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Modeled Exposure Assessment via Inhalation and Dermal Pathways to Airborne Semivolatile Organic Compounds (SVOCs) in Residences

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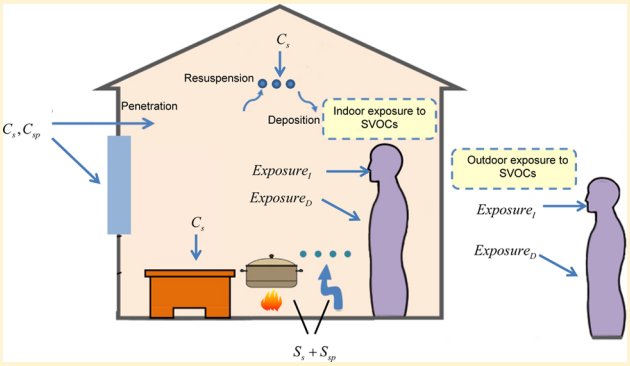
Supporting Information

ABSTRACT: Exposure to airborne semivolatile organic compounds (SVOCs) in indoor and outdoor environments of humans may lead to adverse health risks. Thus, we established a model to evaluate exposure to airborne SVOCs. In this model, SVOCs phase-specific concentrations were estimated by a kinetic partition model accounting for particle dynamics. The exposure pathways to airborne SVOCs included inhalation exposure to gas- and particle-phases, dermal exposure by direct gas-to-skin pathway and dermal exposure by direct particle deposition. Exposures of defined "reference people" to two typical classifications of SVOCs, one generated from both indoor and outdoor sources, represented by polycyclic aromatic hydrocarbons (PAHs), and the other generated mainly from only indoor sources, represented by di 2-ethylhexyl phthalate (DEHP), were analyzed as an example application of the model. For PAHs with higher volatility, inhalation exposure to gas-phase, ranging from 6.03 to 16.4 ng/kg/d, accounted for the most of the exposure to the airborne phases. For PAHs with lower volatility, inhalation exposure to particle-phase, ranging from 1.48 to 1.53 ng/kg/d, was the most important exposure pathway. As for DEHP, dermal exposure via direct gas-to-skin pathway was 460 ng/kg/d, which was the most striking exposure pathway when the barrier effect of clothing was neglected.



INTRODUCTION

More and more chemicals, including semivolatile organic compounds (SVOCs), are introduced into humans' daily life. With the vapor pressures ranging from 10^{-9} to 10 Pa,[1] SVOCs exist not only in gas-phase but also partition with other substances, such as airborne particles and sorption surfaces. SVOCs are omnipresent organic compounds in both indoor and outdoor environments due to multiple sources. A majority of the widely used plasticizers and fire retardants, as well as the main ingredients of pesticides, are SVOCs. Moreover, SVOCs are also generated from anthropogenic activities, such as various combustion activities. Considering the fact that people spend roughly 90% of their time in indoor environments,[2] exposure to indoor SVOCs needs more consideration. Some indoor SVOCs are generated mainly indoors, such as di 2-ethylhexyl phthalate (DEHP), which is added into building materials and commodities as plasticizer. Some other indoor SVOCs are generated not only from indoor but also outdoor sources. Atmospheric polycyclic aromatic hydrocarbons (PAHs) can enter into indoor environments through building envelopes. Apart from outdoor generation, indoor activities, such as cooking and smoking, also contribute to indoor airborne PAHs.

Previous epidemiological and toxicological studies have confirmed that exposure to airborne SVOCs (sum of gas- and particle-phases) may lead to adverse health risks of human beings. For example, several PAHs have been realized to cause animal cancers, gene mutations and reproductive problems of human beings by epidemiological studies.[3,4] High dermal exposure to PAHs was claimed to be responsible for the increasing risk of skin cancer under some occupational situations.[5] Inhalation exposure to DEHP, especially inhalation exposure to particle-phase DEHP, was believed to correlate with increasing diagnosed asthma of children.[6] Some phthalates were claimed to be likely to disrupt endocrine and affect reproductive system from toxicological concern.[7−9] Consequently, appropriate assessment of exposure to airborne SVOCs is indispensable to evaluate the adverse effect on human being's health.

Exposure to SVOCs is normally studied by personal exposure concentration monitoring and biomarkers measurements, which reflect the realistic conditions and provide substantially important information for studying exposure. However, these methods are quite time-consuming and expensive. Modeling is a more flexible tool. It costs less time and money and can be used to study exposure to airborne pollutants after calibration. Guo has built up nonsteady state models for estimating SVOCs phase-specific concentrations.[10,11] The partition between gas-phase SVOCs and airborne particles and the partition between gas-phase SVOCs and sorption surfaces were described in

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separate models. Little et al.[12] set up a model for rapid estimation of exposure to SVOCs indoors. However, they did not account for the airborne particle dynamics, which can affect indoor exposure to airborne SVOCs.[13] In addition, they applied the linear equilibrium assumption for SVOCs partitioning between different phases, which may incorrectly predict SVOCs phase-specific concentration and thus distort the relative importance of exposure pathways. Xu et al.[14] modeled human potential exposure to DEHP via inhalation, ingestion and dermal pathways with a kinetic partition model for SVOCs. Nevertheless, this study also failed to account for the impact of airborne particle dynamics. The influence of ventilation and particle deposition on particle-phase SVOCs was also ignored. Last but not least, apart from direct contact event, mass transfer via direct gas-to-skin pathway and direct particle deposition also contribute to the dermal exposure,[15] which was not considered or partially considered in the researches mentioned above.

