

POLICY STATEMENT

Organizational Principles to Guide and Define the Child Health Care System and/or Improve the Health of All Children

Committee on Environmental Health

Ambient Air Pollution: Health Hazards to Children

ABSTRACT. Ambient (outdoor) air pollution is now recognized as an important problem, both nationally and worldwide. Our scientific understanding of the spectrum of health effects of air pollution has increased, and numerous studies are finding important health effects from air pollution at levels once considered safe. Children and infants are among the most susceptible to many of the air pollutants. In addition to associations between air pollution and respiratory symptoms, asthma exacerbations, and asthma hospitalizations, recent studies have found links between air pollution and preterm birth, infant mortality, deficits in lung growth, and possibly, development of asthma. This policy statement summarizes the recent literature linking ambient air pollution to adverse health outcomes in children and includes a perspective on the current regulatory process. The statement provides advice to pediatricians on how to integrate issues regarding air quality and health into patient education and children's environmental health advocacy and concludes with recommendations to the government on promotion of effective air-pollution policies to ensure protection of children's health. *Pediatrics* 2004;114:1699–1707; *air pollution, adverse effects, children, asthma, environmental health.*

ABBREVIATIONS. PM_{2.5}, particulate matter with a median aerodynamic diameter less than 2.5 μm; PM₁₀, particulate matter with a median aerodynamic diameter less than 10 μm; EPA, Environmental Protection Agency; HAP, hazardous air pollutant; AQI, air quality index.

INTRODUCTION

Although it has been 3 decades since passage of the Clean Air Act in 1970 (Pub L No. 91–604), the air in many parts of the United States is far from clean. Air quality has improved in some areas but decreased in others.¹ In addition, there are important health effects from air pollutants at levels once considered safe. Children and infants are among the most susceptible to many of the air pollutants.

In 2002, approximately 146 million Americans were living in areas where monitored air failed to meet the 1997 National Ambient Air Quality Standards for at least 1 of the 6 “criteria air pollutants”: ozone, particulate matter, sulfur dioxide, nitrogen dioxide, carbon monoxide, and lead (Table 1).¹ Although the standards for ozone and particulate matter were revised in 1997, legal barriers have delayed

timely implementation.² Recent reports have identified adverse health effects at levels near or below the current standards for ozone, particulate matter, and nitrogen dioxide. Thus, the 1997 federal standards may not adequately protect children. Additionally, numerous other toxic air pollutants are of public health concern.³

Outdoor air pollution is also a major problem in developing countries. The World Health Organization found that the air quality in large cities in many developing countries is remarkably poor and that very large numbers of people in those countries are exposed to ambient concentrations of air pollutants well above the World Health Organization guidelines for air quality (www.who.int/ceh/publications/en/11airpollution.pdf).

Scientific understanding of the health effects of air pollution, including effects on children, has increased in the last decade. This statement updates a 1993 American Academy of Pediatrics (AAP) statement titled “Ambient Air Pollution: Respiratory Hazards to Children.”⁴

EFFECTS OF AIR POLLUTION ON CHILDREN

Children are more vulnerable to the adverse effects of air pollution than are adults. Eighty percent of alveoli are formed postnatally, and changes in the lung continue through adolescence.⁵ During the early postneonatal period, the developing lung is highly susceptible to damage after exposure to environmental toxicants.^{5–7}

Children have increased exposure to many air pollutants compared with adults because of higher minute ventilation and higher levels of physical activity.⁸ Because children spend more time outdoors than do adults, they have increased exposure to outdoor air pollution.^{9,10}

Infants, children, the elderly, and those with cardiopulmonary disease are among the most susceptible to adverse health effects from criteria pollutants.^{11–15} Lead is neurotoxic, especially during early childhood. Carbon monoxide interferes with oxygen transport through the formation of carboxyhemoglobin. Other criteria pollutants (ozone, sulfur dioxide, particulate matter, nitrogen dioxide) have respiratory effects in children and adults, including increased respiratory tract illness, asthma exacerbations, and decreased lung function (eg, changes in peak flow).^{11–12} In adults, particulate air pollution is associated with respiratory and cardiovascular hos-

TABLE 1. National Ambient Air Quality Standards for Criteria Air Pollutants, 1997

Pollutant	Primary Standards*
Ozone	
1-h average	0.12 ppm (235 $\mu\text{g}/\text{m}^3$)
8-h average	0.08 ppm (157 $\mu\text{g}/\text{m}^3$)
PM ₁₀	
Annual arithmetic mean	50 $\mu\text{g}/\text{m}^3$
24-h average	150 $\mu\text{g}/\text{m}^3$
PM _{2.5}	
Annual arithmetic mean	15 $\mu\text{g}/\text{m}^3$
24-h average	65 $\mu\text{g}/\text{m}^3$
Sulfur dioxide	
Annual arithmetic mean	0.03 ppm (80 $\mu\text{g}/\text{m}^3$)
24-h average	0.14 ppm (365 $\mu\text{g}/\text{m}^3$)
Nitrogen dioxide	
Annual arithmetic mean	0.053 ppm (100 $\mu\text{g}/\text{m}^3$)
Carbon monoxide	
8-h average	9 ppm (10 mg/ m^3)
1-h average	35 ppm (40 mg/ m^3)
Lead	
Quarterly average	1.5 $\mu\text{g}/\text{m}^3$

Additional information on air quality standards are available at www.epa.gov/air/criteria.html.

* People residing in regions with pollutant concentrations above the primary standard may experience adverse health effects from poor air quality.

pitalizations, cardiovascular mortality,¹⁶ and lung cancer.¹⁷ Air pollution also has effects on indirect health indicators such as health care utilization and school absences.^{11–13}

Although numerous studies have shown that outdoor air pollution exacerbates asthma, the effect of outdoor air pollution on the development of asthma has been less clear. Recently, a prospective study found that the risk of developing asthma was not greater, overall, in children living in communities with high levels of ozone or particulate air pollution. However, in communities with high levels of ozone, there was an increased risk of developing asthma in a small subset of children involved in heavy exercise (participation in 3 or more team sports per year [relative risk: 3.3; 95% confidence interval: 1.9–5.8]). This increased risk with heavy exercise was not seen in low-ozone communities. Time spent outside was also associated with new cases of asthma in high-ozone communities (relative risk: 1.4; 95% confidence interval: 1.0–2.1) but not in low-ozone communities.¹⁸ Additional studies are needed to define the role of outdoor air pollution in the development of asthma.

Children in communities with higher levels of urban air pollution (acid vapor, nitrogen dioxide, particulate matter with a median aerodynamic diameter less than 2.5 μm [PM_{2.5}], and elemental carbon [a component of diesel exhaust]) had decreased lung function growth, and children who spent more time outdoors had larger deficits in the growth rate of lung function.^{19,20} Ambient air pollution (especially particulate matter with a median aerodynamic diameter less than 10 μm [PM₁₀]) has also been associated with several adverse birth outcomes, as discussed in the next section.

