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Model for Screening-Level Assessment of Near-Field Human Exposure to Neutral Organic Chemicals Released Indoors

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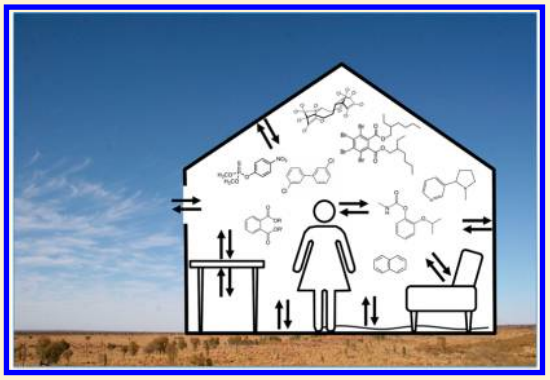
Supporting Information

ABSTRACT: Screening organic chemicals for hazard and risk to human health requires near-field human exposure models that can be readily parametrized with available data. The integration of a model of human exposure, uptake, and bioaccumulation into an indoor mass balance model provides a quantitative framework linking emissions in indoor environments with human intake rates (*iR*s), intake fractions (*iF*s) and steady-state concentrations in humans (*C*) through consideration of dermal permeation, inhalation, and nondietary ingestion exposure pathways. Parameterized based on representative indoor and adult human characteristics, the model is applied here to 40 chemicals of relevance in the context of human exposure assessment. Intake fractions and human concentrations (C_U) calculated with the model based on a unit emission rate to air for these 40 chemicals span 2 and 5 orders of magnitude, respectively. Differences in priority ranking based on either *iF* or C_U can be attributed to the absorption, biotransformation and elimination processes within the human body. The model is further applied to a large data set of hypothetical chemicals representative of many in-use chemicals to show how the dominant exposure pathways, *iF* and C_U change as a function of chemical properties and to illustrate the capacity of the model for high-throughput screening. These simulations provide hypotheses for the combination of chemical properties that may result in high exposure and internal dose. The model is further exploited to highlight the role human contaminant uptake plays in the overall fate of certain chemicals indoors and consequently human exposure.



1. INTRODUCTION

Due to the rapid development of the chemical industry over the past half century, the number of commercially available chemical substances is large and increasing.[1] While chemicals have been promoting economic growth and social development, concerns about their potential negative impact on humans and the environment persist. Chemical management programs therefore seek to assess numerous new and existing chemicals for hazard, exposure and potential risk.[2−4] Whereas biomonitoring (e.g., National Health and Nutrition Examination Survey[5]) can provide valuable information for human exposure assessment, measured data are limited or nonexistent for the vast majority of chemicals,[6,7] necessitating the use of screening-level models for chemical assessment and prioritization.[8,9] Mechanistic mass balance models that combine environmental fate and human food chain bioaccumulation models for simulating far-field human exposure to chemicals in outdoor environmental media (food, water, and air) have been developed[10,11] and applied to thousands of chemicals.[12] Significant human exposure to chemicals also occurs indoors and thus near-field exposure pathways (e.g., inhalation, nondietary ingestion, and dermal permeation) need to be considered when estimating total exposure.[13−17]

Models and metrics to quantify chemical fate[18,19] and human exposure indoors[20,21] have been developed. The chemical intake fraction (*iF*)[22] has been used to evaluate indoor exposure pathways to humans for 15 chemicals for three emission scenarios assuming a pulse release and 20 years of exposure.[20] Intake-to-production ratio, a semiempirical metric linking chemical intake inferred from biomonitoring data to manufactured chemical production volumes, has been proposed to screen for potential human population exposure.[23] Chemical properties and use patterns were found to influence human exposure indoors.[23] Models have been applied to assess exposure to select semivolatile organic chemicals (SVOCs) based on estimates of actual chemical use and release information.[21] Chemicals requiring evaluation display, however, a much greater range of properties than is represented by SVOCs. Models have been used to evaluate the impact of physical removal (e.g., vacuuming, ventilation rates) on chemical residence time[20] in indoor environments and of rates of degradation on *iF*s.[24] The potential for humans to affect the rate of chemical reaction indoors has

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been shown[25] and human intake can comprise a large portion of the quantity of chemical emitted indoors.[21] Thus, indoor fate and human exposure may be affected by the presence of people; however, how the loss of chemical from the indoor environment as a result of human intake and elimination processes may affect near-field exposure has not been examined.

To address the need for mass balance models that link the indoor release of chemicals to human exposure and internal dose, we incorporated a human exposure and uptake model into an indoor chemical fate mass balance model. The new model was applied to two groups of chemicals, namely a set of 40 real indoor contaminants and a large set of hypothetical chemicals comprising the space of neutral organic substances. The model applications illustrate how the relative importance of different exposure pathways depends on chemical properties and how screening results depend on the exposure metric (*iF* vs internal concentration) selected for prioritization. Finally, we also use the model to examine the possibility of human intake and elimination processes influencing chemical fate indoors.

2. METHODS

2.1. Exposure Metrics Calculated by the Model. Models use a variety of metrics to characterize human exposure. Exposure depends on how and how much of a chemical is used, but actual chemical emission rates (E_A, g/d) and use patterns are poorly known for most chemicals requiring assessment. Metrics describing the potential for exposure that are independent of E_A have proven useful for screening and comparing chemicals for exposure hazard. The chemical intake rate (*iR*; g/d) is a product of a human contact rate with the environment (e.g., inhalation of air, m^3/d) and a chemical concentration in that environmental medium (e.g., air concentration, g/m^3) and, depending on the defined contact boundary layer, it may also be a function of the chemical absorption efficiency (AE). The intake rate can be expressed on a body weight basis (i.e., g/kg/d) and all exposure pathways can be summed to estimate aggregate exposure. The intake fraction (*iF*; unitless), which is *iR* normalized to E_A, expresses the incremental intake of a chemical per unit of emission[22] and can be used to examine exposure pathways and exposure potential. Chemical body burden (BB; e.g., ng or mol) and concentration (*C*; e.g., ng/kg or mol/kg) are internal dose metrics of exposure and are a function of *iR* and absorption, distribution, metabolism (biotransformation) and excretion processes. Use of actual emission estimates E_A yields concentrations C_A suitable for exposure and risk assessment, whereas use of a unit emission rate (E_U; g/d) yields unit emission-based human concentration C_U (e.g., ng/kg) suitable for comparing the exposure potential of different chemicals.[12] Although the exposure model introduced here can calculate all of these exposure metrics, the focus of the present study is on the influence of chemical properties on relative exposure potential quantified using *iF* and C_U.

2.2. General Model Description. The Indoor Chemical Exposure Classification/Ranking Model (ICECRM) solves a steady-state, evaluative mass balance to simulate human exposure to organic chemicals in the indoor environment. Details of the model equations and parametrization are presented in the Supporting Information (SI). The model equations, formulated in fugacity notation[26] and programmed in Visual Basic for Application (VBA) in MS Excel 2007, quantify chemical transport, degradation, distribution and exposure processes for up to 10 000 chemicals at a time. The model's indoor environmental fate module and the human exposure module can be run separately or together. The model considers the following exposure pathways to humans from chemicals in the indoor environment: (1) inhalation of gaseous and particulate chemicals in indoor air, (2) nondietary ingestion of dust and through hand-to-mouth contact following indoor surface contact, and (3) dermal permeation.

2.3. Indoor Fate Module. The model describing the fate of chemicals in the indoor environment is based on an existing model[18] which includes five compartments (i.e., air, polyurethane foam, carpet, vinyl floor, organic film on impervious surfaces). Particles are present in all the compartments. A chemical is assumed in equilibrium among different phases of a given compartment (e.g., between gaseous and particulate phases in air). A chemical enters the indoor environment through emission to any of the indoor compartments and is lost from the indoors through degradation in any of these compartments and also through advective loss processes such as particle removal (e.g., cleaning) and exchange of air between the outdoor and indoor environment. This air exchange can also be a source of chemical, if air concentrations are greater outdoors than indoors. In the parametrization used here, the volumes and flow rates are generally representative of a typical contemporary indoor environment (Table S1 in SI). The existing model[18] was modified as follows:

2.3.1. Incorporating Particle Mass Balance. In most previous indoor chemical fate models,[18,19,21,27] mass loadings and size distributions of particles in indoor compartments were model input parameters. Recently, Shin et al.[28] incorporated a particle mass balance in an indoor chemical fate model to estimate rates of particle removal from carpet and vinyl floor based on literature reported particle loadings. Here, we treat particle emission and removal rates as model input parameters and calculate concentrations of different particle size classes. While this approach would allow for the assessment of the effect of variations in particle dynamics on SVOC exposure indoors, this issue is not further addressed here.

2.3.2. Considering the Orientation of Indoor Surfaces. Whereas previous indoor chemical fate models treat all walls and ceilings as one compartment,[18−21,28] rates of particle deposition to surfaces vary with orientation (i.e., vertical or horizontal),[29] potentially resulting in differences in the surface concentrations of both particle bound chemicals and chemicals dissolved in organic films. At the same time, chemicals accumulated on surfaces oriented upward (e.g., desk surface, table tops, and counters), downward (ceiling) and vertically (walls) have different transfer rates to hands due to different contact frequencies (one hardly touches the ceiling or walls while one frequently touches upward oriented surfaces). Thus, the chemical distribution on surfaces influences the potential for human exposure, in particular to SVOCs, for which (i) hand-to-mouth transfer is important and (ii) concentrations on hands are primarily due to hand-surface contact.[30] Therefore, the organic film coated indoor surfaces are treated as three unique compartments (facing upward, downward and vertically). Further information related to the treatment of particles and surface orientation is presented in the SI (Tables S2−S6).

2.3.3. Including Human Feedback on Chemical Mass Balance Indoors. While chemical intake by humans can comprise a large portion of chemical emissions indoors,[21] previous models of indoor fate and human exposure did not consider this potential feedback mechanism. In the modified model, the potential feedback on the indoor concentration by human intake of indoor chemicals is considered by having the

model system comprise the human and the indoor environment. Thus, chemical can also be lost from the system through processes such as biotransformation, renal excretion, fecal egestion, hand washing, and bathing. Chemicals accumulated in the human can re-enter the indoor environment through respiration and dermal permeation.