In this study, an integrated framework was established by combining a kinetic partition model for SVOCs phase-specific concentrations estimation and an exposure assessment model. The airborne particle dynamics was taken into consideration. SVOCs generated from both indoor and outdoor sources, represented by PAHs, and SVOCs generated mainly from indoor sources, represented by DEHP, were taken into account. The USEPA (United States Environmental Protection Agency) has listed 16 PAH congeners as priority pollutants, among which phenanthrene (Phe), pyrene (Pyr), benzo(a)pyrene (BaP), and benzo[g,h,i]perylene (BghiP) were chosen for this study. This is because they are the most abundant tricyclic, tetracyclic, pentacyclic, and hexacyclic PAHs in indoor air according to previous measurements conducted in Chinese residences.[16] DEHP was chosen as the target pollutant because of its wide use[17] and the adverse health risks mentioned above. The counted exposure pathways included inhalation exposure to gas- and particle phases, dermal exposure by gas-to-skin pathway and dermal exposure by direct particle deposition. By constructing the model, the exposure to airborne SVOCs could be more reasonably estimated with less money and time consumption.

MATERIALS AND METHODS

Indoor SVOCs Concentrations Modeling. In our previous study,[18] a kinetic partition model was established to calculate indoor SVOCs gas-phase (C_s) and particle-phase (C_{sp}) concentrations by the following equations:

$$V\frac{dC_p}{dt} = Q_p(PC_{p,o} - C_p) - v_d A C_p + RMA_f \quad (1)$$

$$V\frac{dC_s}{dt} + V\frac{dC_{sp}}{dt} + A\frac{dC_{surf}}{dt} = Q_p(C_{s,o} + PC_{sp,o} - C_s - C_{sp}) \quad (2)$$

$$\frac{dC_{sp}}{dt} = h_{mp}A_{pn}N_{pn}\left(C_s - \frac{C_{sp}}{C_pK_p}\right) + \frac{Q_p}{V}(PC_{sp,o} - C_{sp}) \quad (3)$$

$$\frac{dC_{surf}}{dt} = h_m\left(C_s - \frac{C_{surf}}{K_{surf}}\right) \quad (4)$$

In this model, no indoor source was considered. Equations 1 and 2 describe the mass conservation of indoor airborne particles (C_p) and indoor SVOCs, respectively. The influence of ventilation (Q_p) and particle dynamics, including penetration (P), deposition (v_d) and resuspension (R) were considered. Equation 3 describes the accumulation of indoor particle-phase SVOCs. The first term on the right-hand side represents the dynamic mass transfer between gas-phase SVOCs and airborne particles. The second term on the right-hand side represents the impact of ventilation. The kinetic partition process between gas-phase SVOCs and sorption surfaces is described by eq 4, where K_{surf} is the coefficient describing SVOCs partitioning between surfaces and gas-phase (m), C_{surf} is the surfaces concentration of SVOCs (ng/m^2). However, particle-phase concentrations of SVOCs are also affected by particle deposition, which was not considered in this model. Moreover, indoor source of airborne particles (S_p) and SVOCs (S_s, S_{sp}) should be included to make the model more general. As a result, eqs 1−3 were rephrased in the present study to account for these influences, which are expressed as

$$V\frac{dC_p}{dt} = Q_p(PC_{p,o} - C_p) - v_d A C_p + RMA_f + S_p \quad (5)$$

$$V\frac{dC_s}{dt} + V\frac{dC_{sp}}{dt} + A\frac{dC_{surf}}{dt} = Q_p(C_{s,o} + PC_{sp,o} - C_s - C_{sp}) - v_d A C_{sp} + (S_s + S_{sp}) \quad (6)$$

$$\frac{dC_{sp}}{dt} = h_{mp}A_{pn}N_{pn}\left(C_s - \frac{C_{sp}}{C_pK_p}\right) + \frac{Q_p}{V}(PC_{sp,o} - C_{sp}) - \frac{v_d A}{V}C_{sp} + \frac{S_{sp}}{V} \quad (7)$$

Consequently, the indoor phase-specific concentrations of SVOCs can be estimated by eqs 4−7. The model was previously validated[18] with experimental measurements of indoor PAH phase-specific concentrations reported by Kamens et al.[19] In addition, Benning et al.[20] has conducted an experiment measuring gas- and particle-phase DEHP within a chamber, the results of which were in good agreement with the predicted results by our model, which is shown in the Supporting Information Figure S1. Thus, the rephrased kinetic partition model was judged to be reasonable and reliable to predict indoor SVOCs phase-specific concentrations.

Exposure Assessment Modeling. To study the typical exposure conditions, here we defined the "reference people" who live in the typical residence in Beijing. The "reference people" included typical male adult, typical female adult and typical adult to study the influence of gender. Additionally, the "reference people" were also set up as typical child before school from 0 to 3, typical child at school from 4 to 14, typical adult from 15 to 64, and typical elderly older than 64 to study the influence of age. The "reference people" were all set as nonsmokers. With compiled atmospheric SVOCs concentrations from former literatures, modeled indoor SVOCs phases-specific concentrations and exposure factors, exposure of "reference people" to airborne SVOCs via different pathways can be evaluated by the exposure assessment models, which are described as follows.

The inhalation exposure to gas-phase ($exposure_{I,s}$) and particle-phase ($exposure_{I,sp}$) SVOCs can be assessed by the following equations:[21]

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$$\text{exposure}_{I,s} = \frac{C_s \cdot IR \cdot ED_i + C_{s,o} \cdot IR \cdot ED_o}{24 \cdot BW} \quad (8)$$

$$\text{exposure}_{I,sp} = \frac{C_{sp} \cdot IR \cdot ED_i + C_{sp,o} \cdot IR \cdot ED_o}{24 \cdot BW} \quad (9)$$

where IR is the long-term inhalation rate (m^3/d), ED_i is the indoor exposure duration (h/d), ED_o is the outdoor exposure duration (h/d), and BW is the Body weight (kg).

