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REVIEW ARTICLE

Do indoor environments influence asthma and asthma-related symptoms among adults in homes? A review of the literature

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This review summarizes the results of epidemiological studies focusing on the detrimental effects of home environmental factors on asthma morbidity in adults. We reviewed the literature on indoor air quality (IAQ), physical and sociodemographic factors, and asthma morbidity in homes, and identified commonly reported asthma, allergic, and respiratory symptoms involving the home environment. Reported IAQ and asthma morbidity data strongly indicated positive associations between indoor air pollution and adverse health effects in most studies. Indoor factors most consistently associated with asthma and asthma-related symptoms in adults included fuel combustion, mold growth, and environmental tobacco smoke. Environmental exposure may increase an adult's risk of developing asthma and also may increase the risk of asthma exacerbations. Evaluation of present IAQ levels, exposure characteristics, and the role of exposure to these factors in relation to asthma morbidity is important for improving our understanding, identifying the burden, and for developing and implementing interventions aimed at reducing asthma morbidity.

Introduction

Exposure or sensitization to indoor pollutants, including cigarette smoke,[1] air pollution,[2] and allergens,[3] are important risk factors for asthma morbidity. Although the role of indoor environmental exposure in the development of asthma morbidity and exacerbations is largely unknown, strong evidence suggests that exposure to indoor risk factors plays a key role in triggering and exacerbating asthma, allergic, and respiratory symptoms.

Many studies have examined the association between exposure to indoor factors and asthma, allergic and respiratory symptom among children in school and daycare environments.[4–6] However, adults can be exposed to indoor

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risk factors in environments other than daycare and school settings. Evidence suggests that many houses or occupational settings in China[7] and other countries[8–11] pose significant and serious indoor environmental problems, because adults spend as much as 90% of their lives in these environments.[12]

Although a number of individual studies of indoor air quality (IAQ), exposure characteristics, and the role of these exposures in relation to asthma morbidity in homes or occupational settings have been performed, a comprehensive examination of the existing studies has not been compiled. This review therefore aims to summarize the results of epidemiological studies focusing on the detrimental effects of indoor environmental factors on asthma morbidity in adults. We summarize the key findings from the recent scientific literature, identify the most commonly reported asthma morbidities involving home environments, evaluate the key indoor aero contaminants most likely to be related to asthma morbidity [e.g. nitrogen dioxide (NO_2), volatile organic compounds (VOCs), and biological contaminants], and discuss the role of these exposures in relation to asthma morbidity and the extent to which these exposures affect asthma morbidity and severity.

Study selection

Scientific studies published in journals, conference proceedings, and dissertations, since 1999 were searched using a number of electronic databases, including PubMed, MEDLINE, Proquest Health & Medical Complete and conference proceedings. Relevant studies were identified using the following key words: "Indoor air pollution AND asthma", "Epidemiologic studies AND asthma", "Indoor air pollutant AND asthma and adult", and "Indoor air pollution AND asthma odds ratio (OR) allergy OR respiratory symptom AND adult". All retrieved abstracts were read initially to judge the suitability of the studies. The full texts of the selected references were then retrieved and saved. A total of 162 articles were assessed, of which 72 were finally used in this review.

Chemical contaminants

Environmental tobacco smoke

Environmental tobacco smoke (ETS) is a dynamic, complex mixture of more than 4000 chemicals found in both vapor and particle phases.[13] The 2006 US Surgeon General's report states that ETS is a cause of premature mortality. Living with a smoker is associated with an estimated 20–30% increased risk of lung cancer.[14] Exposure to ETS is common in adults and has also been associated with an increased risk of asthma morbidity.

