**Impact of indoor environment on children’s pulmonary health**

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**Abstract**

A child’s living environment has a significant impact on their respiratory health, with exposure to poor indoor air quality contributing to potentially lifelong respiratory morbidity. These effects occur throughout childhood, from the antenatal period through to adolescence. Children are particularly susceptible to the effects environmental insults, and children living in socioeconomic deprivation across the world are more likely to breathe air both indoors and outdoors which poses an acute and long-term risk to their health. Adult respiratory health is, at least in part, determined by exposures and respiratory system development in childhood, starting *in utero*.

Throughout this review will discuss, from a global perspective, what contributes to poor indoor air quality in the child’s home and school environment and the impact that indoor air pollution exposure has on respiratory health throughout the different stages of childhood. All children have the right to a living and educational environment without the threat of pollution affecting their health. Action is needed at multiple levels to address this pressing issue to improve lifelong respiratory health. Such action should incorporate a child’s rights-based approach, empowering children, and their families, to have access to clean air to breathe in their living environment.

**Executive Summary**

* Indoor air quality in the child’s home is affected significantly by a complex interrelationship between numerous factors including building characteristics, biological and non-biological pollution sources (originating from outside or inside) and dwelling occupancy.
* Exposure to poor indoor quality affects lung development, lung function, respiratory symptoms and risk of respiratory morbidity throughout their life course, beginning *in utero* through to adolescence.
* Antenatal exposure to air pollution impacts life-long respiratory health through direct insults to developing lungs and lung function, by its association with low birth weight, and by driving epigenetic DNA changes which have multigenerational effects. Further research is required to understand the differences between antenatal exposure to indoor, rather than ambient, air pollution.
* Younger children and infants appear particularly vulnerable to the respiratory sequalae of poor indoor air quality. This is likely due to their immature respiratory and immune systems. Younger children also spend a larger proportion of their time indoors. Exposure to indoor pollutants including particulate matter, mould and second-hand smoke increase their risk of respiratory tract infections and respiratory symptoms.
* Wheezing disorders in pre-school aged children are common but the risk of pre-school wheeze and pre-school onset asthma is increased by exposure to poor indoor air quality (for example exposure to particulate matter, mould, second-hand smoke). Exposure to mould in the home also increases the risk of chronic respiratory symptoms including cough and excess sputum production.
* School-aged children are at an increased risk of chronic respiratory symptoms (including cough and wheeze), asthma and respiratory tract infections when exposed to poor indoor air quality. Exposure to indoor air pollution also predisposes children to poorer lung function measures, signifying impact on lung growth.
* Children spend a significant proportion of their waking hours in school, and it is important that we consider the impact on indoor air quality in schools on child respiratory health. There is a complex relationship between building characteristics, classroom occupancy and processes within the classroom which contribute significantly to indoor air pollution, with a negative impact on respiratory outcomes for children.
* Children living in low and middle-income countries face increased challenges in terms of indoor air quality, particularly due to additional sources of indoor pollution including increased reliance on solid fuels, open fires, and rapid urbanisation. This significantly impacts lung development, respiratory symptoms, lung function and respiratory morbidity.

**Introduction**

Over recent decades, our knowledge of how indoor air pollution (IAP) affects respiratory health has grown. There is also greater understanding around the increased vulnerabilities of the respiratory health of children and young people (CYP) to air pollution exposure, due to incomplete development of their respiratory and immune systems, along with a greater minute volume to body surface area when compared to grown adults(1, 2). We have also learnt more on the negative effects of IAP at every stage of childhood even before birth, *in utero*. CYP spend a large amount of time indoors, which makes indoor air quality (IAQ) an even more pressing issue(3).

Due to its significant impact on CYP at every stage of growth and development, air pollution has been recognised as an issue of global importance, with the United Nations (UN) declaring an evaluation of ‘clean air’ as part of the committee of the UN Convention on the Rights of The Child (UNCRC) (4).

This review will explore, from a global perspective including in low and middle-income countries (LMICs), what contributes to IAP and the impact of IAP on the respiratory health of children throughout different stages of their life course.