As for the dermal exposure, it was defined as the amount of SVOCs transported onto skin surfaces in this study. The dermal exposure caused by direct gas-to-skin pathway ($\text{exposure}_{D,s}$) and particle deposition ($\text{exposure}_{D,sp}$) can be evaluated by the following equations:[22]

$$\text{exposure}_{D,s} = \frac{J_{s,i} \cdot SA \cdot f_{SA} \cdot ED_i + J_{s,o} \cdot SA \cdot f_{SA} \cdot ED_o}{BW} \quad (10)$$

$$\text{exposure}_{D,sp} = \frac{J_{sp,i} \cdot SA \cdot f_{SA} \cdot ED_i + J_{sp,o} \cdot SA \cdot f_{SA} \cdot ED_o}{BW} \quad (11)$$

where SA is the skin surface area (m^2) and f_{SA} is the fraction of the skin directly exposed to the air.

For gas-phase, Weschler and Nazaroff[15] have constructed a model to estimate the mass flux of SVOCs transported from air-to-skin-to-blood for steady state. In that model, the mass flux of gas-phase SVOCs transported from the bulk air onto the skin ($J_{s,i}$, $J_{s,o}$) was assumed to be the same as the mass flux of gas-phase SVOCs transported from the bulk air to the blood, which can be calculated using the following equations:

$$J_{s,i} = C_s \cdot k_s$$
$$J_{s,o} = C_{s,o} \cdot k_s \quad (12)$$

The overall permeability coefficient of gas-phase SVOCs from the ambient air through the skin to the blood (k_s) can be calculated as follows:

$$\frac{1}{k_s} = \frac{1}{v_g} + \frac{1}{k_{p\_cb}} + \frac{1}{k_{p\_eb}} \quad (13)$$

where v_g is the external mass transport rate of gas-phase SVOCs from the bulk air through the boundary layer adjacent to the skin (m/h), k_{p_cb} is the permeability coefficient from the boundary layer adjacent to the skin across the stratum corneum to the viable epidermis (m/h), and k_{p_eb} is the permeability coefficient across the viable epidermis to the capillary dermis (m/h).

SVOC-bound particles deposit directly onto human body. The corresponding particle deposition velocity ($v_{d,h}$) is the key parameter quantitatively scaling this transfer rate, with which the mass flux of particle-phase SVOCs transported from the air onto the skin ($J_{sp,i}$, $J_{sp,o}$) can be quantified as follows:

$$J_{sp,i} = C_{sp} \cdot v_{d,h}$$
$$J_{sp,o} = C_{sp,o} \cdot v_{d,h} \quad (14)$$

We previously studied the deposition velocities of particles from 0.01 to 10 μm onto human body surfaces of three typical scenarios, including transition seasons, summer and winter,[23] which can be utilized for the exposure estimation. The fraction of the skin in contact with the air was assumed to be unity, which is discussed in detail in the later section.

Input Parameters and Scenarios. To estimate the indoor SVOCs phase-specific concentrations, the input parameters of eqs 4−7, including aerodynamic parameters of particles, partition coefficients of SVOCs, mass transfer coefficients of gas-phase SVOCs, source strength and window open parameters, were appropriately set according to creditable references. The details were described in the Supporting Information.

Regarding to the atmospheric PAHs concentrations, there existed a gap between the previous literature results and our need. Atmospheric daily PAHs concentrations are assumed to be more appropriate than seasonal ones for indoor PAHs concentrations estimation.[18] However, it is quite difficult to get atmospheric daily PAHs concentrations at present. Therefore, effort was made to acquire the atmospheric daily PAHs concentrations based on some logical assumptions. First, PAHs were considered to combine only with $PM_{2.5}$. Size distributions of atmospheric particle-phase PAHs were measured in Beijing by Zhou et al.[24] and it was reported that more than 80% of the PAHs mass was bound to PM_2, under which circumstances the assumption was reasonable. Second, the atmospheric daily particle-phase PAHs concentrations were assumed to be proportional to the corresponding atmospheric daily $PM_{2.5}$ concentrations, which can be described as the following equation:

$$\frac{C_{sp,o,1}}{C_{sp,o,2}} = \frac{C_{p,o,2.5,1}}{C_{p,o,2.5,2}} \quad (15)$$

where $C_{p,o,2.5}$ is the atmospheric $PM_{2.5}$ concentration (ng/m^3).

Last but not least, atmospheric gas- and particle-phases PAHs were hypothesized to be at equilibrium. Thus, the correlation between the gas- and particle-phases PAHs can be represented as

$$K_p C_{p,o,2.5} C_{s,o} = C_{sp,o} \quad (16)$$

With atmospheric $PM_{2.5}$ concentration as well as corresponding gas-particle partition coefficient, ratio between gas- and particle-phases PAHs can be calculated. Then, ratio among atmospheric daily airborne PAHs concentrations ($C_{airborne}$) can be described as

$$\frac{C_{airborne,1}}{C_{airborne,2}} = \frac{C_{sp,o,1} + C_{s,o,1}}{C_{sp,o,2} + C_{s,o,2}} = \frac{C_{sp,o,1} + C_{sp,o,1}/C_{p,o,2.5,1}K_{p,1}}{C_{sp,o,2} + C_{sp,o,2}/C_{p,o,2.5,2}K_{p,2}} = \frac{C_{sp,o,1} + C_{sp,o,1}/C_{p,o,2.5,1}K_{p,1}}{C_{sp,o,1}C_{p,o,2.5,2}/C_{p,o,2.5,1} + C_{sp,o,1}/C_{p,o,2.5,1}K_{p,2}} \quad (17)$$

