The relationship of early-life household air pollution with childhood asthma and lung function

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Shareable abstract (@ERSpublications)
Increased evidence shows household exposures linked to development of respiratory diseases. Our review highlights the importance of timing of exposure during early life in relation to risk of asthma and reduced lung function in childhood. https://bit.ly/3R5Lfun

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Abstract

The increase in childhood asthma over the past few decades has made it an important public health issue. Poor lung function growth associated with some phenotypes of asthma compounds its long-term impact on the individual. Exposure to early-life household risk factors is believed to be linked with respiratory health while infants' lungs are still developing. This review summarises epidemiological studies and mechanistic evidence focusing on the detrimental effects of early-life household air exposures on the respiratory health of children, in particular effects on asthma and lung function. Many early-life household air exposures, including tobacco smoke, gases from heating and cooking, mould/dampness and cleaning products are associated with childhood asthma development and lung function growth. These exposures may alter structural and mechanical characteristics of infants' lungs and contribute to deficits in later life. In addition, some risk factors, including tobacco smoke and cleaning products, can transmit effects across generations to increase the risk of asthma in subsequent generations. This review supports the hypothesis that risks of asthma and accelerated lung ageing are established in early life. The timing of exposure may be critical in the pathogenesis of respiratory diseases, in terms of future risk of asthma and reduced lung function in adults.

Introduction

Asthma is responsible for a significant health burden in children. Asthma symptoms are experienced by 14% of children worldwide. The highest prevalence of recent wheeze (>20%) was observed in the English-speaking countries of Europe, North America and Australia, as well as parts of Latin America [1]. Phase 3 of the International Study of Asthma and Allergies in Childhood (ISAAC) found that the burden of asthma was greatest for children aged 10–14 years, and it was the most common chronic disease in this age group [2]. Although a large proportion of children with asthma/wheeze in early life go into remission, some asthma persists into adulthood [3]. Compared to doctor-diagnosed late-onset asthma (after the age of 3 years), children with early-onset asthma (up to and including the age of 3 years) are more likely to have persistent respiratory symptoms into later childhood [4] and even adulthood [5]. The characteristic pathological features of asthma, namely increased reticular basement membrane thickness and eosinophilic inflammation, can occur in very young children (aged 1–3 years) with recurrent wheeze [6]. Therefore, exposure to adverse early-life risk factors may not only influence asthma development, but may also contribute to lifetime lung function impairment.

Multi-trigger wheeze in young children has been linked to increased risk of asthma and chronic respiratory diseases in later life [7]. Recurrent viral infection is a common trigger of wheezing in young children, especially in those aged <5 years. In addition, many children wheeze in response to allergic and nonallergic airborne triggers such as tobacco smoke, traffic-related and combustion-related air pollution, animal hair and dander, pollen and mould (fungal) spores, or other pollutant exposures [7, 8]. These

exposures in early life may be related to asthma and lung function through the vulnerable developing immune and respiratory systems. Firstly, infants' immune responses of antigen presentation, phagocytosis and cytotoxicity are not totally understood; however, it is well known that the risk of asthma is enhanced if immune regulation is influenced by certain exposures in very early childhood [9]. Secondly, in addition to their potential influence on the neonatal immune system, early-life exposures may also be detrimental for development of healthy lungs. Newborn lungs are quite immature, and alveoli, capillary networks and airways grow rapidly in number and size until the child is 2 years old. After the age of 2 years, the lung continues to grow into adolescence, mainly by growth in volume of existing structures.

Many early-life household air exposures have been linked to asthma development and lung function impairment, for example tobacco smoke, cooking/heating, mould/dampness, pets and cleaning products. Although these exposures may also be harmful for older children, adolescents and adults, early childhood is a particularly vulnerable period, especially for indoor exposures. Infants are more highly exposed to many important household risk factors than adults. They spend most of their time indoors [10] and have an increased respiratory rate, inhaling more particulate matter relative to their body size [11]. Additionally, they usually breathe air closer to the ground, where airborne exposures are higher [12].

The evidence regarding the associations between early-life household air exposures, childhood asthma and lung function has not been comprehensively presented in previous reviews, as many of them focus on both childhood and adult asthma and/or present a broad overview of the full spectrum of risk factors. Identifying asthma is not easy in young children, as wheeze, the traditional symptom of asthma, may be caused by early-life respiratory infection [13]. This wheeze resolves with resolution of the infection and is often not indicative of asthma. Additionally, most asthma symptoms improve or disappear with age, although it is not currently possible to determine which early wheeze will resolve and which children will go on to have persistent asthma. Given the difficulties in defining childhood asthma in epidemiological studies, this review used a broader definition in conjunction with wheeze to include more asthma-related articles. This review summarises the epidemiological and mechanistic evidence linking early-life household air exposures before 2 years to outcomes of childhood asthma/wheezing and lung function impairment.