Levels of ozone and particulate matter are high enough in many parts of the United States to present health hazards to children.¹ Additionally, National

Ambient Air Quality Standards for nitrogen dioxide may not be protective. Findings on these pollutants are summarized here.

Ozone

Ambient ozone is formed by the action of sunlight on nitrogen oxides and reactive hydrocarbons, both of which are emitted by motor vehicles and industrial sources. The levels tend to be highest on warm, sunny, windless days and often peak in midafternoon, when children are most likely to be playing outside.

Ozone is a powerful oxidant and respiratory tract irritant in adults and children, causing shortness of breath, chest pain when inhaling deeply, wheezing, and cough.¹¹ Children have decreases in lung function, increased respiratory tract symptoms, and asthma exacerbations on days with higher levels of ambient ozone.^{11,21–23} Increases in ambient ozone have been associated with respiratory or asthma hospitalizations,^{24,25} emergency department visits for asthma,²⁶ and school absences for respiratory tract illness.²⁷ In Atlanta, Georgia, summertime children's emergency department visits for asthma increased 37% after 6 days when ozone levels exceeded 0.11 ppm.²⁵ In southern California, school absences for respiratory tract illness increased 63% in association with a 0.02-ppm increase in ozone.²⁷

In healthy adults, ozone causes airway inflammation and hyperreactivity, decrements in pulmonary function, and increased respiratory tract symptoms.¹¹ Ozone exposures at concentrations of 0.12 ppm or higher can result in decrements in lung function after subsequent challenge with aeroallergen.²⁸ Although most of the controlled studies of ozone exposure have been performed with adults, it is reasonable to believe that the results of these findings could be extended to children.

Ozone may be toxic at concentrations lower than 0.08 ppm, the current federal regulatory standard. Field studies suggest potential thresholds of between 0.04 and 0.08 ppm (1-hour average) for effects on lung function.^{29–31} Recent studies of hospitalizations for respiratory tract illness in young children and emergency department visits for asthma suggest that the effects of ozone may occur at ambient concentrations below 0.09 ppm.^{32,33} Another study found associations of ozone and respiratory symptoms in children with asthma at levels below the current US Environmental Protection Agency (EPA) standards.³⁴ If these findings are confirmed, the ozone standards may need additional revision.

In addition to studies on short-term effects, 2 recent studies of college freshmen suggest that increasing cumulative childhood exposure to ozone may affect lung function when exposed children reach young adulthood, particularly in measures of flow in small airways.^{35,36} Early childhood exposures may, therefore, be particularly important.³⁵

Particulate Matter

PM₁₀ is small enough to reach the lower respiratory tract and has been associated with a wide range of serious health effects. PM₁₀ is a heterogeneous

mixture of small solid or liquid particles of varying composition found in the atmosphere. Fine particles (PM_{2.5}) are emitted from combustion processes (especially diesel-powered engines, power generation, and wood burning) and from some industrial activities. Coarse particles (diameter between 2.5 and 10 μm) include windblown dust from dirt roads or soil and dust particles created by crushing and grinding operations. Toxicity of particles may vary with composition.^{37,38}

Particle pollution contributes to excess mortality and hospitalizations for cardiac and respiratory tract disease.^{14,39–41} The mechanism for particulate matter-associated cardiac effects may be related to disturbances in the cardiac autonomic nervous system, cardiac arrhythmias, or increased blood concentrations of markers of cardiovascular risk (eg, fibrinogen).^{16,42}

Daily changes in mortality rates and numbers of people hospitalized are linked to changes in particulate air pollution.^{14,39–41} These studies and others have estimated that for every 10 μg/m³ increase in PM₁₀, there is an increase in the daily mortality rate between 0.5% and 1.6%. Effects were seen even in cities with mean annual PM₁₀ concentrations between 25 and 35 μg/m³. These recent studies suggest that even the current federal standards for PM_{2.5} (24-hour standard = 65 μg/m³; annual standard = 15 μg/m³) and PM₁₀ (24-hour standard = 150 μg/m³; annual standard = 50 μg/m³) should be lowered to protect public health. In 2002, California adopted more stringent standards for particulate matter: the annual average standard for PM_{2.5} is 12 μg/m³ and for PM₁₀ is 20 μg/m³.⁴³

In children, particulate pollution affects lung function^{44–46} and lung growth.¹⁹ In a prospective cohort of children living in southern California, children with asthma living in communities with increased levels of air pollution (especially particulates, nitrogen dioxide, and acid vapor) were more likely to have bronchitis symptoms. In this study, bronchitis symptoms refers to a parental report of “one or more episodes of ‘bronchitis’ in the past 12 months” or report that, “apart from colds, the child usually seems to be congested in the chest or able to bring up phlegm”.⁴⁷ The same mix of air pollutants was also associated with deficits in lung growth (as measured by lung function tests).¹⁹ Recent studies in different countries have also found associations between ambient air pollution (especially particulates and/or carbon monoxide) and postneonatal infant mortality (attributable to respiratory causes and possibly sudden infant death syndrome),^{48,49} low birth weight,^{50–53} and preterm birth.^{51,54–56}

The relative contribution of fine versus coarse particles to adverse health effects is being investigated. In studies of cities on the East Coast, fine particles seem to be important.⁵⁷ In other areas, coarse particles have a stronger or similar effect.⁵⁸ Several studies have found that fine particles from power plants and motor vehicles⁵⁹ or industrial sources⁶⁰ may be more closely associated with mortality.

Nitrogen Dioxide

Nitrogen dioxide is a gaseous pollutant produced by high-temperature combustion. The main outdoor sources of nitrogen dioxide include diesel and gasoline-powered engines and power plants. Levels of nitrogen dioxide around urban monitors have decreased over the past 20 years. Currently, all areas of the country meet the national air quality standard for nitrogen dioxide of 0.053 ppm (100 μg/m³), measured as an annual arithmetic mean. However, national emissions (overall production) of nitrogen oxides have actually increased in the past 20 years because of an increase in nitrogen oxide emissions from diesel vehicles.¹ This increase is of concern, because nitrogen oxide emissions contribute to ground-level ozone (smog) and other environmental problems such as acid rain.¹

Controlled-exposure studies of people with asthma have found that short-term exposures (30 minutes) to nitrogen dioxide at concentrations as low as 0.26 ppm can enhance the allergic response after subsequent challenge with allergens.^{61,62} These findings are of concern, because some urban communities that are in compliance with the federal standards for nitrogen dioxide (annual average) may experience substantial short-term peak concentrations (1-hour average) that exceed 0.25 ppm. Confirmation of these studies is needed.

