

Air Quality Assessments:
Implications for Pediatric Environmental Health

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I. INTRODUCTION

Hippocrates (*ca* 460 BC – *ca* 370 BC), an early Greek medical school instructor, is widely regarded as the founder of medicine. He strove to elevate a practice of magic and superstition to a profession of art and science (1). In his honor, medical students in the modern era still take the solemn Hippocratic Oath as a final step before graduating as physicians. However, Hippocrates should be considered the founder of environmental medicine as well, for he recognized some 2400 years ago the impact of environment on human health in his treatise, entitled *On Airs, Waters, and Places*:

Whoever wishes to investigate medicine properly, should proceed thus: in the first place to consider the seasons of the year, and what effects each of them produces for they are not at all alike, but differ much from themselves in regard to their changes. Then the winds, the hot and the cold, especially such as are common to all countries, and then such as are peculiar to each locality.... But such cities as lie to the west, and which are sheltered from winds blowing from the east, and which the hot winds and the cold winds of the north scarcely touch, must necessarily be in a very unhealthy situation.... [The inhabitants'] voices are rough and hoarse owing to the state of the air.... (2)

Over two millennia later, the relationship between environmental air quality and health remains somewhat nebulous. Numerous organizations have established indoor and outdoor air quality standards for a wide range of pollutants in order to protect human health. These efforts have contributed to the reduction of many pollutants in the environment, including settings such as homes, schools and workplaces. Unfortunately, a

confusing array of acronyms has evolved over the years that renders interpretation of air-quality assessments difficult at best. (Table 1)

As a group, children represent a population that remains particularly vulnerable to the effects, both immediate and long-term, of air pollution. Approximately 10 million children under 16 years of age in the U.S., for example, suffer from asthma, a chronic inflammatory disorder of the airways. Prevalence of asthma has increased 58% since 1980, while mortality has increased 78%; the disease disproportionately affects urban and racial/ethnic minority populations. (3) Protection of children, therefore, has become a priority for regulation and research. The purpose of this monograph is 1) to describe governmental, industrial, and other organizations that establish standards for—and regulation of—air quality; and 2) to review specific air pollutants that affect both indoor and outdoor air quality and children’s environmental health.

II. AIR QUALITY RULES AND RECOMMENDATIONS

A. Indoor Air

1. American Society of Heating, Refrigerating and Air-Conditioning Engineers

The Standards Committee of the *American Society of Heating, Refrigerating and Air-Conditioning Engineers* (ASHRAE), a member of the *American National Standards Institute* (ANSI), periodically publishes a national consensus statement known as Standard 62. The purpose of Standard 62 is to establish ventilation requirements for a variety of indoor spaces that will ensure acceptable air quality for human occupants. Compliance with Standard 62 is “voluntary until and unless a legal jurisdiction makes compliance mandatory through legislation.” (4)

Most recently revised in 1999, Standard 62 defines a number of issues that pertain to indoor air quality. “Acceptable indoor air quality” specifies that contaminants, if present, are below harmful concentrations “as determined by cognizant authorities” and that 80% or more of occupants are subjectively satisfied with the conditions. The Standard acknowledges that indoor air quality may not be universally acceptable to all occupants at a given time, as individual perceptions of air quality, which are dependent on preferences for factors such as air temperature and humidity, may vary. Furthermore, certain populations, such as children, may be more sensitive to conditions that may be tolerated by the general population.

The most recent revision of the Standard clarifies several important points. First, maintenance of comfortable conditions in certain spaces, such as garages, may be neither practical nor necessary. Second, “cognizant authorities” now consider environmental tobacco smoke (ETS) to be a health hazard; therefore, the revised Standard, while not prohibiting smoking, no longer implies that ventilation in building should allow for a moderate amount of smoking. Third, indoor carbon dioxide (CO₂) concentration should not be interpreted as an overall measure of air quality; rather, it is intended as a proxy for all human bioeffluents (i.e., in addition to CO₂, human occupants produce water vapor, particulate matter, biological aerosols, and volatile organic compounds). Standard 62 recommends that ventilation systems maintain indoor CO₂ concentration below 700 parts per million (ppm) by volume in order to meet occupant criteria for comfort and odor. Modern ventilation systems usually vary air flow rates through indoor spaces—effectively, dilution with outdoor air—to achieve acceptable CO₂ concentrations; however, if a closed system instead employs only a specialized filter to remove CO₂, it is

possible that concentrations of the other bioeffluents may rise. Standard 62 includes requirements for the flow of fresh outdoor air that would properly ventilate a variety of indoor spaces, including commercial, institutional and residential facilities (see Table 2).

