



**HAL**  
open science

## Indoor residential exposure to semivolatile organic compounds in France

Maud Pelletier, Nathalie Bonvallot, Olivier Ramalho, Corinne Mandin, Wenjuan Wei, Gaëlle Raffy, Fabien Mercier, Olivier Blanchard, Barbara Le Bot, Philippe Glorennec

► **To cite this version:**

Maud Pelletier, Nathalie Bonvallot, Olivier Ramalho, Corinne Mandin, Wenjuan Wei, et al.. Indoor residential exposure to semivolatile organic compounds in France. *Environment International*, 2017, 109, pp.81-88. 10.1016/j.envint.2017.08.024 . hal-01635777

**HAL Id: hal-01635777**

**<https://hal.science/hal-01635777v1>**

Submitted on 15 Nov 2017

**HAL** is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

1 **Indoor Residential Exposure to Semivolatile Organic Compounds in France**

2

3 Maud Pelletier<sup>1,2</sup>, Nathalie Bonvallot<sup>1,2</sup>, Olivier Ramalho<sup>3</sup>, Corinne Mandin<sup>3</sup>, Wenjuan Wei<sup>3</sup>,

4 Gaëlle Raffy<sup>1,2,4</sup>, Fabien Mercier<sup>1,2,4</sup>, Olivier Blanchard<sup>1,2</sup>, Barbara Le Bot<sup>1,2,4</sup>, Philippe

5 Glorennec<sup>1,2\*</sup>

6

7 <sup>1</sup>EHESP-School of Public Health, Sorbonne Paris Cité, Rennes, France

8 <sup>2</sup>INSERM-U1085, Irset-Research Institute for Environmental and Occupational Health,

9 Rennes, France

10 <sup>3</sup>University of Paris-Est, Scientific and Technical Center for Building (CSTB), Health and

11 Comfort Department, French Indoor Air Quality Observatory (OQAI), 84 Avenue Jean

12 Jaurès, Champs sur Marne, 77447 Marne la Vallée Cedex 2, France

13 <sup>4</sup>LERES-Environment and Health Research Laboratory (Irset and EHESP Technologic

14 Platform), Rennes, France

15 \*Corresponding author:

16 INSERM-U1085, Irset-Research Institute for Environmental and Occupational Health,

17 Rennes, France. EHESP-School of Public Health, Sorbonne Paris Cité, Rennes, France.

18 Tel.: +33-2 99 02 26 80.

19 E-mail address: philippe.glorennec@ehesp.fr

20 **KEY WORDS**

21 Aggregate exposure; cumulative exposure; chemicals; Monte Carlo simulation; indoor air  
22 quality; dermal exposure.

23 **ABSTRACT**

24 Multiple chemicals are emitted in residential accommodation. Aggregate Daily Doses (ADD)  
25 (ng/kg-bw/d) were estimated for 32 semivolatile organic compounds (SVOCs) of different  
26 chemical families that are frequently detected in French dwellings in both air and settled dust.  
27 Daily doses were determined using steady-state models for the population, categorized into 11  
28 age groups covering birth to age 30. Three routes of exposure were taken into account: dust  
29 ingestion, inhalation (gaseous and particulate phases) and dermal contact with the gaseous  
30 phase of air. Contamination levels were preferentially retrieved from large, nationwide  
31 representative datasets. A two-dimensional probabilistic approach was used to assess  
32 parametric uncertainty and identify the most influential factors. For children aged 2 to 3 years,  
33 ADD estimates spanned orders of magnitude, with median values ranging from 8.7 pg/kg-  
34 bw/d for 2,2',3,4,4'-pentabromodiphenylether (BDE 85) to 1.3 µg/kg-bw/d for di-isobutyl  
35 phthalate (DiBP). Inhalation, ingestion and dermal pathway contributed at varying levels, and  
36 depending on compound, air was the dominant medium for 28 of the 32 compounds (either by  
37 inhalation or dermal contact). Indoor exposure estimate variance was mainly driven by indoor  
38 contamination variability, and secondarily by uncertainty in physical and chemical  
39 parameters. These findings lend support to the call for cumulative risk assessment of indoor  
40 SVOCs.

## 41 INTRODUCTION

42 Both consumer product use and the production of chemicals have been rising constantly since  
43 the mid-20<sup>th</sup> century, and many of these chemicals are semivolatile organic compounds  
44 (SVOCs). SVOCs include compounds from various chemical families: phthalates, bisphenols,  
45 polycyclic aromatic hydrocarbons (PAHs), organophosphorus (OPs), organochlorines (OCs),  
46 synthetic musks, polychlorinated biphenyls (PCBs) and polybromodiphenylethers (PBDEs).

47 The health effects of SVOCs have been assessed by numerous studies on both humans and  
48 animals; some are suspected of having reprotoxic (Casas et al. 2013; Rubin 2011), neurotoxic  
49 (Baldi et al. 2001; Blanc-Lapierre et al. 2012; Elbaz et al. 2009; Zaganas et al. 2013) or  
50 carcinogenic effects (Armstrong et al. 2004; IARC 2010a, 2010b).

51 SVOCs are emitted by volatilization from their source materials and contaminate other  
52 compartments; in some cases, they can also migrate directly from source (Sukiene et al.  
53 2017). In the indoor environment, they are found in the gas phase, airborne particles, settled  
54 dust (Weschler and Nazaroff 2008) and on any other available surfaces such as walls, ceiling  
55 and flooring – as well as on human skin and clothing. In addition to dietary exposure and  
56 dermal contact with consumer products, humans are continuously exposed to these chemicals  
57 through various pathways, including inhalation of indoor air (gaseous and particulate phases),  
58 ingestion of settled dust and dermal contact with indoor air and settled dust (on floor and  
59 other surfaces). Many authors have assessed indoor exposure to certain families of SVOCs  
60 (phthalates, PBDEs, etc.), taking into account one or more exposure media via oral,  
61 respiratory (and sometimes dermal) pathways (Bekö et al. 2013; Gaspar et al. 2014; Linares et  
62 al. 2010; Mitro et al. 2016; Roosens et al. 2010; Trudel et al. 2011; Wilson et al. 2003). Mitro  
63 et al. (2016) recently estimated indoor exposures based on US dust surveys and an air

64 contamination model. Here, we seek to use measurement data to assess the exposure of a  
65 large population and estimate the associated uncertainty.

66 The objective of this study was to estimate the indoor exposure of people of various age  
67 groups, to 32 SVOCs from different chemical families frequently detected in French  
68 dwellings (Blanchard et al. 2014; Mandin et al. 2014, 2016). Three routes of exposure were  
69 taken into account: dust ingestion, inhalation of air (gaseous and particulate phases) and  
70 dermal contact with the gaseous phase of air. Contamination levels were preferentially  
71 retrieved from large, representative datasets. A two-dimensional probabilistic approach was  
72 used to assess the uncertainty associated with the different parameters, and identify the most  
73 impacting factors.

## 74 **METHODS**

75 **Target Population:** To address exposure across a broad section of the population, we  
76 estimated exposures for 11 age groups from birth to age 30 (as an example of an adult),  
77 following the U.S. Environmental Protection Agency (U.S. EPA 2005) recommendations as  
78 to which age groups should be considered within a health risk assessment.

79 **Compounds selection:** 32 SVOCs were selected on the basis of their health interest  
80 (Bonvallot et al. 2010), and because they were detected in both the air and the settled dust of  
81 French dwellings (Blanchard et al. 2014; Mandin et al. 2014, 2016).

82 **Exposure model:** The Aggregate Daily Doses (ADD) (ng/kg-bw/d) were assessed by  
83 summing internal (uptake) daily doses from dust ingestion ( $DD_{\text{ing-dust}}$ ), inhalation of air (both  
84 gaseous and particulate phases) ( $DD_{\text{inh-air}}$ ) and dermal contact with gas phase ( $DD_{\text{derm-gas}}$ ).

85 Very few studies included the dermal exposure to dust pathway when assessing aggregate  
86 exposures to SVOCs. Trudel et al. (2011) studied dermal exposure to 8 PBDEs in dust. Even

87 though they overestimated this pathway using *in vitro* experimental data with acetone as  
88 carrier vehicle (Roper et al. 2006), they found the contribution of dermal exposure to dust to  
89 be consistently < 20%, even for the most contaminated region, and for every age group  
90 (below 1 year to 65 years of age). Bekö et al. (2013) estimated indoor exposure to five  
91 phthalates and found a very low (<1%) contribution of dermal exposure to dust, in  
92 comparison to other pathways. Since this pathway is typically found to be minor and required  
93 uptake parameters are ill-suited to dust exposure even when available, dermal exposure to  
94 dust was not addressed in this work.