Very few measurements of ETS in home settings have been reported in the scientific literature.[15,16] In a cohort study in the US, Eisner et al found that ETS levels varied between different settings.[16] The detectable level of 7-day median nicotine concentration was 0.03 $\mu g/m^3$. Measured nicotine concentrations were the highest among the persons who reported home exposure (median 0.61 $\mu g/m^3$), followed by work exposure (0.03 $\mu g/m^3$), outdoor exposure (0.025 $\mu g/m^3$), and no exposure (0 $\mu g/m^3$; $p = 0.03$). In another study, Eisner et al[15] noted that higher levels of second hand smoke exposure, as measured by the personal nicotine badge, were related to greater asthma severity; the second and third tertiles of hair nicotine exposure were associated with a greater risk of hospital admission for asthma [hazard ratio (HR) 3.73; 95% confidence intervals (CI) 1.04–13.30 and HR 3.61; 95% CI 1.0–12.9, respectively].

The effects of ETS on asthma morbidity have been investigated in variety of publications,[1,17–24] and the evidence indicates that adults who are exposed to ETS have a greater risk of developing asthma. In a large study conducted among 3471 residents in Copenhagen, Denmark, aged 18–69 years, Hersoug et al[1] found that persons exposed to ETS for >5 h/d were at increased risk of wheeze, chronic cough, and decreased lung function [forced expiratory volume (FEV)1% predicted]. In another study, also by Hersoug et al,[17] exposure to ETS for 0.5–5 hours was associated with an increased prevalence of rhinitis symptoms, compared with exposure for less than 0.5 hours. Gupta et al[20] reported an OR of 1.22 (CI, 1.08–1.38) for an asthma attack in persons exposed to ETS in India.

A recent French study investigated the relationship between smoking and fraction of exhaled nitric oxide (FeNO) levels in relation to the present asthma and atopy status in 654 adults (268 asthmatic participants). Mean FeNO values increased with asthma, atopy, and eosinophilia ($p < 0.001$), and decreased with smoking. ETS exposure of at least 2 h/d and active smoking were negatively associated with the FeNO levels in nonasthmatic participants ($p < 0.001$) and asthmatic participants ($p = 0.02$).[19]

Exposure to ETS was recently studied together with other potential asthma risk factors in several studies.[22,25–27] One cross-sectional study from Taiwan explored the relative and overall contributions of parental atopy and environmental exposures to adulthood asthma among 24,784 participants. Home exposure (OR, 1.80; 95% CI, 1.08–3.23; and population attributable risk, 28.04%) and parental atopy (OR, 4.47; 95% CI, 3.47–5.75; and percent attributable risk, 31.38%) were associated with new-onset asthma in adults.[25] In Liaoling, in a heavy-industry province of northeastern China, 31,704 adults from six cities were studied. Exposure to smoking (OR, 2.06–5.02), occupational dust (OR, 1.35–1.72), occupational gas (OR, 1.48–1.72), and presence of irritating smoke during cooking (OR, 1.54–2.22) were closely related to persistent cough, persistent phlegm and wheeze, as well as to asthma.[22]

Coal and biomass fuels

Stoves fueled by coal or biomass, which are major sources of indoor combustion, release respiratory irritants such as particulate matter (PM), carbon monoxide (CO), sulfur dioxide, NO_2, and organic toxins. Burning biomass fuels, mainly wood, crop residues, and livestock dung, remain as the important source of exposure to a variety of toxins. According to some estimates, half of the world's population still uses coal or biomass for heating and cooking, most of who live in developing countries, such as China.[28]

Several studies have suggested that pollutants from coal and biomass fuel combustion could be present in environments. In a rural agricultural area of Tibet, China, the daily average $PM_{2.5}$ concentrations in the kitchen, living room, bedroom, and yard were 134.91 $\mu g/m^3$, 103.61 $\mu g/m^3$, 76.13 $\mu g/m^3$, and 78.33 $\mu g/m^3$, respectively. Furthermore, the $PM_{2.5}$ concentrations in kitchens burning dung cake, fuel wood, and methane were 117.41 $\mu g/m^3$, 271.11 $\mu g/m^3$, and 46.96 $\mu g/m^3$, respectively,[29] with measured $PM_{2.5}$ concentrations far in excess of the United States Environmental Protection Agency's[30] standards for 24-hour average $PM_{2.5}$ of 65 $\mu g/m^3$.

