Supporting Information for

Conceptual framework to extend life cycle assessment using near-field human exposure modeling and high-throughput tools for chemicals

Susan A. Csiszar[1], David E. Meyer[2], Kathie L. Dionisio[3], Peter Egeghy[3], Kristin K. Isaacs[3], Paul S. Price[3], Kelly A. Scanlon[4], Yu-Mei Tan[3], Kent Thomas[3], Daniel Vallero[3], Jane C. Bare[2]

[1]Oak Ridge Institute for Science and Education (ORISE) Research Participation Program, hosted at U.S. Environmental Protection Agency, Cincinnati, OH 45268, USA

[2]Office of Research and Development, National Risk Management Research Laboratory, US Environmental Protection Agency, Cincinnati 45268, OH, USA

[3]Office of Research and Development, National Exposure Research Laboratory, US Environmental Protection Agency, Research Triangle Park 27711, NC, USA

[4]AAAS Science & Technology Policy Fellow hosted by the U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Radiation and Indoor Air, Washington, DC 20460, USA

Number of pages: 13

Number of figures: 0

Number of tables: 8

Table of Contents

S.1 Human toxicity impact modeling review

In this section details are given on how characterizations factor (CFs) are calculated in the USEtox model.[1-3] USEtox v.20[3] was used for the life cycle impact assessment (LCIA) calculations and to calculate outdoor intake fractions for the aggregate exposure calculations in the case study. In LCIA, the CFs are combined with the life cycle inventory (LCI) to arrive at impact scores (IS).[4]

The CF (disease cases per $kg_{emitted}$) for chemical *x*, emitted to compartment *i* for a given effect, *eff*, is calculated as

$$CF_{x,i,eff} = \sum_{r} FF_{x,i} \times XF_{i}^{r} \times EF_{eff,x}^{r} \quad \text{(S1)}$$

where FF (days), XF ($days^{-1}$), and EF (disease cases per kg_{intake}) are termed the fate factor, exposure factor, and effect factor, respectively. The values of FF and XF can be multiplied together to form the intake fraction, iF which represents the ratio of the mass of chemical taken in by the exposed population per mass of chemical emitted to compartment *i* via a given exposure route, *r* (i.e. inhalation or ingestion).[1] Once emitted to compartment *i*, the fate and transport to exposure media (e.g., air, drinking water, food) are calculated and average human exposure factors are applied to calculate intake of these media via inhalation or ingestion. The effect factor, EF represents the disease cases per kg of chemical that is taken in by one or more individuals via exposure route *r*.[1, 2] The CF units of disease cases per kg of chemical emitted have been termed "comparative toxicity units" (CTU) per $kg_{emitted}^{1}$ as an indication of the comparative nature of LCIA. The human EF is based on an ED_{50} which represents the lifetime dose at which the probability of cancer or non-cancer disease (*eff*) is increased by 50% as

$$EF^{r}_{eff,x} = \frac{0.5}{ED^{r}_{50\ eff,x}} \quad \text{(S2)}$$

The ED_{50} values in Eq. S2 are derived from *in vivo* toxicity data and converted to lifetime effects for an average adult as described by Rosenbaum et al.[1, 2]

The life cycle inventory (LCI) identifies the emission quantities and emission compartments of chemical release for a given functional unit (FU). Multiplying the chemical and compartment specific mass emitted, $m_{x,i}$ with the $CF_{x,i,eff}$ yields the Impact Score ($IS_{x,eff}$) for emission to that compartment, and the total impact score for a given chemical is then summed across all emission compartments as

$$IS_{x,eff} = \sum_{i} CF_{x,i,eff} \times m_{x,i} \quad \text{(S3)}$$

where $IS_{x,eff}$ is expressed in terms of disease cases or CTUs as described above.

S.2 Potential approaches to incorporate risk-based screening into LCIA

There are a number of approaches that could be used to interpret and incorporate the risk-based screening information within the context of LCIA and this section further discusses some of these potential approaches. As discussed in the main text there are essentially two options for calculating the aggregate exposure: (a) using all anticipated sources and (b) using all anticipated sources minus the FU product contribution to exposure.