95 **Equations for exposure dose estimation:** Daily Doses (DD) can be estimated in steady-state  
96 conditions using the following equations. These were adapted from relationships developed  
97 by Bekö et al. (2013) and Weschler and Nazaroff (2012, 2014).

98 Ingestion of settled dust

$$DD_{ing-dust} = \frac{C_{dust} \times DI \times f_{oral} \times f_{dust} \times t}{BW} \quad (1)$$

99 Where  $C_{dust}$  is the SVOC concentration in settled dust (ng/g), DI is the amount of dust  
100 ingested by an individual per day (g/d),  $f_{oral}$  is the oral bioavailability of the SVOC (-),  $f_{dust}$  is  
101 the bioaccessibility of the SVOC from the dust (-), t is the fraction of time spent in dwellings  
102 (-), BW is the body weight (kg), and  $DD_{ing-dust}$  is expressed in ng/kg-bw/d.

103 Inhalation of indoor air

$$DD_{inh-air} = \frac{(C_{part} + C_{gas}) \times IR \times f_{pulm} \times t}{BW} \quad (2)$$

104  
105 Where  $C_{part}$  is the SVOC particulate phase concentration (ng/ m<sup>3</sup>),  $C_{gas}$  is the SVOC gas phase  
106 concentration (ng/m<sup>3</sup>), IR is the inhalation rate for an individual per day (m<sup>3</sup>/d),  $f_{pulm}$  is the

107 pulmonary bioavailability of the SVOC (-),  $t$  is the fraction of time spent in dwellings (-),  $BW$   
108 is the body weight (kg), and  $DD_{inh-air}$  is expressed in ng/kg-bw/d.

109 Dermal contact with the gas phase

$$DD_{derm-gas} = \frac{C_{gas} \times k_{p-g} \times BSA \times t}{BW} \quad (3)$$

110  
111 Where  $C_{gas}$  is the SVOC gas phase concentration (ng/m<sup>3</sup>),  $k_{p-g}$  is the SVOC transdermal  
112 permeability coefficient (m/h),  $BSA$  is the body surface area (m<sup>2</sup>),  $t$  is the daily duration  
113 exposure (h/d),  $BW$  is the body weight (kg), and  $DD_{derm-gas}$  is expressed in ng/kg-bw/d. The  
114 steady-state model adapted by Weschler and Nazaroff (2012, 2014) used to estimate  $k_{p-g}$  is  
115 described in more detail in Supplemental Material (see S1). This requires use of the SVOC  
116 octanol/water partition coefficients ( $\log(K_{ow})$ ), Henry's law constants ( $H$ ) and coefficients  
117 describing the external transport of a gaseous SVOC from the bulk indoor air to the boundary  
118 layer adjacent to the skin ( $\gamma_d$ ).

119 The ADD for a single SVOC for an individual was then calculated by summing the previous  
120 doses according to the following equation, and expressed in ng/kg-bw/d:

$$ADD = DD_{ing-dust} + DD_{inh-air} + DD_{derm-gas} \quad (4)$$

121  
122 **Parameter estimation for exposure model:** Parameter distributions were constructed or  
123 retrieved from the literature as detailed below. Some of these parameters will be the same for  
124 all SVOCs ( $\gamma_d$ ,  $BW$ ,  $BSA$ ,  $IR$ ,  $DI$  and  $t$ ) while others will vary from one compound to another  
125 ( $f_{oral}$ ,  $f_{dust}$ ,  $f_{pulm}$ ,  $\log(K_{ow})$ ,  $H$ ,  $C_{dust}$ ,  $C_{part}$  and  $C_{gas}$ ).

126 Physical and chemical parameters

127 For each SVOC, measured or estimated values of  $\log(K_{ow})$  and H at 25°C were retrieved  
128 from: online databases - Hazardous Substances Data Bank (HSBD) and ChemIDplus  
129 (<http://toxnet.nlm.nih.gov/>), Chemspider (<http://www.chemspider.com/>), and Chemicalize  
130 (<http://www.chemicalize.org/>); toxicological and environmental data sheets from the French  
131 National Competence Center for Industrial Safety and Environmental Protection (INERIS)  
132 (<http://www.ineris.fr/substances/fr/page/21>); online calculators - Chemexper  
133 (<https://www.chemexper.com/>) and ACD/Labs (<http://www.acdlabs.com/>); EPI Suite software  
134 (U.S. EPA, v4.1) and the Handbook of Physical-Chemical Properties and Environment Fate  
135 for Organic Chemicals (Mackay et al. 2010a, 2010b, 2010c, 2010d). Particular attention was  
136 paid to avoiding duplicates (EPI Suite software, HSBD and ChemIDplus often used the same  
137 sources for these parameters). Furthermore, only values at the reference temperature of 25°C  
138 were selected, in order to obtain comparable data between compounds and estimate DD at a  
139 constant temperature. At least two values were available for each SVOC. Where at least 15  
140 values for  $\log(K_{ow})$  and H were available, distributions were fitted. Otherwise, we used either  
141 triangular distributions having at least three values (minimum, average and maximum), or  
142 uniform distributions for two retrieved values. See Table S1 for corresponding distributions  
143 and input parameters for each SVOC.

#### 144 Contamination data

145 Contamination data were provided from measurements taken in recent French housing  
146 surveys. Concentration levels in settled dust collected from vacuum cleaner bags were  
147 retrieved from a national survey covering the 3.6 million French dwellings that were home to  
148 at least one child aged 6 months to 6 years in 2008-2009, using 145 samples (Mandin et al.  
149 2014). Concentration levels in airborne Particulate Matter (PM) of 10  $\mu\text{m}$  in diameter were  
150 retrieved from a national survey covering the 24.7 million French main residences, using 285  
151 samples (Mandin et al. 2016). PM samples were collected from each living room over a



152 period of one week, and assumed to be representative of each dwelling as a whole. SVOC gas  
153 phase concentrations were not available at national scale; data from 30 French dwellings  
154 (Blanchard et al. 2014) were used as a surrogate.

155 Modeling was established regarding the percentage of samples above the limit of detection  
156 (LOD) or the limit of quantification (LOQ) for the retrieved studies. For settled dust and PM  
157 measurements, lognormal distributions were fitted using summary statistics when > 75% of  
158 data were above LOD (Burmester 1997). This concerns 20 and 14 compounds, for settled dust  
159 and PM measurements respectively. Where detection frequency ranged from 20 to 75%, the  
160 maximum-likelihood estimation (MLE) was used to estimate lognormal distributions (Helsel  
161 2011; Helsel and Hirsch 1992). This statistical method could be used to estimate the  
162 undetected data in the studies of Mandin et al. (2014, 2016), because sample sizes were large  
163 enough - with 145 and 285 samples for dust and PM respectively. The number of compounds  
164 concerned was 7 for settled dust and 11 for PM measurements. Where detection frequency  
165 ranged from 1 to 20%, custom distribution was modeled with discrete probability for the  
166 quantified values, and continuous uniform probability from 0 to LOD and from LOD to LOQ.  
167 The number of compounds concerned was 5 for settled dust and 7 for PM measurements. For  
168 the gas phase measurements, the Blanchard et al. (2014) sample size (n=30) was too small to  
169 use MLE. So, where detection frequency ranged from 1 to 99%, custom distribution was  
170 modeled using continuous uniform probability from 0 to LOQ and discrete probability for the  
171 quantified values. For all three media, a literature survey was conducted to retrieve specific  
172 data when detection frequency was < 1%. Publications were selected in the following order of  
173 preference: conducted in Europe, post-2000 and providing sample size, measurement method,  
174 LOD and/or LOQ values and percentage of samples above the LOD and/or LOQ. Where a  
175 lower LOD or LOQ was used, other contamination information was used instead of the

176 initially-selected data (Blanchard et al. 2014; Mandin et al. 2014, 2016). See Table S2 for  
177 corresponding distributions and input parameters for each SVOC in each medium.

#### 178 Exposure media properties

179 Assessment of  $DD_{\text{derm-gas}}$  (Eq. 3) requires use of a transdermal permeability coefficient,  $k_{p-g}$ ,  
180 estimated using the specific physical and chemical parameters of each SVOC, and a non-  
181 specific mass transport coefficient,  $\gamma_d$ . This last describes the external transport of an SVOC  
182 from the gas phase in the core of a room through the boundary layer adjacent to the skin, see  
183 Eq. 1 (Supplemental Material). A triangular distribution was modeled for  $\gamma_d$ , using the  
184 minimum and maximum values found in the literature and the generally assumed value of 6  
185 m/h as most likely (Pandurangi and Morrison 2008; Tamas et al. 2006; Weschler and Nazaroff  
186 2008). See Table S3 for corresponding distribution and input parameters.

