BFR Exposure: Air, Particles and Food Matters

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Introduction

There are a limited number of brominated flame retardants (BFRs) in commercial use today [1] even though the numbers of individual chemical species among the BFRs, present and previous, are far higher [2-4]. In addition to the parent technically produced BFRs, there are an almost uncountable number of abiotically or metabolically formed secondary products of these chemicals. In relation to all known BFRs and their degradation products only a very limited number of chemicals are scientifically followed regarding the exposure perspective.

The aim of the present mini-review is to give an overview of general human exposure to BFRs and their metabolites, to compare exposure sources and to draw some conclusions from there.

The historical perspective on human exposure to BFRs, i.e. exposures until the millennium change are basically limited to studies on exposure to polybrominated biphenyls related to the accidental distribution of Firemaster BP6 in Michigan 1973 [5], to human exposure to tris(dibromopropyl) phosphate in the 1970's [6] and not much more. The human exposure to these chemicals interlinked with their toxicity led to their ban. The next class of BFRs to be studied for their human exposure were the polybrominated diphenyl ethers (PBDEs). However, very few data were available still in the late 1990's. At this stage the discussion was only related to analysis of levels of PBDEs even though the temporal trend study performed by Norén and co-workers in human milk from mothers in Stockholm [7] made a major case. The BFR exposure research started very much from this point, including studies of exposure routes and discussion about the impact of the different pathways. These are the issues discussed in this mini-review.

Human Exposure to BFRs

The human exposure pathways for anthropogenic compounds are well known. The general view on their relative importance for building up elevated concentrations of lipophilic and persistent organic pollutants (POPs) in the human body is that ingestion of POPs via food is the primary route of exposure. Inhalation may become important for the public population if the compound is volatile. The volatility of a chemical is always an important parameter when humans are exposed under occupational conditions. Dermal uptake is either dependent on work related exposure or use of hygiene and cosmetic products.

Dermal exposure: Dermal exposure is not relevant for humans in general unless a more pronounced use of BFRs come into play in cloths worn close to the body. This was indeed the case for tris(dibromopropyl) phosphate when applied in e.g. pyjamas for babies [8]. Except for this issue, uptake of BFRs through the skin has not really been well studied. However, in the electronics dismantling industry this issue was addressed after elevated blood concentrations of PBDEs were reported [9]. It is questionable if handling of flame retarded rubber had any particular influence on the human exposure to personnel handling DecaBDE treated rubber coated electric wires [10]. Still these personnel have in

some cases very high blood concentrations of BDE-209 and of some octa- and nonaBDEs implying that there may be a link.

Air exposure: Human outdoor exposure to gaseous BFRs is most likely negligible, at least for most BFRs, since their volatilities are low [11] as well as their air concentrations [12,13]. Due to the physicochemical characteristics of most of the neutral BFRs, they are strongly bound to particulates, including dust. This is shown in a series of studies where persons are working in the electronics dismantling industry [14-16]. In the first of these studies, significantly higher concentrations of BDE-183 and BDE-209 were observed [9]. After some reorganisation of the facility new human blood samples showed significantly lower exposures to these highly brominated diphenyl ethers [17]. This is due to lower dust and particle content of the indoor air in the dismantling hall even though lager volumes of electronic waste were handled.

Exposure via dust and/or particulates: Particle and dust matters as shown for the industry workers but also for people in offices and in their homes. Several studies indicate, as reviewed by Hale and coworkers, ppm concentrations of PBDEs in dust [18]. More of the volatile PBDEs are indicated in e.g. air conditioning filters. An extensive study on PBDEs in a standard reference material of indoor dust is reporting on concentrations of approx. 20 PBDE congeners [19]. The levels of the sum of PBDEs are in the low ppm range, with BDE-209 making up more than 50% of the total. BDE-209 was the dominant PBDE congener in all dust samples compiled and recently presented by Karlsson and coworkers [20]. The high concentrations of PBDEs in humans from North America seem to relate primarily the dust and particulates and not to any other presently known source [21]. The importance of dust and particulate exposures are strengthened by the observed high levels of PBDEs in young individuals, young children, as reported on as a case study from a Berkley family [22]. Also other, published [23,24] and hitherto unpublished data indicate high concentrations of the PBDEs among children (Bergman, unpublished).

Independent of the large number of individual BFR chemical class species, including tetrabromobisphenol A (TBBBPA), hexabromocyclododecane (HBCDD), bis(2,4,6-tribromophenoxy) ethane (BTBPE), decabromodiphenyl ethane (DBDPE) or others there are very little known on air and particle and/or dust as sources for human exposure. Only few studies report on TBBPA air concentrations and then primarily in work places [12,15,16,25]. HBCDD is distributed via air and has been reported in house dust [26]. BTBPE has been reported in air sampled at a electronics dismantling facility [16]. However, it is difficult to make links between air or dust and human exposure to any of the BFRs but the PBDEs. One reason for this may also be the very short half life of TBBPA, hours, in humans and in rats (13 h) [27].

