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Human exposure to indoor air pollutants in sleep microenvironments: A literature review

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ABSTRACT

We spend approximately one-third of our lives sleeping, yet little is known as to how human exposure to indoor air pollutants during sleep impacts human health and sleep quality. This paper provides a literature review of the current state-of-knowledge pertaining to human inhalation and dermal exposures while sleeping. An analysis of the duration of sleep exposure periods is provided, demonstrating that the sleep microenvironment is the predominant indoor space where humanity spends most of its time. Mattress dust is found to contain a diverse spectrum of biological particles and particle-bound chemical contaminants and their concentrations in dust can span many orders of magnitude among bed samples. These dust particles can become airborne through particle resuspension associated with body movements in bed. Mattress foam and covers, pillows, and bed frames can emit a variety of volatile and semi-volatile organic compounds, including phthalate plasticizers and organophosphate flame retardants, and emission rates can increase due to localized elevations in surface temperature and moisture near the bed due to close contact with the human body. This literature review demonstrates that human exposures to mattress-released pollutants can be amplified due to the source-proximity effect inherent to the sleep microenvironment, where the human body and breathing zone are in close and intimate contact with potential pollutant sources for prolonged periods. Given the findings of this review, human exposures to indoor air pollutants in the sleep microenvironment should receive more attention and future research is needed to fully understand how sleep exposures affect human health and sleep quality.

Contents

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1. Introduction

Many particulate and gaseous pollutants that are potentially health hazardous originate from indoor sources and concentrations are often much higher indoors than outdoors [133]. Since people spend about 80–90% of their time indoors [77,113,137], human exposures in the indoor environment are often substantially greater than exposures occurring outdoors. Across the world, adults typically sleep 8–9 h per day [46,77,114,159], virtually always indoors, which corresponds to one-third of their lifetime, making the sleep microenvironment particularly important in contributing to both their acute and chronic exposures to indoor pollutants. Mattresses, pillows, bedding materials, and bed frames are possible pollutant sources unique to sleep microenvironments. People are likely to be exposed to elevated concentrations of various chemical contaminants and biological particles since they are in close and intimate contact with these items. Early-life exposures to mattress-released pollutants are a particular concern, given the extended sleep periods of infants (12–14 h per day) and their low body weights [30,50,85,86].

The sleep microenvironment can be defined as the space encompassing a mattress, pillow, bedding materials, bed frame and the volume of air above these items that includes an individual's breathing zone (BZ) and buoyant thermal plume [15,16,88]. The seemingly innocuous sleep microenvironment can be home to a diversity of pollutants that have been shown to impact human health. Mite and animal allergens, human- and animal-associated bacteria, fungi, and semi-volatile organic compounds (SVOCs) can accumulate in settled dust on mattresses, pillows, and bed sheets (see references in Table 1). The materials used to manufacture mattresses and bedding products, such as polyurethane foam and vinyl mattress covers, are possible sources of a myriad of chemical contaminants, including volatile organic compounds (VOCs), plasticizers, flame retardants, and unreacted (free) isocyanates (NCO) (see references in Table 2).

Measuring indoor air pollutant concentrations at a reference location in the bulk air of a room may not be sufficient to characterize indoor exposures (e.g. Ref. [180]). This discrepancy, which is known as the source-proximity effect, is mainly due to the non-uniform distribution of pollutants that arises due to source location, the buoyant human thermal plume, occupant movements, and the overall airflow pattern and mixing conditions within a space [29,43,60,128,130,131]. The source-proximity effect is an important characteristic of the sleep microenvironment, where the human body and BZ are in close proximity to potential pollutant sources for extended periods of time. Therefore, people may be exposed to elevated concentrations of pollutants via inhalation and dermal pathways as they sleep. Research on the underlying physical processes governing the source-proximity effect of the sleep microenvironment is limited.

Human exposure in sleep microenvironments is important, but has not been extensively researched in contrast to exposures in other types of indoor environments, such as classrooms, kitchens, and occupational workplaces. Therefore, it offers challenging research opportunities to advance our understanding of the pollutants commonly found in sleep microenvironments, the mechanisms by which pollutants are transported around the human body to an individual's BZ, pollutant concentrations and exposure levels that individuals experience while sleeping, and the total amount of pollutants that are inhaled or absorbed via dermal exposure. This knowledge is important to investigate subsequent health effects and to develop strategies to promote healthy bedrooms. As a key step in the advancement of this knowledge, we provide a literature review of the current understanding of human exposure in sleep microenvironments, including: exposure characteristics, a summary of the biological and chemical composition of mattress dust, an overview of chemical emissions from mattresses and bedding materials, the potential for elevated exposures due to the source-proximity effect, and the use of personalized ventilation and air cleaning systems for beds.

2. Sleep exposure characteristics

2.1. Exposure pathways

Inhalation exposure can be defined as the contact between an agent (pollutant) and a target (person) [179]. The contact between a pollutant and a person occurs at an exposure point, which we can define as a person's BZ. The inhalation exposure concentration can be defined as the mass of a pollutant (e.g. # of particles or μg of a VOC) in a person's BZ divided by the BZ volume. The volume of the BZ is variable, has been shown to be dependent on the transport dynamics of a specific pollutant e.g. Ref. [97], and is likely influenced by physical obstructions, such as a pillow for a person sleeping in the prone position [89]. However, it is generally defined as a hemisphere with a radius of 30–50 cm, with its center of origin at the mouth or the nose [72,132]. An expression for the time-integrated inhalation exposure, $E_{i,j}$, of a specific pollutant, i, in a particular microenvironment, j, is:

$$E_{i,j} = \int_{t_0}^{t} C_{BZ,i,j}(t)\ dt \quad (1)$$

where is $C_{BZ,i,j}(t)$ is the BZ concentration at time t, although time-averaged BZ concentrations are commonly reported. Two key variables thus define inhalation exposures: the amount of pollutant that enters the BZ and the length of time over which continuous contact occurs between the pollutant and the BZ.

In addition to inhalation exposure assessment, it is necessary to evaluate transdermal uptake of chemical pollutants (e.g. SVOCs) in the sleep microenvironment, given the extended periods of contact between human skin and vinyl mattress covers, pillow protectors, and assorted bedding materials (bed sheets, blankets, duvet covers). Dermal exposures while sleeping can occur via both direct contact transfer with these items and air-to-skin uptake [169], the latter of which is linked to concentrations of gas-phase pollutants released from the mattress. Dermal exposure via direct contact transfer ($E_{Dermal\ Contact}$) can be calculated using Equation (2):

$$E_{Dermal\ Contact} = C_s \cdot SA \cdot TE \cdot AF \quad (2)$$

where C_s is the surface concentration of a pollutant, SA is the contact area, TE is the efficiency of contact transfer, which represents the fraction of the surface pollutant that is transferred to the skin during a contact event, and AF is the fractional absorption factor. Although contact transfer has been well studied, numerical values of TE are difficult to measure accurately and thus remain poorly characterized. The value of TE can be influenced by the chemical compound, physical nature of the bedding surfaces, contact pressure and motion during sleep, sleeping posture, surface concentration of the SVOC, and temperature and humidity (e.g. Ref. [182]). Simulated experiments with full-size human thermal manikins could be used to determine empirical TEs for sleeping people. AF is typically an empirical quantity that is assumed to be a fixed value for a specific chemical regardless of exposure conditions. Although the AF approach has been widely adopted, Frasch et al. [183] discussed its potential limitations and concluded that loading, evaporation or sublimation of VOCs, and experimental duration, may have strong impacts on the fraction absorbed by skin.

Dermal exposure due to direct air-to-skin transport has received attention recently [9,167,169,170,175,181]. The transdermal uptake of a gas-phase pollutant ($E_{Dermal\ Gas}$) can be estimated using Equation (3):

$$E_{Dermal\ Gas} = C_{gas} \cdot k_{p\_g} \cdot BSA \quad (3)$$

where C_{gas} is the gas-phase concentration of a pollutant in proximity of the human body, k_{p_g} is the transdermal permeability coefficient, and BSA is the body surface area. Weschler and Nazaroff [169,181] demonstrated that the abundance of SVOCs on hand wipe samples can be predicted reasonably well from gas-phase concentrations using this expression. They developed equations to estimate k_{p_g} based on the knowledge of physical and chemical properties of indoor pollutants, such as molecular weight (MW), octanol-water partition coefficient (K_{ow}), and Henry's constant, and extended their analysis of transdermal penetration to approximately eighty VOC and SVOC compounds.

2.2. Sleep exposure period

The exposure period can be defined as the time of continuous contact between a pollutant and a person [179]. It is the magnitude of this period that makes the sleep microenvironment particularly important in contributing to human inhalation and dermal exposures to various pollutants originating in mattresses, pillows, and bedding.

The U.S. Environmental Protection Agency (EPA) provides data on the duration of time spent (h/day) in a sleep or nap activity (EPA Exposure Factors Handbook (EFH) 2009). To investigate the dependence of sleep duration on age, we plotted the EFH's data, as shown in Fig. 1. During the first few months to years of life, infants (<1 year of age) and toddlers (1–3 years of age) spend a considerable amount of time sleeping, with an average of 13.3 h/day in the first year of life, 12.6 h/day in the second year, and 12.1 h/day in the third year, also reported in Refs. [37,70]. The data suggests that sleep microenvironments may play a critical role in characterizing exposures of very young children to indoor air pollutants. As expected, sleep duration continues to decline with age through much of adulthood, with an average of 8.2 h/day for the U.S. mean age group of 37 years [184]. An inflection is observed around 50 years of age, after which, sleep duration begins to steadily rise with age to an average of 9.2 h/day for people older than 81 years of age.

For adults, it is also constructive to evaluate how the time spent in the sleep microenvironment compares to other microenvironments in which people commonly spend their time. The National Human Activity Pattern Survey (NHAPS) Study conducted by Ref. [77] surveyed over 9000 adults (>18 years of age) in the U.S. on their activity patterns, finding that Americans spent nearly 87% of their time indoors. The NHAPS data can be used along with the EFH data set to estimate the average percentage of time an adult in the U.S. spends in sleep microenvironments. We assumed that the time spent in the sleep or nap activity (mean of 8.2 h for adult of mean age in the U.S.), as defined by the U.S. EPA, is spent entirely in a person's bedroom, on their mattress or similar bedding, and in their personal residence. As shown in Fig. 2, the modified activity pattern data shows us that adults spend about 34% of their day in the sleep microenvironment, which equates to about 50% of the time they spend in a residence, and 39% of the time they spend indoors. Assuming the “in a residence, other activity” location category can be partitioned among several different microenvironments, such as living areas, kitchen, office, and bathroom, the sleep microenvironment becomes the predominant indoor space where American adults spend most of their time. Furthermore, the percentage of time spent in sleep microenvironments is likely an underestimate, given that the U.S. EPA EFH data is limited to time spent sleeping or napping, and does not include other activities common to this indoor space, such as reading a book, watching television in bed, or using a smartphone/tablet.

Time-activity pattern studies conducted outside of the U.S. have reported similar trends in time spent sleeping. Among Canadian adults, 36% of the day is spent sleeping or napping, predominately in one's residence [92]. In Europe, adult sleeping time varies by country, but is generally between 8 and 9 h/day: Belgium: 8.4 h/day; Bulgaria: 9.1 h/day; Estonia: 8.4 h/day; Spain: 8.6 h/day; France: 8.9 h/day; Italy: 8.3 h/day; Latvia: 8.7 h/day; Lithuania: 8.5 h/day; Poland: 8.5 h/day; Slovenia: 8.4 h/day; Finland: 8.5 h/day; United Kingdom: 8.4 h/day; and Norway: 8 h/day [46]. Adults in Australia and New Zealand spend 8.5 h/day sleeping; in China adults sleep 9 h/day; while in Japan and the Republic of Korea, adults sleep less than 8 h/day [114]. Furthermore, there is additional variability in the time spent indoors and in the home among different countries due to differences in climate and culture, e.g. Refs. [113,137,177]. Notably, Koreans spend 59% and 67% of their time indoors on weekdays and weekends, respectively, much less than North Americans and Europeans [177].

2.3. Exposure of infants and toddlers during sleep: the impact of body weight

Because of their low body weight, infants and toddlers inhale considerably more air per kg of body weight as they sleep compared to adolescents and adults. The volume of air inhaled/kg-day can be estimated with the U.S. EPA EFH data set by taking the product of the mean normalized volumetric breathing rate in the sleep or nap activity (L/h-kg) and the mean duration of time spent in the sleep or nap activity (h/day). The normalized inhaled air volumes, V^*_{Sleep}, are categorized by age group and gender and presented in Fig. 3. It is apparent that infants and toddlers inhale nearly an order of magnitude more air per body mass than adults. During the first and second years of life, they inhale approximately 300 L/kg-day, which decreases to about 244 L/kg-day during the third year of life. Between 21 and < 31 years of age, adults breathe about 30 L/kg-day.

The analysis above helps to understand how the dose of an inhaled pollutant is considerably different between very young children and adults. The daily inhalation intake dose, $D_{i,j}$, (mass of pollutant inhaled per body weight per day) of pollutant i in microenvironment j can be calculated as:

$$D_{i,j} = \frac{\int_{t_0}^{t} Q_{B,j}(t) \times C_{BZ,i,j}(t)dt}{BW} \quad (4)$$

where $Q_{B,j}(t)$ is the volumetric breathing rate (L/h) and BW is body weight (kg). This expression can be simplified for the sleep microenvironment and by assuming time-averaged BZ concentrations and breathing rates. Additionally, the normalized inhaled air volumes during a sleep period, V^*_{Sleep}, can be substituted into the equation to obtain:

$$\overline{D_{i,Sleep}} \cong \frac{\overline{Q_{B,Sleep}} \times t_{Sleep} \times \overline{C_{BZ,i,Sleep}}}{BW} \cong V^*_{Sleep} \times \overline{C_{BZ,i,Sleep}} \quad (5)$$

Thus, to estimate the daily sleeping dose, one can simply take the product of V^*_{Sleep}, as calculated from the U.S. EPA EFH data set, and the average BZ concentration of a pollutant originating in the sleep microenvironment. Therefore, if a very young child (an infant or toddler) and an adult are exposed to the same BZ concentration of a pollutant released from a mattress, the child-normalized dose will be an order of magnitude greater than that of the adult. Similarly, to compare an infant's dose of dermally absorbed pollutants to that of an adult, we can use the skin surface area to body mass ratio (BSA/BW), as presented in Fig. 4. Infants have BSA/BW three times greater than adults, emphasizing the importance of early-life dermal exposures, particularly during sleep periods.