(4)

From a strictly toxicologic point of view, subacute CO₂ exposure may cause somnolence, and the gas may also behave in the acute setting as a “simple asphyxiant” (defined as a substance that displaces oxygen, impairing pulmonary gas exchange and causing hypoxemia). (5) Even in the absence of CO₂, conditions of mild hypoxia—similar to those found aboard a routinely “pressurized” airliner—may have significant clinical effects. In an interventional study that exposed 34 healthy term infants (mean age, 3.1 months) to mild hypoxia, oxygen desaturations were observed. Over a mean exposure period of 6.3 (SD 2.9) hours, baseline oxygen saturations fell from a median of 97.6% (range, 94.0% to 100%) in room air to 92.8% (range, 84.7% to 100%) in 15% oxygen in nitrogen. There was also a statistically significant 3.5-fold increase in the proportion of time spent in periodic apnea. (6)

B. Outdoor Air

1. The Clean Air Act and Amendments

The Clean Air Act (CAA), first passed in 1963, has evolved as a result of amendments in 1970, 1974, 1977, 1990 and 1997. The original legislation established standards to control air pollution from both stationary and mobile sources, but had little power of enforcement. The 1970 amendment to the CAA created National Ambient Air Quality Standards (NAAQS), intended to afford greater protection to the health and

welfare of the population. (7) The CAA directs the *U.S. Environmental Protection Agency* (EPA; see below) to conduct a review of major pollutants every 5 years in order to ensure that state-of-the-art science remains the basis for these standards. In April 1997, President Clinton issued Executive Order 13045, “Protection of Children from Environmental Health Risks and Safety Risks.” (8). The order requires all federal agencies for the first time to consider children and their special risks in the formulation of federal standards. Three months later, the EPA announced revised standards for ozone and the first-ever standards for fine particulate matter, essential components of smog and soot, respectively. EPA Administrator Carol Browner defended the action, arguing that in the face of compelling new scientific evidence that associates these pollutants with significant pulmonary morbidity and mortality, updated standards were required to safeguard Americans—including children—from rising threats to health and welfare. (9) On June 22, 2000, a federal appeals court upheld a challenge to 1998 EPA rules that could remove 1.1 million tons of nitrogen oxides (another contributor to smog) annually. If enacted, these rules could remove, by 2003, the pollution equivalent of 166 million cars at a cost of \$1.7 billion annually over 15 years. The EPA contends that lower medical expenses and better public health could offset the cost. (10)

As a result of these and other actions, industry representatives and a United States Court of Appeals recently have challenged the constitutionality of the government’s mandate to consider only health and welfare issues of air pollution. This has prompted the United States Supreme Court to conduct a review of the CAA. In the fall of 2000, the justices are scheduled to decide whether this key statute should require the federal

government to consider the economic costs in addition to the benefits of reducing air pollution. (11)

2. The Environmental Protection Agency

In 1970, Congress created the United States Environmental Protection Agency (EPA), charged with oversight of regulation of air, water and land pollutants. Despite EPA sovereignty over six principal air pollutants, individual states have responsibility for attaining and maintaining NAAQS. In 1977, the EPA recognized the need to regulate additional “air toxics” and therefore set National Emission Standards for Hazardous Air Pollutants (NESHAPs) (see Hazardous Air Pollutants, below). (7)

The EPA maintains an extensive site on the World Wide Web that covers all aspects of its regulatory and educational missions. (12) Several pages of this Web site are devoted to the EPA’s Office of Air and Radiation (13) and its Office of Air Quality Planning and Standards (14). One page lists provisions of the 1990 CAA, as well as changes that occurred in 1997 (15). Other pages describe the six principal pollutants, often referred to as “criteria” pollutants (16), and the corresponding NAAQS (see Criteria Air Pollutants, below). Other pages of the EPA Web site are designed to educate teachers, students and younger children about various aspects of air pollution (17, 18). The EPA also maintains an Office of Children’s Health Protection, created after President Clinton’s 1997 Executive Order. (8)

3. Other Organizations Involved in Air Quality Rules and Recommendations

a. The *Committee on Environmental Health of the American Academy of Pediatrics* (COEH-AAP) recently published its *Handbook of Pediatric Environmental Health*. This comprehensive guide includes background chapters on special considerations of children and the environmental history. In addition, there are chapters on a variety of specific environmental contaminants and diseases, including air pollutants (indoor and outdoor), asbestos, carbon monoxide, environmental precipitants of asthma, environmental tobacco smoke, ionizing radiation (including radon), lead, and mercury. Other chapters characterize specific environments, such as arts and crafts, childcare, schools, waste sites, and workplaces, all of which may affect children. The Handbook also lists the Committee's Policy Statements that have been published over time in the journal *Pediatrics* (19).

b. Hazardous air conditions often exist in the workplace. Several agencies concern themselves with the health and safety of workers that may include adolescents:

The *American Conference of Governmental Industrial Hygienists* (ACGIH) is a non-governmental professional society concerned with administrative and technical aspects of both occupational and environmental health. In an effort to control health hazards, ACGIH has established guidelines that are published in the form of threshold limit values (TLVs) and biological exposure indices (BEIs). An ACGIH policy statement cautions, however, that only individuals trained in the practice of industrial hygiene should apply TLVs and BEIs as guidelines to improve worker protection; furthermore, TLVs and BEIs were never intended to serve as legal standards (20).

