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Aithal, S. S., Sachdeva, I. & Kurmi, O. P.

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Sathya Swarup Aithal[1], Ishaan Sachdeva[2] and Om P. Kurmi[3,4,5,6]

[1]The Global Health Office, McMaster University, Hamilton, ON, Canada. [2]Department of Mathematics and Sciences, Brock University, St. Catharines, ON, Canada. [3]Department of Medicine, Division of Respirology, McMaster University, Hamilton, ON, Canada. [4]Firestone Institute for Respiratory Health, St. Joseph's Healthcare, McMaster University, Hamilton, ON, Canada. [5]Faculty Centre for Intelligent Healthcare, Coventry University, Coventry, UK. [6]Nexus Institute of Research and Innovation, Lalitpur, Nepal.

Corresponding author: Om P. Kurmi (om.kurmi@coventry.ac.uk)

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Poor air quality is associated with adverse health impacts on children, including impaired lung growth and lower respiratory tract infections. The magnitude of risk is much higher in children in developing countries compared with developed ones. https://bit.ly/3K4N7SN

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Abstract

Air pollution is a leading modifiable risk factor for various cardio-respiratory outcomes globally, both for children and for adults. Children are particularly susceptible to the adverse effects of air pollution due to various physiological and behavioural factors. Children are at a higher risk of outcomes such as acute respiratory infections, asthma and decreased lung function due to air pollution exposure; the risk varies in different geographical regions, depending on the source of air pollution, duration of exposures and concentration. Prenatal exposure to air pollution may also contribute to adverse respiratory outcomes later in life.

Introduction

Air pollution generally refers to the pollution of indoor and outdoor air by any chemical, physical or biological agent harmful to human health and the environment [1].

According to the World Health Organization (WHO), 99% of the world's population lives in areas exceeding the WHO air quality guidelines [1]. The sources of air pollution are diverse and can broadly be categorised into household air pollution (HAP) and ambient air pollution (AAP). Air pollution is a risk factor for many diseases, such as heart disease, stroke, lower respiratory infections, lung cancer, diabetes and COPD. This results in around 7 million premature deaths annually [2]. The burden of disease and mortality from air pollution is disproportionally borne by those in low- and middle-income countries (LMICs), where the exposure tends to be the highest.

More than 40% of the world's population, including 1 billion children aged <15 years, are exposed to HAP. About 93% of the world's children aged <15 years (1.8 billion children) breathe polluted air, hence having adverse health outcomes during the developmental phase of their life [2]. In LMICs, 98% of children aged <5 years are exposed to particulate matter (PM) with aerodynamic diameter <2.5 µm ($PM_{2.5}$) levels above WHO air quality guidelines. About 52% of children aged <5 years in high-income countries are exposed to levels of $PM_{2.5}$ that exceed the WHO air quality guidelines [2]. The WHO publishes air quality guidelines for the pollutants: $PM_{2.5}$, PM_{10}, ozone (O_3), nitrogen dioxide (NO_2), sulfur dioxide (SO_2) and carbon monoxide (CO).

According to the WHO, one in 10 deaths or 600 000 deaths in children aged <15 years were attributed to AAP and HAP joint effects [1]. The fetus, infant and child are uniquely vulnerable and have a higher adverse effect on their health, particularly lung health, when exposed to air pollution. This is due to various physiological, behavioural and environmental factors [3]. In this article, we shall review the sources of air pollution, the reasons underlying the unique susceptibility of children to air pollution, and the various adverse health outcomes experienced by children due to their exposure to air pollution.

Sources of air pollution

Household air pollution

HAP is mainly produced by the inefficient combustion of solid fuels, such as coal and biomass, for cooking and heating. According to the WHO, in 2020, approximately 2.4 billion people or a third of the world's population were exposed to HAP from open fires or inefficient stoves burning kerosene, biomass or coal. Most of these people are the poor from LMICs who do not have access to relatively clean fuels. Although this is highly prevalent in LMICs, rural communities in LMICs that are marginalised and economically poor are the most vulnerable. Other important sources of indoor air pollution include tobacco products, building materials such as asbestos-containing insulation, newly installed carpeting, and certain wood pressed products [4].