2.4. Human Exposure Module. A three-compartment human exposure model was developed and incorporated into the indoor model to calculate exposures and internal doses from indoor chemical concentrations and the chemical exposure pathways humans typically experience indoors. Chemicals on hands and skin surfaces contribute to human exposure indoors via hand-to-mouth transfer and dermal permeation, respectively.[16,30] To describe these two processes,[16,30] hand and other skin surfaces are considered two separate compartments; the rest of the body constitutes the third "main" compartment. Considering both the focus on long-term exposure and the limited capacity to parametrize more sophisticated, multicompartment human bioaccumulation models for a large number of chemicals, chemical equilibrium between all human tissues and organs (other than the hands and skin) is assumed to be achieved instantaneously. Fugacity capacities (Z-values) of hands and skin surfaces are quantified assuming that skin lipids have partitioning properties equivalent to those of octanol at the skin surface temperature of 32 °C. Z-value of the human body compartment is quantified as the volume weighted average Z-values of lipid, nonlipid organic matter and water at the body temperature of 37 °C. While the model is parametrized here for a representative human adult (physiological parameters in SI Table S7), it is also capable of handling multiple age classes (i.e., children, toddlers) or individuals with different activity patterns.

Chemical transfer via hand-surface, mouth-surface and mouth-hand contact is described following the approach by Shin et al.[20] The transfer rate of a contact process i (T_i, ng/h) is quantified as $C_i \times SA_i \times EF_i \times TE_i$, where C_i (ng/m^2) is the chemical concentration on hands or indoor surfaces, SA_i (m^2), EF_i (h^{-1}), and TE_i (unitless), are surface areas, frequencies and chemical transfer efficiencies for hand-surface, mouth-surface, or mouth-hand contacts. Default transfer efficiencies are summarized in SI Table S7. T_i needs to be multiplied with an absorption efficiency (AE_i, unitless) to calculate the rate of chemical intake into the body.

Chemical AE through the skin is modeled following Weschler and Nazaroff[15,16] based on the mass transfer coefficient of dermal permeation from skin surface through the stratum corneum and viable epidermis into the papillary dermis, where blood circulation through the body occurs. Because the skin and hands surfaces are treated as separate compartments from the rest of the body, the AE of dermal permeation is accounted for by modeling processes and chemical mass balances at the skin and hands surfaces (see SI eq S14 and related text for details). For chemical entering the body via nondietary ingestion, AE is treated the same as for chemical dietary intake,[31] i.e., $AE = 1/(1.55 \times 10^{-9} K_{OW} + 1.01)$, where K_{OW} is the octanol–water partition coefficient at 20 °C. Chemicals inhaled in the gas phase are absorbed completely into the body, i.e., AE = 100%, whereas of the chemicals inhaled in particulate form, only those associated with particles that are deposited to the respiratory tract are accessible to the human body. The deposited fraction of particles of various sizes is quantified based on empirical and theoretical studies of particle dynamics in the respiratory tract (values in SI Table S7).[32,33] Pathways of chemical elimination from the modeled human include chemical removal from hand and skin surface via washing and bathing, chemical losses from hand skin surface via desquamation (skin peeling and turnover), chemical elimination from the human body via exhalation, biotransformation, fecal egestion, and renal excretion. Further details of the human model are provided in the SI.

The *iF* defined by Bennett et al.[22] is based on the rate at which a contaminant enters a target after crossing a contact boundary. The boundary layer they defined considers "passage through the nose (or mouth for oral breathing), mouth, and skin".[22,34] In this study, the contact boundary is identical to the boundary of the main human body compartment (i.e., excluding hand/skin surface) with the contents of the respiratory and gastrointestinal tracts falling outside of the body compartment. *iF*s for dermal exposure, nondietary ingestion, and inhalation calculated and discussed in this study therefore reflect chemicals that pass through skin, and are absorbed from the gastrointestinal and the respiratory tract into the body, respectively.

2.5. Chemical Input Parameters. Apart from parameters characterizing an indoor environment and a human, the model requires chemical input information. To be useful in the context of high-throughput screening, the model was designed to require only a minimal set of chemical property values that can be readily obtained from publicly available databases and quantitative structure–activity relationships (QSARs) such as those in the US EPA's Estimation Programs Interface (EPI) Suite.[35] The properties required for *iF* calculations are molar mass (MW; g/mol), molar volume (MV; cm^3/mol), octanol–water partition coefficient (K_{OW}; unitless), octanol-air partition coefficient (K_{OA}; unitless), and reaction half-lives for compartments in the indoor environment (HL_{i}; h). With respect to the latter, it may often be appropriate to provide different half-lives for the gas phase chemical in the indoor air compartment, which might react readily with oxidants, and for the other indoor compartments. The model allows the user to include degradation HLs for specific indoor compartments, i (i.e., air, surfaces and polyurethane foam). For calculations of the internal exposure metrics the model also requires the primary whole body biotransformation half-life (HL_B; h). For actual exposure estimates (i.e., *iR* and C_A) the model further requires an actual emission rate estimate (E_A) to the indoor environment and mode-of-entry (or release) information.

2.6. Model Applications. The model, parametrized for a generic indoor environment and a typical adult living in this environment, was used to evaluate and compare exposure and dose metrics as a function of chemical properties in two separate applications.

Forty chemicals (SI Table S8), primarily used or generated indoors and ranging widely in their chemical properties (SI Figure S1), were selected to illustrate how the model can be used to predict indoor exposure potential and to rank chemicals based on either *iF* or C_U. The model can handle chemical emissions to any of the indoor compartments; however, in these simulations chemicals are assumed to only be released to air. The degradation half-lives of these 40 chemicals were estimated from typical OH radical concentrations reported for indoor air,[19] and the reaction rate constants of the gas phase chemicals with OH radicals estimated using AOPWIN.[35] Chemicals can be degraded via other processes (e.g., NO_3, O_3, hydrolysis) and in other indoor compartments but because such information is generally limited we only used OH radical gas phase reactions in air to parametrize the model for degradation HLs in air for these 40 chemicals. For the few ionogenic organic chemicals we ignore the potential for ionization and use only the properties of the neutral species.

For a second set of model simulations aimed at illustrating how chemical properties influence *iF* and C_U, we developed a large database of hypothetical chemicals capturing a range of chemical properties indicative of in-use chemicals (MW = 50 to 950 (step 50), log K_{OW} = 0 to 12 (step 0.5), log K_{OA} = 2 to 14 (step 0.5), and log HL_B = −1 to 5 (Step 2)). In this case, the same chemical degradation half-life (log HL_I = 1 to 4 (step 1)) was assumed to apply to all indoor compartments and MV was estimated based on a regression with MW (SI Table S8). These simulations illustrate how changes in chemical properties (input parameters) change *iF* and C_U calculations (model output).

3. RESULTS AND DISCUSSION

3.1. Ranking 40 Chemicals Based on their Exposure Potential. The intake fractions calculated for the 40 chemicals (SI Figure S2) range over 2 orders of magnitude (0.26%−34%). These *iF*s are notably higher than those calculated for far-field human exposures,[12] highlighting that human exposure potential to chemicals released indoors can be much higher compared to chemicals released outdoors. Of the 40 selected chemicals, propoxur has the highest *iF* followed by nicotine, dibutyl phthalate and 2,4,4'-tribromodiphenyl ether. Nondietary ingestion dominates the intake of dibutyl phthalate and 2,4,4'-tribromodiphenyl ether. The dominant nondietary ingestion exposure pathway of dibutyl phthalate and 2,4,4'-tribromodiphenyl ether predicted by the model is supported by the detection of these compounds in house dust, which is an important vector of exposure.[36,37] The indirect indoor exposure calculations suggest that nicotine and propoxur are mostly taken up by dermal permeation. Nicotine is a weak base with a $pK_a \approx 8$ and can become partially ionized at the skin surface (pH ∼ 6). The applicability domain of the dermal uptake scheme[16] does not include ionic chemicals. Since the dermal permeation efficiency is higher for neutral than for ionized nicotine,[38] the model may overestimate its exposure potential.[39] Nevertheless, high dermal exposure potential for nicotine and propoxur has been confirmed in other exposure studies.[40−43] Future effort on exposure modeling will be directed to develop a scheme that expands the applicability domain to better address ionizable organic chemicals. A key point is that exposure potential can be high through a variety of different exposure pathways and thus there is a need to consider all exposure pathways.

The contribution to total exposure from inhalation, dermal permeation, and nondietary ingestion for the 40 chemicals is displayed using a ternary plot (Figure 1). The range of exposure pathways covered by the 40 chemicals is related to their different properties. Inhalation is important for chemicals (represented by dots close to the left corner of the triangle) that either have high volatility (log K_{OA} < 5, e.g., toluene and formaldehyde) and therefore are present mostly in the gas phase of indoor air, or have low volatility (log K_{OA} > 10, e.g., Bis(2-ethylhexyl) 3,4,5,6-tetrabromophthalate) and therefore partition to the particles suspended in indoor air (in addition to other condensed indoor phases). Inhalation is the dominant exposure pathway for the latter because their relatively high K_{OW} and MW (e.g., Bis(2-ethylhexyl) 3,4,5,6-tetrabromophthalate) lowers dermal permeation (SI eq S14) and/or their low indoor persistence limits accumulation in indoor compartments contributing to nondietary exposure. For more persistent chemicals with similar physical properties nondietary ingestion will become relatively more significant. Ventilation efficiently removes a large fraction of volatile chemicals (low K_{OA}) emitted indoors and only a small fraction of the involatile chemicals (high K_{OA}) is present in the particle phase in air; therefore, *iF*s for chemicals predominantly taken up by inhalation are generally lower than *iF*s for chemicals that are predominantly taken up by other exposure pathways. For chemicals located further from the left corner of the triangle, the contribution of dermal permeation or nondietary ingestion increases and so does their exposure potential (SI Figure S3).

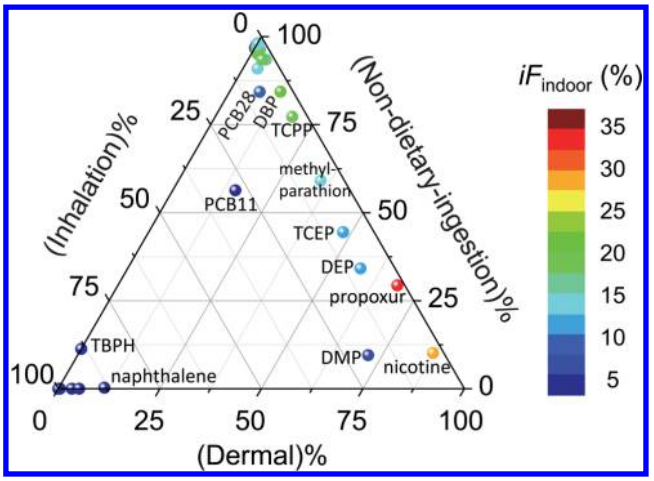

Figure 1. Ternary diagram indicating the contributions of inhalation, dermal permeation, and nondietary ingestion to the total indoor exposure of a human adult to 40 organic chemicals. The color scale represents the total intake fraction (*iF*) from the three indoor exposure pathways. For definitions of chemical acronyms, see Table S8 in the SI.

