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Intake, distribution, and metabolism of decabromodiphenyl ether and its main metabolites in chickens and implications for human dietary exposure☆

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ABSTRACT

Diet is considered as the most important human exposure pathway for polybrominated diphenyl ethers (PBDEs). Metabolism and accumulation patterns of PBDEs in different growth periods of chickens are helpful for evaluating human dietary exposure, but such information is scarce. In this study, female chickens were fed with food spiked with BDE-209 at 85 mg kg^{-1}, and the intake, accumulation, and excretion of BDE-209 and its main metabolites in various tissues were examined. Concentrations of BDE-209 in chicken tissues increased over time in a tissue-specific manner; they were the greatest in liver and generally the lowest in breast meat during the entire exposure period. The kinetic patterns were dependent on both growth-dilution effects and accumulated concentrations of BDE-209. Tissue concentrations of $\sum_8$PBDE (sum of BDE-28, 47, 99, 100, 153, 154, 183, and 209) followed the sequence of liver > blood > skin > intestine > stomach > leg meat > breast meat. Different tissue partition coefficients and perfusion rates for blood may have resulted in different PBDE concentrations in tissues. The absorption efficiency of BDE-209 in chicken tissues followed the sequence of liver (0.15 ± 0.032%) > skin (0.14 ± 0.038%) > intestine (0.071 ± 0.021%) > breast meat (0.062 ± 0.020%) > leg meat (0.059 ± 0.016%) > stomach (0.021 ± 0.0095%), likely due in part to facilitated absorption of BDE-209 by transport proteins (P-glycoproteins). On average, 9.3 ± 1.7% of BDE-209 was excreted in feces. Estimated human average dietary intake via the consumption of chicken tissues of $\sum_8$PBDE for adults and children was 319 and 1380 ng day^{-1} for liver, 211 and 632 ng day^{-1} for leg meat, and 104 and 311 ng day^{-1} for breast meat from the contaminated group. Liver clearly poses the highest exposure risk for human consumption, particularly if chickens are fed with contaminated feed.

1. Introduction

Polybrominated diphenyl ethers (PBDEs) are widely used as flame retardants (Alonso et al., 2012; Pirard and De Pauw, 2007). Penta-, octa-, and deca-BDE commercial mixtures have been gradually banned from manufacture and use by the European Union (EU) since 2004 (EU, 2004, 2008), because PBDEs could result in thyroid toxicity, developmental neurotoxicity, and endocrine disruption on animals and humans (Bellés et al., 2010; Kim et al., 2013; Reverte et al., 2014). Despite these bans and restrictions on them, PBDEs can still be easily released to the environment as they are physically bonded in products (Chen et al., 2007; de Wit, 2002; Domingo, 2012). They biomagnify through the food web (Losada et al., 2009; Mizukawa et al., 2009), resulting in higher PBDE concentrations in humans through consumption of food (Ohta et al., 2002).

Therefore, reducing the concentrations of PBDE in food is

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significant for the protection of human health, which in turn relies on informed knowledge about the bioaccumulation and metabolism of PBDEs in animals.

Previous studies have focused on the concentrations of PBDEs in chicken tissues (Zhao et al., 2016), as well as those of PBDEs, hexabromocyclododecanes, polychlorinated dibenzo-p-dioxins, tetrabromobisphenol, polychlorinated biphenyls, and polychlorinated dibenzofurans in chicken eggs (Piskorska-Pliszczynska et al., 2016; Squadrone et al., 2015; Zeng et al., 2016). The pharmacokinetics of low-brominated PBDEs in chicken (*Gallus gallus domesticus*), a major food item worldwide, were also carefully investigated (McKernan et al., 2010). However data about bioaccumulation kinetics of highly brominated BDE congeners in chicken have remained scarce (Covaci et al., 2011). Pirard and De Pauw (2007) found that concentrations of ∑PBDE (sum of BDE-47, 99, 100, 153, 154, and 183) were higher in adipose than in liver and eggs of laying hens. Voorspoels et al. (2006) indicated that the concentrations of ∑PBDE (sum of BDE-28, 47, 99, 100, 153, 154, and 183) in the tissues of common buzzards (26–130 ng g^{-1} lipid weight) were an order of magnitude lower than those of sparrow hawks (360–1900 ng g^{-1} lipid weight); BDE-209 was found in liver and serum only. Understanding the pharmacokinetics of a wide range of PBDEs in chicken tissues is significant for quantifying the mechanisms of PBDE bioaccumulation in chicken, a major source of proteins for the general population. This information is critical for gauging the magnitude of human health risk through consumption of chickens, because diet is considered as the most important human exposure pathway for PBDEs (Kang et al., 2010; Ni et al., 2012) and BDE-209 is the prominent congener in most animal-derived food products (EFSA, 2011).

