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Indoor air quality and respiratory health of children

Peter J. Franklin*

Centre for Asthma, Allergy and Respiratory Research, University of Western Australia, and WHO Collaborating Centre for Research on Children's Environmental Health, Telethon Institute for Child Health Research & Curtin University of Technology, Perth, WA, Australia; CRC for Asthma and Airways

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Summary Indoor air pollution (IAP) is an important environmental health issue in developing countries and is a major contributor to mortality and morbidity from acute lower respiratory illness in children. In developed countries, IAP in homes is not nearly as severe as it can be in developing countries; however, evidence suggests that it does contribute significantly to the risk of adverse respiratory health in children. Children spend the majority of their time indoors, mostly at home. Homes are built so that air exchange between the indoor and outdoor environments is minimised and there is a large range of pollution emission sources inside. For many pollutants, indoor concentrations regularly exceed those outdoors. Although there has been considerable interest in the health effects of IAP, questions still remain regarding the role of IAP in the exacerbation and/or development of respiratory disease. Prospective, longitudinal studies are required to better clarify the contribution of IAP to the respiratory health of children.

Indoor air pollution (IAP) is a major contributor to the global burden of disease and is the second biggest environmental contributor to ill health worldwide.[1] IAP is an important contributor to acute lower respiratory infections (ALRI), such as pneumonia, in children. Of course, the burden of disease due to IAP is disproportionately borne by children in developing countries where the burning of biomass indoors for cooking and heating contributes to extremely high levels of particulates and combustion-related pollutants. The effect of IAP on children's respiratory health in industrialised countries is much less extreme, more subtle and varies from those observed in poorer households in the developing world. However, there is increasing evidence that IAP contributes to respiratory disease in children in developed countries. Indeed, there is now a considerable body of literature on the health effects of various indoor air pollutants. Despite this the contribution of IAP to respiratory disease remains to be fully resolved.

WHY INDOOR AIR POLLUTION?

Outdoor air pollution has long been considered a considerable risk for human health, particularly since major air pollution events such as the London smog of the early 1950s. Although outdoor air pollution, particularly pollution associated with vehicle exhausts, remains a concern,[2] there has been a growing recognition that IAP is of equal or greater significance to human health.[3] Reasons for this include: the amount of time people spend indoors; the wide and varied range of indoor emission sources; and the increased concentration of some pollutants indoors compared with outdoors.

* Centre for Asthma, Allergy and Respiratory Research, First Floor, B Block, Sir Charles Gairdner Hospital, Hospital Avenue, Nedlands, Western Australia 6009. Tel.: +61 8 9346 7949; Fax: +61 8 9310 4997.
E-mail address: Peter.Franklin@uwa.edu.au.

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Time indoors

In industrialised countries people spend over 80% of their time inside at home, school and the office.[4,5] Children spend most of their time inside at home. On average, over 16 hours a day is spent in this environment.[5] For preschool children the time indoors at home is greater still, often exceeding 85%.[4] Pregnant mothers also spend most of their time inside at home[4] and, therefore, IAP may also be critical for exposures to environmental toxicants during the pre-natal period.

Indoor concentrations

For many air pollutants concentrations are often considerably higher indoors than outdoors. For example, during the operation of unflued gas heaters, peak indoor concentrations have been reported to exceed outdoor maximum allowable levels by an order of magnitude.[6] Important indoor pollutants such as formaldehyde and other volatile organic compounds are consistently found to be higher indoor than outdoors,[7,8] while in some areas indoor particulate concentrations can be twice those measured outdoors.[9]

There are two reasons why indoor concentrations may exceed levels outdoors. First, the large number of emission sources inside homes. Second, the 'tightness' of homes built since the mid 1970s. Table 1 presents the major indoor air pollutants and their sources. It is evident from this table that a large variety of indoor emission sources of chemical pollutants exist inside homes that include all aspects of modern living. Over the past three or four decades there has been a large increase in the number of chemicals produced and used in the manufacture of construction and household products.[10] Although this 'modernisation' has lead to the improved quality of life that we now enjoy, it has also increased the potential for exposure to chemical mixtures with short- or long-term health effects that we do not fully understand.