Human concentrations C_U span 5 orders of magnitude, much wider than the range of *iF*s. While mass concentrations are normally reported from direct measurement, molar concentrations are toxicologically more relevant. Both mass and molar concentrations were calculated (SI Table S9) and result in different chemical rankings. Large differences in rankings based on *iF* or C_U (Figure 2) can be attributed to chemical absorption, biotransformation and excretion processes affecting the C_U

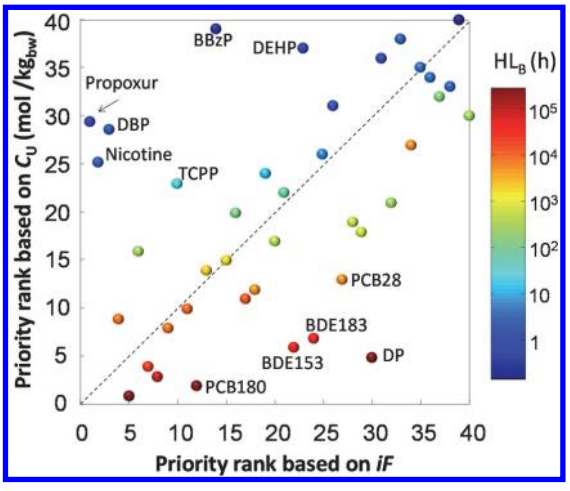

Figure 2. Comparison of the ranking of 40 organic chemicals based either on the indoor intake fraction (*iF*) or on the unit emission based human concentrations (C_U), both calculated by the model for a generic adult living in a typical indoor environment. Large discrepancies in rank are apparent mostly for substances with a short whole body human biotransformation half-life (HL_B, indicated by blue colored dots), whereas ranking of chemicals not subject to fast biotransformation (red dots) correlate highly. For definitions of chemical acronyms see Table S8 in the SI.

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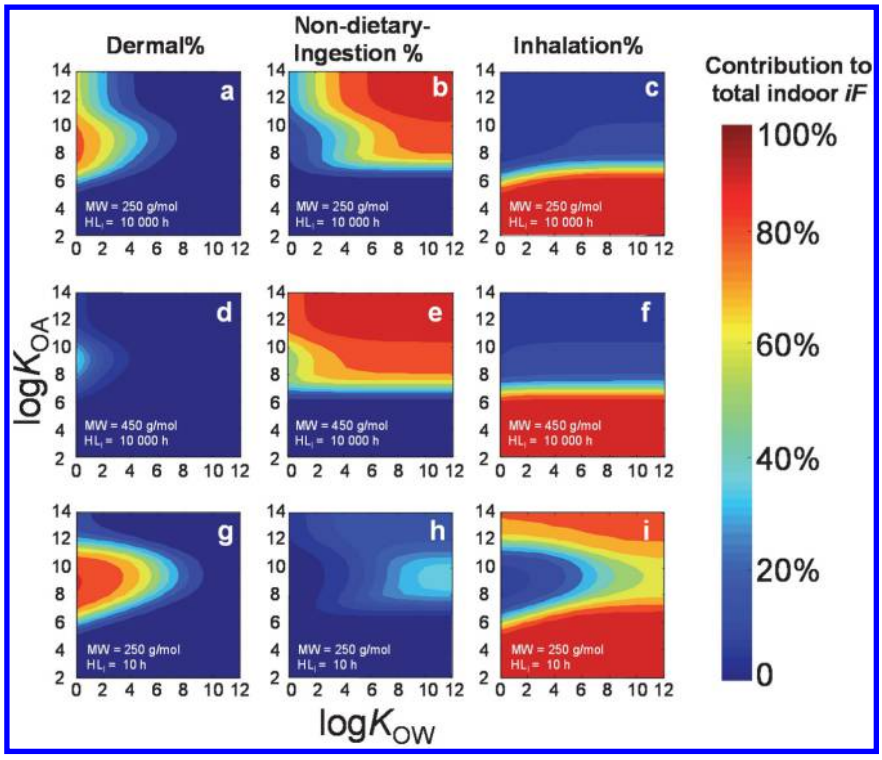

Figure 3. Percent contribution of different exposure pathways (dermal permeation, nondietary ingestion, inhalation) to the total indoor intake fraction (*iF*), calculated by the model as a function of the octanol-air and octanol–water equilibrium partitioning coefficients (K_{OA}, K_{OW}), molecular mass (MW), and the degradation half-life in indoor compartments (HL_I) of hypothetical neutral organic chemicals.

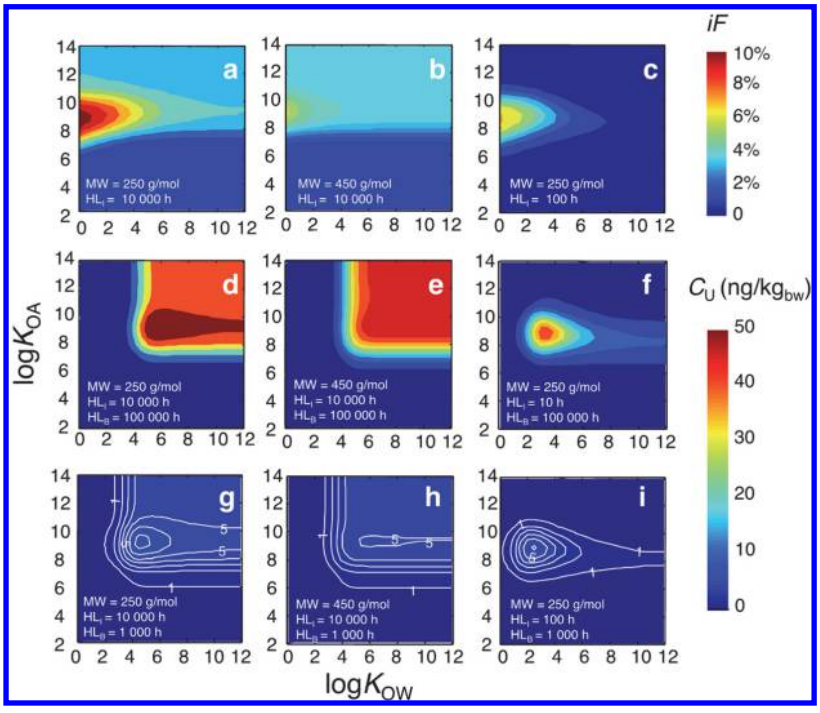

Figure 4. Variations of the total indoor intake fraction (*iF*) and unit emission based human concentrations (C_U) calculated by the model as a function of the octanol-air and octanol–water equilibrium partitioning coefficients (K_{OA}, K_{OW}), molecular mass (MW), the degradation half-life in indoor compartments (HL_I), and the whole body human biotransformation half-life (HL_B) of hypothetical neutral organic chemicals.

metric. For most of the chemicals susceptible to relatively rapid biotransformation (low HL_B, blue colored dots in Figure 2), the rank based on C_U is lower than the one based on *iF*. The chemicals represented by the dots on the upper left of Figure 2 have a high potential to be transferred from indoor sources to humans and thus have high *iF* priority ranks. Their rank based on C_U is lower due to efficient loss from humans via biotransformation (chemicals represented by blue dots) and renal excretion for water-soluble (low K_{OW}) chemicals and respiratory loss for volatile (low K_{OA}) chemicals. For most

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chemicals that are slowly biotransformed (large HL_B, red colored dots in Figure 2), ranks based on *iF* and C_U are highly correlated (Spearman rank correlation coefficient = 0.88).

3.2. Influence of Chemical Properties on Exposure. The relative contributions of inhalation, dermal permeation and nondietary ingestion to the total indoor exposure vary with chemical properties. Inhalation dominates human exposure to volatile chemicals (log K_{OA} < 6), regardless of their other chemical properties (Figure 3c,f,i). Indoor exposure to chemicals with log K_{OA} > 6 occurs primarily through dermal permeation and nondietary ingestion. Dermal permeation dominates exposure to chemicals of low MW, low K_{OW} and intermediate K_{OA} (Figure 3a,g). As K_{OW} and/or MW increases, the overall mass transfer coefficient for skin permeation decreases (SI eq S11) causing the contribution of dermal permeation to the total exposure to decrease. Chemicals with very high K_{OA} (e.g., > 10^{12}) will mostly partition to the condensed indoor phases, such as particles and organic surface films, which results in decreased contribution of dermal permeation caused by the slow permeation through the skin and increased exposure via nondietary ingestion. Indoor exposure of chemicals with high K_{OA} is predominantly the result of nondietary ingestion of dust and surface contact (hand-to-mouth) (Figure 3b,e). In relative terms, for higher K_{OW} and higher K_{OA} chemicals, as HL_I decreases, the nondietary ingestion pathway decreases and the inhalation pathway increases.

The *iF* and C_U calculated for hypothetical chemicals are displayed in contour maps in Figure 4. The two different metrics identify chemicals with different properties as having a high indoor exposure potential (represented by hot colors in Figure 4). Persistent chemicals with low MW (<300), low K_{OW} (<10^4), and intermediate K_{OA} (10^7−10^{11}), i.e., those whose uptake is dominated by the dermal pathway (Figure 3), show a high indoor exposure potential based on *iF* (Figure 4a). In contrast, chemicals with high K_{OW} (>10^5) and K_{OA} (>10^7) are associated with high C_U (Figure 4d,e). The differences can be attributed to chemical absorption, biotransformation and excretion processes within the human body. Chemicals with high *iF* are quite water-soluble (low K_{OW}) and therefore subject to relatively efficient renal excretion. Therefore, some of the persistent chemicals with properties causing elevated *iF* will not be able to maintain a high C_U unless they are poorly eliminated through renal clearance. Chemicals with high C_U are hydrophobic and have quite low volatility and are thus more likely retained in the body unless they are efficiently biotransformed. When chemicals are less persistent indoors (lower HL_I), the area of highest *iF* shifts toward lower K_{OW} (Figure 4a vs 4c). Degradability in the indoor environment prevents high C_U for most chemicals, except for those with an intermediate K_{OW} and K_{OA}, e.g., a log K_{OW} of 2 to 5 and a log K_{OA} around 9, if the *MW* is 250 g/mol (Figure 4f). Examples of chemicals with such partitioning properties are the pesticides atrazine, alachlor, and metolachlor. Chemicals with shorter HL_B have low C_U irrespective of other properties (Figure 4g,h,i).

