

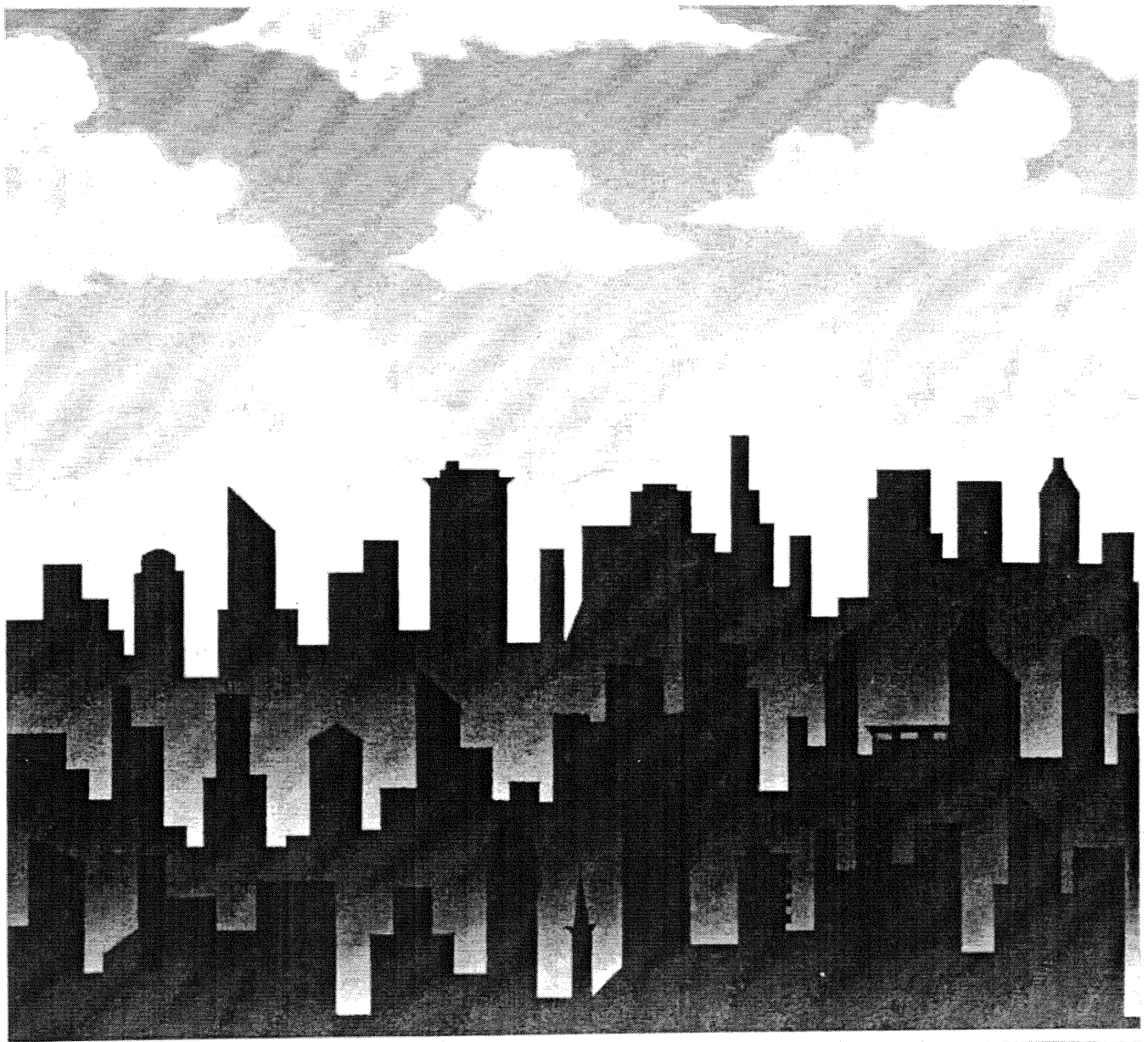
Q-91

# AIR POLLUTION EMERGENCIES

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A Summary of Testimony  
Presented at the Public Hearing  
Sponsored by the  
New Jersey Clean Air Council

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APRIL 15, 1991

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**Being a Summary of Testimony**  
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**SCOPE**

The 1991 public hearing sought information on issues related to air pollution emergencies in New Jersey. The Clean Air Council was particularly interested in ascertaining whether or not the State's ambient air quality guidelines for triggering actions at the four stages of its Air Pollution Emergency system ("Advisory," "Alert," "Warning," "Emergency") are sufficiently protective of public health. Other objectives included assessment of procedures for distributing information to the public and recommendations for actions to be taken by state government.

**BACKGROUND**

The current regional air pollution emergency warning system was developed in 1970 and revised in 1973 by a committee consisting of representatives of the states of Connecticut, New Jersey, and New York, and the Interstate Sanitation Commission. The system sets guidelines and procedures for three stages of an Air Pollution Emergency: these are known as "Alert" (or "Stage I"), "Warning" (or "Stage II"), and "Emergency" (Stage III). These guidelines are related to, but not necessarily identical with, the legal standards for ambient air mandated by the federal Clean Air Act and the state Air Pollution Control Act. New Jersey has incorporated into its system an additional guideline for issuing a "Health Advisory" to the public before the guideline triggering an "Alert" is reached. The system is used as "a tool to prevent contaminant concentrations from reaching an emergency level and not as a substitute for an effective air pollution control program." The prime objective of the program is protection of public health.<sup>2</sup> New Jersey's Air Pollution Emergency Control Act (NJSA 26:2C-26 through 36) was adopted in 1967. The administrative rules implementing this act (NJAC 7:27-12) were last revised in 1972. The guidelines that are currently used were created as part of a joint agreement between New York, New Jersey, and the Interstate Sanitation Commission with some modifications issued by Administrative Order of former DEP Commissioner Richard Sullivan.

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<sup>1</sup> The Clean Air Council was formed under the authority of the New Jersey Clean Air Act of 1954; its members are appointed by the governor; its business is to study and make recommendations to the Department of Environmental Protection and Energy concerning the implementation of federal and state legislation and regulation dealing with air quality and to advise the commissioner of DEPE on air matters. When these hearings were held, the DEPE was simply the Department of Environmental Protection and shall be so called in this summary.

<sup>2</sup> "Status Criteria for a High Air Pollution Alert and Warning System." Interstate Sanitation Commission, New York, NY 10019. 1970, rev. 1973.

## Emergency Air Quality Control Guidelines

Pollutant	Advisory	Alert	Warning	Emergency
Smoke Shade	2.0 COHS† for 24 hours	5.0 COHS for 6 hours or 3.0 COHS for 24 hours	8.0 COHS for 6 hours or 6.0 COHS for 24 hours	7.0 COHS for 24 hours
Carbon Monoxide	9 ppm* for 8 hours	15 ppm for 8 hours	30 ppm for 8 hours	40 ppm for 8 hours
Nitrogen Dioxide	0.25 ppm for 1 hour	0.8 ppm for 1 hour or 0.2 ppm for 24 hours	1.2 ppm for 1 hour or 0.3 ppm for 24 hours	1.6 ppm for 1 hour or 0.4 ppm for 24 hours
Ozone	0.12 ppm for 1 hour		0.25 ppm for 4 hours	0.35 ppm for 4 hours
Sulfur Dioxide	0.14 ppm for 24 hours	0.3 ppm for 6 hours	0.5 ppm for 6 hours	0.6 ppm for 24 hours

† Coefficient of Haze

\* parts per million

★ The various stages of the warning system go into effect when a 36-hour (or longer) air stagnation episode is forecast and air pollution concentrations meet or exceed the above air quality control guidelines. NJ's weather information is received from a subscription service, ACCU Weather. New York relies on its Dept. of Environmental Conservation in Albany. [This service used to be provided to the region by the National Weather Service.] The New Jersey Department of Environmental Protection receives air pollution information from its network of 27 continuous ambient air monitoring stations across the state which transmit data every minute to a central computer in Trenton. Arithmetic average concentration values are used throughout the system. DEP's ozone predictions are available to the public by 9 a.m. every day.

★ Should the governor of New Jersey, on the advice of the DEP commissioner, declare an Air Pollution "Alert," "Warning," or "Emergency," specific departmental actions must be taken to reduce the atmospheric pollutant load. These measures include temporary curtailment of some industrial operations, imposition of driving bans, and the shutdown of various services. The issuing of a "Health Advisory," however, does not require government to take any specific action to reduce source emissions.

## RECOMMENDATIONS

The following general recommendations were developed from many specific recommendations presented to the Council during testimony. A list of these recommendations may be found in Appendix B of this summary.

### Revision of system guidelines

\* The Clean Air Council recommends a comprehensive review and revision of the Air Pollution Emergency Warning System, including the Emergency Air Quality Control Guidelines to ensure reflection of both the current situation in the New Jersey region and new scientific evidence regarding the effect of ambient air pollutants on public health, including the most sensitive groups. We recommend that this revision include the following actions:

--setting the guidelines for triggering a Health Advisory lower than federal or state ambient air standards;

--setting the Health Advisory for Ozone at an actual level of 0.10 ppm or greater for one hour, or a forecast of 0.12 ppm for one hour;

--moving all Alert guidelines closer to the Clean Air Act ambient air standards;

--reinstating the Alert guideline for ozone and setting it at the legal health standard, currently 0.12 ppm for one hour;

--coordinating the Air Pollution Emergency System with relevant features of the Toxic Prevention Control Act; and

--replacing the smoke shade guideline with a real-time measurement of particulate matter (PM-10) as the technology to do so becomes available.

