



HAL
open science

Dietary exposure to perfluoroalkyl acids, brominated flame retardants and health risk assessment in the French infant total diet study

Gilles Rivière, Julien Jean, Sébastien Gorecki, Marion Hulin, Martine Kolf-Clauw, Cyril Feidt, Nicole Picard-Hagen, Paule Vasseur, Bruno Le Bizec, Véronique Sirot

► To cite this version:

Gilles Rivière, Julien Jean, Sébastien Gorecki, Marion Hulin, Martine Kolf-Clauw, et al.. Dietary exposure to perfluoroalkyl acids, brominated flame retardants and health risk assessment in the French infant total diet study. *Food and Chemical Toxicology*, 2019, 131, pp.110561. 10.1016/j.fct.2019.06.008 . hal-03210313

HAL Id: hal-03210313

<https://hal.univ-lorraine.fr/hal-03210313v1>

Submitted on 29 Apr 2021

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.

Dietary exposure to perfluoroalkyl acids, brominated flame retardants and health risk assessment in the French infant total diet study

Gilles Rivière*, Julien Jean, Sébastien Gorecki, Marion Hulin, Martine Kolf-Clauw, Cyril Feidt, Nicole Picard-Hagen, Paule Vasseur, Bruno Le Bizec and Véronique Sirot

French Agency for food, environmental and occupational health & safety. 14 rue Pierre & Marie Curie, F-94700, Maisons-Alfort, France

* : Corresponding author: Gilles Rivière, French Agency for food, environmental and occupational health & safety. 14 rue Pierre & Marie Curie, F-94700, Maisons-Alfort, France. gilles.riviere@anses.fr

The conclusions presented in the following article represent only the opinion of the authors.

Abstract

Perfluoroalkyl acids (PFAAs) and brominated flame retardants (BFRs) are widely used and present in human food. Due to the increased susceptibility to pollutants of the young children, we conducted a total diet study focusing on this population. Around 200 baby and common food composite samples, prepared “as consumed”, have been analysed for PFAAs, hexabromocyclododecanes, polybrominated biphenyls, polybrominated diphenyl ethers and tetrabromobisphenol A. The dietary exposure of 705 children aged 1 to 36 months was assessed. Detection rates were very low for PFAAs (<5%) and varied from 4 to 93% for BFRs, depending on the congeners. Regarding the provisional health-based guidance values set by EFSA in 2018 for PFOA and PFOS at 0.8 and 1.8 ng.kg bw⁻¹.d⁻¹, respectively, 20 to 100% of children exceeded them, depending on the age. Efforts should be made to decrease the PFAAs contamination of food. This study also highlighted that for other PFAAs, toxicological studies are needed to set dietary health-based guidance values, to assess their related health risk. Conversely, dietary exposures to BFRs were much lower than the respective health based guidance values or margins of safety were high enough, and consequently not considered at-risk due to very low contamination of the infant specific foods.

1. INTRODUCTION

Early stages of life, i.e. the fetal and early postnatal periods, correspond to periods of increased susceptibility (Makri *et al.*, 2004; Sly and Flack, 2008; Diamanti Kandarakis *et al.*, 2009). Several pieces of evidence suggest that fetuses and infants may be more sensitive to pollutants (Landrigan *et al.* 2003). The French Agency for Food, Environmental and Occupational Health & Safety (ANSES) launched a specific TDS on infants to complete its overall chemical food safety program for the general population (Hulin *et al.*, 2014). More than 500 chemical substances were analyzed in food products consumed by children under 3 years old, including persistent organic pollutants.

Perfluoroalkyl acids (PFAAs) are a large class of chemical contaminants of anthropogenic origin that includes perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA). These substances are highly stable, thermally, chemically and biologically, due to their strong carbon-fluoride bonds. They have surfactant properties and are used in numerous industrial applications and common consumer products: stain- and water-resistant treatments (clothes, rugs, carpets), non-stick coatings (kitchen utensils, paper including food contact materials) and certain specialized applications (fire-fighting foam). Due to their toxic properties, a group of renowned scientists published a statement, known as the “Madrid Statement”, urging the international

community to cooperate in limiting the production and use of PFAAs and in developing safer non fluorinated alternatives (scientists, governments, chemical and product manufacturers (Blum *et al.*, 2015).

PFAAs contaminate several compartments of the environment (water, soil, air) and accumulate in the food chain (Kowalczyk *et al.*, 2012). Food, particularly seafood products, is a significant source of exposure to PFAAs in humans (Cornelis *et al.*, 2012; EFSA, 2008; Trudel *et al.*, 2008, Rivière *et al.*, 2014). PFOS and PFOA persist in the environment and can accumulate in animals and humans. High concentrations are generally observed in the liver, blood and kidneys. Toxicity studies on PFAAs generally only investigate PFOS and PFOA (Olsen *et al.*, 2007). Their apparent elimination half-lives in humans are approximately 4 to 5 years (Olsen *et al.*, 2007; Bartell *et al.* 2010). The main toxic effects reported in animals have been observed in the liver (Seacat *et al.*, 2003), on reproductive and developmental functions and immune and hormonal systems (Seacat *et al.*, 2002, Kang *et al.*, 2016; Grandjean *et al.*, 2012), neurobehavior (Johansson *et al.*, 2007) as well as on lipid metabolism (EFSA, 2008). PFOS and PFOA have neoplastic effects but have not been shown to be genotoxic (EFSA 2008).

Brominated flame retardants (BFRs) are mixtures of chemical substances produced and incorporated in many products due to their fire-retardant properties. They are incorporated in plastics, textiles as well as in electronic equipments. This class encompasses different compounds, including hexabromocyclododecanes (HBCDDs), polybrominated biphenyls (PBBs), polybrominated diphenyl ethers (PBDEs) and tetrabromobisphenol A (TBBPA). Food appears to be the main route of exposure for some BFRs such as PBDEs (Frederiksen *et al.*, 2009; Domingo, 2012). The characterisation of chronic human toxicity of BFRs is difficult since they are often experimentally studied as mixtures. In experimental studies, these compounds have been shown to trigger neurodevelopmental effects (EFSA, 2011a).

For all these compounds, dietary intakes appear to be a major contributor to the overall exposure. On the other hand, some of these compounds trigger effects that can be of particular importance for the youngest population (behavioral, neurodevelopmental or endocrine disruptor effects). It is consequently deemed of importance to estimate the dietary intake of the youngest population to these compounds and to assess the risk related to these exposures. The objective of the present study is to assess the risk to children aged from 0 to 3 years of age related to the presence of PFAA and BFR compounds in food. Exposures were calculated using concentrations of 16 PFAAs, 8 PBDEs, 3 PBBs, 3 HBCDDs and TBBPA measured in food samples collected in the context of the infant total diet study (TDS) performed in France (Hulin *et al.*, 2014).

2. MATERIALS & METHOD

2.1. Consumption data for children under 3

For children under 3 years of age, consumption data came from a national consumption survey conducted specifically on this population (Fantino and Gourmet 2008). Briefly, this cross-sectional study was conducted from January to March 2005 by the Syndicat Français des Aliments de l'Enfance et de la Nutrition Clinique, © « Etude SOFRES 2005 / Université de Bourgogne – Pr M. Fantino pour le Syndicat Français des Aliments de l'Enfance » (Fantino 2005, Fantino and Gourmet 2008). Individual, consecutive 3-day weight food records were collected from a sample of 705 children selected through proportionate quota sampling based on the living area, the age of the children, the occupation of the mother and the family socio-economic category. Totally or partially breastfed infants (during the survey period) were excluded from the study.

2.2. Food sampling

Food samples were collected as previously described (Hulin *et al.*, 2014). To obtain a representative and general view of infant food consumption, food items were selected based on the results of the consumption survey (Fantino and Gourmet, 2008) based on two main criteria:

- The most consumed food in terms of quantity and/or percentage of consumers
- Food items that are known or supposed to be the main contributors to the exposure to one or more substances of interest

Food items were sampled in the same region and prepared as consumed by the same contractor (i.e. peeled, cooked, etc.) based on a result of a national study on parents' food preparation/cooking processes carried out by Anses (Hulin et al., 2014).

Infant products were updated before selecting food items due to the changing market. Food items included common foods such as vegetables, fruits or cakes as well as specific infant foods such as infant formula or jarred baby food. The sampling plan covered over 95% of the total diet of 0 to 3 years old children. To take into account seasonal variability, possible different flavours, and different ways of consuming or preparing foods, all the analysed samples, referred to as "composites" or "pools", were formed of 12 sub-samples of the same food and of equal weight. The products purchased were prepared in a way that reflected as closely as possible what is done in the home: preparation (removal of the non-edible part, washing of foods, etc.) and cooking (duration and power, addition or not of salt, fat, etc.). Specificities in infant food consumption and habits were considered. The infant formulas were diluted and heated most of the time, prepared baby dishes were reheated, vegetables (excluding raw vegetables), meat and fish were cooked according to the practices reported in the survey on practices (Hulin et al., 2014). The sampling phase took place between July 2011 and July 2012 in the center region of France.

2.3. Analytical procedure

2.3.1. Perfluoroalkyl acids

The analyses were performed in food items known or supposed to be contaminated by PFAAs (EFSA, 2012), corresponding to 199 samples out of 457.

