

# 24

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## *Air Quality Criteria and Standards*

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### I. AIR QUALITY CRITERIA

For any pollutant, air quality criteria may refer to different types of effects. For example, Tables 24.1 through 24.6 list effects on humans, animals, vegetation, materials, and the atmosphere caused by various exposures to sulfur dioxide, particulate matter (PM), nitrogen dioxide, carbon monoxide, ozone, and lead. These data are from the Air Quality Criteria for these pollutants published by the US Environmental Protection Agency (EPA).

Criteria stipulate conditions of exposure and may refer to sensitive population groups or to the joint effects of several pollutants. Air quality criteria are descriptive. They describe effects that can be expected to occur wherever the ambient air level of a pollutant reaches or exceeds a specific concentration for a particular time period. Criteria will change as new information becomes available.

### II. CONVERSION OF EFFECTS DATA AND CRITERIA TO STANDARDS

In developing air pollution cause-effect relationships, we must be constantly on guard lest we attribute to air pollution an effect caused by something else.

TABLE 24.1

## US Ambient Air Quality Criteria for Carbon Monoxide

Percent of carboxyhemoglobin (CoHb) in blood	Human symptoms associated with this CoHb level
80	Death
60	Loss of consciousness; death if exposure is continued
40	Collapse on exercise; confusion
30	Headache, fatigue; judgment disturbed
20	Cardiovascular damage; electrocardiographic abnormalities
5	Decline (linear with increasing CoHb level) in maximal oxygen uptake of healthy young men undergoing strenuous exercise; decrements in visual perception, manual dexterity, and performance of complex sensorimotor tasks
4	Decrements in vigilance (i.e. ability to detect small changes in one's environment that occur at unpredictable times); decreased exercise performance in both healthy persons and those with chronic obstructive pulmonary disease
3-6	Aggravation of cardiovascular disease (i.e. decreased exercise capacity in patients with angina pectoris, intermittent claudication, or peripheral arteriosclerosis)

Source: Henderson, Y., and Haggard, H. W., *Noxious Gases*. Chemical Catalog Co., New York, 1927; and US Environmental Protection Agency, Air Quality Criteria for Carbon Monoxide, EPA/600/8-90/045F. Research Triangle Park, NC, December 1991.

Material damage due to pollution must be differentiated from that due to ultraviolet (UV) radiation, frost, moisture, bacteria, fungi, insects, and animals. Air pollution damage to vegetation has to be differentiated from quite similar damage attributable to bacterial and fungal diseases, insects, drought, frost, soil mineral deviations, hail, and cultural practices. In the principal animal disorder associated with air pollution, i.e. fluorosis, the route of animal intake of fluorine is by ingestion, the air being the means for transporting the substance from its source to the forage or hay used for animal feed. However, the water or feed supplements used may also have excess fluorine. Therefore, these sources and disease states, which may have symptoms similar to those of fluorosis, must be ruled out before a cause-effect relationship can be established between ambient air levels of fluorine and fluorosis in animals. Similarly, there are many instances of visibility reduction in the atmosphere by fog or mist for which air pollution is not a causative factor.

To study damage to materials, vegetation, and animals, we can set up laboratory experiments in which most confusing variables are eliminated and a direct cause-effect relationship is established between pollutant dosage and resulting effect. We are limited to low-level exposure experiments under controlled conditions with human beings for ethical reasons. Our cause-effect relationships

TABLE 24.2a

Summary of Lowest Observed Effect Levels for Key Lead-Induced Health Effects in Adults

Lowest observed effect level (PbB) <sup>a</sup> (μg dL <sup>-1</sup> )	Heme synthesis and hematological effects	Neurological effects	Effects on the kidney	Reproductive function effects	Cardiovascular effects
100–120		Encephalopathic signs and symptoms	Chronic nephropathy		
80	Frank anemia				
60					
50	Reduced hemoglobin production	Overt subencephalopathic neurological symptoms		Female reproductive effects Altered testicular function	
40	Increased urinary ALA and elevated coproporphyrins	Peripheral nerve dysfunction (slowed nerve conduction)			
30					
25–30	Erythrocyte protoporphyrin (EP) elevation in males				Elevated blood pressure (white males, aged 40–59)
15–20	EP elevation in females				
<10	ALA-D <sup>b</sup> inhibition				?

<sup>a</sup>PbB = blood lead concentrations.

dL = deciliter = 0.1 L (a commonly used unit in medical literature).

ALA, ALA-D: aminolevulinic acid dehydrase.

Source: US Environmental Protection Agency, Air Quality Criteria for Lead, EPA-600-8-83/028aF, June 1986.

TABLE 24.2b

Summary of Lowest Observed Effect Levels for Key Lead-Induced Health Effects in Children

Lowest observed effect level (PbB) <sup>a</sup> (µg dL <sup>-1</sup> )	Heme synthesis and hematological effects	Neurological effects	Renal system effects	Gastrointestinal effects
80-100		Encephalopathic signs and symptoms	Chronic nephropathy (aminoaciduria, etc.)	Colic, other overt gastrointestinal symptoms
70	Frank anemia	Peripheral neuropathies		↓
60		↓		
50		?		
40	Reduced hemoglobin synthesis Elevated coproporphyrin	Peripheral nerve dysfunction (slowed NCVs) <sup>d</sup> CNS cognitive effects (IQ deficits, etc.)		
30	Increased urinary ALA <sup>c</sup>	↓		
15	Erythrocyte protoporphyrin elevation	Altered CNS electro-physiological response	Vitamin D metabolism interference	
10	ALA-D <sup>c</sup> inhibition Py-5-N activity inhibition <sup>b</sup>	↓	↓	
	↓	?	?	

