Pace Environmental Law Review

Volume 16
Issue 1 *Winter 1998*

Article 3

January 1998

Scientific Research for Ozone and Fine Particulate Standards

George D. Thurston

Scientific Research for Ozone and Fine Particulate Standards

DR. GEORGE D. THURSTON*

My talk today will focus on the new air quality standards and their health benefits.

EPA has written a Criteria Document[1] on this topic that is the size of several Manhattan phone books, full of information. I will try to condense all the information into a ten minute talk.

As background, the air quality standards, the foundation of the nation's air pollution control program, establishes acceptable air levels as targets, or goals, to ensure the protection of public health and welfare. The Clean Air Act and its Amendments (CAAA)[2] requires national air quality standards to be set at levels sufficient to protect the public's health, including the sensitive populations, with an adequate margin of safety.[3] The legislative history, confirmed by the courts, indicate that the national air quality standards must be set strictly on the basis of public health and welfare, with-

* George D. Thurston is an Associate Professor of Environmental Medicine and Director of Community Outreach, Department of Environmental Medicine, New York University School of Medicine. Dr. Thurston serves as an advisor to the State of New York on air quality matters. He has served on the Air Management Advisory Committee of the New York Department of Environmental Conservation (DEC) since its formation in 1991. He is presently on the National Academy of Science's Committee on Health Effects of Incineration. In 1997, Dr. Thurston was Chairman of Canada's Health and Environment Panel for the Health. He received his A.B., Sc.B. Envtl. Engineering from Brown University in 1974; M.S. Envtl. Health Science, Harvard University, 1978; Sc.D. Envtl. Health Science, Harvard University, 1983.

1. *See* U.S. ENVTL. PROTECTION AGENCY, REP. NOS. EPA/600/AP/93/004a-c, *Air Quality Criteria Document for Ozone and Related Photochemical Oxidants* (1993).

2. Clean Air Act (CAA), § 108, 42 U.S.C. § 7408 (1996).

3. *See* Lead Industries Ass'n v. EPA, 647 F.2d 1130, 1161-62 (D.C. Cir. 1980).

out consideration of cost or technological feasibility to assure that Americans' rights to breathe clean air are protected.

The Clean Air Act requires that the EPA review these standards every five years.[4] It is not something that the EPA just decided to do because they are mean and they want to get industries to clean up. Congress wrote this law and said, "You will do it," although I do not think that Congress will bring the subject up during its hearings on this matter. Indeed, the American Lung Association has had to litigate a few times[5] to get the EPA to revise the particulate matter standard. It has been part of the legislative and legal history since the standards were set in 1970. We have had new standards set, but they have also been revised. Occasionally, EPA review determines that things are fine the way they are. In 1979, EPA relaxed the original ozone standard from 80 parts per billion (ppb) (as an annual maximum) to 120 ppb. However, it usually takes much longer than the legislated five years between administrative reviews.

What I am going to talk about today is the broad body of scientific evidence that indicates that the old National Ambient Air Quality Standards (NAAQS) for ozone[6] and particulate matter (PM) were not sufficient to protect public health. These studies are largely consistent, showing similar results for various geographic locations, different methodologies, different times and different pollution sources.[7]

---

4. *See id.*

5. A court order entered in *American Lung Assoc. v. Browner* required EPA to publish its final decision on the review of the particulate matter NAAQS by July, 1997. *See* American Lung Assoc. v. Browner, 884 F. Supp. 345 (D. Ariz. 1994).

6. The ozone air quality primary and secondary standards were originally set at a level of 0.12 parts per million (ppm), with a one-hour averaging time and a single expected-exceedance form. *See* 40 C.F.R. pt. 50, Appendix H. The standards are attained when the expected number of days per calendar year with a maximum hourly average concentration above 0.12 ppm is equal to or less than one averaged over three years. *See id.* These standards were reviewed in March, 1993, and EPA published a notice of final decision not to revise the existing primary and secondary standards at that time. *See* 58 Fed. Reg. 13,008 (1993).