With poor ventilation in the household environment, the levels of fine PM can reach 100 times higher than the WHO air quality guidelines. Children tend to experience high exposure to HAP for a few reasons. First, breastfeeding infants are more likely to spend their time indoors, in proximity to their mothers, causing greater exposure [5]. Secondly, children may engage in household chores such as cooking and collecting firewood, therefore spending more time exposed to harmful smoke and fuel.

In 2020, 3.2 million deaths globally were attributed to HAP. This included over 237 000 deaths of children aged <5 years [6]. Exposure to HAP nearly doubles children's risk for acute lower respiratory infection. It is responsible for almost half of the pneumonia deaths in children aged <5 years. There are also links between HAP exposure and low birthweight, tuberculosis, cataract, and nasopharyngeal and laryngeal cancers.

Ambient air pollution

AAP is the pollution of the ambient air from various sources, both natural and anthropogenic (man-made). Common anthropogenic sources of AAP include coal-fired power plants, waste incineration, agricultural practices and motor vehicle emissions. Natural sources of AAP include volcanoes, forest fires, wind-blown dust and emissions of volatile organic compounds from plants.

About 93% of all children and 630 million children aged <5 years are exposed to ambient levels of $PM_{2.5}$ above WHO guideline limits. AAP was responsible for 4.2 million premature deaths in 2016. Of these, almost 286 000 were children aged <5 years. Various systematic reviews have shown that AAP affects childhood acute respiratory infections, asthma, pneumonia, lung function, hypertension, and even cancers [7–10].

The sources of AAP can differ in urban and rural settings. In urban environments, the main sources of AAP include energy production, traffic-related air pollution and waste incineration. In rural settings, communities are exposed to AAP from agricultural waste incineration and certain agroforestry activities.

Air pollutants

PM, NO_2, SO_2 and ground-level O_3 are the major air pollutants that have adverse health outcomes. Depending on the aerodynamic equivalent diameter (AED), PM is categorised into coarse particles (PM_{10}), fine particles ($PM_{2.5}$) and ultra-fine PM. Particles of an AED smaller than 10 µm are likely to enter the lower airways, with a smaller AED generally resulting in deeper deposition, such as in the terminal bronchioles and alveoli. A major source of PM is fossil fuel combustion from human activity. Combustion-derived PM typically consists of a central nucleus of elemental carbon coated with polyaromatic hydrocarbons, metals and sulfates. Other sources include wildfires, agricultural activity, dusty roads and pollen. PM exposure can lead to increased pulmonary inflammation and consequent respiratory symptoms due to oxidative stress and direct toxic injury [11]. Exposure to high concentrations of PM can lead to an increased prevalence and exacerbation of diseases such as asthma and COPD. The toxic gaseous pollutants include volatile organic compounds, CO_2, CO, NO_2 and SO_2. Lead, cadmium, chromium, nickel and zinc are the most frequent air pollutants that can be deposited in the lung *via* PM. Air pollutants can have acute and chronic effects on the cardio-respiratory system and influence the development of serious lung pathologies.

Susceptibility of children

Various behavioural and cultural factors can frequently expose a child to HAP or AAP. For instance, breastfeeding mothers or mothers carrying children on their backs or laps while cooking indoors inadvertently expose them to high degrees of HAP. Children also tend to spend more time outdoors, engaging in physical activity such as play and exposing themselves to larger doses of AAP. Physiologically, infants have a higher breathing rate and breathe more air per unit of body mass than adults [12]. The steepest decline in the median respiratory rate is observed in the first 2 years of life, from a median of 44 breaths·min^{-1} at birth to 26 breaths·min^{-1} at 2 years [13]. Additionally, children are more

typically mouth breathers than adults [14] and tend to have a lesser nasal contribution to breathing. Furthermore, they have less efficient nasal filtering than adults [15]. These physiological factors increase the dose of toxic substances in a child's airways and lungs. Obstructive disorders and changes in lung function manifest earlier and more easily in children, as they have narrower airways than adults. Additionally, children born to mothers exposed to air pollution during pregnancy are more likely to have a low birthweight, increasing the complications for infection and cardio-respiratory abnormalities [16, 17]. Lastly, children have immature and developing immune systems, which makes them more susceptible to pathogenic invasion [14].

