Ambient Air Pollution: Health Hazards to Children

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Abstract

Ambient air pollution is produced by sources including vehicular traffic, coal-fired power plants, hydraulic fracturing, agricultural production, and forest fires. It consists of primary pollutants generated by combustion and secondary pollutants formed in the atmosphere from precursor gases. Air pollution causes and exacerbates climate change, and climate change worsens health effects of air pollution. Infants and children are uniquely sensitive to air pollution, because their organs are developing and they have higher air per body weight intake. Health effects linked to air pollution include not only exacerbations of respiratory diseases but also reduced lung function development and increased asthma incidence. Additional outcomes of concern include preterm birth, low birth weight, neurodevelopmental disorders, IQ loss, pediatric cancers, and increased risks for adult chronic diseases. These effects are mediated by oxidative stress, chronic inflammation, endocrine disruption, and genetic and epigenetic mechanisms across the life span. Natural experiments demonstrate that with initiatives such as increased use of public transportation, both air quality and community health improve. Similarly, the Clean Air Act has improved air quality, although exposure inequities persist. Other effective strategies for reducing air pollution include ending reliance on coal, oil, and gas; regulating industrial emissions; reducing exposure with attention to proximity of residences, schools, and child care facilities to traffic; and a greater awareness of the Air Quality Index. This policy reviews both short- and long-term health consequences of ambient air pollution, especially in relation to developmental exposures. It examines individual, community, and legislative strategies to mitigate air pollution.

Introduction

Air pollution exposures are widespread, and children are uniquely vulnerable. Since publication of the 2004 American Academy of Pediatrics policy statement on ambient air pollutants,1 the evidence for child health impacts has expanded considerably. Current levels of air pollutants are associated with many of the most important pediatric morbidities, including asthma incidence and prevalence, adverse birth outcomes, preterm birth, low birth weight, neurodevelopmental disorders, IQ loss, pediatric cancers, and increased risks for adult chronic diseases. These effects are mediated by oxidative stress, chronic inflammation, endocrine disruption, and genetic and epigenetic mechanisms across the life span. Natural experiments demonstrate that with initiatives such as increased use of public transportation, both air quality and community health improve. Similarly, the Clean Air Act has improved air quality, although exposure inequities persist. Other effective strategies for reducing air pollution include ending reliance on coal, oil, and gas; regulating industrial emissions; reducing exposure with attention to proximity of residences, schools, and child care facilities to traffic; and a greater awareness of the Air Quality Index. This policy reviews both short- and long-term health consequences of ambient air pollution, especially in relation to developmental exposures. It examines individual, community, and legislative strategies to mitigate air pollution.


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behavioral and cognitive development, and pediatric cancers, as well as with increased risk for a range of chronic diseases in adult life.2–6

Common sources of air pollution include emissions from traffic, coal-fired power plants, poorly ventilated wood-burning stoves or boilers, and forest fires. Toxicants in these emissions can influence the morphologic and functional development of organ systems from the fetal period to early, middle, and late childhood.7 In addition, children receive higher doses because of their faster breathing rate and proportionately greater air intake per kilogram of body weight compared with adults.8

This policy statement focuses on short- and long-term health effects of key air pollutants that originate outdoors. These pollutants may enter buildings (and vehicles) through open doors and windows, ventilation systems, and cracks in structures, adding to the burden of indoor-derived air pollutants. Studies addressing regulated individual contaminants as well as mixtures or sources (traffic-related air pollution [TRAP], diesel particles, animal agricultural emissions, hydraulic fracting, wood burning) are included. Several other air contaminants are not included but have been addressed in other comprehensive reviews or other American Academy of Pediatrics policy statements (eg, tobacco smoke, mercury, asbestos, etc).9–12

EXPOSURE TO OUTDOOR AIR POLLUTANTS

The US Environmental Protection Agency (EPA) sets air quality standards for 6 criteria air pollutants (Table 1).13 These are periodically updated and include primary standards to protect sensitive populations, including children. The EPA also monitors outdoor concentrations of 187 air toxics known or suspected to cause cancer or other serious health effects, such as birth defects; these are referred to as hazardous air pollutants. Examples include benzene, formaldehyde, polycyclic aromatic hydrocarbons (PAHs), and metals, such as chromium.14

States (or local agencies delegated by the state) have the primary authority to ensure compliance. These state and local air agencies, as well as the local public health agencies, are knowledgeable and can provide information about local and regional concerns.