**Indoor air pollution**

IAP can be influenced by factors from both indoors and outdoors. Indoor sources, as described by the Royal College of Paediatrics and Child Health (UK) and Royal College of Physicians (UK) in their report ‘The inside story: Health effect of IAQ on children and young people’ includes substances such as asbestos, man-made fibres, formaldehyde, mould and mildew, pet dander, dust mites, volatile organic compounds (VOCs) from toiletries and cleaning substances, carbon monoxide (CO), nitrogen dioxide (NO2) and particulates from gas cookers, pathogens and more (3). Exposure to second-hand smoke (SHS) in the home also has a detrimental impact on children’s respiratory health, and the impact of this will be explored further in this review (5).

*Building characteristics*

Poor quality or poorly maintained housing can predispose households to more of these sources e.g., leaks or poor ventilation leading to more moisture, in turn leading to mould and dust mites.

In 2021, nearly a quarter of privately rented homes in the UK failed the “Decent Homes Standard” which states a home is unsuitable for living in if it is in disrepair, cannot reasonably maintain thermal comfort, doesn’t have modern facilities or has a Category 1 hazard as defined by the Housing Health And Safety Rating System (HHSRS) (6, 7). A recent English Housing Survey found that an estimated 11% of privately rented homes had damp, compared to 4% of social housing and 2% of owner occupied homes (7). The same survey also found 8% of social housing was overcrowded, compared to 5% of privately rented and 1% of owner-occupied homes. Overcrowding can lead to increased transmission rates of respiratory infections (8), increased household moisture and therefore increased development of damp, mould and pests (9).

Examining building characteristics, it is important to remember that how and where buildings are constructed can influence the impact of IAP. For example, it is known that higher levels of air pollutants such as particulate matter (PM) are identified in homes with open fires, open stove cooking and even open planned kitchens. PM can be considered according to different size concentrations, with each to some degree associated with both increasing pulmonary inflammation and respiratory symptoms(10). PM from outdoor sources of air pollution can also make its way indoors confounding the impact of indoor sources(3). The proximity with which a house is near a source of outdoor pollution, and the build quality, can influence the degree to which this becomes a problem.

The sources and contributors to IAP can be potentiated due to poorly ventilated homes. Poor ventilation can lead to accumulation of, and therefore both increased concentration and length of exposure to, indoor pollutants. Poor ventilation can therefore increase the risk of childhood respiratory infections and exacerbations of asthma (11). There are special considerations for children when they sleep, as pollutants may increase in concentration at the level of a child’s bed, and when “bunk beds” are used, the child on the lower tier is typically in an environment with reduced ventilation. Families with lower income are more likely to live in areas of higher density such as flat complexes which are more prone to exposure of pollutants from neighbours (12). As discussed above, outdoor air pollution can also come into the home and become IAP, it has been found that the most deprived areas of the country are where 66% of carcinogenic air pollution is emitted(13).

Children with asthma who live in lower quality housing are also more vulnerable to the effects of pests. Those who live in lower quality housing have also been found to be more likely to be exposed to pests such as cockroach(14, 15). Presence of rats has been linked with increased risk of asthma diagnosis in children and exposure to cockroach allergen or mouse antigen is linked with emergency/unscheduled use of healthcare, with cockroach exposure also being linked with more days off school and more days of wheeze (16-19).

*Global inequalities in indoor air pollution*

Levels of IAP and its burden on respiratory health, are considerably higher in the developing world. People living in low- and middle-income countries (LMICs) are exposed to IAP for 3-7 hours longer per day than those in high-income countries (HICs). (1,2) As previously discussed, children are particularly vulnerable to the respiratory effects of IAP exposure. In LMICs, 98% of children under 5 years old are exposed to PM2.5 levels greater than the World Health Organisation (WHO) air quality guidelines (20). And according to the WHO, in 2020, household air pollution was responsible for approximately 3.2 million deaths worldwide, of these over 237,000 were children younger than 5 years old (21).

The greater burden of IAP in LMICs can largely be attributed to the fact that solid fuels (SF) remain the principal source of energy for cooking, lighting and heating for approximately 3 billion people worldwide. 90% of these solid fuel users reside in rural households in LMICs, with solid fuel use in HICs estimated at less than 5% (22, 23).

Socioeconomic status (SES) is fundamental in determining choice of fuel and movement up the “energy ladder”. Cleaner sources of energy such as electricity or kerosene, are more expensive and less widely accessible, particularly in rural communities. While poverty often dictates the use of solid fuels, it is also inextricably associated with other factors that exacerbate IAP, including poor housing conditions, inefficient ventilation, lack of chimneys and the inability to separate kitchens from other rooms in the household (24).