#### 187 Bioaccessibility and bioavailabilities

188 Bioaccessibility in dust ( $f_{\text{dust}}$ ) is the fraction of pollutant released from settled dust into the  
189 gastrointestinal tract and available for absorption (Rostami and Juhasz 2011). For each  
190 SVOC,  $f_{\text{dust}}$  was retrieved from the literature survey performed by Raffy et al. (2016). Oral  
191 bioavailability ( $f_{\text{oral}}$ ) is the fraction of a contaminant reaching the digestive system and  
192 absorbed into systemic circulation (Rostami and Juhasz 2011). Pulmonary bioavailability  
193 ( $f_{\text{pulm}}$ ) is the fraction of a contaminant reaching the alveolar system and absorbed into  
194 systemic circulation. For each SVOC,  $f_{\text{oral}}$  and  $f_{\text{pulm}}$  were retrieved from the following online  
195 databases: the HSDB (<https://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB>) and the Agency  
196 for Toxic Substances and Disease Registry (ATSDR) (<https://www.atsdr.cdc.gov/>). For each  
197 compound displaying at least three available values, triangular distributions were modeled  
198 (minimum, average and maximum values). Where only two values were retrieved, uniform  
199 distributions were modeled (minimum and maximum). Where only a single value was

200 available, triangular distributions were modeled between this value (as the likeliest), and the  
201 minimum and the maximum values retrieved from other compounds belonging to the same  
202 chemical family. Finally, if no value was found for an SVOC, a uniform distribution was  
203 modeled between the minimum and the maximum values found for the other compounds from  
204 the same chemical family - or from the entire studied chemical families taken together if no  
205 other compound from the same chemical family was assessed. See Table S4 for  
206 corresponding distributions and input parameters for each SVOC.

### 207 Human body parameters

208 The World Health Organization (WHO 2006) has identified and suggested common critical  
209 life stages for use in exposure and risk assessment. Because these were not available for every  
210 parameter and age group for populations living in France, we used the U.S. EPA Exposure  
211 Factors Handbook (U.S. EPA 2011). The potential influence of this is covered in the  
212 discussion section. We compiled the distribution of human body weight (BW), body surface  
213 area (BSA), dust ingestion (DI), inhalation rate (IR) and time spent in dwellings (t).  
214 Lognormal distributions were used for BW, BSA and DI. Normal distributions were used for  
215 IR and t. See Table S5 for corresponding distributions and input parameters.

216 **Simulation:** DD from all three routes of exposure (Eq. 1 to 3) and ADD (Eq. 4) were  
217 estimated using Crystal Ball® software (Oracle<sup>®</sup>, version 11.1.1.3.00). Latin Hypercube two-  
218 dimensional simulations were carried out with 500,000 runs for each SVOC, and each age  
219 group. Two-dimensional simulations take into account both the uncertainty (lack of  
220 knowledge about a parameter) and the variability (heterogeneity of a parameter in a  
221 population) of the input parameters. The following parameters were considered variable (i.e.  
222 high variability in relation to uncertainty):  $C_{\text{dust}}$ ,  $C_{\text{part}}$ ,  $C_{\text{gas}}$ ,  $\gamma_{\text{d}}$ , BW, BSA, IR and t, whereas  
223  $\log(K_{\text{ow}})$ , H,  $f_{\text{oral}}$ ,  $f_{\text{dust}}$  and  $f_{\text{pulm}}$ , were considered uncertain. DI was considered both uncertain

224 and variable. Sensitivity analysis was performed in a one-dimensional simulation by assessing  
225 the contribution of each input parameter to ADD variance (the output), using linear  
226 regression.

## 227 **RESULTS**

228 [Figure 1]

229 ADD (ng/kg-bw/d) estimates for the 32 SVOCs for children aged 2 to 3 years are shown in  
230 Figure 1. We are presenting results for this specific segment because young children are more  
231 vulnerable than the rest of the population in terms of exposure to contaminants (due to more  
232 frequent contact with the ground and deposited dust, carrying objects in their mouths, higher  
233 inhalation rates, etc.) and more sensitive in terms of effects (due to ongoing development of  
234 the main organ systems, which continues after birth). Detailed results for ADD and the three  
235 DD from each exposure route for all 11 age groups (birth to age 30) are shown in Tables S8 to  
236 S18. ADD estimations spanned orders of magnitude, with median values ranging from 8.7  
237 pg/kg-bw/d for 2,2',3,4,4'-pentabromodiphenylether (BDE 85) to 1.3  $\mu\text{g/kg-bw/d}$  for  
238 diisobutyl phthalate (DiBP) for children aged 2 to 3 years. Variability of ADD (relative  
239 interpercentile range, see Table S6) ranged from 1.2 for 2,2',4,4',5-pentabromodiphenylether  
240 (BDE 99) to 12 for 2,3,3',4,4'-pentachlorobiphenyl (PCB 105), with a median value of 4. The  
241 median uncertainty on the mean was 40%, ranging from 11% for the benzo[a]pyrene to 230%  
242 for 2,3',4,4',5-pentachlorobiphenyl (PCB 118) (see detailed relative errors in Table S13).

243 The relative contribution of exposure pathways to aggregated median exposure is presented in  
244 Figure 1 (bottom panel) for children aged 2 to 3 years. The relative contributions made by  
245 each route of exposure to total indoor estimates differ by compound, and could be related to  
246 their degree of volatility. For the most volatile SVOCs - that is those compounds having an  
247 octanol-air partition coefficient ( $\log(K_{oa})$ ) value of 9 or less (see Table S1) and expected to be

248 primarily gaseous (Weschler and Nazaroff, 2012), inhalation and dermal contact with the gas  
249 phase were the dominant routes of exposure: fluorene, anthracene, aldrin, dieldrin, tonalide,  
250 galaxolide, tributylphosphate, diethyl phthalate (DEP), dibutyl phthalate (DBP), DiBP and  
251 2,4,4'-trichlorobiphenyl (PCB 28), 2,4',5-trichlorobiphenyl (PCB 31) and 2,2',5,5'-  
252 tetrachlorobiphenyl (PCB 52). For less volatile SVOCs - that is those compounds having a log  
253 ( $K_{oa}$ ) value of 13 or greater (see Table S1) and expected to be primarily in the particle phase  
254 (Weschler and Nazaroff, 2012), dust ingestion was the dominant route of exposure:  
255 diethylhexyl phthalate (DEHP), di-isononyl phthalate (DiNP) and 2,2',4,4',5,6'-  
256 hexabromodiphenylether (BDE 154). The contribution made by different pathways was more  
257 contrasted for SVOCs having a log ( $K_{oa}$ ) value of between 9 and 13 (see Table S1). On the  
258 whole, air was the dominant exposure medium (sum of inhalation and dermal contact > 50%  
259 of ADD) for 28 compounds out of 32, while dust was the major contributor for DEHP, DiNP,  
260 benzyl butyl phthalate (BBP) and 2,2',4,4',5,5'-pentachlorobiphenyl (PCB 101). Overall, dust  
261 ingestion is more contributive to exposure for less volatile chemicals, although this is not the  
262 case with BBP and PCB 101, both of which are found in relatively high concentrations in  
263 dust.

264 Relative contributions of exposure pathways for P95 values were similar to median values.  
265 Notable exceptions were that higher contributions from dust ingestion were found for PCB  
266 118, 2,2',3,4,4',5'-hexachlorobiphenyl (PCB 138) and 2,2',4,4',5,5'-hexachlorobiphenyl (PCB  
267 153), and higher contributions from inhalation were found for benzo[a]pyrene and DBP.

268 [Figure 2]

269 The main sensitivity analysis results are shown in Figure 2 (see Table S7 for detailed results).  
270 These reveal that contamination parameters are most influential:  $C_{gas}$ ,  $C_{part}$  and  $C_{dust}$ , the only  
271 exception being H for BDE 99. Their relative contributions to ADD variance ranged from

272 30% for galaxolide ( $C_{\text{part}}$ ) and BDE 99 (H) to 89% for fluorene ( $C_{\text{gas}}$ ). For most compounds,  
273 the other influential parameters (having contributions higher than 10%) are the other  
274 contamination parameters:  $C_{\text{gas}}$ ,  $C_{\text{part}}$  and  $C_{\text{dust}}$ , and also  $f_{\text{pulm}}$ . For some compounds, other  
275 influential parameters are  $f_{\text{dust}}$  (DEHP and DiNP) alongside physical and chemical  
276 parameters:  $\log(K_{\text{ow}})$  and H (lindane and BDE 99). Furthermore, compounds have been  
277 ranked by volatility in Figure 2 (based on their  $\log(K_{\text{oa}})$  values), from most volatile (fluorene)  
278 to least volatile (DiNP). Among the most volatile SVOCs,  $C_{\text{gas}}$  tended to be the more  
279 influential parameter, while  $C_{\text{dust}}$  and  $C_{\text{part}}$  appeared influential for the least volatile. Lastly,  
280 we note that within each age group, human parameters were not influential.

