985 the number of exceedance days has declined by 74 percent in urban areas outside of California.

Several other facts should also be kept in mind when considering the seriousness of exposure to elevated ozone levels in nonattainment areas. First, localized readings recorded on any single monitor define the extent of a nonattainment area's air quality and, hence, the types of controls it must apply to the precursor pollutants that form ozone. As required by the Clean Air Act, the EPA has classified nonattainment areas as "marginal," "moderate," "serious," "severe," and "extreme," based on their fourth highest monitor reading during the period 1987-89. Cities classified as "severe" or "serious" must implement tougher control strategies than less-polluted "marginal" and "moderate" areas.

Simply by using more up-to-date data, the EPA could alleviate the regulatory burden of numerous cities. Jones points out that if 1988 data were not used, 14 areas would be reclassified as moderate or marginal, and thus could forgo the adoption of the "expensive and onerous" federal clean fuels program. Jones estimates that the resulting reclassification of non-California areas would lower the overall cost of ozone control by nearly 60 percent.

A second factor distorts the picture of ozone's impact on America's health, namely the current method of assessing the severity of nonattainment. The nonattainment classification of an area currently rests on just a single piece of data—the fourth highest one-hour reading from any single monitor in that area during the 1987-89 (or 1988-90) period. Taking the average of a representative sample of monitors would seem a more sensible procedure and could well show the fourth-highest reading to be a fluke, not at all representative of air quality for the area.

Using the three-year data that includes 1988 readings, the EPA estimated that 140 million Americans are living in areas that are technically nonattainment. That figure is misleading, however, since data from 1989 show that only 67 million Americans live in counties that recorded one exceedance or more in that year. In addition, exposures to elevated levels of ozone depend on the amount of time an individual spends outside. Most people spend more than 90 percent of their day indoors at home, school, or work, where ozone concentrations are substantially lower



than those outdoors. Some groups of people, such as construction workers or children, however, spend relatively more time outdoors and thus have a greater chance of being exposed to elevated levels of ozone.

Finally, the number of days that cities are in violation of the ozone standard has been steadily declining for the past decade. According to the EPA's most recent air quality report, the expected number of exceedances at all 532 air pollution monitoring sites decreased 60 percent between 1984 and 1993. For those sites that are located in high pollution-high population areas, the number of exceedances decreased 57 percent.

Hot spots do exist, but are isolated to Southern California, Houston, Chicago, and the Northeast coastal area. The South Coast Basin in California is the only region classified as "extreme." Houston and Chicago are classified as "severe," one category away from "extreme." As was pointed out earlier, even those highly polluted areas have shown marked improvement in air quality.

Ozone's Complex Chemistry. Researchers' lack of understanding of the formation, transportation, and accumulation of ozone further complicates the task of combating America's smog woes. Current atmospheric science does recognize that the *relative* concentrations of

ozone precursors—VOCs and NO_x —in the air can greatly affect the choice of the control strategy that will bring about the best results. Natural sources of VOCs also affect ozone control efforts: if “background” (natural) levels of ozone are significant relative to ozone formed from manmade pollutants, a more stringent standard would be even more difficult to reach.

In 1991 the National Research Council (NRC) analyzed the role of VOCs and NO_x in the formation and control of ground-level ozone. The NRC report suggests that science presently lacks the “knowledge of many of the fundamental processes that govern the formation and distribution of ozone.”

The NRC report emphasizes that the effectiveness of efforts to reduce ozone levels depends on the relative amounts of VOCs and NO_x in the air. Figure 2 shows curves representing maximum ozone concentrations formed from various mixtures of VOCs and NO_x . (These relationships have been derived through experimentation and computer modeling.)