3. Biological and chemical composition of mattress dust

Mattresses, pillows, and bedding materials serve as an accumulation zone for a diverse spectrum of biological particles and particle-bound chemical contaminants. Table 1 provides an overview of the biological and chemical composition of mattress dust in selected field studies (n = 52). These studies are often aimed at identifying associations between the microbial and allergenic content of mattress dust with the development of atopic and allergic diseases in infants and children, e.g. Refs. [21,27,80,125,146,154,160]. Mattress dust is commonly sampled as a surrogate for human exposure in large cohort studies. However, air sampling, particularly near the BZ, may provide more valuable insight in characterizing sleep inhalation exposures, e.g. Refs. [57,102,122,134], as dust levels are not necessarily correlated with concentrations in the air. However, field data on BZ concentrations during sleep is limited.

3.1. House dust mite and animal allergens

Biological matter in mattress dust is comprised of a wide range of organisms and their associated allergens. Among house dust mite (HDM) allergens, Der p 1, Der f 1, Der p2, and Blo t 5, have been identified in many regions of the world across a range of climate zones. The high moisture levels and plentiful supply of desquamated skin particles, or squames, makes the sleep microenvironment an ideal environment for HDMs to flourish. For example, in Brazil, da Silva et al. [35] detected 400-1000 HDM bodies (eggs, larvae, nymphs, and adults) per g of mattress dust. Additionally, cat (Fel d 1), dog (Can f 1), cockroach (Bla g 2), and mouse/rat urinary allergens have also been detected, along with allergens originating outdoors (Ph1 p 5, timothy pollen). Concentrations of these allergens can range over several orders of magnitude, from <1 to > 10^3 μg/g, as shown in Table 1.

The type of mattress and bedding materials has been shown to influence levels of HDM allergens. Van den Bemt et al. [161] reported higher levels on inner spring mattresses, compared to polyester and latex, and waterbeds. In Thailand, [163] found varying levels Der p 1 and Der f 1 in different mattresses, with kapok showing the highest concentrations, followed (in decreasing order) by synthetic, coconut, and polyurethane sponge. Der p 1 levels also tend to increase with mattress age [107,161]. In addition, the frequent use of wool, synthetic, and sheepskin bedding materials was found to be associated with higher levels of Der p 1 allergens [51,107]; and pillow dust can also be a source of Der p 1, Der f 1, and Blo t 5 allergens [172]. Finally, differences in HDM allergen levels have also been observed between the mattresses of mothers and their infants [26], between rural and urban residences [44], before and after home renovation [200], as well as variations among the seasons [150].

Additional studies have reported elevated levels of airborne HDM allergens during sleep periods, suggesting that deposited HDM particles can be released into the air via human-induced resuspension, e.g. Refs. [16,123,141]. Gore et al. [57] measured airborne Der p 1 allergen exposure via nasal air sampling and estimated inhalation exposures of 6 ng/night and 2 μg/year during sleep periods. Sakaguchi et al. [134] reported airborne Der p 1/Der f 1 and Der 2 concentrations of 223 and 87.1 pg/m^3, respectively, in the air directly above a mattress. These levels were found to be a factor of ten greater than those measured in the living room air of the same residence, which may suggest significant resuspension of settled HDM particles from mattresses. However, a recent study has suggested that beds may not be the primary site of HDM exposure [156].

3.2. Fungi

A wide variety of fungal genera and species are commonly identified in mattress dust, including *Penicillium* spp., *Cladosporium* spp., *Aspergillus* spp., *Aspergillus fumigatus* spp., *Alternaria* spp., *Eurotium* spp., *Epicoccum*, and yeasts, among many others (Table 1). Along with dust, many fungal species have been found within mattress and pillow foam, with early studies by Refs. [31,33] recognizing the impact of fungal contamination of pillows on human health. Concentrations of fungal species are typically reported as CFU (colony forming units) per g of mattress dust or number of cells per g of mattress dust, with concentrations in the range of 10^3–10^4 CFU/g and < 10^4 to > 10^7 cells/g, respectively. Studies also report levels of (1–3)-β-Glucan, a component of the cell wall of many fungi (also found in some bacteria) and a common microbial

Table 1
Biological material and chemical contaminants detected in mattress dust in selected field studies ($n = 52$).

Study	Description	Location of Field Study	Detected Biological Material or Chemical Contaminant	Mattress Dust Concentration	Units	Mattress Dust Load, m_0 (g/m^2)
1. Garrett et al. (1998) [51]	Evaluated the impact of bedding characteristics on HDM concentrations	Latrobe Valley, Victoria, Australia	Der p 1	**Wool bedding:** Always: 30.6 (24.8–37.8) Sometimes: 23.6 (20.0–27.7) Never: 11.2 (8.6–14.6) **Type of mattress:** Inner-spring: 25.0 (21.9–28.6) Foam: 13.0 (8.9–19.1)	μg/g mattress dust (Mean (95% CI))	Not reported
2. Douwes et al. (1998) [38]	Relationship between house characteristics and levels of endotoxin and (1–3)-ß-D-Glucan	Erfurt and Hamburg, Germany	Endotoxin	Hamburg: 300 Erfurt: 4200	EU/m^2 mattress area sample (Mean)	0.528 (Hamburg) 1.075 (Erfurt)
			(1–3)-ß-D-Glucan	Hamburg: 317 Erfurt: 862	μg/m^2 mattress area sampled (Mean)	(Mean)
3. Fahlbusch et al. (2000) [47]	Quantified indoor grass pollen allergens in house dust	Hamburg, Erfurt, Zerbst, Bitterfeld & Hettstedt, Germany	Phl p 5	<0.03 (<0.03–0.120)	μg/g mattress dust (Median (25th-75th percentiles))	Not reported
4. Stubner et al. (2000) [149]	Examined the impact of home remediation interventions on lowering allergen levels in low-income households	Birmingham, Alabama, USA	Various allergens (e.g. Der p 1, Der f 1, Fel d 1)	**Home 1, Mattress:** 15 (before intervention), <2 (after intervention) **Home 2, Mattress:** 20 (before intervention), 4 (after intervention) **Home 3, Mattress:** 235 (before intervention), 16 (after intervention) **Home 4, Mattress:** 840 (before intervention), – (after intervention) **Home 5, Mattress 1:** 630 (before intervention), 240 (after intervention) **Home 5, Mattress 2:** 565 (before intervention), – (after intervention) **Home 6, Mattress:** 5 (before intervention), – (after intervention)	μg/mg mattress dust	Not reported
5. Hirsch et al. (2000) [200]	Measured HDM and mold spore concentrations before and after installation of windows and central heating systems	Dresden, Germany	Der f 1	**Before renovation:** 1.56 (0.89–2.74) **After renovation:** 2.40 (1.33–4.36)	μg/g mattress dust (Mean (95% CI))	Not reported
			Cladosporium spp.	**Before renovation:** 130 (50–370) **After renovation:** 60 (20–160)	CFU/g mattress dust (Mean (95% CI))	
			Penicillium spp.	**Before renovation:** 1500 (740-3020) **After renovation:** 1250 (590-2660)		
			Alternaria spp.	**Before renovation:** 10 (0–20) **After renovation:** 10 (10–30)		
			Aspergillus spp.	**Before renovation:** 590 (270-1320) **After renovation:** 820 (400-1710)		
			Aspergillus fumigatus spp.	**Before renovation:** 10 (0–10) **After renovation:** 10 (10–20)		

6. Douwes et al. (2000) [39]	Investigated the relationship between endotoxin, β-Glucans, and HDM allergens in mattress dust with peak expiratory flow in children	Amsterdam, Netherlands	Endotoxin	**Non-symptomatics:** 1820 ± 3.6 (level), 4772 ± 2.8 (load) **Symptomatics:** 2082 ± 4.3 (level), 5696 ± 3.6 (load) **Asthmatics:** 1202 ± 3.3 (level), 3983 ± 1.8 (load) **Cough:** 3402 ± 4.7 (level), 7670 ± 4.3 (load)	Level: EU/m^2 mattress area sampled Load: EU/g mattress dust (Mean ± SD)	Not reported
			(1−3)-ß-D-Glucan	**Non-symptomatics:** 276 ± 2.4 (level), 718 ± 1.8 (load) **Symptomatics:** 293 ± 2.3 (level), 792 ± 2.1 (load) **Asthmatics:** 283 ± 1.9 (level), 903 ± 1.8 (load) **Cough:** 303 ± 2.6 (level), 683 ± 2.3 (load)	Level: µg/m^2 mattress area sampled Load: µg/g mattress dust (Mean ± SD)	
			Der p 1	**Non-symptomatics:** 537 ± 6.1 (level), 1440 ± 6.1 (load) **Symptomatics:** 432 ± 7.5 (level), 1147 ± 6.4 (load) **Asthmatics:** 318 ± 9.0 (level), 960 ± 7.8 (load) **Cough:** 646 ± 5.9 (level), 1481 ± 5.9 (load)	Level: ng/m^2 mattress area sampled Load: ng/g mattress dust (Mean ± SD)	
7. Su et al. (2001) [150]	Measured seasonal concentrations of HDM allergens and endotoxin	Southern Taiwan	Der p 1	**Spring:** 3.78, **Summer:** 5.61, **Fall:** 9.11, **Winter:** 10.15	µg/g mattress dust (Mean)	Not reported
			Der p 2	**Spring:** 1.41, **Summer:** 1.49, **Fall:** 1.96, **Winter:** 3.32		
			Endotoxin	**Spring:** 45.18, **Summer:** 17.96, **Fall:** 50.25, **Winter:** 18.84	ng/mg mattress dust (Mean)	
8. Pitten et al. (2001) [119]	Investigated the impact of mattress encasings on fungal growth	Germany	**Fungal genera and species identified in dust on mattresses with cotton encasings:** *Alternaria* spp., *Aspergillus* spp., *Candida* spp., *Cladosporium cladosporioides*, *Mycelia sterilia*, *Penicillium* spp., *Rhodotorula* spp., *Scopulariopsis* brevicaulis, *Scopulariopsis* spp., *Sirodesmium-like fungus*, *Trichoderma* spp., *Ulocladium* spp. **Fungal genera and species identified in dust on mattresses with synthetic encasings:** *Aspergillus* spp., *Cladosporium cladosporioides*, *Mycelia sterilia*, *Penicillium* spp., *Scopulariopsis* brevicaulis, *Scopulariopsis* spp.			Not reported
9. Oppermann et al. (2001) [117]	Measured levels of HDM allergens and fungi in children's mattresses	East & West Germany	Der f 1	1.36	µg/g mattress dust (Mean)	Not reported
			Der p 1	1.14		
			Common fungal species identified: *Penicillium* spp., *Eurotium* spp.,, *Aspergillus* spp.,, *Alternaria* spp.,, *Epicoccum* spp.,, *Cladosporium* spp.	**Total fungi:** 26,500 (1400 −300,000)	CFU/g mattress dust (Mean (Range))	
10. Mihrshahi et al. (2002) [107]	Measured HDM allergens in residential homes and investigated the impact of mattress and bedding characteristics on concentrations	Sydney, Australia	Der p 1	**Type of mattress:** Foam: 21.39 (13.77−33.25) Inner spring: 14.22 (12.58−16.07) **Mattress Age:** ≤2 years: 10.31 (8.03−13.24) >2 years: 15.47 (13.47−17.67) **Wool blankets:** Yes: 27.18 (21.06−35.08) No: 12.88 (11.38−14.57) **Synthetic blankets:** Yes: 22.18 (17.51−28.11) No: 13.21 (11.64−14.99) **Sheepskin:** Yes: 22.95 (14.37−36.66) No: 13.84 (12.31−15.56)	µg/g mattress dust (Mean (95% CI))	Not reported

(*continued on next page*)

Table 1 (*continued*)