All revisions of system guidelines should be coordinated regionally to encourage continued inter-state cooperation and to avoid public confusion regarding media messages.

→ DISCUSSION W. COMM'R. WEINER

### Public Education and Notification

\* The Council recommends increased attention be given to improving the effectiveness of the messages used to communicate with the public regarding Air Pollution Emergencies. Messages need to be both concise and precise regarding actions individuals should take to protect themselves from adverse health effects when air quality is reduced. General public education



regarding the effect of air pollutants on health needs to be on-going. We recommend that strong consideration be given to establishing a special work group, like that described by Professor Greenberg, to help create public information messages and to test their effectiveness in generating appropriate public responses.

\* The Council also recommends increased attention be given to increasing the speed with which these messages reach the community; particular emphasis should be placed on reaching those segments of the population most sensitive to poor air quality, including the elderly, the young, the chronically ill, the physically active (e.g. athletes), and those who spend time outdoors. A new communications network needs to be created that includes direct media contacts, local health officers, school officials and athletic departments, and the NJ Medical Society.

#### **Actions to be taken by DEP in Response to an Air Pollution Emergency**

\* The Clean Air Council recommends that DEP develop additional speedy, pro-active plans for achieving air pollutant reductions when predictions of high pollutant levels are received. Plans should include procedures for eliciting specific pollutant reductions from the general public as well as from industry. For example, consideration should be given to developing plans for eliciting reduced motor vehicle use during periods of high ozone. Response plans need to be accompanied by an active public education effort.

\* The Council also recommends that a response manual be developed for all elements of the response network and that planned response procedures be tested during air emergency drills patterned on those conducted by the Office of Emergency Management. Response procedures should also include appropriate actions to terminate an Air Pollution Emergency. Plans for response actions should be coordinated regionally.

#### **Data Collection**

\* The Clean Air Council recommends a review of data collection procedures to ensure that the best, most specific information is available for forecasting potential reductions in air quality. This review should pay particular attention to improving the gathering of local weather information, establishing an upper air monitoring system, and using real-time measurements for air pollutants.

\* The Council also recommends that computer models be incorporated into the data collection system, both to improve prediction of high pollution concentrations and to monitor plumes of pollutant that may be missed by the current monitoring stations. Research and development to improve the accuracy and reliability of such models should be encouraged and supported.

## ORAL TESTIMONY

**Scott A. Weiner, Commissioner, New Jersey Department of Environmental Protection**

The NJ Department of Environmental Protection is responsible for testing air quality in New Jersey and for issuing warnings to the public when the air quality fails to meet specified standards. The state warning system has three levels-- "Alerts," "Warnings," and "Emergencies"--as well as various response criteria. This system has not changed significantly since 1973, but our air quality problems have. It may be time to update.

The public is not generally aware that everyone, not just industry, creates air pollution and thus everyone has the power to help reduce air pollution. It is important to drive this idea home. It is a fact that in New Jersey today our most difficult air pollution problem is no longer industry but the individual automobile. Somehow we must reduce the pollution from our individual cars without destroying their benefits.

Some actions can be taken now. I have convened a Clean Air Act Implementation Group, made up of myself, the Attorney General, and the Commissioners of the Departments of Transportation and Commerce. We must make better use of the daily forecast so we can avoid the onset of predicted high pollution concentrations. We need to increase and broaden efforts to educate the public about what they can do about air pollution, perhaps taking as a model the way we have promoted recycling through school programs. And, finally, DEP needs to review and update the air pollution emergency warning system regulations.

**John Penek, M.D., The Breathing Center**

Every day, people move between 15 and 18 thousand liters of air into their bodies; this volume represents the greatest interface between human beings and the environment. The threshold for damage to the body from breathing ozone contaminated air is currently set at 0.12 parts per million, or approximately one molecule of ozone for every ten million air molecules. Because of recent advances in research concerning the effects of ozone on the lung, it is probably time to reconsider this standard, although I cannot say here whether 0.12 ppm is too high or too low.

Ozone (O<sub>3</sub>) is related to oxygen (O<sub>2</sub>). At low concentrations oxygen is good for the body (oxygen represents 21% of the air). But too much oxygen can result in blindness, lung tissue damage, or even fatality.

Breathing is an involuntary process we don't notice until something goes wrong. When lungs are damaged, the small airways go into spasm. When this happens, the exchange of carbon dioxide and oxygen in the alveolar sacs at the ends of the airways cannot take place. The person begins to wheeze. On hot summer days when ozone concentrations are high, we have an increased number of patients coming into our office wheezing. I have a suspicion that ozone may actually trigger wheezing symptoms in otherwise normal individuals.

Ozone is an extremely potent oxidant. In high concentrations, it attaches to cell membranes, causing them to break down and destroying their capacity to protect cell functions from foreign substances that enter the body with the air we breathe--disease agents, particulate matter, allergens.

The delicate hairs, or cilia, on the lining of the entire respiratory tract are extremely sensitive to injury by air pollution. These cilia are made of the same material as cell membrane. For two years I have been studying the damage that occurs to the cilia of human volunteers when they are exposed to varying concentrations of ozone. Damage begins around 0.1 ppm; by 0.4 ppm, the cilia are almost entirely gone from the exposed area. In some ways the damages are similar to those done by smoking. Thus a big problem for epidemiology is separating the various causes of lung injury: smoking, disease, infection, allergy, air pollution. Shortness of breath and bronchospasm are the lung's defenses against damage by pollution. The secretions that occur with asthma and bronchitis arise from the body's (not very productive) attempt to heal injury.

The incidence of respiratory disease in the United States continues to rise, despite a leveling off of smoking in some population age groups. The cause for this rise is, however, unclear. It could be new methods for classifying respiratory disease; it could be new occupational factors; it could be increased air pollution. Moreover, despite better treatments for asthma and bronchitis, the death rate for asthma has risen considerably in recent years. Again, we don't know exactly why. It could be caused by changing classification procedures -- any death caused by an acute bronchospasm is now classed as an asthma death -- or air pollution may be a contributing factor.

In general, the best way to avoid death from asthma is to avoid situations that cause one to go into bronchospasm. This indicates the importance of educating the public. One of the best ways to educate the medical community about the dangers of air pollution is through the Journal of the New Jersey Medical Society. Another is through public seminars.

**Herbert I. Wortreich, New Jersey Clean Air Council**

Air pollution may be classed according to the circumstances in which it occurs: 1) chronic, ubiquitous pollution resulting from routine activities; 2) acute, short-term pollution incidents resulting from abnormal events such as spills or failures; and 3) somewhat longer pollution episodes resulting from weather conditions that prevent normal ventilation and diffusion of pollutants. These various circumstances may be superimposed, increasing the seriousness of air pollution.

Although prevention is the best way to deal with air pollution, prevention has not, until recently, been the government approach. NJ's Air Pollution Control Act of 1954, though a pioneering document, allowed regulatory action to take place only after the fact. The concept of preventive regulation was not introduced until the 1961 amendments.

Chronic situations (1) have generally been dealt with through standard setting. In NJ this method has been particularly successful for sulfur dioxide and suspended particulate matter. Prevention of acute incidents (2) was not addressed until 1986 in the Toxic Catastrophe Prevention Act, motivated by a series of in-state incidents between Oct. 1964 and February 1985 coupled with the disastrous accident in Bhopal, India. Administrative rules were passed in 1988. The TCPA, however, was narrow in its focus.

A number of regional weather-related pollution episodes occurred in the early 1960s, exposing millions of people to abnormal concentrations of SO<sub>2</sub>, NO<sub>x</sub>, NO<sub>2</sub>, HC, and particulate matter. This series of episodes resulted in creation of the Regional Air Pollution Emergency

Warning System in 1964 through cooperation of the states of NJ, NY, and Connecticut, and the Interstate Sanitation Commission. This weather-oriented system focused on sulfur dioxide, carbon monoxide, and a "soiling index" related to particulate matter. Revisions of the system in 1967 resulted in the familiar hierarchical warning system that distinguishes among the several stages of a weather-induced episode of air pollution. A forecast, or advisory, occurs when the Weather Bureau predicts conditions with a potential for increasing concentrations of pollutants. The "Alert," "Warning," and "Emergency" stages are reached as continued unfavorable weather combines with increasing concentrations of pollutants. Notifications cease when the weather clears.

In 1967 NJ adopted an Air Pollution Emergency Control Act, authorizing the governor to restrict activities that "may contribute to an air pollution emergency." This led to the 1969 adoption of criteria defining the three stages of a weather-related episode. These rules were amended in 1973, but have remained static for the last 17.5 years despite much improvement in our understanding of both individual and synergistic effects of pollutants as well as dramatic changes in the quality of NJ's air and the nature of the pollutants of concern.

Since the 1987 public hearing of this Council, ozone has been a particular interest of New Jersey and efforts have been made to advise the public of unhealthy ozone concentrations.

Weather-related episodes will always be with us, since we can never reduce pollution to zero or change the behavior of weather. Therefore, the only strategy left is to slow down the accumulation of pollutants by taking increasingly stringent emission-reducing actions as we proceed through the stages of an episode. At the emergency stage, draconian measures are warranted.