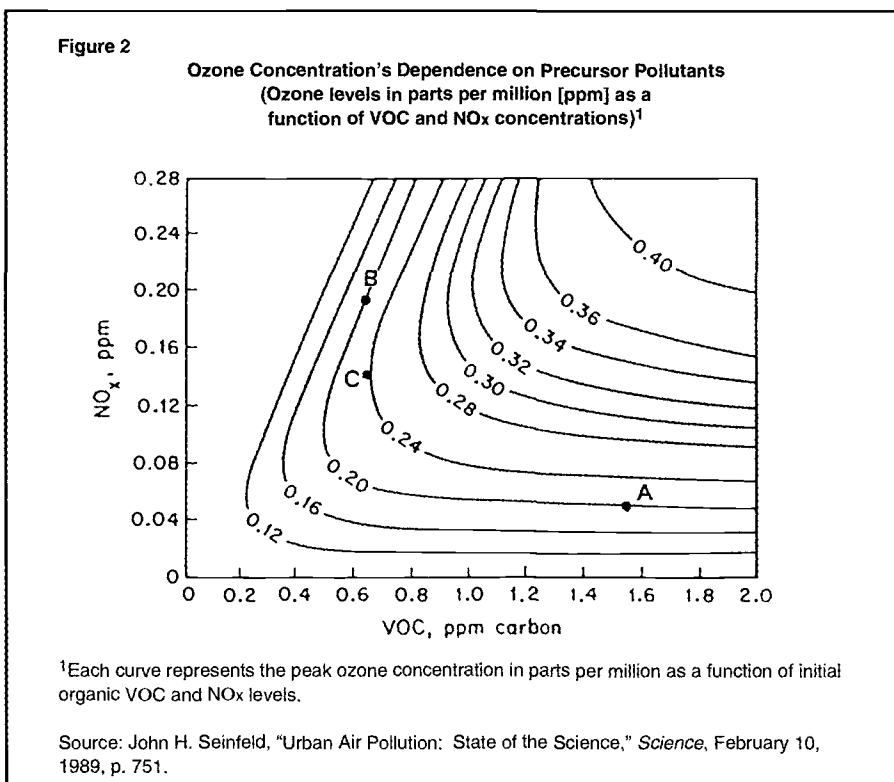
An area with a mixture of VOCs and NO_x similar to that represented by point A has a much higher ratio of VOCs to NO_x than, say, a city with a mixture equivalent to point B. Although both cities would

experience the same peak ozone level, they would require very different control strategies to lower ozone concentrations effectively.

For a city with a relatively high ratio of VOCs to NO_x in its air, such as point A, abating VOC emissions (moving to the left on the graph) will be *relatively* ineffective. Such a city could likely gain much greater benefits by reducing NO_x levels. The case for City B is the opposite: smaller reductions in VOCs provide the same benefit as large NO_x reductions. Additionally, in some cases abating NO_x could actually *increase* ozone concentrations, as shown by moving from point B to point C in Figure 2.

Ozone’s real-world chemistry is even more dynamic than that, however—VOC/ NO_x ratios can change throughout a single day. Ratios also vary by metropolitan area and can differ within sectors of an individual city. Further, the relative amounts of VOCs and NO_x in the air are linked to the location and size of their sources and the movement of air masses. VOCs originate mostly from vehicle emissions, chemical and petroleum refining companies, industrial solvents, and natural vegetation. Some smaller sources include dry cleaners, small repair shops, household paints, and lighter fluid. Major NO_x sources

include motor vehicles and electric power plants. Until recently NO_x emissions received little attention. Abatement emphasis has been placed on controlling VOCs in nearly all nonattainment areas. Most cities apply VOC controls because they are generally cheaper and more readily available. The NRC’s report also states that emissions inventories of manmade VOCs—especially automobile emissions—have been significantly underestimated and the effectiveness of efforts to control VOCs has been overstated. Consequently, the NRC concludes that past ozone-control strategies have been “misdirected.” Most ozone nonattainment



areas have point-A-type air chemistry—*relatively* high levels of VOCs compared to NO_x. Thus, controlling NO_x emissions may provide greater benefits than abating VOCs. Of course, comparing the relative costs of VOC and NO_x controls, which likely vary by region, still would be essential to choose the most efficient abatement strategy.

Atlanta's experience with controlling VOCs in the 1980s offers an example of the consequences of implementing a control strategy without a good understanding of the local smog chemistry. Atlanta spent \$700 million between 1979 and 1985 to cut transportation and stationary-source emissions of VOCs in half. In 1986, however, Atlanta's ozone levels were higher than in 1979. Besides not knowing the importance of VOC/NO_x ratios, city officials appear to have failed to realize the significance of natural emission of VOCs. Researchers have shown that in Atlanta, where almost 60 percent of the metropolitan area is wooded, natural VOC emissions are nearly equivalent to manmade VOC emissions.

Natural Sources of Ozone. As is the case in Atlanta, trees and other vegetation generate a significant amount of VOCs. Such natural VOC emissions are extremely dependent on temperature and thus are highest on days when meteorological conditions also favor ozone formation.

The National Research Council estimates that natural sources of VOC emissions, resulting in background levels of ozone, are of "comparable magnitude" to manmade VOC emissions. The NRC suggests that "in many cities even if manmade VOC emissions are totally eliminated, a background of reactive [natural] VOCs will remain." On days conducive to ozone formation, "this VOC background should be able to generate ozone concentrations that exceed the NAAQS [National Ambient Air Quality Standard] concentration of 0.12 ppm," according to the NRC. Computer-generated simulations have shown that reactions between prevailing levels of NO_x and natural VOCs alone can generate ozone concentrations above 0.08 ppm in the Ohio River Valley and the entire Northeast corridor.

