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Review on Air Pollution and Children's Health

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ABSTRACT

Air pollution remains a significant global public health concern, with mounting evidence highlighting the detrimental impact on children's health and development. This review synthesizes the latest research on the relationship between air pollutants and various adverse health outcomes in children. It examines the effects of exposure to common air pollutants, including particulate matter (PM), nitrogen oxides (NOx), ozone (O3), and other key contaminants, on children's respiratory health, cognitive function, and neurodevelopment. It discusses the heightened vulnerability of children due to their physiological, behavioral, and socioeconomic factors. The review highlights epidemiological studies that have linked air pollution exposure during pregnancy and early childhood to increased incidence of respiratory illnesses, such as asthma, bronchitis, and pneumonia. It explores the emerging evidence on the association between air pollutants and impaired cognitive development, decreased academic performance, and the potential contribution to neurodevelopmental disorders This review emphasizes the need for comprehensive, multidisciplinary approaches to address air pollution, including policy interventions, community-based initiatives, and innovative technological solutions. It underscores the importance of protecting children, the most vulnerable population, from the detrimental effects of air pollution through targeted public health strategies and environmental regulations. It provides a comprehensive overview of the current state of research on the nexus between air pollution and children's health, serving as a valuable resource for policymakers, public health professionals, and researchers working to mitigate the adverse impacts and promote children's wellbeing in the face of this pressing environmental challenge.

Keywords: Review, Air, Pollution, Children, Health

INTRODUCTION

Exposure to fine particulate pollution early in human development, particularly during pregnancy, poses significant risks to children's health and development. Maternal exposure to particulate pollution during pregnancy can harm the developing fetal brain, potentially lowering children's cognitive abilities [1]. Additionally, air pollution exposure during pregnancy increases the chances of premature birth and low birth weight, both of which are risk factors for developmental issues [2, 3]. In infancy and early childhood, exposure to air pollution leads to lung damage, hinders lung growth, and raises the risk of conditions like asthma, pneumonia, and chronic obstructive pulmonary disease later in life [4, 5]. Outdoor air pollution presents a significant challenge in developing countries. The World Health Organization has noted poor air quality in many large cities across these nations, exposing a substantial portion of the population to air pollutant levels well above recommended guidelines [6, 7]. Recent advancements in scientific understanding underscore the health impacts of air pollution, particularly on children. Children experience higher exposure to various air pollutants compared to adults due to their increased minute ventilation and physical activity levels [8]. Moreover, their extended outdoor activities contribute to heightened exposure to outdoor air pollutants [9]. Common air pollutants such as ozone, sulfur dioxide, particulate matter, and nitrogen dioxide have significant respiratory effects on children and adults, leading to increased respiratory illnesses, asthma exacerbations, and reduced lung function [10-16]. In adults,

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particulate air pollution is associated with hospitalizations for respiratory and cardiovascular conditions, cardiovascular mortality [17], and lung cancer [18]. Air pollution also impacts indirect health measures such as healthcare utilization and school absenteeism [10, 19, 20, 21]. Tirana, a city experiencing rapid population growth, faces serious air pollution issues, particularly from particulate matter originating from motor vehicle traffic, construction industry development, and dense population concentrations. This survey aimed to compare the lung function of children residing and attending schools in urban areas of Tirana with those in suburban areas of the city.

Early Vulnerability

Fetuses, infants, and young children are exceptionally vulnerable to environmental pollution, especially during critical stages of early development [22, 23]. Exposure to pollutants during infancy and early childhood can cause enduring harm to cells and tissues, increasing the likelihood of childhood diseases and impacting health throughout their lives [24]. Pollution early in life poses a significant risk by potentially hindering efforts to support children's development through improved nutrition, early education, and better healthcare.

Traditional environmental pollutants, such as waterborne coliforms or air pollution from solid fuels, primarily contribute to diseases like diarrhea, pneumonia, and other infections. In contrast, modern environmental threats are more closely linked to non-communicable diseases such as asthma, neurodevelopmental disorders, birth defects, obesity, diabetes, cardiovascular disease, mental health issues, and pediatric cancer [25]. Children in rapidly industrializing nations face concurrent challenges from both historic and contemporary environmental threats to health [26]. A 1993 report from the US National Academy of Sciences underscored children's heightened vulnerability to environmental pollutants. It emphasized that children breathe more air, consume more water, and ingest more food daily per kilogram of body weight, leading to increased exposure to environmental toxins. Children also have immature metabolic pathways, limiting their ability to efficiently detoxify and eliminate many toxic substances. Their delicate development processes are easily disrupted, especially during critical windows of vulnerability in early human development that are absent in adult life. Even low levels of toxic chemicals or environmental hazards during these sensitive periods can heighten disease risks during childhood and throughout their lifetime.