Six articles were identified in scientific journals that reported associations between coal and biomass fuel combustion and asthma morbidity in households.[9,31–35] Indoor combustion of coal and biomass fuels has been found to affect the development and exacerbation of asthma. In the US, 508 adults aged $\geq$40 years participated in a study that evaluated indoor usage of biomass fuels, using spirometry to assess participants' lung function. Participants exposed to cooking indoors with wood or coal for $\geq$6 months had an OR of 2.3 (1.1–5.0) for reporting current asthma, whereas no effect was found for heating indoors with wood or coal (OR, 0.8; CI, 0.4–1.8).[9]

The relationship between cooking smoke exposures and asthma was also examined in India's Second National Family Health Survey, which was a cross-sectional study (n = 38,595) of a representative sample of elderly persons ($\geq$60 years old) in India.[32] Those exposed to biomass fuels were compared with elderly people in homes using cleaner fuels. Exposure to combustion of biomass fuels was associated with a higher prevalence of asthma (OR, 1.59; 95% CI, 1.30–1.94). In Finland, Kilpeläinen et al[33] investigated the impact of exposure to wood stove heating on subsequent asthma in childhood among 10,667 university students aged 18–25 years. They found a significant negative association between childhood wood stove heating and allergic rhinitis or conjunctivitis in a univariate model (OR, 0.61; 95% CI, 0.61–0.91).

Although asthma is less prevalent among rural populations where biomass fuels are in common use, compared with their urban counterparts, it should not be assumed that smoke exposure is not deleterious in these areas. A cross-sectional study of disadvantaged women in rural Nepal found significant associations between biomass-smoke use and cough, phlegm, breathlessness, wheezing symptoms, and asthma ($p < 0.05$).[36] The prevalence of respiratory illnesses was higher in those living in mud and brick houses compared with those in concrete houses, and was therefore higher in people living in the hills and in rural areas, compared with those in flatter and urban areas.[36] A survey of cooking fuels and health status in China, performed by Peabody et al[37] reported that, in contrast to other fuel types, coal was associated with negative impacts on exhaled CO level, forced vital capacity (FVC), and lifetime prevalence of chronic obstructive pulmonary disease, as well as asthma, in rural households.

In contrast to other studies, one home study from the US found no association between gas stove use and FEV1, FVC or peak expiratory flow, or risk of self-reported cough, wheeze or other respiratory symptoms.[38]

Nitrogen dioxide

NO_2 is a major pollutant produced by combustion, and the major source of exposure is from household appliances fueled by gas, kerosene heaters, coal- and wood-burning stoves, and tobacco smoking. Previous studies indicated that indoor NO_2 exposure might increase bronchial reactivity in asthma patients and increase respiratory infections among the general population.[39,40]

Two journal articles reported NO_2 measurements in houses. Simoni et al[41] reported median NO_2 levels of 20 ppb in winter and 14 ppb in summer; the highest levels occurred in the kitchen in the winter (33 ppb) and summer (20 ppb) in Italian houses. Gilbert et al[42] reported NO_2 measurements from 96 homes in Quebec City, Canada and found concentrations ranging from 3.3–29.1 $\mu g/m^3$ [geometric mean (GM) 8.3 $\mu g/m^3$].