Epidemiologic studies have reported relationships between increased ambient nitrogen dioxide and risks of respiratory tract symptoms^{63,64} and asthma exacerbations.⁶⁵ As noted previously, children with asthma living in communities with increased levels of air pollution (especially nitrogen dioxide, acid vapor, and particulates) were more likely to have bronchitis symptoms.⁴⁷ The same mix of air pollutants was also associated with deficits in lung growth (as measured by lung function tests).¹⁹ These effects were increased in children who spent more time outdoors.

The epidemiologic studies of health effects associated with nitrogen dioxide should be interpreted with caution. Increased levels of ambient nitrogen dioxide may be a marker for exposure to traffic emissions or other combustion-related pollution. An independent role of nitrogen dioxide cannot be clearly established because of the high covariation between ambient nitrogen dioxide and other pollutants. Nonetheless, these studies illustrate that adverse respiratory tract effects are seen in urban areas where traffic is a dominant source of air pollution.

Traffic-Related Pollution

Motor vehicles pollute the air through tailpipe exhaust emissions and fuel evaporation, contributing to carbon monoxide, PM_{2.5}, nitrogen oxides, hydrocarbons, other hazardous air pollutants (HAPs), and ozone formation. Motor vehicles represent the principal source of air pollution in many communities, and concentrations of traffic pollutants are greater near major roads.⁶⁶ Recently, investigators (primarily in Europe and Japan) have found increased adverse health effects among those living near busy roads.

Studies examining associations between adverse respiratory tract health and traffic have been reviewed.⁶⁷ Increased respiratory tract complications in children (eg, wheezing, chronic productive cough, and asthma hospitalizations) have been associated with residence near areas of high traffic density (particularly truck traffic).^{68–71} Other investigators have linked various childhood cancers to proximity to traffic.^{72–74}

Diesel exhaust, a major source of fine particulates in urban areas, is carcinogenic. Numerous studies have found an association between occupational exposure to diesel exhaust and lung cancer.⁷⁵ On the basis of extensive toxicologic and epidemiologic evidence, national and international health authorities, including the EPA and the International Agency for Research on Cancer, have concluded that there is considerable evidence of an association between exposure to diesel exhaust and an increased risk of lung cancer.^{76,77} Additionally, fine particles in diesel exhaust may enhance allergic and inflammatory responses to antigen challenge and may facilitate development of new allergies.^{78,79} Thus, diesel exhaust exposure may worsen symptoms in those with allergic rhinitis or asthma.

School buses operate in proximity to children, and most of the nation's school bus fleets run on diesel fuel. The EPA and some state agencies are establishing programs to eliminate unnecessary school bus idling and to promote use of cleaner buses to decrease children's exposures to diesel exhaust and the amount of air pollution created by diesel school buses (www.epa.gov/cleanschoolbus). A recent pilot study found that a child riding inside a school bus may be exposed to as much as 4 times the level of diesel exhaust as someone riding in a car.⁸⁰ These findings underscore the importance of advocating for school districts to replace diesel buses or retrofit them with pollution-reducing devices and limit school bus idling where children congregate as soon as possible.

Other Air Pollutants

Airborne levels of lead, sulfur dioxide, and carbon monoxide have decreased dramatically because of the implementation of control measures. However, levels of these pollutants may still be high near major sources. For example, high lead levels may be found near metals-processing industries, high sulfur dioxide levels may occur near large industrial facilities (especially coal-fired power plants), and high levels of carbon monoxide may occur in areas with heavy traffic congestion.¹

In addition to criteria air pollutants, there are numerous other air pollutants produced by motor vehicles, industrial facilities, residential wood combustion, agricultural burning, and other sources that are hazardous to children. More than 50000 chemicals are used commercially, and many are released into the air. For most of these chemicals, data on toxicity are sparse.⁸¹ Some pollutants remain airborne or react in the atmosphere to produce other harmful substances. Other air pollutants deposit into and contaminate land and water. Some toxic air pollutants

such as lead, mercury, and dioxins degrade slowly or not at all. These pollutants may bioaccumulate in animals at the top of the food chain, including humans. Children can be exposed to toxic air pollutants through contaminated air, water, soil, and food.³ One example of a persistent pollutant emitted into ambient air that leads to exposure through another route is mercury, a developmental neurotoxicant.⁸² Industrial emissions, especially from coal-fired power plants, are the leading source of environmental mercury. Although the levels of airborne mercury may not be hazardous, mercury deposits into soil and surface waters and ultimately accumulates in fish.⁸²

The HAPs, often referred to as "toxic air contaminants" or "air toxics," refer to 188 pollutants and chemical groups known or suspected to cause serious health effects including cancer, birth defects, and respiratory tract and neurologic illness.^{3,83} The Clean Air Act directs the EPA to regulate HAPs, which include compounds such as polycyclic aromatic hydrocarbons, acrolein, and benzene from fuel or fuel combustion; solvents such as hexane and toluene; hexavalent chromium from chrome-plating facilities; perchloroethylene from dry-cleaning plants; asbestos; metals (eg, mercury and cadmium); and persistent organic pollutants such as polychlorinated biphenyls. In 2001, diesel exhaust was listed as a mobile-source HAP. Many of these compounds are included in a priority list of 33 HAPs that are of special concern because of their widespread use and potential carcinogenicity and teratogenicity.⁸¹ The priority list and general sources of these compounds are available on the EPA Web site (www.epa.gov/ttn/atw/nata).

Limited monitoring data suggest that concentrations of some HAPs may exceed the goals of the Clean Air Act in many cities.⁸⁴ Mobile sources (on- and off-road vehicles) account for approximately half of the emissions³ but may contribute to 90% of the cancer risk (www.scorecard.org/env-releases/hap/us.tcl). A number of studies assessing health risks have found that estimated levels of some of the HAPs are a potential public health problem in many parts of the United States.^{3,84–86} For example, estimated concentrations of benzene, formaldehyde, and 1,3-butadiene may contribute to extra cases of cancer (at least 1 extra case per million population exposed) in more than 90% of the census tracts in the contiguous United States. Additionally, the most recent national cancer-risk assessment for HAPs (1996 data) did not include diesel exhaust in the risk estimates.³ The health risks may also be underestimated, because there is limited information on toxicity values for many of the HAPs,⁸⁷ and the risk models did not consider the potential for increased risk in children. These findings underscore the need for better ways to decrease toxic air emissions and assess exposures and risks.

Air-pollution episodes created by disasters (eg, accidents, volcanoes, forest fires, and acts of terrorism) can also create hazards for children. A discussion of these events and of bioaerosols in ambient air (eg, fungal spores and pollen) is beyond the scope of this

policy statement. Additionally, this statement does not address the hazards of indoor air pollution.