Regulatory monitoring of criteria air pollutants indicates overall improvement in ambient air concentrations (Fig 1).14 National mean levels of the 6 criteria air pollutants have fallen by 70% across the United States since passage of the Clean Air Act in 1970.15 However, in 2016, 62% of children resided in a county with exceedance of at least 1 standard.14 Despite these overall improvements, important socioeconomic and racial disparities in exposure continue to be observed in the trend data. Compared with non-Hispanic white individuals, Asian American or Pacific Islander, Black non-Hispanic, and Hispanic individuals were more likely to reside in counties unable to meet the air quality standards for particulate matter 2.5 μm or less in diameter (PM2.5) and ozone.14 Children living in poverty were more likely to reside in census tract areas above the benchmark for 1 in 10 000 cancer risk from hazardous air pollutants compared with those living in households at or above the poverty level.14 Irrespective of poverty status, a higher percentage of children who identify as Black or Asian American live in census tracts where noncancer health-based benchmarks are exceeded.14 Of note, the trend in levels of PM2.5 shows a slight increase in the United States since 2015, the first recorded deterioration in national air quality in nearly a half century.16

Community air quality may reflect mobile (eg, on- and off-road vehicles) and point sources, including industrial facilities (such as coal-fired power plants and electricity-generating plants), dry-cleaning shops, restaurants (especially those using wood-fire ovens), metal plating facilities, residential wood burning, barbeques, etc, as well as seasonal variability and meteorology. Combustion contributes many primary pollutants (carbon monoxide [CO], nitrogen oxide [NOx], particulate matter [PM], and volatile organic compounds [VOCs]). Combustion processes may also lead to secondary pollutants (ozone, nitrate, or sulfate aerosols) formed in the atmosphere from precursor gases.

TRAP is ubiquitous. These emissions include primary particles representing particulate matter 10

### TABLE 1 Six EPA Criteria Pollutants With National Ambient Air Quality Standards

<table>
<thead>
<tr>
<th>Pollutants</th>
<th>Standard</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO</td>
<td>9 ppm over 8 h, 35 ppm over 1 h</td>
</tr>
<tr>
<td>Lead</td>
<td>0.15 μg/m³ over 3 mo</td>
</tr>
<tr>
<td>NO₂</td>
<td>100 ppb over 1 h, 53 ppb annual average</td>
</tr>
<tr>
<td>PM, including PM₂.₅ and PM₁₀</td>
<td>PM₂.₅ 35 μg/m³ over 24 h; 12 μg/m³ annual average PM₁₀ 150 μg/m³ over 24 h</td>
</tr>
<tr>
<td>SO₂</td>
<td>75 ppb over 1 h</td>
</tr>
<tr>
<td>Ozone</td>
<td>0.070 ppm over 8 h</td>
</tr>
</tbody>
</table>

Permissible levels of the 6 criteria air pollutants (primary standards based on public health protection). These enforceable standards are reevaluated periodically by the EPA. Standards noted are based on information from the EPA National Ambient Air Quality Standards Table. (Available at: https://www.epa.gov/criteria-air-pollutants/naaqs-table. Accessed February 4, 2019.)
μm or less in diameter (PM₁₀), PM₂.₅, and ultrafine PM (<100 nm), which are formed during combustion of fuels and comprise insoluble carbonaceous material (referred to as elemental carbon or black carbon), along with other toxic components, such as metals and PAHs. Diesel particles contain mostly ultrafine PM enriched in black carbon and PAHs as well as VOCs, such as formaldehyde. Resuspended road dust, tire wear, and brake wear represent noncombustion sources of traffic-related PM. In addition, secondary particles may be formed in the atmosphere post emission and include PM in the nanoparticle size range.¹⁷

An emerging source of concern is fossil fuel extraction by hydraulic fracturing, or “fracking.” Water containing proprietary chemicals under high pressure is forced underground to crack deep shale layers and release trapped gas.¹⁸,¹⁹

The volume of natural gas derived from fracking doubled in the United States from 2000 to 2011 and continues to increase.¹⁹ The wellhead, controlled burns (flaring), water storage pits and tanks, sand operations, and diesel-powered equipment and trucks contribute VOCs, PAHs, and criteria air pollutants, such as NOₓ and PM₂.₅.²⁰,²¹

