Toxic volatile organic compounds in 20 homes in Shanghai: Concentrations, inhalation health risks, and the impacts of household air cleaning

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ARTICLE INFO

Keywords:
Nighttime sleep
Formaldehyde
Residence
Cancer risk
Air filtration
Asthmatic children

ABSTRACT

Exposure to VOCs causes a variety of adverse health effects, with much of the exposure occurring at home during sleep. The use of an air cleaner maybe an effective and convenient way to reduce associated exposures and health risks. Studies about the impact of air cleaners on VOCs are limited. The main objective of this paper was to assess the inhalation health risks that toxic VOCs present during nighttime sleep and to estimate the removal effectiveness of an indoor air cleaner on these VOCs. Asthmatic children, who are especially vulnerable to the detrimental effects of air pollutants, were recruited and the VOC concentrations in their bedrooms were measured during two periods - once with a true air cleaner and once with a sham air cleaner. Among the toxic VOCs quantified, formaldehyde, acetaldehyde and toluene had the highest concentrations (median: 18.0, 14.0 and 12.1μg/m³, respectively) in the bedrooms. Health risk assessments were conducted to identify compounds of greatest concern. During nighttime sleep, 7 VOCs presented inhalation cancer risk above the acceptable risk (1×10⁻⁶), 4 VOCs exceeded the non-cancer risk threshold (1) in most of the homes tested. The results indicate that the use of an air cleaner in residences may lead to significant reductions in VOC concentrations indoors, but even with this reduction, the associated health risks are still of concern. This study highlights the need for reductions in toxic VOCs at home, points to the imminent need for improvements in control of VOC sources.

1. Introduction

Most exposure to harmful air pollutants including volatile organic compounds (VOCs) and fine particulate matter (PM₂.₅) occurs indoors as a result of a plethora of sources, low ventilation rates, and the amount of time spent indoors and especially in homes [1,2]. Many studies have been undertaken to characterize PM₂.₅ and have focused on exposure, composition, sources, health impacts and ways to reduce PM₂.₅ [2–5]. In contrast, VOCs, which can be equally damaging to health, have received less attention compared to PM₂.₅.

VOCs can be emitted from vehicle exhaust, gasoline vapor, coal burning, biomass burning, and petrochemical industry and solvent use (includes paint application, printing processes, dry cleaning, solvent evaporation from household products and other industrial processes) [6]. Rapid industrial and economic development in China has resulted in an increase in VOCs indoors with these compounds having a wide range of sources, routes of exposure, and health effects [7–9]. The adverse health effects of VOCs range from acute irritation of the eyes, skin, mucous membranes and respiratory tract to the development of chronic diseases including asthma, cardiovascular disease and even cancer [10–14]. The main route of exposure to most VOCs is through inhalation [15], which leads to more severe health impacts for susceptible people like asthmatic children than for healthy individuals [16]. In addition, exposure to VOCs has been associated with the onset

https://doi.org/10.1016/j.buildenv.2019.04.047
Received 1 March 2019; Received in revised form 19 April 2019; Accepted 23 April 2019
Available online 24 April 2019
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and exacerbation of asthma in children [14,17,18]. Based on a 2011 study of childhood asthma in 10 cities in China, the prevalence of asthma appears to have risen in recent years, with Shanghai having the highest doctor-diagnosed asthma prevalence (9.8%) among 3–6 year olds [19]. This highlights the growing need to understand the environments and exposures that may be affecting these sensitive individuals. A cross-sectional study [20] investigated the association between asthma and common indoor exposures among schoolchildren aged 9–11 years, but the indoor exposures they presented are dampness, visible mold growth, paraffin use for cooking, and passive smoking, no specific chemical exposures are evaluated.

Despite the complexity of exposure in different scenarios, people spend 8.1 ± 1.3 (mean ± standard deviation) hours at home for nighttime sleep [21]. Previous studies have been conducted to measure the concentrations of VOCs in residences in China and assess the health risks, however the environments around sensitive individuals and the VOC exposures overnight have not been fully investigated. Guo et al. [22] measured 100 homes in Hong Kong, China, and observed that formaldehyde and styrene had the highest concentration of all 16 VOCs measured. In a study of 27 homes in Shizuoka, Japan and 14 homes in Hangzhou, China, Ohura et al. [23] found that VOC concentrations in Hangzhou were significantly higher than those in Shizuoka, likely due to potent indoor emission sources in Chinese homes. Other studies have focused instead on the risk assessment of VOCs: cancer and non-cancer risk assessments were used to identify the VOCs of highest concern in residences and public places [7,24–26]. Zhou et al. [24] found that for 12 participants, benzene presented the highest median cancer risk of 10 VOCs measured indoors in Tianjin, China. For 8 newly renovated residences in Shanghai, Dai et al. [27] found concentrations of 1,2-dichloroethane (1,2-DCE), 1,4-dichlorobenzene (1,4-DCB), methylene chloride, and ethylbenzene presented a mean cancer risk above the US EPA proposed acceptable risk level of 1 × 10−6. Du et al. [7] summarized the concentrations of 16 highly prevalent hazardous VOCs in different micro-environments in urban China from 10 research studies (more than 3000 samples), and found that formaldehyde, 1,4-DCB, benzene and 1,3-butadiene presented the highest median cancer risk and only formaldehyde exceeded the non-cancer risk limit. Deng et al. [25] measured the concentrations of 16 carbonyls in five kindergartens in Hong Kong but only assessed the cancer risk of formaldehyde indoors (ranged from 4.5 × 10−5 to 2.1 × 10−4).

Some standards were enacted in China to control VOC concentrations (i.e., GB16297-1996, GB/T-18883-2002). However, they only focused on a less-than-comprehensive list of VOCs. As previously mentioned, the VOC levels in China were still of concern. In spite of suggestive evidence of VOC reduction by filtration, most of exist studies have certain limitations, due to their focus on only single pass efficiency of air cleaners, a less-than-comprehensive list of VOCs, and a very controlled experimental setting [28–31]. Chen et al. [28] evaluated different cleaning technologies of 15 air cleaners for the removal of 16 VOCs in a stainless steel chamber during 12h of use. They found that sorption filtration was the most effective off-the-shelf commercial technology for general removal of VOCs indoors. While these controlled studies can provide insight into the potential for an air cleaner to remove certain compounds, these environments do not reflect the complexity of the indoor environment (such as the home environment), or how well a purifier removes VOCs during an extended period of time. Among the studies that evaluated the VOC removal performance of air cleaners in real-world scenarios, they offered the removal performance of air cleaners in TVOC but did not fully elucidate the removal performance of air cleaners in individual toxic VOC [32,33].