Behavioral and sociodemographic factors in homes and workplaces may have significant impacts on personal exposures to NO_2. In a cross-sectional study (n = 176) of randomly selected inhabitants (25–55 years old) in Finland, factors significantly associated with differences in exposures to NO_2 were home and work location, housing characteristics, traffic volume near home, season, keeping windows open at home, and ETS exposure, as well as the use of a gas stove.[43]

Women with mild-to-severe persistent asthma have been reported in communities exposed to indoor gas cooking in Singapore.[44] In this study, the frequency of cooking during a 2-week period was positively correlated with mean exposure to NO_2 (r = 0.529; p = 0.042), which was associated with a greater use of rescue bronchodilators (r = 0.597; p = 0.031), whereas it was negatively associated with peak expiratory flow rate variability (r = −0.512; p = 0.051) and respiratory symptom severity score (r = −0.567; p = 0.043), probably because of the masking effects of bronchodilator treatment.

Although there is increasing evidence that NO_2 may play a role in the pathogenesis of asthma, the underlying mechanisms are not clear. Bayram et al[45] observed that NO_2 modulated airway inflammation in asthma patients by increasing the release of inflammatory mediators from bronchial epithelial cells. Another study demonstrated that NO_2 was a proinflammatory air pollutant under the conditions of repeated exposure, and found that repeated exposure attenuated the changes in FEV1 and FVC after initial exposure in healthy non-smoking adults.[40]

Carbon monoxide

CO is an asphyxiant, and is known to bind to hemoglobin and interfere with the transport of oxygen. Certain vulnerable populations (e.g. the elderly people, fetuses, and asthmatic people) are particularly sensitive to elevated CO levels. The main indoor sources of CO are gas stoves, furnaces, wood stoves and fireplaces, and cigarette smoking, as well as vehicle exhaust from attached garages.

High levels of CO in homes are closely related to unclean fuel combustion (e.g. biomass fuels) used for heating or

cooking.[46] Park and Lee[47] measured CO in rural residential areas of Costa Rica; when a wood-burning stove was used, average CO concentrations were between 0.5 ppm and 3.3 ppm. Naeher et al[48] measured CO during breakfast, lunch, and dinner in three high-density and four low-density villages in Guatemala. The highest indoor concentrations of CO were observed in homes using open fires (mean, 22.9 ppm and standard deviation, 28.1 ppm), reaching a maximum of >250 ppm, which is far in excess of the United States Environmental Protection Agency's[30] 8-hour average CO standard of 9 ppm or 10 $\mu g/m^3$.

Remarkably few studies to date have evaluated the relationship between indoor CO exposure and asthma morbidity among adults in households.[49,50] In China, Pan et al[50] observed harmful effects of CO levels on pulmonary function in farmers in Anhui Province, China.

Volatile organic compounds

The World Health Organization's[51] definition of VOCs includes all organic compounds (substances made up of predominantly carbon and hydrogen) with boiling temperatures in the range of 50–260°C, excluding pesticides. VOCs, toxic gases or vapors emitted at room temperature from certain solids or liquids, involve a variety of chemicals [e.g. formaldehyde (FA), benzene, and perchloroethylene], some of which may have acute or/and chronic adverse effects. Cigarette smoke, cleaners, floor/wall coverings, paints, and room deodorizers are major sources of indoor VOCs.

Three articles were found in scientific journals with reported measurements of VOCs.[52–54] Arif and Shah[54] reported VOC measurements in households in the US where the GM concentration varied from as low as 0.03 $\mu g/m^3$ for trichloroethene to as high as 14.33 $\mu g/m^3$ for toluene. Levels of indoor toluene measured in Erfurt and Hamburg in Germany were 37.3 $\mu g/m^3$ and 20.5 $\mu g/m^3$, respectively, with a significant difference in levels between the two cities ($p < 0.0001$).[53] Hodgson and Levin[52] assessed the VOC levels in indoor air in North America and reported that average indoor concentrations of some toxic air contaminants such as 1,1,1-trichloroethane have decreased since 1990.