Biomass combustion is an important source of air pollutants, such as PM. Globally, cooking- and heating-related emissions from solid fuel use are significant (eg, wood, charcoal, dung, crop waste). Large health initiatives for alternative fuels and improved cook stoves are targeting high pollution observed in middle- and low-income countries.²² In some communities in North America, wood burning for heat and intentional burning of agricultural waste are important regional contributors to regional air quality.²³

In agricultural communities, significant emissions from industrial-scale animal operations, such as poultry, swine, dairy, or beef, may result from the decomposition of animal waste or application of waste in liquid form as fertilizer. This includes gases and odorous vapors, including ammonia, hydrogen sulfide, and PM, that may contain endotoxin and other bioaerosols.²⁴

Air pollution and climate change are closely linked, and the contribution of climate change to air quality is of concern. Climate change has increased the size and frequency of wildfires in North America, and ground-level ozone formation from precursor gases is enhanced in the presence of increasing temperatures.²⁵ Climate change influences the distribution, quantity, and quality of aeroallergens as well as ozone levels. Among those with allergic disease and asthma, global climate change is estimated to worsen disease, lead to more symptom days, and reduce quality of life.²⁶

Air pollution is experienced by populations as a mixture containing components with various toxicological properties. Several common pathophysiological pathways have been identified as important mechanisms of how air pollution may affect health, including oxidative stress, inflammation, endocrine disruption, genetics, and epigenetics (Fig 2). The effects of air pollution occur at each stage of the life course and may cross generations via epigenetic and genetic factors. The delineation of mechanisms is complicated by the fact that different

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**FIGURE 1**
Percentage of children aged 0 to 17 years living in counties with pollutant concentrations above the levels of the current air quality standards, 1999–2016. avg, average. (Reprinted with permission from US Environmental Protection Agency. ACE: environments and contaminants - criteria air pollutants. Available at: https://www.epa.gov/ace/ace-environments-and-contaminants-criteria-air-pollutants. Accessed February 4, 2019.)
pathways may trigger one another, such as oxidative stress causing inflammation or DNA damage.\textsuperscript{27,28} PM and PAHs have been associated with increased histone acetylation and DNA methylation, respectively.\textsuperscript{29,30} Supporting the role of environmental exposures on epigenetic modifications is the association of exposure to air pollution and increased methylation of the \textit{FOXP3} locus from T regulatory lymphocytes, changes known to increase asthma morbidity.\textsuperscript{30,31}

The subsequent morbidities arising from these pathways in childhood are reviewed in the following sections. The emphasis is on human epidemiological data, much of which are observational. Observational studies of air pollution and regulation of air pollutants are based on causal inferences from observational data with consideration of confounders and application of epidemiological criteria for causality (eg, dose response, biological plausibility, and consistency of evidence).

**BIRTH OUTCOMES**

Multiple studies, many systematic reviews, and a few pooled analyses are available and have supported associations between ambient air pollution exposure during pregnancy and adverse birth outcomes. In most studies, researchers have examined effects on fetal growth (as term low birth weight or small for gestational age) and preterm birth.\textsuperscript{2,32–36} In a pooled analysis comprising multiple studies and examination of multiple pollutants, birth weight decrements of approximately 10 to 30 g and odds ratios of 1.05 to 1.10 for low birth weight and of 1.04 to 1.06 for preterm birth were observed in relation to representative concentrations of higher CO, nitrogen dioxide (NO\textsubscript{2}), PM\textsubscript{10}, and PM\textsubscript{2.5}.\textsuperscript{2}

For context, van der Zee et al\textsuperscript{37} sought to translate ambient air pollution effect sizes to a more familiar air contaminant sharing many constituents, environmental tobacco smoke. The risk of low birth weight associated with a 10 \(\mu g/m^3\) increase in NO\textsubscript{2} or PM\textsubscript{2.5} was estimated to be equivalent to the effect of maternal passive smoking of 1.3 \(\pm\) 0.7 or 3.8 \(\pm\) 2.3 cigarettes, respectively. The public health impact is significant, with an estimated 3.3\% of 2010 preterm births in the United States attributable to airborne PM exposure at a cost of more than $5 billion dollars in medical care and lost economic productivity.\textsuperscript{38}