Critical period for lung growth and asthma development

Household air exposures that contain allergens may increase the risk of atopic sensitisation and lead to asthma through the development of airway inflammation, bronchial hyperresponsiveness and reversible airflow obstruction. Common household allergens include animal dander, mould and dust, and exposure to these in sensitive individuals can trigger an allergic-type immune response. Early-life exposures may also modulate the immune system and can increase the risk of asthma. For instance, tobacco smoke may influence the immune regulation of infants. There is clear evidence that tobacco smoke impairs type 1 T-helper cell (Th1)-type responses with a shift to Th2 immunity together with enhanced secretion of pro-inflammatory cytokines [14]. An immune response polarised to a Th2 type with increased pro-inflammatory interleukin (IL)-1β/IL-17 is a feature observed in childhood asthma [9].

Early-life environmental exposures may also substantially influence lung growth. Neonates' lungs may be especially vulnerable when moving from limited environmental exposures while *in utero* to diverse environmental exposures during and after birth. There is mounting epidemiological evidence that factors in the early-life environment have a major influence on lung health and maximally attained lung function. An Australian birth cohort observed that parental smoking during early life increased the risk of asthma, lung function impairment and lung growth in their children by adolescence, at 12 and 18 years [15]. A birth cohort study in Dunedin, New Zealand, found that >25% of children had wheezing that persisted into adulthood, and that the risk factors in early life included allergies, tobacco smoke exposure and early-age-onset asthma. In this study, adolescents with persistent wheezing had lower lung function in terms of a lower post-bronchodilator forced expiratory volume in 1 s (FEV_1)/forced vital capacity (FVC) ratio (more than two standard deviations below the mean values) than nonasthmatic and nonsmoking peers [3]. Although a newborn's lungs are able to function at the time of birth, the lungs are still quite immature, as the alveoli (the site of gas exchange) are not completely formed. Alveoli start to form after 36 weeks of gestation and grow rapidly in number and size until the child is 2 years old in order to enlarge the gas-exchange surface area [16]. The respiratory tree of bronchi and bronchioles is complete at birth and they increase in size (length and diameter) after this time. Capillary networks including those surrounding alveoli for gas exchange are also formed during the first 2 years of life. Simple lung growth then starts after 2 years, as single lung weight increases from 60 g to 750 g, and the remaining volume of air in the lungs after maximal expiration (functional lung residual capacity) increases from ~80 mL at birth to 3000 mL in early adulthood [16]. Lung growth is largely complete in late-adolescent girls; by contrast, adolescent boys continue to increase their lung volume well into their mid-20s [17]. Given these growth

patterns, early-life adverse exposures may impair the ability to attain maximal lung function at the end of adolescence and may also influence the rate of decline (accelerate decline) in later life.

Common early-life household air exposures

The adverse household factors that have been implicated in asthma pathogenesis and lung growth, particularly during infancy and early childhood, include environmental tobacco smoke, cooking/heating, mould, pet ownership and chemical pollutants.

Tobacco smoke exposure

There is irrefutable evidence that personal smoking has adverse effects on respiratory health, and the association between smoking and lung disease is universally acknowledged, even by the tobacco industry. However, evidence on the impact of passive smoke exposure, especially early-life exposure, is still an active area of research (table 1). *In utero* exposure to maternal tobacco smoking is associated with increased rates of asthma and wheezing in later life [30]. Nicotine has been shown to have detrimental effects on fetal lung development [16, 31]. Associations have been found between both pre- and post-natal exposure to maternal smoking and wheezing and asthma in children; however, the available studies have been unable to untangle distinct contributions of pre- and post-natal smoking [32].