Acute symptoms resulting from the indoor VOC exposure have been reported, including eye and upper respiratory irritation, rhinitis, wheezing, breathlessness, and asthma. Several previous studies have identified relationships between various health outcomes among adults and the indoor VOC exposure. In the US, 550 participants exposed to 10 VOCs collected as part of the National Health and Nutrition Examination Survey 1999–2000 were studied.[54] Adults exposed to aromatic compounds had ORs of 1.63 (95% CI, 1.17–2.27) for doctor-diagnosed asthma, and 1.68 (95% CI, 1.08–2.61) for wheezing attacks.

Various VOCs are known to have adverse effects on pulmonary health, but most studies have reported the impacts of individual VOCs, instead of taking account of their combined effects on health.[54,55] In a recent study in France, 1012 inhabitants (aged ≥15 years) from 490 dwellings were quantitatively evaluated by an IAQ Observatory. Among the adults, asthma (8.6%) was significantly associated with N-undecane and 1,2,4-trimethylbenzene levels, and rhinitis (38.3%) was associated with ethylbenzene, trichloroethylene, and m/p-and o-xylene. The VOC levels in homes were associated with prevalence of asthma and rhinitis in adults in concentration-dependent manners.[56]

Formaldehyde

Acute exposure to FA is highly irritating to the eyes, nose, and throat, resulting in coughing and wheezing symptoms. Further exposure may induce severe allergic reactions of the skin and respiratory tract.[57] The World Health Organization's recommended maximum level for FA in indoor environments is 100 $\mu g/m^3$ in 30 minutes.

Six publications measured FA in houses.[42,58–61] Guo et al[58] reported that the 24-hour average FA concentrations in 37 homes exceeded the good class of the Hong Kong IAQ Objectives (<100 $\mu g/m^3$). Indoor FA and styrene levels in Hong Kong were higher than in other East Asian cities, reflecting the fact that homes in Hong Kong were more affected by household products and materials. FA levels were also measured in another study of 96 homes in Quebec City, Canada. The GM FA concentration for these households was 29.5 $\mu g/m^3$, ranging from 9.6–90.0 $\mu g/m^3$.[42]

Hodgson et al[59] measured FA concentrations in four newly-built houses for a period of 2–9.5 months after completion in North America. FA concentrations were below or near 50 ppb with a GM value for all houses of 40 ppb. Weng et al reported a mean concentration of total carbonyl compounds in residential indoor air in Hangzhou, China of 222.6 $\mu g/m^3$ in summer, which was higher than in winter (68.5 $\mu g/m^3$). Multiple regression analysis indicated that carbonyl concentrations in air depend on the age of the decorations and furniture, as well as on their concentrations in outdoor air.[61]

Several studies focused on the relationship between higher indoor concentrations of FA and asthma morbidity, and produced controversial results.[62–64] Matsunaga et al[62] studied 998 pregnant women in Osaka, Japan and found that exposure to FA of ≥47 ppb was positively associated with an increased prevalence of atopic eczema (OR, 2.25; 95% CI, 1.01–5.01), especially among women with a negative familial allergic history. In another study, Casset et al[63] found that exposure to low levels of FA (<100 $\mu g/m^3$) significantly enhanced bronchial responsiveness to mite allergen in mite-sensitized adult asthmatics.

An additional study, however, found no association between FA exposure and asthma morbidity.[64] In a double-blind crossover study conducted in Paris, France, 12 adults with intermittent asthma and allergy exposed to FA levels as low as 500 $\mu g/m^3$ showed no deleterious effects on airway allergen responsiveness, whereas a trend toward a protective effect was detected.

Biological contaminants

Numerous studies have shown that biological contaminants can be present in the environment. Biological contaminants are frequently detected in homes, but the levels of exposure vary considerably. Biological reservoirs include dust mites, molds and other fungi, bacteria, and human, pet and

pest (e.g. cockroaches, rats) products enhanced by damp conditions, as well as microbial products such as endotoxins, microbial fragments, peptidoglycans, and various allergens. The term "biological contaminants" usually refers to a variety of agents from biological sources found in indoor environments, including bacteria (including endotoxins from bacteria) and fungi (which may contain allergens, toxins, and irritants). Fungi and dampness or mold are usually considered as risk factors for atopy in the home environment.