The analytical method was used to determine the concentration of 5 perfluoroalkyl sulfonates: PFOS, perfluorobutane sulfonate (PFBS), perfluorohexane sulfonate (PFHxS), perfluoroheptane sulfonate (PFHpS) and perfluoro[-1-]decanesulfonate (PFDS), 11 perfluorocarboxylic acids: PFOA, perfluorobutanoic acid (PFBA), perfluoropentanoic acid (PFPA), perfluorohexanoic acid (PFHxA), perfluoroheptanoic acid (PFHpA), perfluorononanoic acid (PFNA), perfluorodecanoic acid (PFDA), perfluoroundecanoic acid (PFUnA), perfluorododecanoic acid (PFDoA), perfluorotridecanoic acid (PFTrDA) and perfluorotetradecanoic acid (PFTeDA) and one perfluoroalkyl sulfonate (PFOSi) (Kadar *et al.*, 2011). Solid food samples were freeze-dried and extracted with methanol. After evaporation, food extracts were purified onto two consecutive solid-phase extraction (SPE) columns (copolymeric reversed phase and charcoal). Final purified extracts were analysed by liquid chromatography tandem mass spectrometry (LC-MS/MS); electrospray ionisation in the negative ion mode was preferred and at least two transitions were monitored per analyte. Water samples (100 mL), were directly loaded on a reversed-phase SPE column. For milk and dairy products, samples were first extracted using acetone. For fish samples, a dispersive SPE based on charcoal particles was used.

Quantification was performed according to isotopic dilution principles. Each sample was spiked by two ¹³C-labelled quantification standards (¹³C₄-PFOA and ¹³C₄-PFOS) for quantification. A recovery standard (fluorometholone) was added at the end of the analytical process. Depending on the matrix, these recoveries ranged from 30 to 80%. Limits of detection (LOD) and quantification (LOQ), were determined based on the signal-to-noise ratio and depended on the matrix and analyte, overall, they ranged from 0.2 pg.g⁻¹ fresh weight (fw) to 3.7 ng.g⁻¹ fw. Repeatability of the signal was also dependent on the matrix, but was always better than 18.6 % for all analytes.

2.3.2. Brominated flame retardants

The analyses were performed in food items known or supposed to be contaminated by BFRs (EFSA, 2010; EFSA, 2011a, b, c), corresponding to 206 samples out of 457.

The extraction of fat was adapted to the physical characteristics of the samples (Cariou *et al.*, 2006; Debrauwer *et al.*, 2005). For all matrices, quantification standards were added before extraction (eight ¹³C₁₂-labelled PBDE congeners, one ¹³C₁₂-labelled PBB congener and three ¹³C₁₂-labelled HBCD congeners). The solid samples were freeze-dried, ground, and extracted by Accelerated Solvent Extraction (ASE) using a Dionex™ ASE 300™. Pressure and temperature were set to 100 bars and 120°C respectively. Liquid samples underwent protein precipitation through the addition of potassium oxalate. Two additional successive extractions using a solvent mixture of ethanol, ether and hexane were performed to extract the fat.

Purification involved three stages involving silica, Florisil® and charcoal-celite® columns. A recovery standard was added to each vial for each class of compounds (¹³C₁₂-PBDE 138 for PBDEs and PBBs and fluorometholone for HBCDDs) just before injection.

Detection and identification of PBDEs and PBBs were carried out using a Hewlett–Packard 6890 gas chromatograph, equipped with a DB-5MS capillary column (30 m x 0.25 mm ID, 0.25 µm film thickness) coupled to a Jeol JMS-800D (Jeol, Tokyo, Japan), operating at a resolving power of 10,000 (10% valley). Concentrations of the following PBDE congeners were measured: BDE-28, -47, -99, -100, -153, -154, -183 and -209. Concentrations of PBB-52, PBB-101 and PBB-153 were also measured. For PBDEs, analytical limits in infant specific food items ranged from 0.01 to 1.4 pg.g⁻¹ fw and from 0.1 to 15 pg.g⁻¹ fw for common food items. For PBBs, analytical limits ranged from 0.007 to 5.4 pg.g⁻¹ fw depending on the matrix. Detection rates were 8, 4 and 12% for PBB-52, -101 and -153, respectively.

HBCDD were analysed by LC-MS/MS. HBCDD isomers were separated by reverse-phase chromatography using a Hypersil Gold column (100 mm × 2.1 mm; 1.9µm, Thermo), with a mobile phase containing acetonitrile/methanol (1:1) (A) and 20 mM ammonium acetate (B). The detection and quantification MS instrument was a triple quadrupole 6410 (Agilent Technologies, Santa Clara, CA, USA), fitted with an electrospray ion source, operating in the negative ion mode. Analytical limits for infant specific food items ranged from 0.007 to 4 pg.g⁻¹ fw. For common food items, the median limit of detection ranged from 1.7 to 5 pg.g⁻¹ fw.

TBBPA was analyzed on a reversed-phase chromatography column (Gemini, 50 mm × 2 mm; 3µm, Phenomenex) with a mobile phase containing acetonitrile+0.1% acetic acid (A) and water+0.1% acetic acid (B). The detection and quantification was achieved on an Orbitrap system (Exactive, Thermo) operating in the negative electrospray mode and full scan acquisition. Analytical limits were highly dependent on the matrix and ranged from 0.1 to 3 ng.kg⁻¹ fw.

2.3.3. Internal quality controls

LABERCA operates an ISO/IEC 17025:2005-certified Quality Assurance system requiring strict controls with regard to personnel, instrument conditions, and experimental situations.

Blank samples and quality control were analyzed and monitored in each series of ten to twenty samples: the signal of each compound in the blanks was checked to avoid contamination throughout the analytical procedure, and concentrations of analytes in quality control were monitored to ensure repeatability and followed in charts. In addition, every year the laboratory takes part in an interlaboratory comparison tests and achieves satisfactory results for both of these methods.

2.4. Exposure calculation

Censored data (results below the limits of detection (LOD) and quantification (LOQ)) were processed according to a substitution method that involved framing the actual level using the lowest (low assumption or lower-bound (LB)) and highest (high assumption or upper-bound (UB)) values possible. The low assumption was calculated by assuming that all values below the LOD were equal to zero and those between the LOD and the LOQ were equal to the LOD. The high assumption was calculated by assuming that all values below the LOD were equal to the LOD and those between the LOD and the LOQ were equal to the LOQ.

Using data on individual consumption and levels of PFAAs and BFRs measured in food, exposure was calculated with the following equation:

$$E_i = \sum_{k=1}^n \frac{C_{i,k} \times L_k}{BW_i}$$

where:

- E_i is the total daily exposure of an individual i ($\mu\text{g.kg body weight}^{-1}.\text{day}^{-1}$),
- $C_{i,k}$ is the daily consumption of the food k by an individual i (g.day^{-1}),
- L_k is the estimated level for the studied contaminant in the food k ($\mu\text{g.g}^{-1}$ fresh weight),
- BW_i is the body weight of the individual i (kg),
- n is the total number of foods consumed by the individual i .

For common food items, concentrations data were completed with data from the second French TDS focusing on the general population (Rivière et al., 2014).

According to WHO recommendations (GEMS/Food-EURO. 2013), exposure data were estimated according to both the upper bound hypothesis (LB and UB, respectively). In the present article health risk was assessed considering the worst case scenario, i.e., considering the UB hypothesis.

Mean and 90th percentile (P90) of exposure were calculated for the population divided into four age groups: 1-4 months, 5-6 months, 7-12 months and 13-36 months. The mean exposure of the 10% of the most exposed children (over the P90) was also calculated. The percentage (and corresponding confidence interval at 95%, CI95%) of children exceeding the respective health based guidance value was calculated for each age group. Margins of safety were calculated (when necessary) as the ration between BMDLs and exposure (mean or P90). The ratio of the exposure through individual food items over the total exposure was calculated to determine the contribution (in %) of each food item to the total exposure. Food items contributing to more than 10% of the total exposure were identified as main contributors to the exposure.

2.5. Collective appraisal

The collective assessment of the risk linked to PFAA and BFR exposure has been conducted with the the French Agency for Food, Environmental and Occupational Health & Safety (ANSES) Expert Committee on food contaminants.

3. RESULTS AND DISCUSSION

3.1. Contamination data

3.1.1.PFAAs

Analyses covered 96% of the diet theoretically contributing to the total exposure. Due to the limited available toxicological data for PFAAs other than PFOS, PFOA, PFBS and PFHxA, only the results from these compounds

are reported in the present article, concentrations of all other PFAAs are available as supplementary materials. Excluding water, overall detection rates were very low (between 0 for PFDS and PFHpS) to 5.9% for PFTrDA. PFAAs were not detected in any of the infant specific food items, so results are only presented for common food items (Table 1). Highest concentrations of PFOA and PFOS were determined in crustaceans and mollusks: respectively, 0.014 ng.g⁻¹ fw and 0.312 ng.g⁻¹ fw (Rivière et al., 2014). The contamination data of common food items were those already reported (Rivière et al., 2014).

In 2012, the European Food Safety Authority (EFSA) reported PFAAs concentration in fish ranging from 0.0064 to 18.2 ng.g⁻¹ fw for PFOA and from 0.04 to 211 ng.g⁻¹ fw for PFOS (EFSA 2012). A Spanish study published in 2010 reported concentrations of PFOA and PFOS ranging from 0.17 to 0.72 ng.g⁻¹ fw and from 0.16 to 1.1 ng.g⁻¹ fw, respectively (Llorca et al. 2010). In both studies, detection rates were low (8% in the study reported by EFSA in 2012 for instance). The concentrations reported in our study are slightly lower than those previously reported.