A few epidemiological studies have investigated whether there is an effect on children's lungs of post-natal exposure to maternal smoking regarding childhood asthma; however, they have provided inconsistent evidence. An Australian study observed 4276 children from birth to age 14 years. In this study, maternal smoking was assessed during pregnancy, 3–5 days post-partum, at 6 months and at 5 years. This study found that pregnancy smoking only (OR 1.98, 95% CI 1.25–3.33) and post-natal smoke exposure only (OR 1.53, 95% CI 1.10–2.13) increased risk of child-reported asthma symptoms at age 14 [24]. Another large cohort study in western Norway (n=3786) found that children exposed to only pre- or only post-natal tobacco smoke did not have a substantial risk of developing adult asthma (self-reported doctor's diagnosis) and respiratory symptoms. However, children exposed to maternal smoking at both periods had significant risks of all respiratory outcomes [33].

TABLE 1 The association between early-life tobacco smoke exposure and respiratory outcomes in birth cohort studies

First author (year) [reference]	Exposure age	Outcome age	Results
Cunningham (1994) [18]	Pre-natal only and current smoking	8–12 years	Pre-natal smoking associated with reduced lung function Current smoking was not associated with reduced lung function
Wang (1994) [19]	The first 5 years	6–18 years	Reduced lung function
Gilliland (2003) [20]	*In utero* to post-natal maternal smoking	7–18 years	Reduced lung function
Rizzi (2004) [21]	During pregnancy and current smoking	Mean age 16 years	Reduced lung function
Jedrychowski (2005) [22]	Maternal smoking post-natally	9 years	Reduced lung function
Magnusson (2005) [23]	The 36th week of gestation	14–28 years	Increased risk of wheezing and hay fever
Alati (2006) [24]	Last trimester, 6 months and 5 years	14 years	*In utero* smoking increased risk of asthma in girls, but post-natal smoking (only) did not increase the risk of asthma No associations were seen in boys
Prabhu (2010) [25]	11 weeks, 20 weeks and 32 weeks of gestation	2 and 5 years	Maternal smoking throughout pregnancy associated with reduced lung function; children whose mothers smoked during the first trimester were at increased risk of asthma at age 2 years, but had normal lung function
Chen (2011) [26]	*In utero* and ETS before 5 years	12–14 years	*In utero* smoking associated with early-onset asthma; ETS before 5 years associated with late-onset asthma
Grabenhenrich (2014) [27]	*In utero* smoking	20 years	Increased risk of asthma
Dai (2017) [15]	Early-life smoking (mean time: 4 days before birth)	12 and 18 years	Reduced lung function, lung growth and increased risk of asthma seen in girls, not in boys
Thacher (2018) [28]	*In utero* smoking, during fetal life, infancy, childhood, adolescence	14–16 years	*In utero* smoking was associated with increased risk of asthma and rhinoconjunctivitis; tobacco smoking after birth was not associated with adolescent-onset asthma or rhinoconjuctivitis
Huang (2018) [29]	ETS before 3 years	8 years	Increased risk of asthma

ETS: environmental tobacco smoke.

Impaired lung growth may explain the subsequent increased risk of incidence in asthma associated with early-life tobacco smoke exposure, as many studies found that FEV_1 was more affected by tobacco smoke exposure than FVC. Dai *et al*. [34] observed that FEV_1, mid-expiratory flow (MEF) and FEV_1/FVC seemed to be reduced more than FVC at 18 years when participants were exposed to early-life maternal smoking (mean exposure time 4 days post-partum). The Dutch population-based Prevention and Incidence of Asthma and Mite Allergy (PIAMA) birth cohort suggested that persistent childhood passive smoking (measured at 3 months after birth, annually from 1 to 8 years and at 11 years) was associated with reduced FEV_1 growth per year from the age of 12 to 16 years, but the association was not seen for FVC. Reductions in these specific lung function parameters (FEV_1, MEF and FEV_1/FVC) are markers of airway obstruction, a classic feature of asthma. Lung function obstruction over time may become irreversible as a consequence of asthma progression [35].

Interestingly, more recent studies have investigated pre-conception smoking exposure on asthma risk for children. A large registry-based cohort recorded smoking habits of women in early pregnancy and followed-up smoking behaviour and asthma in their children and grandchildren. Maternal grandmother's smoking, independent of mother's smoking, was associated with increased risk of early persistent asthma in their grandchildren (at 0–3 and 4–6 years) [36, 37]. In this study, early persistent asthma was defined by purchase of at least two asthma medications before 3 years and at least two after 3 years. Another multigeneration analysis suggested that father's smoking before conception increased risk of parent-reported asthma for children at 10 years. These findings support possible epigenetic transmission of risk from tobacco smoking exposures in previous generations [38].