Fortunately, NJ has been spared even "Alerts" for the past few years, but there is no guarantee that this will continue. Moreover, there is a real question whether the criteria for triggering the stages are still adequate, if they ever were, for protecting public health. Not only must concentrations exceed the National Ambient Air Quality standard (supposed themselves to be a threshold of danger) even for an "Alert" to be issued, but also there are no "Alert" criteria at all related to ozone and none at any stage for lead or particulate matter. Nor do we know whether the emission-reducing measures on the books would work, since they have never been tested. Communications technology, demography, transportation, and public expectations have all changed since the system was devised 17 years ago. In short, it is time for reconsideration. I would suggest, for example, moving the Alert criteria much closer to the National Ambient Air Quality Standards

(Editor's note: Mr. Wortreich has appended to his testimony a list of specific changes to NJSA 26:2C-26 through 36 and the Emergency Air Quality Control Criteria--Administrative Order 38--recommended in a memo to Jorge Berkowitz, DEP, dated Sept. 30, 1987, which is summarized in Appendix A.)

**Alan Mytelka, Ph. D.**  
**Director, Interstate Sanitation Commission (ISC)**

The current warning system for regional episodes of high air pollution works because of the cooperation of all concerned agencies, including the ISC. Only New Jersey, however, has

adopted specific criteria values for moving through the various stages into its Administrative Code. After almost two decades, it may be time to reconsider the parameters for these criteria.

Ozone was dropped as a parameter in 1972; perhaps it should be reinstated. Exactly what values to use, however, is never simply based on medical evidence; it results from compromise and negotiation among scientists, engineers, politicians, and bureaucrats. Any changes that are made in the system must be agreed upon regionally; if they're not, cooperation will break down. Also, a recent fire in Jersey City raises the question of adding even more criteria--parameters for organics, for example. But if we start to extend the list of parameters, how specific should we get? Or should we continue to rely on indicator pollutants only?

Finally, I'd like to make some comments on weather forecasting. The system was set up to use the National Weather Service, an unbiased provider of forecasts. A few years ago, however, the National Weather Service gave up making these small-scale forecasts (20-30 miles radius), and so they are now done in Albany by the New York Dept of Environmental Conservation (DEC). Is this really appropriate for New Jersey? It would cost money, but I think New Jersey ought to have the opportunity to become involved in this forecasting. This is not only a question of fairness, but in New Jersey the various stages of the warning system are legally tied to certain emissions-reducing activities, including production slowdowns. So what happens if you shut industries down, and the forecast is wrong. It's a risky business, and to do it well, we must have the best available information. I think the weather forecasting aspect of the system needs another look.

(Editor's note: Mr. Mytelka appended to his testimony a description of the current warning system, which was used to prepare the "Background" section of this report.)

**John Elston, Assistant Director  
Air Quality Management and Surveillance  
NJ Department of Environmental Protection**

On March 13, 1991, Project Clean Air reported the results of a poll by the Eagleton Institute. According to this poll, 33% of New Jersey residents adjust their behavior when air quality declines; 80% say they can tell when air quality is worse; 52% named some sensory experience associated with poor air quality; and 23% cited some health-related indicator of poor air quality. Nobody mentioned the state's air pollution warning system.

Thirty to sixty times each year, areas of NJ have concentrations of air pollution higher than the National Ambient Air Quality Standards (NAAQS). Other criteria govern the air pollution warning system; that system has not been triggered since 1988. This is perhaps why the public does not associate poor air quality with the warning system. Generally, an air "Alert" is not triggered in New Jersey until the air pollutant reaches twice the standard concentration. It should be noted that in New York, an "Alert" is instituted whenever air quality goes only slightly over the NAAQS--this is what most NJ residents are hearing over TV and radio.

For purposes of the warning system, all pollutant concentrations are normalized into one number, called the Pollution Standard Index (PSI). A PSI of 100 equals the NAAQS. In this



scale 0-50 means "good" air quality; 51-100 means "moderate"; 101-200 means "unhealthy"; 200-300 means "very unhealthy"; 300-400 means "hazardous"; and over 400 means "emergency." The PSI is what you usually hear in press releases.

The values used for calculating the PSI in New Jersey are health based and are slightly more restrictive than the federal standards. In 1972 ozone was formally removed from the PSI calculation by regional agreement. In 1982 New Jersey informally included short-term NO<sub>2</sub> into its calculations (0.5 ppm for 1 hour). In late July 1988, as the result of a very persistent episode of smog, New Jersey also revised its administrative guidelines to allow for incorporation of ozone (0.2 ppm) into the calculation of its "Alert" and for voluntary industry reductions of ozone precursors. In that July we instituted our most recent Alert; we requested a number of large pollution emitters to curtail production; most complied. These requests were made by phone and call took most of one morning.

The ozone values used for the PSI calculation are designed to protect most but not all sensitive individuals. The PSI is intended to be advisory only; it is not regulatory, although some have recommended a more proactive system.

Ozone concentrations are hard to predict. This is partly because of the nature of its formation and transport, but also because of the loss of local weather reporting data through the consolidation of the National Weather Service over the past decade. The wide dispersal of ground pollution monitors also causes loss of some localized episodes of pollution by SO<sub>2</sub>, CO, and particulate matter. To improve the system, we need to improve quick response and real time monitoring.

One thing the Council could do is review medical data for short-term exposure; in other words, we need credible extrapolations of long term health data down to, perhaps, the one-hour level. This would greatly facilitate forecasting. Any new criteria developed as a result of this review, however, should be regionalized.

The DEP needs to make up for the loss of good data from the national weather system, perhaps through more surface meteorology and the establishment of an upper air monitoring system. We are exploring an automated gas chromatograph for ozone precursors as well as a TEOM system (Tapered Element Oscillating Micro-Balance Technique) for particulate matter.

We also need more proactive measures to get pollution curtailment, using industry agreement or administrative order, during periods of high pollution. Actions should be pollutant specific, however, not just across the board. } ★

Monitoring and response to air pollution released should be based on computer modeling of pollution pathways, as they are in the nuclear industry. In other words, if we knew where to look for pollution after a release, we could do better monitoring and make quicker responses. Often narrow plumes of a pollutant are missed because we can't find the plume in our monitoring network. There are many ways to improve the system.

Press releases occur only after the fact. We need better communication with the electronic media, television, and radio to get our message across quickly. Public displays of monitoring information is another idea. The Council could help overcome inertia in this area.

To get the message out to the public, I feel a good privatized system might be better than government. Weather reporting suggests one model, for example, frost reporting in agricultural areas. A business could be made selling air pollution information to people with medical problems. If this doesn't happen, the DEP is obliged to do it. But since it is hard to predict



specific numbers and mistakes in any direction have definite political costs, it is hard to get government to be too aggressive in forecasting.

Information on Air Quality is available daily from the Bureau of Air Monitoring: 609-633-7648 or 800-782-0161.

**Ronald White, Senior Program Manager for Air Conservation  
American Lung Association, Washington, D.C.**

Ozone is New Jersey's most serious outdoor air pollutant. Northern NJ violates the federal standard an average of 17 times each year; southern NJ violates it about 9 times each year. Multiple violations of the 1-hour standard during a single day count as just one violation, so the severity of NJ's problem is really worse than the statistics imply.

When Congress passed the 1970 Clean Air Act, they unambiguously intended it to protect public health "with an adequate margin of safety" for sensitive groups. Costs of control were not intended to be part of the standard setting process; costs were to be considered only during strategy selection and implementation. This basic concept remains in tact in the Clean Air Act of 1990. It should be employed in New Jersey, where neither economic issues nor transport (assigning fault) should be allowed to cloud the discussion of protection of the public's health.

I direct your attention to testimony received by this Council at its 1987 hearing on the ozone standard. At that time you heard evidence that healthy, exercising adults and children experience loss of lung function when exposed to ozone concentrations at or above the federal standard of 0.12 parts per million (ppm) for one to three hours, and that this loss persists for as long as one week after the smog episode has cleared. The U.S. Environmental Protection Agency has estimated that up to 15 percent of the population is so affected. Since 1987, research on children at summer camps has found evidence of lung function loss from exposures below 0.12 ppm. There is also biochemical evidence that inflammatory changes persist for as long as 16 to 18 hours after exposure. Exposures to ozone levels of 0.08 to 0.10 ppm for longer than 7 hours may also result in dramatic losses of lung function, increase in respiratory symptoms (coughs), and inflammation. This is well below the federal one hour ozone health standard.

We conclude from these studies 1) that the current Primary Ambient Air Standard of 0.12 ppm, which is used as the triggering criteria for an Air Pollution Emergency in New Jersey is not protective of public health and should be revised downward to 0.095 ppm (the California criteria), and 2) that NJ's 6 to 8 hour standard should be no higher than 0.08 ppm.

Recent studies of animals exposed to chronically elevated ozone reveals presence of lesions, damage to lung cells, and structural changes associated with loss of lung elasticity. These studies have significant implications for chronic human effects and suggest the need to be conservative when evaluating an ozone standard designed to protect people from acute effects

There is in New Jersey no criteria for an ozone Alert. I do not understand this omission, and I strongly suggest that this represents a danger and a disservice to the health of the people

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of NJ. In Sept. 1990, California adopted an ozone Health Advisory level of 0.15 ppm, retaining the criteria of 0.2 ppm for Alerts (Stage I). Both the Calif. Air Resources Board and the Calif. Dept. of Health Services recommended revising the Alert criteria down to 0.15 ppm. the American Lung Assoc. recommends that NJ adopt an Air Pollution Alert guideline of 0.15 ppm (one hour) and 0.12 ppm (6 hour average).

Recent studies indicate that people with asthma (3-4% of the population) are acutely sensitive to sulfur dioxide. Symptoms occur with exposure to levels as low as 0.25 ppm for 5 minutes. People with allergies (12% more of the population) are nearly as sensitive. Thus, the current federal standard of 0.14 ppm for a 24-hour averaging period is totally insufficient to protect this group when exercising. A one-hour standard below 0.25 ppm is necessary to protect the health of this group, and all four triggering levels for an Air Pollution Emergency in NJ should take this into account.