Limitations to this evidence base include an inadequate understanding of specific windows of vulnerability (eg, trimester or more refined time points) during pregnancy. Current findings are heterogeneous regarding specific trimester effects.\textsuperscript{39,40} Methodologic challenges include accuracy of gestational age measurements, which may be measured by last menstrual period rather than by early ultrasonography.\textsuperscript{39}

Data on other pregnancy and birth outcomes are fewer but accumulating. Maternal air pollution exposure and hypertensive disorders in pregnancy have been systematically reviewed, and most studies support an association.\textsuperscript{41,42} This association provides a mechanistic link to intrauterine growth restriction, perinatal and neonatal mortality, preterm birth, and associated prematurity-related neonatal diseases, all highly associated with maternal hypertensive disorders. In addition, several studies report associations between airborne particulate exposures and increased risk of postneonatal death from respiratory causes.\textsuperscript{43–47} Exposures to airborne sulfur dioxide (SO\textsubscript{2})\textsuperscript{43} and CO\textsuperscript{42} have also been associated with postneonatal mortality. There are conflicting data on associations between ambient air pollution and sudden infant death.
syndrome. There have also been mixed results regarding a possible association between ambient air pollution and risk of stillbirth.

Thus, the evidence reveals that components of air pollution and proximity to traffic reduce birth weight and increase the risk for low birth weight at term, being small for gestational age, and preterm birth. Firm conclusions about congenital anomalies and air pollution are hampered by fewer available studies and inconsistent findings. Increased risk of congenital heart anomalies has been noted most consistently. Emerging data suggest that exposures to fracking may increase risk of certain adverse birth outcomes, including congenital anomalies, preterm birth, and being small for gestational age. Additional evidence needs to be amassed to understand the role of air pollution in infant mortality, stillbirth, sudden infant death syndrome, and congenital anomalies.

NEURODEVELOPMENT
Mechanistic data, large well-designed cohort studies, and systematic reviews of multiple studies support mounting concern for effects of air pollution, largely TRAP, on the developing central nervous system. Recent systematic reviews of the epidemiological studies suggest evidence is currently strongest for pre- or postnatal PAH exposure to decrease neurocognitive function and for PM$_{2.5}$ to increase the risk of autism spectrum disorder. There are also several studies revealing associations of autism spectrum disorder with NO$_2$. Studies in which researchers examine the association of air pollutant exposures with attention-deficit/hyperactivity disorder risk are fewer and have inconsistent results.

Interestingly, reduction in PAH pollution after the closure of a coal-fired power plant in China was reported to resolve previously observed decrements in motor and overall developmental quotients in area children, thus suggesting positive public health effects may be possible with improved community air quality.

Studies of possible associations between neurocognitive development and postnatal exposures to air pollution include a focus on school-based exposures. These studies demonstrate adverse effects of pollution on measures of academic success and neurobehavioral development. Growth in cognitive development assessed by repeat assessments has also been shown to be adversely affected by traffic-related air contaminant exposures at school.

Overall, despite heterogeneity in exposure assessments and outcome metrics, the current literature suggests that exposure to ambient air pollutants and TRAP in early life, particularly during pregnancy, likely plays a role in the genesis of neurodevelopmental disorders in children. Exposures postnatally may also affect the trajectory of normal neurodevelopment. Continued attention to robust characterization of exposures across the pediatric life course and assessment of neurodevelopmental health and functional growth throughout childhood are needed to more adequately assess risks and protective factors.

CHILDHOOD AND ADULT RESPIRATORY ILLNESS
The primary exposure pathway for air pollutants is inhalation, and thus, the respiratory tract represents a sensitive and key target for health effects. The adverse consequences of air pollution on the respiratory system have been recognized for decades, and the particular vulnerability of children is well established.

PM, NO$_x$, ozone, and measures of traffic are most frequently implicated in studies of respiratory compromise. Traffic is a primary source of NO$_x$ in most settings. For children living in urban areas, TRAP is often the most significant air pollution exposure source. For children residing in areas with significant animal agriculture, emerging data suggest emissions from large animal-feeding operations (eg, ammonia, hydrogen sulfide, and PM) may influence respiratory health, including pediatric asthma.