Risk-based screening approaches are being developed to screen and prioritize chemicals for risk based on aggregate exposures from all anticipated sources.[5-8] This screening approach is in alignment with the screening-level goals of the LCA human toxicity impact category.[9, 10] The additional information from the risk screening could be used to identify chemicals for risk which may have not have high impact scores based on exposure to FU source alone. Calculating both LCA impacts and screening-level risks would result in an identification of chemicals with high impact potentials as well as high potentials for absolute risks. Another method would be to inform comparisons and rankings of individual chemical impact scores. For example, if two chemicals have aggregate exposures below a dose limit, a direct impact score comparison could be valid and follow the traditional ‘less is better’ approach. However, if one chemical is above the limit and another is not, a direct comparison of impact scores would not necessarily be as

valid or insightful. Thus, an aggregate exposure and dose comparison could be used as an additional ranking step to give context to standard impact score calculations. These methods would use the (a) option for aggregate exposure calculations.

Option (b) could be used to determine whether the FU product increases the aggregate exposure above a dose limit; if the aggregate exposure including the FU product is below a dose limit; or if the aggregate exposure is already above the limit without including the product FU. These scenarios could then be classified into different CF or impact score regimes for a given chemical in a FU. This approach to including risk-based information would be consistent with the product-centric nature of LCA as the CF regime would be determined by the product. Approaches would need to be developed to calculate regime-specific CFs; a possible approach could be to scale the CF as suggested by Potting et al.,[11] for example, based on the fraction of the exposed population that reaches a dose above a given reference or based on the ratio of the reference dose to estimated background exposures. A potential drawback of this approach is that, in the near-term, it may be difficult to define meaningful regimes and scaling factors within acceptable levels of uncertainty.

S.3 Case study aggregate exposure calculations

Manufacture: Data from the US EPA's Toxics Release Inventory (TRI)[12] was used to obtain emissions of p-DCB to the outdoor environment, and intake fractions from USEtox v2.0[3] model were used for releases to relevant environmental media. We set the population to one person in USEtox so that intake fractions represent the p-DCB intake of one person (rather than the entire population which is USEtox default), noting that we used a point value for outdoor intake fractions for a default North American person (as parameterized in USEtox v2.0). Intake fractions for emissions to urban air (rather than regional air) were used and p-DCB intake was calculated as the product of the emission rate and intake fraction and normalized to body weight. Values used in the calculations are summarized in Table S1.

Table S1: Summary values and calculations used to estimate exposure to p-DCB due to manufacturing releases in the aggregate exposure calculations.

	Emission [a] (kg/y)	**Inhalation iF[b]**	**Ingestion iF[b]**	**Inhalation intake[c] (mg/kg/d)**	**Ingestion intake (mg/kg/d)**
Urban Air	1.2×10^{4}	1.2×10^{-11}	9.1×10^{-16}	5×10^{-6}	4×10^{-10}
Fresh Water	2.3	7.5×10^{-15}	6.5×10^{-16}	6×10^{-13}	5×10^{-14}
Soil	0			0	0
SUM				5×10^{-6}	4×10^{-10}

Notes: [a] Highest facility emission to air and water from the TRI[12] for the year 2012. The highest air and water emissions were from the same facility. [b] Intake fractions from USEtox v2.0[3] for North America and setting the population to one person. [c] Intakes were calculated using an average adult body weight of 80 kg for the general population.[13]

Use: Exposure in the near-field use-stage was calculated using SHEDS-HT[14] as follows. The SHEDS-HT exposure model was used to generate a set of exposure predictions for p-DCB for a population of 5000 simulated individuals. Exposure was assumed to result from use of three different types of products in the home known to contain p-DCB: closet air fresheners, toilet bowl deodorizers, and moth cakes/crystals. Exposures were estimated for an aggregate case (all three products), a "background" that excluded the product being evaluated here (closet air fresheners), and for the air fresheners only.

Default population and human activity data[14] were used. An air emission scenario was added to SHEDS-HT; this scenario was parameterized using the steady state gas-phase concentration (*y0*) at the interface of the article. Since the products were primarily formulated from solid p-DCB (e.g. 95%), *y0* was estimated from the vapor pressure of the chemical at 25 °C (*y0* = 13 g/m^3). Indoor home air concentrations, *y*, were then calculated from estimated emission areas as in Little et al.[15]:

$$y = \frac{y0\, h\, A}{h\, A + Q} \qquad \text{(S4)}$$

where *y0* is the chemical air concentration in equilibrium with the product surface (g/m^3), *h* is a mass transfer coefficient (m/h), *A* is the area of the emitting source (m^2), and *Q* is the effective ventilation rate for air (m^3/h). The *Q* term includes both the standard air exchange rate plus a particulate transport term. The variable *h* was calculated from the molecular weight of p-DCB

using equations 4-21 and 4-35 of Schwope et al.[16] as 2.55 m/h, and set to a normal distribution with a coefficient of variation (CV) of 1. The *Q* term was calculated as

$$Q = \left(1 + K_p\, TSP\right) AER\ V \quad \text{(S5)}$$

where the K_p (air/particulate partition coefficient, unitless), *AER* (air exchange rate, 1/h) and *V* (house volume, m^3) values are computed in SHEDS-HT as part of the indoor fate and transport module.[14]