Respiratory Health Risks

Population growth, urban development, transportation, and various forms of pollution pose significant threats to human health and the environment [27-30]. Among these, air pollution stands out due to its detrimental impacts on cardiovascular and respiratory health [23, 31, 32, 33]. Industrial processes, transportation emissions, waste incineration, agricultural practices, and coal-fired power plants are well-known sources of anthropogenic air pollutants, while natural events like wildfires and volcanic eruptions contribute to atmospheric pollution. Inhalation of pollutants in densely populated megacities can lead to various health effects, prompting governments to implement measures to reduce emissions and issue warnings to vulnerable groups [34-39]. Geographical factors, topography, climate, and land use significantly influence the composition of air pollution. Economic growth, urbanization, household fuel use, industries, and vehicle emissions are major contributors to the emission of pollutants such as PM10, O₃, NO₂, SO₂, metals, and PAHs in megacities [40, 41, 11]. Moreover, Central and West Asia have experienced increased dust storms originating from sources like the Aral Sea region and the Taklimakan Desert, significantly elevating particulate matter (PM) levels and the frequency of dusty days [42]. PM is particularly noted for its harmful effects as an airborne pollutant [43, 44]. Recent studies worldwide, including in the United States, Europe, and Asia, have extensively investigated the health effects of air pollution. Findings consistently demonstrate increased risks of premature death associated with higher concentrations of PM10, O₃, NO₂, SO₂, metals, and PAHs [45-51]. The nervous, cardiovascular, and respiratory systems are particularly sensitive to air pollutants based on epidemiological data. Both short-term and long-term exposures to these pollutants have been linked to disorders affecting major organs such as the lungs, eyes, brain, and heart [52-54]. Symptoms commonly associated with exposure to pollutants like PM10, O₃, NO₂, SO₂, metals, and PAHs include coughing, asthma exacerbations, respiratory and cardiovascular diseases, eye irritation, and strokes [55-61]. [62], highlighted an increase in cardiovascular-related hospital visits during short-term dust storms.

This narrative review aims to explore the mechanisms and clinical manifestations of cardiovascular diseases (CVD) and respiratory diseases (RD) associated with external air pollution agents. It examines how airborne pollutants interact with cardiovascular and respiratory systems, emphasizing current knowledge on their detrimental effects on health.

Longitudinal Studies on Health (Infancy to early childhood)

Understanding the relative impacts of fetal development and childhood growth requires detailed There is a growing focus in medical research on how early life factors, including fetal and childhood conditions, interact with later exposures to affect health outcomes in adulthood analysis of longitudinal data [63]. For instance, researchers might investigate the relationships between birth weight and length, subsequent growth patterns during

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childhood, and health outcomes in early adulthood. Studies have linked low birth weight to a higher risk of asthma and a lower risk of atopic dermatitis in late childhood. Additionally, shorter height and higher body mass index (BMI) in childhood have been associated with increased asthma incidence. Some evidence also suggests that rapid length gain during the first year of life may be positively related to asthma, especially in those born with shorter lengths, but not atopy. Analyzing life course data presents several statistical challenges. Dependencies between repeated measurements on the same individual must be accounted for, and techniques such as multilevel models are now commonly used for this purpose [64]. When there are multiple measures of exposure that relate to a later-life outcome, standard regression models can suffer from multicollinearity due to a lack of independence and serial autocorrelation. Measurement error may also vary over time, with greater absolute error in adulthood than in childhood, and there is often dropout over time due to various factors such as death, illness, emigration, and nonresponse. Common approaches in the programming literature include z-score plots and conditional regression (or life course plots). However, z-score plots do not account for correlations between successive exposures or describe how an individual's exposure changes over time [65]. Additionally, by conditioning on an effect (such as disease status), these plots can create associations that do not reflect causal relationships. Life course plots are often used to illustrate relations between outcomes and both current exposure and changes in exposure, where a change in sign between two times is interpreted as indicating that the change between those times is related to the outcome. However, because successive exposures are autocorrelated, regression coefficients are affected by collinearity, a problem that worsens with more prior exposures conditioned on $\lceil 66 \rceil$. Both methods have major drawbacks: they require the same sample at each time point and simultaneous measurements for all individuals. Therefore, it is essential to develop a robust and flexible framework for analyzing life course data. Our goal was to

Therefore, it is essential to develop a robust and flexible framework for analyzing life course data. Our goal was to use multilevel models to describe the trajectories of weight and length/height gain in early childhood and to demonstrate how these trajectories can be linked to later health outcomes, using the example of rash occurrence in later childhood to illustrate the principles [67].