In summary, the combination of weather conditions, complex chemistry, and natural emissions makes the objective of decreasing smog levels problematic. The NRC report emphasizes that solutions to local ozone problems are not easily prescribed, and a regulatory approach that does not take specific local circumstances into account is likely to produce disappointing results. The lack of progress in Atlanta in the

early 1980s clearly substantiates that conclusion.

Redefining Dirty Air Areas. More than 20 years of research and regulatory efforts have produced some progress toward reaching the current one-hour standard. Research on the air chemistry producing lower average levels of ozone over a longer period of time is less developed. The means for abating those lower concentrations are also poorly understood.

The EPA has analyzed the effects of various new ozone standards on the attainment status of many American cities. If an eight-hour, 0.10 ppm standard were required, the agency estimates that it would be roughly equivalent to reaching the current one-hour 0.12 ppm goal. An eight-hour, 0.08 ppm standard, however, would effectively be tighter than the current standard, resulting in designating as many as 67 additional cities as "dirty air" areas. Over one-third of those areas would record 10 or more violations a year under the revised standard, according to the EPA.

Many areas that were very close to compliance with the current standard in 1989 would have more difficulty reaching the revised target. For example, three Indiana cities—South Bend, Indianapolis, and Evansville—were on the threshold of reaching attainment status in 1989, averaging only 1.1 days a year when the 0.12 ppm standard was not met for more than one hour. Under an eight-hour, 0.08 ppm standard, South Bend, Indianapolis, and Evansville would fail to comply 16, 21, and 25 times a year, respectively.

The Costs and Benefits of Reducing Ozone Levels

It seems intuitive that a lower standard for average ozone concentration over a six- or eight-hour period, as championed by the American Lung Association and others, will be more difficult, that is, more costly, to try to reach. The costs of attempting to meet a new or additional standard and the value of the health benefits to be gained are unknown. Indeed, overall benefit and cost estimates of the present standard have been few and far between because the Clean Air Act does not allow for costs to be considered when setting national air-quality standards.

Of course, even if costs were better documented, we would only have half the puzzle solved—the issue is whether benefits exceed costs. Again, there is a paucity of information about the bene-

fits of abating ozone pollution.

One comprehensive study of acute human health (and agricultural) benefits, completed for the Office of Technology Assessment (OTA) by Alan Krupnick and Raymond Kopp of Resources for the Future, provides numerical estimates associated with various decreases in ambient ozone concentrations. Assuming that benefits are derived only by heavily exercising individuals, the payoff to public health from reaching the 0.12 ppm standard ranges from \$69 million to \$490 million (in 1994 dollars). If all exercising individuals are presumed to receive benefits proportional to exercise levels, the benefits derived as a result of total compliance with the current

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ozone standard range from \$906 million to \$6.4 billion.

The total costs of reaching the standard are incalculable, however. In particular, for Los Angeles to reach the 0.12 ppm standard, citizens would have to alter transportation methods, change patterns of workplace and housing locations, and substantially modify consumption of products that emit VOCs in their production or use. The costs of such lifestyle changes are virtually unquantifiable.

Fortunately, some sense of the comparability of nationwide aggregate benefits and costs can be deduced from benefit and cost estimates for a given level of reduction in VOC emissions. In their report for the OTA, Krupnick and Kopp furnish a benefit estimate for an across-the-board 35 percent reduction in VOCs for all nonattainment areas. That level of abatement was projected to bring only one-third of the areas in mild violation of the ozone standard into attainment by 2004.

Assuming that all exercising individuals receive benefits, a 35 percent reduction in VOC emissions would produce health benefits ranging from \$337 million to \$2.3 billion (in 1994 dollars). Converting a 35 percent VOC reduction

into tons and taking the high end of the acute health benefit estimated to accrue due to the decrease in ozone levels, health benefits would amount to \$610 a ton. That is an *optimistic* estimate of aggregate acute health benefits from reduced ozone levels.

Prior to passage of the 1990 Clean Air Act Amendments, the OTA estimated that reducing VOC emissions by about 35 percent (by 1994) would cost all nonattainment areas between \$4.2 and \$7.1 billion per year (in 1989 dollars). Costs would rise to between \$6.6 and \$10.0 billion (also in 1989 dollars) annually by 2004. Adjusting those estimates to 1994 dollars yields costs ranging from \$7.6 billion to \$11.6 billion by the year 2004.

Based on data from the EPA and the Alliance Technology Corporation, the Center for the Study of American Business (CSAB) calculated its own estimate for a 40 percent reduction in emissions by the year 2005. A 40 percent reduction would be possible at an estimated total nationwide cost of approximately \$10.9 billion a year (in 1994 dollars).