<sup>a</sup> PbB: blood lead concentrations.

<sup>b</sup> Py-5-N: pyrimidine-5 $\epsilon$ -nucleotidase.

<sup>c</sup> ALA, ALA-D: aminolevulinic acid dehydrase.

<sup>d</sup> NCV: nerve conduction velocity.

Source: US Environmental Protection Agency, Air Quality Criteria for Lead, EPA-600, EPA-600-8-83/028aF, June 1986.

TABLE 24.3

## US Ambient Air Quality Criteria for Sulfur Dioxide

Concentration of sulfur dioxide in air (ppm)	Exposure time	Human symptoms and effects on vegetation
400	—	Lung edema; bronchial inflammation
20	—	Eye irritation; coughing in healthy adults
15	1 h	Decreased mucociliary activity
10	10 min	Bronchospasm
10	2 h	Visible foliar injury to vegetation in arid regions
8	—	Throat irritation in healthy adults
5	10 min	Increased airway resistance in healthy adults at rest
1	10 min	Increased airway resistance in asthmatics at rest and in healthy adults at exercise
1	5 min	Visible injury to sensitive vegetation in humid regions
0.5	10 min	Increased airway resistance in asthmatics at exercise
0.5	—	Odor threshold
0.5	1 h	Visible injury to sensitive vegetation in humid regions
0.5	3 h	United States National Secondary Ambient Air Quality Standard promulgated in 1973
0.2	3 h	Visible injury to sensitive vegetation in human regions
0.19	24 h <sup>a</sup>	Aggravation of chronic respiratory disease in adults
0.14	24 h	United States National Primary Ambient Air Quality Standard promulgated in 1971 <sup>b</sup>
0.07	Annual <sup>a</sup>	Aggravation of chronic respiratory disease in children
0.03	Annual	United States National Primary Ambient Air Quality Standard promulgated in 1971 <sup>b</sup>

<sup>a</sup> In the presence of high concentrations of PM.

<sup>b</sup> Source: Air Quality Criteria for Particulate Matter and Sulfur Oxides, final draft, US Environmental Protection Agency, Research Triangle Park, NC, December 1981; Review of the National Ambient Air Quality Standards for Sulfur Oxides: Assessment of Scientific and Technical Information, Draft OAQPS Staff Paper, US Environmental Protection Agency, Research Triangle Park, NC, April 1982.

for humans are based on (1) extrapolation from animal experimentation, (2) clinical observation of individual cases of persons exposed to the pollutant or toxicant (industrially, accidentally, suicidally, or under air pollution episode conditions), and (3) most important, epidemiological data relating population morbidity and mortality to air pollution. There are no human diseases uniquely caused by air pollution. In all air pollution-related diseases in which there is buildup of toxic material in the blood, tissue, bone, or teeth, part or all of the buildup could be from ingestion of food or water containing the material. Diseases which are respiratory can be caused by smoking or occupational exposure. They may be of a bacterial, viral, or fungal origin quite divorced from the inhalation of human-made pollutants in the ambient air. These causes in addition to the variety of congenital, degenerative, nutritional, and

TABLE 24.4

## US Air Quality Criteria for Nitrogen Dioxide

Concentration of nitrogen dioxide in air (ppm)	Exposure time	Human symptoms and effects on vegetation, materials, and visibility
300	—	Rapid death
150	—	Death after 2 or 3 weeks by bronchiolitis fibrosa obliterans
50	—	Reversible, nonfatal bronchiolitis
10	—	Impairment of ability to detect odor of nitrogen dioxide
5	15 min	Impairment of normal transport of gases between the blood and lungs in healthy adults
2.5	2 h	Increased airway resistance in healthy adults
2	4 h	Foliar injury to vegetation
1.0	15 min	Increased airway resistance in bronchitics
1.0	48 h	Slight leaf spotting of pinto bean, endive, and cotton
0.3	—	Brownish color of target 1 km distant
0.25	Growing season	Decrease of growth and yield of tomatoes and oranges
0.2	8 h	Yellowing of white fabrics
0.12	—	Odor perception threshold of nitrogen dioxide
0.1	12 weeks	Fading of dyes on nylon
0.1	20 weeks	Reduction in growth of Kentucky bluegrass
0.05	12 weeks	Fading of dyes on cotton and rayon
0.03	—	Brownish color of target 10 km distant
0.003	—	Brownish color of target 100 km distant

Source: Draft Air Quality Criteria for Oxides of Nitrogen, US Environmental Protection Agency, Research Triangle Park, NC, 1981; Review of the National Ambient Air Quality Standard for Nitrogen Dioxide, Assessment of Scientific and Technical Information, EPA-450/5-82-002. US Environmental Protection Agency, Research Triangle Park, NC, March 1982.

psychosomatic causes of disease must all be ruled out before a disease can be attributed to air pollution. However, air pollution commonly exacerbates preexisting disease states. In human health, air pollution can be the “straw that breaks the camel’s back.”