Zhou and Zhao[25] have obtained the atmospheric seasonal average concentrations of the studied PAHs in Beijing by statistics analyzing previous studies. Thus, atmospheric daily PAHs airborne concentrations can be calculated with atmospheric seasonal PAHs airborne concentrations and proportional relations among the daily PAHs airborne concentration of the same season obtained by eq 17. Once atmospheric daily PAHs airborne concentrations were acquired, corresponding PAHs phase-specific concentrations can be worked out with eq 16. Measuring daily atmospheric PAHs concentrations is quite challenging at this phase. The preliminary assumption made in this study was the first step

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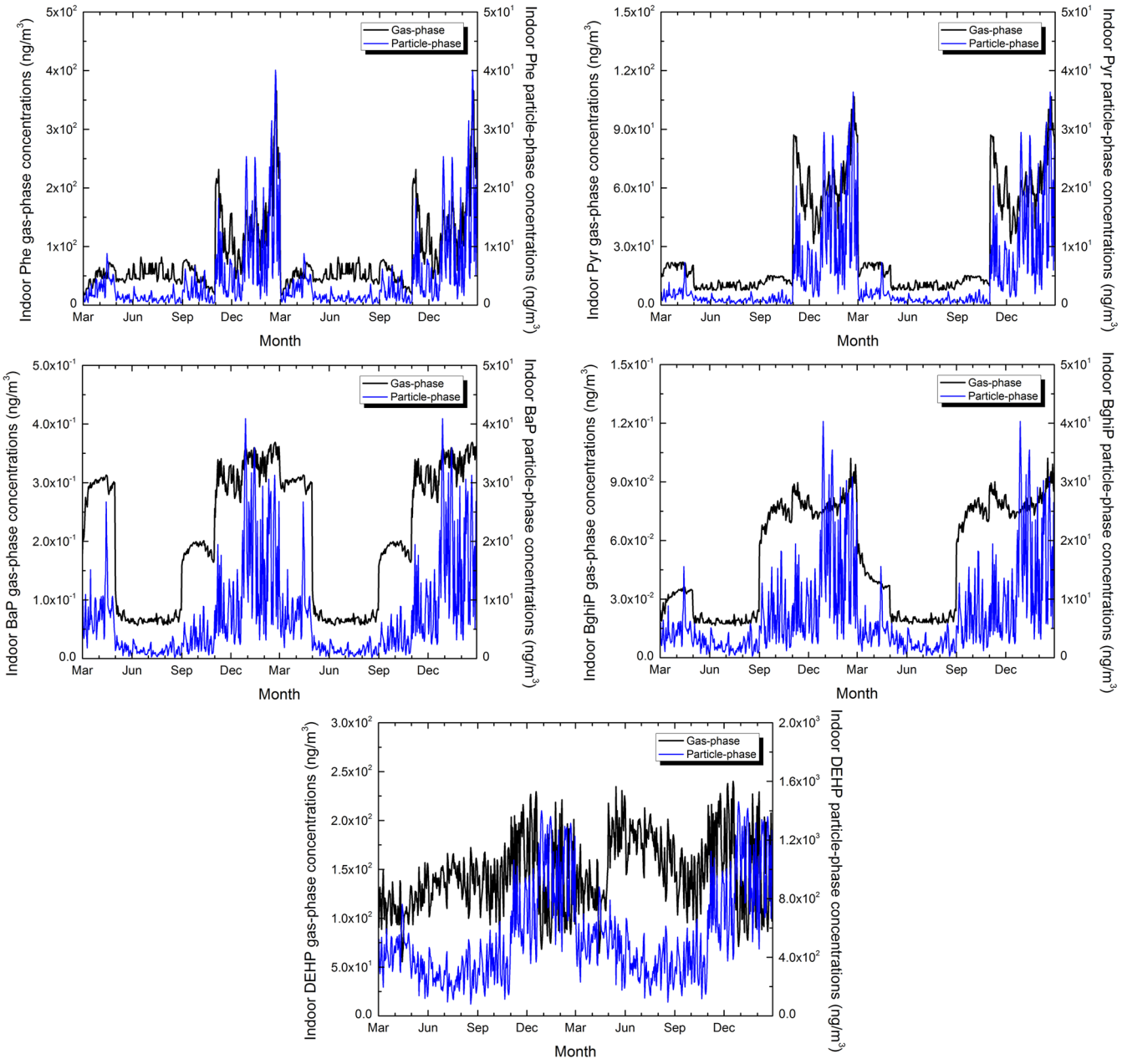

Figure 1. Indoor daily SVOCs phase-specific concentrations. (a) Phe, (b) Pyr, (c) BaP, (d) BghiP, (e) DEHP.

Table 1. Average Daily Indoor Phase-Specific Concentrations of the Studied SVOCs

	current study			reference[16]		reference[27]		reference[29]	
				$C_{airborne}$(ng/m^3)		$C_{airborne}$(ng/m^3)		$C_{airborne}$(ng/m^3)	
compound	C_s(ng/m^3)	C_{sp}(ng/m^3)	$C_{airborne}$(ng/m^3)	minumum	maximum	minimum	maximum	minimum	maximum
Phe	81.8	4.38	86.2	21.1	2103	135.2	963.4		
Pyr	29.7	4.58	34.3	3.04	126	3.6	272.6		
BaP	0.210	6.90	7.11	0.109	12.4	7.7	380.3		
BghiP	0.051	7.12	7.17	0.163	10.5	13.8	442		
DEHP	145	545	690					11.8	1660

to get atmospheric daily concentrations of PAHs, which is acceptable considering the lack of such research.

To estimate exposure to airborne SVOCs with eq 8-11, exposure factors, which evaluate the exposure behaviors, such as exposure pathways, durations etc., need to be reasonably determined. A nation-wide survey was conducted focusing on the exposure factors that included Beijing residents.[26] Thus, the corresponding exposure factors from the survey were applied for the "reference people". Details about determining exposure factors are shown in the Supporting Information.