Exposure to allergens from dust mites [*Dermatophagoides farinae 1* (*D farinae 1*), *Dermatophagoides pteronyssinus* (*D pteronyssinus*)], cats [*Felis domesticus 1* (*F domesticus 1*)], dogs [*Canis familiaris 1* (*C familiaris 1*)], cockroaches [*Blattella germanica 1* (*B germanica 1*), *B germanica 2* (*B germanica 2*)], and mice [*Mus musculus 1* (*M musculus 1*), Mouse urinary protein], as well as to molds in homes are important risk factors for asthma development and exacerbations. Measured values of biological contaminants can vary significantly depending on the sampling techniques and analytical methods used. Antibody-based enzyme-linked immunosorbent assays have been used to quantify allergen concentrations in most scientific studies.

Indoor exposure to dust mite allergens

House dust mites (HDM) are notable sources of inhalant allergens in many parts of the world. Several studies have shown higher concentrations of HDM allergens in residential environments.[65–69] The reported range of cat and dog allergens in house dust ranged from 0.11–24 μg/g. Trakultivakorn et al measured HDM allergens (*D pteronyssinus, D farinae 1*) in collected dust from mattresses and living room floors from 35 Chiang Mai homes, and found GMs of *D pteronyssinus, D. farinae 1*, and total Group I allergens in dust from mattresses and living room floors of 8.61 μg/g, 2.88 μg/g, and 15.81 μg/g and 1.61 μg/g, 0.27 μg/g, and 2.43 μg/g, respectively.[66]

Gross et al[67] subsequently reported that concentrations of HDM allergens were significantly higher on lower floors (2–8 times higher, *D pteronyssinus, D farinae 1*), in older mattresses (3–13 times higher, *D pteronyssinus, D farinae 1*), in post-war buildings (6 times higher, *D pteronyssinus*), in houses without central heating (2 times higher, *D pteronyssinus*), in old carpets (3 times higher, *D pteronyssinus*), and when there was a dog in the house (3 times higher, *D farinae 1*). Nam et al,[69] however, reported lower *D pteronyssinus* levels for floors and mattresses, with GMs of 0.11 μg/g (range: 0.01–4.05) and 0.14 μg/g (range: 0.01–30.0), respectively. Corresponding levels of *D farinae 1* were higher, at 7.46 μg/g (range: 0.01–262.9) and 10.2 μg/g (range: 0.01–230.9) for floors and mattresses, respectively. Furthermore, Su et al[68] reported that 65% of selected mattresses in Taiwan had *D pteronyssinus* levels above 2 μg/g. Most airborne fungal concentrations found in homes were higher than the recommended levels.

Indoor exposure to cat and dog allergens

Cats and dogs are the most popular pets in most parts of world, but keeping these animals at home is associated with a high prevalence of allergic sensitization. Four studies measured cat and dog allergens in home environments,[65,70–72] and elevated levels of *C familiaris 1* and *F domesticus 1* were detected in both dust and air samples. Allergen levels showed high variability between houses, with a reported range of 0.1–200 μg/g.

Arbes et al[71] measured allergens in dust collected from beds, bedrooms floors, living room floors, and living room sofas from 831 US homes. *C familiaris 1* and *F domesticus 1* were detected in 100% and 99.9% of homes, respectively; though only 49.1% of homes had resident animals. Averaged over the sampled sites, GM concentrations (μg/g) were 4.69 for *C familiaris 1* and 4.73 for *F domesticus 1*. Among homes with an indoor dog or cat, GM concentrations were 69 μg/g for *C familiaris 1* and 200 μg/g for *F domesticus 1*, respectively. Among the homes with no indoor pet, GM concentrations were >1.0 μg/g.