281 [Figure 3]

282 ADD (ng/kg-bw/d) estimates for the 32 SVOCs for four age groups are shown in Figure 3:  
283 infants aged 0 to 1 months, infants aged 1 to 3 months, children aged 2 to 3 years and adults  
284 aged 21 to 30 years. As expected, ADD decreases with age because of increasing BW - except  
285 within the [0-1 month] category where the dust ingestion rate equals 0 mg, and ADD may be  
286 lower in comparison with other groups. This is true in particular of compounds making a  
287 major contribution to dust ingestion (PCB 101, BBP, DEHP and DiNP). Detailed results for  
288 ADD across all 11 age groups (birth to age 30) are shown in Tables S8 to S18.

## 289 **DISCUSSION**

290 Indoor exposure to 32 SVOCs was modeled using contamination measurements from  
291 dwellings and human body parameters. ADD spanned orders of magnitude from 8.7 pg/kg-  
292 bw/d to 1.3  $\mu\text{g/kg-bw/d}$ . Inhalation, ingestion and dermal pathway all contributed, though  
293 differently across compounds. Indoor exposure estimations were influenced more by  
294 variability in indoor concentrations ( $C_{\text{gas}}$ ,  $C_{\text{part}}$  and  $C_{\text{dust}}$ ) than by uncertainty in physical and  
295 chemical parameters.

296 Study strengths include: 1) estimation of indoor exposure to numerous SVOCs from different  
297 chemical families via gas- and particle-phase inhalation, dermal contact with gas phase, and  
298 ingestion of settled dust; 2) use of field measurements in dust, particulate and gaseous phases;  
299 3) use, when available, of large and nationwide representative datasets; 4) choice of a two-  
300 dimensional probabilistic approach, and 5) consideration of many age groups.

301 Study limitations include: 1) use of an exposure model that neglects dynamic conditions; 2)  
302 use of different data sets for different exposure media, and 3) uncertainty analysis being  
303 restricted to parameter uncertainty.

304 We used steady-state models to estimate indoor exposure to SVOCs. Because indoor air  
305 measurements were performed over a period of one week, and settled dust being collected in a  
306 vacuum cleaner, we assumed that equilibrium had been reached. Because we frequently move  
307 from one environment into another, or between rooms having different concentrations,  
308 equilibrium is rarely achieved for the transfer from air to skin. A transient model developed  
309 by Gong et al. (2014) considered convective mass transfer resistance in the boundary air layer  
310 adjacent to the skin. Morrison et al. (2016) have recently improved this model by including  
311 skin surface lipids, which increase overall resistance to SVOC uptake from air. However,  
312 because these models require parameters that are not available for all compounds, default  
313 parameters are employed - rendering a probabilistic assessment difficult.

314 For contamination data in the three media when the detection frequency was < 1% (Blanchard  
315 et al. 2014; Mandin et al. 2014, 2016) a literature survey was conducted to retrieve other  
316 contamination information, which was then employed (instead of the initially-targeted data)  
317 where a lower LOD or LOQ was used by the authors (see Table S2). This concerned settled  
318 dust and gas phase concentrations for the 2,4,4'-tribromodiphenylether (BDE 28), and gas  
319 phase concentration alone for 12 other SVOCs: benzo[a]pyrene, PCB 31, PCB 105, PCB 118,

320 PCB 138, PCB 153, BDE 47, BDE 85, BDE 99, 2,2',4,4',6-pentabromodiphenylether (BDE  
321 100), 2,2',4,4',5,5'-hexabromodiphenylether (BDE 153) and BDE 154.

322 To put our results into perspective, this indoor aggregate multimedia and multipathway  
323 exposure assessment was compared to the few other studies investigating the same exposure  
324 pathways, namely: Bekö et al. (2013), Gaspar et al. (2014) and Mitro et al. (2016), who  
325 investigated phthalates and galaxolide for children aged 3 to 6 years in Denmark, California  
326 (US) and 14 states of the US, respectively. Our ADD estimated median values for children  
327 aged 3 to 6 years (see Table S14) were compared to: estimated median values for DBP and  
328 DEHP (Bekö et al. 2013; Gaspar et al. 2014); estimated median values for DiBP, BBP and  
329 DEP (Bekö et al. 2013), and estimated average values for DBP, DEHP, DiBP, BBP, DEP and  
330 galaxolide (Mitro et al. 2016). Similar results were found for DBP, DEHP and DEP. For  
331 DiBP, we found a median value equal to 1.1 µg/kg-bw/d, consistent with the 1.5 µg/kg-bw/d  
332 found by Bekö et al. (2013). Using a dust contamination level for this compound that was  
333 seven times lower, Mitro et al. (2016) found a lower exposure level equal to 0.1 µg/kg-bw/d.  
334 This difference in concentration data between French and US samples may also reflect a shift  
335 in the use of phthalates as a result of changes to the formulation of plasticizers. For BBP, we  
336 found a median value equal to 0.1 µg/kg-bw/d, consistent with the 0.2 µg/kg-bw/d of Mitro et  
337 al. (2016). Using a dust contamination level for this compound that was four times lower,  
338 Bekö et al. (2013) found a lower exposure level equal to 0.01 µg/kg-bw/d. Regarding  
339 galaxolide, our exposure level of 0.05 µg/kg-bw/d is consistent with Mitro et al. (2016) who  
340 found 0.1 µg/kg-bw/d - in line with dust contamination that was twice as high as ours.

341 Pathway contributions were found to be similar to Mitro et al. (2016) for DBP, DEP and  
342 galaxolide, with inhalation as the main route of exposure (> 50%), followed by dermal  
343 absorption from the gas phase and dust ingestion. Pathway contribution estimates from Bekö  
344 et al. (2013) for DiBP, BBP and DEHP are also similar to our findings, with total uptake



345 dominated by dermal absorption from the gas phase for DiBP, and dust ingestion for BBP and  
346 DEHP. The results of the present study were also comparable to Gaspar et al. (2014), with  
347 dust ingestion dominating total uptake of DEHP.

348 In addition to the indoor exposure in dwellings assessed by this study, other sources of  
349 exposure (e.g. diet and personal care products) and other indoor environments (e.g. schools  
350 and offices) for many of these compounds contribute to total exposure, as well as direct  
351 dermal contact with surfaces. The use of personal care products, directly applied to skin or  
352 inhaled from aerosols, could have a non-negligible contribution to total exposure for some  
353 compounds, e.g., for certain phthalates (Romero-Franco et al. 2011) and musks (Zhang et al.  
354 2017). Most of the studies addressing SVOC exposure assessment also looked at dietary  
355 ingestion (Beamer et al. 2012; Duggan et al. 2003; Lorber et al. 2007; Roosens et al. 2010;  
356 Trudel et al. 2011; Wilson et al. 2003; Wormuth et al. 2006). This could naturally lead to  
357 higher ADD estimates and different pathway contributions. Diet has been shown to be an  
358 important source of exposure for certain compounds: e.g. some PBDEs (Trudel et al. 2011)  
359 and some phthalates (Wilson et al. 2003). However, several authors who have performed  
360 exposure assessments on both indoor and diet exposure found that dietary ingestion was not  
361 the main route of exposure for chlorpyrifos (Beamer et al. 2012; Duggan et al. 2003), DEP,  
362 BBP and DiNP (Wormuth et al. 2006), some PBDEs (Lorber et al. 2007), some PCBs and  
363 some OCs (Wilson et al. 2003). For certain compounds, exposure via dietary ingestion could  
364 also equal exposure via non-dietary ingestion: e.g. DEHP (Wormuth et al. 2006), and  
365 anthracene, phenanthrene, benzo[a]pyrene and fluorene (Wilson et al. 2003). To put our  
366 results in perspective, we compared them to those of the French infant total diet study  
367 (ANSES 2016a, 2016b). We compared to lower bound (LB) and upper bound (UB) median  
368 estimates for children aged 2 to 3 years. For 6 PCBs (sum of PCB 28, 52, 101, 138, 153 and  
369 2,2',3,4,4',5,5'-heptachlorobiphenyl (PCB 180)) our median residential indoor ADDs were

370 about one order of magnitude lower than dietary exposure estimates (LB and UB). For 7  
371 PBDEs (sum of BDE 28, 47, 99, 100, 153, 154 and 2,2',3,4,4',5',6-heptabromodiphenylether  
372 (BDE 183)) they were about one order of magnitude higher (LB and UB). For DEHP they  
373 were of the same order of magnitude (LB and UB). For DiNP and lindane they were of the  
374 same order of magnitude as the LB estimates and about one order of magnitude lower than the  
375 UB. For BBP, DEP and DBP they were of the order of magnitude of UB estimates, and one  
376 order of magnitude higher than the LB. For DiBP they were about one or two orders of  
377 magnitude higher than UB and LB respectively. For chlorpyrifos, dieldrin and aldrin they  
378 were of the same order of magnitude, in comparison with the LB estimates, and about two  
379 orders of magnitude lower, in comparison with the UB.