Study	Description	Location of Field Study	Detected Biological Material or Chemical Contaminant	Mattress Dust Concentration	Units	Mattress Dust Load, m_0 (g/m^2)
11. Sidenius et al. (2002) [138]	Distribution of HDMs and their allergens on different surfaces in houses occupied by patients with newly diagnosed allergy to HDM allergen and previously reported high Der 1 concentrations in mattresses	Copenhagen, Denmark	Der 1	86 (30–288) μg/g; 88 (12-1910) mites/g. Note: 100% of samples from mattresses exceeded 10 μg/g; 37.5% of samples from pillows and duvets exceeded 10 μg/g	μg/g mattress dust; mites/g mattress dust (Median)	Not reported
12. Jovanovic et al. (2004) [73]	Measured indoor fungi levels in homes of children with and without a history of allergies	Southwest Germany	Common fungal species identified: *Aspergillus* spp., *Penicillium* spp., *Alternaria* spp., *Cladosporium* spp.	**Total viable mold spores:** 16,250/38,238 (0–2,500,000)	CFU/g mattress dust (Median/Mean (Range))	Children with allergic history: 0.393 Controls: 0.403 (Median)
13. Gehring et al. (2004) [52]	Measured endotoxin concentrations in the mattress dust of mothers and their infants	Munich & Leipzig, Germany	Endotoxin	**Mothers' mattress:** Munich: 2000 (level), 3000 (load) Leipzig: 2200 (level), 3100 (load) **Infants' mattress:** Munich: 900 (level), 4900 (load) Leipzig: 1000 (level), 6600 (load)	Level: EU/m^2 mattress area sampled Load: EU/g mattress dust (~Mean)	Infants' mattress: ~0.2 Mothers' mattress: ~0.7
14. El Sharif et al. (2004) [44]	Measured concentrations of HDM allergens, pet allergens, and endotoxin in mattress dust	Ramallah, Palestine	Der p 1	**City residences:** 958 (load), 4290 (level) **Village residences:** 1941 (load), 3776 (level) **Camp residences:** 3100 (load), 5656 (level)	Load: ng/g mattress dust Level: ng/m^2 mattress area sampled (Mean)	City residences: 0.29 ± 0.20 Village residences: 0.59 ± 0.31 Camp residences: 0.62 ± 0.36
			Der f 1	**City residences:** 48 (load), 206 (level) **Village residences:** 33 (load), 64 (level) **Camp residences:** 24 (load), 42 (level)		
			Can f 1	**City residences:** 100 (load), 451 (level) **Village residences:** 99 (load), 192 (level) **Camp residences:** 164 (load), 298 (level)		
			Fel d 1	**City residences:** 91 (load), 408 (level) **Village residences:** 210 (load), 408 (level) **Camp residences:** 151 (load), 275 (level)		
			Endotoxin	**City residences: -** (load), 18,738 (level) **Village residences:** 19,214 (load), 25,700 (level) **Camp residences:** 23,972 (load), 43,696 (level)	Load: EU/g mattress dust Level: EU/m^2 mattress area sampled (Mean)	
15. Instanes et al. (2005) [71]	Measured concentrations of allergens and endotoxin in mattress dust in day-care centers	Oslo, Norway	Fel d 1 Can f 1	1.5 (load), 0.17 (level) 10.0 (load), 1.7 (level)	Load: μg/g mattress dust Level: μg/m^2 mattress area sampled (Median)	Not reported

			Endotoxin	5.0 (load), 0.9 (level)	Load: ng/g mattress dust Level: ng/m^2 mattress area sampled (Median)	
16. da Silva et al. (2005) [35]	Investigated mite fauna in mattress dust	Londrina, Brazil	HDM bodies: total mites	**Crib:** 404.5 ± 183.3 **Bed:** 1075.8 ± 198.8	Mite bodies/g mattress dust (Mean ± 95% CI)	Not reported
			HDM bodies: Pyroglyphidae mites	**Crib:** 289.9 ± 136.7 **Bed:** 875.0 ± 183.6	Note: bodies represent all life stages: eggs, larvae, nymphs, and adults.	
			Der p 1	**Crib:** 5.8 ± 2.7 **Bed:** 17.5 ± 3.7	μg/g mattress dust (Mean ± 95% CI, estimate)	
17. Hicks et al. (2005) [63]	Analyzed fungal flora and concentrations in settled dust	Northern California, USA	Culturable fungi	62,300 (37,300–104,000)	CFU/g bedspread and furniture dust (Mean (95% CI))	Not reported
			Cladosporium *Penicillium* spp. Yeasts *Aureobasidium* *Aspergillus niger* Nonsporulating fungi *Alternaria* *Epicoccum*	22,000 (ND-180,000) 4000 (ND-120,000) 8800 (NS-200,000) 4300 (ND-68,000) 400 (ND-56,000) 800 (ND-1400) 1800 (ND-12,000) 800 (ND-12,000)	CFU/g bedspread and furniture dust (Median (Range))	
18. van den Bemt et al. (2006) [161]	Evaluated the influence of mattress characteristics on HDM concentrations	Netherlands	Der p 1	**Type of mattress:** Latex: 1.191 (0.744–1.908) Waterbed: 0.433 (0.073–2.559) Polyester: 1.199 (0.592–2.431) Inner spring: 1.418 (0.594–3.387) **Upper layer material:** Cotton: 0.801 (0.343–1.873) Synthetic: 2.606 (1.527–4.448) Cotton/synthetic: 1.382 (0.885–2.158) Other: 0.447 (0.111–1.792) **Mattress age:** ≤5 years: 0.986 (0.517–1.883) >5 years: 1.190 (0.731–1.939) **Relative humidity in bedroom:** ≤50%: 0.796 (0.444–1.426) >50%: 1.975 (1.312–2.974)	log μg/g mattress dust (Mean (95% CI))	Not reported
19. Woodcock et al. (2006) [171]	Identified fungal flora in used (18 months to >20 years) synthetic and feather pillows	United Kingdom	*Penicillium* spp. *Aspergillus vitus* *Rhodotorula mucilaginosa* *Aspergillus glaucus* *Scopulariopsis brevicaulis*	15,100 27,800 69,400 41,700 13,900	CFU/g pillow dust Mean	Not reported
			Aspergillus fumigatus	**Synthetic pillows:** 2745 **Feather pillows:** 1863	CFU/g pillow material (not dust) Mean	
			Aureobasidium pullulans	**Synthetic pillows:** 1926 **Feather pillows:** 5110		
20. Krämer et al. (2006) [80]	Relationship between eczema and exposure to HDM allergens	Augsburg, Germany	Der p 1 Der f 1	0.68 (1.08, 16.0) 0.79 (1.3, 22.0)	μg/g mattress dust Mean (Median, Maximum)	Not reported
21. Chen et al. (2007) [26]	Explored associations between socioeconomic status and the concentrations of indoor bio-contaminants	Munich & Leipzig, Germany	Der p 1	**Mothers' Mattress:** 97.7 (5–608.3) **Infants' Mattress:** 5.0 (5.0–642.9)	ng/g mattress dust (Median (25th-75th percentiles))	Mothers' Mattress: 0.7385 (0.453–1.166)
			Der f 1	**Mothers' Mattress:** 634.1 (125.4–3601.8) **Infants' Mattress:** 525.1 (5.0–2324.7)	ng/g mattress dust (Median (25th-75th percentiles))	

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Table 1 (*continued*)

Study	Description	Location of Field Study	Detected Biological Material or Chemical Contaminant	Mattress Dust Concentration	Units	Mattress Dust Load, m_0 (g/m^2)
			Fel d 1	**Mothers' Mattress:** 180.2 (68.6–820.5) **Infants' Mattress:** 326.0 (84.7–1322.5)	ng/g mattress dust (Median (25th-75th percentiles))	Infants' Mattress:
			Endotoxin	**Mothers' Mattress:** 3008 (1045.5–7913) **Infants' Mattress:** 5866 (2336–14,669)	EU/g mattress dust (Median (25th-75th percentiles))	0.188 (0.120–0.289)
22. Krop et al. (2007) [82]	Assessed the presence of occupational laboratory animal allergens in mattress dust of workers	Netherlands	Rat urinary allergen	**Laboratory animal workers:** 39.3 (19.8–78.0) **Non-laboratory animal workers (controls):** 7.6 (4.7–12.2)	ng/g mattress dust (Median (Range))	Not reported
			Mouse urinary allergen	**Laboratory animal workers:** 29.5 (11.7–74.6) **Non-laboratory animal workers (controls):** 8.8 (4.6–16.8)		
			Mouse urinary protein	**Laboratory animal workers:** 30.9 (12.8–74.8) **Non-laboratory animal workers (controls):** 5.6 (2.0–16.0)		
23. Giovannangelo et al. (2007) [54]	Measured levels of β-Glucans and fungal extracellular polysaccharide in mattress dust of children	Germany, Netherlands & Sweden	(1–3)-β-Glucan	**Germany:** 450 (150-1050) (level), 1050 (900-2050) (load) **Netherlands:** 400 (150-1000) (level), 1050 (750-3000) (load) **Sweden:** 350 (100-1000) (level), 225 (1050–4000) (load)	Level: μg/m^2 mattress area sampled Load: μg/g mattress dust (~Median (95% CI))	Germany: ~0.25 (0.01–1) Netherlands: ~0.25 (0.01–1) Sweden: ~ 0.15 (0.02–0.9) (~Median (Range))
			Fungal Extracellular Polysaccharide (EPS)	**Germany:** 10,000 (2000-50,000) (level), 40,000 (10,500–100,000) (load) **Netherlands:** 9000 (1050–45,000) (level), 40,000 (10,500–100,000) (load) **Sweden:** 2050 (400-10,500) (level), 15,000 (4000–50,000) (load)	Level: EPSU/m^2 mattress area sampled Load: EPSU/g mattress dust (~Median (95% CI))	
24. Rennie et al. (2008) [125]	Investigated associations between endotoxin levels and asthma and wheeze in children	Saskatchewan, Canada	Endotoxin	2069.79 (4650.02) (level), 7.99 (9.85) (load)	Level: EU/m^2 mattress area sampled Load: EU/mg mattress dust (Median (IQR))	0.365 ± 0.26 (Mean ± SD)
25. Nam et al. (2008) [110]	Measured HDM allergen concentrations in house dust	Cheonan, Korea	Der f 1	4.0 (0.01–112.1) (level), 10.2 (0.01–230.9) (load)	Level: μg/m^2 mattress area sampled	Not reported
			Der p 1	0.02 (0.01–8.8) (level), 0.14 (0.01–30.0) (load)	Load: μg/g mattress dust (~Median (Range))	
26. Korthals et al. (2008) [79]	Investigated the occurrence of *Listeria* spp. bacteria in mattress dust of farm children	Bavaria, Germany	*Listeria* spp.	Detected in 8% of mattress dust samples (Culture methods)		Not reported
			L. monocytogenes	Detected in 3% of mattress dust samples (Culture methods) Detected in 8% of mattress dust samples (Real-time PCR methods)		
			L. innocua	Detected in 8% of mattress dust samples (Culture methods)		
27. Vogel et al. (2008) [164]	Identified microbial flora on the mattresses of farm children	Bavaria, Germany	*Bacillus* spp.	5.25 (0.87)	log. CFU (Median (IQR))	Not reported
			Coliform bacteria	5.67 (0.94)		
			Lactobacillus spp.	3.00 (3.30)		
			Enterococcus spp.	3.15 (4.47)		
			Clostridium spp.	0.00 (3.00)		
			Aspergillus spp.	4.00 (1.10)		
			Mucor spp.	3.75 (1.00)		
			Cladosporium spp.	0.00 (3.74)		

			Penicillium spp.	3.00 (4.00)		
			Wallemia spp.	3.48 (4.00)		
28. Soleimani and Rafinejad (2008) [139]	Analyzed HDM contamination in hotels and inns	Bandar-Abbas, Iran	**HDM species:** *D. pteronyssinus* (identified in 84.5% of samples), *D. farinae* (identified in 13% of samples), *C. malaccensis* (identified in 2.5% of samples)			Not reported
29. Heyes et al. (2009) [62]	Measured (1−3)-β-Glucan levels in pillows, duvets, and mattresses	Wellington, New Zealand	(1−3)-β-Glucan	**Mattress:** 15.7 (10.4−23.9) (level), 76.6 (61.4−94.0) (load) **Duvets:** 8.8 (5.4−14.1) (level), 132.1 (68.9−207.9) (load) **Pillows:** 3.5 (2.5−4.8) (level, μg pillow), 132.1 (68.9−207.9) (load) **Synthetic Pillows:** 3.8 (1.4−11.0) (level, μg/pillow), 105.4 (76.9 −144.6) (load) **Feather Pillows:** 2.0 (0.8−5.1) (level, μg/pillow), 76.6 (36.7 −159.9) (load)	Level: μg/m^2 mattress/pillow/duvet area sampled Load: μg/g mattress/pillow/duvet dust (Mean (95% CI))	Not reported
30. Wu et al. (2009) [172]	Measured allergen and microbial bio-contaminant concentrations in homes of asthmatic children	Central Taiwan	Der p 1	**Pillow:** 1.33 (0.91−1.94) **Bamboo side of mattress:** 0.38 (0.25−0.58) **Conventional side of mattress:** 0.77 (0.51−1.17)	μg/g mattress/pillow dust (Mean (95% CI))	Not reported
			Der f 1	**Pillow:** 0.22 (0.14−0.33) **Bamboo side of mattress:** 0.24 (0.14−0.44) **Conventional side of mattress:** 0.55 (0.30−1.01)		
			Blo t 5	**Pillow:** 0.014 (0.011−0.017) **Bamboo side of mattress:** 0.024 (0.017−0.033) **Conventional side of mattress:** 0.165 (0115−0.237)		
			Total HDM allergen	**Pillow:** 2.72 (1.99−3.73) **Bamboo side of mattress:** 1.35 (0.95−1.91) **Conventional side of mattress:** 3.50 (2.65−4.62)		
			Endotoxin	**Bamboo side of mattress:** 163.7 (82.6−244.8) **Conventional side of mattress:** 358.2 (211.7−504.8)	EU/mg mattress dust (Mean (95% CI))	
			β-Glucan	**Bamboo side of mattress:** 27.9 (22.4−34.8) **Conventional side of mattress:** 25.5 (21.9−29.8)	μg/g mattress dust (Mean (95% CI))	
31. Chen et al. (2009) [27]	Investigated associations between infant exposure to endotoxins and development of eczema	Munich & Leipzig, Germany	Endotoxin	**Mothers' mattress:** 2071 (595-6919) (level), 3008 (1046−7913) (load) **Children's mattress:** 1015 (330-3022) (level), 5866 (2336 −14,669) (load)	Level: EU/m^2 mattress area sampled Load: EU/g mattress dust (Median (IQR))	Not reported
32. Täubel et al. (2009) [151]	Explored the origins of bacteria in house dust	Finland	Bacterial phyla and genera	**Gram-positive bacterial groups (phylum:genera) identified in mattress dust:** Actinobacteria: *Corynebacterium, Propionibacterium, Micrococcus* Firmicutes: *Staphylococcus, Lactobacillus, Streptococcus, Lactococcus* Deinococcus-Thermus: *Deinococcus* **Gram-negative bacterial phyla identified in mattress dust:** Proteobacteria: *Paracoccus, Aggregatibacter* Bacteroidetes: *Prevotella, Bacteroides*		Not reported