PREVENTION

Public health interventions to improve air quality can improve health at the population level. A decrease in levels of air pollution in former East Germany after reunification was associated with a decrease in parent-reported bronchitis⁸⁸ and improved lung function.⁸⁹ During the 1996 Summer Olympics in Atlanta, Georgia, extensive programs were implemented to improve mass transportation and decrease anticipated downtown traffic congestion. These programs were successful and were associated with a prolonged decrease in ozone pollution and significantly lower rates of childhood asthma visits during this period.⁹⁰ Closure of a steel mill in Utah Valley and resultant reductions in particulate matter were associated with a twofold decrease in hospitalizations for asthma in preschool children.^{91,92} Finally, lung function improved in children who moved away from communities with high particulate air pollution, compared with those who remained or moved to communities with comparable particulate air pollution.⁹³ These studies provide support for continued efforts to decrease air pollution and improve health via decreases in motor vehicle traffic and industrial emissions. Dietary factors may play a role in modulating the effects of air pollution in children. A recent study in Mexico City, Mexico, found that children with asthma given antioxidant supplements were less affected by ozone compared with a control group that did not receive supplementation.⁹⁴ Additional studies are needed to explore this issue further.

Air Pollution and the Regulatory Process

The Clean Air Act of 1970 mandated the EPA to establish the National Ambient Air Quality Standards (Table 1). Standards were set for criteria air pollutants because they are common, widespread, and known to be harmful to public health and the environment.^{11,12,83,95} The standards are reviewed every 5 years and set to protect public health, including the health of "sensitive" populations such as people with asthma, children, and the elderly. These standards are set without considering the costs of attaining these levels.

The standards for ozone and particulate matter were revised in 1997 on the basis of numerous scientific studies showing that the previous standards were not adequate to ensure health protection. Legal challenges were made by the American Trucking Associations, the US Chamber of Commerce, and other state and local business groups. However, the Supreme Court ultimately supported the EPA and ordered implementation of the standards.² Establishing implementation plans will be a lengthy process that will require the coordinated efforts of the EPA, state and local governments, and industry and environmental organizations.

Population exposures to toxic air contaminants may be of substantial public health concern.^{84,86} In contrast to criteria pollutants, monitoring of toxic air

contaminants is more limited. Exposures are estimated on the basis of reported emissions and may underestimate actual exposures.⁸⁷ The EPA is mandated to develop regulations through a lengthy process that first sets standards to control emissions on the basis of best-available technology. After maximum available control technology emission standards are established, the EPA must assess the risk remaining after emission decreases for the source take effect (residual risk).

To date, the EPA has focused primarily on establishing technology-based emission standards,³ and this has been a slow process for some sources (eg, mobile toxic air contaminants and mercury emissions). Nationwide, emissions of toxic air contaminants have dropped approximately 24% from baseline (1990–1993) because of regulation and voluntary decreases by industry. With the current plans for gradual fleet turnover and implementation of controls for motor vehicles and fuels, the EPA projects that toxic air-contaminant emissions from gasoline-powered and diesel mobile sources will not be decreased to 75% and 90% of baseline (1990–1993) levels, respectively, until the year 2020.³ However, major decreases could be more rapidly achieved simply from a prompt, wider application of existing technology.

Protecting populations from exposure to the harmful effects of air pollutants will require effective control measures. Industry (eg, coal-burning power plants, refineries, and chemical plants) and motor vehicles (both gasoline- and diesel-powered) are major sources of criteria pollutants and HAPs.^{11,12} For example, coal-fired power plants are important sources of nitrogen oxides (precursors of ozone), particulates, and sulfur dioxide and are the largest sources of mercury emission in the United States. Smaller sources such as dry cleaners, auto body shops, and wood-burning fireplaces can also affect air quality locally. Municipal and hospital waste incinerators release toxic air pollutants including mercury, lead, cadmium, and dioxin emissions. Depending on weather conditions and individual physicochemical properties, some pollutants can be carried by air currents to areas many miles from the source.

In numerous cities in the United States, the personal automobile is the single greatest polluter, because emissions from millions of vehicles on the road add up. Despite significant technologic advances that have led to tighter pollution control from vehicles, emissions vary substantially between vehicles, particularly between classes of vehicles, because of differences in fuel-economy standards set by regulatory agencies. For instance, the corporate average fuel-economy standards have less stringent fuel-economy requirements (average: 20.7 miles per gallon) for light-duty trucks, sport utility vehicles, and minivans, compared with passenger cars (average: 27.5 miles per gallon). The former group of vehicles tends to have higher emissions of air pollutants, higher fuel consumption, and higher emissions of greenhouse gases.^{96,97} Information on emissions and fuel-economy ratings for recent models and a

guide for choosing clean, fuel-efficient vehicles are available from the EPA Web site (www.epa.gov/greenvehicles/index.htm). The high levels of particulate emissions from diesel-powered buses and trucks must also be addressed. More than 70% of fine particle emissions from traffic are attributable to diesel-powered buses and trucks.

Driving a private car is probably a typical citizen's most "polluting" daily activity, yet in many cases, individuals have few alternative forms of transportation. Thus, urban planning and smart growth are imperative. Urban sprawl affects land use, transportation, and social and economic development and ultimately has important implications for public health.⁹⁸ Ways in which individuals can help to decrease air pollution are available at www.epa.gov/air/actions and www.arb.ca.gov/html/brochure/50things.htm.

Air Quality Index

The air quality index (AQI) provides local information on air quality and potential health concerns at the observed (or forecasted) levels of air pollution and can be a useful tool for educating families about local air quality and health.⁹⁹ The AQI is reported daily in metropolitan areas, often as part of local weather forecasts on television or radio or in newspapers. The AQI divides air-pollution levels into 6 categories of risk for 5 common pollutants (ozone, PM₁₀, nitrogen dioxide, carbon monoxide, and sulfur dioxide). Each category has a descriptive name reflecting levels of health concern (ranging from good through very hazardous), an associated color, and an advisory statement. Information about air quality in a specific area can be obtained from www.epa.gov/air/urbanair/index.html, www.scorecard.org, or www.weather.com. Although many states and local air districts actively forecast and disseminate health warnings, the challenge is to have people take actions to protect themselves and decrease activities that cause air pollution.

*Pediatric Environmental Health*¹⁰⁰ from the AAP provides additional information about the outdoor air pollutants and the use of the AQI.

CONCLUSIONS

Ambient air pollution has important and diverse health effects, and infants and children are among the most susceptible. Currently, levels of ozone and particulates remain unhealthy in many parts of the United States, and the current National Ambient Air Quality Standards may not protect the public adequately. There is a compelling need to move forward on efforts to ensure clean air for all.