The concentration of IAP is directly related to its emission rate and inversely to air change rate and ventilation efficiency of a building. Since the 1970s, due to the need for greater energy efficiency, homes have become much tighter with extremely low natural ventilation rates. Natural ventilation (or leakage) is measured in air changes per hour (ach) between the outdoor and indoor environments with all external openings (windows and doors) shut. The American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHREA) recommends at least 0.35 ach to provide acceptable indoor air quality (ASHRAE standard 90.2). However, air exchange rates in modern homes can be as low as 0.2 ach.[11]

Personal exposure

As a result of the above factors the indoor environment is a major contributor to personal exposure to many air pollutants including nitrogen dioxide (NO_2),[12] formaldehyde,[8] volatile organic compounds (VOCs)[7] and particulate matter (PM).[13]

IAP AND CHILDREN'S RESPIRATORY HEALTH

Important indoor pollutants were identified in Table 1. Each of these has been associated with adverse respiratory health in children, although in many cases the data remain equivocal. Although indoor biological pollutants are an important contributor to respiratory disease in children,[14] the remainder of this review will focus on the more ubiquitous chemical pollutants in homes.

Environmental tobacco smoke (ETS)

The evidence for increased respiratory morbidity from passive smoking, particularly for children, is fairly consistent. In an extensive review of the literature, the United States

Table 1 Indoor air pollutants and their main emission sources in homes

Pollutant	Domestic sources
Environmental tobacco smoke (ETS)	Cigarettes, cigars, pipes
Biologicals (e.g. house dust mite, animal dander, mould, cockroaches)	Dampness, moisture, floor dust, bedding, insects, pets, pests
Nitrogen dioxide	Combustion sources particularly unvented gas or kerosene appliances
Formaldehyde	Composite wood products such as particleboard, furnishings, combustion sources, ETS, cosmetics, paints
Volatile organic compounds	Cleaning agents, aerosol sprays, pesticides, paints, solvents, building materials, combustion sources, glues
Particulate matter	Outdoors, combustion sources such as cigarettes, wood stoves and candles, cooking, cleaning, general activity
Polycyclic aromatic hydrocarbons	Vehicle exhausts, cigarette smoke, cooking, woodsmoke
Pesticides	Commercial and residential application of insecticides and herbicides, treated wood products
Radon	Soil and bedrock under homes, ground water

Environmental Protection Authority (USEPA) concluded that passive smoking for children was associated with increased prevalence of respiratory symptoms of irritation, increased prevalence of middle ear effusion, a small but significant reduction in lung function, increased risk of lower respiratory tract infections in infants and increased severity of asthma in asthmatic children.[15] There is a high degree of consistency among published studies and a dose–response relationship can be demonstrated, which suggests that the association between parental smoking and respiratory illness in children is very likely to be causal.[15] The role of ETS in the development of childhood asthma is less clear. The USEPA concluded that, although a causal relationship could not be definitively demonstrated, exposure to ETS was considered a probable risk factor for the induction of asthma in childhood.[15] There is also good evidence that early exposure to ETS – *in utero* and early post-natal – has significant impact on lung function in infancy[16] and some evidence that maternal smoking can affect foetal immune function.[17] Parental smoking is associated with wheeze and asthma-like symptoms in young children, but the relationship is not as strong in older children.[18]

Although smoking rates have reduced in many Western countries, it is still an important IAP. Bans on smoking in indoor public and work places have been introduced in many industrialised countries, however, the home remains an unregulated source of ETS exposure and is probably the most important source of exposure for children. Parental smoking behaviour at home does seem to be changing with more parents reporting smoking outdoors.[19] Smoking outdoors can reduce levels of smoking-related IAPs in homes as well as biomarkers of exposure, urinary cotinine, in young children.[20] However, these levels still remain above those found in non-smoking homes and it is unclear whether parental smoking outdoors at home is sufficient to protect children from the adverse effects of ETS.

Nitrogen dioxide

NO_2 is an important constituent of both indoor and outdoor air pollution and there has been considerable research on the effects of both indoor and outdoor exposure to NO_2 on children's respiratory health.[21] In the indoor environment, increased respiratory symptoms in children have been associated with exposure to either gas appliances[22,23] or NO_2 concentrations.[24–26] Some years ago a meta-analysis of the available studies estimated that the risk of lower respiratory tract illness in children exposed to a long-term increase in NO_2 exposure of 15 ppb was increased by about 20%.[27] Since that time the weight of research suggests NO_2 contributes to respiratory symptoms in children, particularly asthmatic children; however, there is still a degree of inconsistency in the data.[28]

One of the difficulties in determining the health impacts of environmental pollutants, such as NO_2, is adequately measuring personal exposure. In homes, NO_2 concentrations will fluctuate depending on sources (e.g. operation of gas appliances). Exposure will also depend on the proximity of children to the source. Personal exposure studies have found that indoor NO_2 concentrations may be a good predictor of exposure in young[12] but not older[29] children. In one of the few studies to measure personal exposure of children to NO_2, Smith *et al.*[26] found significant associations between daily variations in personal NO_2 and asthma symptoms in children. Measuring personal exposure, particularly in children, can be difficult, predominantly due to the difficulties of compliance, but more studies using personal exposures may be required to better determine the health impacts of this pollutant.