3.1.2. BFRs

For PBDEs, overall analyses covered 96% of the contributing diet. Detection rates for the 7 PBDEs (BDE-28+BDE-47+BDE-99+BDE-100+BDE-153+BDE-154+BDE-183) ranged from 20% to 93% for BDE-183 and BDE-99, respectively. Highest mean concentrations were measured in dairy desserts and infant cereals for BDE-209 (respectively 69.57 and 66.94 pg.g⁻¹ fw considering the LB hypothesis) and in vegetables/meat or vegetable/fish ready to eat jars, infant cereals and dairy desserts for the sum of the 7 PBDEs (4.35 pg.g⁻¹ fw, 3.67 pg.g⁻¹ fw and 3.22 pg.g⁻¹ fw, respectively considering the LB hypothesis, Table 2). For common food items, highest BDE-209 mean concentrations were measured in sweet and savoury biscuits and bars (244 pg.g⁻¹ fw) and in margarine (105 pg.g⁻¹ fw). For the sum of the 7 PBDEs, highest mean concentrations were measured in fish (578 ng.g⁻¹ fw). The contamination data of common food items were those already reported (Rivière et al., 2014). In 2011, EFSA reported concentrations of PBDE in fish and seafood ten-fold higher than those reported in the present study (around 400 *versus* 40 pg.g⁻¹ fw; EFSA, 2011a). Similarly in infant food items, EFSA reported concentrations of BDE-209 of 130 pg.g⁻¹ fw *versus* 8 to 70 pg.g⁻¹ fw in the present study.

For HBCDDs, overall analyses covered 96% of the contributing diet. HBCDDs were detected in 82, 36 and 38% of the samples for isomers α , β and γ , respectively. Detection rates were very variable depending on the matrix analyzed (from 0 to 100%, in, respectively, cereals and dairy desserts and milk-based beverages). Measured concentrations in infant specific food items ranged from 2.46 to 42.85 pg.g⁻¹ fw (considering the sum of the three isomers and the UB hypothesis, Table 3). In one sample of infant formula, a concentration of 307 pg.g⁻¹ fw was measured whereas all other infant formula samples had concentrations ranging from 0.22 to 8.15 pg.g⁻¹ fw. A similar situation has been observed in the past where high concentrations of HBCDD were measured in different matrices (especially turkey and eggs, Anses 2012). However, except an occasional contamination, no relevant hypothesis could be drawn to explain this observation. For common food items, highest mean concentrations were measured in fish (between 177 pg.g⁻¹ fw and 185 pg.g⁻¹ fw depending on the hypothesis) and in delicatessen meat (between 140 pg.g⁻¹ fw and 150 pg.g⁻¹ fw, depending on the hypothesis). The contamination data of common food items were those already reported (Rivière et al., 2014). EFSA, in 2011, reported concentrations of HBCDD in infant food in the same order of magnitude (between 10 and 30 pg.g⁻¹ fw, EFSA 2011c) than those reported in the present study.

For PBBs, overall, analyses covered 96% of the contributing diet. The three congeners analyzed were detected in 4 to 12% of samples tested. In infant specific food items, measured concentrations were close to the limits of detection, with values ranging from 0.26 to 4.88 pg.g⁻¹ (considering the UB hypothesis, Table 4) for the sum of the three congeners. Highest PBB-52 concentrations were measured in an infant specific dairy based dessert containing fruits (3.28 pg.g⁻¹ fw) and in a ready to eat jar containing salmon, rice and vegetables (1.23 pg.g⁻¹

fw). For common food items, concentrations were slightly higher than those for infant specific food items with mean concentrations ranging from 0.19 to 20.4 $\mu\text{g}\cdot\text{g}^{-1}$ fw (considering the UB hypothesis). The contamination data of common food items were those already reported (Riviere et al., 2014). These results are consistent with the poor use of these compounds in Europe since their ban in 2000 (EFSA 2010). In 2010, EFSA reported concentrations of PBB in fish in the same order of magnitude than those reported here (between 1.86 and 18.9 $\text{ng}\cdot\text{kg}^{-1}$ fw depending on the congener, EFSA 2010).

Overall, analyses of TBBPA covered 94% of the contributing diet. TBBPA was detected in 30% of the samples. Highest mean concentrations were measured in croissant-like pastries (914 $\text{ng}\cdot\text{kg}^{-1}$ fw, Table 5) and infant and follow-on formulae (between 45 and 60 $\text{ng}\cdot\text{kg}^{-1}$ fw depending on the hypothesis), poultry (between 42 $\text{ng}\cdot\text{kg}^{-1}$ fw and 54 $\text{ng}\cdot\text{kg}^{-1}$ fw) and chocolate (between 32 and 62 $\text{ng}\cdot\text{kg}^{-1}$ fw).

3.2. Exposure data

3.2.1. PFAAs

Considering the UB hypothesis with regards to censored data, mean daily exposures to PFOA ranged from 1.49 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$ (90th percentile 1.77 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$) in the 1 to 4 months of age children to 2.95 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$ (90th percentile 5.16 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$) in the 1 to 3-year old children (Table 6). For PFOS, the mean exposures ranged from 1.61 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$ (90th percentile 3.56 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$) in the 5-6 months of age children to 2.66 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$ (90th percentile 4.63 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$) in the 1 to 3 years of age children. The mean exposure of the most exposed children (whose exposure were higher than the 90th percentile), ranged from 6.25 to 7.45 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$ for PFOA and from 5.54 to 6.37 for PFOS $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$. In 2012, EFSA reported exposures higher than those reported in the present study (EFSA, 2012). In newborns, reported exposures to PFOS and PFOA were 0.16 and 0.29 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$, respectively, whereas in young children these exposures ranged from 0.20 to 17 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$ for PFOA depending of the hypothesis with regards to censored data and from 0.58 to 14 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$ for PFOS. In France, in the last total diet study, exposures to PFOA and PFOS of the 3 to 6 years of age children were estimated at 2.45 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$ and 2.18 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$, respectively (Anses 2011), which is the same range as for 1-3 years-old children of the present study.

Due to the very limited number of food where PFBS and PFHxA were detected, no conclusions can be drawn with regard to the exposures to these compounds.

3.2.2. BFRs

Considering the UB hypothesis, mean daily exposure to BDE-209 ranged from 1.12 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$ to 2.62 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$, for the 1 to 3 years of age children and the 1 to 4 months of age children, respectively (Table 7). For the sum of the 7 PBDEs, mean exposures ranged from 0.448 to 0.926 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$ (Table 8). The 90th percentiles ranged from 1.88 to 3.91 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$ for BDE-209, whereas they ranged from 0.694 to 1.56 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$ for the sum of the 7 PBDEs depending on the age groups (4.30 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$). Two children had exposures of the 7 PBDEs much higher than the mean of their respective age groups. In both cases, these children consumed during the survey high quantities of salmon. Exposures of the 3 to 6 years of age children in France previously reported were in the same order of magnitude than those reported in the present study (ranging from 0.481 to 0.510 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$) whereas exposure to BDE-209 reported by EFSA were higher (ranging from 2.61 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$ to 6.02 $\text{ng}\cdot\text{kg}\ \text{bw}^{-1}\cdot\text{d}^{-1}$ depending on the hypothesis for censored data, EFSA 2011a). It is of interest to note that the ratio of exposures to BDE-209 to exposures to the sum of PBDEs is higher in the present study (around 3-fold) than in some previously reported studies (around 2-fold, Anses

2011, EFSA 2011a). This being possibly due to the increasing use of BDE-209 compared to that of the other congeners.

For the sum of HBCDDs and following the UB hypothesis, mean daily exposure ranged from 0.505 ng.kg bw⁻¹.d⁻¹ for the 1 to 3 years of age children to 8.27 ng.kg bw⁻¹.d⁻¹ for the 1 to 4 months of age children (Table 9). The 90th percentile ranged from 0.880 to 43.2 ng.kg bw⁻¹.d⁻¹ for the same age groups, the last value being due to the atypical contamination value measured in one sample of infant formula. For this reason the median values have also been reported in Table 9. Mean exposures of the 3 to 6 years of age children previously reported in the previous total diet study ranged from 0.35 to 0.49 ng.kg bw⁻¹.d⁻¹ (Anses 2011). EFSA, in 2011, reported similar values (between 0.36 and 1.27 ng.kg bw⁻¹.d⁻¹ for the sum of the three HBCDDs, EFSA 2011c).

Mean exposure to the sum of the three PBBs ranged from 0.049 ng.kg bw⁻¹.d⁻¹ in the 1 to 3 years of age children (90th percentile: 0.074 ng.kg bw⁻¹.d⁻¹) to 0.097 ng.kg bw⁻¹.d⁻¹ in the 1 to 4 months of age children (90th percentile: 0.204 ng.kg bw⁻¹.d⁻¹, Table 10). These exposure values were in the same order of magnitude than those reported for the 3 to 6 years of age children in the previously conducted total diet study (Anses, 2011).

For TBBPA, mean daily exposures, following the UB hypothesis, ranged from 0.968 to 9.94 ng.kg bw⁻¹.d⁻¹ for the 1 to 3 years of age group and the 1 to 4 months of age group, respectively (Table 11). One of the children from the 5 to 6 months of age group had exposure estimated at 77.1 ng.kg bw⁻¹.d⁻¹, i.e. 14 times higher than the mean exposure of that group of age. The reason for this lying in the fact that this child consumed a follow-on formula highly contaminated compared to other formulae. In 2006, the United-Kingdom committee on toxicity of chemicals in food reported exposures similar to those reported in the present study in 1.5 to 4.5 years of age children (between 7.0 and 4.6 ng.kg bw⁻¹.d⁻¹, COT 2006).