The public needs advanced notice when unhealthy air is forecast. They may then make decisions to reduce personal exposure by staying indoors or not exercising, for example. "Same day" advisories, though difficult, are needed early in the day via television and radio spots. The message must be both concise and motivating, with avoidance strategies clearly articulated. When setting criteria for advisories, it is better to err on the side of public health protection.

**Nathan Reiss, Ph.D.**

**Department of Meteorology, Rutgers University**

If we want to curtail source emissions when pollutant levels rise, it is very important to make timely and accurate predictions. Mistakes can be costly; people need time to prepare in order to minimize the impacts of reduced activity on business. To improve subjective forecasts by DEP personnel, we have developed a computer model at Rutgers that provides automated daily predictions. Although the model uses fairly rudimentary statistical procedures, it does take into account a number of variables and produces predictions without human intervention.

Data for this model comes from the National Weather Service, which routinely feeds into our departmental computer via an automated modem connection. We also receive data from DEP's air quality data base and DEP's meteorological data base. The computer extracts from this information (which it keeps on file) the precise items it needs to create an ozone forecast.

One of the most challenging aspects of this model was to develop a system to verify the data automatically, so that errors would not be used to produce wildly erroneous predictions. The data received by the computer comes in highly predictable formats; if the computer finds that the format is wrong, it won't use the data. Missing or garbled data is patched up from a standard data file, although we intend to revise the program so that patching is done from the previous day's data and a more long-range prediction is produced.

The program must also match up the locations of the observations, so that only data from a single location is used to make the prediction for that location.

Forecasts are produced by 8:30 a.m. and are accessed by DEP personnel who may, at the same time, review the data that went into the forecast. Currently, the person at DEP who uses this system doesn't trust the computer forecast at all, but he is assisted by having all data gathered into one place. Also, if the user doesn't trust the weather forecast that is being used,

alternative weather data may be selected and a new ozone prediction generated. Predictions are offered for 15 locations in New Jersey.

Last summer the system proved to be 92.6% accurate. The system rarely failed to predict that the standard would not be exceeded (this is about as impressive as predicting a dry day in the desert), but it often predicted high ozone levels for days when none occurred. This over-prediction occurred because we were using 1988 gasoline volatility data and we had no data regarding the effect of Stage II vapor recovery systems. The effects of these changes have been very noticeable; weather patterns that a few years ago would produced high ozone levels, no longer do. We intend to adjust our data to reflect these improvements. We are also going to adjust the model for local weather phenomena--like sea breezes-- and we will add in a nitrogen oxide variable. We have been working on this model for only three years, under a small grant from DEP. With more support, the model could certainly be improved through smaller scale data and increasing the sophistication of the programs. The computer would be happy to produce predictions continuously through the day.

This automated prediction system is like the one we provide to the Department of Agriculture for frost predictions. It is useful for farmers because it is available in the middle of the night, when experts are not. Farmers can call up our computer and receive both the prediction and a discussion of strategies to protect crops.

**James C. Morford, Vice President for Governmental Relations  
New Jersey State Chamber of Commerce**

The Chamber of Commerce believes that the current NJ ambient air quality standards for both pollutant concentrations and duration are reasonably protective of public health. In some cases they are more stringent than the federal standards, which are set by the Environmental Protection Agency after scientific demonstration of their necessity. Moreover, the air quality in New Jersey has generally improved since 1970. According to DEP the maximum ozone concentration in NJ has fallen 33% since 1973.

Major sources of ozone in NJ are not industrial sources. Regional transport has been addressed by the 1990 Clean Air Act. Emergency criteria used to trigger cutbacks of business activity are below the level of "significant harm" identified by EPA, although the objective of the warning system is to ensure that we never do get to that point. The fact that the state has not declared even an "Alert" in recent years is evidence that air quality has improved. Lowering the criteria that trigger action would thus not provide more protection to the public.

We support the current Emergency Air Quality Control Guidelines. Industry has developed contingency plans. Plans could be developed to reduce emissions from nonindustrial sources as well, both within the state and regionally through cooperative agreements.

We encourage the DEP to establish a series of public discussions regarding how the public could respond to air pollution emergencies. DEP should solicit voluntary reductions of vehicle use by the public, which would further improve air quality without imposing undue burdens on the state's industry.

**Charles Pietarinen**  
**NJ Dept. of Environmental Protection**

Smoke shade or the coefficient of haze (COHS) is currently used to indicate the presence of particulate matter in the atmosphere. Smoke shade, however, is not the same thing as particulate matter. COHS is measured in Smoke Shade Units and involves both a measurement of mass in micrograms per cubic meter and a light effect. Particulate matter is measured only according to mass. Even though smoke shade does not provide a simple equivalent to particulate matter, it is used because there is a real correlation between haze and air quality. Historically, COHS has provided the only way real-time method available for estimating particulate measurements; results based on direct monitoring for the presence of particulate matter can take up to a week to receive. New technologies for near real-time measurement of small particulates (PM-10), however, are being developed—for example the beta gauge and also the Tapered Element Oscillating Micro-Balance (TEOM). We should move ahead towards direct mass measurements of particulate matter on real time.

#### **WRITTEN TESTIMONY**

**Michael Greenberg, Ph.D.**  
**Department of Urban Studies and Community Health**  
**Rutgers University**

I would like to suggest a 4-step process for developing and evaluating public messages regarding air pollutant episodes.

1: Gather a working group of experts to define what action the public should take in response to public messages. This group should include representatives of state government, academia, and the business community. I recommend two state representatives, one academic, one corporate, and one clean air advocate. This group should translate the raw numerical standards into recommended actions. For example, when the ozone level is at such and such a level, people who have trouble breathing or who have heart conditions should not exercise and should walk up stairs slowly. California's approach should be studied, and New Jersey's messages should be coordinated with New York's and Pennsylvania to avoid conflicting communications.

2: The group should meet with one or two representatives of each mass media organization to describe exactly what the public should be understanding and to help media writers compose the message transcripts.

3: Messages should be tested by focus groups of health officers and senior citizens (the former because it deals with individual members of the public; the latter because it is a high risk group). Responses from these test groups should be used for revision.

4: After a message is used, the state should follow it up with a stratified random sample (by age and risk group, weighted for higher risks) to determine the message's effectiveness, and revise accordingly.

**Frances J. Dunston, M.D., M.P.H., Commissioner  
New Jersey Department of Health**

State government's responsibility is two-fold when air pollution levels violate standards and threaten public health. First, the responsibility is to notify the public, giving them the opportunity to take personal action to reduce their exposure. Second, the responsibility is to take measures, as prescribed by law, to reduce unhealthful exposures. Such measures may include temporary closing of industries or limiting of automobile use. NJ's Air Pollution Emergency program partially fulfills these responsibilities.

The NJ Dept. of Health recommends that the state set an ozone Alert level. There is mounting medical evidence that ozone can cause serious injury at levels commonly found in New Jersey during the summer. The current Health Advisory system needs to be augmented by establishing an Alert action level of 0.15 ppm (as recently adopted in California). The adoption of this Alert level would enhance NJDEP's ability to take appropriate legal action to reduce the intensity of exposure to ozone. Appropriate actions for the Alert level include notification to school athletic departments, Public Service Announcements, and encouragement of car pooling or reduced driving days.

The current Ozone Health Advisory system relies on a somewhat passive system for distribution of information via the wire service, United Press International, and the Associated Press. It is just hoped that other media will pick up the message. DOH recommends that NJDEP directly inform all media about ozone and the importance of broadcasting all ozone Advisories, Alerts, etc. DEP staff should develop direct contacts with the few NJ television stations and with major radio stations, and distribute annually updated information packets about the environmental and health effects of ozone. Public Service Announcements (or other communication avenues) for immediate notification to the public should also be developed. Cooperation with the all communications media could produce more effective formats for distribution of ozone information.

Finally, efforts to educate the public and provide behavioral alternatives should be continued on an on-going basis. These activities should include the ozone Health Bulletin mailed each June to all NJ physicians; development of fact sheets, news articles, and pamphlets for distribution to the general public; and active identification and educational outreach to high-risk populations in the state.

**John B. Batchelder, Jr., Senior Air Pollution Specialist  
Health Assessment and Air Quality  
State of California Air Resources Board**

The California Air Resources Board (CARB) has adopted the following revisions into its Air Pollution Emergency Plan: 1) changed the wording in the plan from "oxidant" to "ozone"; 2) formed a new separate Health Advisory level for ozone at 0.15 parts per million, averaged over one hour; 3) transferred the health warning requirements from the 0.20 ppm Stage 1 episode (Alert) to the new Health Advisory level; 4) retained the abatement options at the 0.20 ppm Stage 1 episode level; 5) specified that forecasting of the Health Advisory level not be required in districts with five or fewer occurrences of 0.15 ppm ozone; and 6) deferred consideration of the recommendation to combine oxidant with sulfur dioxide criteria.

In addition, the CARB distributes a sample Ozone Episode information sheet to Air Pollution Control Officers. The sheet describes the effects of ozone exposure and suggests ways individuals may limit their exposures. The sheet is intended to help districts develop their own public notifications for various episode levels.

(Editor's note: Copies of this sample letter and the Proposed Revision of the Air Pollution Emergency Plan for Ozone Episodes, Aug. 1990, are included with the transcript of this Public Hearing.)