In the US, floor dust in houses was analyzed for cat and dog allergens, which ranged from <0.002 ng/m^3 to >5 ng/m^3. Airborne endotoxin levels were significantly lower in homes with a cat, compared with homes with a dog ($p < 0.001$), and were significantly correlated with airborne Can f 1 ($r = 0.50$, $p < 0.01$), but not Fel d 1.[72] In another study, dog allergen concentrations were higher in homes with dogs compared with those without dogs. Mean concentrations of cat allergen were 0.1 μg/g.[65]

Indoor exposure to cockroach and mouse allergens

Cockroach and mouse allergens are ubiquitous in many homes at levels that might contribute to asthma morbidity. Previous studies have examined cockroach and rodent allergen levels in residential environments. GM concentrations of mouse allergens varied from 0.94.6 μg/g in a US study.[73] In another study, detectable levels of mouse allergen were found in 82% of US homes ($n = 831$), and kitchen floor concentrations exceed 1.6 μg/g.[74] Elevated cockroach allergen levels have also been detected in cities in the US[74,75] and some other countries.[76,77] A recent study demonstrated that cockroaches were found in 77% of 324 apartments and apartments with high *B germanica 2* levels had a 1.7-fold increased OR for having an asthmatic resident (95% CI, 1.2–2.3).[75] Furthermore, in a Chinese study, cockroach allergens were detected in 93% of households ($n = 107$) and were higher in living room samples compared with bedding samples. Cockroach allergen levels were higher during winter than in summer.[76]

Indoor exposure to fungal allergens

Fungal allergen levels are generally higher in settled house dust. Exposure to fungal allergens has usually been estimated using fungal culture methods. One study examined indoor fungal spore levels in houses in Mexico,[78] and the most frequently cultured fungi were *Cladosporium* spp. (76%), *Alternaria* spp. (57.5%) and *Aspergillus* spp. (51.3%), whereas another study in Iran identified *Cladosporium* spp. (29.2%), *Aspergillus* spp. (19.0%), and *Penicillium* spp. (18.3%) as the most common indoor fungi.[79]

Indoor exposure to endotoxin

Asthma morbidity is highly prevalent in both urban and rural areas, and its prevalence has been closely related to bacterial endotoxins. In the United States, Roy et al[80] quantified bacterial DNA and endotoxin contents in dust from various geographic locations. The highest bacterial DNA levels were measured in farm barns (mean, 22.1 μg/g dust), followed by rural homes (6.3 μg/g), and farm homes (2.2 μg/g) and urban homes (0.6 μg/g), respectively. Bischof et al[81] measured endotoxin concentrations in settled house dust collected from 405 randomly selected homes in two German cities. The mean concentration of 2.274 μg endotoxin/g dust in living rooms was about two orders of magnitude below mean occupational exposure. Endotoxin concentrations were higher in older buildings (means ratio, 1.52, 95% CI, 1.14—2.04), and in the lower story of the dwelling (means ratio, 1.30, 95% CI, 1.04—1.62).

Relationships between asthma and asthma-related symptoms and exposure to indoor biological contaminants in homes

Numerous studies have shown that indoor allergens are causally related to asthma and asthma-related symptoms.[26,80,82—90] Biological contaminant exposure has clear implications for sensitization and clinical asthma through the development of bronchial hyper-responsiveness and airway inflammation in sensitized subjects. Because people in modern society spend most of their time in residential environments, homes may be important sources of indoor biological contaminant exposure. Indeed allergen levels in homes, which include a variety of potential allergen reservoirs (e.g. bedding, mattresses, carpeting, and upholstered furniture), may be higher than in occupational settings.