380 Compared to more traditional deterministic approaches, probabilistic methods (e.g. Monte  
381 Carlo simulations) have the main advantage of addressing input parameters featuring both  
382 uncertainty and variability. ADD estimations were mainly dependent on variability in indoor  
383 concentrations ( $C_{\text{gas}}$ ,  $C_{\text{part}}$  and  $C_{\text{dust}}$ ), and secondarily by uncertainty in  $f_{\text{pulm}}$  and physical and  
384 chemical parameters. Uncertainty of parameters is related to lack of total knowledge, whereas  
385 data variability refers to true heterogeneity. An interesting finding is that although input  
386 parameter uncertainty has been accused of causing broad uncertainty in exposure estimates  
387 (Pelletier et al. 2017; Salthammer and Schripp 2015), it appeared to contribute less to total  
388 variance of ADD than did variability. For every compound but one, variability of a  
389 concentration explains at least 40% of ADD variance; in half of all cases, this exceeded 60%.  
390 Concentrations of a chemical within dwellings span orders of magnitude, especially as a result  
391 of the presence (or absence) of a source in the dwelling. Physical and chemical parameters  
392 can either be measured experimentally or calculated using other chemical properties.  
393 Depending on which of these methods is used, it follows that values vary by one order of  
394 magnitude or more (Finizio et al. 1997) - and these uncertainties are propagated in the

395 calculation of  $DD_{\text{derm-gas}}$  and then ADD. Up to 34% of ADD variation for aldrin could be  
396 explained by uncertainty in  $f_{\text{pulm}}$  (see Table S7) because data for most of the SVOCs were  
397 unavailable. Indeed, data were available for just two compounds: 2,2',4,4'-  
398 tetrabromodiphenylether (BDE 47) and benzo[a]pyrene, leading to modeling of uniform  
399 distribution between 0 and 100% for most compounds (see Table S4) and to potential  
400 overestimation of inhalation exposure. The same limit was encountered for the other uptake  
401 fractions ( $f_{\text{oral}}$  and  $f_{\text{dust}}$ ), though to a lesser extent. For most SVOCs, in respect of  $f_{\text{oral}}$ , even  
402 where pharmacokinetics studies were available and provided qualitative evidence that the  
403 SVOC was absorbed following oral exposure, no quantitative data describing *in vivo* oral  
404 absorption were available in the literature. Moreover, in laboratory animals, absorption of a  
405 compound can also depend strongly on experimental parameters, such as the carrier vehicle  
406 employed (Huwe et al. 2008). For example, gastrointestinal lindane bioavailability in rats  
407 ranged from 6% when the compound was suspended in water to 99% when given in oil. A  
408 further aspect is that we used  $PM_{10}$  measurements, whereas a smaller sampling fraction would  
409 have been more representative of respirable particles - this may result in an overestimation of  
410 the inhalation pathway for those SVOCs mainly present in the particulate phase (BDE 47,  
411 BDE 99, benzo[a]pyrene, PCB 118, PCB 138 and PCB 153). Human body parameters (BW,  
412 BSA, IR, DI and t) are highly variable and, to a lesser extent, uncertain. Özkaynak et al  
413 (2011) found significant uncertainty on DI, and this result led us to consider both uncertainty  
414 and variability for this parameter in our study. Nevertheless, we found that human body  
415 parameters made a marginal or null contribution (see Table S7) to the variance of ADD for a  
416 child aged 2 to 3 years, for each SVOC. It is however important to bear in mind both that we  
417 ran the model age-group by age-group, and that these parameters can have a broader impact  
418 when applied to a more diverse population.

419 Recent studies have investigated and modeled the clothing effect. Morrison et al. (2016) have  
420 shown that frequency of bathing and changing clothes were influential in terms of dermal  
421 uptake. In addition, introduction to the model of a skin surface lipid film, and its interactions  
422 with clothing, may affect results. The authors assessed the influence of clothing on the dermal  
423 uptake of two phthalates (DEP and DBP). They found that clean clothes were protective  
424 against air pollutants, whereas worn clothes, because they had adsorbed air pollutants,  
425 increased dermal uptake. Because only clean clothes could be considered protective, we  
426 decided to not take into account the role of clothing in this study, making the assumption that  
427 the total body surface area was exposed to the gaseous phase.

## 428 **CONCLUSIONS**

429 This indoor aggregate multimedia and pathway exposure assessment considered a wide range  
430 of pollutants, across a broad population. It reveals that exposure spanned orders of magnitude,  
431 from pg/kg-bw/d to  $\mu\text{g/kg-bw/d}$  and was mainly dependent on indoor SVOC contaminations.  
432 Within the boundaries of the conceptual model we used, exposure variability overwhelmed its  
433 uncertainty. Air was the dominant medium for most compounds, either by inhalation or  
434 dermal contact. Along with evidence of these compounds causing similar toxic effects  
435 (Fournier et al. 2014; Mitro et al. 2016), these findings lend support to the call for cumulative  
436 risk assessment of indoor SVOCs.

437

438 Acknowledgments: this work was supported by a doctoral Grant (Maud Pelletier) from  
439 Fondation de France (Grant Number 2014-00053331) and Agence de l'Environnement et de  
440 la Maîtrise de l'énergie (Grant Number TEZ14-36).

441

442 The authors declare having no competing financial interests.

443 **REFERENCES**

- 444 ANSES (2016a). Etude de l'Alimentation Totale infantile 2 (EATi) – Tome 2 – Partie 3 :  
445 Composés organiques. Rapport d'expertise collective.  
446 <https://www.anses.fr/fr/system/files/ERCA2010SA0317Ra-Tome2-Part3.pdf>
- 447 ANSES (2016b). Etude de l'Alimentation Totale infantile 2 (EATi) – Tome 2 – Partie 4 :  
448 Résultats relatifs aux résidus de pesticides. Rapport d'expertise collective.  
449 <https://www.anses.fr/fr/system/files/ERCA2010SA0317Ra-Tome2-Part4.pdf>
- 450 Armstrong, B., Hutchinson, E., Unwin, J., and Fletcher, T. (2004). Lung cancer risk after  
451 exposure to polycyclic aromatic hydrocarbons: A review and meta-analysis, *Environ.*  
452 *Health Persp.*, 112, 970–978.
- 453 Baldi, I., Filleul, L., Mohammed-Brahim, B., Fabrigoule, C., Dartigues, J.F., Schwall, S.,  
454 Drevet, J.P., Salamon, R., and Brochard, P. (2001). Neuropsychologic effects of long-  
455 term exposure to pesticides: Results from the French Phytoner study, *Environ. Health*  
456 *Persp.*, 109, 839–844.
- 457 Batterman, S., Chen, T.C., Chernyak, S., and Godwin, C. (2009). Design and performance  
458 evaluation of a medium flow sampler for airborne brominated flame retardants  
459 (BFRs). *J. Environ. Monitor.*, 11(4), 858-866.
- 460 Beamer, P.I., Canales, R.A., Ferguson, A.C., Leckie, J.O., and Bradman, A. (2012). Relative  
461 pesticide and exposure route contribution to aggregate and cumulative dose in young  
462 farmworker children. *Int. J. Environ. Res. Public. Health*, 9(1), 73–96.
- 463 Bekö, G., Weschler, C.J., Langer, S., Callesen, M., Toftum, J., and Clausen, G. (2013).  
464 Children's Phthalate Intakes and Resultant Cumulative Exposures Estimated from  
465 Urine Compared with Estimates from Dust Ingestion, Inhalation and Dermal

466 Absorption in Their Homes and Daycare Centers, *PLoS One* 8(4), e62442.

467 Blanchard, O., Glorennec, P., Mercier, F., Bonvallot, N., Chevrier, C., Ramalho, O., Mandin,  
468 C., and Le Bot, B. (2014). Semivolatile Organic Compounds in Indoor Air and Settled  
469 Dust in 30 French Dwellings, *Environ. Sci. Technol.*, 48, 3959–3969.