This summary was prepared for the Council by Linda Howe, Ph.D., Editorial Services  
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## APPENDIX A

Summary of a Memo dated September 30, 1987

To: Jorge H. Berkowitz  
From: Herbert Wortreich,  
Deputy Director, Division of Environmental Quality  
Subject: Air Pollution Emergencies

In response to your request for recommendations, the Task Group submits the following recommendations and suggestions:

### I. Statute and Code

- A. Statute NJSA 26:2C-26 through 36
  - 1. No changes needed
- B. Code NJAC 7:27-12
  - 1. Revise definitions to encompass additional categories
  - 2. Revise tables 1, 2, 3 to focus on ozone and toxics
  - 3. Include provisions to cover accidental releases
  - 4. Revise procedures for stand-by plans to
    - a. expedite process
    - b. impose fees

### II. Pre-emergency preparations

- A. Amend Administrative Order 38
  - 1. Restore Alert guideline for ozone
  - 2. Coordinate with neighboring states
  - 3. Add guidelines for lead, PM10, toxics, etc.
- B. Forecast and Watch
  - 1. Update procedures
    - a. Written SOP
    - b. Personnel assignments
- C. Stand-by plans
  - 1. Update for quality, relevance, and applicability
  - 2. Augment to address ozone precursors
  - 3. Revise to be consistent with revised guidelines
  - 4. Review periodically for relevance and update
- D. Surveillance and enforcement plans
  - 1. Upgrade for current needs and resources
  - 2. Reactivate for full implementation
- E. Communication network
  - 1. Update and close all links
  - 2. Prepare a guidance and procedure manual
    - a. Distribute to all network elements
    - b. Revise periodically

3. Activate and test network annually
- F. Public Information
  1. Expand and improve mechanisms
    - a. Focus on public resistance caused by
      - (1) Inconvenience
      - (2) Life-style modifications
- III. Emergency actions
  - A. Internal communications
    1. Update communications prototypes
      - a. From the Commissioner to the Governor
      - b. Gubernatorial Proclamations and Orders
      - c. Press releases
    2. Develop new prototypes
      - a. For new emergency guidelines
      - b. For termination of episode
  - B. Set procedures for
    1. External communications
    2. Surveillance
    3. Investigation
    4. Enforcement
    5. Episode monitoring
    6. Episode termination
- IV. Conduct post-emergency debriefing and critique
- V. Hold periodic drills for simulated air emergencies

## APPENDIX B

Listed in this appendix is a compilation of all the recommendations presented to the Clean Air Council by those who gave testimony at its 1991 public hearing on the Air Pollution Watch criteria and guidelines. Recommendations have been organized into five categories: 1) Updating the guidelines, 2) Public education and notifications, 3) Required follow-up action by DEP, 4) Weather forecasting, and 5) Data collection improvements.

### I. Updating the criteria

\*Recommend review and revision of the guidelines for declaring an Air Pollution Watch as well as for attendant regulations in order to reflect the current air pollution situation (Weiner, Wortreich, Mytelka)

\*Recommend reconsideration of the Clean Air Act 0.12 ppm ozone standard in light of recent scientific evidence (Penek)

\*Recommend moving Alert guideline closer to the National Ambient Air Quality Standards (Wortreich)

\*Recommend reinstating the Alert guideline for ozone (Mytelka, Wortreich)

\*Recommend that all guideline changes be coordinated regionally to encourage continued cooperation and to avoid public confusions (Mytelka, Elston, Wortreich)

\*Recommend consideration of guidelines for additional air pollutants, for example, organics (Mytelka), lead, toxics, and particulates (Wortreich)

\*Recommend that guidelines be set solely on grounds of protecting the public's health, i.e. avoid discussion of costs and problems resulting from regional transport during this process (White)

\*Recommend changing the guideline for triggering an Ozone Advisory to 0.095 ppm (White)

\*Recommend changing the 6-8 hour standard for ozone to 0.08 ppm (White)

\*Recommend adoption of 0.15 ppm (one hour) and 0.12 ppm (6 hour) as guideline for Ozone Alert (White, Dunston)

\*Recommend revision of the sulfur dioxide guideline for all stages of an Air Pollution Episode to include a short-term (one hour) level below 0.25 ppm (White)

\*Recommend that guidelines be set low enough to protect the most sensitive groups (White)

\*Recommend retaining current guidelines for the Air Pollution Watch system as they are reasonably protective of public health (Morford)

\*Recommend replacing the smoke shade guideline with one for particulate matter, based on real-time mass measurements using new technologies (Pietarinen)

## II. Public Education and Notifications

\*Recommend emphasizing what individuals can do to reduce emissions, particularly those from vehicles (Weiner)

\*Recommend broadening and increasing public education efforts, perhaps using school recycling programs as a model (Weiner, Wortreich)

\*Recommend updating the wording of press release prototypes (Wortreich)

\*Recommend continued effort to educate physicians about the effects of ozone exposure, making more use of the Journal of the NJ Medical Society (Penek)

\*Recommend speedier and better notification of the public, perhaps using public displays of monitoring information (Elston)

\*Recommend privatizing the public notification system (Elston)

\*Recommend public notifications be made earlier in the day so people can plan activities (White)

\*Recommend public messages be concise and include articulation of specific exposure-avoidance strategies (White, Greenberg)

\*Recommend DEP develop direct contacts with media for better communication to the public of ozone Health Advisories, rather than simply relying on wire services to distribute information (Dunston, Greenberg)

\*Recommend development of a Public Service Announcement format for speedier, better communication of Health Advisories and Alerts (Dunston)

\*Recommend continued efforts to communicate information about ozone exposure to NJ's physicians and distribution of fact sheets and pamphlets to the general public (Dunston)

\*Recommend identification of high risk groups and development of specifically targeted outreach programs to educate them regarding avoidance behaviors (Dunston)

\*Recommend establishing a work group to help DEP create better public announcements. (Greenberg)

### III. Required Follow-up actions by DEP

\*Recommend development of more "proactive" follow-up procedures for actions when predictions of high ozone levels are received (Weiner, Elston)

\*Recommend improving response procedures to make them faster (Elston)

\*Recommend more pollutant specific responses (Elston)

\*Recommend preparation of a guidance and procedure manual to be distributed to all elements of the response network. This manual should include information about who, when, where, and how to activate response procedures. The network should be tested annually. (Wortreich)

\*Recommend preparation of written procedures and personnel assignments, including both actions to be taken during an episode and appropriate ways to terminate an episode (Wortreich)

\*Recommend developing plans for voluntary reductions of automobile use when high ozone levels are predicted, motivated by a series of DEP-sponsored public forums on the subject (Morford)

\*Recommend regional agreements regarding response actions when an Alert is declared (Morford)

\*Recommend Alert response actions include direct notification to school athletic departments, Public Service Announcements, and encouragement of car pooling or reduced driving days (Dunston)

\*Recommend periodic drills for simulated air emergencies, including post-drill critiques and debriefings (Wortreich)

### IV. Weather Forecasting

\*Recommend review of weather forecasting procedures in NJ to ensure the best available, most specific information (Weiner)

\*Recommend improvement of weather system, perhaps through more local surface weather data collection and establishment of an upper air monitoring system (Elston)

\*Recommend more financial support for development of the Rutgers computerized ozone production model (Reiss)

## **V. Data collection improvements**

- \*Recommend improvement of real-time monitoring for air pollutants (Elston, Pietarinen)
- \*Recommend review medical literature for data on the effect of short-term ozone exposure, in order to improve ozone forecasts (Elston)
- \*Recommend monitoring and prediction of pollution episodes be based on computer models, like those used in the nuclear industry; this would help avoid missing plumes of air pollution that go in between current monitoring stations (Elston)



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## Effect of low concentrations of ozone on inhaled allergen responses in asthmatic subjects

NESTOR A. MOLFINO STANLEY C. WRIGHT IDO KATZ  
SUSAN TARLO FRANCES SILVERMAN PATRICIA A. MCCLEAN  
JOHN P. SZALAI MARK RAIZENNE ARTHUR S. SLUTSKY  
NOE ZAMEL

The relation between inhalation of ambient concentrations of ozone and airway reactivity to inhaled allergens may be important in asthma, since both agents can produce inflammatory changes in the airways. Seven asthmatic patients (mean age 40 [SD 13] years), with seasonal symptoms of asthma and positive skin tests for ragweed or grass, took part in a study to investigate whether exposure to low concentrations of ozone potentiates the airway allergic response. The patients were studied during 4 separate weeks in the winter. In each week there were 3 study days: on days 1 and 3 methacholine challenges were carried out; and on day 2 the subject received one of four combined challenges in a single-blind design—air breathing followed by inhalation of allergen diluent (placebo); ozone followed by inhalation of allergen diluent; air followed by allergen; or ozone followed by allergen. The ozone concentration was 0.12 ppm during 1 h of tidal breathing at rest, and allergens were inhaled until the forced expiratory volume in 1 s (FEV<sub>1</sub>) had fallen by 15% (PC<sub>15</sub>). There were no significant differences in baseline FEV<sub>1</sub> after exposure to ozone but PC<sub>15</sub> was significantly reduced when allergen was preceded by ozone inhalation: the mean PC<sub>15</sub> after air was 0.013 (SD 0.017) mg/ml compared with 0.0056 (0.0062) mg/ml after ozone ( $p=0.042$ ). Thus, low ozone concentrations, similar to those commonly occurring in urban areas, can increase the bronchial responsiveness to allergen in atopic asthmatic subjects. This effect does not seem to be the result of changes in baseline airway function.