Air emissions during cooking/heating

Exposure to tobacco smoke and lung health has been well studied. However, there is currently a lack of evidence concerning this relationship for cooking/heating emissions in household settings, although both tobacco smoke and solid fuel/biomass/gas may have similar effects, given they generate small-sized particulate matter (diameter <2.5 µm) ($PM_{2.5}$) that can reach smaller airways on inspiration. It is acknowledged that inhaled $PM_{2.5}$, due to daily cooking, may generate air pollution amounts equivalent to smoking one cigarette per day [39]. Combustion of solid fuel, an important contributor to indoor air pollution in developing countries, also releases various gases including nitrogen oxides, carbon monoxide and carbon dioxide, which are all associated with lung damage.

Exposure to cooking/heating smoke occurs throughout life, including critical periods of lung development, and it may even start *in utero*, continuing during childhood and adulthood. Several cross-sectional and longitudinal studies have found that exposure to solid-fuel smoke increases risk of asthma and poor lung growth in children and young adults [40–42]. However, the potential for severe impact of exposure during early life, especially during the critical period for lung growth, has not been adequately addressed [39]. There is currently limited evidence on early-life exposure to fuel smoke from cooking/heating and whether this is likely to lead to adverse respiratory outcomes in later life. For example, the PIAMA birth cohort investigated 3590 children from birth to 8 years and found that ever-exposure gas cooking was associated . with prevalent asthma (parent-reported doctor's diagnosis) only in girls (OR 1.97, 95% CI 1.05–3.72) [40]. A recent African study found that pre-natal and post-natal exposure to firewood/kerosene and ethanol led to poorer lung function, assessed using oscillometry in 2-year-old children [43].

Air filter devices could be an effective strategy for improving household air quality for homes that generate air emissions from cooking/heating. A recent study indicated that sericin-coated polyester based air filters can remove $PM_{2.5}$ and PM_{10} from household fuel burning, reducing particulates from levels of 1000 $\mu g \cdot m^{-3}$ to 5 $\mu g \cdot m^{-3}$ within half an hour of operation [44]. Alternatively, effective house ventilation can also modify adverse respiratory health from cooking/heating exposure in children [45, 46]. However, there is a lack of evidence demonstrating whether any of these interventions can eliminate the impact of household air pollution during the critical window time for lung development in early life.

Mould/dampness

Mould and dampness have been reported to be related to higher risk of childhood asthma in several reviews [47, 48], although a few studies have not reported associations [49, 50]. Higher fungal allergen levels including *Cladosporium* spp., *Alternaria* spp., *Aspergillus* spp. and *Penicillium* spp. are found in houses with visible mould and signs of dampness [51, 52]. These diverse allergens are very small (usually 2–10 µm), allowing easy penetration and lodgement in the airways. They may bind antigen-specific immunoglobulin (Ig)E to mast cells or basophils that are associated with hypersensitivity or allergic reactions. Some fungal spores also release damaging mycotoxins that cause systemic inflammation, which can subsequently induce airway obstruction leading to symptoms of asthma [53]. A meta-analysis of eight

European birth cohorts reported a positive association between early exposure to visible mould and/or dampness during the first 2 years of life and the development of asthma in children aged between 3 and 10 years [54].

Few studies have followed-up participants long enough to assess associations beyond childhood into adolescence. The current evidence does not provide enough data for a firm conclusion on adverse long-term associations. The Barn/Child Allergy Milieu Stockholm Epidemiology (BAMSE) study investigated mould/dampness exposure for 4089 infants at 2 months of age and found increased risk of ISAAC questionnaire-defined asthma at 16 years of age [55]. In contrast, the Dutch PIAMA study (n=1871) found no evidence of an association between early-life mould/dampness and Mechanisms of the Development of Allergy (MeDALL) protocol-defined asthma at the age of 17 years. In the PIAMA study, exposure was measured over a longer period at 3 months and 6 months, and then annually until 8 years [49]. The reason for the inconsistency is unclear. Home dampness may lead to asthma causing exposures including growth of fungi and bacteria, and increased emissions from some chemical pollutants used to treat the mould [56]. Lack of quantitative measurements of total mould counts may also be a reason for inconsistent associations [57].

Early-life mould/dampness may be harmful for lung function growth in childhood. The PIAMA birth cohort suggested that early-life exposure to mould (or dampness) was associated with reduced lung function growth in FEV_1 and FVC between 12 and 16 years, but these associations were not found when exposure was during mid- or later childhood [58]. This study did not specify the mean time and range for early-life exposure.