The assurance of healthy air for children to breathe is beyond the control of an individual pediatrician, and there are no easy solutions. State chapters of the AAP, as well as individual members, can play an important role as advocates for children's environmental health. Areas of involvement might include working with community coalitions in support of strong pollution-control measures and informing local and national representatives and policy makers about the harmful effects of the environment on chil-

dren's health. Advocates for children's health are needed in discussions about land use and transportation issues. Pediatricians can also advocate for energy-saving (and pollution-minimizing) lifestyles to their patients' families, especially regarding vehicles driven.

In communities with poor air quality, pediatricians can play a role in educating children with asthma or other chronic respiratory tract disease and their families about the harmful effects of air pollution. Patients and families can be counseled on following the AQI to determine when local air-pollution levels pose a health concern. Ozone levels tend to be highest in the afternoon, and it may be possible to decrease children's exposure by scheduling strenuous outdoor activity earlier in the day.

As pediatricians become better informed about local air quality issues in their communities (eg, ozone, nearby industrial facilities, traffic, diesel buses, wood burning, etc), these local concerns can provide a starting point for discussion and education.

Pediatricians who serve as physicians for schools or for team sports should be aware of the health implications of pollution alerts to provide appropriate guidance to school and sports officials, particularly in communities with high levels of ozone.

RECOMMENDATIONS

1. The National Ambient Air Quality Standards are designed to protect the public. To achieve this, the following points should be addressed:
 - The revised standards for ozone and particulate matter adopted by the EPA in 1997 should be promptly implemented.
 - During implementation, the standards should not be weakened in any way that decreases the protection of children's health.
 - Because recent studies suggest that current standards for PM₁₀, PM_{2.5}, ozone, and nitrogen dioxide may not be protecting children, the standards should be promptly reviewed and revised.
 - Because the law requires that the most vulnerable groups be protected when setting or revising the air quality standards, the potential effects of air pollution on the fetus, infant, and child should be evaluated, and all standards should include a margin of safety for protection of children.
2. The current measures to protect children from exposures to HAPs are not effective and should be critically reevaluated. The EPA should focus on prompt implementation of the Clean Air Act Amendments of 1990 (Pub L No. 101-549) to decrease HAPs. Additional monitoring for HAPs should be undertaken to allow more accurate characterization of children's exposures to these compounds. Risk assessments for HAPs should be reviewed to ensure that goals are protective of children. Control measures that specifically protect children's health should be implemented.
3. States and local air districts with air quality concerns should actively implement forecasting and

dissemination of health warnings in ways that help people take actions to protect themselves and decrease activities that cause air pollution.

- Children's exposure to diesel exhaust particles should be decreased. Idling of diesel vehicles in places where children live and congregate should be minimized. Ongoing programs to fund conversion of diesel school bus fleets to cleaner alternative fuels and technologies should be pursued.
- Industrial emissions of mercury should be decreased.
- Federal and state governments' policies should encourage reductions in mobile and stationary sources of air pollution, including increased support for mass transit, carpooling, retiring or retrofitting old power plants that do not meet current pollution-control standards, and programs that support marked improvements in fuel emissions of gasoline- and diesel-powered vehicles. Additionally, the development of alternative fuel fleets, low-sulfur diesel, and other "low-emission" strategies (eg, retrofit of existing diesel engines) should be promoted. Before promoting new alternative fuels, these alternative fuel sources should be critically evaluated and determined by governmental authorities to have a good safety profile.
- The same overall fuel-economy standard should apply to all passenger vehicles. Programs that allow certain passenger vehicles to be exempt from the usual fuel-economy standards should be abolished.
- City and land-use planning should encourage the design and redevelopment of communities to promote mass transit, carpooling, pedestrian walkways, and bicycle use.
- Siting of school and child care facilities should include consideration of proximity to roads with heavy traffic and other sources of air pollution. New schools should be located to avoid "hot spots" of localized pollution.

COMMITTEE ON ENVIRONMENTAL HEALTH, 2003–2004

Michael W. Shannon, MD, MPH, Chairperson
Dana Best, MD, MPH
Helen J. Binns, MD, MPH
Christine L. Johnson, MD
*Janice J. Kim, MD, PhD, MPH
Lynnette J. Mazur, MD, MPH
David W. Reynolds, MD
James R. Roberts, MD, MPH
William B. Weil, Jr, MD

Sophie J. Balk, MD
Past Committee Chairperson
Mark Miller, MD, MPH
Past Committee Member
Katherine M. Shea, MD, MPH
Past Committee Member

CONSULTANT

Michael Lipsett, MD
California Department of Health Services

LIAISONS

Robert H. Johnson, MD
Centers for Disease Control and Prevention/Agency
for Toxic Substances and Disease Registry

Martha Linet, MD
National Cancer Institute
Walter Rogan, MD
National Institute of Environmental Health Sciences