### Introduction

High levels of air pollution have been related to increasing mortality and morbidity rates from asthma.<sup>1,2</sup> Ozone (O<sub>3</sub>) is the major oxidant in the photochemical smog<sup>3</sup> and it increases bronchial responsiveness in both healthy<sup>4</sup> and asthmatic<sup>5</sup> subjects. The effects are dose dependent,<sup>6</sup> and Dimeo and colleagues<sup>7</sup> calculated that the threshold concentration of ozone that caused hyperreactivity was between 0.20 and 0.40 ppm, which is higher than concentrations usually encountered in the environment. It is still not clear whether lower concentrations of ozone directly trigger a bronchoconstrictive response, or whether they increase the responsiveness of the airways to other agents such as allergens. Koenig and colleagues<sup>8</sup> found no difference in peak expiratory flow rates and pulmonary function tests after exposing ten asthmatic patients and ten normal subjects to an ozone concentration of 0.12 ppm for 60 min at rest. By contrast, Goldsmith and Nadel<sup>9</sup> found that bronchoconstriction occurs in some people at rest on exposure to an ozone concentration as low as 0.10 ppm, which may be detected in large urban centres. Relations between asthma attacks, ambient temperature, and levels of pollutants have been reported in Los Angeles, USA,<sup>10</sup> and in Canada.<sup>11,12</sup>

Allergens are well-known triggers of asthma and there is increasing evidence that allergic mechanisms are more important in asthma than had been previously thought.<sup>13</sup> Furthermore, atopic patients may have more severe

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asthma<sup>14</sup> and a higher mortality rate than non-atopic asthma patients.<sup>15</sup>

We have investigated whether low ozone concentrations increase the airway response to allergens in atopic asthmatic patients.

### Subjects and methods

Ten patients with mild, stable, atopic asthma and clinically confirmed seasonal histories were enrolled in the study after they signed a consent form approved by the University of Toronto Ethics Committee. Three patients were excluded from the study and their results were not analysed: in two the variation in baseline forced expiratory volume in 1 s (FEV<sub>1</sub>) did not meet the day-to-day protocol criteria, and in the other an upper respiratory infection affected her response to the combination ozone/allergen. Demographic and screening data for the seven patients whose results were analysed are shown in table 1. Six subjects had never smoked, and patient 3 was an ex-smoker. Their mean age was 40 (SD 13) years. The allergen used for testing was ragweed in patients 1-6 and grass in patient 7.

Changes in FEV<sub>1</sub> and partial expiratory flow volume curves<sup>16</sup> were used to determine the effects of the four different combined challenges and the provocative concentration of methacholine that produced a 20% drop in FEV<sub>1</sub> (PC<sub>20</sub>) or a 40% fall in flow at 40% of vital capacity (PC<sub>40</sub>V<sub>40</sub>). Since the effects of previous inhalation of ozone on the airway response to allergens were unknown, during allergen provocation, PC<sub>20</sub> (the dose that elicited a 15% drop in FEV<sub>1</sub>) was used instead of PC<sub>40</sub> for safety reasons.

A sample size of ten patients was chosen based on the number of subjects needed to measure pulmonary function differences at 0.12 ppm in a previous study.<sup>9</sup> The study was carried out during the winter months only (December-March, 1988-90). Ozone concentrations in Toronto each month are given in table II. For each patient the 13 study days included a first day to verify the seasonal history of asthma, and to confirm a positive methacholine test and positive skin responses to grass or ragweed. The other 12 days were divided into four 3-day study sets, during which all the patients had to attend the laboratory at the same time of day.

On days 1 and 3 of each set methacholine challenge was carried out and on day 2 of each set, in a single-blind fashion, the subjects were exposed to one of the following combinations: 1 h of breathing air followed by inhalation of allergen diluent (ie, placebo); 1 h breathing air followed by inhalation of allergen; 1 h of breathing ozone followed by inhalation of allergen diluent; and 1 h of breathing ozone followed by inhalation of allergen. The criterion for entry to either day 1 or day 2 was a baseline FEV<sub>1</sub> within 10% of that measured on day 1 of the first week. If this was not possible, the entire study set was rescheduled. Since day 3 of each set was the post-intervention day no criteria for baseline were established. On all second days, after the provocative combination was given, FEV<sub>1</sub> and partial flow volume curves were measured in the laboratory for 6 h.

The sequence and time between allergen challenges are shown in table 1. At the start of the study a randomised design was used for

TABLE II—OZONE CONCENTRATIONS IN TORONTO\* DURING STUDY PERIOD AND IN OTHER LARGE NORTH AMERICAN CITIES†

	1988	1989	1990
<i>Toronto</i>			
January	0.033	0.029	0.028
February	0.044	0.030	0.030
March	0.049	0.040	..
April	0.057	0.041	..
May	0.117	0.054	..
June	0.116	0.139	..
July	0.159	0.191	..
August	0.111	0.088	..
September	0.063	0.064	..
October	0.060	0.051	..
November	0.029	0.036	..
December	0.028	0.025	..
<i>City</i>	<i>Ozone concentration</i>	<i>City</i>	<i>Ozone concentration</i>
Anaheim	0.24	New York	0.18
Atlanta	0.17	Philadelphia	0.20
Baltimore	0.19	Washington	0.18
Boston	0.17	Chicago	0.22
Detroit	0.16	Houston	0.22
Los Angeles	0.33		

\*Monthly maximum reading 1 h concentration in ppm: Ontario Ministry of the Environment-Air Resources Branch.

†Second daily maximum 1 h concentration in ppm: National Air Quality and Emissions Trends Report, 1988, United States Environmental Protection Agency.

allocation of study days. However, in the second subject studied (excluded from data analysis) there was an increase in asthma symptoms and airway reactivity that lasted for 3 months after ozone-allergen exposure. To avoid this possible long-term potentiation of airway reactivity, we administered ozone/allergen as the last challenge combination for the rest of the subjects. No other subject had this long-lasting increase in asthma symptoms after ozone/allergen.

Methacholine challenge was carried out according to the protocol of Cockcroft et al.<sup>17</sup> At least five total and partial flow-volume curves were obtained as baseline in each subject and all the American Thoracic Society recommendations for inhalation provocation tests<sup>18</sup> were followed. The Wright nebuliser was used with compressed air at 8 l/min to obtain an output of 0.13-0.16 ml/min. Doubling concentrations of methacholine were inhaled through a mouthpiece during tidal breathing for 2 min with the patient wearing a nose-clip. The response was expressed as the concentration that caused a 20% fall in FEV<sub>1</sub> in non-cumulative units.

For ozone and air (placebo) exposure at least three total and partial flow volume curves were obtained before and immediately afterwards. Seated subjects breathed for 1 h at rest in an 800 litre environmentally controlled chamber. On test days (day 2), chamber and environmental ozone concentrations were measured with a Bendix 8002 ozone analyser. Ozone was produced by passage of 100% oxygen (O<sub>2</sub>) through an ultraviolet ozone generator and concentrations were maintained at 0.12 (SD 0.02) ppm.

TABLE I—ANTHROPOMETRIC AND SCREENING DATA

Subject	Sex	Age (yr)	Drugs*	FEV <sub>1</sub> †	PC <sub>20</sub> ‡	Test order§	Time (wk)¶
1	M	36	S, C	92	0.45	P, A, O, OA	4
2	M	32	F	107	0.60	O, A, P, OA	36
3	F	49	S, T, C, B	65	0.50	O, A, P, OA	4
4	F	64	S	98	0.19	P, A, O, OA	2
5	M	43	None	113	0.23	P, O, A, OA	1
6	F	35	S, T, AH	99	0.70	P, O, OA, A	2
7	M	21	S	77	0.03	P, A, O, OA	3

\*AH = antihistamine; B = beclomethasone; C = sodium cromoglycate; F = fenoterol; S = salbutamol; T = theophylline.

†% predicted

‡in mg/ml = methacholine concentration causing 20% fall in FEV<sub>1</sub>.

§P = air/diluent; A = air/allergen; O = ozone/diluent; OA = ozone/allergen.

¶Time between the two allergen tests.

TABLE III—PC<sub>20</sub> METHACHOLINE ON DAYS 1 AND 3 (mg/ml)

Subject	Placebo		Ozone		Allergen		Ozone/allergen	
	Day 1	Day 3	Day 1	Day 3	Day 1	Day 3	Day 1	Day 3
1	0.45	1.00	0.45	0.23	0.82	0.19	0.43	0.20
2	0.60	0.25	0.80	0.30	1.30	0.75	0.27	0.29
3	1.00	0.90	1.30	2.00	1.10	0.30	0.60	0.50
4	0.19	0.25	0.25	0.31	0.25	0.32	0.50	0.40
5	0.65	0.18	0.32	0.55	0.30	0.55	0.38	0.45
6	0.70	2.40	1.20	2.10	1.60	1.30	0.65	1.35
7	0.03	0.02	0.03	0.03	0.02	0.01	0.05	0.05
Mean	0.52	0.71	0.62	0.79	0.77	0.52	0.41	0.46
SD	0.30	0.77	0.43	0.81	0.55	0.39	0.19	0.39

p > 0.50 for all test combinations between days 1 and 3 by one group / tests on change scores calculated as

$$\frac{PC_{20} \text{ day 1} - PC_{20} \text{ day 3}}{PC_{20} \text{ day 1}}$$

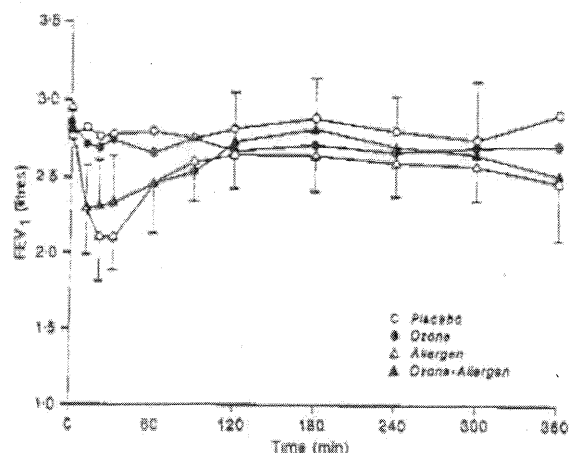


Fig 1—Time course of responses to the four challenges.  
Values = mean and SEM.