In homes where there are often open fires and poor ventilation, burning solid fuels (e.g., wood, charcoal, manure, crop residue) results in incomplete combustion and the production of pollutant emissions, including PM2.5, polycyclic aromatic hydrocarbons, sulfur dioxide (SO2), NO2, CO, VOCs and smoke condensate (22).

Worldwide the average person spends 80-90% of their time indoors, in LMICs women typically spend the most amount of time indoors, particularly those in rural households. Carrying the responsibility for cooking and maintaining heat and lighting, whilst often accompanied by their pre-school aged children, leads to significant health detriment to women and children in LMICs (22, 24).

Other important contributors to IAP in LMICs include rapid urbanisation and indoor smoking. Rapid urbanisation and industrialisation are well-known contributors to ambient air pollution, and when there is a high concentration of pollutants outdoors these can infiltrate into indoor environments. A 2011 Sri Lankan study demonstrated that indoor concentrations of NO2, SO2 and PM2.5 were higher in houses located in congested urban areas compared to those which used clean fuels in a semi-urban area (3, 24).

 As per the 2021 Intergovernmental Panel on Climate Change report, urbanisation is occurring most rapidly in LMICs as a result of high birth rates and increasing migration into cities (25).

*Second-hand smoke as an indoor air pollutant*

Up to 40% of children are exposed to SHS globally with the undisputedly detrimental impact on lung development, lung function and respiratory health widely reported. SHS exposure affects respiratory health throughout a child’s life course from the antenatal period to adolescence(5, 26). SHS contains thousands of harmful chemicals contributing to poor IAQ principally in three ways; i) smoke emitted from a lit cigarette (‘side-stream’ smoke), ii) smoke exhaled from a cigarette-user (‘mainstream’ smoke) and iii) chemicals from side and mainstream smoke attaching to carpet, furniture, soft furnishings, and dust (27, 28). The latter forms third-hand smoke (THS), contributing to longitudinal poor IAQ even after a smoker has extinguished their cigarette (29, 30). Children are once again particularly vulnerable to THS owing to their higher respiratory rate and inhale comparatively twice as much dust as adults (31). Furthermore, infants and younger children are more likely to be affected by exposure to THS through placing objects and toys from the floor to their mouths, crawling, and when walking, breathe air closer to carpets and furniture (32, 33). Taking measures such as smoking out of a window or in a room when a child is not present does not provide adequate protection from SHS and cigarette smoke can be present in a room for up to 2.5 hours after smoking one cigarette (34-37). Children from more socioeconomically deprived backgrounds are more likely to be exposed to SHS (38), not only due to increased risk of household smoking but also being more likely to live in a smaller home with less access to outside space (29). Within this review, we consider the contribution of SHS (and THS) exposure as a pollutant in the context of IAQ and as such will not describe the effects *per se* of maternal smoking in pregnancy or uptake of smoking in CYP.

Several studies have highlighted the significant impact of SHS on IAP. In 2018, Bui *et al* published a study on lung function trajectories of children exposed to parental smoking over a 60-year period. They demonstrated that exposure to parental smoking in childhood was associated with reduced lung function both in childhood and adulthood, as well as increased risk of developing COPD in adulthood (39, 40).

As a country’s SES rises, the cigarettes they use become more engineered, with filters and ventilation techniques that reduce emission levels. A 2010 study showed that 95% of cigarettes in HICs had ventilated filters compared to only 44% in low-income countries (41). The current prediction is that by 2030, over 80% of tobacco-related deaths will occur in LMICs (41). Given the increasing prevalence of smoking, less engineered cigarettes and the lack of legislation surrounding indoor smoking in developing countries, the likelihood that children will be exposed to SHS is significant. 40% of children worldwide are exposed to SHS, and approximately 165,000 children under 5 years old die from SHS-related illnesses annually: with two-thirds of these deaths occurring in developing countries (24, 42).

**Effects of exposure to indoor air pollution on respiratory health in children**

As previously described, exposure to poor IAQ affects CYP at different stages during their life course. As such, we will explore the effects of IAP on respiratory health at different stages of childhood, starting before birth, in the antenatal period.