Effects of air pollution on children's health

Acute lower respiratory tract infection, including pneumonia

Pneumonia is an inflammatory state affecting the lung. It is characterised by an accumulation of pus and fluid in the alveoli, obstructing normal breathing. Pneumonia can be a result of bacterial, viral and fungal infections. In children, bacterial pneumonia is most often caused by *Streptococcus pneumoniae* bacteria, followed by *Haemophilus influenzae* type b (Hib). Respiratory syncytial virus (RSV) is the most common cause of viral pneumonia among children. The most common cause of pneumonia among infants with HIV is *Pneumocystis jirovecii*, accounting for a quarter of all pneumonia deaths among HIV-infected infants [18].

Air pollutants are thought to compromise the child's immune system against pathogens in the respiratory tract. In a healthy airway, typically, the epithelial cells lining the alveoli specialise in generating an inflammatory response against invading pathogens *via* the release of cytokines and radicals [19]. The subsequent recruitment of the inflammatory cells, such as macrophages and phagocytes, to the site phagocytoses and digests the pollutants and/or pathogens. However, exposure to air pollutants can compromise this inflammatory mechanism and render one more susceptive to developing acute lower respiratory infections [20].

Globally, pneumonia is the leading infectious disease of paediatric mortality, especially among children under the age of 5 years. In 2019, pneumonia accounted for 14% of the deaths among children under the age of 5 years and 22% of the deaths among children aged 1–5 years. 44% of pneumonia-related deaths in children aged <5 years are attributed to HAP. The risk of developing lower respiratory infections doubles in children exposed to HAP [21]. Literature also supports the association between the incidence of acute respiratory infections and AAP exposure. Ambient PM, in particular, has been associated with acute upper and lower respiratory infections [8, 22, 23]. Even short-term exposures to AAP (PM_{10}, $PM_{2.5}$, SO_2, NO_2 and O_3) were consistently associated with paediatric hospitalisations for pneumonia [24, 25].

It was noted during the coronavirus disease 2019 (COVID-19) pandemic that air pollution was linked with increased mortality from COVID-19, with every 1 $\mu g \cdot m^{-3}$ increase in $PM_{2.5}$ associated with an 8% increase in the risk of death [26]. Suggested plausible mechanisms for this include impaired epithelial barrier function [27], impaired macrophage function [28] and reduced innate immune antiviral response [29].

Asthma

Asthma is the most common chronic disease among children. This chronic condition is characterised by a variable degree of airway inflammation associated with excessive mucus production, reversible bronchoconstriction, and airway hyperreactivity. The asthma symptoms can include a combination of a wheeze, cough, shortness of breath and chest tightness.

Pollution-induced oxidative stress and consequent lung injury are thought to contribute to children's susceptibility to asthma from air pollution. There is compelling evidence linking exposure to AAP to the incidence and exacerbation of asthma [9, 30, 31]. Specifically, exposure to AAP has been associated with increased exacerbation, hospitalisation and poor control of asthma [32]. The increased exposure to AAP has been associated with higher rates of medication use [33], increased hospitalisation [34] and outpatient visits, and increased duration of hospitalisation [35].

Traffic-related air pollution (a significant source of AAP) was shown to be responsible for 14% of all incident cases and 15% of all childhood asthma exacerbations in 10 European cities. Similarly, in Bradford, UK, 18–38% of all asthma cases in children could be attributed to AAP [36]. Children who live or attend school in areas with high levels of AAP have a greater incidence and severity of asthma [12, 37].