STAFF
Paul Spire

*Lead author

REFERENCES

- US Environmental Protection Agency. Latest findings on national air quality: 2000 status and trends. Research Triangle Park, NC: Environmental Protection Agency; 2001. Publication No. EPA 454/K-01-002. Available at: www.epa.gov/airtrends/reports.html. Accessed August 8, 2003
- US Environmental Protection Agency. Supreme Court upholds EPA position on smog, particulate rules [press release]. Available at: www.epa.gov/airlinks/rehear.htm. Accessed October 29, 2004
- US Environmental Protection Agency. About air toxics, health and ecologic effects. Available at: www.epa.gov/air/toxicair/newtoxics.html. Accessed August 8, 2003
- American Academy of Pediatrics, Committee on Environmental Health. Ambient air pollution: respiratory hazards to children. *Pediatrics*. 1993;91:1210–1213
- Dieter RR, Etzel RA, Chen D, et al. Workshop to identify critical windows of exposure for children's health: immune and respiratory systems work group summary. *Environ Health Perspect*. 2000;108(suppl 3):483–490
- Plopper CG, Fanucchi MV. Do urban environmental pollutants exacerbate childhood lung diseases? *Environ Health Perspect*. 2000;108:A252–A253
- Pinkerton KE, Joad JP. The mammalian respiratory system and critical windows of exposure for children's health. *Environ Health Perspect*. 2000;108(suppl 3):457–462
- Plunkett LM, Turnbull D, Rodricks JV. Differences between adults and children affecting exposure assessment. In: Guzelian PS, Henry CJ, Olin SS, eds. *Similarities and Differences Between Children and Adults: Implications for Risk Assessment*. Washington, DC: ILSI Press; 1992: 79–96
- Wiley JA, Robinson JP, Piazza T, et al. *Activity Patterns of California Residents: Final Report*. Sacramento, CA: California Air Resources Board; 1991. Publication No. A6-177-33
- Wiley JA, Robinson JP, Cheng YT, Piazza T, Stork L, Pladsen K. *Study of Children's Activity Patterns: Final Report*. Sacramento, CA: California Air Resources Board; 1991. Publication No. A733-149
- American Thoracic Society, Committee of the Environmental and Occupational Health Assembly. Health effects of outdoor air pollution. Part 1. *Am J Respir Crit Care Med*. 1996;153:3–50
- American Thoracic Society, Committee of the Environmental and Occupational Health Assembly. Health effects of outdoor air pollution. Part 2. *Am J Respir Crit Care Med*. 1996;153:477–498
- Bates DV. The effects of air pollution on children. *Environ Health Perspect*. 1995;103(suppl 6):49–53
- US Environmental Protection Agency. *Air Quality Criteria for Particulate Matter, Vol. II*. Research Triangle Park, NC: Environmental Protection Agency; 2001. Publication No. EPA/600/P-99/002bB
- US Environmental Protection Agency. *Air Quality Criteria for Ozone and Related Photochemical Oxidants, Vol. III*. Research Triangle Park, NC: Environmental Protection Agency; 1996. Publication No. EPA/600/P-93/004a-cF
- Dockery DW. Epidemiologic evidence of cardiovascular effects of particulate air pollution. *Environ Health Perspect*. 2001;109(suppl 4): 483–486
- Pope CA III, Burnett RT, Thun MJ, et al. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *JAMA*. 2002;287:1132–114118.
- McConnell R, Berhane K, Gilliland F, et al. Asthma in exercising children exposed to ozone: a cohort study [published correction appears in: *Lancet*. 2002;359:896]. *Lancet*. 2002;359:386–391
- Gauderman WJ, McConnell R, Gilliland F, et al. Association between air pollution and lung function growth in southern California children. *Am J Respir Crit Care Med*. 2000;162:1383–1390
- Gauderman WJ, Gilliland GF, Vora H, et al. Association between air pollution and lung function growth in southern California children: results from a second cohort. *Am J Respir Crit Care Med*. 2002;166:76–84