3.3. Influence of Human Intake on Indoor Chemical Fate and Exposure. Results of an earlier modeling study[21] suggested that the rate of chemical uptake in humans could be significant relative to other chemical transport and transformation processes occurring indoors (see the SI of ref 21 for detail). If true, not accounting for human uptake in the indoor mass balance could overestimate indoor concentrations and thus human exposure. To examine this hypothesis, we used ICECRM to quantify the relative change to steady-state indoor concentrations (thus estimated human uptake) and residence time of the 40 illustrative chemicals that occurs with and without considering human uptake as an indoor chemical loss process (SI Figure S4). Including a human in the indoor mass balance lowers concentrations and residence times by up to 2 orders of magnitude for chemicals with low volatility, if it is assumed that no degradation occurs in the particle phase and in compartments serving as major reservoirs of the chemicals (e.g., PUF, carpet, and vinyl floor). When the chemical-specific air degradation half-lives were assumed applicable to all indoor compartments, the indoor chemical concentrations and residence times for chemicals with high indoor persistence and *iF*s such as hexabromobenzene still decreased by up to 60%. Humans may affect the mass balance for many chemicals in an indoor setting because the capacity of the indoor compartments is much lower than compartments in an environmental fate model. This in turn is a result of the small size and low fugacity capacity of indoor compartments for many substances. We therefore suggest that in some cases human processes need to be incorporated in the indoor chemical mass balance when modeling indoor human exposure. Better quantification of human activity patterns and chemical reaction kinetics in different indoor compartments would help reduce the uncertainties in modeling this process.

3.4. Influence of Indoor Surface Orientations on Modeled Human Exposure. The set of 40 chemicals illustrate how the changes in indoor surface orientation can affect exposure calculations (see SI). For chemicals primarily in the gas phase (log K_{OA} < 10), modeled indoor exposures are insensitive to differences in the orientation of indoor surfaces. However, for some particle-bound chemicals (log K_{OA} > 10 and log K_{OW} < 8), modeled indoor exposure can be underestimated by a factor of about 10 if indoor surface orientation is ignored (assuming all surfaces are only vertically oriented).

3.5. Perspectives on Modeling of Indoor Chemical Exposure for Priority Setting. The different exposure metrics have their merits and limitations for applications in exposure and risk assessment and for comparing, screening and prioritizing chemicals for more comprehensive evaluations. The *iF* helps elucidate key exposure pathways and target indoor sources important for measurement depending on the chemical properties. C_U shows greater discrimination for ranking. The exposure potential metrics *iF* and C_U can be used for exposure-based screening and prioritization in the absence of definitive data for E_A, which are generally lacking for most chemicals. The focus here has been on exposure potential, but the exposure model output can be combined with use and emissions data for exposure estimates and these data compared with effects data for high-throughput screening-level risk estimates. When reliable estimates for E_A, including mode-of-entry, are available, *iR* and C_A can be calculated directly by the model. Alternatively, when E_A and E_U are expressed in the same units and apply to the same mode-of-entry, the model results for a unit emission rate can simply be multiplied by E_A/E_U (e.g., $C_A = C_U \times E_A/E_U$), because the steady-state calculation is linear.

Similar to many other far-field and near-field screening-level models,[20,21,44,45] ICECRM is based on a fixed representative "unit world"[46] environment and assumes equilibrium within each model compartment and steady-state between them. Whether equilibrium and steady-state are reached within an indoor system depends on many factors including chemical properties, characteristics of the indoor environment (e.g., residence time of air and dust), and activity patterns. Studies have shown that SVOCs with high K_{OA} may not reach equilibrium between particles and the surrounding air.[47−49] Assuming equilibrium

could lead to an average of 1 order of magnitude overestimation of the particulate phase concentrations for SVOCs with log K_{OA} >12[47] and thus affect exposure metrics used for chemical screening. The exposure metrics can also be influenced by temporal and spatial variation. For example, more recent OH radical concentrations[50] are 1 order of magnitude higher than the 10^5 molecules/cm^3 reported previously,[51] as used here and elsewhere.[19,20] More data and models are needed to obtain better estimates for degradation rates in various indoor compartments to address uncertainty in indoor human exposure estimates.

Simplifying assumptions are required for screening-level models because of significant data gaps. Oftentimes, model variation and uncertainty may be reduced by more realistic process descriptions; however, improved process resolution inevitably requires additional, and often uncertain, input parameters. Uncertainty introduced by poorly characterized additional parameters could compromise efforts of reducing model uncertainty. Here we have sought a balance between model complexity and key processes governing chemical fate in indoor environments and subsequent exposure and concentrations in humans. Conservative assumptions are also often warranted for screening-level models to minimize false negatives. Here we assumed the efficiency of transfer from indoor environmental media to the hands is the same for all chemicals and that clothing does not constitute a barrier to dermal uptake from the air. The uncertainty inherent in such simplifications is not expected to compromise the model's capacity for screening-level assessment in the absence of measured exposure information; however, ICECRM and other indoor exposure models[20,21] require evaluation. Evaluations with biomonitoring data may be facilitated with ICECRM's unique capacity to quantify internal doses. Because C_A will vary with activity patterns and the characteristics of the indoor environment (e.g., cleaning frequency, ventilation rates), such comparisons should use model parametrizations specific to the individuals and populations sampled in the biomonitoring study instead of the generic values used here. Relevant and reliable E_A estimates are critical for any meaningful comparisons with measured concentrations in indoor environments (air, dust, surfaces) and in human tissues (blood) and fluids (urine). Subsequent model evaluations with appropriate data sets will provide a basis for model refinement.

■ ASSOCIATED CONTENT

Supporting Information

Detailed model descriptions, parameters, and additional results are available free of charge via the Internet at http://pubs.acs.org. ICECRM is available for download at http://www.utsc.utoronto.ca/labs/wania/downloads/.

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Supporting Information for

Model for Screening-Level Assessment of Near-Field Human Exposure to Neutral Organic Chemicals Released Indoors

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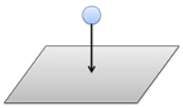
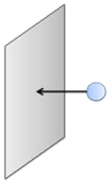
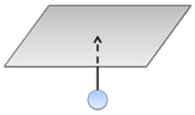
Table S1. Characteristics of the evaluative indoor environment

Parameter	Value	Note/Reference
room area (m^2)	25	default value for evaluation
room height (m)	3	default value for evaluation
PUF area (m^2)	2	default value for evaluation
PUF thickness (m)	0.05	default value for evaluation
vinyl floor area (m^2)	15	default value for evaluation
vinyl floor thickness (m)	5.00E-04	default value for evaluation
carpet area (m^2)	10	default value for evaluation
carpet (fiber only) thickness (m)	0.005	default value for evaluation
area of upward-facing surface (m^2)	60	default value for evaluation
area of vertical surface (m^2)	100	default value for evaluation
area of downward-facing surface (m^2)	40	default value for evaluation
surface-film thickness (m)	1.00E-07	*Ref.*[1]
PUF density (kg/m^3)	22	*Ref.*[2]
vinyl floor density (kg/m^3)	1500	*Ref.*[3]
carpet density (kg/m^3)	65	Derived based on *Ref.*[4]
film density (kg/m^3)	1200	*Ref.*[1]
PUF FOC (fraction of organic carbon, m^3OC/m^3Solid)	0.65	Estimated based on the PU polymer structure of $(C_{17}H_{16}N_2O_4)_n$
vinyl floor FOC	0.37	Estimated based on the vinyl polymer structure of $(C_2H_3Cl)_n$
carpet FOC	0.64	Estimated based on the nylon polymer structure of $(C_6H_{11}NO)_n$
film FOC	0.2	*Ref.*[5]
volume fraction of air in PUF	0.98	*Ref.*[6]
volume fraction of air in carpet	0.8	Estimated by the density of nylon fiber polymer[7] and bulk carpet density
air exchange rate (h^{-1})	0.75	*Ref.*[5]
room temperature (°C)	25	default value for evaluation
thickness of BL (boundary layer) above PUF (m)	0.005	
thickness of BL above floor (m)	0.005	Geometric mean of the 0.01-0.001m
thickness of BL above carpet (m)	0.005	BL thickness above indoor surface
thickness of BL above up facing surf (m)	0.005	suggested in *ref*[8]
thickness of BL above vertical facing surf (m)	0.005	
thickness of BL above down facing surf (m)	0.005	

Particle mass balance modeling in the indoor environment

Particle mass balance in the indoor environment was modeled following the same approach adopted by Shin et al.[9] For the air compartment, the direct emission of particles, resuspension of particles from the other compartments, and the transport of particles from the outdoor environment due to air exchange were considered as the particle input into the air compartment; the transport of particles from indoors to outdoors via air exchange and the deposition of particles from air to the other indoor compartments were considered as particle output from the air. For indoor compartments other than air, particle deposition from the air and transport of particles from outdoor soil to carpet and vinyl floor (soil track-in) are considered as the particle input into the corresponding compartment; particle resuspension and removal due to cleaning from the compartments are considered as the particle output from the corresponding compartment. The default values related to particle deposition, resuspension, emission, transfer from outdoors to indoors, and removal via cleaning are given in Table S2, Table S3, Table S4 and Table S5 respectively. Some of these default values were based on literature data while other data were not available in the literature and were based on our best estimate (professional judgment). In particular, the predicted particle mass concentrations in the indoor compartments are compared with literature reported values (if available) to ensure the predicted values are within reasonable ranges of measured or observed values (Table S6). Note that in reality, many of the assumed default values selected here can vary significantly among different indoor environments and with different resident habits. In this study, the purpose is to evaluate and rank indoor organic chemicals exposure based on their physicochemical properties in a consistent and assumed representative environment. Future model simulations and sensitivity analyses may provide guidance for refining the current assumed values.