*Antenatal period*

The Barker hypothesis popularised the concept of foetal origins of adult disease, in his 1991 study he established a correlation between low birth weight and reduced adult FEV1. Several subsequent studies have corroborated this association between low birthweight and subsequent respiratory ill health, including chronic lung disease in adulthood (43, 44). A 2017 meta-analysis of thirteen studies demonstrated a strong link between low birthweight and low adult FVC, indicating significant lifelong respiratory repercussions to antenatal IAP exposure (45). Studies have also shown that air pollutants can alter the genetics of a foetus; altering pulmonary function, increasing the risk of respiratory infections and diseases. The susceptibility of an individual regarding the effects of air pollutants is variable with genetic factors known to play a role in a person’s response(46). Pollution including PM can lead to epigenetic changes such as DNA methylation, leading to effects such as changes to surfactant protein expression, untoward effects on the development of an unborn child’s lungs and therefore correlation with long term respiratory health outcomes such as developing lung diseases (47). Epigenetic changes are transgenerational, the effects of which may still be seen in generations down the line.

Despite this, there is a lack of published high-quality evidence correlating intrauterine exposure to specific IAQ (rather than indoor and outdoor pollution) and childhood respiratory outcomes, with most evidence being centred around exposure to PM, carbon monoxide and SHS.

Intrauterine exposure to PM has been linked with decreased lung function in newborns and will go on to have effects further into childhood. If a mother, when pregnant, was exposed to higher amounts of PM, there is a greater chance of the offspring developing wheeze as an infant(48), and asthma – particularly when exposure occurs in the second trimester (when small airways are developing *in utero*) (49).

As well as PM, antenatal exposure of chemicals found in the house used for maintenance or improvements is also associated with a greater risk of developing wheeze when compared with exposure to the same chemicals as an infant (3, 50). Prenatal exposure to carbon monoxide also leads to decreased lung function in childhood (3, 46-51)

Evidence relating exposure to SHS in non-smoking mothers during pregnancy and respiratory outcomes in their children is sparser in comparison to the effects of antenatal smoking. However, specifically addressing the effect of SHS contributing to IAQ in the antenatal period, a large, pooled analysis of 15 European birth cohorts examined nearly 28,000 mother-infant pairs. This demonstrated that exposure to SHS in non-smoking pregnant women was an *independent* risk factor for wheezing in their children at 2 years of age (52). Furthermore, there is evidence that showing that pregnant women exposed to SHS are more likely to give birth to infants of low birth weight or small for gestational age which increases the risk for subsequent respiratory morbidity (53, 54).

Multiple studies have established a relationship between maternal IAP exposure during pregnancy and low infant birthweight – an independent risk factor for lifelong respiratory ill health as per Barker. Pope *et al* conducted a meta-analysis in 2010, of studies done in Guatemala, India, Pakistan and Zimbabwe, demonstrating that antenatal exposure to IAP was associated with a mean reduction of birthweight by 96.6g (22, 55). Whilst the pathophysiology of this relationship is not well understood, a study performed in Hong Kong – known to have high levels of ambient air pollution, showed that healthy post-partum placental cells contained nanoscale, carbonaceous, metal-bearing particles, supporting the concept that maternal exposure to air pollution directly correlates with foetal exposure (56, 57).

Importantly, some studies have looked at the impact of addressing sources of IAP for pregnant women, both on air quality and infant outcomes. The Household Air Pollution Intervention Network (HAPIN) trial recruited 3,200 pregnant women (9 to 20 weeks’ gestation) from rural Guatemala, India, Peru and Rwanda. They were randomised to an intervention or control group. The intervention group received a liquified petroleum gas (LPG) stove with fuel, and the control group continued using solid fuels for the duration of their pregnancy.

Primary outcomes measured for children in this trial include birthweight, incidence of severe pneumonia and growth in the first year of life. Secondary outcomes for children are pre-term birth, foetal growth, infant linear growth and development.

They measured the level of exposure to PM2·5, carbon monoxide, and black carbon (BC) pre- and post-intervention, as well as birth weight. Initial results of the RCT show that the exposure to fine PM in the intervention group was 66% lower than the control group, however, did not demonstrate significantly higher birthweights in the intervention group. However, a companion exposure-response analysis did find an association between increased exposure to PM2·5 and BC and lower birthweights. It showed an inter-quartile increase in antenatal exposure to PM2.5 was associated with a 14·8g reduction in birthweight and increased antenatal exposure to BC was associated with a 21·9g reduction of birthweight (58, 59).

The study also showed that infants born to mothers who received the intervention prior to 18 weeks’ gestation were on average 33.8 g heavier than those born to women who received the intervention later. This suggests that earlier implementation of LPG use may have been more protective (58, 59).