7. *See* J. SCHWARTZ, HEALTH EFFECTS OF AIR POLLUTION FROM TRAFFIC: OZONE AND PARTICULATE MATTER *in* HEALTH AT THE CROSSROADS: TRANSPORT POLICY AND URBAN HEALTH (T. Fletcher & A.J. McMichael eds. 1997).

Now, what is ozone air pollution? Ozone (O_3) is an invisible irritant gas that is formed in the atmosphere in the presence of sunlight and air pollutants.[8] So, it is not what we call a primary pollutant. It does not come right out of the stacks of a pollution source or out of the tailpipe of a car. Rather, it is nitrogen oxides, hydrocarbons and other pollutants that are emitted by these sources. These chemicals, in the presence of sunlight (which provides the energy for reactions of these primary pollutants), are then converted to a secondary pollutant, ozone.[9] The precursor pollutants, like nitrogen oxides and hydrocarbons, come from a variety of sources, like automobiles, power plants and industry.[10]

Particulate matter, or particles, are composed of two types of particles: primary and secondary particles.[11] Some particles come directly out of the tailpipes and stacks, and those are primarily carbonaceous particles. Then there are secondary particles that are formed in the atmosphere from gaseous pollutants, such as sulfates from sulfur dioxide.[12] Power plants emit sulfur dioxide that interacts in the atmosphere with other pollutants, sunlight and moisture, and is then converted into sulfuric acid droplets to become a sulfate particle, or aerosol, that can have adverse health effects when inhaled.

Prior to 1997, the ozone standard was 120 ppb daily, as a one-hour maximum.[13] That is an important aspect to understand. The old standard was a one-hour maximum standard.

---

8. *See* U.S. ENVTL. PROTECTION AGENCY, REP. NO. EPA/400/K/93/001 (1993).

9. *See* U.S. Natl. Oceanic and Atmospheric Admin., Reports To The Nation On Our Changing Planet: Our Ozone Shield (1992), (visited Dec. 4, 1998) <http://www.ogp.noaa.gov/OGPFront/mono2.html>.

10. *See* U.S. ENVTL. PROTECTION AGENCY, REP. NO. EPA/454/R/97/013, NATIONAL AIR QUALITY AND EMISSIONS TRENDS REPORT, (1996).

11. *See* U.S. ENVTL. PROTECTION AGENCY, REP. NO. EPA/600/P-95-001A-C, AIR QUALITY CRITERIA FOR PARTICULATE MATTER (1996), *available in* NAT'L TECHNICAL INFO. SERV., DOC. NO. PB-96-168224 (1996).

12. *See* NATIONAL RESEARCH COUNCIL, COMM. ON MED. AND BIOL. EFFECTS OF ENVTL. POLLUTANTS, AIRBORNE PARTICLES (1979).

13. *See* 58 Fed. Reg. 13,008 (1993).

The new standard is an eight-hour average.[14] The equivalent eight-hour average to the old standard's one-hour maximum value is about 90.

Some people look at the new ozone standard and say, "Wow! It went from 120 to 80." Believe me, it has often been said, "This is a big reduction, going down a third in the standard," but not really. You are also changing the averaging time, and when you do that, the equivalent to the 120, in an eight-hour basis, is about 90. So, the ozone standard is only down by about ten percent.

The old PM standard was set for particles less than ten microns, or micrometers, in diameter.[15] A hair is about 100 microns in diameter. So these are particles smaller than one-tenth that of the diameter of the average human hair, which are very small particles. The old daily maximum PM_{10} standard, in comparison, was 150 micrograms per cubic meter.

The new $PM_{2.5}$ standard went down to 65 $\mu g/m^3$, but these are a three year average of the fourth highest value. By doing so, we are allowing more exceedances. The regulation is not based on a one year time frame anymore. As a result, there is not as much of a tightening as it might first appear.