Emission areas for all the chemicals were obtained from Guerrero et al.[17] Since the size of the house is a Monte Carlo sampled variable in SHEDS-HT, emission areas were set in the model as a fraction of house floor area. This fractional floor area was determined by making the assumption of the use of 3 closet air fresheners, 3 moth products, and 1 toilet bowl deodorizer per 1000 square feet. This fractional area was assumed to be the mean of a normal distribution with a CV of 50%. A summary of the estimation of emission areas and SHEDS-HT inputs are given in Table S2. Once air concentrations were estimated, partitioning into particulates and to surfaces was estimated as in Isaacs et al.[14] using a two-compartment fugacity-based indoor fate and transport model. Exposures from inhalation, dermal, and non-dietary ingestion routes were then calculated in Isaacs et al.[14] Exposure estimates from SHEDS-HT are summarized in Table S3. We report values for the general population.

Table S2: Summary inputs specific to products associated with p-DCB used in SHEDS-HT.

Product	**Num Products**	**Emission area (ft^2 per product)**	**Emission area (fraction of house with area of 1000 ft^2)**	**Mean**	**CV**
Closet Air Freshener	3	0.025	0.000025	0.000025	0.5
Moth Products	3	0.045	0.000045	0.000045	0.5
Toilet Bowl Deodorizer	1	0.13	0.00013	0.00013	0.5

Out-the-window emissions due to product use were calculated based on the median residential air concentration modeled in SHEDS-HT (Table S4) as

$$E_{air}^{outdoor,use} = C_{air}^{indoor} \times VR \times N_{house} \quad \text{(S6)}$$

where C_{air}^{indoor}, VR, and N_{house} are the residential indoor air concentration (g/m^3), the air ventilation rate (5×10^3 m^3/d), and the number of households in an urban area (4×10^5), respectively. The latter two parameters were based on USEtox v2.0[3] parameterization for North America. The exposure was then calculated by multiplying the emission by the urban air intake fractions for an individual from USEtox v2.0[3] and normalized to body weight,[13] to arrive at an intake in mg/kg/d (Table S4).

Table S3: Summary values from SHEDS-HT for exposure to p-DCB during the use-stage.

Mean (2.5^{th} – 97.5^{th} percentiles)	Inhalation intake (mg/kg/d)	Ingestion intake (mg/kg/d)	Dermal intake (mg/kg/d)	Total intake (mg/kg/d)	Residential air conc (mg/m^3)
Aggregate	2.9×10^{-2} (4.8×10^{-3} – 9.6×10^{-2})	2.7×10^{-4} (1.7×10^{-7} – 2.0×10^{-3})	3.9×10^{-5} (7.7×10^{-7} – 2.0×10^{-4})	2.9×10^{-2} (4.8×10^{-3} – 9.8×10^{-2})	0.22 (0.06 – 0.59)
Background without air fresheners				2.4×10^{-2} (3.1×10^{-3} – 8.8×10^{-2})	
Air fresheners only				5.3×10^{-3} (3.4×10^{-4} – 2.2×10^{-2}	

Table S4: Summary values used to estimate exposure to p-DCB from out-the-window emissions.

	Emission (mg/d)	Inhalation iF[a]	Ingestion iF[a]	Inhalation intake (mg/kg/d)	Ingestion intake (mg/kg/d)	Total intake (mg/kg/d)
Urban Air (out the window)	4×10^{8}	1.2×10^{-11}	9.1×10^{-16}	6×10^{-5}	4×10^{-9}	6×10^{-5}

Notes: [a] Intake fractions calculated using USEtox v2.0[3] for North America and setting the population to one person. [c] Intakes were calculated using an average adult body weight of 80 kg for the general population.[13]

Table S5: Total estimated aggregate exposure (mean, 2.5^{th} – 97.5^{th} percentile, mg/kg/d) to p-DCB from the manufacturing and use stage releases.