In this study, we quantified the concentrations of VOCs outdoors and in the bedrooms of 20 asthmatic children in Shanghai, China. Inhalation health risk assessments of toxic VOCs were conducted to identify VOCs of highest concern during nighttime sleep. Monte Carlo simulation was used to quantify the uncertainty of risk assessment model. We evaluated the impact of air cleaner use on the levels of toxic VOCs, by comparing measurements conducted during two periods: one with a fully functional air cleaner (with both a HEPA filter and an activated carbon filter) and one with a sham air cleaner (where both filters were removed).

2. Methodology

2.1. Participant recruitment and study design

Participants were recruited from individuals that attended the outpatient clinic of the Shanghai First People’s Hospital. The study protocol was approved by the Ethics Committee of Shanghai First People’s Hospital, the Duke University Institutional Review Board, and registered at ClinicalTrials.gov (NCT03282864). To be eligible, participants need to be 5–14 years old, had doctor-diagnosed asthma, and had experienced at least one asthma attack during the past 12 months. Individuals who had chronic diseases other than asthma were excluded from participation. All participants provided oral assent, and their guardians gave written consent. We measured VOCs among 20 participants, (35% female) aged from 5 to 12 with mild or moderate asthma. The bedroom sizes of recruited residential buildings were 36 ± 10 (mean ± standard deviation) m². The common window types are sliding and casement windows, with no carpet used in any of the families.

A double-blind, randomized crossover trial was conducted. A commercially available air cleaner (Atmosphere®, Amway, USA) comprising a pre-filter, a high efficiency particulate air (HEPA) filter, and an activated carbon filter was used in this study. Between February 14th and April 24th, 2017, each participant use two different air cleaners in their bedroom: a true air cleaner (pre-filter + HEPA + activated carbon) and a sham air cleaner (with pre-filter only, as placebo). Each indoor air cleaning intervention lasted 14 days and a two-week washout period separated the two interventions. The order of true and sham interventions was randomized for each participant. During the interventions, the participant and their families were instructed to keep doors and windows in their bedroom closed as they usually did in winter, and refrain from smoking indoors at home. They were also instructed to keep the air cleaner running continuously, at a flow rate of 2.8 m³/min.

2.2. Sample collection and analysis

VOC samples were collected simultaneously indoors and outdoors during each intervention. Samples were collected at least two days after the initiation of air cleaner use so that the VOC concentrations could reach steady state prior to sampling. Tenax TA and 2, 4-dinitrophenylhydrazine (DNPH) cartridges were used for sampling, at a flow rate of 200 mL/min and 600 mL/min, respectively, using low-cost pump packages designed and built at Duke University. The 90-min integrated sample was collected in the breathing zone in the early morning to represent the indoor VOC concentrations during sleep. Off-the-shelf timers were used to specifically initiate sampling so that the entire 90-min completes before the family woke up to minimize the influence of human behaviour. The execution of the VOC sampling was shown in Fig. S1 in the Supplementary data. The Tenax-TA cartridges were analysed by a thermal desorption-gas chromatography-mass spectrometry (TD-GC/MS, 7890-5975, Agilent, USA). The DNPH cartridges were extracted with acetonitrile and analysed by a high-performance liquid chromatography (HPLC, 1260, Agilent, USA) with UV absorption. The target species were quantified using a multipoint internal calibration method. Indoor and outdoor temperature (T), relative humidity (RH) and CO₂ concentrations were simultaneously and continuously monitored by low-cost air quality monitors [34]. SPSS 17 (IBM Corp., Somers, USA) was used for data analysis.
2.3. Risk assessment method

This study is a part of a broader project about air pollution and the health impacts of indoor air filtration for asthmatic children, the health parameters from asthma children with and without air purification will be discussed in another paper. Here, inhalation cancer and non-cancer risk were chosen as health index to identify VOCs of greatest concern during nighttime sleep. Although the VOC concentrations were obtained in asthmatic children’s bedroom, the target population of the risk assessment in this paper, however, is not asthma children but people who exposed to the same VOC concentrations during nighttime sleep.

To estimate the elevated inhalation health risks by inhalation exposure during nighttime sleep, we followed the guidance proposed by US EPA as described in Dai’s study [27]. A time-adjusted exposure model was used to represent continuous exposure, as shown in Equation (1):

\[ E_i = \sum_j C_{ij} \times \frac{EF}{NY} \times \frac{ED}{AL} \]

where, \( E_i \) is the adjusted chronic daily personal exposure to compound \( i \) during nighttime sleep in μg/m³; \( C_{ij} \) is the measured concentration of compound \( i \) in bedroom in μg/m³; \( t_j \) is the time fraction of nighttime sleep; \( EF \) is the exposure frequency in days/year; \( NY \) is the number of days per year, 365 days/year; \( ED \) is the exposure duration in years; and \( AL \) is the average lifetime (70 years) in years. According to the surveys of Chinese Center for Disease Control and Prevention [21,35], people spend 8.1 ± 1.3 (mean ± standard deviation) hours for nighttime sleep. Detailed distribution about nighttime sleep duration for each age group is listed in Table S1 in the Supplementary data, and this information was used in the calculation for \( t_j \). \( EF/NY \) is taken as 1 to estimate the chronic daily exposure in this study.

Inhalation unit risk (IUR) is the excess cancer risk resulting from continuous exposure to a unit increase of a compound via inhalation. The IUR listed in Table 1 is derived from previous studies by the US EPA for the general population with a default body weight of 70 kg and a default inhalation rate of 20 m³/day (i.e., 13.9 L/min) [36,37]. To estimate the potential cancer risk of different population, IUR can be adjusted as:

\[ \text{IUR}_a = \frac{\text{ADAF} \times \text{IR}_d \times \text{BW}_a \times \text{IUR}_i}{\text{BW}_d} \]

where \( \text{IUR}_a \) is the adjusted inhalation unit risk of compound \( i \) by inhalation rate, body weight and age, per μg/m³; \( \text{IUR}_i \) is the inhalation unit risk with a default body weight and inhalation rate, per μg/m³; \( \text{ADAF} \) is the age-dependent adjustment factor, US EPA [38] recommends applying ADAF of 10 × 3 × 4 for IUR for children ages 0 to < 2 years and 2 to < 16 years, respectively; \( \text{IR}_d \) is the default inhalation rate of 13.9 L/min, \( \text{IR}_d \) is the age-specific inhalation rate of the target population, L/min; \( \text{BW}_a \) is the default body weight of 70 kg, \( \text{BW}_d \) is the age-specific body weight of the target population, kg. The distribution of inhalation rate and body weight for each age group are listed in Table S1 in the Supplementary data.

