Estimation of *per capita* intake of phosphorous flame retardants (PFRs) using Swedish market basket food samples

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Introduction

Brominated flame retardants (BFRs), such as polybrominated diphenyl ethers (PBDEs) and hexabromocyclododecane (HBCD), have been used as additives in furniture, electronics, building materials, textiles, etc. to reduce the risk of fire spreading. These chemicals have been ubiquitously found both in environmental and food matrices (Wei et al. 2015). Due to evidence of their toxicity and persistency, and to increasing health and environmental concerns, the main BFRs were gradually banned worldwide, leading to the increased usage of phosphorous flame retardants (PFRs) as alternatives (Marklund et al. 2010). As a consequence, PFRs are found in many environmental matrices, such as air, dust, surface water, sediments, and biota (Zeng et al. 2014; Brandsma et al. 2015; Malarvannan et al. 2015; Kim et al. 2013). Knowledge about the environmental persistence, human exposure and toxicity of PFRs are, however, still limited (Wei et al. 2015). Foodstuffs might be contaminated with PFRs by bioaccumulation in food of animal origin and during the food treatment processes and packaging (Campone et al. 2010). However, information on the presence of PFRs in foodstuffs is still scarce.

In this study, we investigated the route of exposure to PFRs *via* diet. In total, 8 PFRs (namely, tris(1,3-dichloro-2-propyl) phosphate (TDCIPP), tris(1-chloro-2-propyl) phosphate (TCIPP), tris(2-chloroethyl) phosphate (TCEP), tri-n-butyl phosphate (TNBP), tris(2-ethylhexyl) phosphate (TEHP), triphenyl phosphate (TPHP), tris(2-butoxyethyl) phosphate (TBOEP), 2-ethylhexyl diphenyl phosphate (EHDPHP)) were analyzed in 53 food samples belonging to 13 different food categories (including cereals, pastries, meat, fish, dairy products (fluid and solid), eggs, fats/oils, vegetables, fruit, potatoes, sugar/sweets, and beverages), from a recent Swedish food market basket study (2015). Based on the analysis results, the human *per capita* exposure to PFRs from food was estimated.

Materials and methods

The food samples were freeze dried and 0.50 g of dry sample, or 0.10 g of fats/oils, was spiked with a proper solution of internal standard (IS) and extracted by solid-liquid extraction in 5 mL of acetonitrile. The extract was cleaned up through dispersive solid phase extraction (d-SPE) and Florisil. The analysis of the target compounds was performed by GC-MS in the electron-impact (EI) mode. The mass spectrometer was run in SIM mode with 2-3 characteristic ions acquired for each analyte and the corresponding IS: TAP was used as IS for TEHP, TNBP; TCEP-d12 was used for TCEP and TCIPP (2 isomers); TBOEP-d6 was used for TBOEP; TPHP-d15 was used for TPHP and EHDPHP; TDCIPP-d15 was used for TDCIPP.

The calculation of the *per capita* exposure was based on the *per capita* consumption, which represents the calculated mean population consumption of various food groups derived from Swedish sales and production statistics. The *per capita* intake of the respective PFRs was derived by multiplying the *per capita* consumption amount of a specific food category by the concentration of the actual compound found in the food homogenate of this category. The average body weight of the Swedish population (67.2 kg) was used to present the data on a body weight basis.

Results and discussion

The results showed detectable levels of PFRs in most of the 13 considered food groups. Differently from PBDEs, usually present at high levels in foods of animal origin, the highest levels of PFRs were

measured in highly processed foods such as cereals, pastries, fats/oils, and sugar/sweets (ΣPFRs up to 19.1 ng/g wet weight). A possible explanation could be a low bioaccumulation/concentration of these compounds in biota, and consequently in the human food chain (Greaves & Letcher 2016), and the PFR contamination during food processing/storage. Among the analyzed PFRs, EHDPHP had the highest median concentrations (9 ng/g ww) and was detected in most food groups, possibly due to the use of EHDPHP in food packaging materials (US FDA 2006). It was followed by TPHP (2.6 ng/g ww), TDCIPP (1.0 ng/g ww), TCEP (1.0 ng/g ww), and TCIPP (0.80 ng/g ww). TEHP, TNBP and TBOEP were not detected in the analyzed food samples.

For the average adult population, the calculated daily *per capita* intakes of the five different PFR compounds from food ranged from 406 to 3266 ng/day (from 6 to 49 ng/kg bw/day). The major contributor to the total intake was EHDPHP (57 %), followed by TDCIPP (14 %), TPHP (11%), TCIPP (10 %), TCEP (7 %), whereas the food categories contributing most to the total PFR intake were cereals (26 %), beverages (17 %), sugar/sweets (11 %) and pastries (10 %). The estimated *per capita* dietary intake values of the analyzed PFRs were compared to the available reference doses (RfD) for the target PFRs (Ali et al. 2012), resulting in levels several orders of magnitude lower than the indicated RfD-values.

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