	Inhalation (mg/kg/d)	Ingestion (mg/kg/d)	Dermal (mg/kg/d)	Sum across routes (mg/kg/d)	Margin of Exposure (MOE)
Manufacture	5×10^{-6}	4×10^{-10}	--	5×10^{-6}	--
Far-field use	6×10^{-5}	4×10^{-9}	--	6×10^{-5}	--
Near-field use	2.9×10^{-2} (4.8×10^{-3} – 9.6×10^{-2})	2.7×10^{-4} (1.7×10^{-7} – 2.0×10^{-3})	3.9×10^{-5} (7.7×10^{-7} – 2.0×10^{-4})	2.9×10^{-2} (4.8×10^{-3} – 9.8×10^{-2})	--
Sum across stages	2.9×10^{-2} (4.8×10^{-3} – 9.6×10^{-2})	2.7×10^{-4} (1.7×10^{-7} – 2.0×10^{-3})	3.9×10^{-5} (7.7×10^{-7} – 2.0×10^{-4})	2.9×10^{-2} (4.8×10^{-3} – 9.8×10^{-2})	110 (660 - 32)

Notes: Margin of Exposure (MOE) is the ratio of a dose limit to estimated dose. We used the minimum oral equivalency dose (OED) of 3.16 mg/kg/d from Wetmore et al.[7] as the dose limit

S4. Case study LCIA calculations

Life Cycle Inventory

Functional Unit (FU): The FU is defined as 160 g air freshener used in a home for seven weeks.[17, 18] We used a chemical content of 95% from the Consumer Product Chemical Profile database (CPCPdb[19]) and combining the mass and chemical contents gives a chemical mass per FU of 152 g (0.152 kg) (assuming that the reported mass does not include the plastic casing). The Life Cycle Inventory for the FU is summarized in Table S7.

Manufacture: To calculate the outdoor emissions per FU we combined data from the US EPA's Chemical Data Reporting (CDR)[20] database and US EPA's TRI.[12] There was one facility in the CDR with a reported volume used of p-DCB (the latest version of the CDR represents the year 2012). The product category for manufactured product was 'Air Care Product'. The same facility also had reported releases to air, land, water, and facility transfer from the TRI (Table S6). Subtracting the amount released or transferred from the mass used yields the amount of chemical available for incorporation into the consumer product (2.25×10^6). Dividing this mass by the mass of p-DCB per FU (0.152 kg) yields the number of FUs that can be produced from the mass used by the facility. Finally, dividing the mass of chemical release to air by the number of FUs

produced yields an emission to air per FU of 2.3×10^{-5} kg to air/FU. The TRI indicated that there were no releases to land and water for p-DCB.

Use: Several empirical studies exist to estimate the emissions of p-DCB from air fresheners or moth balls.[17, 18, 21] Guerrero & Corsi[17] reported emissions of 0.15 g/h for hanging-type air fresheners, respectively. To be consistent with the risk-based screening exposure estimate, we also estimated the emission rate as used in SHEDS-HT, following the mass balance of Little et al.[15]:

$$E_{air}^{indoor,use} = y0 \times h \times A \quad \text{(S7)}$$

where *y0, h,* and *A* are defined with Equation S4 and parameterized as in the SHEDS-HT calculations. Based on the FU of seven weeks this yields a total gaseous p-DCB release of 74 – 152 g per FU. We note that the mass released to the outdoors from indoors is estimated in USEtox as the removal of chemical from indoor air[22] and is thus not a separate inventory.

Disposal: The amount of chemical left in the product after 7 weeks of use was estimated as the original mass of chemical in the product minus the amount of chemical emitted to air. This yields a p-DCB mass of 0 – 78 g which we assumed was sent to landfill as household waste.[23] We further assumed that 100% of this mass was released to urban air from the landfill.

Table S6: p-DCB mass used and released or transferred from a facility reported in the CDR database.

(kg)	Mass used	Air release	Land release	Water release	Facility transfer
p-DCB	2.25×10^{6}	3.40×10^{2}	0	0	6.58×10^{2}

Table S7: Life cycle inventory for the p-DCB release per functional unit (kg/FU).

kg/FU	Urban air	Indoor air	Water	Notes
Manufacture	2.3×10^{-5}	--	--	
Use	--	0.074 – 0.152	--	
Disposal	0 – 0.078	--	0 – 0.078	For disposal assumed either air or water emission compartments.

Characterization Factors and Impact Scores

We used the default North America parameterization provided in USEtox v2.0 for releases to both outdoor media and indoor air. Impact scores were derived by multiplying the appropriate LCI by the CF (Eq. S3) and aggregating cancer and non-cancer scores. Results are summarized in Table S8.