*Infants*

Those at the highest risk of respiratory ill-health secondary to IAP exposure are children younger than 3 years old (60). Various physiological explanations for this are provided, including that young children are of shorter stature and being closer to the ground they inhale a greater proportion of heavy PM. They also have a higher respiratory rate, favour mouth breathing – bypassing nasal filtration, and inhale a higher volume per unit body mass compared to adults, in addition to having immature respiratory and immune systems (20, 22, 61). It has been shown that the respiratory health of younger children is more vulnerable to the effects of air pollution than that of older children (62, 63).

There are few studies that have shown the impact of IAQ on respiratory health in infants.

A number of factors have been associated with having effects on the respiratory health of infants, summarised by the joint report by the Royal Colleges of Paediatrics and Child Health and Physicians (UK), ‘The inside story’ (3), with housing being a key related factor. The European birth cohort found a link between moisture damage in kitchens or visible mould with increased risk of wheezing in those aged up to 18 months, although a birth cohort in the USA did not find the same results (64-66). Exposure to chemicals including those used in home cleaning and maintenance or repair are associated with increased risk of airway inflammation and wheeze, with some lasting for long periods in the home and some leading to long term health implications which are not solely respiratory in nature (3, 50, 67, 68).

The difficulty in homes heating and retaining warmth, or for people affording to heat homes can also affect the respiratory health of children. Colder homes are also at more risk of damp, and infectious pathogens survive better in cooler homes compared to warmer ones. Cooler temperatures can also affect the innate capabilities of the immune system due to causing bronchoconstriction and decreased mucociliary clearance. A longitudinal study conducted in Ireland found infants living in ‘cold homes’ were more likely to experience wheeze (41% more so), as well as any respiratory illness (47% more so) (69).

Exposure to SHS in the home during infancy has been shown to result in an increased risk of respiratory tract infections by up to 50% and hospitalisation with a lower respiratory tract infection (70, 71).

Particularly in LMICs, younger children tend to spend more time indoors, alongside their mothers, and in closer proximity to open fires and kitchens (24, 72).

Dherani *et al* demonstrated an increased risk of acute respiratory infections by 78% in children exposed to IAP (60). A meta-analysis undertaken by Po *et al* showed that children from homes using solid fuels were three times more likely to develop acute respiratory infections compared to those from homes that used any alternative cleaner fuel. Whilst the same study showed that women exposed to SF were over twice as likely to develop COPD, the results for the incidence of asthma in children exposed to SF were inconclusive (73).

*Pre-school children*

Pre-school children often are affected by respiratory infections that can contribute to children suffering from viral induced wheeze, asthma, and lower respiratory tract infections (pneumonia). When analysing the effects that dampness and mould have on pre-school children, a systematic review by Dick *et al* assessing 135 papers identified a consistent correlation between increased risk of wheeze by the age of 2 with visible damp or mould (OR 1.4) (62). Persistent wheeze has been shown to also be associated with mould in children up to the age of 2 in a Polish birth cohort study (74). Children exposed to damp early in their life have been shown to be at a significant higher risk of developing asthma by age of 3(62). A study undertaken in Finland of 2568 children aged 1 to 6 years of age highlighted that mould noted to be either history of water damage, presence of moisture or visible mould or perceived mould odour was noted to increase the risk of respiratory symptoms; persistent cough (OR 2.17), wheezing (OR 2.62) and phlegm (OR 2.2) (75). Symptoms were further noted by the study team be linked in a dose-response relation with increased frequency of damp exposure over days linked to an increase occurrence in respiratory symptoms (75). The Pollution and the Young (PATY) study that examined twelve cross-sectional studies found positive associations between mould and children’s respiratory health consistently across the studies (76). Conversely, there are studies that have found no such association between adverse respiratory health outcomes and presence of dampness and mould in the home. A Swedish study found no link between asthma in children and levels of mould found in a child’s bedroom (77). In a study undertaken in the United States there was no association between mould in the home and recurrent night-time cough in children aged three years and under (78). Nonetheless there is compelling evidence demonstrating the link between indoor environmental mould exposure and wheezing and asthma in pre-school children. It is important to note that this association is also found in children without evidence of allergic sensitisation to moulds, suggesting that mould can act as either an allergen or irritant to the airways (79).