Table S2. Default values of particle deposition rates (m h^{-1}) towards different orientations [a]

	Deposition rate(m h^{-1}) towards different orientations		
Particle size fraction (μm)			
			Negligible
0-1	5.18E-02	1.60E-02	(1.00E-10)
1-2.5	1.97E-01	7.87E-03	(1.00E-10)
2.5-10	1.04E+01	3.63E-03	(1.00E-10)
10-65	1.33E+02	1.57E-03	(1.00E-10)
65-150	1.08E+03	9.95E-04	(1.00E-10)
150-2000	1.08E+05	6.42E-05	(1.00E-10)

[a] calculated based on Ref [10]

Table S3. Default values of particle resuspension rates (h^{-1})

Particle size fraction (μm)	Resuspension rate (h^{-1})					
	PUF [a]	Vinyl floor [a]	Carpet [a]	Upward-facing surface [b,c]	Vertical surface [b,c]	Downward- facing surface [b,c]
0-1	2.6E-06	2.6E-06	2.6E-06	2.6E-07	2.6E-07	2.6E-07
1-2.5	1.1E-05	1.1E-05	1.1E-05	1.1E-06	1.1E-06	1.1E-06
2.5-10	1.6E-04	1.6E-04	1.6E-04	1.6E-05	1.6E-05	1.6E-05
10-65	6.9E-04	6.9E-04	6.9E-04	6.9E-05	6.9E-05	6.9E-05
65-150	1.0E-04	1.0E-04	1.0E-04	1.0E-05	1.0E-05	1.0E-05
150-2000	1.0E-04	1.0E-04	1.0E-04	1.0E-05	1.0E-05	1.0E-05

[a] resuspension rates compiled by Bennett and Furtaw.[5]

[b] one order of magnitude lower for particles on organic film coated surface to account for the less influence by resuspension due to human activity induced air flow.

[c] resuspension rates were assumed not changing with the orientations of surface because among the forces that affect particle resuspension, the adhesive force of particles is about orders of magnitude larger than the gravity force. [11]

Table S4. Default values of parameters on particles that enter the indoor environment through indoor emissions and advective transfer from the outdoors

Particle size fraction (μm)	Parameters [a]				
	Mass concentrations of particles in outdoor air (μg/m^3)	Penetration efficiency of outdoor particles to indoors due to air exchange	Rate of particle emission to indoor air (μg/h)	Rate of particle track-in on floor (μg/h)	Rate of particle track-in on carpet (μg/h)
0-1	2	0.85	1000	0	0
1-2.5	5	0.85	1000	50	500
2.5-10	7	0.65	4000	100	1000
10-65	10	0.1	2000	200	2000
65-150	6	0.1	2000	200	2000
150-2000	0	0.1	2000	200	2000

[a] best estimate based on ref.[9]

Table S5. Default values of particle removal rates (h^{-1})

Particle size fraction (um)	Removal rate (h^{-1}) [a]						
	air [b]	PUF	Vinyl floor	Carpet	Upward-facing surface	Vertical surface	Downward-facing surface
0-1	0	7.5E-05	7.5E-03	7.5E-04	7.5E-04	7.5E-05	negligible
1-2.5	0	7.5E-05	7.5E-03	7.5E-04	7.5E-04	7.5E-05	negligible
2.5-10	0	7.5E-05	7.5E-03	7.5E-04	7.5E-04	7.5E-05	negligible
10-65	0	7.5E-05	7.5E-03	7.5E-04	7.5E-04	7.5E-05	negligible
65-150	0	2.0E-05	2.0E-03	2.0E-04	2.0E-04	2.0E-05	negligible
150-2000	0	2.0E-05	2.0E-03	2.0E-04	2.0E-04	2.0E-05	negligible

[a] particle removal via cleaning. Removal rates for vinyl floor and carpet are based on ref [9]. The removal rate for upward-facing surface was assumed the same as carpet. The removal rates for PUF and vertical surface were assumed an order of magnitude lower and that for downward-facing surface was assumed negligible.

[b] particle removal via indoor air purifier (particle filter), not considered here

Table S6. Model predicted particle mass concentrations in different indoor environmental compartments and comparisons with empirical data

	Particle mass concentrations (mg/m^3 for air or mg/m^2 for others)						
	air	PUF	Vinyl floor	Carpet	Upward-facing surface	Vertical surface	Downward-facing surface
This study	0.05 (TSP)	1000	70	500	700	6	negligible
Ref [9]	0.03		58	380			
Ref [12]	0.06						
Ref [13]			$50\text{-}7x10^3$	$300\text{-}10^5$			
Ref [14]			390	6800			

Human Exposure Module of ICECRM

In the fugacity-based modeling approach, uptake and elimination processes in the human exposure model are described with D-values. Derivations and quantitative descriptions of these D-values are provided below (Equations S4 to S16). With these D-values, the mass balance equations (Equation S1 to S3) for the three human compartments can be derived and solved concurrently with the chemical mass balance in the indoor environment. When the steady-state mass balance of chemicals in the modeled indoor environment and human are solved, matrices of exposure potential such as *iF* and C_U can be calculated.

Mass balance of chemicals in the three-compartment human model at steady state

Hands surface: $\sum_X D_{XToHand} f_X = (D_{loss,hand} + D_{HandToMouth} + D_{HandDermalAbsorb}) f_{Hands}$ (Equation S1)

where X represents the indoor compartments, f is the fugacity of the compartment represented by the subscript.

Skin surface: $D_{AirToSkin} f_{Air} = (D_{loss,skin} + D_{SkinToBody}) f_{Skin}$ (Equation S2)

Human body:

$(D_{HandToBody} + D_{HandDermalAbsorb}) f_{Hands} + D_{SkinToBody} f_{Skin} + D_{Inh} f_{Air} = D_{LossBody} f_{Body}$ (Equation S3)

D values for chemical transport processes relevant to human exposure

Inhalation

$D_{\text{Inh}} = IR \times FT_{\text{Indoor}} \times (Z_{\text{Vapor}} + \Sigma Z_{\text{Particle, i}} \times PVFA_{\text{i}} \times PDFR_{\text{i}})$ (Equation S4)

where D_{Inh} (mol Pa^{-1} d^{-1}) is the D-value for inhalation, IR (m^3 d^{-1}) is the inhalation rate, FT_{Indoor} is the fraction of time spent indoors, Z_{Vapor} (mol m^{-3} Pa^{-1}) is the fugacity capacity of the vapor phase, $Z_{\text{Particle, i}}$ is the fugacity capacity of particles, $PVFA_{\text{i}}$ is the particle volume fraction in air, $PDFR_{\text{i}}$ is the particle deposition fraction in the respiratory system, whereby the subscript i refers to different particle size fractions. Calculations of , Z_{Vapor} and $Z_{\text{Particle, i}}$ are demonstrated in Zhang et al. (2009) [2]

Indoor-Human transfer

$D_{\text{XToHand}} = CA_{\text{XToHand}} \times d_{\text{X}} \times CTE_{\text{XToHand}} \times FC_{\text{XToHands}} \times Z_{\text{X}}$ (Equation S5)

where a subscript X represents the indoor environmental media of foam, carpet, vinyl floor, and organic film coated impervious surface, D_{XToHand} (mol Pa^{-1} d^{-1}) is the D-value for chemical transfer from X to hands, CA_{XToHand} (m^2) is the contact area between X and hands, d_{X} (m) is the thickness/depth of the environmental media, CTE_{XToHand} is the chemical transfer efficiency via one-time hand contact, FC_{XToHands}

(d^{-1}) is the frequency of contact, Z_X (mol m^{-3} Pa^{-1})is the fugacity capacity of X. The equations to calculate Z_X are listed in Zhang et al. (2009) [2]

$D_{SurfToMouth} = CA_{SurfToMouth} \times d_{Surf} \times CTE_{SurfToMouth} \times FC_{SurfToMouth} \times Z_{Surf}$ (Equation S6)

where $D_{SurfToMouth}$ (mol Pa^{-1} d^{-1}) is the *D*-value for chemical transfer from indoor surface to mouth, $CA_{SurfToHand}$ (m^2) is the contact area between mouth and surface, which was assumed as 10% of the hands area, d_X (m) is the thickness organic film on indoor surface, $CTE_{SurfToHand}$ is the chemical transfer efficiency from surface to mouth via a one-time contact, $FC_{SurfToMouth}$ (d^{-1}) is the frequency of mouth-surface contact, Z_{Surf} (mol m^{-3} Pa^{-1}) is the fugacity capacity of organic film coated impervious surfaces. Note that $CTE_{SurfToHand}$ may depend on chemical properties and the force of contact, but there is presently no quantitative approach that could be integrated into the model. Thus we adopted the default values used by a previous study [15]

$D_{AirToHand} = B_{Air} \times 24$ (h/d) / $\delta \times SA_{hands} \times Z_{Vapor}$ (Equation S7)

$D_{AirToSkin} = B_{Air} \times 24$ (h/d) / $\delta \times SA_{skin} \times Z_{Vapor}$ (Equation S8)

where $D_{AirToHand}$ and $D_{AirToSkin}$ (mol Pa^{-1} d^{-1}) are the *D*-values for chemical transfer from air to hands surface and skin surface, respectively, B_{Air} (m^2 h^{-1}) is the diffusivity of the chemical molecules in air, δ (m) is the thickness of the boundary layer above the skin and hands surface, and SA_{hands} and SA_{skin} (m^2) is the surface area of the hands and skins, respectively.

Losses from human

$D_{loss,hand} = FST \times V_{lipid,\ hand} \times Z_{SL} + FHW \times EHW \times V_{lipid,\ hand} \times Z_{SL}$ (Equation S9)

$D_{loss,skin} = FST \times V_{lipid,\ skin} \times Z_{SL} + FB \times EB \times V_{lipid,\ skin} \times Z_{SL}$ (Equation S10)

where $D_{loss,hand}$ and $D_{loss,skin}$ (mol Pa^{-1} d^{-1}) are the D-values for chemical losses from hands and skin surface, respectively, *FST* (d^{-1}) is the frequency of skin turnover, $V_{lipid,\ hand}$ and $V_{lipid,\ skin}$ (m^3) are the volumes of lipid on the hands and skin surface, respectively, Z_{SL} (mol m^{-3} Pa^{-1}) is the fugacity capacity of skin lipid, *FHW* and *FB* (d^{-1}) are the frequencies of hand washing and bathing, respectively, and *EHW* and *EB* are the efficiency of removal of the chemicals by hand washing and bathing, respectively.