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Table 1 (*continued*)

Study	Description	Location of Field Study	Detected Biological Material or Chemical Contaminant	Mattress Dust Concentration	Units	Mattress Dust Load, m_0 (g/m^2)
			Endotoxin	1.4 ± 3.7 (load), 194.1 ± 5.8 (level)	Load: EU/mg mattress dust Level: EU/m^2 mattress area sampled (Mean ± SD)	
			Lipopolysaccharide (LPS)	0.031 ± 1.379 (load), 4.396 ± 1.944 (level)	Load: nmol/mg mattress dust Level: nmol/m^2 mattress area sampled (Mean ± SD)	
			Muramic acid	15.5 ± 1.7 (load), 2191.2 ± 3.3 (level)	Load: ng/mg mattress dust Level: ng/m^2 mattress area sampled (Mean ± SD)	
33. Eberlein et al. (2009) [42]	Measured endotoxin and allergen levels in hospitals and adjacent homes	Bavaria, Germany	Endotoxin	**Hospitals:** 321,720 (230,500−518,700) **Adjacent Homes:** 243,980 (156,520−399,020)	EU/g mattress dust (Median (IQR))	Not reported
			Der p 1	**Hospitals:** 0 (0−208) **Adjacent Homes:** 163 (0−391)	ng/g mattress dust (Median (IQR))	
			Der f 1	**Hospitals:** 409 (152−839) **Adjacent Homes:** 791 (320-2074)		
			Fel d 1	**Hospitals:** 663 (117-2426) **Adjacent Homes:** 1227 (261-2824)		
34. Spertini et al. (2009) [140]	Evaluation of the reduction in allergens in houses with improved energy performance	Lausanne, Switzerland	Der f 1	**Control Buildings (CB):** 954; **Low Energy Use Buildings (LEB):** 67	ng/g mattress dust (Median)	Not reported
			Der p 1	**CB:** 73; **LEB:** 51		
			Mite Group 2	**CB:** 15; **LEB:** 5		
35. Visitsunthorn et al. (2010) [163]	Measured HDM allergen concentrations in different types of mattresses	Bangkok, Thailand	Der p 1 + Der f 1	**Coconut mattress:** 0.96 (0.39−1.45) (month 1), 16.60 (0−33.7) (month 12) **Kapok mattress:** 1.44 (1.06−1.83) (month 1), 31.30 (17.40−42.80) (month 12) **Sponge (polyurethane foam) mattress:** 0.96 (0.23−1.75) (month 1), 12.90 (1.00−17.90) (month 12) **Synthetic mattress:** 1.30 (0.80−1.89) (month 1), 25.30 (12.10−32.50) (month 12)	µg/g mattress dust (Median (IQR))	Not reported
36. Doyen et al. (2011) [40]	Measured endotoxin concentrations in mattresses at time of birth of newborn and at 6 months of life	Belgium	Endotoxin	**At birth, mattresses in newborn's bedroom:** 17.6 (0.4−346.7) (Site A), 20.4 (0.8−26.3) (Site B) **6 months of life, mattresses in newborn's bedroom:** 79.6 (3.8−518.8) (Site A), 101.8 (6.5−634.4) (Site B)	EU/mg mattress dust (Median (Range)	Not reported
37. Leung et al. (2011) [93]	Measured allergen levels in settled dust in residences of asthmatic children	Hong Kong	Fel d 1	0.06 (0.03−0.10)	µg/g mattress dust (Median (IQR))	Not reported
			Bla g 2	24.8 (14.2−47.7)	ng/g mattress dust (Median (IQR))	
			Blo t 5	0.26 (0.14−0.79)	µg/g mattress dust (Median (IQR))	

38. Tischer et al. (2011) [153]	Investigated exposure of children to endotoxins, β-Glucans, and EPS mold components in house dust	Germany & Netherlands	Endotoxin	**German LISA & GINI Studies:** 3053 (1521–6015) (level), 12,222 (7379–21,337) (load) **Dutch PIAMA Study:** 2356 (1461–4208) (level), 10,608 (6550–17,366) (load)	Level: EU/m^2 mattress area sampled Load: EU/g mattress dust (Median (IQR))	German LISA & GINI Studies: 0.257 (0.139–0.471) Dutch PIAMA Study: 0.247 (0.148–0.366) (Median (IQR))
			(1–3)-ß-D-Glucan	**German LISA & GINI Studies:** 421 (238–865) (level), 1859 (1277–2396) (load) **Dutch PIAMA Study:** 380 (199–625) (level), 1662 (1135–2205) (load)	Level: μg/m^2 mattress area sampled Load: μg/g mattress dust (Median (IQR))	
			Extracellular polysaccharides (EPS)	**German LISA & GINI Studies:** 1008 (4458–25,904) (level), 40,792 (24,235–65,371) (load) **Dutch PIAMA Study:** 8257 (3890–17,310) (level), 34,696 (20,364–58,156) (load)	Level: EPSU/m^2 mattress area sampled Load: EPSU/g mattress dust (Median (IQR))	
39. Hsu et al. (2012) [68]	Explored associations between indoor phthalate concentrations and mold growth	Tainan, Taiwan	DEHP	**w/o visible mold:** 656.6 (459.4–980.9) **w/ visible mold:** 933.3 (580.0–1404.9)	μg/g mattress dust (Median (25^{th}-75th percentiles))	Not reported
			BBzP	**w/o visible mold:** 2.1 (1.0–6.4) **w/ visible mold:** 3.7 (1.0–6.1)		
			DBP	**w/o visible mold:** 16.6 (10.4–22.8) **w/ visible mold:** 20.2 (12.4–40.9)		
40. Ege et al. (2012) [45]	Identified environmental bacteria in dust on children's mattresses	Bavaria, Germany	**Bacterium identified which are related to a physician's diagnosis of asthma:** *Corynebacterium mycetoides*, *Zoogloea* sp., *Duganella* sp., *Aurantimonas ureolytica* sp., *Serratia marcescens*, *S. nematodiphila*, *Serratia* sp., *Pseudomonas uorescens*, *Corynebacterium tuberculostearicum*, *Gardnerella vaginalis*, *Lactobacillus curvatus*, *Lactobacillus sakei*, *Streptococcus* sp., *Moraxella* sp., *Staphylococcus sciuri* sp., *Jeotgalicoccus* sp., *Salinicoccus* sp., *Macrococcus brunensis*, *Bacillus* sp. **Bacterium identified which are related to hay fever:** *Aerobacter ureolytica*, *P. fluorescens*, *Corynebacterium mucifaciens*, *C. freiburgense*, *C. variabile*, *C.* sp. *Triatoma infestans*, *Neisseria meningitidis*, *N. mucosa*, *N. subflava* **Bacterium identified which are related to atopic sensitization:** *Enterobacter cloacae*, *E. aerogenes*, *E. cancerogenus*, *E. ludwigii*, *Pantoea* sp., *Kluyvera cryocrescens*, *Erwinia persicina*, *Aurantimonas altamirensis*; *A. ureolytica*, *Mesorhizobium* sp., *Lactobacillus iners*, *Acinetobacter lwoffii*			Not reported
41. Barnig et al. (2012) [5]	Measured endotoxin concentrations in the dust of rural and urban residences	Strasbourg & Haut-Doubs, France	Endotoxin	**Type of farm:** Non-modern: 3700 ± 4900 Modern: 1200 ± 1600 **Barn position:** Adjacent to house: 3400 ± 4700 Separated to house: 2200 ± 3600 **Storage hay mode:** Round bales: 3200 ± 4700 In bulk: 1100 ± 900 Other: 2700 ± 3600	ng/g mattress dust (Mean)	Not reported
42. Begum et al. (2012) [7]	Evaluated the prevalence of fungi in used pillows	Perth, Australia	Fungi: *Alternaria* sp., *Aureobasidium* sp., *Cladosporium* sp., *Epicoccum* sp., *Monilia* sp., *Penicillium* sp., Yeast, *Rhizopus* sp.	**Total fungal counts:** 241 (22–563) (Yeast most prevalent)	CFU/pillow Mean (Range)	0.06–0.36 g pillow dust/pillow
43. Lawson et al. (2012) [91]	Measured endotoxin concentrations and loading in mattress dust	Humboldt, Saskatchewan, Canada	Endotoxin	**Children with asthma or wheeze (cases):** 19.6 (15.6–24.8)	EU/mg mattress dust (Mean (95% CI))	Not reported

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Table 1 (*continued*)

Study	Description	Location of Field Study	Detected Biological Material or Chemical Contaminant	Mattress Dust Concentration	Units	Mattress Dust Load, m_0 (g/m^2)
				Children without asthma or wheeze (controls): 21.1 (15.6–24.8)		
				Children with asthma or wheeze (cases): 240.5 (212.1–370.9)	EU/m^2 mattress area sampled (Mean (95% CI))	
44. Ali et al. (2012) [2]	Identified and quantified flame retardants in floor and mattress dust	Wellington, Wairarapa, Christchurch, & North Canterbury, New Zealand	OPFRs	**Children without asthma or wheeze (controls):** 376.2 (324.4–436.2)	TEP: 10 (<10–235), TnBP: 65 (20-1920), TCEP: 38 (<20–475), TCPP: 250 (133-1920), TBEP: 1545 (635-3610), TPhP: 240 (20–35,190), TDCPP: 103 (20-6465), TCP: 157 (<50-2155)	ng/g mattress dust (Median (Range))
			Not reported	HBCDs	α-HBCD: 41 (3–345), β-HBCD: 9 (<2–122), γ-HBCD: 80 (18-1260)	
				NBFRs	BTBPE: 1 (<2–37), TBB: 3 (<2 –40), TBPH: 1 (<2–50), DBDPE: 9 (<5–220)	
BFRs	BDE-47: 35 (2–290), BDE-99: 40 (8–540), BDE-183: 6 (<2–20), BDE-197: 4 (<2 –18), BDE-209: 735 (106-21,960)					
45. Wu et al. (2012) [173]	Assessed the impact of daily vacuuming of mattresses to reduce levels of HDM allergens, bacterial endotoxin and fungal β-Glucans	Taiwan	Der p 1	**Week 0:** 0.68 (0.32–1.40), **Week 8:** 0.46 (0.23–0.92)	μg/g mattress dust (Mean (95% CI))	1.5 (1.12–2) at Week 0, to 0.27 (0.18–0.41) at Week 8 (Mean (95% CI))
			Der f 1	**Week 0:** 1.18 (0.60–2.30), **Week 8:** 0.69 (0.41–1.14)		
			Endotoxin	**Week 0:** 91 (64–130), **Week 8:** 97 (75–125)	EU/mg mattress dust (Mean (95% CI))	
			β-Glucan	**Week 0:** 63.2 (46.1–86.6), **Week 8:** 75.1 (54.1–104.0)	μg/g mattress dust (Mean (95% CI))	
46. Gehring et al. (2012) [53]	Assessed the impact of mite-impermeable mattress covers on allergen exposure	Netherlands	Der f 1	**Placebo mattress cover:** 2849 ± 5.7	ng/g mattress dust (Mean ± SD)	Not reported
				Active mattress cover: 886 ± 3.5		
			Der p 1	**Placebo mattress cover:** 410 ± 4.7		
				Active mattress cover: 397 ± 3.5		
47. Casas et al. (2013) [23]	Analyzed the results of the European HITEA project (Health Effects of Indoor Pollutants: Integrating microbial, toxicological, and epidemiological approaches) with 4 European birth cohorts	Finland, Germany, Netherlands, & Spain	Endotoxin	**German LISA Study:** 23.0 (21.1 –25.1)	EU/mg mattress dust (Mean (95% CI))	Not reported
				Dutch PIAMA Study: 22.0 (19.8 –24.5)		
				Spanish INMA Study: 3.2 (2.7 –3.9)		
				Finnish LUKAS2 Study: 17.6 (15.8–19.5)		
			Fungal Extracellular Polysaccharide (EPS)	**German LISA Study:** 47.5 (43.7 –51.6)	EPSU/mg mattress dust (Mean (95% CI))	
				Dutch PIAMA Study: 30.7 (27.8 –33.8)		
				Spanish INMA Study: 116.7 (107.2–127.0)		
				Finnish LUKAS2 Study: 39.6 (34.1–46.0)		
			(1–3)-β-Glucan	**German LISA Study:** 0.9 (1.8–2.0)	μg/mg mattress dust (Mean (95% CI))	
				Dutch PIAMA Study: 1.7 (1.5 –1.8)		