470 Blanc-Lapierre, A., Bouvier, G., Garrigou, A., Canal-Raffin, M., Raheison, C., Brochard, P.,  
471 and Baldi, I. (2012). Chronic central nervous system effects of pesticides : state-of-  
472 the-art, *Rev. Epidemiol. Sante.*, 60(5), 389–400.

473 Bonvallot, N., Mandin, C., Mercier, F., Le Bot, B., and Glorennec, P. (2010). Health ranking  
474 of ingested semi-volatile organic compounds in house dust: an application to France,  
475 *Indoor Air*, 20, 458–472.

476 Burmaster, D.E., and Hull, D.A. (1997). Using lognormal distributions and lognormal  
477 probability plots in probabilistic risk assessments. *Hum. Ecol. Risk Assess.*, 3(2), 235-  
478 255.

479 Casas, M., Chevrier, C., Den Hond, E., Fernandez, M. F., Pierik, F., Philippat, C., Slama, R.,  
480 Toft, G., Vandentorren, S., Wilhelm, M., and Vrijheid, M. (2013). Exposure to  
481 brominated flame retardants, perfluorinated compounds, phthalates and phenols in  
482 European birth cohorts: ENRIECO evaluation, first human biomonitoring results, and  
483 recommendations. *Int. J. Hyg. Envir. Heal.*, 216(3), 230-242.

484 Duggan, A., Charnley, G., Chen, W., Chukwudebe, A., Hawk, R., Krieger, R.I., Ross, J., and  
485 Yarborough, C. (2003). Di-alkyl phosphate biomonitoring data: assessing cumulative  
486 exposure to organophosphate pesticides. *Regul. Toxicol. Pharmacol.*, 37(3), 382–395.

487 Elbaz, A., Clavel, J., Rathouz, P.J., Moisan, F., Galanaud, J.P., Delemotte, B., Alperovitch,  
488 A., and Tzourio, C. (2009). Professional Exposure to Pesticides and Parkinson  
489 Disease, *Ann. Neurol.*, *66*, 494–504.

490 Finizio, A., Mackay, D., Bidleman, T., and Harner, T. (1997). Octanol-air partition coefficient  
491 as a predictor of partitioning of semivolatile organic chemicals to aerosols, *Atmos.*  
492 *Environ.*, *31*, 2289–2296.

493 Fournier, K., Glorennec, P., and Bonvallot, N. (2014). An exposure-based framework for  
494 grouping pollutants for a cumulative risk assessment approach: Case study of indoor  
495 semi-volatile organic compounds. *Environ. Res.*, *130*, 20-28.

496 Gaspar, F.W., Castorina, R., Maddalena, R.L., Nishioka, M.G., McKone, T.E., and Bradman,  
497 A. (2014). Phthalate Exposure and Risk Assessment in California Child Care  
498 Facilities. *Environ. Sci. Technol.*, *48*(13), 7593–7601.

499 Gong, M., Zhang, Y., and Weschler, C.J. (2014). Predicting dermal absorption of gas phase  
500 chemicals: transient model development, evaluation, and application, *Indoor Air*, *24*,  
501 292–306.

502 Helsel, D.R. (2011). *Statistics for censored environmental data using Minitab and R* (Vol. 77).  
503 John Wiley & Sons.

504 Helsel, D.R., and Hirsch, R.M. (1992). *Statistical methods in water resources* (Vol. 49).  
505 Elsevier.

506 Huwe, J.K., Hakk, H., Smith, D.J., Diliberto, J.J., Richardson, V., Stapleton, H.M., and  
507 Birnbaum, L.S. (2008). Comparative absorption and bioaccumulation of  
508 polybrominated diphenyl ethers following ingestion via dust and oil in male rats.  
509 *Environ. Sci. Technol.*, *42*(7), 2694-2700.



510 IARC (2015a). Polychlorinated biphenyls and polybrominated biphenyls, *IARC Monogr.*  
511 *Eval. Carcinog. Risk Chem. Hum.*, Vol. 107.  
512 <http://monographs.iarc.fr/ENG/Monographs/vol107/index.php>

513 IARC (2015b). Some organophosphate insecticides and herbicides: diazinon, glyphosate,  
514 malathion, parathion, and tetrachlorvinphos, *IARC Monogr. Eval. Carcinog. Risk*  
515 *Chem. Hum.*, Vol. 112. [http://](http://monographs.iarc.fr/ENG/Monographs/vol112/index.php)  
516 <http://monographs.iarc.fr/ENG/Monographs/vol112/index.php>

517 Linares, V., Perelló, G., Nadal, M., Gómez-Catalán, J., Llobet, J. M., and Domingo, J.L.  
518 (2010). Environmental versus dietary exposure to POPs and metals: A probabilistic  
519 assessment of human health risks. *J Env. Monit*, 12(3), 681–688.

520 Lorber, M. (2007). Exposure of Americans to polybrominated diphenyl ethers. *J. Expo. Sci.*  
521 *Environ. Epidemiol.*, 18(1), 2–19.

522 Mackay, D., Shiu, W.Y., Ma, K.C., and Lee, S.C. (2010a). Handbook of physical-chemical  
523 properties and environmental fate for organic chemicals, Vol. 1, Introduction and  
524 hydrocarbons. CRC Press.

525 Mackay, D., Shiu, W.Y., Ma, K.C., and Lee, S.C. (2010b). Handbook of physical-chemical  
526 properties and environmental fate for organic chemicals, Vol. 2, Halogenated  
527 hydrocarbons. CRC Press.

528 Mackay, D., Shiu, W.Y., Ma, K.C., and Lee, S.C. (2010c). Handbook of physical-chemical  
529 properties and environmental fate for organic chemicals, Vol. 3, Oxygen containing  
530 compounds. CRC Press.

531 Mackay, D., Shiu, W.Y., Ma, K.C., and Lee, S.C. (2010d). Handbook of physical-chemical  
532 properties and environmental fate for organic chemicals, Vol. 4, Nitrogen and sulfur

533 containing compounds and pesticides. CRC Pressed.

534 Mandin, C., Mercier, F., Ramalho, O., Lucas, J. P., Gilles, E., Blanchard, O., Bonvallot, N.,  
535 Glorennec, P., and Le Bot, B. (2016). Semi-volatile organic compounds in the  
536 particulate phase in dwellings: A nationwide survey in France. *Atmos. Environ.*, *136*,  
537 82-94.

538 Mandin C., Mercier F., Lucas J-P., Ramalho O., Blanchard O., Bonvallot N., Raffy G., Gilles  
539 E., Glorennec P., and Le Bot B. ECOS-POUSS: A Nationwide Survey of Semi-  
540 Volatile Organic Compounds in Home Settled Dust. Proceedings of the Indoor Air  
541 conference 2014, July 7-12 2014, Hong Kong, Vol. VI, 143-148.

542 Mitro, S.D., Dodson, R.E., Singla, V., Adamkiewicz, G., Elmi, A. F., Tilly, M. K., Zota, A.R.,  
543 (2016). Consumer Product Chemicals in Indoor Dust: A Quantitative Meta-analysis of  
544 U.S. Studies. *Environ. Sci. Technol.*, *50*(19), 10661–10672.

545 Morrison, G.C., Weschler, C.J., and Bekö, G. (2016). Dermal uptake directly from air under  
546 transient conditions: advances in modeling and comparisons with experimental results  
547 for human subjects. *Indoor air*, *26*(6), 913-924.

548 Özkaynak, H., Xue, J., Zartarian, V. G., Glen, G., and Smith, L. (2011). Modeled estimates of  
549 soil and dust ingestion rates for children. *Risk Anal.*, *31*(4), 592-608.

550 Pandrangi, L.S., and Morrison, G.C. (2008). Ozone interactions with human hair: Ozone  
551 uptake rates and product formation, *Atmos. Environ.*, *42*, 5079–5089.

552 Pelletier, M., Bonvallot, N., Ramalho, O., Blanchard, O., Mercier, F., Mandin, C., Le Bot, B.,  
553 and Glorennec, P. (2017). Dermal absorption of semivolatile organic compounds from  
554 the gas phase: Sensitivity of exposure assessment by steady state modeling to key  
555 parameters. *Environ. Int.*, *102*, 106-113.