PM and other pollutants are found both inside and outside a child’s home, with outdoor sources contributing to the levels of pollutants found inside the home. High levels of NO2 and VOCs within the home have been associated with increased risk of developing wheeze before the age of 5 in children (80). A study examining 150 asthmatic children found that higher NO2 concentrations were associated with increased cough (IRR 1.1) and nocturnal symptoms (IRR 1.09) (81). A case-control study in Australia analysing VOC concentrations in asthmatic children’s homes found that there were higher levels of VOCs in children with asthma compared to non-asthmatic controls (82). On a US register, 75% of children with bronchopulmonary dysplasia who had required hospitalized due to respiratory illness were exposed to at least one air pollutant (83). Exposure to PM, both PM2.5 and PM2.5-10, increased respiratory symptoms and the need for asthma rescue medications amongst 150 asthmatic children aged 2-6 years in United States (84).

Exposure to SHS through parental smoking is also associated with increased risk of wheezing disorders in early childhood (5).

Overcrowding increases the levels of moisture and air pollutants, children under the age of 10 years do not count into the room standard or count as less than one person. A systematic review led by the World Health Organization (WHO) highlighted the increasing risk associated with overcrowding and respiratory illness in paediatric populations (8). A review undertaken by Antova *et al* found that overcrowding in households was found to be significantly associated with respiratory symptoms in children (76). A Swedish study that utilised mathematic simulations of different occupancy levels found an increase in occupancy positively correlated to an increase in moisture within homes (9).

*School-aged children*

School-aged children are exposed to indoor environmental factors at their homes as well as in the school environment.

An Italian study of 2529 school children with a mean of age of 10 years old showed that cough was associated with condensation on windows (OR 1.56) and phlegm was associated with mould (OR 1.56) and condensation on windows (OR 1.49) (85). A Macedonian study utilizing questionnaire data amongst school age children, found an associated risk between signs of mould in the house and children experiencing wheeze and a night-time dry cough(86). A systematic review by Groot *et al* analysing respiratory tract infections (RTIs) in studies undertaken in HICs showed a weak to moderate effect of damp and mould on RTI’s in children, however, this review did highlight limitations due to the majority of studies being cross-sectional in nature (87). Early exposure to damp and mould was strongly associated in an Italian study with asthma (OR 1.8), wheeze (OR 1.98) and cough/phlegm in children (88). Adolescents in this study were also shown to have symptoms of wheeze (OR 1.56) and asthma (OR 1.89) with early exposure to damp or mould. A systematic review undertaken by Dick *et al* found a correlation of developing asthma before the age of 9 and visible mould in the home (OR 1.5) (62). Castro-Rodriguez *et al* highlighted that older children exposed to damp or mould in the home were 50% more likely to have wheeze or asthma (89). This finding is substantiated with 14% of childhood asthma cases being linked to the presence of damp or mould in the home (90-92). Children are at increased risk of hospitalisation with pneumonia with exposure to damp or mould in the home (93). A prospective study in New Zealand utilized the Respiratory Hazard Index (RHI) and found that those with a higher RHI had an increased risk of hospitalization. The study further estimated that by reducing damp or mould exposure, admission rates would be reduced by 19% (94). By using the RHI, the study team also found a dose-response relationship amongst wheeze episodes and asthma attacks after adjustment for household crowding, age, sex and smoking. Children had an increased risk of 11% for both wheeze and asthma attacks over a year (63). A meta-analysis undertaken in the United States by Fisk *et al* found that where damp or mould was reported, there was an increased risk in upper respiratory tract symptoms (OR 2.04), cough (OR 1.65), wheeze (OR 1.66) and current asthma (OR 1.45) compared to homes without damp and mould (95). Damp and mould are important factors that can exacerbate respiratory symptoms and illness in children. The above studies highlight how there are multiple different aspects of household environmental factors that are associated with respiratory symptoms and diseases in school-aged children. Associations have been found with damp and mould and respiratory health in children highlighting that environmental prevention strategies may be beneficial in improving the respiratory health on children with strategies starting early in life.

In the United States, high levels of PM2.5 was associated with an increased risk of cough and asthma diagnoses in school-aged children (96). PM exposure in pregnancy was associated with increased risk of respiratory infections amongst seven-year olds (97). In asthmatic children, increasing concentrations of PM2.5 in the house was associated with decreased lung function (98). When examining VOCs, associated were found with high levels in relations to reduced lung function (99), non-atopic asthma (100, 101) and other respiratory symptoms (102). A longitudinal study highlighted that amongst 1300 asthmatic children with asthma higher levels of NO2 were associated with increased risk of asthma severity (103).