Congress enacted the Occupational Safety and Health Act in 1970, which gave rise to the *National Institute for Occupational Safety and Health* (NIOSH) of the Centers for Disease Control and Prevention (CDC) and the Department of Health and Human Services. NIOSH is a research agency devoted to the prevention of work-related illnesses and injuries. (21) NIOSH creates Recommended Exposure Limits (RELs) and evaluates possible health hazards in workplaces through its Health Hazard Evaluation (HHE) Program. NIOSH prefers the general term “indoor environmental quality” (IEQ) (22) to the more specific “indoor air quality” (IAQ), “sick building syndrome” (SBS) or “building-related illness” (BRI; see below).

The Occupational Safety and Health Act of 1970 also created the *Occupational Safety and Health Administration* (OSHA) in the U.S. Department of Labor (DOL), responsible for creating and enforcing safety and health regulations in the workplace. (23) OSHA develops Permissible Exposure Limits (PELs) for a variety of chemicals that workers may encounter. Industrial hygienists employ PELs as a primary tool in disease prevention in these settings. The DOL maintains a Safe Work / Safe Kids Web site in the interest of working teenagers. (24)

The *American Industrial Hygiene Association* (AIHA), like ACGIH, is a technical-professional organization concerned with environmental factors arising in the workplace that may affect both workers and members of the community. (25) AIHA issues Workplace Environmental Exposure Levels (WEELs) and published in 1998 a “White Paper” that was critical of the current use of OSHA PELs, the majority of which were developed three decades ago (new PELs have been developed for only about two dozen substances in over 25 years). AIHA believes that a variety of organizations should

generate scientific recommendations for air quality (NIOSH's RELs; ACGIH's TLVs; AIHA's WEELs) from which OSHA should review and update their PELs on a three- to five-year cycle. (26)

None of these standards for air quality are formulated specifically for children. Even if older working adolescents have adult-like physiology, they may nevertheless have increased susceptibility to many airborne hazards. For example, some hazards may pose an ongoing threat to health, and damage may be cumulative (chronicity), or an insult early in life may express itself decades later (latency).

III. SPECIFIC AIR POLLUTANTS

A. Indoor Air

1. Asbestos

Asbestos refers to a group of naturally occurring fibrous minerals that includes actinolite, amosite, anthophyllite, chrysotile, crocidolite and tremolite. Although no studies are available on the short-term effects of asbestos, chronic exposure may cause asbestosis, an impairment of respiratory function seen primarily in asbestos workers, as well as a variety of cancers. Neoplasms associated with asbestos include lung cancer, mesothelioma (affecting the thin membranes of the lung and abdominal cavity) and possible gastrointestinal cancers. The EPA has classified asbestos as a Group A, or known, human carcinogen. The *International Agency for Research on Cancer* (IARC) of the *World Health Organization* (WHO) lists asbestos as a Group 1 carcinogen (sufficient evidence for carcinogenicity to humans). (27) Exposure to asbestos may occur through erosion of natural deposits in rock or soil, or from disintegration of automotive

products (brake and clutch pads) or building materials (asbestos-cement pipes, insulation, roofing, floor and ceiling tiles). Fibers are usually inhaled as particles suspended in the air but may be ingested when they enter water supplies. Permissible indoor air concentrations range from 1 to 200 ng/m³. The EPA banned new uses of asbestos in 1989, and requires that school systems remove or cover existing asbestos. (28, 29)

2. Combustion Products

a. Carbon Monoxide

Carbon monoxide (CO) is a gas that results from the incomplete combustion of any hydrocarbon-containing fuel. CO is also generated in vivo: normal catabolism of hemoglobin produces blood carboxyhemoglobin (COHb) concentrations of up to 3%, and may exceed 10% in smokers. Although high levels of CO may be found in the ambient air of some metropolitan areas, the general trend of CO emissions and concentrations is downward (see Criteria Air Pollutants, below). (30) CO may pose a significant hazard in closed or indoor environments; approximately 600 unintentional deaths from CO are reported annually in the U.S., and the number of intentional deaths may be 10 times as high. CO deaths may occur when heating, ventilation, cooking, or automotive exhaust systems malfunction or are used inappropriately. (31)

An odorless, colorless and nonirritating gas, CO is notorious for producing symptoms similar to those of viral syndromes. Most individuals will not experience symptoms or health effects at ambient CO concentrations under 15 parts per million (ppm). Effects appear insidiously as gas concentration rises. In cases of severe CO poisoning, a syndrome of delayed neurologic sequelae (DNS) may emerge between two

and 30 days following exposure, although hypoxia alone does not adequately explain the syndrome. (32) Death may follow acute exposures associated with COHb concentrations above 35%. Duration of exposure, however, also plays a role in producing symptoms. For example, the COHb concentration of a nonsmoker will be approximately 10% after 15 minutes in an ambient CO concentration of 400 ppm, but the same COHb may be reached after approximately 90 minutes at a lower CO concentration of 100 ppm. (33)