A typical residence for a family of three in Beijing, China was set up to estimate indoor SVOCs concentrations. The

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description of the residence is shown in the Supporting Information Table S2. To make the scenario more general, nonsmoking residence was set up for the example application of the model, under which circumstances cooking was considered as the only indoor source of airborne particle. Indoor airborne PAHs were set to come from the atmosphere as well as indoor cooking activity. Indoor airborne DEHP was set to be generated by the emission from indoor vinyl flooring. It is noteworthy that the influence of other indoor sources can be evaluated by the model once the detail information on other sources can be learnt.

RESULTS AND DISCUSSION

Indoor SVOCs Phase-specific Concentrations. With the determined input parameters, two-year (730 days) daily indoor PAHs and DEHP phase-specific concentrations of the typical residence were estimated by eq 4-7, the results of which are shown in Figure 1. The estimated average indoor phase-specific concentrations of the studied SVOCs are shown in Table 1. Considering the fact that the input parameters may vary from scenario to scenario, sensitivity analysis of several modeled parameters, including ventilation rate, partition coefficients, source strength, was conducted in the Supporting Information.

Zhu et al.[16] have measured PAHs of the residential air in Hangzhou. The major sample sites of their measurements were nonsmoking families, being consistent with this study. Lv et al.[27] have conducted experiments to analyze indoor PAHs concentrations in Xuanwei and Fuyuan, China. The results of both previous measurements were compared with the predicted concentrations of our study. As it is shown in Table 1, the predicted average PAHs airborne concentrations fell into the range of the reported data. Hangzhou is a provincial capital located in East China. Xuanwei and Fuyuan are in Yunnan Province and were claimed to be suffering from PAHs pollution problems since 1980s.[28] Accordingly, it is believable that the results of these two studies offered a reasonable range of indoor PAHs airborne concentration. Moreover, the mass fraction of particle-phase within the total airborne concentration of Phe, Pyr, BaP, and BghiP were 0.03, 0.31, 0.95, and 0.96 according to Zhu et al.'s measurement.[16] Meanwhile, the average mass fraction of particle-phase Phe, Pyr, BaP, and BghiP were 0.05, 0.13, 0.97, and 0.99 for the current prediction, which is quite close to the experimental results except for Pyr. However, the sensitivity analysis indicated that the mass fraction of particle-phase Pyr ranged from 0.02 to 0.50 when the input parameters varied within their reasonable ranges. The mass fraction of particle-phase Pyr from Zhu et al's experiment still fell in this range.

The PAHs daily concentrations, as shown in Figure 1, varied between 0.245 and 406 ng/m^3. For PAHs with higher volatility, the daily indoor airborne concentrations were within the range from 17.8 to 406 ng/m^3 for Phe and from 7.85 to 137 ng/m^3 for Pyr. Winter corresponded to the highest indoor airborne concentrations and there is no obvious seasonal change among the rest, which was consistent with the varying trend of the atmospheric concentrations. The most abundant existing phase of more volatile PAHs is gas-phase. For Phe, the daily indoor gas-phase concentrations were 4−192 times larger than those of particle-phase. For Pyr, the daily indoor gas-phase concentrations were 1−61 times larger than the corresponding particle-phase concentrations. For less volatile PAHs, the daily indoor airborne concentrations of BaP were between 0.245 and 41.3 ng/m^3 while the daily indoor airborne concentrations of BghiP were between 0.262 and 40.4 ng/m^3. There was a prominent difference of airborne concentrations between different seasons, which was also consistent with the seasonal atmospheric concentrations variation. The highest daily indoor airborne concentrations were in winter and the lowest were in summer. The indoor PAHs concentrations still reflected increases in outdoor concentrations even in seasons when windows would typically be closed. This was mainly because the penetration coefficient of $PM_{2.5}$ was as high as 0.8, under which circumstances the majority of outdoor PAHs, bound to $PM_{2.5}$, penetrate into indoor environments even though windows are closed. The most abundant existing phase of less volatile PAHs is particle-phase. For BaP, the particle-phase concentrations were 1 to 113 times larger than those of gas-phase. For BghiP, the particle-phase concentrations were 9−541 times larger than those of gas-phase.

The predicted and previously measured indoor DEHP concentrations are shown in Table 1. Kanazawa et al.[29] have measured indoor SVOCs concentrations of detached houses in Sapporo, Japan in 2006−2007. According to their results, the indoor DEHP airborne concentrations ranged from 11.8 to 1660 ng/m^3. In addition, Fromme et al.[30] have studied the occurrence of persistent environmental contaminants in the apartments and kindergartens. The mean airborne DEHP concentration for apartments was 191 ng/m^3 while the maximum was 615 ng/m^3. In this study, the estimated average indoor DEHP airborne concentration was 690 ng/m^3. The predicted concentrations were at the same magnitude as the measured ones. Moreover, average indoor DEHP gas-phase concentrations in this study were within the range compiled from the literature by Xu et al.[14]