Air quality standards prescribe pollutant levels that cannot legally be exceeded during a specific time period in a specific geographic area. Air quality standards are based on air quality criteria, with added safety factors as desired.

The main philosophical question that arises with respect to air quality standards is what to consider an adverse effect or a cost associated with air pollution. Let us examine several categories of receptors to see the judgmental problems that arise.

Two important pollutants that have undergone significant changes to air quality criteria are PM and ozone. Under the Clean Air Act, they are among the six principal (or “criteria”) pollutants for which EPA has established National Ambient Air Quality Standards (NAAQS). Periodically, EPA reviews

TABLE 24.5

## US Ambient Air Criteria for Ozone

Concentration of ozone in air (ppm) <sup>a</sup>	Human symptoms and vegetation injury threshold
10.0	Severe pulmonary edema; possible acute bronchiolitis; decreased blood pressure; rapid weak pulse
1.0	Coughing; extreme fatigue; lack of coordination; increased airway resistance; decreased forced expiratory volume
0.5	Chest constriction; impaired carbon monoxide diffusion capacity; decrease in lung function without exercise
0.3	Headache; chest discomfort sufficient to prevent completion of exercise; decrease in lung function in exercising subjects
0.25	Increase in incidence and severity of asthma attacks; moderate eye irritation
0.15	For sensitive individuals, reduction in pulmonary lung function; chest discomfort; irritation of the respiratory tract, coughing and wheezing. Threshold for injury to vegetation
0.12	United States National Primary and Secondary Ambient Air Quality Standard, attained when the expected number of days per calendar year with maximum hourly average concentrations above 0.12 ppm is equal to or less than 1, as determined in a specified manner

<sup>a</sup> 1 ppm: 1958  $\mu\text{g}\cdot\text{m}^{-3}$  ozone.

Source: Air Quality Criteria for Ozone and Other Photochemical Oxidants, EPA 600/8-78-004. US Environmental Protection Agency, Research Triangle Park, NC, April 1978; Revisions to National Ambient Air Quality Standards for Photochemical Oxidants. *Fed. Reg.* Part V, February 9, 8202-8237 (1979).

40 CFR § 50, July 1992.

the scientific basis for these standards by preparing an Air Quality Criteria Document (AQCD).<sup>1</sup>

The Clean Air Act requires an update and revision of the AQCD for PM every five years. The most recent started in 1998, following July 1997 promulgation of a new PM NAAQS. The PM AQCD is the scientific basis for the additional technical and policy assessments that form the basis for EPA decisions on the adequacy of the current PM NAAQS and the appropriateness of new or revised standards for PM. The original NAAQS for PM, issued in 1971 as "total suspended particulate" (TSP) standards, were revised in 1987 to focus on protecting against human health effects associated with exposure to ambient particles with aerodynamic diameters less than or equal to 10 microns ( $\leq 10\mu\text{m}$ ). These are able to deposit in the thoracic (tracheobronchial and alveolar)

<sup>1</sup> The sources for the discussion in this section are the US EPA criteria documents for PM and ozone: US EPA, Air Quality Criteria for Particulate Matter, Report No. EPA/600/P-99/002aF, 2004; and US EPA, Air Quality Criteria for Ozone and Related Photochemical Oxidants, Report No. EPA 600/R-05/004aF, 2006.

TABLE 24.6

## US Ambient Air Quality Criteria for Particulate Matter

Concentration of PM in air ( $\mu\text{g m}^{-3}$ )				
Total suspended TSP > 25 $\mu\text{m}$	Thoracic TP > 10 $\mu\text{m}$	Fine FP > 2.5 $\mu\text{m}$	Exposure time	Human symptoms and effects on visibility
2000	—	—	2 h	Personal discomfort
1000	—	—	10 min	Direct respiratory mechanical changes
—	350	—		Aggravation of bronchitis
	150	—	24 h	United States Primary National Ambient Air Quality Standard as of September, 1987
180	90	—		Increased respiratory disease symptoms
	150	—	24 h	United States Primary National Ambient Air Quality Standard as of September, 1987
110	55	—	24 h	Increased respiratory disease risk
	50	—	Annual geometric mean	United States Primary National Air Quality Standard as of September, 1987
—	—	22	13 weeks	Usual summer visibility in eastern United States, nonurban sites

Source: Air Quality Criteria for Particulate Matter and Sulfur Oxides, Draft Final. US Environmental Protection Agency, Research Triangle Park, NC, December 1981; Review of the National Ambient Air Quality Standard for Particulate Matter: Assessment of Scientific and Technical Information, EPA-450/5-82-001. US Environmental Protection Agency, Research Triangle Park, NC, January 1982. 40 CFR § 50, July 1992.

portions of the lower respiratory tract. The  $\text{PM}_{10}$  NAAQS set in 1987 ( $150 \mu\text{g m}^{-3}$ , 24 h;  $50 \mu\text{g m}^{-3}$ , annual average) were retained in modified form and new standards ( $65 \mu\text{g m}^{-3}$ , 24 h;  $15 \mu\text{g m}^{-3}$ , annual average) for particles  $\leq 2.5 \mu\text{m}$  ( $\text{PM}_{2.5}$ ) were promulgated in July 1997.

The current criteria document has pointed out a number of areas of uncertainty that need to be addressed. These are provided in Table 24.7. Though much has been learned about the hazards, exposure, effects, and risks of particulates, much still needs to be known.