The chronic inhalation cancer risk (CR) is the increased probability of developing cancer as a result of a specific exposure to a certain compound and was calculated as follows [39]:

\[ \text{CR}_i = E_i \times \text{IUR}_i \]

The inhalation non-cancer risk was calculated using the following equation [40–42]:

\[ \text{HQ}_i = \frac{E_i}{\text{RfC}_i} \]

where \( \text{HQ}_i \) is the hazard quotient of compound \( i \); \( \text{RfC}_i \) is the chronic reference concentration (RfC) of compound \( i \) in μg/m³. To avoid chronic health effects, \( \text{HQ}_i \) should not exceed 1.

IUR, and RfC, values can be obtained from the Integrated Risk Information System (IRIS) and the Office of Environmental Health Hazard Assessment (OEHHA) chemical database as shown in Table 1.

### Table 1

<table>
<thead>
<tr>
<th>CAS No.</th>
<th>Chemical</th>
<th>Group</th>
<th>RfC (μg/m³)</th>
<th>IUR (per μg/m³)</th>
</tr>
</thead>
<tbody>
<tr>
<td>50-00-0</td>
<td>Formaldehyde</td>
<td>1</td>
<td>9</td>
<td>OEHHA</td>
</tr>
<tr>
<td>75-07-0</td>
<td>Acetaldehyde</td>
<td>2B</td>
<td>9</td>
<td>IRIS</td>
</tr>
<tr>
<td>123-38-6</td>
<td>Propanal</td>
<td>-</td>
<td>8</td>
<td>IRIS</td>
</tr>
<tr>
<td>78-93-3</td>
<td>MEK</td>
<td>-</td>
<td>5000</td>
<td>IRIS</td>
</tr>
<tr>
<td>71-43-2</td>
<td>Benzene</td>
<td>1</td>
<td>3</td>
<td>OEHHA</td>
</tr>
<tr>
<td>100-41-4</td>
<td>Ethylbenzene</td>
<td>2B</td>
<td>1000</td>
<td>IRIS</td>
</tr>
<tr>
<td>108-88-3</td>
<td>Toluene</td>
<td>3</td>
<td>300</td>
<td>OEHHA</td>
</tr>
<tr>
<td>1330-20-7</td>
<td>Xylenes</td>
<td>3</td>
<td>100</td>
<td>IRIS</td>
</tr>
<tr>
<td>100-42-5</td>
<td>Styrene</td>
<td>2B</td>
<td>900</td>
<td>OEHHA</td>
</tr>
<tr>
<td>68-12-2</td>
<td>DMF</td>
<td>2B</td>
<td>30</td>
<td>IRIS</td>
</tr>
<tr>
<td>78-87-5</td>
<td>1,2-DCP</td>
<td>1</td>
<td>4</td>
<td>IRIS</td>
</tr>
<tr>
<td>106-46-7</td>
<td>1,4-DCB</td>
<td>2B</td>
<td>800</td>
<td>OEHHA</td>
</tr>
<tr>
<td>107-06-2</td>
<td>1,2-DCE</td>
<td>2B</td>
<td>400</td>
<td>OEHHA</td>
</tr>
</tbody>
</table>

Notes:
- CAS No., Chemical abstracts service number.
- Group 1, carcinogenic to humans; 2A, probably carcinogenic to humans; 2B, possibly carcinogenic to humans; 3, not classifiable based on carcinogenicity to humans.
- Chronic Reference Exposure Levels (REL) from OEHHA, μg/m³, with equivalent definition of RfC from IRIS.

Where IUR\(_a\) is the adjusted inhalation unit risk of compound \( i \) by inhalation rate, body weight and age, per μg/m³; IUR\(_i\) is the inhalation unit risk with a default body weight and inhalation rate, per μg/m³; ADAF is the age-dependent adjustment factor, US EPA [38] recommends applying ADAF of 10 × 3 × 4 for IUR for children ages 0 to < 2 years and 2 to < 16 years, respectively; \( \text{IR}_d \) is the default inhalation rate of 13.9 L/min, \( \text{IR}_d \) is the age-specific inhalation rate of the target population, L/min; \( \text{BW}_a \) is the default body weight of 70 kg, \( \text{BW}_d \) is the age-specific body weight of the target population, kg. The distribution of inhalation rate and body weight for each age group are listed in Table S1 in the Supplementary data.

The chronic inhalation cancer risk (CR) is the increased probability of developing cancer as a result of a specific exposure to a certain compound and was calculated as follows [39]:

\[ \text{CR}_i = E_i \times \text{IUR}_i \]

The inhalation non-cancer risk was calculated using the following equation [40–42]:

\[ \text{HQ}_i = \frac{E_i}{\text{RfC}_i} \]

where \( \text{HQ}_i \) is the hazard quotient of compound \( i \); \( \text{RfC}_i \) is the chronic reference concentration (RfC) of compound \( i \) in μg/m³. To avoid chronic health effects, \( \text{HQ}_i \) should not exceed 1.

IUR, and RfC, values can be obtained from the Integrated Risk Information System (IRIS) and the Office of Environmental Health Hazard Assessment (OEHHA) chemical database as shown in Table 1.

### 2.4. Quality assurance and quality control (QA/QC)

The sampling flow rate was measured and calibrated using a flow calibrator (Bios Defender 520, Mesa Laboratories, NJ, USA) at the beginning and the end of each sampling session. The average value was used as the flow rate during sampling. During the field survey, blanks were collected to identify problems of contamination with the samples. Travel blanks (N = 4) and loading blanks (N = 13) showed little if any contamination. The limit of detection (LOD) was 2 μg/m³ for each VOC analysed by TD-GC/MS and HPLC.

To quantify the uncertainty associated with the risk assessments, we performed Monte Carlo simulations and sensitivity analyses using Crystal Ball (Oracle, USA), and used probability distributions for the input data rather than a single point value [43]. A lognormal distribution was used for VOC concentrations according to our measurement as well as literature [7,27]. A triangle distribution was used to describe the IUR and RfC characteristics, as in previous health risk assessments [7,27]. Sensitivity analyses were conducted to identify the contribution of the input parameters to the variance of the risks.