Numerous studies have reported that exposure of sensitized asthmatics to high levels of allergens caused worsening of pulmonary function.[82—84] A recent study found that many asthma triggers (specifically mold) were as prevalent or more so in the homes of New Yorkers with asthma, compared with control households. A direct association was identified among the presence of mold (adjusted OR, 2.5; 95% CI, 1.8—3.4), air cleaners (adjusted OR, 2.2; 95% CI, 1.7—2.8), humidifiers (adjusted OR, 1.4; 95% CI, 1.1—1.8), and current asthma.[88] Another study in the United Kingdom demonstrated that adults both sensitized and exposed to high levels of sensitizing allergens had significantly lower predicted FEV1% (mean, 83.7% *vs.* 89.3%; $p = 0.03$), higher FeNO values (GM, 12.8 ppb *vs.* 8.7 ppb; $p = 0.001$), and more severe airway reactivity (PD_{20} GM, 0.25 μg *vs.* 0.73 μg; $p < 0.001$) compared with unsensitized and unexposed individuals.[89] A recent Swedish study examined if exposure to indoor allergens was associated with allergic sensitization prevalence. The study identified a positive association between asthma symptoms and cat allergen levels (OR, 1.53; 95% CI, 1.04—2.24), whereas levels of viable molds were significantly associated with increased bronchial responsiveness. The lower prevalence of allergic sensitization in Reykjavík may be partly related to lower indoor allergen exposure.[10] However, a cohort study ($n = 245$) in New York found no association between sensitization to indoor allergens and increased asthma morbidity in inner-city adults.[90]

It is possible that the farming environment and rural lifestyle might be associated with an unknown "protective farming" effect in terms of prevalence of asthma morbidity.[80,86,87] In a study of Norwegian farmers, farmers had lower risks of allergic rhinitis, atopic sensitization, and sensitization against pollen and mites (ORs: 0.63; 0.86; 0.51; and 0.80, respectively) compared with urban residents. Exposure to endotoxins and fungal spores appears to have a protective effect against atopic asthma, but not nonatopic asthma, in farmers.[86] Roy et al[80] reported that DNA from farm barn dust augmented the immunomodulatory effects of endotoxin and may, together with other microbial components, mitigate allergy and asthma development.

Several studies have suggested that poor IAQ caused by moisture (or dampness) and mold problems, might be related to asthma morbidity among adults.[26,84,85] A recent meta-analysis examined the association between indoor dampness and mold contamination and adverse health effects. Building dampness and mold were associated with increases of approximately 30—50% in a variety of respiratory and asthma-related health outcomes.[26] Another study in Canada found that the prevalence of chronic wheeze, wheeze with shortness of breath, and allergy were higher in women who exposed to damp housing compared with those without exposure to damp housing ($p < 0.05$).[85]

Although previous studies suggest that exposure to aeroallergens in homes may significantly affect pulmonary health, the underlying mechanisms responsible for the observed health effects are not well understood.

Summary and conclusions

Exposure to indoor aero contaminants in residential environments is common. Available measurements of indoor pollutants in homes suggest that asthma-related indoor pollution exists in many homes. A review of the literature shows that levels of aero contaminants vary greatly in terms of physical (e.g. temperature and humidity), structural (e.g. housing quality) and behavioral factors (e.g. cleaning, smoking, and pet holding). It is clear that indoor aero contaminants are potent triggers of asthma and asthma-related symptoms in homes. Numerous studies of indoor environments generally suggest a relationship between exposure to indoor aero contaminants and asthma morbidity in adults. The levels of indoor aero contaminants from coal and biomass combustion, microbes, and tobacco use exceeded exposure thresholds in most studies and were associated with asthma morbidity. These indoor risk factors suggest that some measures should be put in place to reduce asthma morbidity in the adult population.

A variety of studies have strongly suggested that widespread environmental inadequacies in homes present potential risk factors for asthma and asthma-related symptoms among adults. Home environments might thus be

major sources of aero contaminants in terms of adult indoor exposure. Further studies are therefore needed to determine the indoor environmental aero contaminant load in homes and its effect on adults.

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