3.3. Risk assessment

3.3.1. PFAAs

Both PFOA and PFOS bind to plasma protein and are non-genotoxic, therefore allowing the setting of a health based guidance value based on a threshold effect (US-EPA, 2009; EFSA 2018). US-EPA in 2009 set a Reference Dose (RfD) at 0.08 µg.kg bw⁻¹.d⁻¹ for PFOS based on the effects observed on thyroid in a 182-day study in monkeys (Seacat *et al.*, 2002). For PFOA, the American environmental protection agency (US-EPA) in 2009 also established a reference dose for PFOA set at 0.2 µg.kg bw⁻¹.d⁻¹ based on a developmental study performed in mice (Lau *et al.*, 2006). On the other hand, the EFSA set provisional tolerable daily intakes for PFOA and PFOS at, respectively, 0.8 and 1.8 ng.kg bw⁻¹.d⁻¹ (EFSA, 2018). These health based guidance values were based on, the increase in serum total cholesterol in adults and a decrease in antibody response at vaccination in children for PFOS and on the increase in serum total cholesterol for PFOA.

Anses recently reported some health based guidance values for Perfluorobutanesulfonic acid (PFBS) and Perfluorohexanoic acid (PFHxA) (0.08 and 0.32 mg.kg bw⁻¹.d⁻¹, both based on renal toxicity; Anses, 2017a and 2017b).

With regard to PFOA and PFOS, based on the RfD set by the US EPA in 2009 for PFOA (0.2 µg.kg bw⁻¹.d⁻¹) and PFOS (0.08 µg.kg bw⁻¹.d⁻¹) the situation appears to not be of health concern (Table 6). However, in 2016, the US EPA reported lower RfD values, based on the result of pharmacokinetic modelling of serum values in animals in conjunction with the criteria of selected effects and their extrapolation to humans (respectively 20 and 30 µg.kg bw⁻¹.d⁻¹ for PFOA and PFOS, respectively, US-EPA, 2016a and 2016b). We observed that for PFOS, no

children included in the present study exceeded this RfD whereas for PFOA, only one child exceeded it. Considering the provisional health based guidance values set by EFSA in 2018 for PFOA and PFOS, high percentages of the 4 populations showed exceedances of the respective health based guidance values when considering the upper bound hypothesis (from 20% to almost 100% of the populations, Table 6).

For PFBS and PFHxA, mean and 90th percentile exposures were much lower than the respective health based guidance values set by Anses (Anses, 2017a and 2017b). For the other PFAAs, given the absence of data that would enable a toxicological point of departure to be established, it was not possible to reach any conclusions as to the health risk associated with dietary exposure to these substances.

3.3.1.BFRs

None of the compounds analyzed in the present study are genotoxic, therefore allowing the setting of health based guidance values based on threshold effects (EFSA 2011). These compounds are lipophilic and accumulate in the adipose tissues.

Regarding the 209 congeners of PBDEs, their chemical structure being similar to that of non-dioxin-like polychlorinated biphenyls (NDL-PCBs) and due to their common mechanism of action (Pellacani *et al.*, 2012; Miller *et al.*, 2012; Kodavanti *et al.*, 2005) and pending the definition of a health-based guidance value for PBDEs, the French Agency for Food, Environmental and Occupational Health & Safety (ANSES) Expert Committee on food contaminants compared exposure to the following congeners: BDE-28,-47, -99, -100, -153, -154, -183 with the threshold of 10 ng/kg bw/day set for the six NDL-PCBs that are the most frequently found in food (AFSSA, 2007, Rivière *et al.*, 2014). The European Food Safety Authority (EFSA) also set benchmark dose lower confidence limits (BMDLs) for a benchmark response of 10% (BMDL₁₀) for the four most “of concern” PBDEs based on their neurodevelopmental effects: 172 ng.kg bw⁻¹ for BDE-47, 4.2 ng.kg bw⁻¹ for BDE-99, 9.6 ng.kg bw⁻¹ for BDE-153 and 1,700 µg.kg bw⁻¹ for BDE-209 (EFSA, 2011a). Regarding HBCDDs, EFSA identified neurodevelopmental effects on behavior as the critical endpoint, and derived a BMDL₁₀ of 0.79 mg.kg bw⁻¹. Due to the limitations and uncertainties in current knowledge, EFSA concluded that it was inappropriate to use this BMDL to establish a health-based guidance value, and instead recommended using the margin of safety (MOS) approach for the risk characterisation of HBCDDs by comparing dietary intake with the estimated dietary human intake associated with the body burden at the BMDL₁₀, set at 0.003 ng.kg bw⁻¹.d⁻¹. In its opinion on PBBs, EFSA could not define a health-based guidance value, but suggested comparing data on exposure to PBBs with a no observed adverse effect level (NOAEL) of 0.15 mg/kg bw/day based on the induction of hepatic carcinomas in rats (EFSA, 2010a). With regards to TBBPA, the toxicology point of departure (BMDL₁₀) was established in rat reprotoxicity study and based on the effects on the homeostasis of thyroid hormones (decrease of the circulating levels of hormone T4 in the first generation). It was set at 16 mg.kg bw⁻¹.d⁻¹ (EFSA 2011b). Due to its specific characteristics in terms of pharmacokinetics and toxicity, BDE-209 was considered separately from the other seven congeners, whose chemical structure and mechanism of action are similar to those of NDL-PCBs.

For the six NDL-PCBs the threshold value (10 ng/kg bw/day) was not observed to have been exceeded (Table 8). The margins of safety calculated for BDE-209 (with regard to the BMDL₁₀ of 1 700 µg.kg bw⁻¹.d⁻¹) ranging from 430,000 to 1,5 million, are much larger than the 2.5 margin deeming tolerable by EFSA (Table 7). For PBDEs, the situation is therefore deemed to be tolerable considering these health-based guidance values.

Regardless of the age group, the margins of safety calculated with regard to the BMDL₁₀ established by EFSA for the sum of the three HBCDDs (3 000 ng.kg bw⁻¹.d⁻¹) ranged 70 for the 1-4 months using the 90th percentile of exposure to 5940 using the median of exposure of the 13-36 months (Table 9). They were higher than the critical margin of safety value set at 25 to consider the exposure as being of no health concern.

The margins of safety calculated using the no adverse effect level observed by EFSA for the sum of the three PBBs ($0.15 \text{ mg.kg bw}^{-1}.\text{d}^{-1}$) ranged 740,000 to more than 3,000,000, depending on the age group and the exposure value (mean or P90) (Table 10). They are then considered as high enough to conclude that the situation is of no health concern.

For TBBPA, the margins of safety calculated using the BMDL_{10} established by EFSA ($16 \text{ mg.kg bw}^{-1}.\text{d}^{-1}$), ranging 511,000 to more than 16 million (Table 11), are also large enough (higher than 1 000 considered by EFSA as the critical margin of exposure) to conclude that the situation is of no health concern.

Overall, the risk related to the dietary exposures to these compounds appeared not to be of concern, for infants, similarly to the general population older than 3 years old (Rivière et al., 2014). However, it seems of importance to note that the present analysis focused on general infant population and then does not take into account some specific situations such as accidental contaminations. Moreover, the present study does not take into account breastfed infants where lipophilic compounds are known to accumulate (Colles et al., 2008).

3.4. Uncertainties

Some uncertainties are important to mention in parallel to the presented results. Indeed, risk analysis processes suppose some hypotheses that can lead to over or underestimation of the risk. The main identified uncertainties are the following. First, the available data to set health based guidance values. For many perfluoroalkyl acids and brominated flame retardants, there is, as of today, not enough robust toxicological data to establish robust health based guidance values. Additionally, considering the population of interest of this study, one has to mention that some toxicological endpoints specific to this population could exist due to the development of the organisms. Efforts have been made to consider values that should be protective to the infant population, however, experimental data to come could lead to lower health based guidance values than the one considered in the present study, as it was the case for PFOS and PFOA with decreasing health-based guidance values with time. Moreover, synergistic or antagonistic effects could occur due to the exposure of substances from the same chemical family.

Some uncertainties are due to the consumption data used to perform exposure calculations. Since only main practices are considered in the present study, specific dietary habits that could lead to different exposure patterns are not observed.

Finally, analytical methodology could lead to uncertainties due to the precision of the method but also due to the limits of detection and quantification that can be achieved. This, indeed, has some impact on the exposure calculation as seen above for PFOS and PFOA. In the case of a limit of detection far above the concentrations measured in the food items, the upper bound hypothesis would lead to a significant overestimation of the exposure.

3.5. Conclusion

The results presented above allow to assess the risk related to the dietary exposure of some PFAAs and of some BFRs. For the compounds analyzed, dietary exposures appear to be of no health concern in the 0 to 3 years of age children with regard to the selected health based guidance values. However, considering the newly established RfD for PFOS and PFOA by US-EPA, for some children with specific diets the risk can not be excluded. This analysis also reveals that for some congeners (especially for some PFAAs), no health based guidance values are as of today available due to the paucity of robust toxicological data, preventing to perform a risk assessment. It is also of importance to consider that new toxicological data becoming available, health based guidance values could be lowered, especially for compounds for which some endocrine disruptor effects have been reported but not characterized enough as of today to set a health based guidance value.

4. ACKNOWLEDGMENTS

The authors are grateful to the Anses expert committee panel in charge of assessing the chemical risk in food and the experts from the infant TDS scientific committee, namely: Jean-Pierre Cravédi, Claude Atgié, Pierre-Marie Badot, Jacques Bélégaud, Catherine Bennetau-Pelissero, Valérie Camel, Guillaume Duflos, Camille Dumas, Jérôme Gay-Queheillard, Philippe Glorennec, Thierry Guérin, Laurence Guldner, Laïla Lakhal, Claude Lambré, Raphaëlle Le Garrec, Catherine Leclerc, Eric Marchioni, César Mattéi, André Mazur, Sakina Mhaouty-Kodja, Fabrice Nesslany, Alain-Claude Roudot, Patrick Sauvegrain, Rémy Slama, Karine Tack, Eric Verdon and Jean-Paul Vernoux.