21. Kinney PL, Thurston GD, Raizenne M. The effects of ambient ozone on lung function in children: a reanalysis of six summer camp studies. *Environ Health Perspect.* 1996;104:170–174
22. Thurston GD, Lippmann M, Scott MB, Fine JM. Summertime haze air pollution and children with asthma. *Am J Respir Crit Care Med.* 1997; 155:654–660
23. Ostro BD, Lipsett MJ, Mann JK, Braxton-Owens H, White MC. Air pollution and asthma exacerbations among African-American children in Los Angeles. *Inhal Toxicol.* 1995;7:711–722
24. Thurston GD, Ito K, Hayes CG, Bates DV, Lippmann M. Respiratory hospital admissions and summertime haze air pollution in Toronto, Ontario: consideration of the role of acid aerosols. *Environ Res.* 1994; 65:271–290
25. White MC, Etzel RA, Wilcox WD, Lloyd C. Exacerbations of childhood asthma and ozone pollution in Atlanta. *Environ Res.* 1994;65:56–68
26. Tolbert PE, Mulholland JA, MacIntosh DL, et al. Air quality and pediatric emergency room visits for asthma in Atlanta, Georgia, USA. *Am J Epidemiol.* 2000;151:798–810
27. Gilliland FD, Berhane K, Rappaport EB, et al. The effects of ambient air pollution on school absenteeism due to respiratory illnesses. *Epidemiology.* 2001;12:43–54
28. Molfino NA, Wright SC, Katz I, et al. Effect of low concentration of ozone on inhaled allergen responses in asthmatic subjects. *Lancet.* 1991;338:199–203
29. Castillejos M, Gold DR, Damokosh AI, et al. Acute effects of ozone on the pulmonary function of exercising schoolchildren from Mexico City. *Am J Respir Crit Care Med.* 1995;152:1501–1507
30. Chen PC, Lai YM, Chan CC, Hwang JS, Yang CY, Wang JD. Short-term effect of ozone on the pulmonary function of children in primary school. *Environ Health Perspect.* 1999;107:921–925
31. Korrick SA, Neas LM, Dockery DW, et al. Effects of ozone and other pollutants on the pulmonary function of adult hikers. *Environ Health Perspect.* 1998;106:93–99
32. Burnett RT, Smith-Doiron M, Stieb D, et al. Association between ozone and hospitalization for acute respiratory diseases in children less than 2 years of age. *Am J Epidemiol.* 2001;153:444–452
33. Stieb DM, Burnett RT, Beveridge RC, Brook JR. Association between ozone and asthma emergency department visits in Saint John, New Brunswick, Canada. *Environ Health Perspect.* 1996;104:1354–1360
34. Gent JF, Tiche EW, Holford TR, et al. Association of low-level ozone and fine particles with respiratory symptoms in children with asthma. *JAMA.* 2003;290:1859–1867
35. Kunzli N, Lurmann F, Segal M, Ngo L, Balmes J, Tager IB. Association between lifetime ambient ozone exposure and pulmonary function in college freshmen—results of a pilot study. *Environ Res.* 1997;72:8–23
36. Galizia A, Kinney PL. Long-term residence in areas of high ozone: associations with respiratory health in a nationwide sample of non-smoking young adults. *Environ Health Perspect.* 1999;107:675–679
37. Ghio AJ, Silbajoris R, Carson JL, Samet JM. Biologic effects of oil fly ash. *Environ Health Perspect.* 2002;110(suppl 1):89–94
38. Pandya RJ, Solomon G, Kinner A, Balmes JR. Diesel exhaust and asthma: hypotheses and molecular mechanisms of action. *Environ Health Perspect.* 2002;110(suppl 1):103–112
39. Dockery DW, Pope CA III. Acute respiratory effects of particulate air pollution. *Annu Rev Public Health.* 1994;15:107–132
40. Schwartz J. Air pollution and daily mortality: a review and meta analysis. *Environ Res.* 1994;64:36–52
41. Samet JM, Dominici F, Currier FC, Coursac I, Zeger SL. Fine particulate air pollution and mortality in 20 U.S. cities, 1987–1994. *N Engl J Med.* 2000;343:1742–1749
42. Schwartz J. Air pollution and blood markers of cardiovascular risk. *Environ Health Perspect.* 2001;109(suppl 3):405–409
43. California Air Resources Board. June 20, 2002 board meeting summary. Sacramento, CA: California Air Resources Board; 2002. Available at: www.arb.ca.gov/research/aaqs/std-rs/bdsum620/bdsum620.htm. Accessed August 8, 2003
44. Hoek G, Dockery DW, Pope A, Neas L, Roemer W, Brunekreef B. Association between PM10 and decrements in peak expiratory flow rates in children: reanalysis of data from five panel studies. *Eur Respir J.* 1998;11:1307–1311
45. Ostro B, Lipsett M, Mann J, Braxton-Owens H, White M. Air pollution and exacerbation of asthma in African-American children in Los Angeles. *Epidemiology.* 2001;12:200–208
46. Yu O, Sheppard L, Lumley T, Koenig JQ, Shapiro GG. Effects of ambient air pollution on symptoms of asthma in Seattle-area children enrolled in the CAMP study. *Environ Health Perspect.* 2000;108: 1209–1214
47. McConnell R, Berhane K, Gilliland F, et al. Air pollution and bronchitic symptoms in Southern California children with asthma. *Environ Health Perspect.* 1999;107:757–760
48. Woodruff TJ, Grillo J, Schoendorf KC. The relationship between selected causes of postneonatal infant mortality and particulate air pollution in the United States. *Environ Health Perspect.* 1997;105:608–612
49. Bobak M, Leon DA. The effect of air pollution on infant mortality appears specific for respiratory causes in the postneonatal period. *Epidemiology.* 1999;10:666–670
50. Ritz B, Yu F. The effect of ambient carbon monoxide on low birth weight among children born in southern California between 1989 and 1993. *Environ Health Perspect.* 1999;107:17–25
51. Bobak M. Outdoor air pollution, low birth weight, and prematurity. *Environ Health Perspect.* 2000;108:173–176
52. Dejmek J, Solansky I, Benes I, Lenicek J, Sram RJ. The impact of polycyclic aromatic hydrocarbons and fine particles on pregnancy outcome. *Environ Health Perspect.* 2000;108:1159–1164
53. Wang X, Ding H, Ryan L, Xu X. Association between air pollution and low birth weight: a community-based study. *Environ Health Perspect.* 1997;105:514–520
54. Ritz B, Yu F, Chapa G, Fruin S. Effect of air pollution on preterm birth among children born in Southern California between 1989 and 1993. *Epidemiology.* 2000;11:502–511
55. Ha EH, Hong YC, Lee BE, Woo BH, Schwartz J, Christiani DC. Is air pollution a risk factor for low birth weight in Seoul? *Epidemiology.* 2001;12:643–648
56. Xu X, Ding H, Wang X. Acute effects of total suspended particles and sulfur dioxides on preterm delivery: a community-based cohort study. *Arch Environ Health.* 1995;50:407–415
57. Schwartz J. Air pollution and hospital admissions for respiratory disease. *Epidemiology.* 1996;7:20–28
58. Ostro BD, Broadwin R, Lipsett MJ. Coarse and fine particles and daily mortality in the Coachella Valley, California: a follow-up study. *J Expo Anal Environ Epidemiol.* 2000;10:412–419
59. Laden F, Neas LM, Dockery DW, Schwartz J. Association of fine particulate matter from different sources with daily mortality in six US cities. *Environ Health Perspect.* 2000;108:941–947
60. Ozkaynak H, Thurston GD. Associations between 1980 U.S. mortality rates and alternative measures of airborne particle concentration. *Risk Anal.* 1987;7:449–461
61. Strand V, Svartengren M, Rak S, Barck C, Bylin G. Repeated exposure to an ambient level of NO₂ enhances asthmatic response to a nonsymptomatic allergen dose. *Eur Respir J.* 1998;12:6–12
62. Tunnicliffe WS, Burge PS, Ayres JG. Effect of domestic concentrations of nitrogen dioxide on airway responses to inhaled allergen in asthmatic patients. *Lancet.* 1994;344:1733–1736
63. Hajat S, Haines A, Goubet SA, Atkinson RW, Anderson HR. Association of air pollution with daily GP consultations for asthma and other lower respiratory conditions in London. *Thorax.* 1999;54:597–605
64. Shima M, Adachi M. Effect of outdoor and indoor nitrogen dioxide on respiratory symptoms in schoolchildren. *Int J Epidemiol.* 2000;29: 862–870
65. Lipsett M, Hurley S, Ostro B. Air pollution and emergency room visits for asthma in Santa Clara County, California. *Environ Health Perspect.* 1997;105:216–222
66. Zhu Y, Hinds WC, Kim S, Sioutas C. Concentration and size distribution of ultrafine particles near a major highway. *J Air Waste Manag Assoc.* 2002;52:1032–1042
67. Delfino RJ. Epidemiologic evidence for asthma and exposure to air toxics: linkages between occupational, indoor, and community air pollution research. *Environ Health Perspect.* 2002;110(suppl 4):573–589
68. Edwards J, Walters S, Griffiths RK. Hospital admissions for asthma in preschool children: relationship to major roads in Birmingham, United Kingdom. *Arch Environ Health.* 1994;49:223–227
69. van Vliet P, Knappe M, de Hartog J, Janssen N, Harssema H, Brunekreef B. Motor vehicle exhaust and chronic respiratory symptoms in children living near freeways. *Environ Res.* 1997;74:122–132
70. Brunekreef B, Janssen NA, de Hartog J, Harssema H, Knappe M, van Vliet P. Air pollution from truck traffic and lung function in children living near motorways. *Epidemiology.* 1997;8:298–303
71. Ciccone G, Forastiere F, Agabiti N, et al. Road traffic and adverse respiratory effects in children. SIDRIA Collaborative Group. *Occup Environ Med.* 1998;55:771–778
72. Feychting M, Svensson D, Ahlbom A. Exposure to motor vehicle exhaust and childhood cancer. *Scand J Work Environ Health.* 1998;24: 8–11