$D_{loss,body} = IR \times Z_{AirInBody} + GR\ Z_{Body} + UR \times Z_{WaterInBody} + LER \times 0.001$ (kg/g) / $\rho_{BodyLipid} \times Z_{LipidInBody} + BTR \times V_{Body} \times Z_{Body}$ (Equation S11)

where $D_{loss,body}$ (mol Pa^{-1} d^{-1}) is the D-value for chemical losses from the human body, *IR* (m^3 d^{-1}) is the inhalation rate, $Z_{AirInBody}$ (mol m^{-3} Pa^{-1}) is the fugacity capacity of air in the human body, *GR* (m^3 d^{-1}) is the body growth rate, *UR* (m^3 d^{-1}) is the urination rate, Z_{Body} (mol m^{-3} Pa^{-1}) is the fugacity capacity of the human body (method of calculating Z_{Body} is described in ref.[16]), *LER* (g_{lipid}/d) is the lipid excretion rate

from the body, $\rho_{BodyLipid}$ (kg/m^3) is the density of the body lipids, $Z_{LipidInBody}$ (mol m^{-3} Pa^{-1}) is the fugacity capacity of the body lipids, BTR (d^{-1}) is the biotransformation rate of the chemicals in the human body, V_{Body} (m^3) is the volume of the human body.

Skin/hands surface to the internal human body

$$D_{HandToMouth} = CA_{HandToMouth} \times d_{Hand\ lipid} \times CTE_{HandToMouth} \times FC_{HandToMouth} \times Z_{SL} \quad \text{(Equation S12)}$$

where $D_{HandToBody}$ (mol Pa^{-1} d^{-1}) is the D-value for chemical transfer by hand to mouth contact, $CA_{HandToMouth}$ (m^2) is the contact area between mouth and hands, $d_{Hand\ lipid}$ (m) is the thickness of the lipid layer on the hand surface, $CTE_{HandToMouth}$ is the chemical transfer efficiency from hand to mouth via a one-time contact, $FC_{HandToMouth}$ (d^{-1}) is the frequency of mouth-hand contact, Z_{SL} (mol m^{-3} Pa^{-1}) is the fugacity capacity of lipid at hands/skin surface.

$$D_{SkinToBody} = D_{SkinDermalAbsorb} = SA_{skin} \times k_{derm} \times Z_{SL} \quad \text{(Equation S13)}$$

where $D_{SkinToBody}$ (mol Pa^{-1} d^{-1}) is the D-value for chemical transfer from skin surface to the internal human body, which equals $D_{SkinDermalAbsorb}$, the D-value for chemical transfer via skin dermal absorption, SA_{skin} (m^2) is surface are of skin, k_{derm} (m/d) is the mass transfer coefficient for chemical permeation from skin surface to the internal human body, Z_{SL} (mol m^{-3} Pa^{-1}) is the fugacity capacity of lipid at hands/skin surface. k_{derm} can be quantified using the resistors-in-series approach reviewed by Weschler & Nazaroff (2012)[17]

$$k_{derm} = \frac{1}{K_{LW}} \times 0.01\ (m/cm) \times 3600(s/h) \times 24(h/d) \times 2.6 \times 10^{0.7 logKow - 0.0722 MW^{2/3} - 5.252} / (2.6 + 10^{0.7 logKow - 0.0722 MW^{2/3} - 5.252} MW^{0.5}) \quad \text{(Equation S14)}$$

where K_{LW} is the partition coefficient between skin surface lipid and water, which is the octanol-water partition coefficient, MW is the molecular weight of an organic chemical.

$$D_{HandDermalAbsorb} = SA_{Hands} \times k_{derm} \times Z_{SL} \quad \text{(Equation S15)}$$

$$D_{HandToBody} = D_{HandToMouth} \times AE + D_{HandDermalAbsorb} \quad \text{(Equation S16)}$$

where $D_{HandDermalAbsorb}$ (mol Pa^{-1} d^{-1}) is the D value for chemical transfer for chemical transfer via dermal absorption from hands skin, $D_{HandToBody}$ (mol Pa^{-1} d^{-1}) is the D value for chemical transfer from hands to the internal human body, AE is the absorption efficiency/bioaccessibiliity of chemicals in the gastrointestinal tract, SA_{hands} (m^2) is surface area of hands.

Table S7. Default values of the human exposure related parameters

Parameter	**Symbol**	**Unit**	**Value**	**Note**
Skin temperature	T_{skin}	K	305	Based on ref [17]
Body temperature	T_{body}	K	310	
Fraction time spent indoors	FT_{Indoor}		0.9	Canadian average[18]
Frequencies of Contact	$FC_{X\text{-}Y}$			Best default estimate
hands→PUF	$FC_{hands\text{-}PUF}$	event/d	10	
hands→floor	$FC_{hands\text{-}Floor}$	event/d	2	
hands→carpet	$FC_{hands\text{-}Carpet}$	event/d	2	
hands→surface	$FC_{hands\text{-}Surface}$	event/d	100	
mouth→surface	$FC_{mouth\text{-}Surface}$	event/d	0[a]	
mouth→hand	$FC_{Moutn\text{-}PUF}$	event/d	10	
Frequency of hand washing	*FHW*	event/d	6	Best default estimate
Frequency of bathing	*FB*	event/d	1	Best default estimate
Frequency of skin turnover	*FST*	1/d	0.067	self-renewal every 15 days[19]
Body Weight	BW	kg	70	RAIDAR default [16]
Density of human	ρ_{Human}	kg/m^3	1000	RAIDAR default [16]
Lipid mass fraction	Φ_{Lipid}	kg/kg	0.2	RAIDAR default [16]
Density of lipid	ρ_{lipid}	kg/m^3	900	RAIDAR default [16]
Water mass fraction	Φ_{Water}	kg/kg	0.6	RAIDAR default [16]
Density of water	ρ_{water}	kg/m^3	1000	RAIDAR default [16]
Mass fraction of non-lipid organic matter	Φ_{NLOM}	kg/kg	0.2	RAIDAR default [16]
Density of non-lipid organic matter	ρ_{NLOM}	kg/m^3	1000	RAIDAR default [16]
Skin surface area	SA_{Skin}	m^2	2	Based on ref [20]
Hands area	SA_{Hands}	m^2	0.1	5% of skin surface area[21]
Skin lipid volume	$V_{lipid,\ skin}$	m^3	$1.2\cdot10^{-6}$	0.6 μm thick for adults[20]
Hands lipid volume	$V_{lipid,\ hands}$	m^3	$6\cdot10^{-8}$	0.6 μm thick for adults[20]
Inhalation rate	IR	m^3/d	18.1	RAIDAR default [16]
Growth rate	GR	m^3/d	$1.2\cdot10^{-5}$	RAIDAR default [16]
Urination rate	UR	m^3/d	$2.0\cdot10^{-3}$	RAIDAR default [16]
Fecal lipid excretion rate	LER	g_{lipid}/d	0.7	Based on ref.[22]

Table S7 continued. Default values of the human exposure related parameters

Parameter	Symbol	Value	Note
Fraction of inhaled particles depositing in the respiratory system	$PDFR_i$		Based on ref.[23,24]
size fraction <1 μm		0.85	
size fraction 1-2.5 μm		0.65	
size fraction 2.5-10 μm		0.65	
size fraction 10-65 μm		0.85	
size fraction 65-150 μm		0.5	
Chemical transfer efficiencies	*CTE*		
PUF→hands		0.03	assumed the same as carpet→hands
floor→hands		0.03	default based on ref.[15]
carpet→hands		0.03	default based on ref.[15]
surface→hands		0.03	default based on ref.[15]
surface→mouth		0.5	default based on ref.[15]
hands→mouth		0.5	default based on ref.[15]
hand washing		0.8	default based on ref.[25]
bathing		0.8	default based on ref.[25]

[a] the current model application is for an adult, for children, particularly toddlers this value should not be zero.[15]

Table S8. Properties of chemicals selected to illustrate ICECRM applications (MW: molecular mass in g/mol, MV: molar volume in cm^3/mol, HL_I: indoor reaction half-life in hours, HL_B: human metabolic half-life in hours, K_{AW},K_{OW}, K_{OA} are unit-less partition coefficient between air, water and octanol).

Chemical name	Indoor use category	MW	MV[a]	logK_{AW}[b]	logK_{OW}[b]	logK_{OA}[b]	HL_I[c]	HL_B[d]
1,2,5,6,9,10-Hexabromocyclododecane (HBCD)	Flame retardant	642	274	-4.3	5.8	10.5	350	2000
Tris(2-chloroethyl) phosphate (TCEP)	Flame retardant	285	176	-6.2	1.4	7.6	80	6
Tris(chloropropyl) phosphate (TCPP)	Flame retardant	328	218	-5.9	2.6	8.5	39	30
Tris(1,3-dichloro-2-propanyl) phosphate (TDCPP)	Flame retardant	431	255	-7.0	3.7	10.6	97	100
2-Ethylhexyl 2,3,4,5-tetrabromobenzoate (TBB)	Flame retardant	550	276	-3.6	8.8	12.3	160	700
Hexabromobenzene (HBB)	Flame retardant	551	177	-4.3	6.1	10.4	150000	2000
2,2-bis(chloromethyl) trimethylene bis[bis(2-chloroethyl) phosphate] (V6)	Flame retardant	583	355	-12.2	3.3	15.5	23	90
Dechlorane Plus (DP)	Flame retardant	654	348	-3.2	9.0	12.3	85	300000
Bis(2-ethylhexyl) 3,4,5,6-tetrabromophthalate (TBPH)	Flame retardant	706	410	-4.9	12.0	16.9	80	300
BDE-28	Flame retardant	407	191	-3.1	5.9	9.5	1200	9000
BDE-47	Flame retardant	486	208	-3.4	6.3	10.3	1700	10000
BDE-99	Flame retardant	565	226	-3.6	6.7	11.1	3200	10000
BDE-153	Flame retardant	644	243	-3.9	7.1	12.0	7600	40000
BDE-183	Flame retardant	722	261	-4.2	7.5	12.8	5800	40000
BDE-209	Flame retardant	959	313	-5.1	8.7	15.3	52000	600
PCB-11	Building material	223	157	-1.8	5.3	7.1	500	300
PCB-28	Building material	258	169	-1.9	5.9	7.9	1500	6000
PCB-52	Building material	292	181	-2.0	6.3	8.2	2400	6000
PCB-101	Building material	326	194	-2.1	6.8	8.8	5200	9000
PCB-138	Building material	361	206	-2.0	7.7	9.7	11000	90000
PCB-153	Building material	361	206	-2.1	7.3	9.5	11000	300000
PCB-180	Building material	395	218	-2.5	7.7	10.2	17000	300000
Dimethyl phthalate (DMP)	Plastizer	194	143	-5.1	1.6	6.7	3100	1
Diethyl phthalate (DEP)	Plastizer	222	171	-5.0	2.4	7.4	510	2
Dibutyl phthalate (DBP)	Plastizer	278	227	-4.4	4.5	8.9	190	2
Butylbenzyl phthalate (BBzP)	Plastizer	312	246	-5.9	4.7	10.6	160	0.3
Di-2-ethylhexyl phthalate (DEHP)	Plastizer	391	340	-4.1	7.6	11.7	80	0.9
Propoxur	Pesticide	209	165	-7.2	1.5	8.8	55	0.9
Chlorpyrifos	Pesticide	351	215	-4.1	5.0	9.1	19	300
p,p'-DDT	Pesticide	354	222	-3.3	6.4	9.7	510	20000
Methyl parathion	Pesticide	263	172	-5.1	2.9	7.9	30	10