				Spanish INMA Study: - **Finnish LUKAS2 Study:** 2.4 (2.2–2.7)		
48. Callesen et al. (2014) [21]	Evaluated associations between selected allergens and asthma, rhinoconjunctivitis, and atopic dermatitis in preschool children	Odense, Denmark	Der p 1	**Healthy Controls:** 110.0 **Asthma:** 132.2, **s-IgE -:** 145.4, **s-IgE +** : 132.0 **Rhinoconjunctivitis:** 119.4, **s-IgE -:** 164.9, **s-IgE +** : 104.8 **Atopic dermatitis:** 131.8, **s-IgE -:** 176.0, **s-IgE +** : 151.0	ng/g mattress dust (Median)	Not reported
			Der f 1	**Healthy Controls:** 977.2 **Asthma:** 988.4, **s-IgE -:** 471.8, **s-IgE +** : 1726 **Rhinoconjunctivitis:** 1,251, **s-IgE -:** 173.7, **s-IgE +** : 988.4 **Atopic dermatitis:** 751.9, **s-IgE -:** 672.4, **s-IgE +** : 1211		
			Der p 2	**Healthy Controls:** 121.5 **Asthma:** 119.5, **s-IgE -:** 69.2, **s-IgE +** : 135.5 **Rhinoconjunctivitis:** 153.4, **s-IgE -:** 158.1, **s-IgE +** : 130.4 **Atopic dermatitis:** 119.5, **s-IgE -:** 88.3, **s-IgE +** : 130.0		
			Fel d 1	**Healthy Controls:** 114.5 **Asthma:** 76.7, **s-IgE -:** 210.7, **s-IgE +** : 42.1 **Rhinoconjunctivitis:** 85.6, **s-IgE -:** 61.6, **s-IgE +** : 99.6 **Atopic dermatitis:** 150.4, **s-IgE -:** 170.9, **s-IgE +** : 116.6		
			Can f 1	**Healthy Controls:** 81.6 **Asthma:** 71.1, **s-IgE -:** 77.7, **s-IgE +** : 71.8 **Rhinoconjunctivitis:** 54.2, **s-IgE -:** 28.5, **s-IgE +** : 66.4 **Atopic dermatitis:** 53.0, **-IgE -:** 53.3, **s-IgE +** : 57.2		
49. Valkonen et al. (2015) [160]	Evaluated associations between bacterial exposures and atopy and asthma in children	Germany, Austria, and Switzerland	*Mycobacterium* spp.	**Asthma:** Farm Children (FC): 1,252,815, Exposed Non-Farm Children (ENFC): 738,266, Non-Exposed Non-Farm Children (NENFC): 326,199 **No asthma, but atopy:** FC: 872,049, ENFC: 583,315, NENFC: 360,554 **No asthma or atopy:** FC: 868,964, ENFC: 876,830, NENFC: 690,282	cells/m^2 mattress area sampled (Median)	Not reported
			Bifidobacteriaceae spp.	**Asthma:** FC: 7,853,091, ENFC: 4,933,515; NENFC: 4,337,218 **No asthma, but atopy:** FC: 6,882,662; ENFC: 2,054,042; NENFC: 3,317,576 **No asthma or atopy:** FC: 5,168,782; ENFC: 4,330,676, NENFC: 6,571,929		

(*continued on next page*)

Table 1 (*continued*)

Study	Description	Location of Field Study	Detected Biological Material or Chemical Contaminant	Mattress Dust Concentration	Units	Mattress Dust Load, m_0 (g/m^2)
			Clostridium cluster I	**Asthma:** FC: 264,510; ENFC: 32,028; NENFC: 31,438 **No asthma, but atopy:** FC: 131,510; ENFC: 30,665; NENFC: 14,547 **No asthma or atopy:** FC: 106,416; ENFC: 32,147; NENFC: 73,044		
			Clostridium cluster XI	**Asthma:** FC: 277,263; ENFC: 54,883; NENFC: 54,362 **No asthma, but atopy:** FC: 172,193; ENFC: 24,904; NENFC: 35,117 **No asthma or atopy:** FC: 271,469; ENFC: 54,771; NENFC: 90,370		
50. Tischer et al. (2015) [154]	Evaluated associations between bacteria and fungi concentrations and asthma and lung function	Iceland (Reykjavik), Sweden (Uppsala, Umea, Gothenburg), Estonia (Tartu), Germany (Hamburg, Erfurt), UK (Norwich, Ipswich), Belgium (Antwerp Centre, Antwerp South), France (Paris, Grenoble), Switzerland (Basel), Italy (Verona, Pavia, Turin), and Spain (Oviedo, Galdakao, Barcelona, Albacete, Huelva)	*Cladosporium herbarum*	64 (2; 3776)	cells/mg mattress dust (Median (Minimum; Maximum))	Not reported
			Penicillium spp./*Aspergillus* spp./*Paecilomyces variotii* group	39,340 (638; 261,600,000)		
			(1−3)-β-Glucan	0.87 (0.08; 7)	μg/mg mattress dust (Median (Minimum; Maximum))	
			Mycobacterium spp.	5413 (258; 524,700)	cells/mg mattress dust (Median (Minimum; Maximum))	
			Muramic acid	15 (1; 116)	μg/mg mattress dust (Median (Minimum; Maximum))	
			Gram-Positive bacteria	568,400 (16,480; 65,870,000)	cells/mg mattress dust (Median (Minimum; Maximum))	
			Gram-Negative bacteria	66,070 (1945; 6,900,000)		
51. Canbaz et al. (2016) [22]	Evaluated associations between organophosphate and polybrominated diphenyl ether flame retardants in dust and asthma	Sweden	OPFRs	**Asthmatic children:** TCEP: 102 (49−263), TCPP: 116 (60−243), TDCIPP: 141 (85−427), TBOEP: 3138 (1082−5593), TPhP: 163 (107−276), EHDPHP: 163 (107−276), mmp-TMPP: 192 (90−437), ΣOPFRs: 6449 (3718−12,865) **Control group:** TCEP: 107 (52−207), TCPP: 96 (44−362), TDCIPP: 162 (88−374), TBOEP: 3723 (1434−7069), TPhP: 631 (308-1493), EHDPHP: 172 (111−421), mmp-TMPP: 288 (102−790), ΣOPFRs: 6795 (3575−13,000)	ng/g mattress dust (Median (25th-75th percentiles))	Not reported
			BFRs	**Asthmatic children:** BDE-47: 11 (6−24), BDE-99: 14 (7−36), BDE-100: 2 (1−5), BDE-153: 7 (3−20), BDE-183: 3 (1−9), BDE-209: 118 (49−306), ΣBFRs: 259 (107−498) **Control group:** BDE-47: 11 (7−24), BDE-99: 13 (8−34), BDE-100: 3 (2−6), BDE-153: 6 (3−16), BDE-183: 3 (1−10), BDE-209: 122 (36−389), ΣBFRs: 239 (91-7910		

52. Stein et al. (2016) [146]	Assessment of asthma risk in Amish and Hutterite farm children	Indiana and South Dakota, USA	**Bacterial families with a relative abundance ≥ 2% in Amish or Hutterite dust, relative proportion in each group shown in parentheses:** Corynebacteriaceae (0.026 vs. 0.067), Micrococcaceae (0.045 vs. 0.04), Prevotellaceae (0.001 vs. 0.022), Bacillaceae (0.041 vs. 0.021), Staphylococcaceae (0.037 vs. 0.08), Lactobacillaceae (0.003 vs. 0.02), Streptococcaceae (0.022 vs. 0.065), Clostridiaceae (0.007 vs. 0.046), Lachnospiraceae (0.005 vs. 0.026), Ruminococcaceae (0.014 vs. 0.047), Bartonellaceae (0.347 vs. 0), Rhodobacteraceae (0.025 vs. 0.016), Sphingomonadaceae (0.033 vs. 0.042), Enterobacteriaceae (0.029 vs. 0.021), Moraxellaceae (0.032 vs. 0.026)	Not reported

Nomenclature: **HDM:** House Dust Mite; **CFU:** Colony Forming Units; **EU:** Endotoxin Units; **EPSU:** Fungal Extracellular Polysaccharide (EPS) Units; **PCR:** Polymerase Chain Reaction; **IgE:** Immunoglobulin E antibody; **CI:** Confidence Interval; **IQR:** Interquartile Range; **ND:** Not Detected; **DEHP:** Bis(2-ethylhexyl) phthalate; **BBzP:** Benzyl butyl phthalate; **DBP:** Dibutyl phthalate; **OPFRs:** Organophosphate flame retardants; **TEP:** tri-ethyl-phosphate; **TnBP:** tri-*n*-butyl-phosphate; **TCEP:** tris-(2-chloroethyl)-phosphate; **TCP:** tri-cresyl-phosphate; **TBEP:** tri-(2-butoxyethyl)-phosphate; **TCPP** tris-(1-chloro-2-propyl) phosphate; **TDCPP:** tris-(2,3-dichloropropyl)-phosphate; **TDCIPP:** tris(1,3-dichloroisopropyl) phosphate; **TBOEP:** tris(2-butoxyethyl) phosphate; **TPhP:** tri-phenyl-phosphate; **EHDPHP:** 2-ethylhexyldiphenyl phosphate; **mmp-TMPP:** meta, meta, para-tricresylphosphate; **BFRs:** Brominated flame retardants; **HBCDs:** Hexabromocyclododecanes: α-HBCD, β-HBCD, γ-HBCD; **NBFRs:** Novel Brominated flame retardants; **BTBPE:** 1,2-bis(2,4,6-tribromophenoxy)ethane; **DBDPE:** decabromodiphenyl ethane; **TBB:** 2-ethylhexyl-2,3,4,5-tetrabromobenzoate; **TBPH:** bis(2-ethylhexyl)-3,4,5,6-tetrabromophthalate; **Der p 1**: Dermatophagoides pteronyssinus Group 1 (European HDM); **Der p 2**: Dermatophagoides pteronyssinus Group 2 (European HDM); **Der f 1**: Dermatophagoides farinae Group 1 (American HDM); **Can f 1**: Canis familaris Group 1 (Dog allergen); **Fel d 1**: Felis domesticus Group 1 (Cat allergen); **Phl p 5**: Phleum pretense Group 5 (Common timothy pollen); **Bla g 2**: Aspartic protease Group 2 (Cockroach allergen); **Blo t 5**: Blomia tropicalis Group 5 (Blomia tropicalis HDM); **LISA Study**: Lifestyle Related Factors on the Immune System and the Development of Allergies in Childhood (Germany); **GINI Study**: German Infant Nutritional Intervention (Germany); **PIAMA Study**: Prevention and Incidence of Asthma and Mite Allergy (Netherlands); **INMA Study**: INfancia y Medio Ambiente (Environment and Childhood) (Spain); **LUKAS2 Study**: Lapsuuden kasvuympäristö ja allergiat 2 (Childhood environment and allergies) (Finland).

marker used to assess fungal exposure. Typical concentrations are in the range of $10–10^3$ μg/g of mattress dust. Mattresses and pillows are an ideal fungal culture medium, given the high moisture levels induced by human sweating (~100 L of sweat per year) and elevated temperatures (>30 °C) [36,171]. Fungi may consume HDM fecal matter and squames in mattress dust, providing them with necessary nutrients; and subsequently, HDMs may consume fungi, leading to a "miniature ecosystem" within mattresses and pillows, as suggested by Refs. [78,171].

Similar fungal species have been detected in mattress dust throughout the world, including the United Kingdom [171], Northern California, U.S. [63], Germany [73,119,164,200], and Australia [7]. Refs. [23,54,153] found small differences in (1–3)-β-Glucan levels in mattress dust in large cohort studies across several European countries. Additionally, the fungal composition of mattress dust can vary with bedding material, as observed by Ref. [119] for dust on cotton and synthetic mattress encasings. Higher concentrations of *Aspergillus fumigatus* and (1–3)-β-Glucans have been reported in synthetic compared to feather pillows [171,62].

3.3. Bacteria

The sleep microenvironment is also home to an array of bacterial phyla and genera, many of which are associated with human origins (skin, oral, intestinal/fecal, and genital) and specifically, the shedding of human skin, e.g. Refs. [45,66,151,154]. Common bacterium identified in mattress dust include: *Staphylococcus*, *Lactobacillus*, *Streptococcus* sp., *Lactococcus*, *Bacillus* sp., *Listeria* spp., *Zoogloea* sp., *Duganella* sp., *Aurantimonas ureolytica* sp., *Serratia marcescens*, *Pseudomonas fluorescens*, *Corynebacterium tuberculostearicum*, *Gardnerella vaginalis*, *Lactobacillus curvatus*, *Moraxella* sp., *Staphylococcus sciuri* sp., *Macrococcus brunensis*, *Aerobacter ureolytica*, *P. fluorescens*, *Triatoma infestans*, and *Neisseria meningitidis*, among a host of others. Concentrations can range from $< 10^6$ to $> 10^8$ cells per g of mattress dust (Table 1). Differences in the bacterial composition of mattress dust have been observed among Amish and Hutterite families in the United States [146] and among children from farming and non-farming households in Europe [160]. In addition to identifying specific bacterium, endotoxin concentrations are also commonly reported in mattress dust. Endotoxin is the biologically active lipopolysaccharide (LPS) of gram-negative bacteria and is a common marker to assess bacterial exposure (although it only represents defined subpopulations of bacteria and does not differentiate between human and non-human sources) [151] and may also serve as an indicator of inflammatory potential of settled dust [100]. Endotoxin levels are reported as endotoxin units (EU) per gram of mattress dust, and concentrations are typically in the range of 10^3 to 10^5 EU/g. Greater levels of bacterial endotoxin loading have been found in infant mattresses, compared to those of their mothers [26,52]. Furthermore, Doyen et al. [40] found endotoxin levels to increase in infants' bedrooms over the first 6 months of life. As with fungal (1–3)-β-Glucans, small variability in endotoxin levels have been found among large cohort studies in Europe [23,153].

3.4. Plasticizers and flame retardants

Due to their low volatility and high molecular weight, SVOCs, such as plasticizers and flame retardants, tend to accumulate in house dust, e.g. Refs. [69,167]. Although many field studies have measured the concentrations of various SVOCs in settled dust on flooring, only a few have explicitly reported levels in mattress dust. [68] detected several phthalate plasticizers in mattress dust in Taiwan, including DEHP, BBzP, and DBP (nomenclature listed in

Table 1). Concentrations are on the order of $10–10^2$ μg/g of mattress dust, with higher levels reported in the presence of visible mold on the mattress. [2,22] identified numerous flame retardants in mattress dust, including polybrominated diphenyl ethers (e.g. BDE 47, BDE 99, BDE 183, BDE 197, and BDE 209), organophosphates (e.g. TEP, TCEP, TCPP, and TDCPP, among others), HBCDs, and novel flame retardants (BTBPE, TBB, and TBH, among others). Concentrations for the various flame retardants ranged over several orders of magnitude, from <1 to 10^3 ng/g mattress dust. Plasticizers and flame retardants may be found as chemical additives in mattresses, especially infant crib mattresses [17,32,144]. It is likely that some of these compounds partitioned directly into settled mattress dust as they migrated from the underling mattress foam and encasing, whereas others may have originated from common indoor sources, such as vinyl flooring, foam furniture, and electronic casings, and then deposited (after partitioning to airborne particles) on the mattress and bedding.