556 Raffy G., Mercier F., Glorennec P., Mandin C., and Le Bot B. Human exposure to semi-  
557 volatile organic compounds (SVOCs) via dust ingestion: a review of influencing  
558 factors. Indoor Air conference, Ghent, 2016.  
559 <http://www.isiaq.org/docs/Papers/Paper853.pdf>

560 Romero-Franco, M., Hernández-Ramírez, R. U., Calafat, A. M., Cebrián, M. E., Needham, L.  
561 L., Teitelbaum, S., Wolff M.S., and López-Carrillo, L. (2011). Personal care product  
562 use and urinary levels of phthalate metabolites in Mexican women. *Environ. Int.*,  
563 *37*(5), 867-871.

564 Roosens, L., Cornelis, C., D'Hollander, W., Bervoets, L., Reynders, H., Van Campenhout, K.,  
565 Van Den Heuvel, R., Neels, H., and Covaci, A. (2010). Exposure of the Flemish  
566 population to brominated flame retardants: Model and risk assessment. *Environ. Int.*,  
567 *36*(4), 368–376.

568 Roper, C.S., Simpson, A.G., Madden, S., Serex, T.L., and Biesemeier, J.A. (2006).  
569 Absorption of [<sup>14</sup>C]-tetrabromodiphenyl ether (TeBDE) through human and rat skin  
570 in vitro. *Drug Chem. Toxicol.*, *29*(3), 289-301.

571 Rostami, I., and Juhasz, A.L. (2011). Assessment of persistent organic pollutant (POP)  
572 bioavailability and bioaccessibility for human health exposure assessment: a critical  
573 review. *Crit. Rev. Env. Sci. Tec.*, *41*(7), 623-656.

574 Rubin, B.S. (2011). Bisphenol A: An endocrine disruptor with widespread exposure and  
575 multiple effects, *J. Steroid Biochem.*, *127*, 27–34.

576 Salthammer, T., and Schripp, T. (2015). Application of the Junge- and Pankow-equation for  
577 estimating indoor gas/particle distribution and exposure to SVOCs, *Atmos. Environ.*,  
578 *106*, 467–476.

579 Sukiene, V., von Goetz, N., Gerecke, A. C., Bakker, M. I., Delmaar, C. J., & Hungerbuhler,  
580 K. (2017). Direct and Air-Mediated Transfer of Labeled SVOCs from Indoor Sources  
581 to Dust. *Environmental Science & Technology*, 51(6), 3269-3277.

582 Shi, S., and Zhao, B. (2015). Estimating indoor semi-volatile organic compounds (SVOCs)  
583 associated with settled dust by an integrated kinetic model accounting for aerosol  
584 dynamics. *Atmos. Environ.*, 107, 52-61.

585 Tamas, G., Weschler, C.J., Bako-Biro, Z., Wyon, D.P., and Strom-Tejsten, P. (2006). Factors  
586 affecting ozone removal rates in a simulated aircraft cabin environment, *Atmos.*  
587 *Environ.*, 40, 6122–6133.

588 Trudel, D., Scheringer, M., von Goetz, N., and Hungerbühler, K. (2011). Total Consumer  
589 Exposure to Polybrominated Diphenyl Ethers in North America and Europe. *Environ.*  
590 *Sci. Technol.*, 45(6), 2391–2397.

591 U.S. EPA (2013). Estimation Program Interface (EPI) Suite (last updated on 15.3.2013,  
592 Version EPI 4.11).

593 U.S. EPA (2011). Exposure Factors Handbook.

594 U.S. EPA (2005). Guidance on selecting age groups for monitoring and assessing childhood  
595 exposures to environmental contaminants. National Center for Environmental  
596 Assessment, Washington, DC; EPA/630/P-03/003F. Available from: National  
597 Technical Information Service, Springfield, VA, and online at <http://epa.gov/ncea>.

598 Weschler, C.J., and Nazaroff, W.W. (2014). Dermal Uptake of Organic Vapors Commonly  
599 Found in Indoor Air, *Environ. Sci. Technol.*, 48, 1230–1237.

600 Weschler, C.J., and Nazaroff, W.W. (2012). SVOC exposure indoors: fresh look at dermal

601 pathways, *Indoor Air*, 22, 356–377.

602 Weschler, C.J., and Nazaroff, W.W. (2010). SVOC partitioning between the gas phase and  
603 settled dust indoors, *Atmos. Environ.*, 44, 3609–3620.

604 Weschler, C.J., and Nazaroff, W.W. (2008). Semivolatile organic compounds in indoor  
605 environments, *Atmos. Environ.*, 42, 9018–9040.

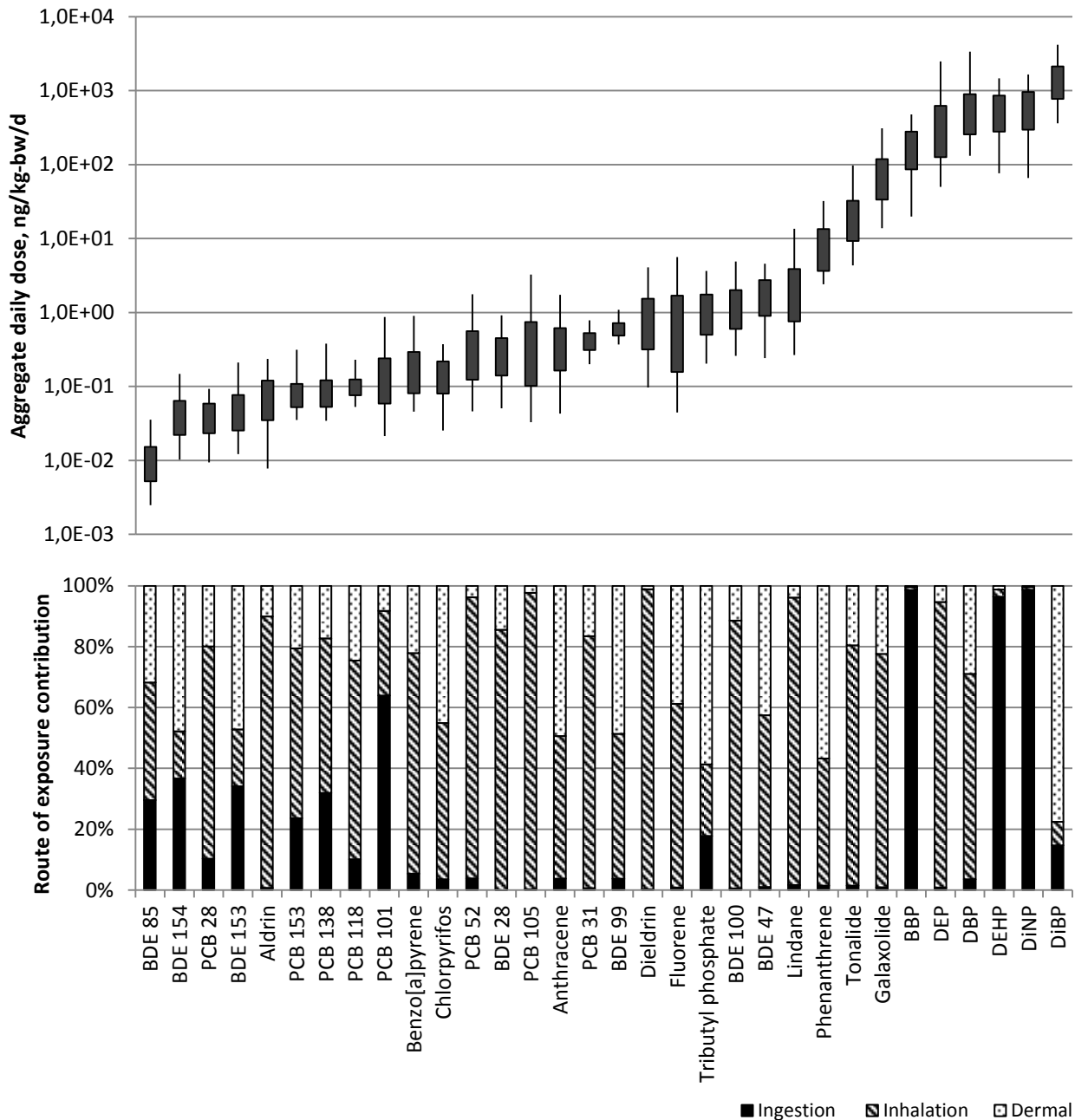
606 Wilson, N.K., Chuang, J.C., Lyu, C., Menton, R., and Morgan, M.K. (2003). Aggregate  
607 exposures of nine preschool children to persistent organic pollutants at day care and at  
608 home. *J. Expo. Anal. Environ. Epidemiol.*, 13(3), 187–202.