The infant TDS was supported by the Ministry for food, agriculture and fisheries, the Ministry for health the Ministry for ecology and sustainable development and the French Agency for Food, Environmental and Occupational Health & Safety (ANSES).

5. REFERENCES

AFSSA (2007). Avis de l'Agence française de sécurité sanitaire des aliments relatif à l'établissement de teneurs maximales pertinentes en polychlorobiphényles qui ne sont pas de type dioxine (PCB « non dioxin-like », PCBNDL) dans divers aliments. AFSSA, Maisons-Alfort, France.

Anses (2011). Avis de l'Anses et rapport d'expertise relatifs à l'étude de l'Alimentation Française 2 (EAT2) - Tome 1 : Contaminants inorganiques, minéraux, polluants organiques persistants, mycotoxines, phytoestrogènes. Maisons-Alfort: Anses. 305 p.

Anses (2012). Avis de l'Anses relatif aux analyses de retardateurs de flamme bromés (RFB) à mettre en œuvre dans le cadre des prochains plans de surveillance. (saisine n°2010-SA-0225). Maisons Alfort: Anses. 12 p.

Anses (2017a) Elaboration de VTR chronique par voie orale pour l'acide per-fluorohexanoïque (PFHxA). available at www.anses.fr

Anses (2017b) Elaboration de VTR chronique par voie orale pour l'acide perfluorobutane sulfonique (PFBS). available at www.anses.fr

Bartell SM, Calafat AM, Lyu C, Kato K, Ryan PB and Steenland K (2010) Rate of decline in serum PFOA concentrations after granular activated carbon filtration at two public water systems in Ohio and West Virginia. *Environ Health Perspect* 118:222-8. doi: 10.1289/ehp.0901252.

Blum A, Balan SA, Scheringer M, Trier X, Goldenman G, Cousins IT ... Weber R (2015). The Madrid statement on poly- and perfluoroalkyl substances (PFASs). *Environmental Health Perspectives*, 123: A107-A111. DOI: 10.1289/ehp.1509934

Colles A, Koppen G, Hanot V, Nelen V, Dewolf MC, Noël E, Malisch R, Kotz A, Kypke K, Biot P, Vinkx C and Schoeters G (2008) Fourth WHO-coordinated survey of human milk for persistent organic pollutants (POPs): Belgian results, *Chemosphere*, Volume 73: 907-914, doi: 10.1016/j.chemosphere.2008.07.002.

Cornelis C, D'Hollander W, Roosens L, Covaci A, Smolders R, Van Den Heuvel R, Govarts E, Van Campenhout K, Reynders L, Bervoets L (2012). First assessment of population exposure to perfluorinated compounds in Flanders, Belgium. *Chemosphere*, 86: 308-314.

COT (2006) Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment. Brominated Chemicals: UK dietary intake. COT statement 10/06.

Diamanti Kandarakis E, Bourguignon JP, Giudice LC, Hauser R, Prins GS, Soto AM, Zoeller RT, Gore AC. 2009. Endocrine-disrupting chemicals: an endocrine society scientific statement. *Endocr Rev.* 30:293–342.

- Domingo J (2012). Polybrominated diphenyl ethers in food and human dietary exposure: a review of the recent scientific literature. *Food Chem Toxicol.* 50: 238-49
- EFSA (2008) Scientific Opinion of the Panel on Contaminants in the Food chain. Perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA) and their salts (Question No EFSA-Q-2004-163). EFSA, Parma, Italy.
- EFSA (2010a) Scientific Opinion on Polybrominated Biphenyls (PBBs) in Food. EFSA, Parma, Italy.
- EFSA (2010) Scientific Opinion of the EFSA Panel on Contaminants in the Food Chain on Polybrominated Biphenyls (PBBs) in Food. In *The EFSA journal*, N°8 (10). Parma: EFSA.
- EFSA (2011a) Scientific Opinion on Polybrominated Diphenyl Ethers (PBDEs) in Food (2011). EFSA Panel on Contaminants in the Food Chain (CONTAM). European Food Safety Authority (EFSA), Parma, Italy
- EFSA (2011b). Scientific Opinion of the EFSA Panel on Contaminants in the Food Chain on Tetrabromobisphenol A (TBBPA) and its derivatives in food. In *The EFSA journal*, N°9 (12). Parma: EFSA.
- EFSA (2011c). Scientific Opinion of the EFSA Panel on Contaminants in the Food Chain on Hexabromocyclododecanes (HBCDDs) in Food. In *The EFSA journal*, N°9 (7). Parma: EFSA.
- EFSA (2012). Scientific report of EFSA on Perfluoroalkylated substances in food: occurrence and dietary exposure. In *The EFSA journal*, N°10 (6). Parma: EFSA.
- Fantino M. 2005. Etude SFAE sur la consommation alimentaire des nourrissons et enfants en bas âge français de 1 mois à 36 mois – Analyse des données nutritionnelles. Not published, Etude SOFRES 2005/Université de Bourgogne–Pr M. Fantino for the French Association for Children’s Food.
- Fantino M, Gourmet E. 2008. Apports nutritionnels en France en 2005 chez les enfants non allaités âgés de moins de 36 mois. *Archives de Pédiatrie.* 15:446–455.
- Frederiksen M, Vorkamp K, Thomsen M, Knudsen LE (2009). Human internal and external exposure to PBDEs—a review of levels and sources. *Int J Hyg Environ Health.* 212: 109–34.
- GEMS/Food-EURO. 2013. Addendum 2013 - Second Workshop on Reliable Evaluation of Low-Level Contamination of Food. Workshop in the frame of GEMS/Food-EURO. Technical report. Kulmbach, Germany: WHO.
- Grandjean P, Andersen EW, Budtz-Jørgensen E, Nielsen F, Mølbak KR, Weihe P, Heilmann C (2012). Serum vaccine antibody concentrations in children exposed to perfluorinated compounds. *JAMA – J. Am. Med. Ass.:* 307: 391-97
- Hulin M, Bemrah N, Nougadère A, Volatier JL, Sirot V and Leblanc JC (2014) Assessment of infant exposure to food chemicals: the French Total Diet Study design, *Food Additives & Contaminants* 31: 1226-1239, DOI: 10.1080/19440049.2014.921937
- Johansson N, Fredriksson A, Eriksson P (2008) Neonatal exposure to perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) causes neurobehavioural defects in adult mice, *NeuroToxicology*, 29: 160-169. <http://dx.doi.org/10.1016/j.neuro.2007.10.008>.
- Kadar H, Veyrand B, Barbarossa A, Pagliuca G, Legrand A, Boshier C, Boquien CY, Durand S, Monteau F, Antignac JP, Le Bizec B (2011). Development of an analytical strategy based on liquid chromatography–high resolution mass spectrometry for measuring perfluorinated compounds in human breast milk: Application to the generation of preliminary data regarding perinatal exposure in France. *Chemosphere.* 85:473-80
- Kang JS, Choi JS and Park JW (2016) Transcriptional changes in steroidogenesis by perfluoroalkyl acids (PFOA and PFOS) regulate the synthesis of sex hormones in H295R cells, *Chemosphere*, 155: 436-443, <http://dx.doi.org/10.1016/j.chemosphere.2016.04.070>.
- Kodavanti PR, Ward TR, Ludewig G, Robertson LW, Birnbaum LS (2005). Polybrominated diphenyl ether (PBDE) effects in rat neuronal cultures: 14C-PBDE accumulation, biological effects, and structure-activity relationships. *Toxicol Sci.* 88 : 181-92.