### 3. Results and discussions

#### 3.1. Identification of target VOCs

Seventy-nine valid sampling sessions were conducted: 40 outdoors, 20 indoors under the sham intervention and 19 indoors under the true intervention. In each of the four sampling groups (i.e., indoor true, indoor sham, outdoor true, and outdoor sham), 43 VOCs were detected above LOD during > 25% of the time. As functional groups determine the characteristics of a compound, these 43 VOCs were further classified into alkanes (n = 6), alkenes (n = 6), aromatics (n = 6), halogenated VOCs (x-VOCs, n = 3), and oxygenated VOCs (o-VOCs, n = 20). In addition to those categories, two compounds (benzoic acid and dimethylformamide) were also measured. The 20 o-VOCs comprised 11 aldehydes, 3 ketones, 3 alcohols, and 3 esters. Detailed information about the detection frequency and concentrations of the 43 VOCs under the true and sham interventions are shown in Table S2 in the Supplementary data. During the interventions, the mean concentration of TVOC was 86.0 μg/m³ (range: 22.1–594.0 μg/m³) outdoors, 338.2 μg/m³...
m$^2$ (range: 83.3–987.8 μg/m$^2$) indoors during the sham intervention and 155.8 μg/m$^2$ (range: 68.6–491.3 μg/m$^2$) indoors during the true intervention. Fig. 1 shows the mean mass concentrations and proportions of different functional groups of VOCs. For sampling conducted outdoors, the dominant groups of VOCs were aromatics, which made up 34% of the VOCs detected, followed by aldehydes (21%) and esters (16%) (all percentage listed were by mass). Indoors during the sham intervention, the dominant groups were aldehydes (46%), followed by alkenes (16%) and x-VOCs (11%). During the use of the true air cleaner, the dominant groups remained the same as those during the use of sham air cleaner indoors, but the concentration of each group was lower. Thirteen of forty-three VOCs were selected as target VOCs for risk assessment, based on the following criteria: (1) VOCs with frequencies of detection above the LOD > 25% during either the true or sham intervention, and (2) VOCs classified by the International Agency for Research on Cancer (IARC) in Groups 1 (carcinogenic to humans), 2A (probably carcinogenic to humans), or 2B (possibly carcinogenic to humans), or with confirmed inhalation toxicity as defined in the US EPA IRIS or the California EPA OEHHA chemical database. For these 13 target compounds, concentrations measured below the LOD were replaced with LOD/2 for statistical analysis. Naphthalene is not identified as a target compound based on our criteria, since almost all samples (n = 36) had naphthalene concentrations below the LOD. The 13 target VOCs are formaldehyde; acetaldehyde; propanal; methyl ethyl ketone (MEK); benzene; toluene; ethylbenzene; xylenes (the latter 4 referred to in combination as BTEX); styrene; dimethylformamide (DMF); 1,2-dichloropropane (1,2-DCP); 1,4-dichlorobenzene (1,4-DCB); and 1,2-dichloroethane (1,2-DCE). Their associated toxicity values are presented in Table 1. IUR and RfC values were obtained from the OEHHA or IRIS databases [36,37] for the following inhalation risk assessments.

### 3.2. Characteristics of target VOCs

To better understand the target VOCs listed in Table 1, we examined the concentrations of VOCs outdoors, and the concentrations of VOCs indoors during the sham intervention - this can provide a snapshot of the daily VOC concentrations in the homes of these asthmatic children in the absence of filtration. Note that although the HEPA and activated carbon filters, which are the most likely to affect concentrations of VOCs, are removed during sham filtration the pre-filter (consisted of coarse plastic meshes) is retained during this intervention. We also calculated the indoor to outdoor ratio (I/O) for each compound. This can serve as an indicator regarding the likelihood of whether the compounds originated indoors or outdoors.

### 3.2.1. Outdoor concentrations

Outdoor concentrations of the 13 target VOCs sampled during the entire study are summarized in Fig. 2, with data from both interventions combined. Most compounds had lognormal distribution, hence the median was selected to better represent central tendencies [44]. Toluene had the highest concentration outdoors with a median value of 8.6 μg/m$^3$, followed by xylenes (including m/p/o-xylenes, median: 5.2 μg/m$^3$), acetaldehyde (median: 4.6 μg/m$^3$) and formaldehyde (median: 3.8 μg/m$^3$). Outdoor concentrations of BTEX varied more than outdoor concentrations of aldehydes. This may be because BTEX measured outdoors are mainly exhausted from traffic [45] and the sampling time in this study (usually between 5 and 8am) included the morning traffic rush hour period; day-to-day variations in traffic likely led to this range of BTEX concentrations, while the aldehydes, which have different sources than the BTEX compounds, did not show as much temporal variation. Concentrations of VOCs measured outdoors are summarized in greater detail in Table S3 in the Supplementary data. Concentrations measured in this study were of the same magnitude as those from other studies conducted in multiple cities in China and the United States [7,44,46].

### 3.2.2. Indoor concentrations during sham intervention

During the sham intervention, the sum of concentrations indoors for the 13 target VOCs varied considerably among homes, from 34.3 to 472.9 μg/m$^3$, with a mean of 135.1 μg/m$^3$ and a median of 88.0 μg/m$^3$. As shown in Table 2 and Table 3, the indoor concentrations of individual compounds varied greatly in this study, especially for formaldehyde, acetaldehyde and 1,4-DCB. Variation in the concentrations measured can be attributed to differences in locations of the homes, interior decoration in the homes, and activities in each family that may contribute to the emission or generation of VOCs (e.g., cooking, cleaning, using personal care products). The median concentrations of formaldehyde, acetaldehyde and toluene, which had higher concentrations than other compounds, were 18.0, 14.0 and 12.1 μg/m$^3$, respectively. The indoor concentrations of formaldehyde, benzene and 1,2-DCP exceeded the reference concentration for chronic toxicity (RfC) as listed in Table 1 and 9 out of 13 target VOCs have potential inhalation cancer risks.

As shown in Table 2, the concentrations of VOCs in this study were similar to those measured in previous studies of VOCs in the homes of asthmatics. Specifically, formaldehyde concentrations in this study (mean ± standard deviation: 28.7 ± 28.3 μg/m$^3$) were similar to those measured by Huang et al. [48] in the bedrooms of 186 asthmatic...
children in Shanghai, China (mean ± standard deviation: 22.4 ± 20.8μg/m³). In addition, our values were of the same magnitude as measurements of 56 VOCs in the bedrooms of 126 asthmatic children made by Chin et al. [1] in Detroit, Michigan, USA, as listed in Table 2.