Lung function

Multiple studies have reported on the effects of air pollution on decreased lung function in children [38, 39]. Even at low levels of exposure, there is evidence that exposure to air pollution affects lung

function in children. A recent review suggested that short-term exposures to O_3, PM_{10} and $PM_{2.5}$ was associated with lower lung function indices (forced expiratory volume in 1 s (FEV_1), forced vital capacity (FVC) and FEV_1/FVC). Similarly, studies support the association between early-life or long-term air pollutant exposure and decreased lung function. A recent large birth cohort study reported that higher exposure to air pollution in the early years was associated with reduced lung function, even in adolescence or 15-year-old children [40]. Importantly, lung function is a subclinical marker for long-term implications for lung health across a lifespan. The decreased growth in lung function due to air pollution could be predictive of COPD, other respiratory deficits, and higher morbidity or mortality later in life [41].

Effects of air pollution during pregnancy

The prenatal period is one of organogenesis and organ development. The fetal cells are uniquely susceptible to the effects of air pollutants due to the prenatal period having higher rates of cell division and differentiation and increased sensitivity to surrounding signals [42]. The pollutants inhaled by the mother can translocate to the developing fetus [32]. Inhaled pollutants have been observed in the placenta [43] and organs within the fetus [44].

The morphogenesis of the lung begins at 4–7 weeks of gestation. At around 9 months of gestational age, the alveolar phase is reached. However, the maturation of the lung continues into early adulthood [45]. Therefore, exposure to air pollution during early pregnancy can disrupt differentiation and morphogenesis. Later in pregnancy, air pollution affects the structural and functional growth of the lung. This exposure can lead to impaired lung function and development even after birth. A large prospective birth cohort study showed that air pollution exposure during pregnancy was associated with decreased lung function, high respiration rates and higher respiratory minute volume at 5 weeks of neonatal age. There is also limited evidence demonstrating the effects of prenatal air pollution exposure on lung function during childhood. These studies reported that exposure to $PM_{2.5}$ during pregnancy is negatively associated with FVC, FEV_1 and forced expiratory flow at 25–75% of FVC ($FEF_{25–75}$) between the ages of 5 and 11 years [46, 47].

Additionally, disruptions to the lungs' functional growth and repair mechanisms can render them more susceptible to acute respiratory infections and asthma during childhood. Various studies have associated intra-uterine exposure to pollutants such as $PM_{2.5}$, PM_{10} and NO_2 with the incidence of asthma and other atopic conditions in the first 3–6 years of a child's life [48–50]. Similarly, studies have shown an increased risk of respiratory symptoms and recurrent acute respiratory infections in the first 1–7 years of life due to intra-uterine exposure to PM [51, 52]. However, prenatal exposures are often correlated with postnatal exposures to air pollution. It is, therefore, critical to differentiate or adjust for postnatal exposures in these studies.

Key points

- 93% of the world's children under the age of 15 years, or 1.8 billion children, breathe polluted air, placing their health at risk. Children from LMICs are more likely to be exposed to air pollution.
- Due to various physiological, cultural and behavioural factors, children's susceptibility to adverse health outcomes from air pollution is increased.
- Among the adverse respiratory outcomes of air pollution on children are increased risks of acute lower respiratory tract infections and asthma, and decreased lung function.
- Prenatal exposure to air pollution may be associated with an increased risk of adverse respiratory outcomes during childhood. However, there is a need for further research concerning prenatal exposure to air pollution.

Self-evaluation questions

1. Why are children more susceptible to the effects of air pollution?
    a) Children tend to spend more time outdoors, engaging in physical activity such as play.
    b) Physiologically, children have a higher breathing rate and breathe more air per unit of body mass than adults.
    c) Mothers carrying children on their backs or laps while cooking indoors inadvertently expose them to high degrees of HAP.
    d) All the above
2. What is the main challenge in evaluating the impact of prenatal exposure to air pollution?
    a) Calculating the effective exposure to the fetus.
    b) Differentiating between the impact of prenatal and postnatal exposure.
    c) Assessing adverse fetal outcomes.
    d) The impact of prenatal exposure to air pollution is well established.

3. What is an important implication of decreased lung function in childhood?
   a) Decreased lung function growth returns to normal levels by adolescence, so there are no implications.
   b) Decreased lung function growth in childhood predicts higher mortality later in life.
   c) Decreased lung function growth in childhood is associated with an increased incidence of COPD and asthma later in life.
   d) Both b) and c).

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Suggested answers

1. d.
2. a.
3. d.