Table S8: Characterization factors for p-DCB for release to urban and indoor air from USEtox v2.0.

	Indoor air	**Urban air**
iF (kg intake/kg emitted)	3.8×10^{-3}	2.4×10^{-5}
CF (CTU/kg emitted) Cancer + non-cancer	3.7×10^{-5}	2.3×10^{-7}
Impact Score (cases)		
Manufacture	--	5×10^{-12}
Use	$3 – 6 \times 10^{-6}$	--
Disposal (assumed 100% to urban air)	--	$0 – 2 \times 10^{-8}$

Notes: Impact scores were calculated using these CFs and the inventory listed in Table S7. The iFs are the intakes aggregated across the inhalation and ingestion routes. The CFs and impact scores are aggregate cancer and non-cancer effects.

References

(1) Rosenbaum, R.; Bachmann, T.; Huijbregts, M.; Jolliet, O.; Juraske, R.; Koehler, A.; Larsen, H.; MacLeod, M.; Margni, M.; McKone, T.; Payet, J.; Schuhmacher, M.; van de Meent, D.; Hauschild, M., USEtox - The UNEP-SETAC toxicity model: recommended characterisation factors for human toxicity and freshwater ecotoxicity. *Int. J. Life Cycle Assess.* **2008,** *7*, 532-546.

(2) Rosenbaum, R., M. Huijbregts, A. Henderson, M. Margni, T.E. McKone, D. van de Meent, M. Hauschild, S. Shaked, D.S. Li, L.S. Gold, O. Jolliet, USEtox human exposure and toxicity factors for comparative assessment of toxic emissions in life cycle analysis: sensitivity to key chemical properties. *Int. J. Life Cycle Assess.* **2011,** *16*, 710-727.

(3) USEtox *USEtox Model version 2.0: UNEP/SETAC scientific consensus model for characterizing human and ecotoxicological impacts of chemical emissions in life cycle impact assessments*, www.usetox,org, 2015.

(4) Bare, J., TRACI 2.0: the tool for the reduction and assessment of chemical and other environmental impacts 2.0. *Clean Technol. Environ. Policy* **2011,** *13*, (5), 687-696.

(5) Shin, H. M.; Ernstoff, A.; Arnot, J. A.; Wetmore, B. A.; Csiszar, S. A.; Fantke, P.; Zhang, X. M.; McKone, T. E.; Jolliet, O.; Bennett, D. H., Risk-Based High-Throughput Chemical Screening and Prioritization using Exposure Models and in Vitro Bioactivity Assays. *Environ. Sci. Technol.* **2015,** *49*, (11), 6760-6771.

(6) Thomas, R. S.; Philbert, M. A.; Auerbach, S. S.; Wetmore, B. A.; Devito, M. J.; Cote, I.; Rowlands, J. C.; Whelan, M. P.; Hays, S. M.; Andersen, M. E.; Meek, M. E.; Reiter, L. W.; Lambert, J. C.; Clewell, H. J., 3rd; Stephens, M. L.; Zhao, Q. J.; Wesselkamper, S. C.; Flowers, L.; Carney, E. W.; Pastoor, T. P.; Petersen, D. D.; Yauk, C. L.; Nong, A., Incorporating new technologies into toxicity testing and risk assessment: moving from 21st century vision to a data-driven framework. *Toxicol. Sci.* **2013,** *136*, (1), 4-18.

(7) Wetmore, B. A.; Wambaugh, J. F.; Allen, B.; Ferguson, S. S.; Sochaski, M. A.; Setzer, R. W.; Houck, K. A.; Strope, C. L.; Cantwell, K.; Judson, R. S.; LeCluyse, E.; Clewell, H. J.; Thomas, R. S.; Andersen, M. E., Incorporating High-Throughput Exposure Predictions With Dosimetry-Adjusted In Vitro Bioactivity to Inform Chemical Toxicity Testing. *Toxicol. Sci.* **2015,** *148*, (1), 121-36.

(8) Wetmore, B. A.; Wambaugh;, J. F.; Ferguson;, S. S.; Sochaski;, M. A.; Rotroff;, D. M.; Freeman;, K.; H.J. Clewell, I.; Dix;, D. J.; Andersen;, M. E.; Houck;, K. A.; Allen;, B.; Judson;, R. S.; Singh;, R.; Kavlock;, R. J.; Richard;, A. M.; Thomas, R. S., Integration of dosimetry, exposure, and high-throughput screening data in chemical toxicity assessment. *Toxicol. Sci.* **2012,** *125*, (1), 157-174.