Temperature and humidity were measured by a 'Hygrotest' 6200 and were maintained within the same range on all test days. Oxygen (Ametek S-3A1) and carbon dioxide (Ametek CD-3A) concentrations were monitored and kept at approximately ambient levels. For air (placebo) exposures, an initial small concentration of ozone was added to the chamber to produce the characteristic odour. Otherwise, the concentration of ozone during this sham exposure was below 0.005 ppm throughout the challenge.

After the chamber exposure, diluent or allergen challenges were carried out. The initial concentration of allergen extract for inhalation was determined for each individual from the prick tests with the same extract and the  $PC_{20}$  methacholine on the entry day.<sup>20</sup> The starting concentration of allergen for inhalation was two doubling concentrations below that predicted to cause a 20% fall in  $FEV_1$ . However, when the  $PC_{20}$  was known from the previous allergen challenge, the initial dose used was two doubling concentrations below this dose. The early response was defined as a 15% drop in  $FEV_1$  within 60 min of inhalation. Doubling concentrations of allergen were inhaled for 2 min and  $FEV_1$  was measured 10 min after each inhalation. If the  $FEV_1$  had fallen by 10% or more, it was remeasured every 10 min until no further fall in  $FEV_1$  was observed before inhalation of a further concentration was allowed. When the  $FEV_1$  had fallen by 15% or more, no further allergen was given and  $FEV_1$  was measured at 20 min, 30 min, then every 30 min to 2 h, then every 1 h to 6 h. On a placebo day, the patient had three 2 min inhalations of the diluent (phosphate-buffered saline with 1.4% benzyl alcohol) followed by measurements of  $FEV_1$  according to the same schedule as that followed on the allergen days.

Repeated measures analysis of variance (ANOVA) was used for testing the effects of exposure on the ratio scale dose-responses. To test the hypothesis that a mean value is discernibly different from zero, the one group  $t$  test (two-tailed) was used. Before the data were submitted to ANOVA or  $t$  test, the distributions of the postulated populations were scrutinised by means of the Shapiro-Wilk test of normality. Whenever a deviation from a Gaussian distribution was found, transformation of the dependent variable, if possible, was attempted. For all statistical tests a probability of 0.05 was taken as significant. The SAS version 6.03 was used.

## Results

The mean (SD) indoor concentrations of ozone in our laboratory during the study days were 0.006 (0.005) ppm on the ozone/allergen days and 0.008 (0.007) ppm on the allergen days. The mean of the hourly highest outdoor levels of ozone on the study days was 0.022 (SD 0.009) ppm (range 0.008–0.049 ppm; Ontario Ministry of the Environment, Air Resources Branch).

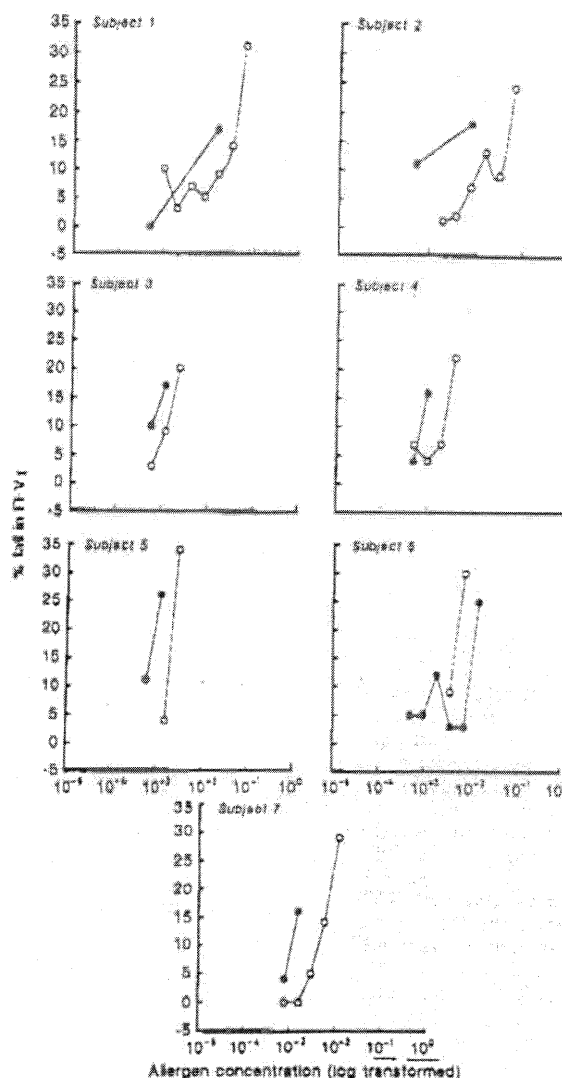


Fig 2—Dose-response relation to allergen in each subject after breathing air without ozone (○) or air with ozone added (●).

The four different exposures on day 2 (placebo, ozone, allergen, and ozone/allergen) and the two levels of before and after factor ( $FEV_1$  and  $V_{50}$ , before and after the ozone chamber or before methacholine challenge on day 1 and day 3) yielded a  $4 \times 2$  repeated measures design. Analyses of variance did not yield statistically significant interactions or main effects on these baseline values.

Results from methacholine challenges before and after the different provocative combinations showed no significant changes even if the more sensitive  $V_{50}$  was used (table III).

The four different exposures gave a single factor repeated measures design. Although allergen alone produced a greater early response than ozone/allergen, no statistically significant main effects were shown by ANOVA (fig 1). Moreover, no mean change score for a condition was significantly different from zero. When allergen was given alone, three of the seven subjects had a late response measured by a drop of 15% in  $FEV_1$  3–6 h after the inhalation. Similarly, three subjects showed a late response after the ozone/allergen combination.

Fig 2 shows the dose-response curves relating the percentage fall in  $FEV_1$  to dose of allergen after air or ozone.

The distributions of the provocative concentrations  $PC_{15}$  and  $PC_{40}V_{40}$  allergen were significantly skewed and natural-log transformation was applied. Mean (SD)  $PC_{15}$  was 0.013 (0.017) mg/ml after allergen exposure compared with 0.0056 (0.0062) mg/ml after ozone/allergen ( $p=0.041$ ); for  $PC_{40}V_{40}$  the corresponding values were 0.011 (0.014) mg/ml and 0.0053 (0.0059) mg/ml ( $p=0.032$ ).

### Discussion

Our results suggest that low ozone concentrations, similar to those commonly found in large urban centres (table II), can increase the bronchial responsiveness to allergens in atopic asthma patients without affecting baseline pulmonary function. After exposure to 0.12 ppm ozone for 1 h at rest, the dose of inhaled allergen necessary to elicit the same early allergic response was half that for allergen preceded by air inhalation. We used two indices to assess the effect of allergen on pulmonary function.  $PC_{15}$  is based on FEV<sub>1</sub> and thus reflects the resistance of large and small airways,<sup>21</sup> whereas  $PC_{40}V_{40}$  is more sensitive because it is not affected by a previous deep inhalation and reflects more specifically the dynamics of the small airways.<sup>14</sup>

Ideally the study should have had a randomised, double-blind, crossover design. However, since we were concerned that the combined ozone and allergen challenges could lead to excessive airway obstruction, we changed the study design so that the combined ozone/allergen challenge was the last exposure. Thus, when a patient was exposed to the combination we already knew his or her response to each of these agonists. It is unlikely that there was a carryover effect of the first allergen challenge, since no methacholine challenges (carried out before and after each step) showed a significant change. Bel et al<sup>22</sup> have shown no significant change in allergen response repeated 2 weeks later and no differences in the airway response to allergen challenges repeated weekly (unpublished). This evidence suggests that, despite the non-randomised design, our results cannot be explained as secondary to the sequence of allergen challenges.

We found no statistically significant bronchoconstrictor effect related to ozone exposure alone in comparison with the placebo combination (air/diluent). Therefore, although four of the seven patients received ozone in two successive weeks, we do not think that this sequence had an important bearing on the results. Others have shown that a long-term effect on pulmonary function<sup>17</sup> requires frequent small doses of ozone or a single dose much higher than that we used. The effects on airway responsiveness after only one exposure to 0.6 ppm ozone concentration last from 24 h<sup>23</sup> to 3-4 days.<sup>3</sup> It is unlikely that repeated exposure to ambient ozone concentrations was sufficient to cause the changes we observed, since these levels never exceeded 0.05 ppm during the study period (table II). Furthermore, on the study days, the mean indoor concentrations were as low as 0.006 ppm and 0.008 ppm.