Food exposure: The BFR exposure that matters for humans in general and all over the world is intake via food sources. An extensive number of reports on levels in food and internal exposure levels in humans are available. Our own studies have concentrated to fish, eggs, pilot whale blubber and mothers milk. Two recent food basket surveys were published 2006 [28,29]. A comparison even between these two studies is difficult due to differences in their design. The Swedish study is limited to the five PBDE congeners #47, 99, 100, 153 and 154 while the U.S. study include an additional number of PBDE congeners, including BDE-209. This is important since several dairy products do indeed contain BDE-209 as well as chicken eggs, chicken and calf liver and butter. In these matrices the BDE-209 is only somewhat lower except for calf liver where it is higher than any of the other PBDE congeners. A comparison of ΣPBDEs (not including BDE-209) in a few food items or basket groups

Table 1. Comparison of some fresh weight concentrations (pg/g f.w.) of PBDEs in some food products from the U.S. and Sweden. Data shown on basis of the references [28-30].

Food Items	Origin	ΣPBDEs[1)]	BDE-209	ΣPCBs	Ref.
Dairy products	U.S.A.	76	40	n.a.[2)]	Schecter et al., 2006 [28]
Dairy products		26	n.a.	70	Darnerud et al., 2006 [29]
Beef	U.S.A.	76	n.a.	n.a.	Huwe et al., 2005 [30]
Calf liver	U.S.A.	25	80	n.a.	Schecter et al. 2006 [28]
Meat		60	n.a.	530	Darnerud et al., 2006 [29]
Egg	U.S.A.	75	10	n.a.	Schecter et al. 2006 [28]
Egg		47	n.a.	1260	Darnerud et al., 2006 [29]

[1)] BDE-209 concentration not included; [2)] n.a. = not analysed/not available

of food are extracted from the two studies and shown in Table 1. Even though there are some differences between the Swedish and the U.S. these differences seem to be marginal. Concentrations of ΣPCBs are given for comparison on the Swedish food basket.

It is difficult to find any major reasons for differences in PBDE concentrations between Sweden and the U.S. as well as most other developed countries. Interestingly, Schecter and co-workers show the importance of BDE-209 in food [28]. This is somewhat surprising since BDE-209 has a short half life in

several mammals, including humans [31-33] and accordingly this compound may be expected to behave similarly in other mammals. It can not be excluded that DecaBDE is contaminating food during processing. This has however not been unambiguously shown even though there is an indication of this in the study performed by Schecter and co-workers, showing high BDE-209 levels in processed food [28]. Human milk in the U.S.A. contains high concentrations of PBDEs, primarily lower brominated diphenyl ethers. BDE-209 concentrations in mother's milk are in general low but possibly too few studies have so far included BDE-209 in the analyses. In our study [34] we were unable to determine any temporal trend for BDE-209 due to low levels of this compound in the human milk analysed.

The human PBDE concentrations are stressing differences in exposure to these compounds around the world. Today there is PBDE concentration data from a large number of countries. Still the by far highest human concentrations are observed in the U.S., Canada and Nicaragua [23,35]. The concentrations differ to other countries in the range of one to two orders of magnitude. To the best of my understanding this is due to inhalation and/or ingestion of dust and particles coated with PBDEs. The support for this conclusion is the high levels in workers subjected to PBDE exposure, the dump site children in Nicaragua being exposed to high concentrations of particles and working at a site continuous on fire and the high concentrations in small children in the U.S. [22] and elsewhere [24].

BFR metabolite exposure: The PBDE exposure of humans, independent of the external route, is leading to exposure to PBDE metabolites. We have recently been able to show that humans are transforming lower brominated diphenyl ethers to OH-PBDEs [36]. The qualitative analysis of human serum is showing the retention of almost 20 OH-PBDEs in the blood compartment. Further, debromination of BDE-209 is most likely occurring as shown by Thuresson in personnel exposed to DecaBDE under occupational conditions [10]. Unfortunately there is a lack of standards for identification of more than a smaller number of all indicated PBDE metabolites.

As stated above TBBPA is rapidly metabolized in rats and humans [27] and accordingly not bioaccumulative. On the contrary, HBCDD is bioaccumulative but so far only low concentrations are present in e.g. human blood [37]. The temporal trend of HBCDD in Swedish mother's milk is showing an increase over time but with a uncertain trend of levelling off or possibly decreasing at the latest time points [34]. Exposure route and internal exposure data for other BFRs are scarce, if at all existing.

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