Regarding to the daily indoor concentrations of DEHP, the airborne concentrations varied from 251 to 1530 ng/m^3. Although it was assumed that there were no outdoor DEHP sources, some seasonal differences in indoor DEHP concentrations were predicted by the model. In the studied scenario, natural ventilation is the only strategy to extract the indoor DEHP. Hence, the longest window-open time in summer, benefiting for exhausting indoor DEHP, led to the lowest indoor DEHP airborne concentrations. Conversely, the shortest window-open time in winter led to the highest. The octanol-air partition coefficients of DEHP in typical indoor temperatures are at the order of 10^{13}−10^{14}, in accord with which DEHP is assumed to be abundant in particle-phase. But the estimated indoor DEHP gas-phase concentrations were comparable with those of particle-phase for the studied scenario, the average of which was 145 ng/m^3 and 545 ng/m^3, respectively (Table 1). The only source of DEHP is the emission from vinyl flooring of the residence in this model. Gas-phase DEHP is generated from the vinyl flooring then sorbs to other indoor substances. Considering the large gas-particle partition coefficient of DEHP, quite a long time is needed for gas- and particle-phase DEHP to reach equilibrium. According to the estimation by Weschler and Nazaroff,[1] the equilibrium time scale of less volatile SVOCs (such as DEHP) to adsorb to airborne particles with the diameter around 1 μm is longer than 1 day. However, the indoor airborne particles are not permanently suspended. The fate of indoor particles is influenced by their built-in aerodynamic features and ventilation rate. The integrated effects determines the diameter dependent residence time of particles in indoor environments, which is much shorter than the equilibrium time scale of DEHP. Under such circumstances, there is not enough time for gas-phase DEHP to react with

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Table 2. Exposure to Airborne SVOCs

compound		exposure(ng/kg/d)				
		inhalation (gas-phase)	inhalation (particle-phase)	dermal (gas-phase)	dermal (particle-phase)	total
Phe	average	16.4	1.30	17.4	0.0705	35.2
	male	16.2	1.31	16.8	0.0680	34.4
	female	16.6	1.31	18.1	0.0735	36.1
Pyr	average	6.03	1.04	13.1	0.0404	20.2
	male	5.96	1.03	12.7	0.0389	19.7
	female	6.11	1.05	13.6	0.0421	20.8
BaP	average	0.0413	1.48	0.587	0.0211	2.13
	male	0.0408	1.47	0.567	0.0204	2.10
	female	0.0419	1.50	0.611	0.0219	2.17
BghiP	average	0.0115	1.53	0.174	0.0233	1.74
	male	0.0114	1.51	0.168	0.0216	1.71
	female	0.0116	1.55	0.180	0.0231	1.76
DEHP	average	25.1	93.9	460	1.63	581
	male	24.6	92.1	442	1.56	560
	female	25.6	95.8	482	1.70	605

suspended particles. Hence, the mass ratio between gas- and particle-phases DEHP in a dynamic environment is much larger than that of DEHP at equilibrium.

Exposure Assessment for SVOCs. With the appropriately estimated indoor SVOCs phase-specific concentrations, exposure to airborne SVOCs of the "reference people" under studied scenarios can be assessed with eqs 8−11. Take indoor inhalation exposure to gas-phase Phe for typical adults as an example. The average indoor daily gas-phase concentration (C_s) of Phe was 81.8 ng/m^3. The long-term inhalation rate (IR) and the body weight (BW) of typical adults were 12.7 m^3/d and 62.29 kg, respectively, while the corresponding indoor and outdoor exposure duration (ED) were 20.35 h/d and 3.65 h/d, respectively. According to eq 8, the indoor inhalation exposure for average adults to gas-phase Phe was 14.1 ng/kg/d while that of outdoor was 2.26 ng/kg/d. Thus, the inhalation exposure to gas-phase Phe was 16.4 ng/kg/d. The results for people of different genders are shown in Table 2. And the results for people of different ages are shown in Figure 2.

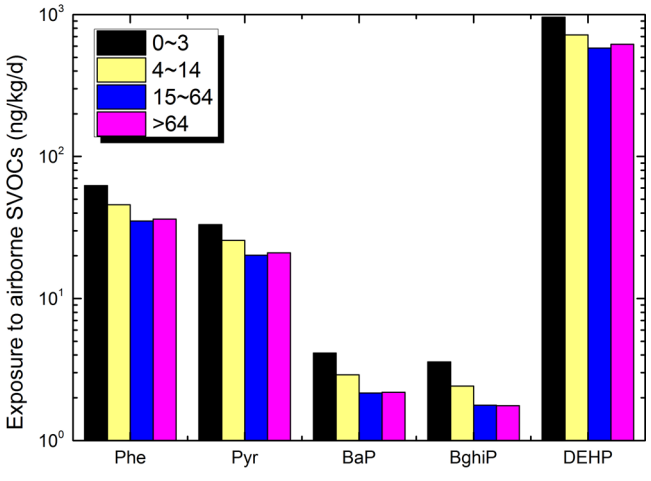

Figure 2. Exposures to airborne SVOCs for people of different ages.

Referring to the results for average adults, the highest daily exposure to airborne SVOCs was 581 ng/kg/d for DEHP and the lowest was 1.74 ng/kg/d for BghiP. The daily exposure of females was a bit higher than the average while the daily exposure of males was a bit lower. The Long-term inhalation rate of males is 17% higher than that of females. The average surface area of males is 12% larger than that of females. Although males are expected to have larger body weight, which may reduce the daily exposure adjusted for body weight. This effect cannot reverse the larger exposure to airborne SVOCs of males due to larger inhalation rates and body surface areas.

For exposure of different ages, the highest daily exposure corresponded to the children before school age, followed by children at school age, elderly people and adults (Figure 2). The daily exposure to airborne BaP and BghiP for children before school age was 2 times as large as that of adults. Children before school age are expected to have smaller inhalation rate, body surface area and body weight. Even though smaller inhalation rate and body surface area would tend to lead to lower exposures, the much smaller body weight compared to adults resulted in higher exposures to airborne SVOCs adjusted for body weight. Considering that children may be more susceptible to effects from air pollutants than healthy adults, pollution control strategies should pay special attention to protection of children.