The concentrations of ozone needed to change human airway function are controversial. It has been reported that concentrations as low as 0.1 ppm can, in some individuals, induce bronchoconstriction<sup>6</sup> as well as biochemical and cellular changes in bronchoalveolar lavage fluid.<sup>17</sup> Our results accord with those of Dimeo, Koenig, and colleagues,<sup>7,8</sup> in that we failed to find significant changes in air flow after 1 h exposure at rest to ozone at 0.12 ppm. However, at concentrations higher than those commonly occurring, ozone seems to produce inflammation in

animals,<sup>24</sup> changes in airway reactivity in subjects at rest,<sup>24,25</sup> and increases in airway reactivity to antigen inhalation in dogs.<sup>27</sup>

The mechanisms producing the changes we observed are not clear. It is possible that the low concentrations of ozone we used might have been associated with a low grade inflammatory reaction with vasodilatation and greater absorption of allergen.<sup>27,28</sup> This hypothesis would accord with the inflammatory response previously reported after exposure to ozone<sup>24-26</sup> which seems to act through epithelial substances that attract neutrophils; these cells release cyclo-oxygenase and lipo-oxygenase substances.<sup>27,29</sup> It is also possible that ozone produced a short-lived increase in non-allergic bronchial responsiveness, which in turn might have led to a change in response to allergen, shifting the dose-response curve to the left without affecting its slope (fig 2). The effects of ozone on airway methacholine responsiveness can be detected as early as 90 min after the exposure<sup>3</sup> and the biochemical changes in bronchoalveolar lavage fluid can persist as long as 18 h.<sup>17</sup> Our study was not designed to investigate this mechanism and we could have missed this effect because we carried out the methacholine challenges 24 h after the exposure to ozone. Alternatively, exposure to ozone could have produced an immunological change<sup>30</sup> or an increased influx of specific cells that bring about the response to the antigen.<sup>29</sup>

Whatever the exact mechanisms, these findings might help to explain the rise in hospital admissions of asthmatic subjects<sup>11,13</sup> that occurs with increases in air pollution in urban areas where an oxidant such as ozone is a part of the environment. These results may have important public health implications for air quality standards, since the dose of ozone used was similar to concentrations present in many urban areas. However, we suggest that the experiment be independently replicated before major policy changes on air quality are contemplated.

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## HIV in pregnant women and their offspring: evidence for late transmission

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To assess the role of maternal viraemia in vertical transmission of HIV and the extent to which viraemia occurs during the various stages of pregnancy, we have attempted to detect human immunodeficiency virus (HIV) in 44 pregnant HIV-1 infected women during 47 pregnancies (30 continued, 17 aborted) and in 30 children and 12 fetuses. Infectious HIV was detected at some time during pregnancy in 59% of women from plasma and in 83% from either peripheral blood mononuclear cells or plasma. HIV was not isolated from any of the newborn babies (0/27) at birth. The mothers had a significantly higher frequency of viraemia during pregnancy than their children had by 6 months of age ( $p=0.002$ ); by this time HIV was recovered from 5 (26%) of 19 infants. HIV was not detected by virus isolation, in-situ hybridisation, or polymerase chain reaction (PCR) in 10 fetuses; the other 2 fetuses were positive either by in-situ hybridisation or by PCR but neither result could be confirmed in a second organ or by the other methods of detection. The findings show that there is no consistent spread of HIV across the placenta during maternal viraemia, and indicate that in most cases transmission occurs close to or at delivery.

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### Introduction

Paediatric human immunodeficiency virus (HIV)-1 infection is acquired largely from the mother either in utero or at birth;<sup>1</sup> the relative risk of vertical transmission has been

estimated to be between 15 and 40%.<sup>2-4</sup> What is the frequency of transmission in utero (and at which stage of pregnancy) compared with that during birth when there is exposure to maternal blood and body fluids; and which factors are important for vertical transmission? Maternal antibodies to regions on gp120 have been associated with protection against transmission.<sup>5-8</sup> Although maternal stage of infection is believed to be important in vertical transmission of HIV,<sup>9</sup> the role of viraemia and the extent to which it occurs during pregnancy are not known.

We have tried to answer some of these questions in a study of the relation between HIV in offspring and HIV viraemia in women during pregnancy, at delivery or abortion, and 6 months later.

### Subjects and methods

#### Subjects

In Sweden nearly all pregnant women have been offered HIV-testing since 1987. All known HIV-1 seropositive women in Sweden who gave birth to a child between Jan 1, 1987 and March 16, 1991, took part in this study, which was approved by the ethics committee of the Karolinska Institute. Women primarily seeking abortion were often not included in the study.

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# te(E.P.A. Urged to Broaden Air Pollution Studies

Report says agency should consider noncancer risks.

**A**N Environmental Protection Agency report suggests that the agency allocate more resources to the study of noncancer health risks of air pollution.

The report said lack of research makes it difficult to assess these risks. The report, "Toxic Air Pollutants and Noncancer Health Risks: Screening Studies," was produced to help the agency decide how to allocate resources to carry out the Clean

## Air Act.

As originally passed in 1970, the act focused on cancer-causing effects of air pollution, said Dr. Ila L. Cote, of the E.P.A.'s Office of Research and Development in Durham, N.C. But she said air pollution could also cause emphysema, birth defects and a variety of other problems.

## State and Local Data

In 1987, E.P.A. researchers studying noncancer health risks began assembling data from state and local government, medical literature and environmental groups. They used computer models and drew on air monitoring data and estimates of public health impact.

"We found a lot of gray areas where concentrations of toxics nationally weren't low enough to be

safe, but weren't high enough so that it was an obvious problem," said Dr. Cote.

Dr. Cote said she and her colleagues were concerned that the lack of data provided a fuzzy picture of noncancer health risks, especially on a local level. They were concerned that the combined effects of multiple sources of toxic air pollutants in small areas might be ignored in their national study.

To examine this issue, they constructed a computer model of an industrialized county, based on Cook County, Ill., where government and environmental groups had gathered good data on air pollution.

## A Model County

They did not have precise emission data for all the facilities in the county,

so they matched the facilities in Cook County with similar ones in other parts of the country that had known emissions. The model that resulted was not exactly like Cook County but it was a model of an industrialized county.

The results showed that multiple sources of toxic air pollution, chemical mixtures and exposure to large short-term emissions should cause concern about noncancer health risks.

But again they were frustrated by a lack of data. These means of exposure and many pollutants that may have posed noncancer health risks were not monitored, Dr. Cote said. "It's like the guy who only looks under the light to find his keys, when he knows he has dropped them outside the light," she said.

# Citing Medical Evidence on Smog, California Lowers Threshold for Its Health Alerts

By ROBERT REINHOLD  
Special to The New York Times

LOS ANGELES, Sept. 13 — Citing cumulative medical evidence that smog can cause serious injury even to healthy, vigorous adults, the California Air Resources Board ordered all local pollution control districts in the state today to issue health alerts at much lower smog levels than before.

The unanimous order of the agency's board gave new urgency to the state's smog problem by broadening health concerns beyond children and the elderly to adults who exercise on days of high ozone pollution.

The action was based on a recent review of 13 medical studies completed in the last decade that found short- and long-term health damage caused by smog. "All showed that even vigorous adults in good health who exercise strenuously, like jogging five miles or playing basketball, are affected by alert levels below what we thought adequate until now," said Bill Sessa, chief spokesman for the powerful agency,

whose ruling on automobile tail-pipe emissions have had national reverberations. The agency oversees 41 air pollution control districts throughout California.

## More Alerts in More Areas

The alerts apply to ozone, the main and most hazardous air pollutant, which is formed when hydrocarbons from automobile exhaust and nitrogen oxides from industrial processes combine under sunlight. Ozone is known to cause sensations of breathlessness, decreased lung capacity, coughing, nose and throat irritation. It also aggravates respiratory illnesses like emphysema.

The order is expected to mean that smog alerts would now be issued in parts of the state that have never had them before and that had considered their air comparatively clean, including southern Santa Barbara County, Ventura County and San Diego County.

But the largest effect will be in the highly polluted Los Angeles Basin,

## Pollution is harming more Californians, the state says.

where the local South Coast Air Quality Management District already issues more than 60 smog alerts during the summer ozone season.

Under a smog alert, schools are notified and urged to reschedule athletic practices, and sensitive people are advised to remain indoors and in some cases consult their doctors. In addition, some car pooling plans are activated by employers when smog alerts are expected.

The state agency estimated that the new lower standard would result in 172 alerts a year in the Los Angeles Basin alone, based on smog trends over the last three years. It also expects from it

to 14 warnings a year in the Sacramento area and the increasingly urban San Joaquin Valley in the central part of the state, which has been largely agricultural.

Under the ruling, which is binding on local agencies, alerts must be issued when one-hour levels of ozone reach 155 parts per million of air, substantially lower than the current standards of 172 parts per million. The state's long-range goal, through tighter pollution controls and other measures under way, is to reduce ozone to 60 parts per million on the worst days. The new rules are expected to be in place by the next smog season, next summer.

A spokeswoman for the air quality district here said that the high for ozone was recorded at 34 parts per million in 1989 and 35 parts in 1988.

The agency's review of medical evidence suggested that ozone — which is formed from hydrocarbons and nitrogen oxides — causes health problems for some people at levels lower than we previously believed, and those people need added protection that an earlier warning would provide.

more insidious than earlier believed, it is believed to speed aging of the body.

For example, three studies conducted at the University of California at Santa Barbara in 1983, 1984 and 1988 found decreased lung function and feelings of chest tightness among 24 men and 14 women, all healthy, at ozone levels below 2 parts per million. Another study by the Environmental Protection Agency in 1983 found increased coughing, chest pain and shortness of breath at ozone levels as low as .12 parts.

"Not everyone responds to the same amount of pollution in the same way," said the chairwoman of the state board, Janine Sharpless, in a statement. "What can be a minor irritation at best for one person can be a serious health threat for others. The increased number of health warnings does not mean smog is getting worse but that it causes health problems for some people at levels lower than we previously believed, and those people need added protection that an earlier warning would provide."



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