Dogs and cats

Higher household allergens have clear asthma implications through the development of bronchial hyperresponsiveness and airway inflammation in sensitised subjects [59]. Given that homes with cats and dogs have elevated levels of allergens in both dust and air samples, including *Canis familiaris 1* and *Felis domesticus 1* [60–62], having pets at home is expected to have an impact on asthma. However, epidemiological studies have reported contradictory associations between early-life cat and dog exposure and asthma and lung function, depending on the study design and establishment of time sequence between exposure and disease. In general, birth cohorts have not found cat or dog exposure during early life to be associated with increased risk of childhood asthma or impaired lung function [63–66] (table 2). However,

TABLE 2 The association between early-life cat/dog exposure and respiratory outcomes in birth cohort studies

First author (year) [reference]	Exposure age	Outcome age	Results
Nafstad (2001) [65]	Cat/dog ownership at birth	4 years	No association was found on asthma
Remes (2001) [67]	Cat/dog ownership at birth	0–12 years	Dog exposure was inversely associated with wheezing
Almqvist (2003) [64]	Cat/dog ownership at birth	4 years	Dog exposure was inversely associated with asthma; no association for cat exposure
Heissenhuber (2003) [68]	Fel d 1 from mother's mattress at 3 months	0–2 years	Increased risk of wheezing
Lowe (2004) [63]	Cat/dog ownership at birth	3 years	No association was found on lung function
Sandin (2004) [69]	Cat/dog ownership at 1 year	1–4 years and 3–4 years	No association was found on wheezing
Hagendorens (2005) [70]	Cat/dog ownership pre-natal and post-natal	1 year	No association was found on wheezing
Campo (2006) [71]	Cat/dog ownership at birth	1 year	No association was found on wheezing
Chen (2008) [66]	Dog ownership in the first year and first 4 years	4 years, 5 years and 6 years	No association was found on asthma outcomes
Pohlabeln (2007) [72]	Cat/dog ownership at birth	2 years	Inverse association was found on asthma
Herr (2012) [73]	Cat exposure at 3 months	3, 6, 9, 12 and 18 months	Inverse association with any history of wheeze, mild wheeze and severe wheeze
Gaffin (2012) [74]	Cat exposure at 6 months	3 years	No association was found on asthma
Collin (2015) [75]	Cat/dog ownership during pregnancy (up to 28 weeks' gestation), 8 months, 2, 3, 4 and 7 years	6 months to 7 years for wheezing phenotypes 8 years for lung function	Cat exposure was inversely associated with wheezing No association was found on lung function
Hu (2017) [76]	Any pet ownership *in utero*, and during the first 2 years	11 years	*In utero* exposure was not associated with lung function impairment Pet ownership during the first 2 years was associated with reduced forced vital capacity

early-life exposure may have effects on respiratory health that are different from the effects of exposures at other times, as it is an important window for respiratory system development. The PIAMA cohort found that early-life cat exposure was associated with reduced FVC growth in adolescence between 12 and 16 years, but there was no significant influence on MeDALL protocol-defined asthma at 17 years, while other timings of exposure during childhood had no impacts on asthma and lung function in adolescence [49, 58]. A cohort study of 7326 school-aged children in China found that pet exposure in the first 2 years of life was associated with reduced FVC [76]. Adverse associations were more likely to be reported in cross-sectional studies or nonbirth cohorts [76, 77]. This inconsistency may be due to reverse causation, where people with asthma choose not to have pets, or recall bias among participants with current asthma. Ascertaining pet ownership retrospectively is more likely to lead to recall bias and a positive association between pet keeping and increased risk of asthma. Longitudinal study designs can more accurately ascertain the direction of causality for the relationship between early-life cat and dog exposure and asthma. Other than study design, there may also be issues with the way cat and dog exposures are measured. This is often by questionnaires that make no direct measurement of the level or type of allergen exposure.

Interestingly, 60% of all wheezing symptoms before 6 years of age resolve by later childhood [78]. The possible adverse outcomes for asthma development linked to pet exposure may also disappear over this time. The Lifestyle Immune System Allergy (LISA) birth cohort study suggested that cat allergens at 3 months were associated with early wheezing at 2 years, but these associations disappeared at 4 and 6 years [79]. In the longer term, many studies show that early-life exposure is protective for asthma. A Swedish birth cohort showed that keeping a dog in the first year of life had an inverse association with late onset wheezing at 4 years [69]; a UK population-based study also found that cat ownership during pregnancy (at 28 weeks) was associated with reduced risk in offspring of persistent wheezing from 6 months to 7 years of age [75]. One hypothesis is that limited exposure to cat or dog allergens may induce immune tolerance and reduce the risk of developing elevated IgE against other allergens during later life [80]. However, the threshold of exposure that may induce harm is unclear.