Impact of Specific Pollutants

The study integrated various techniques, including statistical analysis of spatial and temporal trends, population attributable fraction using the AIRQ+ model developed by the WHO, and burden of disease assessment using Disability-Adjusted Life Years (DALYs). Air quality data for pollutants such as SO₂, NO, NO₂, O₃, H₂S, benzene, PM10, PM2.5, CO, benzo(a)pyrene, and metals, collected from the Camp de Tarragona County air quality monitoring network between 2005 and 2017, were analyzed. Health impacts were assessed using the AIRQ+ model, and the burden of disease was calculated by determining Years of Lost Life (YLL) and Years Lost due to Disability (YLD). While air quality generally met European standards, it failed to meet WHO guidelines, particularly for O₃, PM10, and PM2.5. Temporal trends showed decreases in NO, NO₂, SO₂, PM10, and benzene levels, while O₃ levels increased. A correlation between unemployment rates and air pollutant levels suggested that the economic crisis (2008-2014) influenced pollution levels. Meeting WHO guidelines for PM2.5 in Camp de Tarragona County could reduce adult mortality by 23 to 297 cases annually, accounting for 0.5% to 7% of all mortality in the area. For lung cancer, ischemic heart disease, stroke, and chronic obstructive pulmonary disease linked to PM2.5 levels above WHO thresholds, the estimated DALYs were 240 years, translating to approximately 80 DALYs per 100,000 people annually between 2005 and 2017. The methodologies of population attributable fraction (PAF) and burden of disease (DALYs) are valuable tools for regional and national policymakers to make decisions on air pollution prevention and control and evaluate the cost-effectiveness of interventions. For decades, research has investigated the effects of ambient particulate matter (PM), volatile organic compounds (VOCs), metals, and polycyclic aromatic hydrocarbons (PAHs) on mortality [68]. Numerous epidemiological studies have shown a strong correlation between these pollutants and cardiovascular and respiratory diseases [57-61]. Various mechanisms have been proposed to explain how PM, VOCs, metals, and PAHs impact human respiratory physiology [69]. One such mechanism involves chronic inflammation resulting from the exposure of the respiratory system to air pollutants, which includes the binding of pathogenic antibodies to pro-inflammatory cell receptors. Ultrafine particulate matter (UFPM) inhaled from air pollution can enter the central nervous system (CNS) via the olfactory nerve or through the tight endothelial junctions of the blood-brain barrier. The blood-brain barrier's permeability may increase due to elevated levels of peripheral oxidative stress and inflammation [70]. Additionally, increased peripheral inflammation and macrophage activation can exacerbate existing neuroinflammation, further increasing the permeability of the blood-brain barrier. Research from epidemiological and animal studies indicates that young individuals are particularly vulnerable to the neurotoxic effects of air pollution [71-76]. Studies in Mexico City have shown that children exposed to high levels of air pollution exhibit elevated levels of neuroinflammatory markers in their brains and experience cognitive deficits **[**73**]**.

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Neurodevelopmental Concerns

Epidemiological and animal studies indicate that young individuals may be particularly vulnerable to neurotoxicity induced by air pollution [71-76]. Research in Mexico City has shown increased levels of neuroinflammatory markers in the brains of children exposed to high pollution levels, along with cognitive deficits [71-73], [77], found that early exposure to traffic-related air pollution was linked to hyperactivity in 7-year-old children. A retrospective cohort study in Catalonia, Spain, also found a connection between living within 300 meters of a motorway and the incidence of attention deficit hyperactivity disorder (ADHD) [78]. Conversely, a large European study of eight birth/child cohorts found no link between air pollution exposure and ADHD [79], with similar negative results reported in a Swedish study [80]. A recent systematic review concluded that the association between air pollution and ADHD is weak and requires further high-quality studies for clarification [81]. In six European cohorts, prenatal exposure to air pollution was associated with delayed psychomotor development [75]. Additionally, a study found an inverse relationship between traffic-related air pollution and sustained attention in adolescents [82] and reported lower cognitive development in primary school children [83]. The BREATH project in Catalonia, Spain, linked exposure to traffic-related air pollution to behavioral problems in children, including cognitive development issues, with the APOEE4 genotype identified as a risk factor [84]. Another study in the U.K. indicated that children with intellectual disabilities were more likely to live in areas with high levels of diesel exhaust particles (DE-PM), NO₂, and CO [85].