Exposure to SHS through parental smoking is associated with increased risk of developing chronic respiratory symptoms including cough, sputum production, wheezing and breathlessness in school-aged children as well as increased risk of having asthma (5). Moreover, in children with asthma, SHS exposure increases the risk of having asthma attacks and poorly controlled asthma and children exposed to SHS are twice as likely to be hospitalised with asthma compared to those not exposed (104, 105). SHS exposure has been shown to be associated with lower spirometry measurements in school-aged children and exposure from early postnatal life throughout childhood has been shown to be potentially linked to reduced attained FEV1 at age 16 years (106, 107). In severe childhood asthma, exposure to SHS has been shown to be potentially linked to steroid-insensitive disease (108). It is important that we also turn our attention to the effects of e-cigarette exposure, given their sharp rise in use. As a relatively new entity in comparison to cigarette smoking, data surrounding passive exposure to e-cigarette smoke on child respiratory health is lacking. However, evidence is already emerging demonstrating an increased risk of wheeze, asthma, uncontrolled asthma, and asthma attacks in children due to second-hand e-cigarette exposure (109, 110).

School aged children in both North India and China who live in homes that used SF were recorded to have consistently lower FVC and FEV1 than children in homes using clean fuels (24). Poor lung function in childhood is associated with a multitude of poor respiratory health outcomes, including wheeze, asthma and airway hyperreactivity. Low lung function in childhood is also reflective of adult lung function, increasing the risk of adult asthma and COPD (39, 72).

Children spend a significant proportion of their waking hours in school – in 2018, children in OECD countries spent an average of around 8000 hours of compulsory classroom time over an average of 9 years (111). Any conversation around the impact of IAQ on child health needs to therefore consider school air quality (AQ). School AQ is dependent on multiple interrelated factors, including building standards, levels and type of ventilation, and local outdoor air quality. These building-related factors are then compounded by high levels of occupier density in classrooms, and additional pollutant-producing equipment and activities such as photocopiers, blackboards/dry erase markers, and science and art equipment (3). School AQ is therefore often inadequate in comparison to office buildings and homes.

Whilst heterogeneity exists in findings due to variations in school buildings and air quality guidelines (112), there is evidence that children are exposed to substandard IAQ in schools across the world. This is a particular problem during winter/’heating’ months, where natural ventilation is limited and NO2, carbon dioxide (CO2) and PM levels are all higher. One of the largest multicentre studies, SINPHONIE, assessed IAQ in 115 schools across 23 European countries found that the mean levels of PM2.5, PM10, CO2 and VOCs in classrooms all exceeded the WHO and EU guidelines. Furthermore, there was a correlation between increased levels of PM2.5, CO, ozone, radon, benzene and limonene and respiratory tract, eye and systemic symptoms. Risk of respiratory symptoms significantly increased with VOC score in the study. Multiple studies have also found a correlation between absenteeism and academic performance, and classroom AQ and ventilation (112, 113).

The combination of poor ventilation and elevated humidity in school buildings also increases the risk of exposure to mould. A study of 32 schools and 33 homes in the US found that levels of mould were higher in schools than in home, and school mould levels were associated with higher asthma prevalence for students (114). The Health Effects of the School Environment study found that fungal DNA and viable mould (vm) were present in each of the 46 classrooms sampled (115). 33% of classrooms had more than maximum standard for VM (300cfm/m3), which was associated with a three-fold risk of night-time cough, and presence of aspergillus or penicillium DNA in classrooms were significantly associated with wheeze and rhinitis.

Many studies looking at school AQ have also examined levels of CO2 in classrooms. Whilst CO2 is not considered an air pollutant, it is a useful marker for ventilation levels and there is a direct correlation between rising CO2 levels and poor academic performance (116). The SINPHONIE study also found a significant correlation between high levels of CO2 and upper/lower respiratory symptoms and systemic symptoms in European school children (112). A study of 8 primary schools in England in 2017 found that that during heating season CO2 levels were above acceptable levels (CO2 >1000 PPM as set by UK government’s Health Safety Executive) 60% of the time (117).