- Kowalczyk J Ehlers S, Furst P, Schafft H, Lahrssen-Wiederholt M (2012). Transfer of Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS) From Contaminated Feed Into Milk and Meat of Sheep: Pilot Study. *Arch Environ Contam Toxicol*. 63: 288-298.
- Landrigan PJ, Kimmel CA, Correa A, Eskenazi B. 2003. Children's health and the environment: public health issues and challenges for risk assessment. *Environ Health Perspect*. 112:257-265.
- Lau C, Thibodeaux JR, Hanson RG, Rogers JM, Grey BE, Stanton ME, Butenhoff JL and Stevenson LA. (2003). Exposure to perfluorooctane sulfonate during pregnancy in rat and mouse. II: postnatal evaluation. *Toxicol Sci* 74:382-92. doi: 10.1093/toxsci/kfg122.
- Le Bot B, Lucas JP, Lacroix F and Glorennec P (2013). Population-based metals concentrations in tap water consumed by young children in France. Available at: <http://dx.doi.org/10.1289/ehp.ehbasel13>, Basel, Switzerland, August 19-23, 2013.
- Llorca M, Farre M, Pico Y, Teijon ML, Alvarez JG and Barcelo D (2010). Infant exposure of perfluorinated compounds: levels in breast milk and commercial baby food. *Environ Int* 36:584-92. doi: 10.1016/j.envint.2010.04.016.
- Makri A, Goveia M, Balbus J, Parkin R. 2004. Children's susceptibility to chemicals: a review by developmental stage. *J Toxicol Environ Health, Part B*. 7:417-435.
- Miller VM, Sanchez-Morrissey S, Brosch KO, Seegal RF (2012). Developmental coexposure to polychlorinated biphenyls and polybrominated diphenyl ethers has additive effects on circulating thyroxine levels in rats. *Toxicol Sci*. 127:76-83.
- Olsen GW, Burriss JM, Ehresman DJ, *et al.* (2007). Half-Life of Serum Elimination of Perfluorooctanesulfonate, Perfluorohexanesulfonate, and Perfluorooctanoate in Retired Fluorochemical Production Workers. *Environ Health Perspect*. 115:1298-305.
- Pellacani C, Tagliaferri S, Caglieri A, Goldoni M, Giordano G, Mutti A, Costa LG (2012) Synergistic interactions between PBDEs and PCBs in human neuroblastoma cells. *Environ Toxicol*. 2012 Mar 20. doi: 10.1002/tox.21768.
- Rivière, G, Sirot, V, Tard A, Jean J, Marchand P, Veyrand B, *et al.*, 2014. Food risk assessment for perfluoroalkyl acids and brominated flame retardants in the French population: results from the second French total diet study. *Sci. Total Environ*. 491: 176-183 <http://dx.doi.org/10.1016/j.scitotenv.2014.01.104>.
- Seacat AM, Thomford PJ, Hansen KJ, Olsen GW, Case MT, Butenhoff JL (2002). Subchronic toxicity studies on perfluorooctanesulfonate potassium salt in cynomolgus monkeys. *Toxicol Sci*. 68: 249-264.
- Seacat AM, Thomford PJ, Hansen KJ, Clemen LA, Eldridge SR, Elcombe CR, Butenhoff JL (2003). Sub-chronic dietary toxicity of potassium perfluorooctanesulfonate in rats. *Toxicology* 183: 117-131.
- Sly PD, Flack F. 2008. Susceptibility of children to environmental pollutants. *Ann N Y Acad Sci*. 1140:163-183.
- Trudel D, Horowitz L, Wormuth M, Scheringer M, Cousins IT, Hungerbühler K (2008). Estimating Consumer Exposure to PFOS and PFOA. *Risk Anal*. 28: 251-269.
- US-EPA (2009). The toxicity of Perfluorooctanoic Acid (PFOA) and Perfluorooctane sulfonate (PFOS). Washington DC, USA: US EPA.
- US-EPA (2016a) Drinking water health advisory for perfluorooctanoic acid (PFOA). Washington DC, USA: US EPA.
- US-EPA (2016b) Drinking water health advisory for perfluorooctanoic acid (PFOA). Washington DC, USA: US EPA.

Table 1 Mean concentrations (ng.kg⁻¹ fw) of PFOA and PFOS in common and infant specific food items

Food category	N	PFBS						PFHxA				PFOA				PFOS					
		LB		UB		a	Mean ±SD	(Min- Max)	Mean ±SD	(Min- Max)	a	Mean ±SD	(Min- Max)	Mean ±SD	(Min- Max)	a	Mean ±SD	(Min- Max)	Mean ±SD	(Min- Max)	
		0	0±0	(0-0)	9.19±0																(9.19- 9.19)
Common food																					
Non-alcoholic beverages	1	0	0±0	(0-0)	9.19±0	(9.19- 9.19)	0	0±0	(0-0)	9.19 ±0	(9.19- 9.19)	0	0±0	(0-0)	13.78 ±0	(13.78- 13.78)	0	0±0	(0-0)	9.18 ±0	(9.18- 9.18)
Water	12	0	0±0	(0-0)	0.462±0.006	(0.454- 0.474)	0	0±0	(0-0)	0.462 ±0.006	(0.454- 0.474)	0	0±0	(0-0)	0.462 ±0.0061	(0.454- 0.474)	0	0±0	(0-0)	1.08 ±0.53	(0.91- 2.77)
Dairy-based desserts	1	0	0±0	(0-0)	27.55±0	(27.55- 27.55)	0	0±0	(0-0)	27.55 ±0	(27.55- 27.55)	0	0±0	(0-0)	41.33 ±0	(41.33- 41.33)	0	0±0	(0-0)	27.55 ±0	(27.55- 27.55)
Milk	1	0	0±0	(0-0)	4.54±0	(4.54- 4.54)	0	0±0	(0-0)	4.54 ±0	(4.54- 4.54)	0	0±0	(0-0)	4.54 ±0	(4.54- 4.54)	0	0±0	(0-0)	45.40 ±0	(4.54- 4.54)
Vegetables (excluding potatoes)	1	0	0±0	(0-0)	12.35±0	(12.35- 12.35)	0	0±0	(0-0)	12.35 ±0	(12.35- 12.35)	0	0±0	(0-0)	18.53 ±0	(18.53- 18.53)	0	0±0	(0-0)	12.35 ±0	(12.35- 12.35)
Mixed dishes	1	0	0±0	(0-0)	42.66±0	(42.66- 42.66)	0	0±0	(0-0)	42.66 ±0	(42.66- 42.66)	0	0±0	(0-0)	63.99 ±0	(63.99- 63.99)	0	0±0	(0-0)	42.66 ±0	(42.66- 42.66)
Fish	1	0	0±0	(0-0)	25.91±0	(25.91- 25.91)	0	0±0	(0-0)	25.91 ±0	(25.91- 25.91)	100	30.14 ±0	(0.030139- 0.030139)	30.14 ±0	(30.14- 30.14)	100	277.4 ±0	(277.4- 277.4)	277.4 ±0	(277.4- 277.4)
Ultra-fresh dairy products	1	0	0±0	(0-0)	21.76±0	(21.76- 21.76)	0	0±0	(0-0)	21.76 ±0	(21.76- 21.76)	0	0±0	(0-0)	32.64 ±0	(32.64- 32.64)	0	0±0	(0-0)	21.76 ±0	(21.76- 21.76)
Meat	1	0	0±0	(0-0)	37.97±0	(37.97- 37.97)	0	0±0	(0-0)	37.97 ±0	(37.97- 37.97)	0	0±0	(0-0)	56.95 ±0	(56.95- 56.95)	0	0±0	(0-0)	37.97 ±0	(37.97- 37.97)
Poultry and game	2	0	0±0	(0-0)	37.22±1.59	(36.09- 38.34)	0	0±0	(0-0)	37.22 ±1.59	(36.09- 38.34)	0	0±0	(0-0)	55.82 ±2.38	(54.14- 57.51)	0	0±0	(0-0)	37.22 ±1.59	(36.09- 38.34)
Infant food																					
Milk-based beverage	8	0	0±0	(0-0)	16.22±2.92	(12.53- 19.34)	0	0±0	(0-0)	16.22 ±2.92	(12.53- 19.34)	0	0±0	(0-0)	32.24 ±5.84	(25.05- 38.69)	0	0±0	(0-0)	16.22 ±2.92	(12.53- 19.34)
Cereals-based food	5	0	0±0	(0-0)	36.93±0.17	(36.66- 37.11)	0	0±0	(0-0)	36.93 ±0.17	(36.66- 37.11)	0	0±0	(0-0)	36.93 ±0.17	(36.66- 37.11)	0	0±0	(0-0)	18.46 ±0.084	(18.33- 18.56)

Milk-based dessert	6	0	0±0	(0-0)	20.66±1.49	(13.35-23.29)	0	0±0	(0-0)	20.66±1.49	(13.35-23.29)	0	0±0	(0-0)	41.31±2.97	(38.70-46.57)	0	0±0	(0-0)	20.66±1.49	(19.35-23.29)
Growth milk	9	0	0±0	(0-0)	4.57±0.041	(4.49-4.63)	0	0±0	(0-0)	4.57±0.041	(4.49-4.63)	0	0±0	(0-0)	4.57±0.041	(4.49-4.63)	0	0±0	(0-0)	4.57±0.041	(4.49-4.63)
Soup, purée	11	0	0±0	(0-0)	11.30±2.72	(5.85-14.02)	0	0±0	(0-0)	11.30±2.72	(5.85-14.02)	0	0±0	(0-0)	16.96±4.07	(8.78-21.02)	0	0±0	(0-0)	11.30±2.72	(5.85-14.02)
Fruit purée	4	0	0±0	(0-0)	17.28±2.13	(14.80-19.43)	0	0±0	(0-0)	17.28±2.13	(14.80-19.43)	0	0±0	(0-0)	34.56±4.25	(29.61-38.87)	0	0±0	(0-0)	12.92±5.23	(8.14-19.43)
Pots légumes	27	0	0±0	(0-0)	13.11±3.63	(7.02-21.67)	0	0±0	(0-0)	13.11±3.63	(7.02-21.67)	0	0±0	(0-0)	26.22±7.26	(14.03-43.33)	0	0±0	(0-0)	12.95±8.42	(3.51-28.09)
Vegetable-based ready-to-eat meal	45	0	0±0	(0-0)	19.36±6.69	(11.97-32.04)	0	0±0	(0-0)	19.36±6.69	(11.97-32.04)	0	0±0	(0-0)	26.98±2.68	(22.13-34.85)	0	0±0	(0-0)	22.58±7.18	(11.97-34.85)
Infant formula	28	0	0±0	(0-0)	9.20±2.14	(4.57-18.17)	0	0±0	(0-0)	9.20±2.14	(4.57-18.17)	0	0±0	(0-0)	6.25±3.12	(4.53-18.17)	0	0±0	(0-0)	8.03±2.92	(4.53-18.17)
Follow-on formula	33	0	0±0	(0-0)	8.40±4.60	(4.50-18.50)	0	0±0	(0-0)	8.40±4.60	(4.50-18.50)	0	0±0	(0-0)	7.70±4.77	(4.50-18.50)	0	0±0	(0-0)	6.59±4.84	(4.50-18.50)