Dietary habits also vary by country; e.g., viscera organs such as liver and intestines are widely consumed in some countries such as Korea, France, and China (Hoffmeister et al., 2007; Xing et al., 2010), but are relatively uncommon in Japan and North America. Because chicken offal is often used to feed other livestock, this practice would eventually cause exposure risk to humans. Therefore, distribution patterns of PBDEs in chicken tissues at different growth stages are of great significance for assessing human health effects. Distribution patterns of PBDEs in chicken visceral organs, and particularly in liver, would provide insights into metabolism, migration, and accumulation of PBDEs in chickens. A previous study on rats suggested that lipophilic tissues such as skin, adipose, and gastrointestinal tract were the major reservoirs of PBDEs (Orn and Klasson-Wehler, 1998). Currently, no data are available on the accumulation and transformation patterns of PBDEs in poultry of different growth stages.

To address the above-mentioned knowledge gap, we carried out a series of studies on the bioaccumulation kinetics of BDE-209 in chicken domesticated in a farmland. The objectives of the present study were to (1) obtain concentrations of BDE-209 in chicken tissues of different growth stages; (2) compare the distributions of BDE-209 and its main metabolites accumulated in different tissues of chicken; and (3) estimate the dietary intakes of PBDEs for children and adults via the consumption of chicken muscle and liver and related potential health risk.

2. Materials and methods

2.1. Materials

Eight target PBDE standard solutions, including BDE-28, 47, 99, 100, 153, 154, 183, and 209), sum of which is designated as $\sum_8$PBDE, three internal standards, i.e., BDE-69, 3-fluoro-2,2′,4,4′,5,6-hexabromodiphenyl ether (F-BDE-139), and 4′,6-difluoro-2,2′,3,3′,4,5,5′,6′-octabromodiphenyl ether (F-BDE-201), and three surrogate standards, including BDE-51, 115, and 4′-fluoro-2,2′,3,3′,4,5,5′,6,6′-nonabromodiphenyl ether (F-BDE-208), were all purchased from AccuStandard (New Haven, CT, USA). Sulfuric acid was obtained from Damao (Tianjin, China). Dichloromethane and *n*-hexane of HPLC grade were obtained from Oceanpak (Gothenburg, Sweden), and acetone of analytical reagent grade was obtained from GongDong Hing Wah (Guangdong, China). Bio-Beads SX-3 was obtained from AnPel (Shanghai, China). Acetone was redistilled in an all-glass system before use, and the Bio-Beads were soaked in dichloromethane until use.

Sunflower oil (Hebei, China) was spiked with BDE-209 and mixed with 8.5 kg of commercially available feed under constant agitation (150 rpm at 20 °C) to prepare contaminated feed with final nominal concentrations of 85 mg kg^{-1} for BDE-209, with impurities of 0.04 and 4.4 mg kg^{-1} for BDE-47 and 99, respectively. The concentration of BDE-209 prepared in contaminated feed was similar to that in soil (mean: 58.7 mg kg^{-1}; range: 17–146 mg kg^{-1}) near a manufacturing plant (Li et al., 2015). The control feed was treated in an analogous manner with no BDE-209 added.

2.2. Accumulation of BDE-209 in chicken

Two groups of 30-day old female chickens, each with 15 individuals, were raised indoors under controlled conditions in individual hencoops in a chicken farm at Luogang (Guangzhou, China), equally separated as contaminated and control groups and fed with contaminated and non-contaminated feeds, respectively. After a 10-day period of adaptation, each individual chicken was reared with 90 g of feed per day. At each 10-day exposure time, three chickens from contaminated and control groups, respectively, were slaughtered. During the entire exposure period of 50 days, the mass of the chickens was weighed and chicken feces were collected.

2.3. Sample preparation and extraction

For each batch of slaughtered chickens, blood was collected in polytetrafluoroethylene tubes and agitated at 3500 rpm at 20 °C. Serum was collected and stored at −80 °C. Tissue samples (liver, intestine, stomach, skin, leg meat, and breast meat) and feces samples were excised and wrapped by aluminum-foil, vacuumed, and stored at −80 °C until processing. At that time, each freeze-dried tissue sample was ground into fine homogeneous powders using a grinding miller, wrapped by aluminum-foil, vacuumed, and stored at −80 °C until extraction. The contaminated and control groups were processed separately to prevent cross contamination.

Each sample of 5 g for the control group or 0.5 g for the contaminated group chicken was spiked with surrogate standards and sonicated three times with a mixture of hexane: dichloromethane: acetone (2:2:1 in volume). Twenty percent of the combined extract was used to determine lipid content. Sulfuric acid was added to the remaining extract to remove organics. The supernatant of the sulfuric acid extract was concentrated to 2 g, then fractionated with 30 mL of dichloromethane: hexane (1:1 in volume) through a gel permeation chromatographic column containing 6 g of Bio-Beads SX-3. The fraction eluted at 15–30 mL was reduced to 100 μL under a gentle nitrogen stream. Finally, internal standards were spiked before instrumental analysis.