Additionally, the new PM standard for particles is less than 2.5 microns in diameter.[16] These are the particles that get into the deepest part of the lungs. Actually, I should have said that the PM_{10} are those particles that are small enough to get past the trachea and enter the lungs. Therefore, it makes sense to regulate these smaller particles as opposed to all particles, since not all particles can reach the lungs. Note, that the original PM standard was set for total suspended particles (TSP) in the late 1970s, and it was not until the mid-80s that it was changed to a PM_{10} standard.[17] Now we are going even lower, to a $PM_{2.5}$ standard, which moves us closer and closer to the particle group that has the greatest

14. National Ambient Air Quality Standards for Ozone, 40 C.F.R. pt. 50 (1997).

15. *See id.*

16. *See id.*

17. Ambient Air Quality Surveillance for Particulate Matter, 52 Fed. Reg. 24,736 (1987).

impact on health. Thus, the EPA regulations are focusing more and more on the portion of PM that is thought to pose the greatest health risk.

To review, the current standard is a three year average of the 99th percentile.[18] One percent of the year is approximately 3-4 days, so, again, you are letting some days go by as opposed to before, and the 15 microgram annual average is again a three year average. Now it is the 98th percentile. The values are averaged over three years. Also, $PM_{2.5}$ is a subset of PM_{10}. So, you cannot look at the numbers and directly evaluate the percent reduction, because the way that the standard is applied has changed as well.

With regard to the ozone standards, if we look at the old standard you will find that we are already out of compliance in the New York area. Ozone pollution is very focused in the Northeast and in Southern California. The problem is that these are regional pollutants. The ozone and $PM_{2.5}$ problems in New York City are caused not only locally, but upwind in New Jersey, Pennsylvania, Ohio and Indiana. It is very difficult for East Coast locations to be in compliance without controlling the upwind sources that also have an adverse impact. This is the reason behind EPA/NESCAUM negotiations for an agreement concerning transported pollution. They are trying to get the upwind places, like the Midwest, to clean up also.

The new standard will help achieve that goal. Places that are out of compliance under the new standards in the Midwest will now have to take action. We will see more progress in cleaning up the air in the Northeast than before, because Midwest sources that are causing part of the problem will now also have to clean up.

Of course, another way to look at this is that, based on the new health studies, we now know that the health of people in the Midwest is being threatened by the pollution they cause when they are in non-compliance with the new ozone and $PM_{2.5}$ standards. They too will now be protected. To support this view, I will show you what is happening among chil-

18. *See id.*

dren by using the results of studies like the one that we recently conducted at a camp for children with asthma that we followed over time.[19] The study examined what happened to these children when the pollution levels were high, versus when these levels were low, and whether the occurrence of asthma, or other adverse effects, were correlated.

Controlled exposure chamber studies have also been done extensively with ozone. The results are very confirmatory to the epidemiology, which is the work I do. Epidemiology is the study of populations and the statistical association of a cause with an effect or effects. Controlled exposure chamber studies are sometimes considered to be more useful, because you can control them better. For example, you can change one factor at a time, and thereby control your study. The only problem is that you rarely get permission to put the most sensitive populations in a chamber. It is not ethical to take a very sick person and put them in a chamber and expose them to something that might make them sicker. So you really are limited in what you can do in those studies, but you can observe various activities under very well controlled conditions.

In the early 1980's, we did a field study where we showed that there were lung function declines, a decreased ability of children to breathe and exhale air, from O_3 air pollution below the level of the old O_3 standard.[20] Other researchers did chamber experiments and they said, "We have studied this. We just do not see it." But they had healthy people sitting quietly in the chamber. We challenged this by observing that children are active and are exposed throughout the day, not just for one hour. So, EPA went back to repeat the study. This time, they put people in the chamber and had them bicycle for six hours on and off, and exposed them to air pollution.

The new EPA research did not study a potentially sensitive population (such as children), but they were still able to reproduce what we saw in the epidemiology. A lot of times,

19. *See* George D. Thurston et al., *Summertime Haze Air Pollution and Children with Asthma*, 155 AM. J. RESPIRATORY CRITICAL CARE MED. 654 (1997).