Advances in sensor and microprocessor technology have allowed the manufacture of inexpensive CO detectors, and in 1989, the *U.S. Consumer Product Safety Commission* (CPSC) urged the *Underwriters Laboratory* (UL) to create a standard for detectors. A UL-listed detector must sound an 85-decibel alarm (sufficiently loud to awaken the average person) if, for example, an ambient CO concentration of 100 ppm is exceeded for 16 minutes. A detector must also pass a simulated “rush hour” test, during which it must ignore a CO concentration of 35 ppm for at least one hour, then fresh air for six hours, and finally another hour at 35 ppm. (33)

b. Environmental Tobacco Smoke

Despite claims of the tobacco industry to the contrary, the public health community has for over 30 years recognized the dangers of tobacco smoking. In more recent years, attention has turned to the threat of environmental tobacco smoke (ETS), a mixture of many substances, which the EPA now classifies as a Class A (known) carcinogen. (34) Children are unusually susceptible to the health effects of this pervasive pollutant, and rarely have control over the air quality of their immediate environment, particularly if parents or caregivers smoke. The AAP issued a position statement on the

subject, citing strong epidemiologic evidence that ETS exposure in children is associated with increased rates of middle ear effusion, lower respiratory illness, asthma, and sudden infant death syndrome (SIDS). Furthermore, childhood exposure to ETS increases the potential for latent effects, such as cancer in adulthood. (35)

c. Particulate Matter

A variety of sources may contribute to the existence of particulate matter, a mixture of solid particles and liquid droplets that are suspended in the air. Relatively large particles ($> 2.5 \mu\text{m}$ in diameter) may be generated from windblown dust and grinding operations, while fine particles ($= 2.5 \mu\text{m}$ in diameter, or $\text{PM}_{2.5}$) tend to arise from the combustion of fuels. Prior to 1987, the EPA regulated only “total suspended particulates” that included particles $> 10 \mu\text{m}$ in diameter. Evidence accumulated by the late 1980s implicated particulate matter $= 10 \mu\text{m}$ in diameter, or PM_{10} , in the etiology of significant health effects. These particles may penetrate deeper into the lung to aggravate respiratory diseases such as asthma that effect children disproportionately, and the EPA subsequently focused its monitoring efforts on smaller particles. More recent research has determined that the finest particles, $\text{PM}_{2.5}$, are more strongly associated with the most significant health effects (see discussion of particulate matter under Outdoor Air section, below) (36).

Concentrations of PM_{10} in some indoor settings may exceed those concentrations outdoors. Although some PM_{10} results from combustion of fuels, one study in the Netherlands determined that the source of PM_{10} in classrooms was resuspended coarse

particles or suspension of soil particles, and that the children's activity probably caused the suspension. (37)

3. Lead

The element lead (Pb) is a potent toxin that has been studied extensively. Lead causes a broad array of problems, but earns its notoriety for disturbances of the central nervous system. In children, neurodevelopmental effects may be subtle but irreversible. The hazards of lead have been known since antiquity, and the special risks to children have been known since the 19th century. Children who ingest lead-containing paint chips are at risk for elevated blood lead levels, and many countries banned or restricted the use of lead paint early in the 20th century. The lead industry in the U.S. failed, however, to bridle its use in paint for interior walls and woodwork, and actually developed advertising strategies for white lead in the 1920s that featured children in the campaign. (38) Over the last three decades, removal of lead from paint, gasoline and other sources, as well as abatement of lead-painted houses, has decreased lead exposure of U.S. children; blood lead levels have fallen dramatically. Many children, however, are still exposed to unacceptable quantities of lead that persist in the environment. In fact, lead-contaminated house dust represents a significant source for children with low-level elevations of blood lead. In one study of children 12 to 31 months of age, investigators used dust lead standards of 5 µg/sq. ft, 20 µg/sq. ft, and 40 µg/sq. ft on non-carpeted floors (well below EPA standards). The percentage of children with blood lead levels of at least 10 µg/dL was 4%, 15% and 20%, respectively. (39)

4. Molds

Fungal spores are ubiquitous components of both indoor and outdoor air, and are recognized as significant respiratory allergens. One study of 48 schools in the United States revealed the consistent presence of five fungal genera in the *outdoor* air:

Cladosporium, *Penicillium*, *Chrysosporium*, *Alternaria*, and *Aspergillus*. At 20 of the schools, concomitant air sampling in *indoor* complaint areas revealed significant elevations of *Penicillium* species compared with outdoor air. In another 11 schools, *Stachybotrys atra* (now known as *Stachybotrys chartarum*), was isolated from areas of visible growth. (40) The presence of these fungi may explain, in part, symptoms of the “building-related illness” (BRI; see below). In April 2000, the New York City Department of Health released a document on assessment and remediation of indoor fungi. The significance of this document is that it represents an expansion of the Department’s previous document that dealt only with *Stachybotrys atra*. The new document includes discussions of *Aspergillus*, *Penicillium*, *Fusarium*, *Trichoderma* and *Memmoniella* species, all of which produce toxic cyclic metabolites known as mycotoxins. High concentrations of these fungi may also cause the Organic Dust Toxic Syndrome (ODTS), hypersensitivity pneumonitis (HP) and allergic reactions. (41)