School AQ is also determined by the location of the school and outdoor air pollution levels. A Spanish study using continuous and passive monitoring of indoor air pollutants found that indoor levels of PM2.5, PM10 and VOCs were significantly affected by school location, with schools in lower density urban areas with lower traffic levels persistently performing better (118). A 2017 study modelling PM2.5 levels and school locations in England found that approximately a third of all schools were in areas with higher than WHO recommended annual average of PM2.5 , with a correlation between increasing PM2.5 exposure and increasing deprivation using the Indices of Deprivation Affecting Children Index (IDACI) (119).

**Conclusion**

This review summarises the impact of IAP on children worldwide at various stages of their childhood. Multiple studies correlate exposure to childhood and antenatal IAP with lifelong negative respiratory consequences. Individual exposure is determined by numerous factors, including socioeconomic status, exposure to smoking, quality of housing and access to clean fuels. These exposures have been demonstrated to have a multitude of effects, from epigenetic DNA changes to increased incidence of adult COPD. These effects have been found to be intergenerational. Whilst the evidence included in this review demonstrates the negative impact of childhood exposure to IAP, there is still scope for further research, particularly in the understanding of breadth of the consequences antenatal IAP exposure. Other areas for future research include e-cigarettes, as aforementioned data on the effect of these on IAQ and the subsequent effects on child respiratory health is sparse.

**Expert opinion**

Over the last decade landmark rulings in two cases in the UK have for the first time highlighted direct links between air quality and children’s respiratory mortality. In 2020 a coroner concluded that air pollution from road traffic made a significant contribution to Ella Adoo Kissi-Debrah’s death from asthma in 2013 (120). In 2022 a coroner ruled that prolonged exposure to mould within the home caused the death of two-year-old Awaab Ishak (121). These cases have brought the quality of the air which children breathe into news and with it given the opportunity to examine factors that may affect this.

This paper offers a unique take on exposure to indoor air pollution and its multitude of effects on the respiratory system throughout the life course of children worldwide. WHO data from 2020 attributed household air pollution as responsible for approximately 3.2 million deaths worldwide, with over 237,000 of these being in children younger than 5 years of age (21). Evidence on IAP and its links to a wide spectrum of respiratory morbidity are presented, from acute respiratory infections and pneumonia to pre-school wheeze, asthma, and later COPD.

There is a plethora of evidence presented around more widely studied problems such as mould and damp and their relationship with wheezing disorders in early childhood and asthma. Less studied areas include antenatal effects of IAP – although there are well recognised effects of maternal smoking in pregnancy there is less clear evidence on the impact of second-hand smoke on the developing fetus. On a similar theme second-hand smoke has well established links to respiratory morbidity in infants and pre-school children, however e-cigarettes and vaping are a relatively new entity in comparison to cigarette smoking and data surrounding passive exposure to e-cigarette smoke on child respiratory health is lacking. There is an urgent need to further understand how e-cigarette aerosols and vapour contribute to IAQ and subsequent respiratory morbidity in children.

Evidence presented on IAQ in schools, where children spend a vast amount of time, is presented and demonstrates some alarming data in what is an understudied area. Air quality in schools has been found to be poor with international standards on acceptable levels of pollutants being far from met in the classroom – a place where other social inequalities should be lessened. This should be a focus for further research in the future.

Inequalities within developed countries are highlighted with IAQ being adversely affected both within the home and school by outdoor factors, and urban areas being disproportionately affected. Low-income families are more likely to live in areas with worse outdoor air quality and are also more likely to live in poor quality housing predisposing to many other adverse factors including damp, mould, pests, poor ventilation and overcrowding, giving cumulative effects both within the home and school environments. On a global scale the reliance upon the use of solid fuel leads to a huge burden of IAP with women and children having the greater exposure due to their time spent inside, with greater adverse effects on their respiratory health.

The problems with indoor air quality highlighted in this paper are multifaceted and do not have a simple resolution. In order to improve the respiratory health of children (and adults) indoor air quality must be addressed, with strategies aiming to provide national legislation relating to housing standards as well as legislation around tobacco and e-cigarette exposure being likely to benefit our youngest and most vulnerable members of society. The recent cases in the UK have highlighted the ability to hold to account those who are aware of poor living conditions causing detrimental health effects in the most extreme cases (121). This needs to expand to ensure new, affordable housing is of an adequate standard, whilst action is taken to identify and assist those in whom poor air quality in substandard housing is rectified or new accommodation provided. Clinicians should be aware of all potential adverse factors and address these in their consultations with children and young people presenting with respiratory symptoms.

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