Chemical name	Indoor use category	MW	MV[a]	logK_{AW}[b]	logK_{OW}[b]	logK_{OA}[b]	HL_I[c]	HL_B[d]
Permethrin	Pesticide	391	282	-5.9	6.5	12.4	76	400
Nicotine	Tobacco use	162	137	-6.7	1.2	7.9	19	2
Formaldehyde	Building/ Furniture material	30	27	-2.4	0.4	2.8	220	0.1
Hexanal	Building/ Furniture material	100	97	-2.1	1.8	3.9	61	3
Acrolein	Biocide	56	50	-3.0	0.0	3.0	68	3
Toluene	Paint	92	86	-0.4	2.7	3.2	330	4
Naphthalene (NAP)	Fumigant	128	109	-1.7	3.4	5.2	81	3
Trichloroethene	Vapor intrusion from contaminated ground water or soil	131	71	-0.1	2.4	2.5	2200	200
8:2 Fluorotelomer alcohol (8:2 FTOH)	Leather protectants and stain-resistant	464	188	2.1	5.6	3.5	400	6000

[a] McGowan's characteristic volume predicted by ADME Suite 5.0 (ACD Lab)[26]. MV and MW for the 40 chemicals are correlated according to: MV/(cm^3/mol) = 98.6 ln[MW/(g/mol)] - 358.4, R_{adj}^2 =0.73. This relationship was used to derive MV from MW for the hypothetical chemicals.
[b] Literature reported final adjusted values (shown in bold for HBCD,[27] DP,[28] PBDEs,[29] PCBs,[30] DDT,[31] and NAP[32]) or EPI Suite (v4.1)[33] predicted values at a temperature of 298 K.
[c] based on pseudo-first order reaction rates in cm^3/(molecule·s) predicted by AOPWin (v1.92) in EPI Suite (v4.1)[33] and indoor air concentrations of OH radical of 1.1×10^5 molecule/cm^3 (ref [5])
[d] Human metabolic biotransformation half-life (h) selected estimate based on ref. [34]

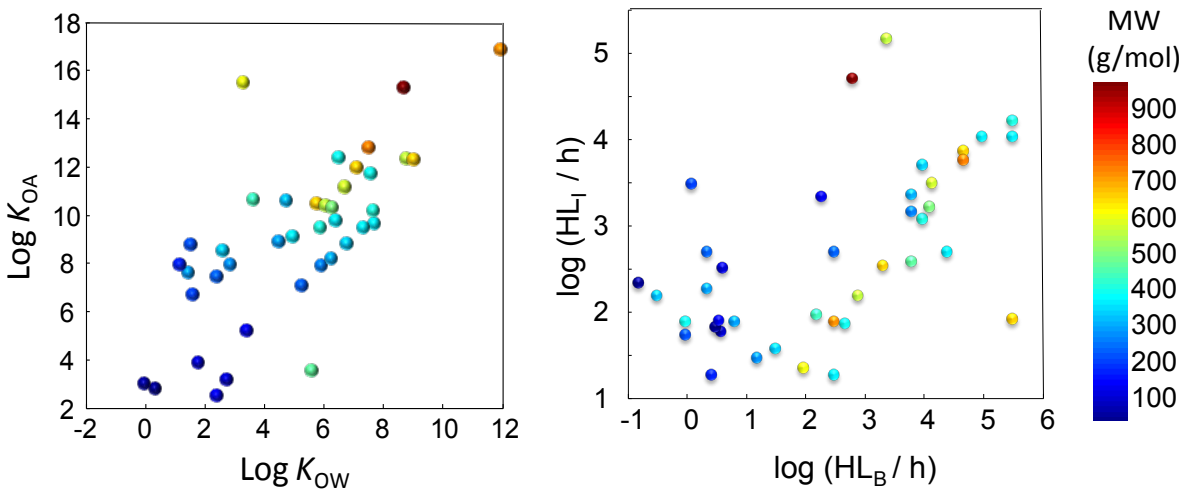


Figure S1. Diverse chemical properties of K_{OW}, K_{OA}, MW, HL_I, and HL_B among the 40 selected chemicals used as an illustration for screening of indoor exposure potential using the Indoor Chemical Exposure Classification/Ranking Model (ICECRM).

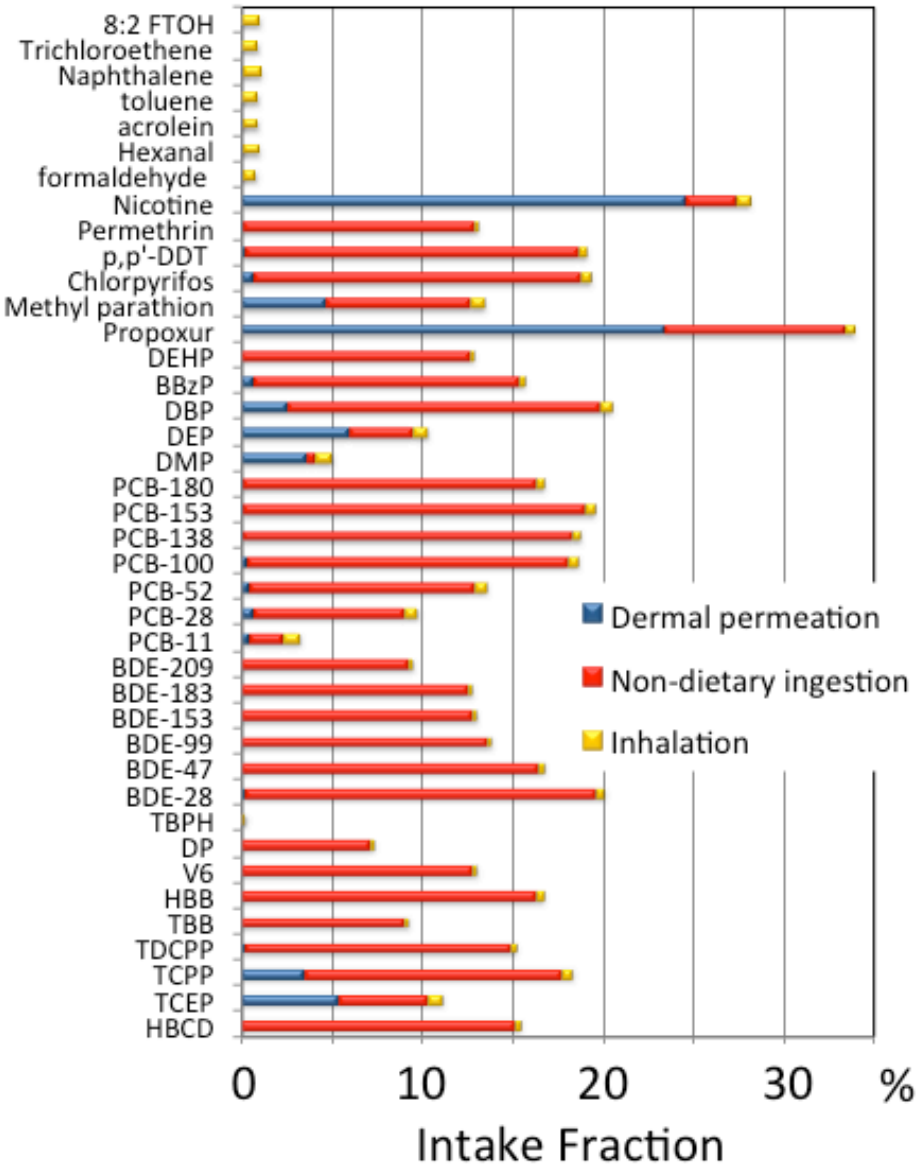


Figure S2. Calculated indoor intake fractions (*iF*) for the 40 illustrative indoor chemicals.

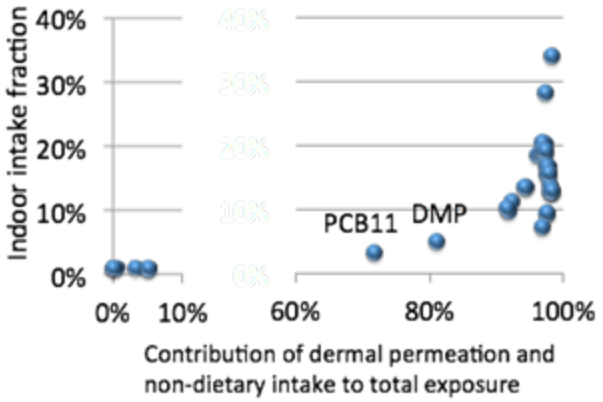


Figure S3. Increasing indoor intake fraction with the contribution of dermal permeation and non-dietary intake to total exposure. Dots with low to high *iF* represent: TBPH, formaldehyde, acrolein, Trichloroethene, toluene, Hexanal, 8:2 FTOH, Naphthalene, PCB-11, DMP, DP, TBB, BDE-209, PCB-28, DEP, TCEP, BDE-183, DEHP, BDE-153, V6, Permethrin, Methyl parathion, PCB-52, BDE-99, TDCPP, HBCD, BBzP, HBB, PCB-180, BDE-47, TCPP, PCB-100, PCB-138, p,p'-DDT, Chlorpyrifos, PCB-153, BDE-28, DBP, Nicotine, Propoxur respectively.