Based on a comparison of our values with measurements reported previously for the homes of non-asthmatics, most of the VOC concentrations indoors were lower in our study than in other residences. The median concentration of each VOC in this study was 54–88% lower than the median concentration in 8 renovated homes in Shanghai in Dai’s study [27], except for 1,2-DCP and benzene. The low concentrations of benzene in the newly renovated homes [27] may indicate a reduction in the use of benzene in decorative materials. Du et al. [7] summed the concentrations of VOCs published prior to August 2013 in multiple cities of China and Duan et al. [44] assessed the concentrations of VOCs in 50 homes in Beijing. Both studies reported higher concentrations indoors than those measured in our study, with the exception of acetaldehyde and 1,2-DCP. The higher 1,2-DCP value in this study can be due to the using of solvents and pesticide fumigant in these homes. In both our study and Chin’s study [1], concentrations of 1,4-DCB were highly variable, potentially due to the use of pest repellents and deodorants indoors [1].

3.2.3. I/O ratios during sham intervention

The ratio of indoor to outdoor concentrations (I/O ratio) during the sham intervention was calculated to identify the origin of each VOC [49]. If the I/O ratio is near unity (1 ± 0.5), it indicates that the compound arises primarily from outdoor sources [50]. If the I/O ratio is higher than 5, it indicates that the compound is mainly emitted by indoor sources [49]. In addition, the Wilcoxon matched pairs test was used to analyze if there was a significant difference between indoor and outdoor VOCs. The I/O ratios as well as the detection frequency of target VOCs in each location (i.e., indoor or outdoor) were used in combination to determine the origin of each VOC as shown in Table 4.

Table 2
Indoor concentrations of target VOCs in this study and other studies (μg/m³).

<table>
<thead>
<tr>
<th>Location/reference</th>
<th>Remarks</th>
<th>Sampling method</th>
<th>Sample size</th>
<th>Statistical parameter</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shanghai, China</td>
<td>Sham intervention</td>
<td>Active sampling for 90 min</td>
<td>20 homes</td>
<td>Median</td>
</tr>
<tr>
<td>Detroit, USA</td>
<td>Homes of asthmatics</td>
<td>Passive sampling for 7 days</td>
<td>126 homes</td>
<td>Median</td>
</tr>
<tr>
<td>Shanghai, China</td>
<td>Renovated within 1 year</td>
<td>Canister sampling for 45 min</td>
<td>8 homes</td>
<td>Median</td>
</tr>
<tr>
<td>Multiple cities, China</td>
<td>Review of 10 studies</td>
<td>Canister/active/passive sampling for 1 h–5 days</td>
<td>-</td>
<td>Median</td>
</tr>
<tr>
<td>Beijing, China</td>
<td>Canister/diffusive sampling for 24 h</td>
<td>-</td>
<td>50 homes</td>
<td>Median</td>
</tr>
</tbody>
</table>

Parameter | Sham filtration, μg/m³ | True filtration, μg/m³ | n S/T (n pairs) | P value
--- | --- | --- | --- | ---
Formaldehyde | 28.7 | 28.3 | 10.3 | 18 | 137.5 | 9.9 | 6.6 | 16 | 53.8 | 0.016 | 20/19 (19)
Acetaldehyde | 28.2 | 44.7 | 4.5 | 14 | 207.9 | 5.4 | 5.5 | 11.2 | 30 | 0.062 | 20/19 (19)
Toluene | 14.6 | 9.9 | BDL | 12 | 39.1 | 7.5 | 7.2 | BDL | 6.7 | 0.020 | 20/19 (19)
Xylenes | 6.9 | 4 | BDL | 6 | 13.7 | 3.1 | 2.5 | BDL | 2.3 | 0.005 | 20/19 (19)
MEK | 5.8 | 3.1 | BDL | 5 | 13 | 2.3 | 2.1 | BDL | BDL | 9.2 | 0.000 | 20/18 (18)
1,2-DCP | 4.4 | 2.7 | BDL | 5 | 8.5 | 3.8 | 2.3 | BDL | 3.4 | 0.480 | 7/10 (5)
DMF | 3.4 | 3 | BDL | 3 | 14.6 | 1.3 | 0.7 | BDL | BDL | 2.9 | 0.000 | 20/14 (14)
Ethylbenzene | 3 | 1.7 | BDL | 3 | 6.4 | 1.6 | 1.2 | BDL | BDL | 4.3 | 0.019 | 20/19 (19)
Styrene | 2.8 | 1.5 | BDL | 2.7 | 5.6 | 5.6 | 1 | 0 | BDL | BDL | 19/16 (16) | 0.001
Propanol | 3.8 | 5.9 | BDL | 2.5 | 26.8 | 1.7 | 1.2 | BDL | BDL | 4.5 | 0.023 | 18/13 (10)
Benzene | 3.1 | 1.9 | BDL | 2.4 | 7.4 | 2.6 | 2.4 | BDL | BDL | 2.3 | 0.632 | 18/19 (17)
1,4-DCB | 32 | 88.2 | BDL | 2.2 | 394.5 | 28.2 | 89.8 | BDL | BDL | 4.5 | 0.001 | 20/17 (17)
1,2-DCE | 2.2 | 1.6 | BDL | 2 | 6.9 | 3.5 | 6 | BDL | BDL | 2.2 | 0.850 | 19/19 (18)

Notes
BDL: below detection limit.
* n S/T (n pairs): number of homes with compound detected indoor under sham/true intervention and their paired number.
† For the difference by intervention status.
3.2.3.1. VOCs with dominating indoor sources. As shown in Table 4, the mean level. Boxes show 10th, 25th, 50th, 75th, and 90th percentile.

3.2.3.2. VOCs with both indoor and outdoor sources. This is in contrast to Zhou et al.’s study [24] in which they measured the BTEX concentrations in 12 residences in Tianjin, China in 2008 and found ratios much higher than 1. This difference between measurements taken almost a decade apart may be attributed to the decreasing use of BTEX in indoor products.

Chlorinated compounds had I/O ratios mostly near unity in this study. The high I/O ratio of 1,4-DCB (maximum of 81.5) in this study may result from the use of pest repellents and deodorants indoors [1]. 1,2-DCE had ratios around 1, and was detected mainly indoors.

3.3. Risk assessment results for the sham intervention

The cancer risks of 8 VOCs and non-cancer risks of 13 VOCs with published IUR and RfC parameters were assessed using Equations (3) and (4). With the probability distributions of the Ci, IURi, and RfCi, we applied Monte Carlo simulation to quantify their uncertainty in the risk assessments [7,27,43].