The main focus of the PM document is the evaluation and interpretation of pertinent atmospheric science information, air quality data, human

TABLE 24.7

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**Key Areas of Uncertainty that Need to Be Addressed for PM as an Air Pollutant**


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- RESEARCH TOPIC 1. OUTDOOR MEASURES VERSUS ACTUAL HUMAN EXPOSURES**
- What are the quantitative relationships between concentrations of particulate matter and gaseous co-pollutants measured at stationary outdoor air monitoring sites and the contributions of these concentrations to actual personal exposures, especially for subpopulations and individuals?
- RESEARCH TOPIC 2. EXPOSURES OF SUSCEPTIBLE SUBPOPULATIONS TO TOXIC PARTICULATE MATTER COMPONENTS**
- What are the exposure to biologically important constituents and specific characteristics of particulate matter that cause responses in potentially susceptible subpopulations and the general population?
- RESEARCH TOPIC 3. CHARACTERIZATION OF EMISSION SOURCES**
- What are the size distributions, chemical composition, and mass-emission rates of particulate matter emitted from the collection of primary-particle sources in the United States, and what are the emissions of reactive gases that lead to secondary particle formation through atmospheric chemical reactions?
- RESEARCH TOPIC 4. AIR-QUALITY MODEL DEVELOPMENT AND TESTING**
- What are the linkages between emission sources and ambient concentrations of the biologically important components of particulate matter?
- RESEARCH TOPIC 5. ASSESSMENT OF HAZARDOUS PARTICULATE MATTER COMPONENTS**
- What is the role of physicochemical characteristics of particulate matter in eliciting adverse health effects?
- RESEARCH TOPIC 6. DOSIMETRY: DEPOSITION AND FATE OF PARTICLES IN THE RESPIRATORY TRACT**
- What are the deposition patterns and fate of particles in the respiratory tract of individuals belonging to presumed susceptible subpopulations?
- RESEARCH TOPIC 7. COMBINED EFFECTS OF PARTICULATE MATTER AND GASEOUS POLLUTANTS**
- How can the effects of particulate matter be disentangled from the effects of other pollutants? How can the effects of long-term exposure to particulate matter and other pollutants be better understood?
- RESEARCH TOPIC 8. SUSCEPTIBLE SUBPOPULATIONS**
- What subpopulations are at increased risk of adverse health outcomes from particulate matter?
- RESEARCH TOPIC 9. MECHANISMS OF INJURY**
- What are the underlying mechanisms (local pulmonary and systemic) that can explain the epidemiologic findings of mortality/morbidity associated with exposure to ambient particulate matter?
- RESEARCH TOPIC 10. ANALYSIS AND MEASUREMENT**
- To what extent does the choice of statistical methods in the analysis of data from epidemiologic studies influence estimates of health risks from exposures to particulate matter? Can existing methods be improved? What is the effect of measurement error and misclassification on estimates of the association between air pollution and health?
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Source: National Research Council, *Research Priorities for Airborne Particulate Matter. III. Early Research Progress*. National Academy Press, Washington, DC, 2001.

exposure information, and health and welfare effects information published since what was assessed during the development of the 1996 PM AQCD. A number of draft versions of AQCD chapters have been evaluated via expert peer-review workshop discussions and peer reviews.

The document is a rich resource for anyone interested in PM and air pollution in general. It is divided into two volumes: Volume I (Chapters 1 through 5, EPA/600/P-99/002aD) and Volume II (Chapters 6 through 9, EPA/600/P-99/002bD). After the brief general introduction in Chapter 1, Chapters 2 and 3 provide background information on physical and chemical properties of PM and related compounds; sources and emissions; atmospheric transport; transformation and fate of PM; methods for the collection and measurement of PM; and ambient air concentrations; Chapter 4 describes PM environmental effects on vegetation and ecosystems, impacts on man-made materials and visibility, and relationships to global climate change processes; and Chapter 5 contains factors affecting exposure of the general population. Chapters 6 through 8 evaluate information concerning the health effects of PM. Chapter 6 discusses dosimetry of inhaled particles in the respiratory tract; and Chapter 7 assesses information on the toxicology of specific types of PM constituents, including laboratory animal studies and controlled human exposure studies. Chapter 8 discusses epidemiologic studies. Chapter 9 integrates key information on exposure, dosimetry, and critical health risk issues derived from studies reviewed in the prior chapters.

Tropospheric or "surface-level" ozone ( $O_3$ ) is also the subject of a high profile AQCD. Following the review of criteria as contained in the EPA document, *Air Quality Criteria for Ozone and Other Photochemical Oxidants* published in 1978, the chemical designation of the standards was changed from photochemical oxidants to ozone ( $O_3$ ) in 1979 and a 1-h  $O_3$  NAAQS was set. The 1978 document focused mainly on the air quality criteria for  $O_3$  and, to a lesser extent, on those for other photochemical oxidants (e.g. hydrogen peroxide and the peroxyacetal nitrates), as have subsequently revised versions of the document.