Children with asthma are highly vulnerable to the respiratory effects of air pollutants. Ozone, NO$_x$, PM, and TRAP have been consistently associated with reduced asthma control manifested as increased symptoms, such as wheezing, rescue medication use, and decreased lung function, as well as increased use of medical services and school absences. In addition, 2 of the most common infectious disease problems of early childhood, bronchiolitis and otitis media, have been linked with ambient air pollution. Air pollution may adversely affect individuals with cystic fibrosis by increasing the risk of pulmonary exacerbations and related antibiotic use as well as by increasing the risk for a decline in lung function.

There is a rapidly developing evidence base supporting the role of early-life exposures (including exposures in utero) in the development of asthma and allergic disease in childhood and in reduced lung function. In multiple large cohort studies in a variety of locations in Europe and North America, authors have investigated early-life exposure to NO$_x$, PM, and/or measures of traffic and found consistent associations with increased risk of development and exacerbation of
asthma. This reflects exposure based on home residence as well as exposure based on school proximity to roads with a high density of traffic. Similarly, several studies have reported associations between measures of air pollution exposure and lung function impairment in children. Long-term decrements in lung function growth have been demonstrated for pediatric cohorts both with and without asthma. In a systematic review of this body of evidence, the authors concluded that there is strong support for adverse effects on lung function growth in childhood and adolescence. The evidence base includes natural experimental studies that reveal improvements in lung function growth associated with relocation of children to less polluted residential settings or secular trends in air quality improvement, highlighting the potential public health impact of interventions to improve air quality.

In addition to asthma, other atopic conditions (eczema, allergic rhinitis) have been associated with exposure to TRAP. In addition to epidemiological studies, experimental data from studies of animals, in vitro systems, and human volunteers provide evidence of diesel exhaust particle induction of airway inflammatory reaction and enhancement of immunologic response to allergens. In the Canadian Healthy Infant Longitudinal Development (CHILD) prospective birth cohort study, exposure to NO2 during the first year of life (derived from a traffic-focused land use model) was positively associated with atopy at 1 year of age; exposure during pregnancy was not significantly associated. A recent meta-analysis revealed no association of TRAP with sensitization to indoor allergens; however, PM was associated with sensitization to outdoor allergens, such as pollen and grass.

In summary, ambient air pollution is responsible for a significant public health burden of respiratory disorders in children, including not only asthma and cystic fibrosis exacerbations but also the development of asthma and allergic disease as well as impaired lung functional development. These consistent findings support ongoing efforts and targeted interventions to reduce air pollution exposures among pregnant women and children in various locations (e.g., home, school, and while commuting).

LONG-TERM HEALTH EFFECTS

Cancer

Ambient air pollution and some of its constituents have been identified as human carcinogens that are specifically associated with increased risk of childhood leukemia. In 2013, the International Agency for Research on Cancer separately classified (1) outdoor air pollution and (2) PM, an important component of outdoor air pollution, as known human carcinogens, in addition to diesel and gasoline engine exhausts and some nitroarenes, which were designated in 2014. Authors of recent meta-analyses found a significant increase in risk of childhood leukemia associated with proximity of traffic and petrol stations to residence in the postnatal period. Increased risk of leukemia was not, however, associated with the same exposures in the prenatal period. The specific pollutants NO2 and benzene have been associated with childhood leukemia. Links between air pollution and other childhood cancers have been difficult to assess in meta-analyses because of the paucity of studies. Given emerging evidence regarding the association between postnatal exposure to TRAP and childhood cancers, ongoing research is merited to confirm these findings and examine other types of childhood cancers in relation to traffic proximity and specific pollutants.

Obesity

The relationship between childhood obesity and environment is multifactorial and complex. The effects of air pollution may be direct, through toxicological properties of particulate pollutants, or indirect, through, for example, avoidance of physical activity in areas of high traffic density because of safety concerns. Although the body of literature is small, several well-designed prospective studies have begun to describe this multidimensional relationship between TRAP and childhood obesity and emphasize the need for further studies to help clarify this relationship.