As for the contribution of each exposure pathway, the results varied from compound to compound. Inhalation exposure to gas-phase along with dermal exposure to gas-phase was the most striking exposure pathways for more volatile PAHs. For Phe, inhalation exposure to gas-phase accounted for 47% of the exposure to airborne phases and dermal exposure via direct gas-to-skin pathway accounted for 50%. As for Pyr, inhalation exposure to gas-phase accounted for 30% of the exposure to airborne phases and dermal exposure via direct gas-to-skin pathway accounted for 65%. Gas-phase exposure is higher than particle-phase exposure because of the high gas-phase concentrations predicted for Phe and Pyr.

For less volatile PAHs, inhalation exposure to particle-phase was the most crucial exposure pathway. The contributions of

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this pathway of the exposure to airborne phases for BaP and BghiP were 70% and 88%, respectively. For BaP, the less important exposure pathways, following inhalation exposure to particle-phase, were dermal exposure via direct gas-to-skin pathway, inhalation exposure to gas-phase and dermal exposure via direct particle deposition. For BghiP, the descending order by the importance of the rest exposure pathways was dermal exposure via direct gas-to-skin pathway, dermal exposure via direct particle deposition and inhalation exposure to gas-phase. These two PAH congeners are abundant in particle-phase in indoor environments. Additionally, the inhalation rate (m^3/h) is much larger than the overall permeability coefficient for both dermal exposure pathways, leading to the important role of inhalation exposure to particle-phase. The sensitivity analysis indicated that gas-particle partition coefficient is the most influencing parameter for exposure to airborne PAHs.

The estimated exposures to studied PAHs were compared with those from previous estimations. Menzie et al.[31] have assessed human exposures to eight carcinogenic PAHs, including BaP and BghiP, from food, water and air. The inhalation exposure to the carcinogenic PAHs ranged from 20−3000 ng/d with median at about 160 ng/d. The sum of inhalation exposure to airborne BaP and BghiP in this study was 190 ng/d. From Zhu et al.'s[16] experiments about indoor PAHs concentrations in China, BaP and BghiP accounted for about 4−24% of the total carcinogenic PAHs mass in residences. Using those ratios, the inhalation exposure to eight carcinogenic PAHs in this study were estimated to be 800−4750 ng/d, which was at the same magnitude as the former estimation.

For DEHP, exposure via direct gas-to-skin pathway contributed 79% of the exposure to airborne phases, followed by inhalation exposure to particle-phase, contributing 16% to the total, inhalation exposure to gas-phase, contributing 4.3% to the total, and dermal exposure via direct particle deposition, contributing 0.28% to the total. Two reasons were assumed to be responsible for the significant status of dermal exposure via direct gas-to-skin pathway: (1) gas-phase concentrations were comparable with particle-phase concentrations for DEHP under dynamic conditions (there was insufficient time to achieve equilibrium partitioning between gas-phase and airborne particles); (2) DEHP has a relatively large overall permeability coefficient through the skin, enhancing the transportation of gas-phase DEHP from environment through the skin. The sensitivity analysis indicated that the DEHP concentration of the vinyl flooring is the most influencing parameter for exposure to airborne DEHP.

The estimation of this distribution was different from that of Little et al.'s estimation,[12] the only DEHP source of which was also emission from vinyl flooring. According to their results, inhalation exposure pathway was the most important. This is mainly because Little et al. calculated DEHP phase-specific concentrations based on the equilibrium partitioning assumption. Hence, in their study, the DEHP particle-phase concentration was about 63 times larger than that of gas-phase. In the present study, the DEHP particle-phase concentration was about 3 times larger than that of gas-phase based on the kinetic partition model. This reduced the importance of inhalation exposure to particle-phase DEHP and enhanced the importance of dermal exposure via the direct gas-to-skin pathway.

It should be noteworthy that particles containing DEHP can also be introduced into indoor environments via several other pathways. For example, atmospheric organic aerosols containing DEHP can enter into indoors through ventilation. The abrasion of PVC materials indoors may also lead to the generation of particle-phase DEHP. However, Wang et al.[32] have measured the outdoor DEHP concentrations of total suspended particles (TSP) in Nanjing, another metropolitan city in China. The airborne particle-bound DEHP concentrations were around 33 ng/m^3, which were much smaller than the estimated indoor DEHP particle-phase concentrations. In addition, the generation of particle-phase DEHP due to abrasion is highly dependent on the material indoors and human activity, which is therefore quite difficult to determine for a typical condition. As a result, these two sources were excluded in this study. Nevertheless, the model will still be applicable with these sources. And the distribution of different exposure pathways may be altered in some way due to the introduction of DEHP particle-phase sources.

In addition to the four studied exposure pathways, human beings can also be exposed to SVOCs via dietary ingestion, incidental ingestion of settled-dust and dermal exposure via contact events. From Menzie et al's estimation,[31] exposure to carcinogenic PAHs by dietary ingestion is the most important exposure pathway, which is 3000 ng/d. And Little et al.[12] also pointed that incidental ingestion is most important for children. Although these pathways are beyond the scope of this study, they still need to be considered in assessments of total exposure to SVOCs.

The Barrier Effect of Clothing. Dermal exposure estimation in this study is based on the assumption that all the skin surface area of the "reference people" was exposed to the air. It is no doubt that the barrier effect of clothing is a substantially important factor influencing dermal exposure. Previous researches have indicated that the adsorption coefficient of phenol were quite similar between naked and clothed subjects, which implied that the barrier effect of clothing of phenol was likely to be neglected.[33,34] Thus, the assumption that clothing left little impact on dermal exposure may be reasonable to some extent. The barrier influence of clothing for particle-phase SVOCs transportation is still unknown. From the results of this study, particle deposition made little contribution to the exposure to the airborne SVOCs for the studied scenario. Thus, the ignorance of the clothing effect may make little difference.