609 WHO (2006). Principles for Evaluating Health Risks in Children Associated with Exposure to  
610 Chemicals. International Programme on Chemical Safety, World Health Organization,  
611 Geneva (Environmental Health Criteria 237).

612 Wormuth, M., Scheringer, M., Vollenweider, M., and Hungerbühler, K. (2006). What Are the  
613 Sources of Exposure to Eight Frequently Used Phthalic Acid Esters in Europeans?  
614 *Risk Anal.*, 26(3), 803–824.

615 Zaganas, I., Kapetanaki, S., Mastorodemos, V., Kanavouras, K., Colosio, C., Wilks, M.F., and  
616 Tsatsakis, A.M. (2013). Linking pesticide exposure and dementia: What is the  
617 evidence? *Toxicology*, 307, 3–11.

618 Zhang, X., Yu, Y., Gu, Y., Li, X., Zhang, X., and Yu, Y. (2017). In vitro determination of  
619 transdermal permeation of synthetic musks and estimated dermal uptake through  
620 usage of personal care products. *Chemosphere*, 173, 417–424.



623 **Figure 1.** Top panel of the graph shows Aggregate daily dose of each SVOC for a child aged  
 624 2 to 3 years (ng/kg-bw/d), percentiles 5<sup>th</sup>, 25<sup>th</sup>, 75<sup>th</sup> and 95<sup>th</sup> are presented in box plot format.  
 625 The bottom panel of the graph shows the contribution made by each route of exposure  
 626 (ingestion, inhalation and dermal exposure from air) to total indoor exposure, based on the  
 627 median value estimated for each SVOC for children aged 2 to 3 years (ng/kg-bw/d).

SVOC	Relative contribution (%)							
	[10-20]	[20-30]	[30-40]	[40-50]	[50-60]	[60-70]	[70-80]	[80-90]
Fluorene								<b>Cgas</b>
DEP	<b>Cdust</b>	<b>fpulm</b>			<b>Cgas</b>			
Anthracene								<b>Cgas</b>
Phenanthrene	<b>Cdust</b>						<b>Cgas</b>	
PCB 28			<b>fpulm</b>	<b>Cgas</b>				
PCB 31		<b>fpulm</b>			<b>Cgas</b>			
Tonalide		<b>fpulm</b>		<b>Cpart</b>				
Aldrin			<b>fpulm</b>		<b>Cgas</b>			
Dieldrin			<b>fpulm</b>		<b>Cpart</b>			
Galaxolide		<b>fpulm Cgas</b>	<b>Cpart</b>					
Tributylphosphate							<b>Cgas</b>	
DiBP		<b>Cdust</b>				<b>Cgas</b>		
PCB 52	<b>Cdust</b>	<b>fpulm</b>			<b>Cpart</b>			
DBP		<b>fpulm</b>			<b>Cpart</b>			
Lindane	<b>fpulm H</b>	<b>Cpart</b>	<b>Cgas</b>					
Chlorpyrifos		<b>fpulm</b>			<b>Cgas</b>			
BBP	<b>Cpart</b>				<b>Cdust</b>			
PCB 101	<b>Cgas</b>					<b>Cdust</b>		
BDE 28	<b>fpulm</b>	<b>Cpart</b>		<b>Cgas</b>				
PCB 138		<b>Cgas</b>				<b>Cdust</b>		
PCB 153			<b>Cgas</b>	<b>Cdust</b>				
PCB 118		<b>Cdust</b>			<b>Cgas</b>			
PCB 105	<b>fpulm</b>					<b>Cpart</b>		
BDE 47								<b>Cgas</b>
Benzo[a]pyrene								<b>Cpart</b>
BDE 99		<b>Kow</b>	<b>H</b>					
BDE 85							<b>Cgas</b>	
BDE 100	<b>fpulm</b>					<b>Cpart</b>		
BDE 153	<b>Cdust</b>					<b>Cgas</b>		
DEHP	<b>fdust</b>					<b>Cdust</b>		
BDE 154		<b>Cdust</b>				<b>Cgas</b>		
DiNP			<b>fdust</b>	<b>Cdust</b>				

629

630 **Figure 2.** Relative contribution (%) of key parameters ( $C_{gas}$ ,  $C_{part}$ ,  $C_{dust}$ ,  $f_{pulm}$ ,  $f_{dust}$ ,  $\log(K_{ow})$ )  
631 and H) to total variation of Aggregate daily doses from exposures to 32 indoor SVOCs  
632 (ng/kg-bw/d) for children aged 2 to 3 years. Only relative contributions above 10% are  
633 represented. Bold font indicates the more influential parameter for each SVOC. Compounds  
634 are ranked according to their volatility (based on their  $\log(K_{oa})$  values) from the most volatile  
635 (fluorene) to the least volatile (DiNP).

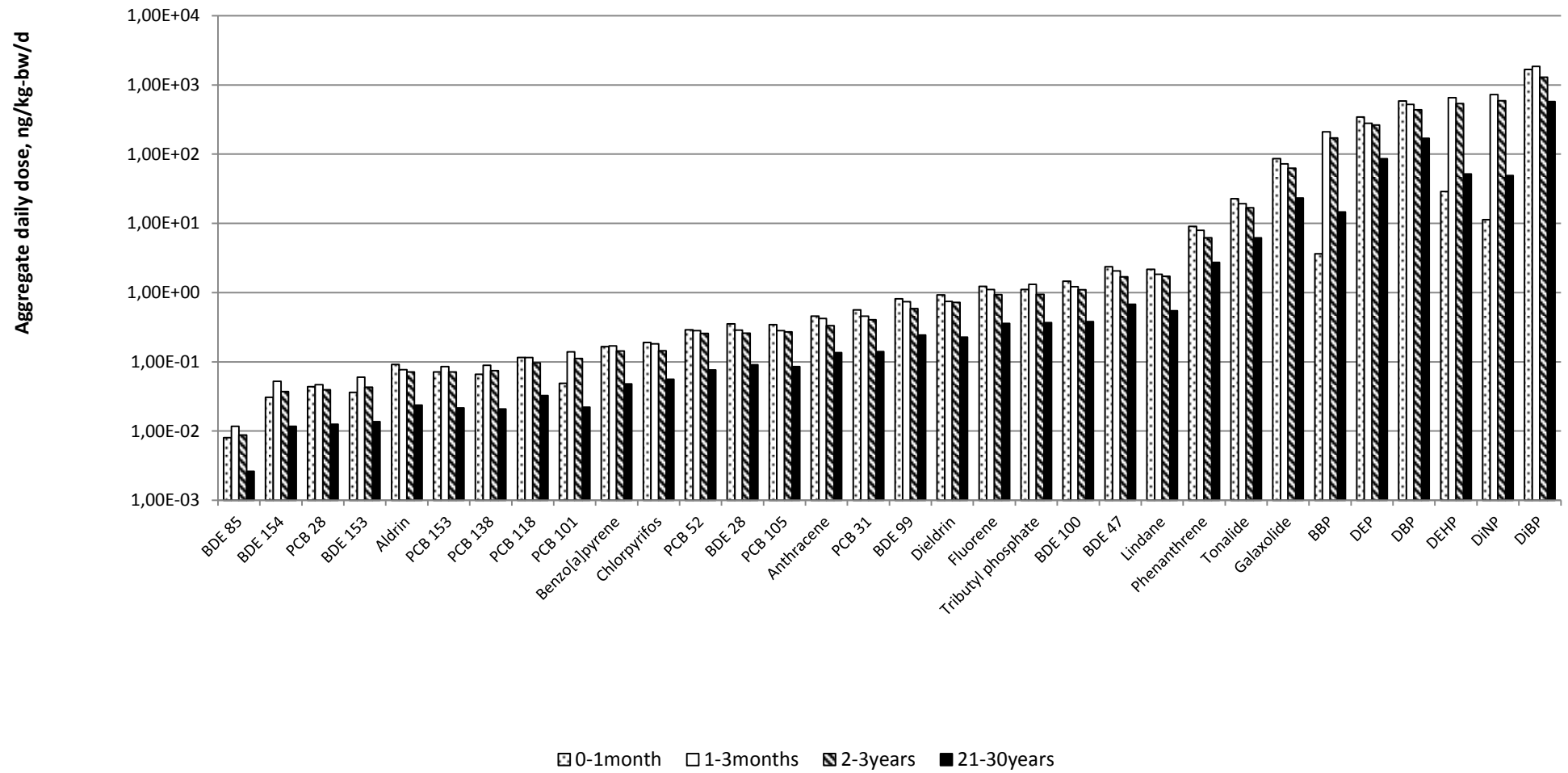


Figure 3. Aggregate daily dose of each SVOC for four age groups (ng/kg-bw/d).