(9) Pennington, D.; Crettaz, P.; Tauxe, A.; Rhomberg, L.; Brand, K.; Jolliet, O., Assessing human health response in life cycle assessment using ED10s and DALYs: part 2--Noncancer effects. *Risk Anal.* **2002,** *22*, (5), 947-63.

(10) Hertwich, E. G.; Mateles, S. F.; Pease, W. S.; McKone, T. E., Human toxicity potentials for life-cycle assessment and toxics release inventory risk screening. *Environ. Toxicol. Chem.* **2001,** *20*, (4), 928-39.

(11) Potting, J.; Hauschild, M.; Wenzel, H., "Less is better" and "only above threshold": Two incompatible paradigms for human toxicity in life cycle assessment? *Int. J. Life Cycle Assess.* **1999,** *4*, (1), 16-24.

(12) U.S. Environmental Protection Agency Toxics Release Inventory (TRI) Program https://www.epa.gov/toxics-release-inventory-tri-program, 2016.

(13) U.S. EPA *Exposure Factors Handbook 2011 Edition (Final)*; EPA/600/R-09/052F; National Center for Environmental Assessment, Office of Research and Development, U.S. Environmental Protection Agency: Washington, DC, 2011.

(14) Isaacs, K. K.; Glen, W. G.; Egeghy, P.; Goldsmith, M. R.; Smith, L.; Vallero, D.; Brooks, R.; Grulke, C. M.; Özkaynak, H., SHEDS-HT: An Integrated Probabilistic Exposure Model for Prioritizing Exposures to Chemicals with Near-Field and Dietary Sources. *Environ. Sci. Technol.* **2014,** *48*, (21), 12750-12759.

(15) Little, J. C.; Weschler, C. J.; Nazaroff, W. W.; Liu, Z.; Cohen Hubal, E. A., Rapid methods to estimate potential exposure to semivolatile organic compounds in the indoor environment. *Environ. Sci. Technol.* **2012,** *46*, (20), 11171-8.

(16) Schwope, A.; Goyday, R.; Reid, R. *Methods for Assessing Exposure to Chemical Substances Volume 11. Methodology for Estimating the Migration of Additives and Impurities from Polymeric Materials*; EPA 560/5-85-015; U.S. Environmental Protection Agency: Washington, DC, 1990.

(17) Guerrero, P. A.; Corsi, R. L., Emissions of p-dichlorobenzene and naphthalene from consumer products. *J Air Waste Manag Assoc* **2012,** *62*, (9), 1075-84.

(18) Shinohara, N.; Ono, K.; Gamo, M., P-dichlorobenzene emission rates from moth repellents and leakage rates from cloth storage cases. *Indoor Air* **2008,** *18*, (1), 63-71.

(19) Goldsmith, M. R.; Grulke, C. M.; Brooks, R. D.; Transue, T. R.; Tan, Y. M.; Frame, A.; Egeghy, P. P.; Edwards, R.; Chang, D. T.; Tornero-Velez, R.; Isaacs, K.; Wang, A.; Johnson, J.; Holm, K.; Reich, M.; Mitchell, J.; Vallero, D. A.; Phillips, L.; Phillips, M.; Wambaugh, J. F.; Judson, R. S.; Buckley, T. J.; Dary, C. C., Development of a consumer product ingredient database for chemical exposure screening and prioritization. *Food Chem. Toxicol.* **2014,** *65*, 269-79.

(20) U.S. Environmental Protection Agency 2012 Chemical Data Reporting Results. U.S. EPA, Washington, D.C. https://www.epa.gov/chemical-data-reporting/2012-chemical-data-reporting-results, 2014.

(21) Chin, J. Y.; Godwin, C.; Jia, C.; Robins, T.; Lewis, T.; Parker, E.; Max, P.; Batterman, S., Concentrations and risks of p-dichlorobenzene in indoor and outdoor air. *Indoor Air* **2013,** *23*, (1), 40-9.

(22) Rosenbaum, R. K.; Meijer, A.; Demou, E.; Hellweg, S.; Jolliet, O.; Lam, N. L.; Margni, M.; McKone, T. E., Indoor Air Pollutant Exposure for Life Cycle Assessment: Regional Health Impact Factors for Households. *Environ. Sci. Technol.* **2015,** *49*, (21), 12823-12831.

(23) Slack, R. J.; Gronow, J. R.; Voulvoulis, N., Household hazardous waste in municipal landfills: contaminants in leachate. *Sci. Total Environ.* **2005,** *337*, (1–3), 119-137.