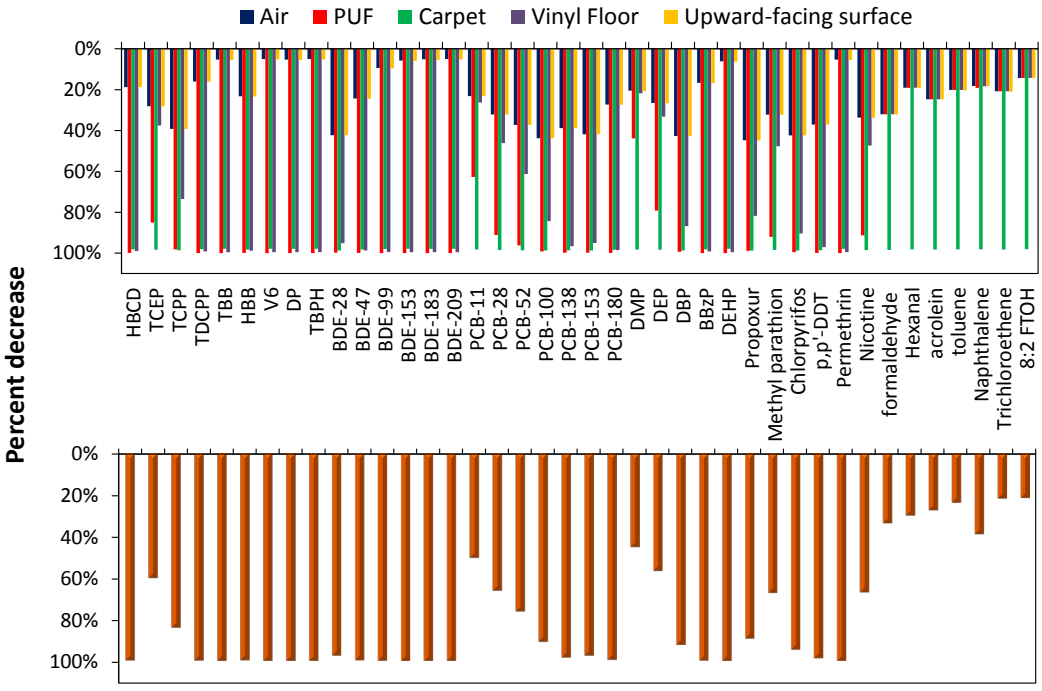


Figure S3. Relative decrease of the chemical concentrations in indoor environmental media (a) and of the overall indoor residence time (b) when chemical intake by human is considered a chemical loss pathway in the indoor environment.

Table S9. Modeled unit emission based concentrations (C_U) in the human body and corresponding priority ranks of the 40 illustrative indoor chemicals. Chemical acronyms are explained in Table S8.

Chemical	C_U (priority rank)			
	mol/m^3	ng/m^3	$ng/g_{body\ weight}$	ng/g_{lipid}
HBCD	9.1E-9 (15)	5821.2 (15)	5.8 (15)	19.4 (15)
TCEP	4.6E-11 (26)	13.3 (28)	0.0 (28)	0.0 (28)
TCPP	3.3E-10 (23)	109.3 (23)	0.1 (23)	0.4 (23)
TDCPP	1.0E-9 (20)	451.7 (20)	0.5 (20)	1.5 (20)
TBB	2.5E-9 (18)	1363.3 (16)	1.4 (16)	4.5 (16)
HBB	1.3E-8 (14)	7244.0 (14)	7.2 (14)	24.1 (14)
V6	4.0E-10 (22)	233.2 (21)	0.2 (21)	0.8 (21)
DP	1.3E-7 (5)	82605.7 (4)	82.6 (4)	275.4 (4)
TBPH	2.2E-11 (30)	15.5 (25)	0.0 (25)	0.1 (25)
BDE-28	7.8E-8 (9)	31860.1 (9)	31.9 (9)	106.2 (9)
BDE-47	7.0E-8 (10)	34137.4 (8)	34.1 (8)	113.8 (8)
BDE-99	5.5E-8 (11)	31102.2 (10)	31.1 (10)	103.7 (10)
BDE-153	1.1E-7 (6)	71060.1 (5)	71.1 (5)	236.9 (5)
BDE-183	9.6E-8 (7)	69506.0 (6)	69.5 (6)	231.7 (6)
BDE-209	1.2E-9 (19)	1123.9 (19)	1.1 (19)	3.7 (19)
PCB-11	8.6E-10 (21)	192.6 (22)	0.2 (22)	0.6 (22)
PCB-28	4.2E-8 (13)	10690.6 (13)	10.7 (13)	35.6 (13)
PCB-52	5.1E-8 (12)	14896.4 (12)	14.9 (12)	49.7 (12)
PCB-100	9.1E-8 (8)	29568.7 (11)	29.6 (11)	98.6 (11)
PCB-138	4.1E-7 (3)	146972.7 (3)	147.0 (3)	489.9 (3)
PCB-153	6.1E-7 (1)	220431.3 (1)	220.4 (1)	734.8 (1)
PCB-180	4.8E-7 (2)	189000.7 (2)	189.0 (2)	630.0 (2)
DMP	6.2E-12 (36)	1.2 (34)	0.0 (34)	0.0 (34)
DEP	1.9E-11 (31)	4.3 (31)	0.0 (31)	0.0 (31)
DBP	3.1E-11 (28)	8.5 (29)	0.0 (29)	0.0 (29)
BBzP	3.0E-12 (39)	0.9 (35)	0.0 (35)	0.0 (35)
DEHP	5.9E-12 (37)	2.3 (32)	0.0 (32)	0.0 (32)
Propoxur	2.9E-11 (29)	6.1 (30)	0.0 (30)	0.0 (30)
Methyl parathion	1.5E-10 (24)	40.1 (24)	0.0 (24)	0.1 (24)
Chlorpyrifos	3.3E-9 (16)	1148.3 (18)	1.1 (18)	3.8 (18)
p,p'-DDT	1.9E-7 (4)	67874.0 (7)	67.9 (7)	226.2 (7)
Permethrin	3.0E-9 (17)	1162.0 (17)	1.2 (17)	3.9 (17)
Nicotine	8.6E-11 (25)	13.9 (27)	0.0 (27)	0.0 (27)
Formaldehyde	8.4E-13 (40)	0.0 (40)	0.0 (40)	0.0 (40)
Hexanal	6.9E-12 (35)	0.7 (37)	0.0 (37)	0.0 (37)
Acrolein	9.5E-12 (33)	0.5 (39)	0.0 (39)	0.0 (39)
Toluene	7.2E-12 (34)	0.7 (38)	0.0 (38)	0.0 (38)
Nap	5.6E-12 (38)	0.7 (36)	0.0 (36)	0.0 (36)
Trichloroethene	9.8E-12 (32)	1.3 (33)	0.0 (33)	0.0 (33)
8:2 FTOH	3.2E-11 (27)	15.0 (26)	0.0 (26)	0.1 (26)

Influence of indoor surface orientations on modeled human exposure - additional discussion and analysis

Bulk concentrations of chemicals on indoor surfaces are relevant to human exposures for certain chemicals.[35] To illustrate how the orientation of impervious surfaces in the indoor environment affects particle mass balance indoors[10] and hence human exposures to chemicals associated with these particles, we compared the results of two model scenarios with the 40 evaluative chemicals used in this study. The first model scenario (i) considered the orientation of impervious surfaces and the second model scenario (ii) did not consider surface orientation. Scenario (ii) assumes all impervious surfaces have the same characteristics as the vertical surface in scenario (i). As shown in Figure S5, the modeled indoor exposure matrices (both *iF* and C_U) are not sensitive to the orientation of surfaces for chemicals with log K_{OA} < 10 because such chemicals are mainly present in the gas phase and their indoor fate is not affected by the change of indoor particle mass balance influenced by indoor surface orientation. The difference in the modeled indoor exposure between the two scenarios increases rapidly with log K_{OA} increasing from 10 to 12. The increasing difference is attributed to the increasing modeled non-dietary exposure of scenario (i) due to increasing chemical transfer rate from surface to hand and from hand to mouth resulting from increased particle (and chemical) deposition to horizontal surfaces that are more frequently contacted by human hands (i.e., table tops, counters, etc). The modeled dermal exposures show little absolute change because dermal exposure is not the dominant exposure pathway for chemicals with log K_{OA} > 10. Modeled indoor exposures through inhalation are lower for particle-bound chemicals in scenario (i) because the horizontal surface of this scenario traps more particles and lowers particle concentrations in air and thus inhalation exposure to chemicals on the air-borne particles. Chemicals with log K_{OA} > 12 are predominantly particle bound and for most of these chemicals the differences between the two scenarios are high but not changing with K_{OA}. For certain chemicals with high K_{OA} and high K_{OW} (e.g. TBPH with log K_{OA} > 12 and log K_{OW} > 8), the estimated indoor exposures are less sensitive to assumptions of indoor surface orientations because these chemicals have reduced bioavailability from the gastrointestinal tract, thus limiting the human intake of such chemicals.

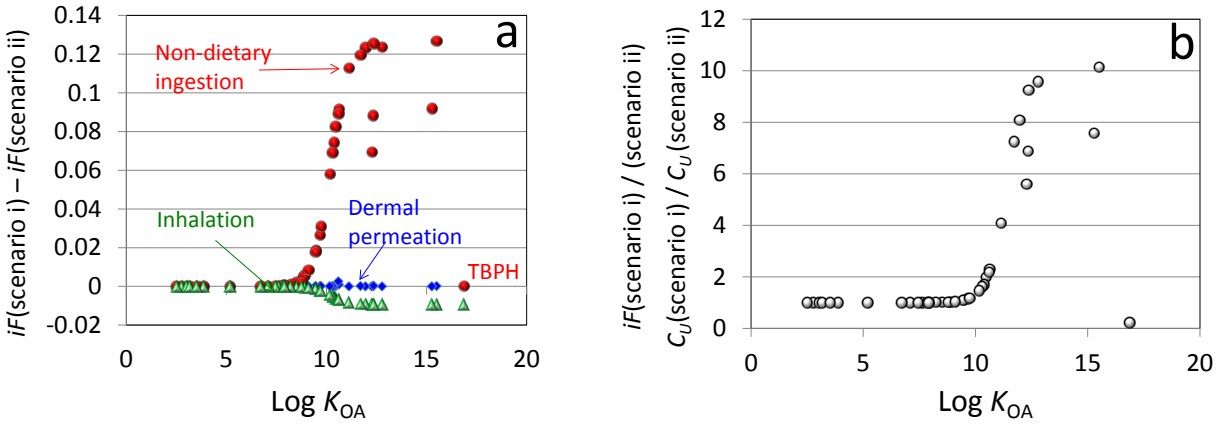


Figure S5. Sensitivity analysis of modeled indoor exposures to different assumptions regarding indoor surface orientation. Scenario i considers the orientation of impervious surfaces and scenario ii does not consider the orientation of impervious surfaces and assumes all impervious surfaces have the same characteristics as the vertical surface in scenario i. (a) Comparison on the changes of intake fraction of non-dietary ingestion, dermal permeation and inhalation between the two scenarios; (b) comparison on the relative differences of total indoor exposure (*iF* of C_U as the exposure matrices) between the two scenarios.

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