Table 3 shows cost-benefit ratios assuming the most inclusive health benefit estimate from the Krupnick and Kopp study, compared to OTA and CSAB projections of the cost-effectiveness of control techniques. The comparisons are very rough, however, and should be considered only as indicators. Using OTA estimates, the cost-benefit ratio ranges from 3.3 to 5.1. That is, for every dollar of benefit derived from the ozone provisions of the 1990 Clean Air Act Amendments, the cost to consumers and taxpayers is between \$3.30 and \$5.10. The CSAB's estimate yields a cost-benefit ratio of roughly 4.1, right in the middle of the OTA's range.

To be certain, no definitive conclusion should be drawn from such crude estimates, but average cost-benefit ratios substantially above 1.0 should raise a warning flag. In and of themselves, cost-benefit ratios as high as these for ozone abatement indicate that consumer and taxpayer dollars are being allocated improperly.

Cost-benefit ratios for greater reductions in ozone precursors (VOCs and NO_x), which would be needed to attain the standard nationally, would be even more unfavorable, because the costs of abating added increments of ozone-producing pollutants would rise at an increasing rate. Such is the case with most environmental cleanup projects. The pulp and paper industry,

Table 3

**Comparing Acute Health Costs to Benefits
(1994 dollars)**

Costs	Abatement Costs	VOC Reduction in Areas of Nonattainment²	Average Cost per Ton
OTA	\$7.6-11.6 billion	35% (3.8 million metric tons)	\$2,000-3,100
Center for the Study of American Business	\$10.9 billion	40% (4.3 mmt)	\$2,500
Benefits	Health Benefits¹	VOC Reduction in Areas of Nonattainment²	Average Benefit Per Ton
Krupnick and Kopp	\$2.3 billion	35% (3.8 mmt)	\$610
Cost-to-Benefit Ratio			
	OTA/Krupnick and Kopp	3.3 - 5.1	
	CSAB/Krupnick and Kopp	4.1	

¹The highest estimate of benefits from a 35 percent rollback of VOCs is \$2.3 billion in 1994 dollars, the high estimate from clinical studies.

²VOC reductions are for a baseline of 1985 levels (10.8 million metric tons for nonattainment areas).

Source: The Center for the Study of American Business.

for example, spent \$3 billion to reduce water pollution by 95 percent to comply with federal clean-water standards. To reach a 98 percent reduction, the industry would have to spend \$4.8 billion or more.

The converse is true for incremental benefits. In the case of ozone, it would be more beneficial in terms of avoiding acute physiological responses to reduce the level of one-hour exposures from, say, 0.24 ppm to 0.20 ppm, rather than from 0.16 ppm to 0.12 ppm.

If the current standard were tightened, every city now in violation, and a large number of new areas currently considered to be in compliance, would face a more difficult task to reach ozone attainment. Further, unless longer exposures are demonstrated to cause chronic effects, the benefits of going beyond the current standard to a six- or eight-hour, 0.08 ppm standard will be less than a comparable drop in ozone levels to reach

the current one-hour standard.

Conclusion

The Clean Air Act prohibits policymakers from considering economic factors when setting air-quality standards. Instead, the act requires that the standards for ground-level ozone ensure an "adequate margin of safety" against "any adverse health effects," regardless of cost. If medical evidence of adverse health effects of longer exposures to lower levels of ozone is demonstrated, the Clean Air Act requires the EPA to revise the standard. But if the adverse effects are temporary, and if the public places a value on their avoidance that is far less than the cost, resources could be better spent on more threatening environmental or public-health problems.

As ozone-policy analyst and former EPA official Dr. Milton Russell has said, "The way the law

[the Clean Air Act] is now written, it is almost as if a cancer were equivalent to a cold, one expected cancer were indistinguishable from an epidemic and as much social disruption . . . and economic costs were to be imposed to avoid the one as to avoid the other." William Ruckelshaus, former two-time EPA administrator, refers to the act's objective as an unattainable "standard of perfection."

While the amended 1990 Clean Air Act took a step in the right direction by defining categories of nonattainment and varying the prescriptions and proscriptions accordingly, it did not go far enough. Since the duration and peak concentrations of ozone and the percentage of the population exposed to elevated ozone levels are site-specific, cost-benefit ratios will differ by region. Thus, a more efficient policy would be to allow each region to weigh the potential gains from improving its air quality against the costs.

Ultimately, the high-cost pursuit of perfection cannot be avoided unless Congress revises the

Clean Air Act's fundamental objective. The goal of the act should be to protect the public against *unreasonable* risk of *significant* adverse health effects. Unless chronic effects of prolonged exposures to elevated ozone can be demonstrated (and after two and a half decades of research, they have not been), it would be extremely wasteful to insist on a tighter air-quality standard.

Selected Readings

Chilton, K. and Sholtz, A. *Battling Smog: A Plan for Action*. St. Louis: Center for the Study of American Business, 1989.

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