3.5. Particle deposits on mattresses and bedding materials

The mass of dust that accumulates on mattresses, pillows, and bedding is an important parameter that can affect exposure and surface-to-air transport of the settled particles, e.g. Refs. [14,152]. Field studies that reported mattress dust loads are listed in Table 1, which are typically in the range of 0.1–1 g/m^2. These values are comparatively less than dust loads on indoor flooring materials, which are often between 1 and 10 g/m^2 [14]. Daily vacuuming of the mattress has been shown to reduce dust loading, from 1.5 g/m^2 initially, to 0.27 g/m^2 after 8 weeks of vacuuming [173].

There have been no published studies that have explicitly measured the size distribution of settled mattress dust, although it likely spans over four orders of magnitude, from sub-1 μm to >100 μm particles, given the diverse spectrum of biological and inorganic particles that can exist: allergen particles, fungal spores and fragments, bacterial cells and their agglomerates, squames, bedding fiber fragments, dust mite bodies (~200–300 μm), and particles originating elsewhere in the residence that have deposited on, or tracked-in to, the bed. Mite and animal allergens, such as Der p 1, Der f 1, Fel d 1, Bal g 2, Mus m 1 (mouse), and Rat n 1 (rat), are primarily carried on particles ~ 5–10 μm in size, although their size distribution can range from <1 to >20 μm [25,34,116,120]. [126] measured the size of various fungal species and found that the spores are typically in the range of 1–4 μm in aerodynamic diameter. Smaller fungal fragments less than 1 μm, are also common [127]. Górny et al. and Meklin et al. [58,105] reported the size distribution of various bacteria, finding aerodynamic diameters to range from 1 to > 7 μm. It is likely many single bacterial cells exist as larger agglomerates in settled dust, 2–4 μm in aerodynamic diameter, e.g. Ref. [11]. As discussed in Ref. [168]; human body sheds about 5×10^8 skin cells per day, a significant fraction of which will accumulate in mattress dust during extended sleep periods, with the average skin flake about $40 \times 30 \times 2$ μm in size.

4. Chemical emissions from mattresses, pillows, and bed/crib frames

Although there is a robust body of literature on compositional analysis of mattress dust, there is comparatively less research on characterizing emissions of chemical contaminants from mattresses, pillows, and bed/crib frames. Several recent studies have found that infant crib mattresses are a source of a variety of chemical additives, including VOCs, plasticizers, flame retardants, and unreacted isocyanates [15,17,95], which can enter the infant sleep microenvironment through volatilization as they partition from the material-phase to the gas-phase. Table 2 provides an overview of chemical contaminants detected in bedding products and their associated material-phase concentrations and area-specific emission rates (*SERs*) to air ($n = 14$).

4.1. Volatile organic compounds (VOCs)

The composition of a typical crib mattress includes a thick layer of polyurethane or polyester foam padding (inner-springs are also used) encased within a thin, waterproof plastic cover to protect the mattress foam and to provide an easy-to-clean surface. Studies by Refs. [3,4,15] have shown that infant crib mattresses and mattress covers are a source of a variety of VOCs, as presented in Table 2. Additionally, bed frames and wooden cribs may release formaldehyde, due to its presence in the resin of composite wood products used to construct the bed structure [100]. Low-VOC emitting bedding products can be found through numerous certification programs around the world, such as GREENGUARD [157], STANDARD 100 by OEKO-TEX® [115], CertiPUR-US® [24], and the Finnish M1 Emission Class for Building Materials [124].

4.2. Flame retardants

Crib mattresses manufactured with polyurethane foam are flammable and flame retardants are added so that they meet various flammability standards. To be effective, flame retardants are often present in the finished product at percent to tens-of-percent levels by weight [32,144,145]. Concerns over flame retardant additives in children's sleep products arose in the 1970s, when researchers demonstrated that children who wore sleepwear impregnated with the flame retardant tris(2,3-dibromopropyl) phosphate (tris-BP) were dermally exposed to the compound [13]. Throughout the next few decades, polybrominated diphenyl ether (PBDE) flame retardants, specifically pentaBDE congeners, were commonly added to crib mattress foam. Crib mattresses containing PBDEs may play a role in the elevated exposures that have been reported among infants relative to adults [61,155]. Concern over the negative health impacts of pentaBDE flame retardants led industry to end production in the U.S. in 2004. Although no longer found in new crib mattresses, these persistent compounds are still present in many older mattresses and can continue to volatilize and enter the indoor environment. This is especially important considering the long lifetime of a crib mattress (~10 years) and their common reuse in families with multiple children. Flame retardants currently added to crib mattresses as pentaBDE replacements include organophosphates, such as TDCPP, TCEP, and TCPP, and the Firemaster® 550 mixture [145]; Table 2. Recently, the State of California revised their residential flammability standard, Technical Bulletin 117 (TB-117), to require that all furniture, including mattresses for infants and adults, be sold with a label indicating the use of a chemical flame retardant additive (CA TB117-2013) [32].

4.3. Plasticizers

Most crib mattresses have a plastic cover for waterproofing and antibacterial purposes, and phthalates are extensively used as plasticizers to enhance the softness and flexibility of the cover. Because they are not chemically bound to the polymer matrix, they slowly volatilize from the material and migrate into surrounding environments [94,95,174,176,198]. Phthalate plasticizers identified in crib mattress covers include bis(2-ethylhexyl) phthalate (DEHP), bis(2-ethylhexyl) isophthalate (iso-DEHP), diisononyl phthalate (DINP) (Table 2).

Recently, the Consumer Product Safety Improvement Act was enacted in the U.S., restricting the use of certain phthalates (i.e., di-n-butyl-phthalate (DnBP), butyl-benzyl-phthalate (BBP), and

Table 2
Chemical contaminants detected in bedding products and their material-phase concentrations and area-specific emission rates (*SERs*) ($n = 14$).

Study	Bedding Product	Identified Chemical Contaminants in Product or Emissions to Air	Material-Phase Concentration in Product (mg/g)[a]	*SER* to Air (μg/m^2 h)
1. Anderson and Anderson (1999) [4]	Crib mattress covers	**Air:** toluene; 2,6-dimethyl-3-heptene; m-xylene; 1-methyl-cis-4-ethylcyclohexane; 1,3,5-trimethylbenzene; phenol; trichloroethylene; ethylbenzene; o-xylene; isopropylbenzene; diisobutylene	Not reported	Not reported
2. Anderson and Anderson (1999) [3]	Crib mattresses: foam and cover	**Air:** styrene; isopropylbenzene; limonene; ethylbenzene; 1,3-p-menthadiene; 1,3-dichlorobenzene; dipentene; 1,2,4-trimethylbenzene; nitrobenzene; 1-methyl-2-ethylbenzne; β-ocimene	Not reported	Not reported
3. Krone et al. (2003) [81]	Mattress pad foam	**Product:** total reactive isocyanate group **Product:** 2,4-toluene diisocyanate	19×10^{-3} 20.3×10^{-3}	Not reported
4. Kemmlein et al. (2003) [75]	Mattress Upholstery foam	**Air:** TCPP **Air:** TCPP	Not reported	23 °C: 0.012 23 °C: 77
5. Salthammer et al. (2003) [135]	Soft upholstery foam	**Air:** 1-chloro-2-propanol **Air:** 1,2-dichloropropane **Air:** TVOCs	Not reported	23 °C: 150 (24 h) 23 °C: 10 (24 h) 23 °C: < 10 (168 h)
6.-7. Hillier et al. (2003, 2009) [64,65]	Mattress foam	**Air (selected compounds):** dichloromethane; 2-methyl-2-propenal; hexane; 1-butanol; toluene; styrene; 1,3-dioxane; 1,4-dioxane; dodecane; DEP; methylcyclopentane; 3-methyl hexane; trimethylpentene; m-xylene; p-xylene; ethylbenzene; 3-methyl-nonane; limonene; ethanol; butanal; ethyl acetate; heptane; trichloroethylene; trimethyl-silanol; siloxane; tetrakis(trimethylsiloxy)silane; dodecamethyl; phenyl methyl ester formic acid; benzaldehyde; tetrachloroethene; 4-methyl-morpholine; 2,2,1,3-dioxolane; 3-methyl-pentane; 2-propenal; butanamine; n,n dimethylethanolamine; 1-chloro-2-propanol	Not reported	Not reported
8. Madsen and Gibson (2008) [100]	Crib	**Air:** formaldehyde	Not reported	Emission factors (μg/h per crib): 5–3680
9. Stapleton et al. (2009) [144]	Mattress pad foam Sofa bed foam Futon foam Pillow foam	**Product:** TDCPP **Product:** TDCPP **Product:** PentaBDE **Product:** TDCPP	12 13 5 28	Not reported
10. Stapleton et al. (2011) [145]	Infant sleep positioners Crib mattresses Nursing pillows	**Product:** TCEP, TCPP, TDCPP, V6 **Product:** TCEP, TCPP, TDCPP, V6, U-OPFR, TPP, TBB/TBPH, PentaBDE **Product:** TCEP, TCPP, TDCPP, V6	>1	Not reported
11. Vangronsveld et al. (2013) [162]	Flexible polyurethane foam	**Product and Air:** 2,4-toluene diisocyanate **Product and Air:** 2,6-toluene diisocyanate	11×10^{-6} 45×10^{-6}	n.d.
12. Boor et al. (2014) [15]	Crib mattresses: foam and cover	**Air:** phenol **Air:** isooctanol **Air:** neodecanoic acid **Air:** 2-ethyl-hexanoic acid **Air:** 3-methyl-1-heptanol **Air:** d-limonene **Air:** 2,6-bis(1,1-dimethylethyl)-4-(1-oxopropyl)phenol **Air:** (s)-3-ethyl-4-methylpentanol **Air:** linalool **Air:** nonanal	Not reported	23 °C: < 1–62 36 °C: 3–257 23 °C: < 1–6 36 °C: 4–7 23 °C: 3–22 36 °C: 9–40 23 °C: < 1–55 36 °C: 5–213 23 °C: 7–21 36 °C: 7–22 23 °C: 4–11 36 °C: 9–18 23 °C: 4–14 36 °C: 12–61 23 °C: 2–6 36 °C: 3–9 23 °C: 3–41 36 °C: 10–44

(*continued on next page*)

Table 2 (*continued*)

Study	Bedding Product	Identified Chemical Contaminants in Product or Emissions to Air	Material-Phase Concentration in Product (mg/g)[a]	*SER* to Air (μg/m^2 h)
				23 °C: < 1–5 36 °C: 2–10
		Air: decanal		23 °C: < 1–5 36 °C: 2–10
		Air: isopropyl myristate		23 °C: < 1–3 36 °C: 3–11
		Air: palmitic acid		23 °C: 2–10 36 °C: 12–43
		Air: 2-ethylhexanol		23 °C: 3–6 36 °C: 7–8
		Air: TVOCs		23 °C: 87.1 (new), 22.1 (used) 36 °C: 218.8 (new), 52.1 (used)
13. Liang and Xu (2014b) [95]	Crib mattress covers	**Air:** DINCH	Not reported	25 °C: 0.3 36 °C: 0.7
		Air: DEHA		25 °C: 0.4 36 °C: 4
14. Boor et al. (2015b) [17]	Crib mattresses: foam and cover	**Product:** DEHP	Cover: 104.4 (new), 21 (used) Foam: 1.7 (new), 5.5 (used)	Not reported
		Product: iso-DEHP	Cover: 314.5 (new), 60 (used) Foam: n.d. (new), 14.4 (used)	
		Product: DINP	Cover: 28.5 (new), 136.8 (used) Foam: n.d. (new), 22.3 (used)	
		Product: DINCH	Cover: 53.2 (new), 103.1 (used) Foam: 0.1 (new), 0.1 (used)	
		Product: DEHA	Cover: 4.8 (new), 31.1 (used) Foam: n.d. (new), 0.2 (used)	
		Product: PentaBDE	Foam: 7.46 (used)	
		Product: NCO	Not reported	

Nomenclature: **n.d.:**not detected; **TVOCs:** total volatile organic compounds; **DEHP**: Bis(2-ethylhexyl) phthalate; **DEP**: diethyl phthalate; **iso-DEHP:** Bis(2-ethylhexyl) isophthalate; **DINP:** Diisononyl phthalate; **DINCH:** Diisononyl 1,2-cyclohexanedicarboxylic acid; **DEHA:** Bis(2-ethylhexyl) adipate; **TCEP:** tris-(2-chloroethyl)-phosphate; **TCPP:** tris-(1-chloro-2-propyl) phosphate; **TDCPP:** tris-(2,3-dichloropropyl)-phosphate; **TPP**: triphenyl phosphate; **V6**: 2,2-bis(chloromethyl)propane-1,3-diyl-tetrakis(2-chloroethyl)bis(phosphate); **PentaBDE**: pentabromodiphenyl ether; **U-OPFR:** 2,2-bis(chloromethyl)propane-1,3-diyl tetrakis(1-chloropropan-2-yl)bis(phosphate); **TBB:** 2-ethylhexyl-2,3,4,5-tetrabromobenzoate; **TBPH:** bis(2-ethylhexyl)-3,4,5,6-tetrabromophthalate; **NCO**: unreacted isocyanates.

[a] mg of compound per g of material.