20. *See id.*

although people consider chamber studies to be more definitive, they are not always representative. So that is one of the problems they have. Yet, we can learn from both and put them together to come to a conclusion. That is basically what EPA has done in the standard setting process, concluding that the evidence indicates that the old O_3 and PM standards were not sufficiently protective.

In another study we conducted, we looked at daily hospital emergency room visits and hospital admissions over time. The question we asked was whether there was an increase in hospital admissions on days when there was a high level of pollution. Conversely, were there lower hospital admissions on days of low pollution? We also looked similarly at mortality. On days of high pollution, do we see more mortality? These studies have found that there are higher incidences of hospital admissions and deaths on days of high pollution.

I now ask the question: which populations are most at risk? These include children, people with chronic lung disease, like chronic bronchitis and emphysema, the elderly, people with asthma, and people with allergies. They are among the most sensitive populations, and the law says we should protect them, not just healthy workers, which would utilize a different standard.

The documented pollution effects for people with respiratory disease include reduced lung function in children and adults, and lung/airway inflammation. Inflamed airways are especially a problem for people with asthma. One of the hallmarks of asthma is the inflammation of the lungs, which makes them much more susceptible to having an asthma attack. In chamber experiments, it has been shown that, if someone is first exposed to ozone, they are likely to have a stronger reaction to an allergen than they would if they were not exposed to ozone first.

Basically, physicians often give steroids, such as corticosteroids, to children and adults with asthma, to try and decrease the inflammation and reduce the chance that a person will have a flare-up or an asthma attack. Ozone works against that medicine and makes it more likely that someone having an asthma attack will end up in the emergency room

or be admitted to the hospital. This has been shown by an increase in hospital visits and/or hospital admissions. In fact, more recent studies, that have not been fully considered by EPA when setting this standard, have shown an increased incidence of deaths due to ozone.

I will now discuss some results from our children's asthma camp study. We looked at their peak flows, which is a measure of lung function, or how much air you can breathe out in liters per second. Normally, when they get up in the morning, a person will not have as good lung function as they do in the afternoon. Your lungs are just getting going for the day. This is especially true of asthmatics. They have diminished lung function first thing in the morning and they improve in the afternoon. So, on the lowest ozone days, they have the greatest improvement during the day. As the ozone level increases, you tend to see less and less improvement, indicating a direct effect. Now, when the same individuals are followed over time, we see that their chest symptoms, the number of symptoms they reported, such as coughing, are also greater on days with higher pollution than on days with lower pollution.

A similar trend was seen with asthma attacks. This study examined the use of beta-agonists medication, which is prescribed to asthmatics by a physician for severe asthma attacks. In fact, this was a very well controlled study, because the patients did not have their own medications. They had to go see a physician, who would evaluate them and say, "Yes, this person is having an asthma attack. Let them have more medication." So they did not carry the medication around and use it at their own discretion. Rather, the diagnosis was made by a physician. Again, there were more asthma attacks on days with higher ozone levels.

Then we looked at New York City hospital admissions and mortality rates. The relative risk, 1.13, indicates a thirteen percent increase per hundred ppb of ozone. As you go up in ozone, your risk rises, relative to the average. In addition,

we see a big jump in respiratory mortality on days of high ozone.[21]

So, what we are really seeing is a pyramid of effects (*see* Figure 1). When you look at the most severe outcomes, which are at the top of the pyramid, versus the less severe outcomes, there are fewer of the most severe effects and more of the least severe effects.[22] You see a whole range of effects. The literature confirms that there are consistent effects across these various outcomes.