Formaldehyde

Formaldehyde is classified as a human carcinogen and high occupational exposures are considered a risk for nasopharyngeal cancer. At lower concentrations, formaldehyde has mostly been associated with irritation of the eyes, nose and upper airways.[30] The role of formaldehyde in lower respiratory symptoms and asthma is controversial. Formaldehyde has been considered a potential agent for occupational asthma, although the incidence of formaldehyde-induced asthma seems to be rare.[30]

Interest in domestic exposure to formaldehyde arose in the early 1980s due to complaints from residents in homes with urea-formaldehyde foam insulation (UFFI). Early studies comparing UFFI and non-UFFI homes found increased rates of eye, nose and throat irritation but were unable to find consistent differences in reported respiratory symptoms in children.[31] However, since the early 1990s there have been a number of studies that have reported associations between formaldehyde concentrations in homes and schools with asthma,[32,33] asthma severity,[34,35] allergy[34,36,37] and airway inflammation[38] in children. The data is still limited and not all studies have reported adverse impacts of domestic formaldehyde exposure.[39]

There is some evidence that formaldehyde is a respiratory sensitiser, although this has been subject to some debate. Formaldehyde is a low-molecular-weight chemical (MW 30) and is too small to stimulate the formation of specific antibodies. However, it can bind with human serum albumin (HSA) to form an antigenic conjugate.[30] Research into the development of formaldehyde-HSA antibodies due to occupational exposure to formaldehyde has been controversial.[40,41] In the past 10 years, there has been some evidence that low level exposure to formaldehyde in homes[34,36] and schools[37] is associated with markers of allergy in children. For example, Wantke *et al.*[37] found an exposure dependent specific immunoglobulin E (IgE)-mediated sensitisation in children associated with formaldehyde concentrations in schools. In homes, Garrett *et al.*[34] reported an association between formaldehyde levels and atopy, as determined by the presence of positive skin-prick reactions, as well as the severity of reactions, in children.

Erdei *et al.*[36] also found increased blood monocytes and bacteria-specific IgG in asthmatic children living in homes with high formaldehyde concentrations. None of these studies have been able to demonstrate an association between markers of sensitisation and respiratory symptoms in children. The implications of these observations need to be explored further.[42]

Volatile organic compounds

VOCs are organic chemicals that easily vaporise at room temperature. The sources of VOCs in homes are many and varied and over 300 individual compounds have been measured in indoor environments. Individual compounds have been associated with a variety of health effects including irritation, neurologic and respiratory symptoms.[43] Some compounds, such as benzene, dichloromethane and tetrachloromethane, are known carcinogens.[43] For children, exposure to VOCs occurs predominantly in the home and there is now a small, but growing, body of research linking domestic exposure to VOCs to adverse respiratory health outcomes in children. In the literature, exposure to VOCs has been determined by using either proxy measures, such as building materials and household products,[44,45] or actual VOCs concentrations.[35,46] Associations between these measures of exposure and poor respiratory health have been observed in infants,[44,45,47], preschool[48] and school-aged[49] children. Again, these findings are not universal.[35,39]

Interestingly, there is now some evidence that early life exposures to VOCs may impact on the developing foetal and infant immune system and increase the risk of the development of allergic disease in young children.[45,47,48,50] There is evidence from two separate birth cohort studies, conducted by the same research group, of an association between: (1) maternal exposure to VOCs in homes and increased T-helper (Th)2 and decreased Th1 cytokines in cord blood;[50] and (2) home renovations just prior to birth or during the first year of life and increased risk of allergic symptoms and eczema in infants.[47] Furthermore, domestic exposure to VOCs[48] or household chemical products[45] can increase the risk of asthma-like wheeze in preschool children. Whether early life exposures contribute to the development of asthma that persists into later childhood is not yet known.