Antecedents of Adult Cardiovascular Disease

PM is a well-established risk factor for cardiovascular disease and mortality in adults, but data on childhood exposure to ambient air pollution and subsequent cardiovascular disease risk in adulthood are few. Prehypertension (3 measurements above the 90th percentile for age) in youth and adolescents is associated with PM, noise pollution, and secondhand tobacco smoke exposure. There is also a small emerging literature suggesting a potential link between childhood exposure to PM and development of short-term elevated blood pressure. Future longitudinal studies are needed to identify critical windows of exposure and whether elevated blood pressure attributed to air pollution in childhood persists into adulthood.

Modifiers of Health Effects

The ultimate toxicity of air pollution on individual children and populations is modified by developmental processes (timing of exposure during sensitive windows of development) and cofactors,
including other toxicant exposures and genetic polymorphisms. An ongoing emphasis of air pollution research in the area of developmental origins of health and disease will be useful in understanding the early life-course vulnerabilities and potential effects into adulthood (Fig 2). In addition, many studies are identifying potentiating effects of coexposure to social stressors. Genetic polymorphisms in several oxidative stress genes (GSTM1, GSTP1, and NQO1) and inflammation genes (TNF) have been associated with differences in toxic effects of ozone and PM. Future genome-wide association studies and whole-genome sequencing may be more helpful in understanding the mechanisms of health outcomes related to air pollution exposure.

There is emerging literature examining antioxidant properties of vitamins C, D, and E mitigating air pollution oxidative effects, but human data are few. In a clinical trial of children in Mexico City, investigators observed that genetically vulnerable children (those with severe asthma with the GSTM1 deletion in the area coding for glutathione transferase enzyme, which protects against oxidative stress) revealed less severe ozone-related decreases in forced expiratory flow when supplemented with antioxidant vitamins C and E.

**PREVENTION OF EXPOSURE AND EFFECTS**

Several natural experiments have demonstrated that reducing community air contaminants can lead to improved health outcomes. In the landmark study of 6 US cities, longitudinal decreases in PM resulted in significant decreases in mortality. Childhood asthma exacerbations were significantly reduced during the 1996 Olympics in Atlanta, Georgia, when initiatives surrounding the games included increased public transportation and telecommuting, which resulted in significantly less air pollution. In Beijing, China, when reduced air pollution measures for the 2008 Olympics occurred, birth weights averaged 23 g higher.

Regulatory protections under the Clean Air Act and the National Ambient Air Quality Standards have helped reduce the percentages of children living in areas with concentrations above the annual standard of PM (from 37.5% to 3.3%), the 8-hour ozone standard (from 66.1% to 57.8%), the 1-hour SO2 standard (from 31.1% to 3.0%), and the 1-hour NO2 standard (from 23.2% to 2%) from 1999 to 2016. Major ongoing contributors to air pollution, besides power plants and industrial fossil fuel combustion, include increasing urban populations and motor vehicles on the roads and deforestation that decreases plant use of CO2. Some strategies to address the growing motor vehicle fleet includes maintaining emissions standards, anticipation and planning of transportation efficiencies to match needs, availability of clean fuels (including electric), and requirements for inspections and vehicle maintenance.

Furthermore, catalytic converter use has reduced ambient air pollution (ie, ozone, mobile air toxics, and PM). Interestingly, when school buses in select Washington State communities used clear air technologies, participating elementary school students had both better lung function and less school absenteeism.

Other ways to decrease exposure would be to adjust behavior on the basis of degree of ambient air pollution. The Air Quality Index (AQI), calculated by the EPA on the basis of daily ambient concentrations of National Ambient Air Quality Standards and then grouped and color coded to 6 categories ranging from good (green) to hazardous (maroon), provides a tool for this.

On the basis of local air quality at any particular time, individuals may make decisions to reduce time spent in exertional activities outside or elect to do indoor activities. Although much of the general public may not recognize the AQI implications, the AQI would be a powerful tool for physicians to help patients alter behavior as needed if better awareness could be achieved. In addition to the AQI, policies limiting proximity of schools, child care facilities, and residences to traffic corridors, as well as anti-idling policies at schools, could reduce exposure in vulnerable populations in areas where they spend significant time. These strategies are important because many US children attend schools in high-traffic areas or encounter diesel school buses with exposure to traffic-related pollutants.

The EPA has reviewed costs and benefits of air quality regulation since 1997, and their first retrospective assessment demonstrated a mean benefit savings of $22.2 trillion, including health, quality of life, and agricultural benefits, versus a cost of regulation and compliance of $523 billion. Most recently, in 2011 an EPA prospective analysis of the years 1990–2020, a $2 trillion benefit and $65 billion in costs were calculated.