As is true for PM, to meet Clean Air Act requirements noted above for periodic review of criteria and NAAQS, the  $O_3$  criteria document, *Air Quality Criteria for Ozone and Other Photochemical Oxidants*, was next revised and released in August 1986; and a supplement, *Summary of Selected New Information on Effects of Ozone on Health and Vegetation*, was issued in January 1992. These documents were the basis for a March 1993 decision by EPA that revision of the existing 1-h NAAQS for  $O_3$  was not appropriate at that time. That decision, however, did not take into account newer scientific data that had become available after completion of the 1986 criteria document. Such literature was assessed in the next periodic revision of the  $O_3$  AQCD which was completed in 1996 and provided scientific bases supporting the setting by EPA in 1997 of the current 8-h  $O_3$  NAAQS. The purpose of this revised AQCD for  $O_3$  and related photochemical oxidants is to critically evaluate and assess the latest scientific information published since that assessed

in the 1996 O<sub>3</sub> AQCD, with the main focus being on pertinent new information useful in evaluating health and environmental effects data associated with ambient air O<sub>3</sub> exposures. However, other scientific data are also discussed in order to provide a better understanding of the nature, sources, distribution, measurement, and concentrations of O<sub>3</sub> and related photochemical oxidants and their precursors in the environment.

Like the PM document, the ozone AQCD has a wealth of information for the air pollution professional and student.

The Executive Summary summarizes key findings and conclusions as they pertain to background information on O<sub>3</sub>-related atmospheric science and air quality, human exposure aspects, dosimetric considerations, health effect issues, and environmental effect issues.

Chapter 1 provides a general introduction, including an overview of legal requirements, the chronology of past revisions of O<sub>3</sub>-related NAAQS, and orientation to the structure of this document.

Chapters 2 and 3 provide background information on atmospheric chemistry/physics of O<sub>3</sub> formation, air quality, and exposure aspects to help to place ensuing discussions of O<sub>3</sub> health and welfare effects into perspective.

Chapters 4 through 7 then assess dosimetry aspects, experimental (controlled human exposure and laboratory animal) studies, and epidemiologic (field/panel; other observational) studies. Chapter 8 then provides an integrative synthesis of key findings and conclusions derived from the preceding chapters with regard to ambient O<sub>3</sub> concentrations, human exposures, dosimetry, and health effects.

Chapter 9 deals with effects of O<sub>3</sub> on vegetation, crops, and natural ecosystems, whereas Chapter 10 evaluates tropospheric O<sub>3</sub> relationships to alterations in surface-level UVB flux and climate change and Chapter 11 assesses materials damage (these all being key types of welfare effects of relevance to decisions regarding secondary O<sub>3</sub> NAAQS review).

### III. CONVERSION OF PHYSICAL DATA AND CRITERIA TO STANDARDS

Although air quality standards are based predominantly on biological criteria, certain physical criteria also deserve consideration.

Most materials will deteriorate even when exposed to an unpolluted atmosphere. Iron will rust, metals will corrode, and wood will rot. To prevent deterioration, protective coatings are applied. Their costs are part of the economic picture. Some materials, such as railroad rails, are used without protective coatings. There are costs associated with the decrease in their life in a polluted atmosphere as compared to an unpolluted one. One may argue that for materials on which protective coatings are used, only pollution levels that damage such coatings are of concern. One may further argue that some air pollution damage to protection coatings is tolerable, since by their

very nature such coatings require periodic replenishment to maintain their protective integrity or appearance; therefore, only coatings that require more frequent replenishment than they would in an unpolluted atmosphere should enter into the establishment of deterioration costs and air quality standards. This argument certainly does hold with respect to the soiling of materials and structures. In fact, it is frequently the protective coatings themselves that require replacement because they become dirty long before their useful life as protectants has terminated. It can readily be shown that there are costs associated with soiling, including the cost of removing soil, the cost of protective coatings to facilitate the removal of soil, the premature disposal of material when it is no longer economical or practicable to remove soil, and the growth inhibition of vegetation due to leaf soiling. However, decision-making for air quality standards related to soiling is based less on economic evaluation than on esthetic considerations, i.e. on subjective evaluation of how much soiling the community will tolerate. This latter determination is judgmental and difficult to make. It may be facilitated by opinion surveys, but even when the limit of public tolerance for soiling is determined, it still has to be restated in terms of the pollution loading of the air that will result in this level of soiling.

An important effect of air pollution on the atmosphere is change in spectral transmission. The spectral regions of greatest concern are the UV and the visible. Changes in UV radiation have demonstrable adverse effects; e.g. a decrease in the stratospheric ozone layer permits harmful UV radiation to penetrate to the surface of the earth. Excessive exposure to UV radiation results in increases in skin cancer and cataracts. The worldwide effort to reduce the release of stratospheric ozone-depleting chemicals such as chlorofluorocarbons is directed toward reducing this increased risk of skin cancer and cataracts for future generations.

The fact that after a storm or the passage of a frontal system the air becomes crystal clear and one can see for many kilometers does not give a true measure of year-round visibility under unpolluted conditions. Between storms, even in unpolluted air, natural sources build up enough PM in the air so that on many days of the year there is less than ideal visibility. In many parts of the world, mountains are called "Smoky" or "Blue" or some other name to designate the prevalence of a natural haze, which gives them a smoky or bluish color and impedes visibility. When the Spanish first explored the area that is now Los Angeles, California, they gave it the name "Bay of the Smokes." The Los Angeles definition of air quality before the advent of smog was that "You could see Catalina Island on a clear day." The part of the definition that is lacking is some indication of how many clear days there were before the advent of smog.