Particulate matter

Most research into the health effects of PM has concentrated on the outdoor environment.[2] There are very few data on the health impacts of indoor PM. Of course, there has been a lot of research into factors that contribute to increased indoor PM concentrations such as ETS and cooking and heating appliances but very little of this research has focused on indoor PM *per se*. Recently, there has been an attempt to characterise the separate toxicity of indoor- and outdoor-generated particles.[51] Interestingly in a recent series of animal exposure studies, a greater inflammatory response to indoor compared with outdoor PM samples was reported,[51] however, in epidemiological studies, ambient, but not indoor, generated fine particulates were associated with decreased lung function in adults[52] and increased exhaled nitric oxide in children.[53] More work is required to separate the contribution of indoor- or outdoor-generated particles on the health of children.

DOES IAP CAUSE ASTHMA?

Indoor allergens are considered by some, but not all, to be an important cause of asthma and allergy in children.[14] In contrast, indoor chemical air pollutants are considered more likely to be, at worst, a trigger for asthmatic symptoms. A causative role for IAP in asthma is difficult to demonstrate, however, evidence is emerging that suggests some air pollutants may indeed contribute to the development of asthma and other allergic diseases in children. For example, exposure of pregnant women to air pollutants such as ETS,[17] VOCs[50] and polycyclic aromatic hydrocarbons[54] are associated with an increased Th2 bias in cord blood. Furthermore, early exposure to VOCs[48] or VOC-emitting sources, such as new building materials[47] and household chemicals,[45] has been associated with increased allergic and asthma-like symptoms in infants and young children. Finally, exposure to fume-emitting heating sources – such as unflued gas heaters – in infancy has been associated with asthma in 7-year-old[55] and 8–11-year-old[56] children. It is possible that there is an interaction between indoor pollutants and indoor allergens[57] but this has not yet been adequately explored. A number of longitudinal birth cohort studies involving early life exposure to IAP are planned or are underway[44,45,47,58–60] and should help shed some light on the role of IAP in childhood asthma. None of these studies have yet reported on asthma in later childhood.

CONCLUSION

In 2000, the US Institute of Medicine published a book on asthma and indoor air exposures,[61] which concluded that for asthma exacerbations there was: sufficient evidence of a causal relationship with ETS; sufficient evidence of an association with NO_2; and suggestive evidence of an association with formaldehyde. For asthma initiation, the authors concluded that there was no evidence of a causal relationship with any of the chemical pollutants although there was suggestive evidence of an association with ETS. Since the publication of that book, not enough data have been published to re-assess these conclusions, although the evidence for the adverse effects of air toxics – such as VOCs and formaldehyde – is growing.[33,38,44,46,47] To date, most studies investigating the respiratory health effects of IAP on children have been cross-sectional and there are

important questions that cannot be answered using this type of study design. For example: what are the threshold concentrations, if any, for triggering symptoms in children with asthma?; and what are the important periods of exposure that may impact on the development of disease? These questions can only be answered by longitudinal, prospective studies, such as the birth cohort studies mentioned in the previous section. Other issues that need to be considered in future IAP studies include the interaction between indoor allergens and chemical pollutants[57] and the role of gene–environment interactions.[62]

FINAL REMARKS

Children spend the majority of their time indoors at home and this is unlikely to change in the near future. Homes are still built to reduce air 'leakage' and increase energy efficiency. Again this remains a dominant factor in building design. The exchange between indoor and outdoor air, therefore, relies on the opening of external windows and doors. However, there is an increased reliance on mechanical means to control indoor climate, reducing the need for active ventilation. Some form of heating has been essential for homes for many decades but in recent times there has been an explosion in the use of air conditioning for cooling in summer. For example, between 1999 and 2005, home air-conditioner ownership rose by more than 50% in Australian homes, with this increase projected to continue.[63] In the US, the use of central air conditioning in homes doubled from 27 to 55% between 1980 and 2001.[64] Obviously this makes life more comfortable for residents but there is a potential legacy with regard to exposure to IAP. In 1998, Turner *et al.*[65] published the results of a study that found that the use of air conditioners in homes was associated with a 15-fold increased risk (95% confidence interval 1.3 to 166) of a near fatal asthma attack. The authors concluded that this finding was unlikely to be due to air conditioning *per se* but possibly as a result of a build-up of pollutants and allergens in the homes of people who regularly use air conditioning.[65] This was a small study and similar results have not been reported elsewhere, however, in light of current trends regarding the penetration of air conditioners in homes, this suggests that domestic air pollution may be an issue of concern for the foreseeable future.

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