5. Radon

The decay of naturally occurring uranium and thorium in rock and soil produces radon-222, a radioactive gas. Radon undergoes further decay into two radon daughters, isotopes of the element polonium, both of which emit alpha particles. Alpha particles travel very short distances into human tissue, but are nevertheless capable of causing lung

cancer in humans; they account for an estimated 14,000 deaths per year. Inhalation of polonium isotopes allows their ionizing radiation to initiate carcinogenesis in bronchial epithelial cells. A synergistic effect is seen among smokers, probably because the isotopes travel deeper into the lungs, attached to smoke particles. Convincing evidence of radon's health effects has been acquired over decades. (42)

Radon gas may enter a building dissolved in well water or through cracks or other structural elements of the foundation. Radon that escapes to the outdoors is diluted to ambient concentrations of 0.2 – 0.7 picoCuries/L (pCi/L). In a tightly sealed house, however, radon concentrations—and the directly proportional risk of lung cancer—may rise. Although no level is considered “safe,” the EPA’s current “action level” of 4 pCi/L is derived from the limitations of current mitigation technology to prevent radon entry or improve its exhaust. Exposure to this action level over a lifetime would result in lung cancer in two of 1000 non-smokers exposed over a lifetime (this proportion would be approximately 29/1000 in smokers). Like CO, radon is colorless and odorless, and the EPA recommends that all homes be tested for radon on levels below the third floor. (42)

6. Building-Related Illness

The concept of “building-related illness” (BRI) or the “sick building syndrome” (SBS) has emerged over the last two decades. The WHO has defined it as an excess of work-related irritation of the skin and mucous membranes of occupants of modern office buildings, although schools and other institutions have been studied. (40, 43) This definition of BRI has now been broadened to include other health complaints, including allergic rhinitis, difficulty breathing, headaches, flu-like symptoms, and watering of the

eyes. Typically no single cause can be identified. Implicated are a variety of respiratory irritants, such as nitrogen and sulfur oxides, fungi, hydrocarbons, particulates, known or suspected carcinogens (asbestos, ETS, formaldehyde, radon), and chemicals released from building materials. (40) Thermal discomfort, work-related stress, noise and poor lighting may contribute substantially to BRI health complaints. (43) A study of children followed for up to 18 months of age in their home or daycare environments found that mothers were more likely to complain of BRI-like symptoms in their children who had a history of atopy, including eczema, dry skin, and reactions to food. (44)

7. Volatile Organic Compounds

Volatile organic compounds (VOCs) are vapors or gases emitted at ambient temperatures from products such as building materials, clothing, upholstery, spray cans, paint, and office machine toner. These products may contain hazardous constituents, including benzene, carbon tetrachloride and formaldehyde. VOCs typically act as ocular or respiratory irritants. They may also cause renal or hepatic damage, or act as teratogens or carcinogens. (40)

B. Outdoor Air

1. Aerosols

An aerosol is defined as a small droplet or particle suspended in the atmosphere. Natural (e.g. volcanic) and industrial emissions are the main contributors to the production of aerosols. These aerosols should not be confused with consumer aerosol products that rely on a pressurized gas to propel a substance from a container.

Commercial propellants, incidentally, have not used ozone-depleting chemicals in the U.S. since the late 1970s (see next section) as a result of switching to non-ozone-depleting hydrocarbons or other compressed gases. (45)

2. Chlorofluorocarbons

Chlorofluorocarbons (CFCs), chemicals that contain chlorine and fluorine, formerly were used ubiquitously as propellants but are now restricted to non-consumer products such as refrigerants and solvents. CFCs tend to remain stable in layers of the atmosphere closest to earth (troposphere). In the stratosphere, however, ultraviolet radiation degrades CFCs, releasing chlorine atoms that deplete the ultraviolet-shielding ozone layer. (45)

3. Criteria Air Pollutants

The CAA required the EPA to set NAAQS for six principal or “criteria” pollutants in 1990. The criteria pollutants are carbon monoxide (CO), lead (Pb), nitrogen dioxide (NO₂), particulate matter (= 10 μm in diameter, PM₁₀, and = 2.5 μm in diameter, PM_{2.5}), and sulfur dioxide (SO₂). Table 3 lists NAAQS for the six criteria pollutants. (46) NAAQS were most recently updated in 1997, although industry groups have challenged the NAAQS for ozone and fine particulate matter in a federal lawsuit. (11)

a. Carbon Monoxide

In 1995, transportation sources accounted for 81% of total carbon monoxide (CO) emissions. However, long-term improvements were seen in the U.S. between 1986 and