N : number of composite samples analysed

LB : Lower bound hypothesis ; UB :Upper bound hypothesis

a: %age of detection

Table 2 Mean concentrations ($\mu\text{g}\cdot\text{g}^{-1}$ fw) of PBDEs in food

Food item	N	BDE-209			Sum of the 7 PBDE		
		% detection	LB	UB	% detection	LB	UB
Infant specific food							
Milk-based beverage	8	75	18.18	21.78	21	0.95	3.96
Cereals	5	60	66.94	90.41	43	3.67	11.64
Milk-based desserts	6	100	69.57	69.57	45	3.22	5.74
Growing-up milk	9	44	5.64	10.11	33	1.27	2.49
Soups and puree	11	73	16.58	19.35	40	2.43	4.43
Fruit puree	4	75	32.86	35.52	11	0.56	4.81
Vegetable-based ready-to-eat meal	20	60	17.61	22.30	19	1.02	3.61
Meat/fish-based ready-to-eat meal	45	64	16.94	20.77	36	4.35	6.64
Infant formula	28	61	11.37	15.43	38	2.33	4.38
Follow-on formula	33	73	18.38	21.78	33	2.19	4.45
Common food							
Offal	16	69	48.4	55.7	62	12.2	17
Butter	4	75	19.6	38.8	71	56.5	62.6
Non-alcoholic beverages	1	100	9.68	9.68	43	0.828	1.34
Delicatessen	56	98	84.2	84.3	88	58.1	61.1
Seasonings and sauces	1	0	0	28.2	29	10.8	32.5
Crustaceans and mollusks	21	57	20.5	23.2	85	82.2	84.3
Dairy-based desserts	13	92	244	272	57	16.9	19.9
Cheese	10	100	43.9	43.9	57	23.6	29.5
Oil	2	0	0	14.7	29	10.1	39.5
Milk	1	0	0	8.93	29	0.655	2.49

Vegetables (excluding potatoes)	7	100	69.9	69.9	86	26.5	27.8
Margarine	4	50	105	114	57	39.1	51.1
Eggs and derivatives	16	50	43	52.9	71	22.3	25.1
Pizzas, quiches and savoury pastries	4	100	68.5	68.5	64	16.6	19.5
Mixed dishes	13	92	38.1	50.6	76	11.8	15.9
Fish	25	80	38.6	43.3	90	578	579
Ultra-fresh dairy products	59	88	35.3	36.7	69	8.33	10.1
Meat	33	88	45.4	57.7	84	26	27.9
Poultry and game	7	100	38.6	38.6	57	17.7	22.1

N : number of composite samples analysed

LB : Lower bound hypothesis ; UB :Upper bound hypothesis

Table 3 Mean concentrations ($\mu\text{g}\cdot\text{g}^{-1}$ fw) of HBCDDs in food

Food item	N	HBCDD α			HBCDD β			HBCDD γ			Sum of the HBCDDs		
		% detection	LB	UB	% detection	LB	UB	% detection	LB	UB	% detection	LB	UB
Infant specific food													
Milk-based beverage	8	100	14.27	14.27	88	3.36	3.39	88	5.14	5.31	92	22.77	22.97
Cereals	5	40	11.04	12.31	0	0.00	0.44	0	0.00	1.85	13	11.04	14.59
Milk-based desserts	6	100	26.81	26.81	83	6.94	7.03	83	8.87	9.02	89	42.61	42.85
Growing-up milk	9	89	3.56	3.63	67	0.80	0.91	67	0.96	1.21	74	5.32	5.74
Soups and puree	11	18	0.96	1.48	9	0.25	0.38	9	0.27	0.61	12	1.49	2.46
Fruit puree	4	75	4.28	4.55	25	0.48	1.01	50	1.24	1.84	50	5.99	7.40
Vegetable-based ready-to-eat meal	20	70	5.16	5.67	50	1.04	1.27	45	1.35	1.71	55	7.54	8.65
Meat/fish-based ready-to-eat meal	45	76	8.90	9.16	49	1.75	1.87	44	2.25	2.54	56	12.89	13.57
Infant formula	28	61	8.41	8.74	18	1.95	2.20	18	2.46	2.89	32	12.81	13.83
Follow-on formula	33	33	2.45	3.13	12	0.48	0.72	12	0.67	1.03	19	3.60	4.88

Food item	N	HBCDD α			HBCDD β			HBCDD γ			Sum of the HBCDDs		
		% detection	LB	UB	% detection	LB	UB	% detection	LB	UB	% detection	LB	UB
Common food													
Offal	16	62.5	20.3	23.2	31.3	0.372	1.71	25	4.44	10.7	40	25.1	35.6
Butter	4	75	4.99	8.72	0	0	8.4	25	1.25	16.6	33	6.24	33.7
Non-alcoholic beverages	1	100	7.98	7.98	100	2.07	2.07	100	2.49	2.49	100	12.5	12.5
Delicatessen	56	100	135	135	53.6	2.12	3.91	57.1	3.17	11.5	70	140	150
Seasonings and sauces	1	100	2.27	2.27	0	0	14.1	0	0	44.3	33	2.27	60.7
Crustaceans and mollusks	21	76.2	43.4	44.4	23.8	3.6	4.82	19	5.67	9.49	40	52.7	58.7
Dairy-based desserts	13	61.5	3.63	5.23	38.5	0.676	1.31	15.4	0.429	3.95	38	4.74	10.5
Cheese	10	100	8.08	8.08	30	0.387	2.59	0	0	12.6	43	8.47	23.2
Oil	2	0	0	23.5	0	0	15	0	0	47.4	0	0	85.8
Milk	1	100	1.59	1.59	0	0	0.059	0	0	0.122	33	1.59	1.77
Vegetables (excluding potatoes)	7	100	58.5	58.5	71.4	0.974	1.32	71.4	2.76	4.17	81	62.3	64
Margarine	4	25	1.5	9.37	0	0	6.5	0	0	18.8	8	1.5	34.6
Eggs and derivatives	16	100	23.1	23.1	18.8	0.085	1.43	0	0	5.75	40	23.1	30.2
Pizzas, quiches and savoury pastries	4	100	22.5	22.5	50	0.397	1.3	0	0	6.9	50	22.9	30.7
Mixed dishes	13	100	27.5	27.5	46.2	3.16	3.82	23.1	3.89	6.74	56	34.5	38
Fish	25	100	163	163	48	5.21	10.1	68	9.38	11.7	72	177	185
Ultra-fresh dairy products	59	67.8	3.95	5.13	15.3	0.557	2.07	25.4	0.901	3.57	36	5.41	10.8
Meat	33	97	13.6	14.9	30.3	0.411	1.72	15.2	0.788	4.24	47	14.8	20.9
Poultry and game	7	100	76.1	76.1	42.9	0.235	1.2	14.3	0.348	5.54	52	76.7	82.9

N : number of composite samples analysed

LB : Lower bound hypothesis ; UB :Upper bound hypothesis

Table 4 Mean concentrations ($\mu\text{g}\cdot\text{g}^{-1}$ fw) of PBBs in food

Food item	N	BB 52	BB 101	BB 153	Somme des PBB
-----------	---	-------	--------	--------	---------------

		% detection	LB	UB	% detection	LB	UB	% detection	LB	UB	% detection	LB	UB
Infant specific food													
Milk-based beverage	8	0	0.00	0.13	0	0.00	0.18	0	0.00	0.15	0	0.00	0.46
Cereals	5	0	0.00	0.67	0	0.00	0.78	0	0.00	0.77	0	0.00	2.22
Milk-based desserts	6	17	0.08	0.40	0	0.00	0.38	0	0.00	0.31	6	0.08	1.09
Growing-up milk	9	0	0.00	0.09	0	0.00	0.14	0	0.00	0.16	0	0.00	0.39
Soups and puree	11	0	0.00	0.06	0	0.00	0.10	0	0.00	0.10	0	0.00	0.26
Fruit puree	4	25	0.82	2.06	0	0.00	1.29	0	0.00	1.54	8	0.82	4.88
Vegetable-based ready-to-eat meal	20	5	0.02	0.37	0	0.00	0.40	0	0.00	0.40	2	0.02	1.17
Meat/fish-based ready-to-eat meal	45	4	0.03	0.17	2	0.01	0.20	0	0.00	0.16	2	0.04	0.53
Infant formula	28	14	0.07	0.20	0	0.00	0.24	0	0.00	0.20	5	0.07	0.63
Follow-on formula	33	0	0.00	0.23	0	0.00	0.24	0	0.00	0.20	0	0.00	0.68
Common food													
Offal	16	0	0	0.406	0	0	1.21	81.3	1.46	1.99	27	1.46	3.61
Butter	4	0	0	1.05	0	0	1.78	0	0	4	0	0	6.83
Non-alcoholic beverages	1	0	0	0.047	0	0	0.045	0	0	0.1	0	0	0.192
Delicatessen	56	0	0	0.41	0	0	1.29	17.9	0.132	1.72	6	0.132	3.41
Seasonings and sauces	1	0	0	3	0	0	7.2	0	0	6.3	0	0	16.5
Crustaceans and mollusks	21	47.6	0.373	0.581	0	0	0.861	9.5	0.117	1.15	19	0.491	2.6
Dairy-based desserts	13	0	0	0.483	0	0	0.72	0	0	0.996	0	0	2.2
Cheese	10	0	0	0.55	0	0	1.85	10	0.126	2.72	3	0.126	5.12
Oil	2	0	0	4.65	0	0	8.6	0	0	7.1	0	0	20.4
Milk	1	0	0	0.174	0	0	0.133	0	0	0.089	0	0	0.396
Vegetables (excluding potatoes)	7	0	0	0.333	0	0	1.01	14.3	0.036	1.29	5	0.036	2.63
Margarine	4	0	0	1.33	0	0	3.08	0	0	10.5	0	0	14.9
Eggs and derivatives	16	0	0	0.225	0	0	0.844	0	0	1.34	0	0	2.41
Pizzas, quiches and savoury pastries	4	0	0	0.2	0	0	0.85	0	0	1.45	0	0	2.5
Mixed dishes	13	0	0	0.19	0	0	0.731	7.7	0.024	1.03	3	0.024	1.95
Fish	25	76	3.05	3.14	64	2.27	2.63	68	2.15	2.56	69	7.46	8.33
Ultra-fresh dairy products	59	0	0	0.2	0	0	0.342	0	0	0.605	0	0	1.15
Meat	33	3	0.001	0.623	0	0	0.957	27.3	0.07	0.98	10	0.071	2.56
Poultry and game	7	0	0	0.347	0	0	0.824	0	0	0.933	0	0	2.1