There are costs associated with loss of visibility and solar energy. These include increased need for artificial illumination and heating; delays, disruptions, and accidents involving air, water, and land traffic; vegetation growth reduction associated with reduced photosynthesis; and commercial

losses associated with the decreased attractiveness of a dingy community or one with restricted scenic views. However, these costs are less likely to be involved in deciding, for air quality standard-setting purposes, how much of the attainable visibility improvement to aim for than are esthetic considerations. Just as in the previously noted case of soiling, judgment on the limit of public tolerance for visibility reduction still has to be related to the pollutant loading of the atmosphere that will yield the desired visibility. Obviously, the pollutant level chosen for an air quality standards must be the lower of the values required for soiling or visibility, otherwise one will be achieved without the other. Whether the level chosen will not be lower than the atmospheric pollutant level required for prevention of health effects will depend on the esthetic standards of the jurisdiction.

#### IV. CONVERSION OF BIOLOGICAL DATA AND CRITERIA TO STANDARDS

There is considerable species variability with respect to damage to vegetation by any specific pollutant. There is also great geographic variability with respect to where these species grow naturally or are cultivated. Because of this, it is possible that in a jurisdiction none of the species particularly susceptible to damage by low levels of pollution may be among those indigenous or normally imported for local cultivation. As an example, the pollution level at which citrus trees are adversely affected, while meaningful in setting air quality standards in California and Florida, is meaningless for this purpose in Minnesota and Wisconsin. In like manner, a jurisdiction may take different viewpoints with respect to indigenous and imported species. It might set its air quality standards low enough to protect its indigenous vegetation even if this level is too high to allow satisfactory growth of imported species. Even if a particularly susceptible species is indigenous, it may be held in such low local esteem commercially or esthetically that the jurisdiction may be unwilling to let the damage level of that species be the air quality standard discriminator. In other words, the people would rather have that species damaged than assume the cost of cleaning up the air to prevent the damage. This same line of reasoning applies to effects on wild and domestic animals.

A jurisdiction may base part of its decision-making regarding vegetation and animal damage on esthetics. Its citizens may wish to grow certain ornamentals or raise certain species of pet birds or animals and allow these wishes to override the agricultural, forestry, and husbandry economics of the situation. Usually, however, economic considerations predominate in decision-making. Costs of air pollution effects on agriculture are the sum of the loss in income from the sale of crops or livestock and the added cost necessary to raise the crops or livestock for sale. To these costs must be added the loss in value of agricultural land as its income potential decreases and

the loss suffered by the segments of local industry and commerce that are dependent on farm crops and the farmer for their existence. An interesting sidelight is that when such damage occurs on the periphery of an urban area, it is frequently a precursor to the breakup of such farmland into residential development, with a financial gain rather than a loss to the landowner. When the crop that disappears is an orchard, grove, or vineyard that took years to establish, and when usable farm buildings are torn down, society as a whole suffers a loss to the extent that it will take much time and money to establish a replacement for them at new locations. To some industries, air pollution costs include purchase of farm and ranch land to prevent litigation to recover damages, annual subsidy payments to farmers and ranchers in lieu of such litigation, and maintenance of air quality monitoring systems to protect themselves against unwarranted litigation for this purpose.

There is a range of ambiguity in our human health effects criteria data. In this range there is disagreement among experts as to its validity and interpretation. Thus, from the same body of health effects data, one could adopt an air quality standard on the high side of the range of ambiguity or one on the low side. Much soul searching is required before one accepts the results of questionable human health effects research and is accused of imposing large costs on the public by so doing, or of rejecting these results and being accused of subjecting the public to potential damage of human health.

## V. AIR QUALITY STANDARDS

Since air pollution is controlled by air quality and emission standards, the principal philosophical discussions in the field of air pollution control focus on their development and application.

The US Clean Air Amendments of 1977 defined two kinds of air quality standards: primary standards, levels that will protect health but not necessarily prevent the other adverse effects of air pollution, and secondary standards, levels that will prevent all the other adverse effects of air pollution (Table 24.8). The amendments also define air quality levels that cannot be exceeded in specified geographic areas for "prevention of significant deterioration" (PSD) of the air of those areas. Although they are called "increments" over "baseline air quality" in the law, they are in effect tertiary standards, which are set at lower ambient levels than either the primary or secondary standards (Table 24.9). The PSD program applies to any "major emitting facility" in attainment areas. For 28 named categories a major emitting facility is one with a "potential to emit" 100 tons or more per year of any regulated air pollutant. However, any source is regulated under PSD if it has a potential to emit 250 or more tons per year. Emission potential assumes maximum design capacity (42 USC § 7479).

Increments are said to be "consumed" as new sources are given permits that allow pollution to be introduced into these areas. Jurisdictions with

TABLE 24.8

US Federal Primary and Secondary Ambient Air Quality Standards (NAAQS). *Primary Standards Set Limits to Protect Public Health, Including the Health of "Sensitive" Populations Such as Asthmatics, Children, and the Elderly. Secondary Standards Set Limits to Protect Public Welfare, Including Protection Against Decreased Visibility, Damage to Animals, Crops, Vegetation, and Buildings*