Cleaning products

In the past decade, increasing evidence has suggested that exposure to cleaning products may cause airway irritation and chronic inflammation, subsequently leading to asthma symptoms [81–83] and reduced lung function [84, 85]. A longitudinal prospective birth cohort of 3455 children in Canada observed increased risk of recurrent wheeze and physician-diagnosed asthma at 3 years in children living in homes with a higher frequency of use of cleaning products during infancy [86]. This Canadian study raised concerns about irritative effects of cleaning products on the respiratory system during early life. In fact, the cleaning products may influence the risk even before birth. A recent study suggested that exposure starting around conception, pregnancy and birth was associated with questionnaire-defined asthma for offspring at 10 years, but exposure only starting after birth was not associated with asthma risk [87]. Such airway irritants may pose hazards to developing airways for unborn babies through maternal transfer *via* the placenta. Studies looking at longer-term outcomes associated with cleaning product exposure after school age are required to explore possible long-term associations.

Understanding cleaning product ingredients is important to precisely explain the mechanisms of risk. Many fragrant cleaning products contain volatile organic compounds (VOCs) [88]. Some VOCs have been linked to asthma, atopic dermatitis and allergies [82, 88]. Other common active ingredients in disinfectants include quaternary ammonium compounds and sodium hypochlorite, which have also been linked to reduced airway function [89]. Spray products such as air fresheners may be particularly hazardous, because they facilitate aerosolised exposure [90]. Secondary exposures may be created when VOCs are mixed with other household air pollution [90], but the influence of secondary exposure is still not fully understood.

Implications and recommendations

Establishing an asthma-friendly home environment for infants and young children

Early-life exposures including tobacco smoke, mould and cleaning products are associated with long-term risks of asthma and lung function deficits. This evidence has the potential to be translated into important public messages to avoid or minimise household air exposures that lead to adverse respiratory health. Clinicians could advise parents of infants and young children to modify their homes to ensure an “asthma-friendly” home environment, when assessing children with early wheeze or asthma and developing management plans. Asthma-friendly home environments are those that endeavour to keep the indoor air as clean as possible. A key component of an asthma-friendly home environment is that it is smoke-free. Indoor air should be free of tobacco smoke, which contains irritants and chemicals known to be harmful for respiratory health. Public health messages should continue to advocate for protection from tobacco smoke exposure for pregnant women and infants.

Energy source selection at home is also linked to risk of childhood asthma and lung function deficits. Combustion of gas and wood generates a complex mixture of carbon-based particles and gases that may have health effects in children. Electric technologies can reduce the release of particulates from combustion activities in the home. It is particularly important to ventilate houses when using home heating and cooking systems that can generate high levels of pollutants, such as gas cooking and solid fuel combustion.

Based on this review, there is little evidence that early-life exposure to cats or dogs has an impact on the development of asthma and lung function impairment. The evidence is currently inconsistent, and no recommendations can be made.

Using cleaning products safely for young children

Although the link between cleaning products and respiratory health is suggestive, cleaning products are still extremely popular, particularly during flu season and during the coronavirus disease 2019 pandemic. To avoid the risks, it may be important to reduce the frequency of cleaning product use and select less harmful products. However, a lack of information and product labelling means that consumers are often not aware of potentially harmful chemicals present in these products [88]. Unfortunately, current disclosure regulations have led to a lack of transparency on cleaning products.

However, given the accumulating evidence, the American Lung Association recommends using cleaning products that "don't have VOCs, fragrances, irritants or flammable ingredients" [91]. Many strategies may be applied to minimise exposure for infants and young children; parents may avoid using cleaning products around children, allow adequate ventilation during and after cleaning activities, use dilute concentrations, avoid using multiple products together and rinse surfaces with water following product use [90].

Conclusions

In conclusion, many early-life household exposures in terms of tobacco smoke, mould and cleaning products are associated with childhood asthma development and reduced lung function growth. This literature review supports the hypothesis that asthma and lung function development and decline are programmed early in life and advances our understanding of timing of exposure on respiratory health. The household environment during early life is of critical importance in ensuring healthy lung development, and it may be important to develop preventive strategies for infants to avoid potentially adverse exposures and maximise their future respiratory health.

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