1995. The EPA NAAQS for CO is 9 ppm, measured as an annual second-maximum 8-hour average concentration. (30) In December 1994, a temperature inversion in Chicago trapped CO in the atmosphere for over 24 hours. Ambient levels rose above 15 ppm from a baseline of < 4 ppm, and the city reported 1800 CO detector alarms in a single day. (33)

b. Lead

The phase-out of lead in gasoline, completed in 1995, has resulted in a dramatic improvement in lead emissions from vehicles, decreasing 99% from 1988 to 1997. Ambient lead concentrations in the U.S. decreased 67% over the same time period. The NAAQS for lead is currently an annual maximum quarterly average of $1.5 \mu\text{g}/\text{m}^3$; violations tend to occur in the vicinity of point sources such as lead smelters. (47)

c. Nitrogen Oxides

Combustion of fossil fuels produces oxides of nitrogen (NO_x), which react with atmospheric water, oxygen and oxidants to form nitric acid, a component of acid rain. (48) The NO_x family includes nitric oxide (NO), nitrous oxide (N_2O) and nitrogen dioxide (NO_2). Although all three gases have effects on human physiology, NO_2 is of greatest environmental significance and has been shown to reduce pulmonary capacity. A study of risk factors for heat stroke in Tokyo determined that maximum daily temperature and concentrations of NO_2 were the most significant risk factors for the development of heat stroke in all age groups, including children 0-14 years of age. Although heat stroke is formally defined as core body temperature $> 40.6^\circ \text{C}$ with multiple organ dysfunction,

the authors broadened the definition to include diagnoses of heat cramps and heat exhaustion. (49)

d. Ozone

Among the most dangerous and pervasive of air pollutants is ground-level ozone (O_3), commonly referred to as smog. The modifier “ground-level” distinguishes O_3 from the ozone that exists at high levels of the atmosphere and protects the earth from harmful ultraviolet radiation. O_3 , which forms when sunlight acts on NO_x and VOCs, reacts slowly with water to form reactive hydroxyl radicals, which can oxidize a variety of biomolecules, impairing cellular architecture and energy production, particularly in the pulmonary tree. Investigators in Taiwan have studied the short-term effects of O_3 on the pulmonary function of nearly 1000 primary school children, 8 to 13 years of age. Spirometry performed once on each child in this cross-sectional study revealed a negative association of peak O_3 concentration measured the previous day with forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1). The slope of lung function decrease with respect to O_3 concentration for these children was approximately 1 mL/parts per billion (ppb). (50) In a study of the long-term effects of O_3 , investigators followed a cohort of 1150 California school children for three years. Lung function was recorded before and after summer in each of three successive years (1994-1996), and regression analyses were used to separate the effect of ozone from confounders such as study site, gender, atopy, passive smoking, baseline lung function, and increase in height. Summertime ozone was associated with lesser increases in several lung function parameters, an effect not seen with other criteria air pollutants. (51) Investigators in

Southern California followed children 6 to 12 years of age over a one-year period in order to compare personal O₃ exposures in two geographic areas. One group consisted of children living near sea level at the eastern edge of the Los Angeles air basin; the other group included those living in mountainous communities at least 1.2 km above sea level. For six consecutive days each month, the children wore passive O₃ samplers and kept diaries of activity. This longitudinal study of personal O₃ exposure revealed concentrations 0-12 ppb higher in children who lived in the mountainous communities in the “O₃-months” (May through September). No significant differences were seen in the “non-O₃-months” (October through April). (52)

Mounting evidence of O₃ effects on lung function prompted the EPA in 1997 to lower the NAAQS for O₃ to 80 ppb over 8 hr from the previous 120 ppb measured over 1 hr (roughly equivalent to 90 ppb over 8 hr). (53) In some studies, however, a decrease in children’s pulmonary function can occur at peak hourly O₃ concentrations below the new NAAQS of 80 ppb/8 hr. (50) The *American Lung Association* (ALA) recently released its State of the Air 2000 report, which focuses on O₃. State of the Air 2000 rates cities and counties on the number of days with unacceptable O₃ levels for the years 1996-1998, and states that over 132 million Americans (including 29 million children and 7 million asthmatics), live in areas that received a “failing” grade. Unlike levels of carbon monoxide, which have declined, levels of O₃ are on the rise in many communities and are unacceptable for 72% of those who live in monitored areas. (54)

e. Particulate Matter

Prior to 1997, NAAQS existed for soot, or particulate matter = 10 μm in diameter (PM_{10}). Recent evidence, however, implicates even smaller particles in the pathogenesis of the most severe human pulmonary disease and premature death; consequently, the EPA has established two new standards for particulates = 2.5 μm in diameter ($\text{PM}_{2.5}$). The NAAQS for $\text{PM}_{2.5}$ are 65 $\mu\text{g}/\text{m}^3$, based on the 3-year average of the 98th percentile of 24-hr $\text{PM}_{2.5}$ concentrations within an area, and 15 $\mu\text{g}/\text{m}^3$, based on the 3-year average of annual arithmetic mean $\text{PM}_{2.5}$ concentrations within an area. The PM_{10} standard is now based on the 99th percentile of 24-hr PM_{10} concentrations at each monitor within an area. (55)