3.3.1. Age-specific cancer risk

According to the collected basic information (i.e., body weight, inhalation rate during sleep, nighttime sleep duration) of Chinese population, 13 age groups are considered: 0–1, 1–2, 2–3, 3–4, 4–5, 5–6, 6–9, 9–12, 12–15, 15–18, 18–45, 45–60, 60–70 years old. The detailed information about the 13 age groups are shown in Table S1 in the Supplementary data.

Younger people are more susceptible to toxic VOCs. Take formaldehyde for example, as shown in Fig. 3, when exposed to the same formaldehyde level at night for one year, higher cancer risk is posed to younger group. The cancer risks posed in children ages 0–2 years and 2–15 years are 25.7–26.4 and 3.9–7.1 times of that posed in people ages 18–45. Same conclusion can be found for other compounds.

To analyze the influence of ADAF, increased cancer risk after one year’s exposure to the mean formaldehyde level (i.e., 28.7 μg/m³) during sleep were calculated for different age groups. As shown in Fig. 3, although the application of ADAF enlarges the differences of risks posed in different age groups, it does not alter the decreasing trend of risk posed in older group.

3.3.2. Cancer risk-based ranking

Lifetime cancer risk of a compound is an accumulation of cancer risk at each age [27,54]. As shown in Fig. 4, the median total cancer risk obtained by summing up the risks of 8 VOCs is 1.56×10⁻⁴ (range: 4.31×10⁻⁵ to 5.98×10⁻⁴), meaning that we could expect 156 additional cases of cancer per million people exposed to the measured VOC levels during sleep. The total cancer risks calculated were all

---

**Table 4**
The I/O ratios of target VOCs under sham intervention. (n total = 20).

<table>
<thead>
<tr>
<th>Main source</th>
<th>Group</th>
<th>Pollutant</th>
<th>min</th>
<th>max</th>
<th>median</th>
<th>mean</th>
<th>sd</th>
<th>n pairs</th>
<th>n in²</th>
<th>n out¹</th>
<th>n n.d.²</th>
<th>P ³</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indoor</td>
<td>Carboxyls</td>
<td>Formaldehyde</td>
<td>0.99</td>
<td>42.97</td>
<td>4.79</td>
<td>7.22</td>
<td>9.28</td>
<td>20</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Acetaldehyde</td>
<td>0.94</td>
<td>35.24</td>
<td>2.97</td>
<td>4.71</td>
<td>7.42</td>
<td>20</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0.005</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Propional</td>
<td>1</td>
<td>3</td>
<td>1.13</td>
<td>1.53</td>
<td>1.78</td>
<td>17</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0.023</td>
</tr>
<tr>
<td></td>
<td></td>
<td>MEK</td>
<td>0.23</td>
<td>12.5</td>
<td>2.09</td>
<td>2.68</td>
<td>2.82</td>
<td>17</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0.023</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Styrene</td>
<td>0.65</td>
<td>5.1</td>
<td>2.7</td>
<td>2.65</td>
<td>1.37</td>
<td>11</td>
<td>8</td>
<td>1</td>
<td>0</td>
<td>0.273</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ethylbenzene</td>
<td>0.13</td>
<td>4.9</td>
<td>1.05</td>
<td>1.85</td>
<td>1.63</td>
<td>10</td>
<td>7</td>
<td>2</td>
<td>0</td>
<td>0.139</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Benzene</td>
<td>0.13</td>
<td>4.9</td>
<td>1.13</td>
<td>1.65</td>
<td>1.33</td>
<td>10</td>
<td>7</td>
<td>2</td>
<td>0</td>
<td>0.139</td>
</tr>
<tr>
<td>Both indoor and outdoor</td>
<td>BTEX</td>
<td>Xylenes</td>
<td>0.14</td>
<td>6.52</td>
<td>1.43</td>
<td>1.82</td>
<td>1.41</td>
<td>20</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0.179</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Toluene</td>
<td>0.2</td>
<td>12.1</td>
<td>1.4</td>
<td>2.13</td>
<td>2.54</td>
<td>20</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0.138</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ethylbenzene</td>
<td>0.13</td>
<td>4.9</td>
<td>1.05</td>
<td>1.85</td>
<td>1.63</td>
<td>10</td>
<td>7</td>
<td>2</td>
<td>0</td>
<td>0.139</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Styrene</td>
<td>0.65</td>
<td>5.1</td>
<td>2.7</td>
<td>2.65</td>
<td>1.37</td>
<td>11</td>
<td>8</td>
<td>1</td>
<td>0</td>
<td>0.273</td>
</tr>
<tr>
<td></td>
<td></td>
<td>DMF</td>
<td>0.34</td>
<td>5.5</td>
<td>2.6</td>
<td>2.51</td>
<td>1.86</td>
<td>11</td>
<td>9</td>
<td>0</td>
<td>0</td>
<td>0.179</td>
</tr>
</tbody>
</table>

**Notes.**

* n pairs: number of homes with compound detected both indoors and outdoors.
* n in: number of homes with compound only detected indoors.
* n out: number of homes with compound only detected outdoors.
* n n.d.: number of homes with compound neither detected indoors nor outdoors.
* P: P values of Wilcoxon matched pairs test for the relationship of indoor and outdoor VOCs.

![Fig. 3](image-url) Increased cancer risks of formaldehyde exposure during sleep for one year for different age groups. The dot means ADAFs (i.e., age-dependent adjustment factors) were not applied during calculation under mean level (i.e., 28.7 μg/m³), the square means ADAFs were applied during calculation under mean level. Boxes show 10th, 25th, 50th, 75th, and 90th percentile.
above the US EPA proposed acceptable cancer risk of $1 \times 10^{-6}$ [55]. For individual compounds, formaldehyde, 1,4-DCB, benzene, acetaldheyde, 1,2-DCE, 1,2-DCP and ethylbenzene presented a median cancer risk above the acceptable level. Formaldehyde presented the highest median cancer risk at $6.75 \times 10^{-5}$ and accounts for 43% of the total inhalation cancer risk, followed by 1,4-DCB (19%), benzene (13%), acetaldheyde (10%), 1,2-DCE (7%), 1,2-DCP (6%) and ethylbenzene (2%). Formaldehyde and benzene are listed as target pollutants in China's national indoor air quality standard [47]. In this study, the inhalation cancer risk for 1,4-DCB is higher than that for benzene, and in combination, formaldehyde, 1,4-DCB and benzene account for 75% of the cancer risk. Thus, priority should be given to these three compounds to protect asthmatic children. We suggest that 1,4-DCB should be added to China's national indoor air quality standards to limit the use of products that may be emitting 1,4-DCB.