Pollutant	Primary standards	Averaging times	Secondary standards	Provisions
Carbon monoxide	9 ppm (10 mg m <sup>-3</sup> )	8 h	None	Not to be exceeded more than once per year.
	35 ppm (40 mg m <sup>-3</sup> )	1 h	None	Not to be exceeded more than once per year.
Lead	1.5 µg m <sup>-3</sup>	Quarterly average	Same as primary	
Nitrogen dioxide	0.053 ppm (100 µg m <sup>-3</sup> )	Annual (arithmetic mean)	Same as primary	
Particulate matter (PM <sub>10</sub> )	Revoked	Annual (arithmetic mean)		Due to a lack of evidence linking health problems to long-term exposure to coarse particle pollution, the agency revoked the annual PM <sub>10</sub> standard in 2006 (effective December 17, 2006).
	150 µg m <sup>-3</sup>	24 h		Not to be exceeded more than once per year on average over 3 years.
Particulate matter (PM <sub>2.5</sub> )	15.0 µg m <sup>-3</sup>	Annual (arithmetic mean)	Same as primary	
	35 µg m <sup>-3</sup>	24 h		To attain this standard, the 3-year average of the 98th percentile of 24-h concentrations at each population-oriented monitor within an area must not exceed 35 µg m <sup>-3</sup>
Ozone	0.08 ppm	8 h	Same as primary	To attain this standard, the 3-year average of the fourth-highest daily maximum 8-h average ozone concentrations measured at each monitor within an area over each year must not exceed 0.08 ppm.

(continued)

TABLE 24.8 (Continued)

Pollutant	Primary standards	Averaging times	Secondary standards	Provisions
	0.12 ppm	1 h (applies only in limited areas)	Same as primary	(a) The standard is attained when the expected number of days per calendar year with maximum hourly average concentrations above 0.12 ppm is $\leq 1$ , as determined by appendix H. (b) As of June 15, 2005 EPA revoked the 1-h ozone standard in all areas except the fourteen 8-h ozone nonattainment Early Action Compact (EAC) Areas.
Sulfur oxides	0.03 ppm	Annual (arithmetic mean)	—	To attain this standard, the 3-year average of the weighted annual mean PM <sub>2.5</sub> concentrations from single or multiple community-oriented monitors must not exceed 15.0 $\mu\text{g m}^{-3}$ .
	0.14 ppm	24 h	—	Not to be exceeded more than once per year.
	—	3 h	0.5 ppm (1300 $\mu\text{g m}^{-3}$ )	Not to be exceeded more than once per year.

Source: 40 CFR Part 50, July 1999.

TABLE 24.9

## US Federal Prevention of Significant Deterioration (PSD) Increments

<i>Class I PSD increments<sup>a</sup></i>	
Sulfur dioxide	Increment ( $\mu\text{g m}^{-3}$ )
Annual arithmetic mean	2
24-h maximum	5
3-h maximum	25
Nitrogen dioxide	Increment ( $\mu\text{g m}^{-3}$ )
Annual arithmetic mean	25
Particulate matter (PM <sub>10</sub> )	Increment ( $\mu\text{g m}^{-3}$ )
Annual arithmetic mean	4
24-h maximum	8
<i>Class II PSD increments<sup>b</sup></i>	
Sulfur dioxide	Increment ( $\mu\text{g m}^{-3}$ )
Annual arithmetic mean	20
24-h maximum	91
3-h maximum	512
Nitrogen dioxide	Increment ( $\mu\text{g m}^{-3}$ )
Annual arithmetic mean	25
Particulate matter (PM <sub>10</sub> )	Increment ( $\mu\text{g m}^{-3}$ )
Annual arithmetic mean	17
24-h maximum	30
<i>Class III PSD increments<sup>c</sup></i>	
Sulfur dioxide	Increment ( $\mu\text{g m}^{-3}$ )
Annual arithmetic mean	40
24-h maximum	182
3-h maximum	700
Nitrogen dioxide	Increment ( $\mu\text{g m}^{-3}$ )
Annual arithmetic mean	50
Particulate matter (PM <sub>10</sub> )	Increment ( $\mu\text{g m}^{-3}$ )
Annual arithmetic mean	34
24-h maximum	60

<sup>a</sup> Class I areas are pristine, such as national parks, national seashores, and natural wilderness areas.

<sup>b</sup> Class II areas allow moderate deteriorations (unless otherwise designated, all areas are considered Class II).

<sup>c</sup> Class III are specifically designated for heavy industrial uses.

Source: 40 CFR § 51.166 (c).

authority to issue permits may choose to “allocate” portions of a PSD increment (or of the difference between actual air quality and the primary or secondary standard) for future consumption, rather than to allow its consumption on a first-come, first-served basis.

The states are required to submit to the federal EPA plans, known as State Implementation Plans (SIP), showing how they will achieve the standards in their jurisdictions within a specified time period. If after that time period there

are areas within the states where these standards have not been attained, the states are required to submit and obtain EPA approval of revised plans to achieve the standards in these "nonattainment" areas. EPA also designates certain areas where the standards are being met, but which have the potential for future nonattainment, as Air Quality Maintenance Areas (AQMA). Such regions have stricter requirements than attainment areas for the granting of permits for new sources of the pollutant not in attainment status.

The Canadian Clean Air Act allows the provincial minister to formulate air quality objectives reflecting three ranges of ambient air quality for any contaminant. The *tolerable* range denotes a concentration that requires abatement without delay. The *acceptable* range provides adequate protection against adverse effects. The *desirable* range defines a long-term goal for air quality and provides the basis for a nondegradation policy for unpolluted parts of the country (Table 24.10). The Canadian ambient  $PM_{2.5}$  and ozone standards shown in Table 24.11 are to be implemented by 2010.