Experimental studies support the hypothesis that air pollution is a developmental neurotoxicant. [86], suggested that developmental exposure to diesel exhaust (DE) may lead to neurotoxicity. In utero exposure to high levels of DE (1.0 mg/m3) caused motor activity alterations, motor coordination issues, and impulsive behavior in male mice $\lceil 87, 88 \rceil$. Early postnatal exposure of mice to concentrated ambient particulate matter resulted in behavioral changes and long-term impairment of short-term memory and impulsivity-like behavior [89]. Further studies in mice demonstrated that post-natal exposure to DE-PM caused changes in GFAP expression in various brain regions [90], while lower exposure to ultrafine particulate matter (UFPM) led to male-specific learning and memory dysfunctions [91]. Prenatal exposure to urban air nanoparticles resulted in depression-like responses in mice [92]. Additional studies showed that developmental DE exposure altered motor activity, spatial learning, memory, and novel object recognition ability, and caused gene expression changes, neuroinflammation, and oxidative damage [93-96]. Another study found that prenatal exposure to PM0.2 caused male rats to exhibit activated microglia, impaired hippocampal neurogenesis, blood-brain barrier alterations, behavioral symptoms of depression, and contextual memory deficits [97]. Prenatal exposure to DE in rabbits resulted in changes in olfactory bulb morphology and olfactory-based behaviors, along with alterations in monoaminergic neurotransmission [98]. Overall, findings in humans and various animal models suggest that air pollution can harm the developing brain and potentially contribute to neurodevelopmental disorders. Autism spectrum disorder (ASD) is a major neurodevelopmental disorder, and evidence from epidemiological and controlled animal studies suggests a possible association between air pollution and its etiology. The following sections will thus focus on ASD and the potential contribution of air pollution to this disorder.

Economic Burden of Illnesses

The economic burden of asthma cases and exacerbations linked to air pollution is significant, totaling around \$18 million annually, with nearly half attributable to TRP-related asthma cases. Traditional risk assessments often overlook these costs. Clinically, this is crucial as the financial burden largely falls on families with asthmatic children. The yearly expense per asthma case, estimated between \$3,800 and \$4,000, constitutes 7% of the median household income in Riverside and 8% in Long Beach. These findings are concerning, as a sustainable healthcare expenditure is generally considered to be 5% of a family's income. Public decision-makers frequently utilize health impact assessments (HIA) to evaluate the effects of various regulatory policies. However, traditional HIAs often neglect to consider that chronic diseases (CDs) can both arise and be aggravated by a common factor, typically focusing only on exacerbations. For instance, exposure to near road traffic-related pollution (NRTP) can trigger the onset of CDs, while general ambient or urban background air pollution (BP) can worsen these conditions. We propose a comprehensive HIA approach that explicitly includes both acute and long-term effects, enabling the calculation of the overall disease burden attributable to air pollution. A case study applies both traditional and comprehensive HIA methods to two CDs-asthma in children and coronary heart disease (CHD) in adults over 65— in Ten European Cities, encompassing 1.89 million children aged 0-17 and 1.85 million adults aged 65 and over. We compare current health impacts with hypothetical scenarios where NRTP exposure for those living near busy roads is as low as that for those living farther away, and annual mean concentrations of PM10 and NO2indicators of general urban air pollution-do not exceed 20 µg/m3. The HIA based solely on acute effects estimates a cost of $\notin 0.55$ million (95% CI 0–0.95), accounting for only about 6.2% of the annual hospitalization burden computed with the comprehensive method $\in 8.81$ million (95% CI 3-14.4)], and approximately 0.15% of the overall economic burden of air pollution-related CDs T€ 370 million (95% CI 106-592)]. These findings

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suggest that morbidity effects have a more substantial and direct impact on the health system than previously recognized. This comprehensive understanding may highlight the extensive benefits of public health or environmental policies addressing CDs caused and exacerbated by common factors.

CONCLUSION

Air pollution significantly impacts children's health, posing risks from prenatal stages through early childhood and beyond. Key pollutants such as particulate matter (PM), nitrogen oxides (NOx), ozone (O_3) , and sulfur dioxide (SO_2) contribute to respiratory illnesses, neurodevelopmental disorders, and cognitive impairments in children. Page | 5 Studies highlight their heightened vulnerability due to physiological and behavioral factors, with long-term effects potentially extending into adulthood. Additionally, air pollution imposes a substantial economic burden, particularly on families and healthcare systems. Emerging evidence links air pollution to developmental disorders like ADHD and autism, with both human and animal studies supporting its neurotoxic effects. Economic analyses reveal the need for comprehensive health impact assessments to account for both acute and chronic disease burdens. Addressing these challenges requires multidisciplinary approaches, including stringent environmental regulations, innovative technological solutions, and public health strategies, to mitigate air pollution's effects and safeguard children's well-being.

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