Direct comparisons with other studies on inhalation cancer risks are difficult because studies include different compounds, and risk assessments are calculated using different approaches and with different populations in mind. Instead of a direct comparison, we have compared the ranking of the target VOCs in each study. Cancer risks from some toxic VOCs for general population have been estimated by Sax et al. [56] and Loh et al. [57] in the United States. Forty-six subjects in New York City and 41 in Los Angeles participated in Sax et al.’s study, while Loh et al.’s study is based on modeling. They both found that formaldehyde and 1,4-DCB were the primary risk contributors, similar to the results in this study. The risk ranking of the carcinogenic VOCs also agrees with Chin's study [1] in homes of 126 asthmatic children in Detroit, Michigan, USA, with 1,4-DCB > benzene > 1,2-DCE > ethylbenzene > styrene. Formaldehyde, acetaldheyde and 1,2-DCP were not studied by Chin. Sarigiannis et al. [39] reviewed and compiled measurements of BTEX, styrene and carbonyls in indoor environments over Europe. The literature research covered studies published from 1990 to 2008, with more than 2000 samples for each compounds. Sarigiannis et al. reported that the associated cancer risks of BTEX and carbonyls are always higher than the acceptable risk threshold (i.e., $10^{-6}$), while that's not the case for styrene. For the target VOCs in this study, the inhalation cancer risk-based ranking agrees well with that in Du's study [7] for females and males in urban China – for both studies, formaldehyde, 1,4-DCB and benzene were the primary contributors to cancer risk.

### 3.3.3. Non-cancer risk-based ranking

The estimated non-cancer risks for DMF, xylenes, toluene, 1,4-DCB, 1,2-DCE, ethylbenzene, styrene and MEK were below the inhalation non-cancer risk threshold of 1 even at the 90th percentile (Fig. 5), indicating that these compounds may not be responsible for non-cancer health effects at the concentrations measured in these homes. In contrast, for formaldehyde, acetaldehyde, and 1,2-DCP - which can affect the respiratory system, and benzene - with known effects on developmental, hematologic, immune and nervous systems [36,37], the risks exceeded the threshold (1) for most of the families. In other studies, the non-cancer risks of VOCs (except formaldehyde) rarely reach or exceed the most conservative safety threshold 1, even in newly decorated homes [7,27,39]. In contrast to what has been previously documented, formaldehyde, acetaldehyde, 1,2-DCP and benzene were measured in our studied homes at levels that may present health risk to occupants during sleep. As such, actions should be taken to prioritize reductions in the concentrations of these four compounds in the home as they may be adversely affecting the health of occupants.

### 3.4. Performance of air cleaner on removing target VOCs

#### 3.4.1. Variations in concentrations

Concentrations of most target VOCs were 1.4–4.4 times higher during the sham intervention than during the true intervention (Table 3). Wilcoxon matched pairs tests were used to determine if these differences were significant. The concentration of TVOC was, on average, 39% lower with the operation of the true air cleaner (mean value decreased from 338.2 μg/m³ to 155.8 μg/m³). The use of a true air cleaner appeared to reduce the VOC concentrations in most of the families, although it should be noted that the true and sham interventions were separated by a number of weeks. For 9 of 13 target VOCs (formaldehyde, toluene, xylenes, MEK, DMF, ethylbenzene, styrene, propanal, and 1,4-DCB) there were significant differences ($p < 0.05$) in concentrations indoors with the use of a true air cleaner, with these concentrations being lower during the true than the sham intervention. The mean and median concentrations of formaldehyde, toluene, xylenes, MEK, DMF, ethylbenzene, styrene, propanal, and 1,4-DCB with and without the operation of the true air cleaner are shown in Table 3. Acetaldehyde, 1,2-DCP, benzene and 1,2-DCE did not differ significantly by intervention status.

Xu et al. [33] reported an average removal effectiveness of 59% (from 1135 μg/m³ to 468 μg/m³) for TVOC by an air cleaning/ventilating unit (HEPAiRx) in a child's bedroom but did not analyze the intervention effect for individual VOCs. Batterman et al. [32] and Chin et al. [1] characterized the effect of using a HEPA filter on the respiratory health of asthmatic children in 126 homes in Detroit, USA. Fifty six VOCs were quantified and there were no significant differences detected by filtration status [1]. This apparent lack of VOC removal may be due to the fact that HEPA filtration is designed mainly for removing particles rather than VOCs. The air cleaner we used in this study included both a HEPA filter and an activated carbon filter which
likely accounts for the higher removal effectiveness observed in our study.

Gallego et al. [58] evaluated the performance of a commercially available activated carbon filter on the removal of VOCs and assessed how performance differed with different pollutant concentrations and under different relative humidity. For the sampling events with similar relative humidity and input concentrations as in this study, the single pass efficiencies for BTEX, styrene, MEK and DMF in Gallego’s study were between 55 and 91%, although the temperature in their study (22–30°C) was higher than in this study (mean ± standard deviation: 19 ± 2.3°C) and they did not measure the removal effectiveness of the air filter for the whole room.

3.4.2. Variations in health risks

With the use of a true air cleaner, the median total inhalation cancer risk was 1.25 × 10⁻⁴, compared to 1.56 × 10⁻⁴ during the sham intervention, or 20% lower during the true intervention (Fig. 6). The inhalation cancer risk for most compounds (1,2-DCP and 1,2-DCE excluded) was lower with the use of a true air cleaner, but in general the health risks were still relatively high. For example, the median cancer risk of formaldehyde reduced from 6.75 × 10⁻⁵ to 5.34 × 10⁻⁵ after the use of true air cleaner, but it is still an order of magnitude higher than the acceptable cancer risk of 1 × 10⁻⁶ during true filtration. The changes in non-cancer risks for formaldehyde, acetalddehyde, benzene and 1,2-DCP during the true and sham interventions were similar to what was observed for the cancer risks (Fig. 7).

Even with the lower indoor concentrations with the use of true air cleaner, formaldehyde, acetalddehyde, 1,2-DCP and benzene still exceeded the inhalation non-cancer risk threshold of 1 in most homes, and formaldehyde, 1,4-DCB and benzene still ranked top 3 for inhalation cancer risks. Based on our source identification, 1,2-DCP, benzene and 1,4-DCB likely have both indoor and outdoor sources, while formaldehyde and acetalddehyde have dominant indoor sources. As some of these compounds may be emitted by products used in the home, individuals can take action to minimize the use of products that may be emitting these compounds or choose products with lower emissions of VOCs. To address this issue on a larger scale, the government could set restrictions on the use of these harmful compounds, and specifically formaldehyde, acetalddehyde, 1,2-DCP, benzene and 1,4-DCB, in products.