In a study in six North Carolina communities located in the neighborhood of waste incinerators, however, incinerator emissions had insignificant impact on monitored pollutants, including $\text{PM}_{2.5}$, and no evidence of altered lung function was found. Children eight years of age and older were included in this study. (56) A study of asthma in children in Seattle found that a change of 11 $\mu\text{g}/\text{m}^3$ in estimated $\text{PM}_{2.5}$ concentrations was associated with significantly higher emergency department visit rates. This finding is relevant because these relationships were noted *below* the new NAAQS of 15 $\mu\text{g}/\text{m}^3$ for $\text{PM}_{2.5}$. (57). Intriguing new evidence suggests that polycyclic aromatic hydrocarbons (PAHs), which may “piggyback” on particles of $\text{PM}_{2.5}$, could act as immune mediators and explain, in part, the rise in reactive airway disease prevalence. (58)

f. Sulfur Dioxide

Like the NO_x compounds, sulfur dioxide (SO_2) is produced from the combustion of fossil fuels and contributes to the development of acid rain. (48) SO_2 may also play a

role in reactive airways disease in children. In a cross-sectional retrospective study of Norwegian school children, exposure during infancy to SO₂ emitted from an aluminum smelter (compared to children in similar but non-industrialized valley) was identified as a risk factor for airway hyper-responsiveness at 7-13 years of age. The odds ratio for a 10-μg/m³ increase in SO₂ at 0-12 months of age was 1.62 (95% CI, 1.11-2.35). (59) A prospective cohort study investigated the effect of a government air quality intervention on airway responsiveness of primary school children 8-11 years of age in a factory-polluted district of Hong Kong. Comparison was made to a control group of children in a less polluted district. The intervention, which began in July 1990 and limited sulfur content of fuels to 0.5%, reduced ambient SO₂ levels by 84%. Furthermore, airway hyper-reactivity after histamine challenge was reduced significantly in the study group one and two years after the intervention. (60)

4. Hazardous Air Pollutants

The majority of air toxics, or hazardous air pollutants (HAPs), are emitted from man-made (industry and vehicle emissions) and natural sources (forest fires and volcanic eruptions). HAPs are pollutants that are known or suspected to cause serious health problems, and include metals, particulate matter (PM), gases adsorbed onto PM, and vapors. Health effects from HAPs may range from reversible eye irritation to exacerbation of asthma to cancer. A list of the original HAPs under the CAA may be found on the EPA Web site. (61). The EPA has published a brochure that describes, in plain language, progress in setting “maximum achievable control technology” (MACT)

for HAPs. MACT standards are based on emission performance of better-controlled and lower-emitting sources in a particular industry. (62)

IV. CONCLUSION

The purpose of this monograph is to provide an overview of air quality assessments in the context of children's environmental health. A comprehensive understanding of air quality and its impact on human health must consider the special susceptibilities of children. Historically, air quality policies were derived largely from the experience of government and industry, and children's physiology, growth and development were not always considered. Only recently (1997) has an executive order of the President mandated children's environmental health a priority for research and regulation. In the years ahead, air quality research may shed new light on the health of children, and regulation may afford additional protections.

This monograph and the references that follow represent a mere sampling of information available. Governmental, industrial and medical literature have provided vast quantities of data, but the World Wide Web has emerged as a powerful tool that offers unprecedented flexibility and accessibility for the dissemination of new information. Table 4 is a compilation of a number of home pages of Web sites that are relevant to the interpretation of air quality assessments, and updates to these sites will prove useful in a rapidly changing world.

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Note: the author has made reference to numerous sites on the World Wide Web and believes that they represent reputable organizations as well as timely and accurate information; Web content, however, may vary with time and may not always represent current information.

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Table 1. Abbreviations relevant to the interpretation of air quality assessments.

AAP	American Academy of Pediatrics
ACGIH	American Conference of Governmental Industrial Hygienists, Inc.
AIHA	American Industrial Hygiene Association
ALA	American Lung Association
ANSI	American National Standards Institute
ASHRAE	American Society of Heating, Refrigerating and Air-Conditioning Engineers
ATSDR	Agency for Toxic Substances and Disease Registry (Centers for Disease Control and Prevention, U.S. Department of Health and Human Services)
BEIs	biologic exposure indices
BRI	building-related illness
CAA	Clean Air Act
CDC	Centers for Disease Control and Prevention (US Department of Health and Human Services)
CFCs	chlorofluorocarbons
CO	carbon monoxide
CO ₂	carbon dioxide
COEH-AAP	Committee on Environmental Health of the American Academy of Pediatrics
COHb	carboxyhemoglobin
CPSC	U.S. Consumer Product Safety Commission
DNS	delayed neurologic sequelae

DOL	U.S. Department of Labor
DHHS	U.S. Department of Health and Human Services
ETS	environmental tobacco smoke
EPA	U.S. Environmental Protection Agency
FVC	forced vital capacity
FEV ₁	forced expiratory volume in one second
HAPs	hazardous air pollutants
HHE	Health Hazard Evaluation program
HP	hypersensitivity pneumonitis
IAQ	indoor air quality
IARC	International Agency for Research on Cancer
IEQ	indoor environmental quality
MACs	maximum allowable concentrations
MACT	maximum achievable control technology
NAAQS	national ambient air quality standards
NESHAPs	national emission standards for hazardous air pollutants
NIOSH	National Institute for Occupational Safety and Health
NO	nitric oxide
NO ₂	nitrogen dioxide
NO _x	nitrogen oxides
O ₃	ground-level ozone
OAQPS	Office of Air Quality Planning and Standards (U.S. Environmental Protection Agency)