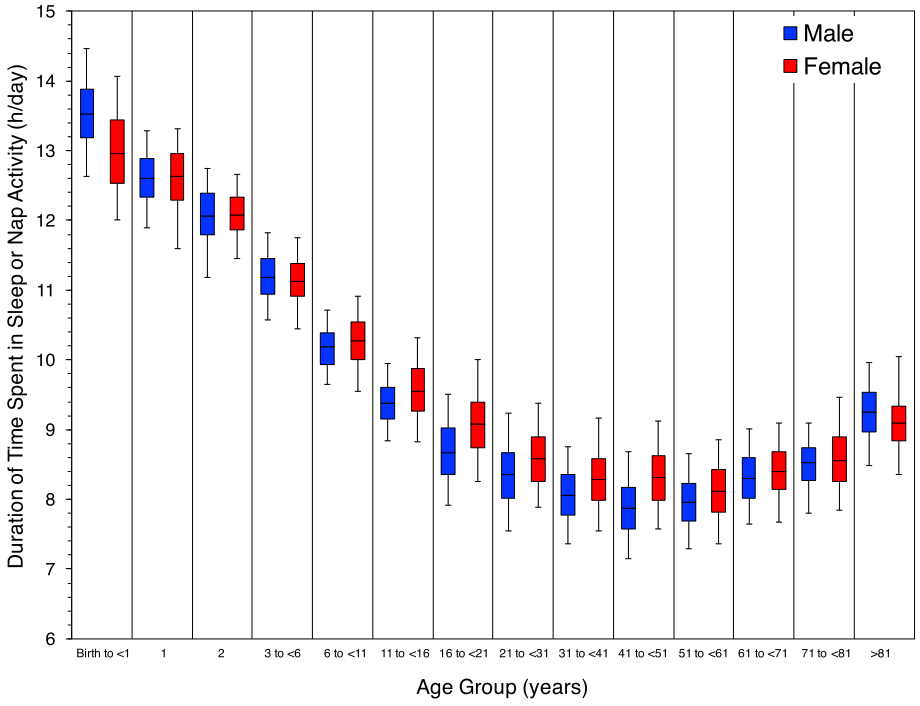

Fig. 1. Duration of time (h/day) spent in sleep or nap activity [159]; data ID = 14500). Box plots represent interquartile range and whiskers represent the 5th and 95th percentiles.

DEHP) in toys and child care articles (66). However, there is some debate as to whether or not crib mattresses are included in the definition of "child care articles" [158,201]. In addition, the ban on DINP and di-iso-decyl-phthalate (DIDP) only applies to children's toys that can be placed in the mouth. Alternative plasticizers that have a chemical structure similar to phthalates, such as di(2-ethylhexyl) adipate (DEHA) and diisononyl cyclohexane-1,2-dicarboxylate (DINCH), have emerged recently and have been identified in new crib mattress covers [17]; Table 2.

4.4. Unreacted isocyanates

An excess of toluene diisocyanate (TDI, the predominate diisocyanate used in polyurethane foam production [162]), may be added during the manufacture of polyurethane foam, above which is necessary for it to react with hydroxyl groups of polyols and water [185]. As such, unreacted isocyanate may be present in the final polyurethane foam product [20,81,185]. Krone et al. [81] identified NCO in polyurethane foam-containing consumer products, including a mattress and sofa padding. NCO was also detected in polyurethane-foam containing crib mattresses via PAS-FT-IR analysis in Ref. [17]. The foam in crib mattresses is somewhat protected from excess moisture released from the infant body, which can react with the isocyanates [10].

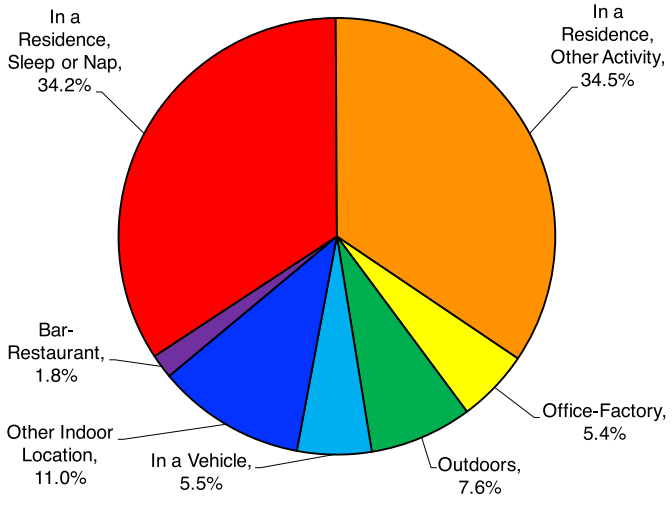

Fig. 2. Fraction of time adults spent sleeping or napping in a residence [77,159].

5. The source-proximity effect of the sleep microenvironment

In the sleep microenvironment, there are several key factors that contribute to the source-proximity effect (Fig. 5), including the spatial proximity of the BZ to the source, incomplete mixing and poor ventilation of room air, concentrations gradients near an actively emitting source, the personal cloud due to human body movement-induced particle resuspension, the buoyant human thermal plume, heat transfer from the human body to the source, which may elevate the emissions of gaseous pollutants, and direct dermal contact with the source.

5.1. Spatial proximity and concentration gradients near actively emitting sources

The spatial proximity of a person's BZ to their mattress and bedding can lead to elevated concentrations in their BZ relative to the bulk bedroom air. The spatial proximity effect has been evaluated in numerous studies, which typically release inert tracer gases from point or area sources and use sampling arrays to measure the uniformity of the dispersion of the tracer gas in a space, e.g. Refs. [1,83,103]. Collectively, these studies have shown that tracer gas concentrations near actively emitting sources are greater than those at locations further from the source, typically by a factor greater than 2.

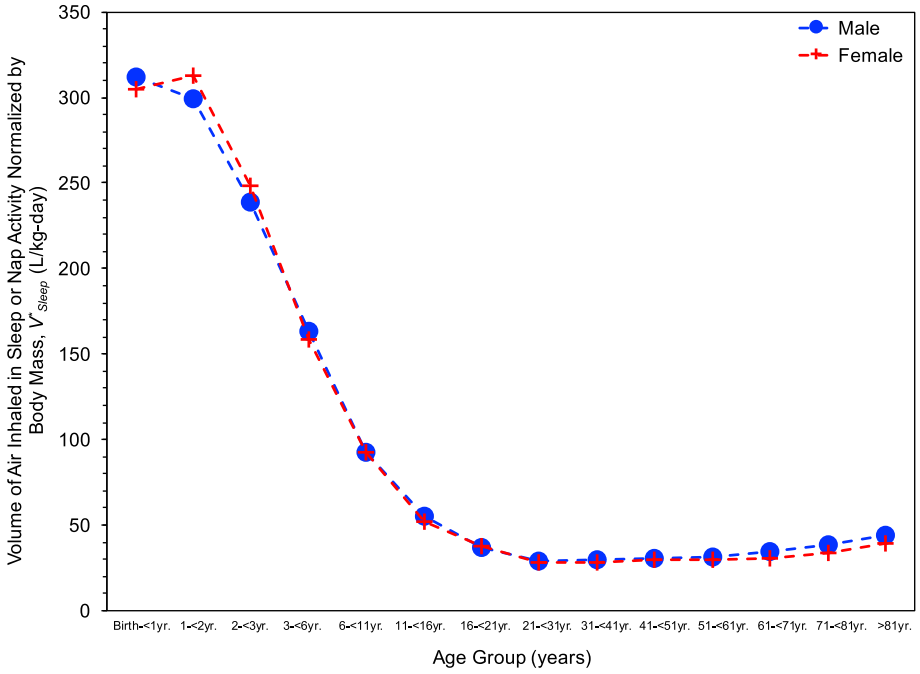

Fig. 3. Volume of air inhaled during sleep or nap activity per day, normalized by body mass for each age group and gender (calculated using U.S. EPA EFH data set, [159]).

Laverge et al. [88] highlighted the important role of the spatial proximity effect on inhalation exposure to pollutants originating in the sleep microenvironment. A breathing thermal manikin was positioned on a twin-size mattress with a pillow, from which sulfur hexafluoride (SF_6) was uniformly released to simulate the emission of gaseous pollutants. The relative intake fraction, defined as the ratio of the BZ concentration to the well-mixed chamber concentration, was reported for different sleeping positions and bedding arrangements. BZ SF_6 concentrations were found to be significantly greater than those measured in the bulk air, with mean relative intake fractions for the mattress source of 1.24 for the supine position, 1.39 for the lateral position, and 1.73 for the prone position. Higher mean relative intake fractions were reported for the pillow source with 2.16 for the supine position and between 3.39 and 4.02 for the lateral position. Additionally, covering the entire manikin's body and head with a blanket increased the relative intake fraction to 32.7. Boor et al. [15] reported similar findings for VOCs emitted from a crib mattress, with BZ TVOC concentrations greater than those in the bulk chamber air by factors in the range of 1.8–2.4. In regard to resuspended mattress dust particles, [16] also found BZ

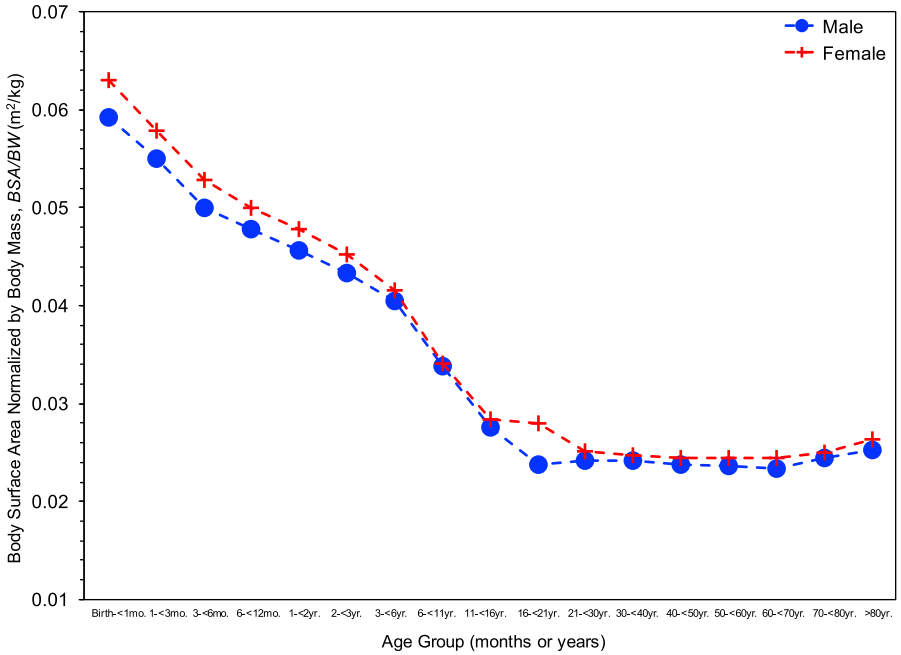

Fig. 4. Infant dermal exposure dose considerations in the sleep microenvironment: body surface area normalized by body mass (*BSA*/*BW*, m^2/kg) for each age group and gender (calculated using U.S. EPA EFH data set, [159]).

concentrations to be greater than bulk air concentrations by factors of 1.07–1.94, depending on particle size. The studies demonstrate that a person will receive elevated exposures to particulate and gaseous pollutants that are released from their mattresses, beyond what would be estimated from bulk bedroom air measurements.

5.2. Incomplete mixing of bedroom air

The spatial proximity effect is due in part to incomplete mixing of the released pollutant with the bulk air, which leads to the development of concentration gradients in the space, as suggested in some of the aforementioned studies. Additional studies by Refs. [18,41,49,101,129,166,178]; among others, have found that incomplete mixing conditions are associated with non-uniform pollutant concentration distributions in indoor environments.

Bedrooms, when compared to the rest of the home, have been found to be poorly ventilated, e.g. Refs. [6,8,90,199]. Incomplete mixing may be aided by the low ventilation rates that have been reported for bedrooms and the reduced run time of ventilation systems at night. Bekö et al. [8] measured the ventilation rates in the naturally ventilated bedrooms of 500 children in Denmark and reported a mean ventilation rate of 0.46 h^{-1}, with rates as low as 0.1 h^{-1}. In addition, [147] found that the fractional operation time of residential ventilation systems is the lowest during the late evening/early morning hours (10PM to 7AM). As the ventilation system run time is reduced, a residence relying on forced mechanical ventilation to deliver fresh (or filtered) air to the bedroom during sleep periods may see poor mixing conditions in the space (e.g. more stagnant air due to reduced ventilation and less human activity and movement to enhance mixing) and inadequate dilution of pollutants released within the sleep microenvironment, leading to the accumulation of pollutants in the air. Furthermore, it is expected that for an actively emitting diffusion-controlled source, such as a mattress, concentrations in very close proximity (several cm) will be greater than those in the bulk air, as a chemical compound partitions from the source to the air immediately above it. Thus, it cannot be assumed that a pollutant released from a mattress or bedding will be well mixed with the bulk bedroom air, and BZ concentrations should be measured to accurately estimate inhalation exposure to pollutants originating in this microenvironment.

5.3. Personal cloud effect and human-induced resuspension of mattress dust

The personal cloud effect, whereby people can be envisioned as being surrounded by a cloud of particles, many of which are of biological origin, is another factor that may lead to elevated exposures in the sleep microenvironment [12,96,104]. The personal cloud effect was used to explain the consistently higher personal exposures to particles compared to stationary monitoring locations [165]. Rabinovitch et al. [122] provides one example in which personal (approximate BZ) bacterial endotoxin concentrations measured with personal monitors are about 3 times greater than those measured at stationary indoor locations, suggesting that endotoxin exposures are better correlated with sources in close proximity to the person and their BZ than those in the bulk indoor air. Human-induced particle resuspension from indoor surfaces, including mattresses and bedding, is one of the physical processes contributing to the personal cloud effect, leading to elevations in BZ concentrations beyond those in the bulk air. In the sleep microenvironment, the personal cloud effect is likely to be enhanced due to the close proximity of a person's BZ to the surfaces from which particle resuspension is occurring, an important consideration for larger particles (>10 μm) that have high settling velocities (on the order of ~10^{-3} m/s), and likely short trajectories upward from a surface. Investigations by Spilak et al. and Boor et al. [16,141] have shown that human body movements in bed, such as rolling from the prone to supine position, can resuspend significant quantities of settled particles on mattresses, pillows, bed sheets, and blankets. Spilak et al. [141] demonstrated that a pillow cover (225-thread count) does not serve as an effective barrier to the transport of resuspended particles from the underlying pillow surface, however, the impact of impermeable mattress covers on particle resuspension and transport to the BZ has yet to be investigated.