TABLE 24.10  
National Ambient Air Quality Standards for Canada

Pollutant		Averaging time			Published	Reviewed
		1 h	24 h	Annual		
Carbon monoxide ( $mg\ m^{-3}$ )	D	15	6 (8 h)		1974	1996
	A	35	15 (8 h)		1974	1996
	T		20 (8 h)		1978	1996
Hydrogen fluoride ( $\mu g\ m^{-3}$ )	RL		1.1	0.5(7 d)	1997	
Nitrogen dioxide ( $\mu g\ m^{-3}$ )	D			60	1975	1989
	A	400	200	100	1975	1989
	T	1000	300		1978	1989
Ozone ( $\mu g\ m^{-3}$ )	D	100	30		1974	1989
	A	160	50	30	1974	1989
	T	300			1978	1989
	RL				*	
PM < 10 ( $\mu g\ m^{-3}$ )	RL		25		1998	
PM < 2.5 ( $\mu g\ m^{-3}$ )	RL		15		1998	
Sulfur dioxide ( $\mu g\ m^{-3}$ )	D	450	150	30	1974	1989
	A	900	300	60	1974	1989
	T		800		1978	1989
Total reduced sulfur compounds	RL				*	
	AQO				*	
Total suspended particulates ( $\mu g\ m^{-3}$ )	D			60	1974	1989
	A		120	70	1974	1989
	T		400		1978	1989

\* Reviews in progress.

Notes: D, desirable; A, acceptable; T, tolerable; RL, reference level; AQO, air quality objective.

Source: Canadian Council of Ministers of the Environment, Canadian national ambient air quality Objectives: process and status. In *Canadian Environmental Quality Guidelines*. Canadian Council of Ministers of the Environment, Winnipeg, Canada, 1999.

Some examples of air quality standards for other countries are given in Table 24.12.

TABLE 24.11

**Canada Wide Standards (CWSs) Have Been Established for Ozone and PM Is to Minimize the Risk to Human Health and the Environment**

Ozone	65 ppb ( $130 \mu\text{g m}^{-3}$ ) averaged over an 8-h period. Achievement will be based on the 4th highest measurement annually as averaged over three consecutive years.
PM <sub>2.5</sub>	$30 \mu\text{g m}^{-3}$ averaged over a 24-h period. Achievement will be based on the 98th percentile ambient measurement annually, averaged over three consecutive years.

*Note:* These standards represent a balance between the desire to achieve the best health and environmental protection possible in the near term with the feasibility and costs to reduce pollutant emissions contributing ozone and PM in the ambient air. These CWSs for ozone and PM<sub>2.5</sub> are to be implemented by the year 2010.

TABLE 24.12

**Air Quality Standards for Selected Pollutants in Several Countries and for International Organizations Around the World**

Country	Suspended particulate matter (SPM), $\mu\text{g m}^{-3}$		Sulphur dioxide (SO <sub>2</sub> ), $\mu\text{g m}^{-3}$		Oxides of Nitrogen (NO), $\mu\text{g m}^{-3}$	
	24 h	1 year	24 h	1 year	24 h	1 year
India						
Industrial	500	360	120	80	120	80
Residential	200	140	80	60	80	60
Sensitive	100	70	30	15	30	15
China						
Class III	500		250		150	
Class II	300		150		100	
Class I	150		50		50	
Australia		90		60		
Japan	100		100		100	
USA	260		365	80		100
EU	300	150	250	80		200
WHO	150–230	60–90	100–150	40–60	150	

*Source:* CONCAWE (Conservation of Clean Air and Water in Europe), *Potential for Exhaust After Treatment and Engine Technologies to Meet Future Emissions Limit*, Report No. 99/55. Brussels, Belgium, 1999.

## SUGGESTED READING

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- US Environmental Protection Agency, *Air Quality Criteria for Particulate Matter*, Report No. EPA/600/P-99/002aF, 2004.
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- World Health Organization, *Air Quality Guidelines for Europe*. World Health Organization, Copenhagen, 1987.

## QUESTIONS

1. Why are air quality criteria descriptive?
2. Why are air quality standards prescriptive?
3. Evaluate the use, effectiveness, and equity of local, state, provincial, or national air quality standards in your community.
4. Prepare a table similar in format to Tables 24.1 through 24.6 for another pollutant not yet required by the administrator of the US EPA to have a criteria document.
5. Discuss the relative merits of stating air quality standards as 1-h, 3-h, 8-h, 24-h, and annual averages.
6. Discuss the relative merits of national versus local air quality standards.
7. Discuss the differences in approach in using air quality standards (as in the United States), air quality objectives (as in Canada), and air quality goals (as in certain other countries).
8. Discuss the advantages and disadvantages of promulgating only one set of air quality standards (as in most countries) and of employing secondary and tertiary (PSD) standards, as in the United States.
9. Discuss the problem caused by cigarette smoking in the evaluation of epidemiological data on the effect of air pollution on respiratory disease.
10. Compare the NAAQS and NAAQO for US and Canada, respectively. What do they have in common and how do they differ?
11. How have air quality standards changed since those promulgated under the Clean Air Act of 1970?
12. Why have ozone and PM been the attention of intensive scrutiny since the mid-1990s? How have the AQCDs changed to address the research findings?