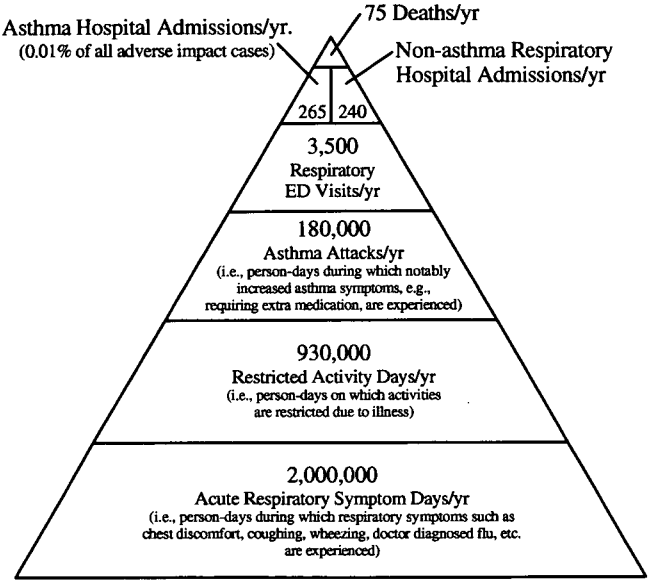

*Figure section sizes not drawn to scale.

From: Ozone and Particulate Matter Standards: Hearings on the Clean Air Act Before the Subcomm on Clean Air, Wetlands, Private Property and Nuclear Safety and the Comm. on Env't and Public Works, 105th Congress (1977)

Figure 1[23]

21. *See* George D. Thurston et al., *A Multi-year Study of Air Pollution and Respiratory Hospital Admissions in Three New York State Metropolitan Areas*, 2 J. EXPERIMENTAL ANAL. ENVTL. EPIDEMIOLOGY 429-540 (1992).

22. *See Ozone and Particulate Matter Standards: Hearings on the Clean Air Act Before the Subcomm. on Clean Air, Wetlands, Private Property and Nuclear Safety and the Comm. on Env't and Public Works*, ISBN 0-16-055638-4 105th Congress (1997) (statements of George D. Thurston), U.S. Gov't Printing Off., Doc. No. ISBNO-16-055638-4, at 124 (1997).

23. *See id.*

However, causality is one of the things that cannot be proved by epidemiology alone. You have often heard that correlation does not necessarily mean cause and effect, but there are criteria that people use to say, "Well, is this correlation something real or not?" A test of this is coherence across different outcomes in different places. Coherence. Consistency. We do see this kind of coherence across all the various outcomes and we do tend to see consistent effects in different places. They may not be the same percentage change, but they are the same kinds of effects.

We also looked for effects in other places. In Ontario, they have national health care, so there is a lot of health data collected. The government has all the records, therefore Canada is an ideal place to look at health effects. What they found was similar; as the ozone levels went up, the respiratory admissions would also rise.

This list gives you an idea of the large number of studies that we are talking about. Again, these results are presented as relative risks. We are looking at different researchers. We are looking at Buffalo, Ontario, New Haven, New York, Spokane, and Tacoma. You will notice that all the relative risks are greater than one. So, it is saying that your risk always rises with higher pollution. It is not a random phenomenon. If it were inconsistent, you would see some that were less than one. You do not. When a chi-square test is performed on the data, the statistics show that they are not significantly different. It is basically consistent and they are no different when you consider the statistical variability from one study to another. You see the same thing with pulmonary and pneumonia admissions. Multiple studies, multiple places, similar kinds of association. Consistency.

Now, what about particulate matter? A lot of analyses have been done, and the biggest benefits of the new standards will probably be due to the reduction of particulate matter. Who is the most at risk from PM? Again, as for O_3, very similar populations. That is the elderly and people with cardiovascular and chronic pulmonary disease. There has not been a lot of good research into the question of to what extent air pollution affects people's chance of having a heart attack. I

think that this is an area that is going to receive a lot of attention in the next few years, and one that has not been fully addressed.