OAR	Office of Air and Radiation (U.S. Environmental Protection Agency)
ODTS	Organic Dust Toxic Syndrome
OSHA	Occupational Safety and Health Administration (U.S. Department of Labor)
PAHs	polycyclic aromatic hydrocarbons
Pb	lead
pCi/L	picoCuries/L
PELs	permissible exposure limits
PM	particulate matter
PM _{2.5}	particulate matter = 2.5 micrometers in diameter
PM ₁₀	particulate matter = 10 micrometers in diameter
ppb	parts per billion
ppm	parts per million
RELs	recommended exposure limits
SBS	sick building syndrome
SIDS	sudden infant death syndrome
SO ₂	sulfur dioxide
TLVs	threshold limit values
TLV-C	threshold limit value—ceiling
TLV-STEL	threshold limit value—short-term exposure limit
TLV-TWA	threshold limit value—time-weighted average
UL	Underwriters Laboratory, Northbrook IL
UV	ultraviolet

VOCs volatile organic compounds
WEELs workplace environmental exposure levels
WHO World Health Organization

Table 2. Outdoor air requirements for ventilation. Adapted with permission from ASHRAE Standard 62-1999, Ventilation for Acceptable Indoor Air Quality. Copyright 1999 American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc., Atlanta. (3)

<u>Commercial facilities</u>	<u>L/s•person</u>	<u>L/s•m²</u>
Kitchens	8	
Parking garages (enclosed)		7.5
Dormitory sleeping areas		8
Offices		10
Locker rooms		2.5
Malls and arcades		1
Shipping and receiving		0.75
Warehouses		0.25
Supermarkets	8	
Spectator areas	8	
Game rooms	13	
Ice arenas		2.5
Swimming pools		2.5
Gymnasiums	10	
Vehicles	8	

Institutional facilities L/s•person

Classrooms	8
Laboratories	10
Libraries	8
Auditoriums	8

Residential facilities L/s•person L/s

Living areas = 7.5
(0.35 air changes per hour)

Kitchens	50 intermittent or 12 continuous or openable windows
Bathrooms	25 intermittent or 10 continuous or openable windows
Garages	50 per car

Key:

L/s•person, liters per second per person

L/s•m², liters per second per meter squared

L/s, liters per second

Table 3. National Ambient Air Quality Standards (NAAQS). Figures listed indicate only *primary* standards, set to protect the health of “sensitive” populations, including asthmatics and children (*secondary* standards set limits to protect animals, crops, buildings, and air visibility). Adapted from U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. (15)

<u>Pollutant</u>	<u>Standard Value</u>	
Carbon Monoxide (CO)		
8-hour average	10 $\mu\text{g}/\text{m}^3$	(9 ppm)
1-hour average	40 $\mu\text{g}/\text{m}^3$	(35 ppm)
Lead (Pb)		
Quarterly average	1.5 $\mu\text{g}/\text{m}^3$	
Nitrogen Dioxide (NO ₂)		
Annual arithmetic mean	100 $\mu\text{g}/\text{m}^3$	(53 ppb)
Ozone (O ₃)		
1-hour average	235 $\mu\text{g}/\text{m}^3$	(120 ppb)
8-hour average	157 $\mu\text{g}/\text{m}^3$	(80 ppb)
Particulate Matter < 2.5 μm in diameter (PM _{2.5})		
Annual arithmetic mean	15 $\mu\text{g}/\text{m}^3$	
24-hour average	65 $\mu\text{g}/\text{m}^3$	
Particulate Matter < 10 μm in diameter (PM ₁₀)		
Annual arithmetic mean	50 $\mu\text{g}/\text{m}^3$	
24-hour average	150 $\mu\text{g}/\text{m}^3$	
Sulfur Dioxide (SO ₂)		

Annual arithmetic mean	80 $\mu\text{g}/\text{m}^3$	(30 ppb)
24-hour average	365 $\mu\text{g}/\text{m}^3$	(140 ppb)

Key:

$\mu\text{g}/\text{m}^3$, micrograms per meter squared

ppm, parts per million

ppb, parts per billion

Table 4. World Wide Web home pages of organizations concerned with air quality.

<u>Organization</u>	<u>Uniform Resource Locator</u>
American Academy of Pediatrics	http://www.aap.org
American Conference of Governmental Industrial Hygienists, Inc.	http://www.acgih.org
American Industrial Hygiene Association	http://www.aiha.org
American Lung Association	http://www.lungusa.org
American Society of Heating, Refrigerating and Air-Conditioning Engineers	http://www.ashrae.org
Agency for Toxic Substances and Disease Registry	http://www.atsdr.cdc.gov
Centers for Disease Control and Prevention	http://www.cdc.gov
U.S. Consumer Product Safety Commission	http://www.cpsc.gov
U.S. Department of Labor	http://www.dol.gov
U.S. Department of Health and Human Services	http://www.dhhs.gov
U.S. Environmental Protection Agency	http://www.epa.gov
International Agency for Research on Cancer	http://www.iarc.fr
National Institute for Occupational Safety and Health	http://www.cdc.gov/niosh/homepage.html
Occupational Safety and Health Administration	http://www.osha.gov
World Health Organization	http://www.who.int