3.5. Limitations and perspectives

Quantifying and understanding VOCs in the home environment can be challenging due to an abundance of sources and the complexity of reactions indoors that lead to the degradation and/or the formation of VOCs. While there were no large-scale changes (e.g., addition of furniture, renovation) in any of the homes during our study, it is possible that other smaller-scale differences in sources or behaviours (e.g., use of personal care products, cleaning, cooking) occurred before or during the sleeping periods and contributed to some of the variations in the measurements independent of a potential effect of filtration. To minimize the influence of human activities on VOCs measured, all samples were collected in the early morning before the family got up and started their daily routine. In future studies, a rigorous inventory of home products or a time activity guide to understand factors affecting VOCs in the home would be useful to better account for potential differences in activity and product use during the two intervention periods.

Changes in temperature and relative humidity indoors may impact the emission rates of VOCs from building materials [59,60]. Using a Wilcoxon matched pairs test, we determined that there were no significant differences in temperature and relative humidity by intervention status (Table 5). Ventilation rate also has a significant impact on VOC removal in buildings. During the interventions, the participants and their families were instructed to keep doors and windows in their bedroom closed. The overnight ventilation rates were measured in this study by CO2 tracer gas method, using sleeping people as constant CO2 sources [61]. The measured results show that the ventilation rates can be taken as the same for the two interventions (0.4 ± 0.4/h and 0.4 ± 0.3/h for true and sham interventions, respectively). The distribution of the measured ventilation rates for true and sham interventions is shown in Fig. S2 in the Supplementary data.

In this study, the inhalation cancer risks during sleep were expressed as the output of IUR, indoor VOC concentration (Cin), and the proportion of time spent in sleeping (t). The inhalation non-cancer risks were expressed as the output of RfC, Cin and t. High uncertainty exists in risk assessments especially when only single point values of relevant inputs are used to estimate the risk for a population [7,43]. To address this uncertainty, we used Monte Carlo simulations based on the given probability distributions of the input factors. Fig. S3 and Fig. S4 in the Supplementary data show the contributions of IUR, RfC and indoor VOC concentrations to the variance of risk assessments. The results

### Table 5

<table>
<thead>
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<th>Parameter</th>
<th>SHAPE</th>
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<th>Wilcoxon test</th>
</tr>
</thead>
<tbody>
<tr>
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<td>Mean</td>
<td>SD</td>
<td>Median</td>
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<tr>
<td>T (°C)</td>
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<td>3.2</td>
<td>19.0</td>
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<tr>
<td>RH (%)</td>
<td>54.7</td>
<td>6.9</td>
<td>55.7</td>
</tr>
</tbody>
</table>
indicate that the indoor concentration has greater impact on inhalation health risks. The limitation of this study is that only exposure during nighttime sleep were considered, exposures in other micro-environments (e.g., at school, in transit, in other rooms at home) and other periods were not assessed. Larger scale measurements are needed to refine the exposure assessments in the future.

IUR and RfC also showed great contribution to the risk assessments for benzene, 1,2-DCP and 1,2-DCB because of their low thresholds. The IUR and RfC are derived by the US EPA for the general population with benzene, 1,2-DCP and 1,2-DCE because of their low thresholds. The limitation of this study is that only exposure assessments in the indoor environment have greater impact on inhalation health risks of toxic VOCs measured during nighttime sleep. This study provides insight not only about the concentrations and associated inhalation health risks of toxic VOCs measured during nighttime sleep but also the performance of an air cleaner as a means to reduce individual toxic VOC in the real home environment. It highlights the need for reduction in the exposure to toxic VOCs.

In the homes tested, we found that formaldehyde accounts for 43% of the total inhalation cancer risk (median: 1.56 × 10−4) during nighttime sleep, followed by 1,4-DCB (19%), benzene (13%), acetaldehyde (10%), 1,2-DCB (7%), 1,2-DCP (6%) and ethylbenzene (2%). Most VOC concentrations were lower in these homes than in the homes of non-asthmatics, with the exception of acetaldehyde, 1,2-DCP, benzene and 1,4-DCB. Formaldehyde, acetaldehyde, 1,2-DCP and benzene exceeded the non-cancer risk threshold in most homes, which might exacerbate asthma as these compounds have the potential to damage the respiratory and immune systems. To protect occupants, actions should be taken to prioritize reductions in formaldehyde, acetaldehyde, 1,2-DCP, benzene and 1,4-DCB – these compounds account for more than 95% of the total non-cancer risk and 90% of the total cancer risk in these homes. TVOC concentrations indoors were 39% lower with the use of a true air cleaner than during the sham intervention, although even at these lower concentrations, the top 5 priority VOCs still presented high inhalation risks in this study. Thus, improvements in the control of sources of VOCs indoors and further research on the performance of individual air cleaners are imminently needed. Given the risk associated with these compounds, priority should be given to reducing the concentrations of formaldehyde, acetaldehyde, 1,2-DCP, benzene and 1,4-DCB in the real home environment. We suggest that 1,4-DCB should be added to China's national indoor air quality standards to limit the use of products that may be emitting 1,4-DCB. In addition to government policies improvement, individuals should use products with lower emissions of these 5 VOCs. There is imminent need for improvements in air cleaning technologies in removing these 5 VOCs.

Acknowledgements

We gratefully acknowledge our participants in this study and our research staff members who helped with field work. Thanks to Aika Davis and other team members from Underwriters Laboratories Inc. for providing analytical quantification of the VOCs collected. Thanks to Corinne Mandin for her advice in the data analysis. The research was supported by Underwriters Laboratories Inc, the National Key Research and Development Program of China (Grant No. 2017YFC0702700) and the Natural Science Foundation of China (Nos. 51420105010, 51722087, and 51521005).

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.buildenv.2019.04.047.

References

[18] J.L. Zhang, C.J. Sun, W. Liu, Z.J. Zou, Y.P. Zhang, B.Z. Li, Z.H. Zhao, Q.H. Deng, L. Fang, et al., Development Program of China (Grant No. 2017YFC0702700) and the Natural Science Foundation of China (Nos. 51420105010, 51722087, and 51521005).

Acknowledgements

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