N : number of composite samples analysed

LB : Lower bound hypothesis ; UB :Upper bound hypothesis

Table 5 Mean concentrations (ng.g⁻¹ fw) of TBBPA in food

Food item	N	% detection	LB	UB
Infant specific food				
Milk-based beverage	8	0	0.00	10.40
Cereals	5	0	0.00	34.77
Milk-based desserts	6	0	0.00	11.57
Growing-up milk	9	100	21.70	21.70
Soups and puree	11	0	0.00	8.37
Fruit puree	4	0	0.00	13.67
Vegetable-based ready-to-eat meal	20	10	0.78	10.35
Meat/fish-based ready-to-eat meal	45	9	4.05	12.68
Infant formula	28	61	45.24	48.60
Follow-on formula	33	73	57.25	60.08
Common food				
Butter	1	0	0.00	46.11
Non-alcoholic beverages	1	0	0.00	4.04
Delicatessen	3	0	0.00	20.52
Chocolate	2	50	31.61	61.90
Dairy-based desserts	2	0	0.00	21.17
Cheese	3	0	0.00	31.99
Milk	4	0	0.00	10.78
Eggs and derivatives	1	0	0.00	13.74
Mixed dishes	4	25	7.38	26.88
Fish	3	33.3	5.55	18.43
Potatoes and potato products	1	0	0.00	11.86
Pasta	1	0	0.00	20.77
Ultra-fresh dairy products	5	0	0.00	12.30
Meat	2	50	10.69	25.35

Food item	N	% detection	LB	UB
Croissant-like pastries	1	100	913.64	913.64
Poultry and game	2	50	41.92	53.58

N : number of composite samples analysed

LB : Lower bound hypothesis ; UB :Upper bound hypothesis

Table 6 Dietary exposure to PFOA and PFOS and risk assessment (Upper bound hypothesis)

Substance	Age group	Reference dose (ng.kg bw ⁻¹ .d ⁻¹)	Mean (ng.kg bw ⁻¹ .d ⁻¹)	Standard deviation (ng.kg bw ⁻¹ .d ⁻¹)	90 th percentile (ng.kg bw ⁻¹ .d ⁻¹)	Percentage of individuals exceeding the reference dose
PFOA	1-4 mo	200	1.49	3,19	1.77	0
	5-6 mo		2.10	2,29	4.59	
	7-12 mo		2.42	1,84	4.71	
	13-36 mo		2.95	1,60	5.16	
PFOS	1-4 mo	80	1.83	2,66	2.04	0
	5-6 mo		1.61	1,99	3.56	
	7-12 mo		1.81	1,58	3.94	
	13-36 mo		2.66	1,43	4.63	
PFOA	1-4 mo	0.8	1.49	3,19	1.77	50.4 (39.4-61.5)
	5-6 mo		2.10	2,29	4.59	86.1 (75.4-96.9)
	7-12 mo		2.42	1,84	4.71	94.8 (90.8-98.8)
	13-36 mo		2.95	1,60	5.16	98.5 (97.3-99.6)
PFOS	1-4 mo	1.8	1.83	2,66	2.04	18.1 (9.6-26.7)
	5-6 mo		1.61	1,99	3.56	23.1 (10.0-36.2)
	7-12 mo		1.81	1,58	3.94	25.8 (17.8-33.7)
	13-36 mo		2.66	1,43	4.63	66.2 (62.0-70.5)
PFBS	1-4 mo	80 000	2.44	6.16	2.04	0
	5-6 mo		2.15	3.95	2.89	
	7-12 mo		2.36	2.57	5.13	
	13-36 mo		3.84	2.33	7.32	
PFHxA	1-4 mo	320 000	2.42	5.84	2.13	0
	5-6 mo		2.29	3.84	5.6	
	7-12 mo		2.7	3.24	6.69	
	13-36 mo		4.25	2.84	8	

Table 7 Dietary exposure to BDE-209 and risk assessment (Upper bound hypothesis)

Substance	Age group	BMDL ₁₀ (ng.kg bw ⁻¹ .d ⁻¹)	Mean (ng.kg bw ⁻¹ .d ⁻¹)	Standard Deviation (ng.kg bw ⁻¹ .d ⁻¹)	90 th percentile (ng.kg bw ⁻¹ .d ⁻¹)	Margin of safety at the mean (ng.kg bw ⁻¹ .d ⁻¹)	Margin of safety at the 90 th percentile (ng.kg bw ⁻¹ .d ⁻¹)
BDE 209	1-4 mo	1 700 000	2.62	1,63	3.78	650 000	450 000
	5-6 mo		2.23	1,96	3.91	760 000	430 000
	7-12 mo		1.96	1,36	3.20	870 000	530 000
	13-36 mo		1.12	0,78	1.88	1 500 000	910 000

BMDL= Lower limit of the benchmark dose

Table 8 Dietary exposure to the sum of the 7 PBDEs and risk assessment (Upper bound hypothesis)

Substance	Age group	Tolerable daily intake	Mean (ng.kg bw ⁻¹ .d ⁻¹)	Standard Deviation	90 th percentile (ng.kg bw ⁻¹ .d ⁻¹)	Percentage of individuals
-----------	-----------	------------------------	---	--------------------	--	---------------------------

		(ng.kg bw ⁻¹ .d ⁻¹)		(ng.kg bw ⁻¹ .d ⁻¹)		exceeding the reference dose
Sum of the 7 PBDE	1-4 mo	10	0.926	0,455	1.56	0
	5-6 mo		0.553	0,330	1.01	
	7-12 mo		0.499	0,428	0.812	
	13-36 mo		0.448	0,477	0.694	

Sum of the 7 PBDE = BDE-28 + -47 + -99 + -100 + -153 + -154 + -183)

Table 9 Dietary exposure to HBCDDs and risk assessment (Upper bound hypothesis)

Age group	BMDL ₁₀ (ng.kg bw ⁻¹ .d ⁻¹)	Mean (ng.kg bw ⁻¹ .d ⁻¹)	Standard Deviation (ng.kg bw ⁻¹ .d ⁻¹)	Median (ng.kg bw ⁻¹ .d ⁻¹)	90 th percentile (ng.kg bw ⁻¹ .d ⁻¹)	Margin of safety at the mean (ng.kg bw ⁻¹ .d ⁻¹)	Margin of safety at the median (ng.kg bw ⁻¹ .d ⁻¹)	Margin of safety at the 90 th percentile (ng.kg bw ⁻¹ .d ⁻¹)
1-4 mo	3000	8.27	18,27	0.662	43.2	363	4 530	70
5-6 mo	3000	0.883	0,962	0.588	1.78	3 400	5 100	1 690
7-12 mo	3000	0.827	0,696	0.574	1.79	3 630	5 230	1 680
13-36 mo	3000	0.505	0,317	0.418	0.88	5 940	7 180	3 410

Table 10 Dietary exposure to PBBs and risk assessment (Upper bound hypothesis)

Age group	NOAEL (ng.kg bw ⁻¹ .d ⁻¹)	Mean (ng.kg bw ⁻¹ .d ⁻¹)	Standard Deviation (ng.kg bw ⁻¹ .d ⁻¹)	90 th percentile (ng.kg bw ⁻¹ .d ⁻¹)	Margin of safety at the mean (ng.kg bw ⁻¹ .d ⁻¹)	Margin of safety at the 90 th percentile (ng.kg bw ⁻¹ .d ⁻¹)
1-4 mo	150 000	0.097	0,062	0.204	1 500 000	740 000
5-6 mo		0.076	0,038	0.112	2 000 000	1 300 000
7-12 mo		0.063	0,028	0.098	2 400 000	1 500 000
13-36 mo		0.049	0,022	0.074	3 100 000	2 000 000

Table 11 Dietary exposure to TBBPA and risk assessment (Upper bound hypothesis)

Age group	BMDL ₁₀ (ng.kg bw ⁻¹ .d ⁻¹)	Mean (ng.kg bw ⁻¹ .d ⁻¹)	Standard Deviation (ng.kg bw ⁻¹ .d ⁻¹)	90 th percentile (ng.kg bw ⁻¹ .d ⁻¹)	Margin of safety at the mean (ng.kg bw ⁻¹ .d ⁻¹)	Margin of safety at the 90 th percentile (ng.kg bw ⁻¹ .d ⁻¹)
1-4 mo	16 000 000	9.94	12,72	31.3	1 610 000	511 000
5-6 mo		5.60	9,37	16.3	2 857 000	982 000
7-12 mo		3.28	5,02	8.39	4 878 000	1 907 000
13-36 mo		0.968	0,794	1.80	16 529 000	8 889 000

BMDL : Lower limit of the benchmark dose