No talk on the health effects of air pollution would be complete without discussing the London studies.[24] It was something that had been going on for a long, long time. It just happened to be that there was a confluence of a big pollution episode and the fact that they were recording air pollution in the 1950's that brought pollution's effects to the public's attention. There is documentation of similar pollution for years and years prior. One of the interesting things is that some of the biggest and most observable effects occurred during the annual livestock show in London. A lot of the prized livestock died as a result of this pollution episode. They were actually able to look at them and see the effects on the lungs, and so forth. In December 1952, there was an interesting phenomena. As you know, London had fog. On days of fog, the pollution levels went up. That was no coincidence. The reason for the fog was the pollution. The pollution was the nuclei for the water to collect on, thus causing the fog.[25] When they cleaned up this pollution, all that "Sherlock Holmes" kind of fog disappeared from London. So we lost all the ambiance, but it is better now. So, you see, what they call smoke, which is the particles, rises, and the death rate goes up and the emergency bed admissions per day also jump, indicating an association between air pollution and adverse health effects.

I would like to turn now to some work that my group has done. Arden Pope followed up on our work and did a real nice analysis, and obtained similar results.[26] This is a plot of total suspended particles versus mortality death rates in various cities. What you see is a cloud of data. No apparent relation-

24. *See* MINISTRY OF HEALTH OF GREAT BRITAIN, REPORT ON PUB. HEALTH AND MED. SUBJECTS: MORTALITY AND MORBIDITY DURING THE LONDON FOG OF DECEMBER 1952 (Her Majesty's Stationary Off., London 1954).

25. *See id.*

26. *See* C. A. Pope, III et al., *Particulate Air Pollution is a Predictor of Mortality in a Prospective Study of U.S. Adults,* 151 AM. J. RESPIRATORY CRITICAL CARE MED. 669-674 (1995).

ship there. It is because you are not focusing on the fine particles that are the problem. When you focus, in this case, on sulfates from sulfur dioxide (they are a big component of fine particles), you see a slope develop in which the higher the sulfate, the higher the mortality. Arden Pope corrected the analysis for things like smoking. We could not correct for smoking, but he did. You can also correct for socio-economic class, education, and other socio-economic barriers, and you still see the relationship. Generally, what you see is that, as you go to smaller and smaller particles, from TSP to inhalables below 10 μm, and down to 2.5 μm and smaller particles, and then to sulfates, you see bigger and bigger effects. Of course, we did this in the mid-1980s, when EPA first set the PM_{10} standard. The knowledge base has grown since then and we are now focusing, I think appropriately so, on fine particles below 2.5 μm in diameter.

Here is a summary plot that shows the concentrations at cities where mortality associations have been found, from 27 micrograms up to almost 120. So, you can see there will be a lot of cities still at risk in the United States if we stayed with the old standard of 150 Mg/m^3. Interestingly, the $PM_{2.5}$ standard, is about fifty to sixty percent of the PM_{10}. Based on this evidence, EPA's $PM_{2.5}$ standard of 65 $\mu g/m^3$ looks appropriate.

Hospital admissions associations with PM are very similar. This is kind of an interesting study, which I think a lot of people found compelling. It is Arden Pope's work in Utah, where a steel mill was shut down for one year.[27] The mill operated during the winter of 1985-1986. When it stopped operating, the PM_{10} levels and hospital admissions dropped dramatically. The next winter, when it was back in operation, asthma and pneumonia hospital admissions again increased along with PM_{10} levels. As an operating steel mill, it was a dominant pollution source in the area. It is off. It is back on again. You turn the light switch off and on, and the light goes off and on. Do you think there is a connection?

27. *See* C.A. Pope, III, *Respiratory Disease Associated with Community Air Pollution and a Steal Mill, Utah Valley*, 74 AM. J. PUBLIC HEALTH 623 (1989).

Just to summarize, I, and a couple dozen other researchers, sent a letter to the President when this debate started. Our message was that exposure to ozone and PM air pollution had been linked to many significant adverse health effects. The old standards were not protecting the public health. We asked President Clinton and his administration to listen to the medical and scientific community on this issue, and that is what has happened. In conclusion, there are some health effects that still occur below the old standards. Therefore, we need the new ozone and particulate matter standards to properly protect the sensitive populations.