73. Pearson RL, Wachtel H, Ebi KL. Distance-weighted traffic density in proximity to a home is a risk factor for leukemia and other childhood cancers. *J Air Waste Manag Assoc.* 2000;50:175-180
74. Raaschou-Nielsen O, Hertel O, Thomsen BL, Olsen JH. Air pollution from traffic at the residence of children with cancer. *Am J Epidemiol.* 2001;153:433-443
75. Lipsett M, Campleman S. Occupational exposure to diesel exhaust and lung cancer: a meta-analysis. *Am J Public Health.* 1999;89:1009-1017
76. US Environmental Protection Agency. *Health Assessment Document for Diesel Engine Exhaust.* Washington, DC: Office of Research and Development NCFEA; 2002. EPA/600/8-909/057F
77. International Agency for Research on Cancer. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Diesel and Gasoline Engine Exhausts and Some Nitroarenes.* Vol 46. Lyon, France: International Agency for Research on Cancer; 1989:458
78. Diaz-Sanchez D, Garcia MP, Wang M, Jyrala M, Saxon A. Nasal challenge with diesel exhaust particles can induce sensitization to a neoallergen in the human mucosa. *J Allergy Clin Immunol.* 1999;104:1183-1188
79. Nel AE, Diaz-Sanchez D, Ng D, Hiura T, Saxon A. Enhancement of allergic inflammation by the interaction between diesel exhaust particles and the immune system. *J Allergy Clin Immunol.* 1998;102:539-554
80. Solomon GM, Campbell T, Feuer GR, Masters J, Samkian A, Paul KA. *No Breathing in the Aisles: Diesel Exhaust Inside School Buses.* New York, NY: Natural Resources Defense Council; 2001. Available at: www.nrdc.org/air/transportation/schoolbus/sbusinx.asp. Accessed August 8, 2003
81. Leikauf GD. Hazardous air pollutants and asthma. *Environ Health Perspect.* 2002;110(suppl 4):505-526
82. American Academy of Pediatrics, Goldman LR, Shannon MW, Committee on Environmental Health. Technical report: mercury in the environment: implications for pediatricians. *Pediatrics.* 2001;108:197-205
83. Suh HH, Bahadori T, Vallarino J, Spengler JD. Criteria air pollutants and toxic air pollutants. *Environ Health Perspect.* 2000;108(suppl 4):625-633
84. Woodruff TJ, Axelrad DA, Caldwell J, Morello-Frosch R, Rosenbaum A. Public health implications of 1990 air toxics concentrations across the United States. *Environ Health Perspect.* 1998;106:245-251
85. Nazemi MA. *Multiple Air Toxics Exposure Study (MATES-II) in the South Coast Air Basin.* Diamond Bar, CA: South Coast Air Quality Management District; 2000
86. Morello-Frosch RA, Woodruff TJ, Axelrad DA, Caldwell JC. Air toxics and health risks in California: the public health implications of outdoor concentrations. *Risk Anal.* 2000;20:273-291
87. Kyle AD, Wright CC, Caldwell JC, Buffler PA, Woodruff TJ. Evaluating the health significance of hazardous air pollutants using monitoring data. *Public Health Rep.* 2001;116:32-44
88. Heinrich J, Hoelscher B, Wichmann HE. Decline of ambient air pollution and respiratory symptoms in children. *Am J Respir Crit Care Med.* 2000;161:1930-1936
89. Frye C, Hoelscher B, Cyrus J, Wjst M, Wichmann HE, Heinrich J. Association of lung function with declining ambient air pollution. *Environ Health Perspect.* 2003;111:383-387
90. Friedman MS, Powell KE, Hutwagner L, Graham LM, Teague WG. Impact of changes in transportation and commuting behaviors during the 1996 Summer Olympic Games in Atlanta on air quality and childhood asthma. *JAMA.* 2001;285:897-905
91. Pope CA III. Respiratory hospital admissions associated with PM10 pollution in Utah, Salt Lake, and Cache Valleys. *Arch Environ Health.* 1991;46:90-97
92. Pope CA III. Particulate pollution and health: a review of the Utah valley experience. *J Expo Anal Environ Epidemiol.* 1996;6:23-34
93. Avol EL, Gauderman WJ, Tan SM, London SJ, Peters JM. Respiratory effects of relocating to areas of differing air pollution levels. *Am J Respir Crit Care Med.* 2001;164:2067-2072
94. Romieu I, Sienna-Monge JJ, Ramirez-Aguilar M, et al. Antioxidant supplementation and lung functions among children with asthma exposed to high levels of air pollutants. *Am J Respir Crit Care Med.* 2002;166:703-709
95. US Environmental Protection Agency. *The Plain English Guide To The Clean Air Act.* 1993. EPA-400-K-93-001. Available at: www.epa.gov/oar/oaqps/peg_caa/pegcaain.html. Accessed October 26, 2004
96. National Research Council. *Effectiveness and Impact of Corporate Average Fuel Economy (CAFE) Standards.* Washington, DC: National Academies Press; 2002. Available at: www.nap.edu/books/0309076013/html. Accessed August 8, 2003
97. Hwang R, Millis B, Spencer T. *Clean Getaway: Toward Safe and Efficient Vehicles.* New York, NY: Natural Resources Defense Council; 2001. Available at: www.nrdc.org/air/transportation/cape/cafexinx.asp. Accessed August 8, 2003
98. Jackson RJ, Kochtitzky C. *Creating a Health Environment: The Impact of the Built Environment on Public Health.* Washington, DC: Sprawl Watch Clearinghouse. Available at: www.sprawlwatch.org/health.pdf. Accessed August 8, 2003
99. US Environmental Protection Agency, Office of Air and Radiation. *Air Quality Index: A Guide to Air Quality and Your Health.* Research Triangle Park, NC: Environmental Protection Agency; 2000. Publication No. EPA-454/R-00-005. Available at: www.epa.gov/airnow/aqibroch. Accessed August 8, 2003
100. American Academy of Pediatrics, Committee on Environmental Health. Outdoor air pollutants. In: Etzel RA, Balk SJ, eds. *Pediatric Environmental Health.* 2nd ed. Elk Grove Village, IL: American Academy of Pediatrics; 2003;69-86

All policy statements from the American Academy of Pediatrics automatically expire 5 years after publication unless reaffirmed, revised, or retired at or before that time.

Ambient Air Pollution: Health Hazards to Children

Pediatrics 2004;114;1699

DOI: 10.1542/peds.2004-2166

Updated Information & Services

including high resolution figures, can be found at:
<http://pediatrics.aappublications.org/content/114/6/1699>

References

This article cites 81 articles, 6 of which you can access for free at:
<http://pediatrics.aappublications.org/content/114/6/1699#BIBL>

Subspecialty Collections

This article, along with others on similar topics, appears in the following collection(s):

Current Policy

http://www.aappublications.org/cgi/collection/current_policy

Council on Environmental Health

http://www.aappublications.org/cgi/collection/council_on_environmental_health

Environmental Health

http://www.aappublications.org/cgi/collection/environmental_health_sub

Permissions & Licensing

Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at:

<http://www.aappublications.org/site/misc/Permissions.xhtml>

Reprints

Information about ordering reprints can be found online:

<http://www.aappublications.org/site/misc/reprints.xhtml>

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN™



PEDIATRICS®

OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

Ambient Air Pollution: Health Hazards to Children

Pediatrics 2004;114;1699

DOI: 10.1542/peds.2004-2166

The online version of this article, along with updated information and services, is located on the World Wide Web at:

<http://pediatrics.aappublications.org/content/114/6/1699>

Pediatrics is the official journal of the American Academy of Pediatrics. A monthly publication, it has been published continuously since 1948. Pediatrics is owned, published, and trademarked by the American Academy of Pediatrics, 141 Northwest Point Boulevard, Elk Grove Village, Illinois, 60007. Copyright © 2004 by the American Academy of Pediatrics. All rights reserved. Print ISSN: 1073-0397.

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN™