We conducted sensitivity analysis of clothing permeability coefficient of exposure to airborne DEHP in the Supporting Information. The exposure to airborne DEHP declined and the contribution of inhalation exposure to particle-phase increased as the clothing permeability coefficient declined. Contaminants can be transported through clothing via aerosol penetration as well as liquid transportation.[35] These processes are quite dependent on the chemical properties of both SVOCs and clothing, making the permeation coefficient of the clothing a very intricate parameter. Thus, we acknowledge that study of clothing permeability coefficient of SVOCs is still in need of study to fully understand exposure of human beings.

Implications for Further Work. This model accounts for the kinetic considerations to estimate exposures to airborne SVOCs. It may be expected to be applicable to estimate exposure to airborne phases of other SVOCs in different scenarios with the appropriate input parameters. Nevertheless, gas-phase SVOCs can also sorb to settled dust, which can be incidentally ingested by human beings. Xu et al.[14] considered settled dust as a kind of "sorption surface" and estimated the

dust concentrations of DEHP as well as the corresponding ingestion exposure. Guo[11] suggested that settled dust can be seen as a special kind of airborne particles. Both of the models mentioned above did not consider the mass transfer between settled-dust and airborne particles because of deposition and resuspension. A more comprehensive model that considers mass transfer between settled dust and gas- and particle-phase SVOCs, and exposures from resuspended particles, is anticipated to be very complicated and beyond the scope of the present study. Considering the fact that the main focus of this study is exposure to airborne SVOCs, the present model is judged to be reasonably appropriate. Further efforts will be made to develop a more complete model which accounts for sorption to settled particles and subsequent resuspension and exposures.

■ ASSOCIATED CONTENT

Supporting Information

Description of the material. This material is available free of charge via the Internet at http://pubs.acs.org/

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Notes

The authors declare no competing financial interest.

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■ NOMENCLATURE:

A	Inner surface area of the residence (m^2)
A_f	Floor area of the residence (m^2)
A_{pn}	Surface area of individual particle (m^2)
BW	Body weight (kg)
$C_{airborne}$	Sum of gas- and particle- SVOCs concentrations (ng/m^3)
C_p	Indoor airborne particle concentration (ng/m^3)
$C_{p,o}$	Outdoor airborne particle concentration (ng/m^3)
C_s	Indoor SVOCs gas-phase concentration (ng/m^3)
$C_{s,o}$	Outdoor SVOCs gas-phase concentration (ng/m^3)
C_{sp}	Indoor SVOCs particle-phase concentration (ng/m^3)
$C_{sp,o}$	Outdoor SVOCs particle-phase concentration (ng/m^3)
C_{surf}	Indoor SVOCs surface concentration (ng/m^2)
ED_i	Indoor exposure duration (h/d)
ED_o	Outdoor exposure duration (h/d)
$exposure_{D,s}$	Dermal exposure to gas-phase SVOCs (ng/kg/d)
$exposure_{D,sp}$	Dermal exposure to particle-phase SVOCs (ng/kg/d)
$exposure_{I,s}$	Inhalation exposure to gas-phase SVOCs(ng/kg/d)
$exposure_{I,sp}$	Inhalation exposure to particle-phase SVOCs(ng/kg/d)
f_{SA}	Fraction of the skin directly exposed to the air
h_m	Mass transfer coefficient of SVOCs at sorption surface (m/h)
h_{mp}	Mass transfer coefficient of SVOCs at particle surfaces (m/h)
IR	Long-term inhalation rate (m^3/d)
$J_{s,i}$	Indoor mass flux of gas-phase SVOCs from the ambient air to the skin ($ng/m^2/h$)
$J_{s,o}$	Outdoor mass flux of gas-phase SVOCs from the ambient air to the skin ($ng/m^2/h$)
$J_{sp,i}$	Indoor mass flux of particle-phase SVOCs from the ambient air to the skin ($ng/m^2/h$)
$J_{sp,o}$	Outdoor mass flux of particle-phase SVOCs from the ambient air to the skin ($ng/m^2/h$)
K_p	Gas-particle partition coefficient of SVOCs (m^3/ng)
k_{p_cb}	Permeability coefficient from the boundary layer adjacent to the skin across the stratum corneum to the viable epidermis (m/h)
k_{p_eb}	Permeability coefficient across the viable epidermis to the capillary dermis (m/h)
k_s	Permeability coefficient of the gas-phase SVOCs from the ambient air through the skin to the blood (m/h)
K_{surf}	Coefficient that describes SVOC partitioning between surfaces and the gas phase (m)
M	Particle mass loading on surface (ng/m^2)
N_{pn}	Number concentration of airborne particles (m^{-3})
P	Particle penetration coefficient from outdoor air into indoor air
Q_p	Ventilation rate (m^3/h)
R	Particle resuspension rate (h^{-1})
SA	Skin surface area (m^2)
S_p	Indoor particle source strength (ng/h)
S_s	Indoor gas-phase SVOCs source strength (ng/h)
S_{sp}	Indoor particle-phase SVOCs source strength (ng/h)
t	Time (h)
V	Volume of the residence (m^3)
v_d	Particle deposition velocity onto indoor building surfaces (m/h)
$v_{d,h}$	Particle deposition velocity onto human body surfaces (m/h)
v_g	External mass transport rate of gas-phase SVOCs through the boundary layer adjacent to the skin (m/h)

Abbreviations

BaP	Benzo[a]pyrene
BghiP	Benzo[g,h,i]perylene
DEHP	Di 2-ethylhexyl phthalate
PAHs	Polycyclic aromatic hydrocarbons
Phe	Phenanthrene
Pyr	Pyrene
SVOCs	Semivolatile Organic Compounds
TSP	Total suspended particle
USEPA	United States Environmental Protection Agency

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