5.4. Body movements during sleep periods

Mattress dust resuspension is driven by human body movements in bed. Human activity in bed can occur during periods of wakefulness, e.g. as a person enters a bed and prepares themselves for sleep, and throughout their extended sleep state. Although the absence of voluntary motor behavior is a characteristic of the sleep state, movements are commonly reported, ranging from about ten significant body posture shifts (e.g. Ref. [186]), to several hundred smaller body movements (e.g. Ref. [187]). The cause of body movements during sleep periods is largely unknown, although several theories suggest that movements occur to relieve muscular discomfort during extended periods of immobility [67,188].

Sleep is typically divided into several sleep stages: stage W (Wakefulness), NREM stage 1 (non-rapid eye movement), NREM stage 2, NREM stage 3 or SWS (Slow Wave Sleep, e.g. “deep sleep”), and REM (rapid eye movement) (e.g. Ref. [189]). It is important to understand the dependency of body movements on sleep stages, as

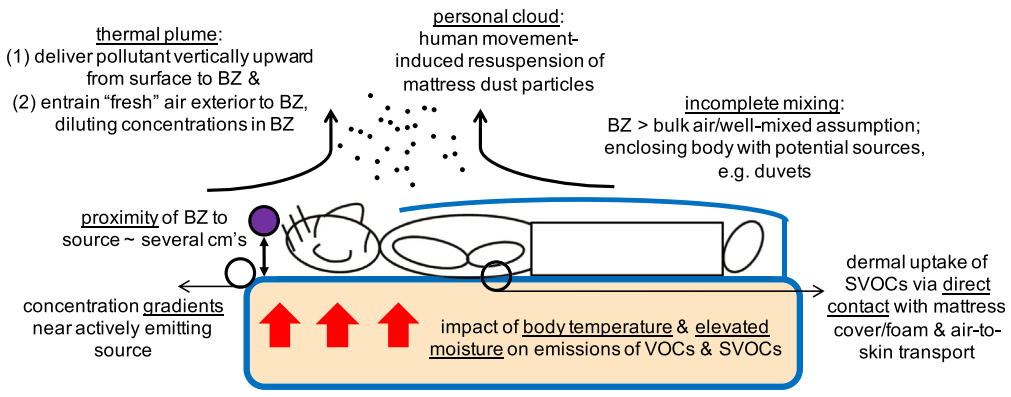

Fig. 5. The source-proximity effect of the sleep microenvironment.

body movements are responsible for inducing the necessary removal forces (aerodynamic, mechanical) to resuspend mattress dust particles [121]. Shimohira et al. [190] found a strong dependency of body movement intensity on sleep stage among children 4–12 years of age. Body movements were classified into four groups, GM-1: axial rotation of the body, GM-2: movement of limbs and trunk (torso) muscles without rotation, GM-3: movement of two or more limbs but not trunk muscles, and GM-4: movement of only one limb. The authors found that movements are much more frequent during both NREM stage 1 and REM sleep, with a decrease during SWS (deep sleep). The most commonly reported movement was GM-1, in which the child rotates some degree, with up to 13 movements/hour during NREM 1 sleep.

Wilde-Frenz and Schulz [191] observed a similar trend among adults, 21–36 years of age, where body movement frequency was the greatest during stage W, followed by NREM 1, then REM, then NREM 2, and SWS. Body movement frequency ranged from <0.1 to 0.8 body movements per sleep minute (bm/min), depending on the sleep stage. Giganti et al. [188] found that the number of body movements were greatest during NREM 1 sleep, with movement frequency remaining nearly constant during NREM 2, SWS, and REM sleep. Body movements were found to decrease with age, with an average of 0.25 bm/min for age group 1 and 0.09 bm/min for age group 2, equating to an average of 86 body movement during the entire sleep period for group 1 (mean sleep time of 342 min) and 27 movements for group 2 (mean sleep time of 297 min).

The findings of these studies suggest that body movement frequency is a function of both sleep stage and age. Thus, the potential for human-induced particle resuspension would vary throughout a sleep period, and may be greater during periods of more frequent activity (e.g. NREM 1 and REM stages) than during deep sleep stages (SWS). [16] demonstrated that more intense body movements in bed, such as a full 360° rotation of the torso, are associated with higher surface vibrations and emissions of resuspended particles compared to less intense movements (e.g. sitting on the mattress). Studies have suggested that asthmatic children and adults have more disturbed sleep patterns [192,193], and Zanobetti et al. [194] found sleep-disordered breathing, defined as the recurrent episodic disruption of normal breathing during sleep, and decreases in sleep efficiency to be associated with increases in short-term variation in the airborne concentration of 10 μm particles. People with asthma and other breathing ailments may have a greater frequency of body movements during their disrupted sleep period, which may put them at greater risk for elevated exposure to resuspended mattress dust particles.

5.5. Human thermal plume

In the sleep microenvironment, the airflow pattern is a result of two different flows and their interaction: the buoyant human thermal plume and transient flows generated by breathing [195,196]. While the thermal plume depends on the metabolic rate of a sleeping person and the sleep stage, the transient flow is mainly dependent on whether the sleeping person inhales/exhales via nose or mouth. The buoyant human thermal plume may also be responsible for elevated BZ concentrations for pollutant sources in close proximity of the human body. Rim and Novoselac [128] and Rim and Novoselac [129] demonstrated that the thermal plume is an effective mechanism for transporting pollutants, released from various locations in close vicinity to the human body, vertically upward, toward to BZ. Thus, the thermal plume that develops around a sleeping person may aid in transporting pollutants released from the mattress surface, upward and toward their BZ. However, the thermal plume may also serve the secondary role of entraining “fresh” air exterior to the sleep microenvironment, thereby diluting concentrations of mattress pollutants in the BZ, as suggested by Ref. [88]. The thermal plume also interacts with the shape of the breathing zone, especially the inhalation zone, reducing it to a shallow area of only a few centimeters wide stretching down around the cheeks of a person resting in supine position [89].

5.6. Impact of body temperature and sweating on chemical emissions from mattresses

Another unique attribute of the sleep microenvironment that may play an important role in enhancing the source-proximity effect is the warming of nearby objects via heat exchange with the human body. Along with warming the surrounding air via convective heat exchange, leading to the development of the thermal plume, our bodies will also transfer heat to surfaces that are in direct contact via conduction and those in close proximity via radiation. Lu et al. [99] monitored the surface temperature of a mattress around sleeping adult male subjects with a matrix of 16 thermistors. Maximum surface temperatures were found to range from 31 to 36 °C beneath and around the lower limbs and between 34 and 36 °C beneath and around the waist. Thus, it is to be expected that the sleeping human body will increase the surface temperature of their mattress, pillow, and bedding materials. The emissions of gaseous pollutants, such as VOCs and SVOCs, from building materials has been shown to increase with temperature (e.g. Refs. [98,197,198]), with recent studies by Refs. [15,95] observing this effect in crib mattresses and mattress covers. Thus, the close proximity of the human body to potential pollutant sources may enhance volatilization, and subsequently, further increase BZ concentrations. Throughout the sleep period, the sleeping thermal environment is a permanently humid environment due to the production of large quantities of moisture through sweating and the large buffering capacity provided by mattresses and bedding materials. Elevated moisture levels may also have a strong impact on emissions of VOCs and SVOCs from mattresses, as well as their desorption from bedding materials that can serve as sorptive reservoirs during the day (e.g. Ref. [112]).

5.7. Direct dermal contact with mattresses and bedding materials

Transdermal uptake of pollutants, such as SVOCs, via contact transfer and air-to-skin transport, is another contributing factor to the source-proximity effect. During sleep periods, a person's body, and exposed skin, is in intimate contact with their pajamas, mattress, mattress cover, and bedding materials. Dermal exposure is an important pathway for certain SVOCs, including plasticizers that are added to mattress covers [109,169]. If the gas-phase concentrations of these pollutants are greater near the source or the skin, dermal uptake via air-to-skin transport may be enhanced [108]. Laverge et al. [88] demonstrated that concentrations of mattress-released gaseous pollutants (SF_6) under the bedding (where most of the body is exposed) can be up to 30 times higher than those in the bulk air of the room. Additionally, during the day, SVOCs found in mattresses, as well as in the bulk bedroom air, may partition to bedding fabric fibers and pajamas [108]. Fabric fibers may also be coated with a surface film of detergents and fabric softeners that are used for cleaning. Temperature elevations during sleep periods may promote desorption of the sorbed SVOCs, thereby offering an additional route of transdermal uptake [169].

5.8. Personalized ventilation and air cleaning systems for beds

An important factor that can affect pollutant transport dynamics and exposures while sleeping is the use of personalized ventilation

(PV) and air cleaning systems for beds. Fan-forced decentralized ventilation systems commonly found in homes can provide filtered and conditioned air for a sleeping person, e.g. Ref. [90]. However, they may not necessarily be sufficient in reducing concentrations of mattress-released pollutants within the sleep microenvironment or in improving sleep quality among people with asthma and allergies [56,59,76]. PV systems can deliver a relatively small amount of air (less than 0.3 h^{-1}) directly to the BZ of a sleeping person, thereby offering an alternative strategy to reduce sleep exposures. Several PV designs for beds have been introduced, including: bedside PV [84]; overhead PV [106]; and a temperature-controlled laminar airflow (TLA) PV [143]. Melikov et al. [106] found overhead PV to reduce exposures of medical staff to the exhaled air of a patient in a bed at significantly lower ventilation rates compared to a decentralized ventilation system. Spilak et al. [143] found TLA PV to reduce BZ concentrations of airborne particles with a sleeping thermal manikin beyond what can be achieved with a portable air cleaner. Furthermore, TLA PV has been shown to reduce the severity of asthma symptoms among patients with persistent asthma [19,118,136]. Along with PV, portable HEPA filtration devices positioned close to the bed (e.g. on a night-stand) have been shown to reduce BZ concentrations of coarse and fine airborne particles [28,142].

6. Conclusions and future directions

We spend nearly one-third of every day asleep. Indoor air quality (IAQ) in the sleep microenvironment, and more broadly, in bedrooms, may have important implications for human health and well-being, especially among children and adults with asthma and allergies. Human exposures to mattress-released pollutants can be augmented as air is inhaled just centimeters above the source, with breathing zone concentrations typically greater than those in the bulk air by a factor of two, and in specific conditions (cover over head), as high as 30. Beds serve as reservoirs for a complex mixture of biological- and chemical-laden dust, which can be readily stirred-up upon tossing and turning during sleep. House dust mite allergens and fungi may be generated in-situ, while other particles, such as bacterial cells and animal allergens, may be generated by, or tracked-in with, people and their pets. The sleep thermal environment can promote fungal growth and the proliferation of dust mites. Settled dust particles can accumulate over time, especially given the low cleaning frequencies of mattresses and pillows. Exposure to SVOCs via inhalation and dermal pathways during sleep may be significant, given the ubiquity of SVOC additives in infant and adult bedding products (foams and plastic covers) and their enhanced volatilization in and under bedding due to elevated localized surface temperatures (~30 °C) and relative humidities.

This review provided an overview of literature pertaining to the emerging field of what can be referred to as *sleep IAQ*, the study of indoor air pollutant dynamics and exposures in sleep microenvironments. However, the existing research on *sleep IAQ* is insufficient to elucidate the impact of sleep exposures to indoor air pollutants and biological material on human health and sleep quality. In reviewing the extant literature, the following research needs were identified:

(1.) Field studies are needed to understand the variation in exposures occurring during sleep among diverse populations and age-groups and how these exposures compare to exposures occurring in the remaining two-thirds of the day. In particular, the contribution of early-life sleep exposures to microbes, allergens, and SVOCs to total exposures to these agents should be evaluated. This work would necessitate personal exposure monitoring (BZ sampling) of particles and gases. Improved sleep exposure assessment will permit more reliable associations with health outcomes, beyond what can be achieved through basic mattress dust sampling and analysis.

(2.) Research is needed to elucidate the impact of human exposure to mattress-released pollutants, human bioeffluents, and pollutants originating elsewhere in the residence or outdoors on sleep itself. Several recent studies have indicated that poorly ventilated bedrooms can be associated with higher CO_2 concentrations, which can adversely affect sleep quality and next day performance [87,148], and that exposure to traffic pollution (e.g. black carbon) may disturb sleep (e.g. Refs. [48,55]. These efforts can be extended to address the full range of particulate and gaseous indoor air pollutants.

(3.) Little is known on the role of multiple bed occupants and pets in influencing sleep exposures and pollutant transport dynamics. According the National Sleep Foundation (NSF) Sleep in America Poll, 48–72% of Americans sleep with their significant other and 2–16% sleep with their pets, depending on ethnicity [111]. Multiple people or pets in bed may impact the microbial composition of mattress dust and the extent to which it is aerosolized throughout the night, as well as the interaction of multiple buoyant thermal plumes in the air in and around the bed.

(4.) Research in the *sleep IAQ* domain should follow a mechanistic approach and include new research on the following topics: experimental and modeling studies on the emissions of VOCs and SVOCs from new and used bedding products on the market, including eco-friendly mattresses and low-cost air mattresses; transient sorption-desorption cycling of these chemical contaminants with bedding and pajama fabrics during periods of occupancy and non-occupancy in bed, as the thermal environment changes with time; size-resolved resuspension fractions of biological particles from new and soiled bedding materials; and improved assessment of inter-zonal airflow between the BZ and bulk bedroom air under a range of bedroom ventilation conditions.

(5.) Future investigations should explore exposures in unique sleep microenvironments, such as neonatal intensive care units (NICUs), hospital beds, tents for camping (which can contain high levels of flame retardants, [74]), hotel beds, and bedrooms with many occupants, such as shared dormitory rooms.

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