**CONCLUSIONS**

 Ambient air pollution is increasingly recognized as a preventable risk factor for a spectrum of pediatric health concerns. Not only do health effects manifest as exacerbations of chronic diseases (eg, asthma) but air pollution also appears to be associated with the development of major pediatric diseases, including adverse birth outcomes, abnormal lung and neurodevelopment, and pediatric cancer, as well as obesity and cardiovascular disease risk.
Proposed mechanisms involve immune, inflammatory, and oxidative pathways as well as gene-environment interactions and epigenetic changes combined with sociodemographic cofactors. Exposure and resultant health effects may be ameliorated most effectively through policy changes to reduce exposure across the life course. In addition, education around modifiable behaviors may contribute to exposure and toxicity reduction. Evidence demonstrates that children and adults are exposed to potential environmental toxicants from distant as well as proximal sources. This mandates preventive action at the level of state, national, and international policy development, regulation, and enforcement. Although inequalities of exposure to children in certain neighborhoods from local sources of pollution occur, children everywhere experience heightened risk. Continued efforts to unravel the complexity of combined exposures, exposure timing, moderators of effect, and key sources will serve to further focus the most effective policies and approaches for exposure prevention.

RECOMMENDATIONS

1. Pediatric Practice
   a. Recognize air quality concerns and resources in your practice area and for individual patients.
   b. Serve as a role model and practice model in reducing contributions to poor air quality by using and promoting active transport (eg, walking, cycling) and alternative transportation to gasoline-powered motor vehicles.
   c. Use the AQI as a tool in helping educate families of potential protective behaviors. The AQI may be the most useful in especially vulnerable patients with medical risk factors, such as preexisting asthma. Additional information, clinical scenarios, and patient education tools may be found through the EPA Web site at https://www.epa.gov/pmcourse/learn-about-particle-pollution-and-your-patients-health-course.
   d. Serve as a source of expertise in your community on the vulnerability of children to air pollution and the importance of primary prevention.

2. Research
   a. Evaluate programs and policies designed to reduce exposure to ambient and infiltrated air pollution for effectiveness and health economic analyses.
   b. Increase monitoring and health impact assessment in areas with historically less emphasis or enhanced vulnerability (areas with animal agricultural production, child care facilities, and low-income communities).
   c. Develop reliable biomarkers of exposure to key traffic constituents and biomass sources.
   d. Enhance understanding of genetic and nongenetic modifiers of air pollution effects.
   e. Elucidate effects of exposure to mixtures of air pollutants and modifying influences of nonchemical stressors, genetic factors, and nutrition.

3. Regulation and Policy
   a. Continue to uphold and strengthen the Clean Air Act, which has demonstrated success in reducing air pollution from mobile and stationary sources.
   b. Advocate for reversing rollbacks of emission limits for coal, gas, and oil industries.
   c. Ensure consideration of pediatric and fetal stages of the life course in all program and regulatory standard reviews related to ambient air contaminants.
   d. Promote school and child care facility siting policies that reduce exposure to traffic-derived air contaminants or other influential proximal sources.
   e. Incorporate active transport and air pollution exposure reduction considerations into land use planning.
   f. Promote lower-emission technology and approaches for transit.
   g. Review regulatory approaches for addressing increasing evidence of community health effects from air contaminants derived from animal-feeding operations.
   h. Advocate for 100% renewable energy.
   i. Require all states to have emissions standards and vehicle inspections to enforce these standards.
   j. Ensure adequate federal and state governmental allocation of resources:
      i. To optimize effective enforcement of current and future regulations of air pollutants.
      ii. To incentivize curtailing of polluting industries.
      iii. To facilitate adoption of lower-emission technology.

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ABBREVIATIONS

AQI: Air Quality Index
CO: carbon monoxide
EPA: US Environmental Protection Agency
NO2: nitrogen dioxide
NOx: nitrogen oxide
PAH: polycyclic aromatic hydrocarbon
PM: particulate matter
PM2.5: particulate matter ≤2.5 μm in diameter
PM10: particulate matter ≤10 μm in diameter
SO2: sulfur dioxide
TRAP: traffic-related air pollution
VOC: volatile organic compound

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