2.4. Instrumental analysis

Extracts were analyzed with an Agilent 7890A gas chromatograph coupled to a 5975C mass spectrometer in the negative chemical ionization mode. A DB-5HT capillary column (15 m × 0.25 mm i.d; 0.1 μm film thickness) was used to separate

the target analytes. The column temperature was programmed from 80 °C (held for 1.0 min) to 200 °C at 30 °C min^{-1} (held for 4.0 min), increased to 260 °C at 10 °C min^{-1} (held for 1.0 min), and finally elevated to 310 °C at 15 °C min^{-1} (held for 5.0 min). One microliter of each extract was injected automatically in the splitless/spilt mode with a split time of 1.0 min. Ultrahigh purity helium served as carrier gas at a flow rate of 1.5 mL min^{-1}. The ion source and interface temperatures were set at 200 °C and 250 °C, respectively. Quantitative analysis was conducted in selected ion monitoring mode. The reporting limits for tetra-to octa-BDEs and BDE-209 were 0.025 ng g^{-1} and 0.25 ng g^{-1}, respectively, for 20 g wet sample weight.

2.5. Quality control and quality assurance

For each batch of 20 samples, one procedural blank, one matrix duplicate, two spiked blanks, and two matrix spiked samples were analyzed. The recoveries of BDE-51, BDE-115, and F-BDE-208 were 88 ± 15%, 86 ± 12%, and 85 ± 13% in field samples, and 93 ± 10%, 96 ± 7%, and 89 ± 6% in blank samples, respectively. The concentrations of BDE congeners in all tissue samples were all normalized to wet sample weights. Only BDE-28 and BDE-47 were detected in the procedural blank samples, with average concentrations of 0.026 and 0.030 ng g^{-1}. These values were fairly low compared to those in the chicken tissues. Concentrations of BDE-28 and BDE-47 in the procedural blank samples in the same batch, which exceeded the reporting limits, were corrected for their measured concentrations in the chicken tissue samples. All concentrations greater than the reporting limits were reported without correction for surrogate standard recoveries.

2.6. Data analysis

Data comparison in chicken tissues was performed with *t*-test (Excel 2007; Microsoft, Washington, USA). Statistical significance was defined at $p < 0.05$. The growth rate for chicken stomach or liver was estimated by fitting the mass data of each tissue to a logarithmic model (Neely, 1980):

$$\ln m = a + b \times t \quad (1)$$

Where m is the tissue mass (g; wet weight); a is a constant; b is the growth rate (day^{-1}); and t is the time (day).

Daily consumption rates of chicken meat and liver products were 10 and 5 g day^{-1} wet weight for children (Labunska et al., 2014) and 16 and 5.5 g day^{-1} wet weight for adults (Labunska et al., 2014; Xing et al., 2010). The average standard 3-year-old boy and male adult body weights of 14.65 kg (Ministry of Health of China, 2012) and 70 kg (Ferrante et al., 2016) were used for the calculation. The estimated daily intake (EDI; ng kg^{-1} bw day^{-1}) was calculated:

$$EDI = C \times CR / BW \quad (2)$$

Where C is the concentration of $\sum_8$PBDE or an individual congener in chicken meat or liver (ng g^{-1} wet weight); CR is the consumption rate of chicken meat or liver (g day^{-1}); and BW is the body weight (kg).

3. Results and discussion

3.1. Distribution of BDE-209 and its main metabolites in chicken tissues

The concentrations of BDE-209 in all tissues were the highest among the target BDE congeners (Table 1 and Table S1; "S" indicates tables in the Supplementary data afterwards), accounting for up to 78–96% and 68–98% of the total amounts in the contaminated and control groups, respectively. The average concentrations of $\sum_8$PBDE (sum of BDE-209 and its main metabolites BDE-28, 47, 99, 100, 153, 154, and 183) in different tissues of 90-day old chickens in the contaminated group ranged from 456 ng g^{-1} in breast meat to 4050 ng g^{-1} in liver (Table 1). The average concentrations of $\sum_8$PBDE in contaminated chicken tissues followed the sequence of liver > blood > skin > intestine > stomach > leg meat > breast meat (Fig. 1). Liver contained the highest $\sum_8$PBDE concentrations during the entire exposure period, whereas breast meat in general contained lower $\sum_8$PBDE concentrations than other issues (Fig. 2). The concentrations of $\sum_8$PBDE in skin and intestine were not significantly different ($p > 0.05$) and no significant difference ($p > 0.05$) of $\sum_8$PBDE concentrations between stomach and leg meat was observed.

The inter-tissue distribution of organic pollutants is generally related to the tissue partition coefficients and perfusion rates of organic pollutants in blood (Barron et al., 1990). For instance, blood flow rates for viscera organs in aquatic animals, such as stomach, liver, and intestine, are several mL min^{-1} per g of tissue, but are substantially slower for adipose tissue, i.e., fat (Barron et al., 1990). Once absorbed, PBDEs are readily distributed to blood-rich tissues, i.e., the liver and intestinal wall (Morck et al., 2003). Higher concentrations of organochlorine pesticides and polycyclic aromatic hydrocarbons were also reported in pork and chicken livers than in other tissues upon dietary exposure (Covaci et al., 2004; Naf et al., 1992). Hence, PBDEs in food enter the liver via the first-pass effect for detoxification (Yang et al., 2010), resulting in the highest average concentration of $\sum_8$PBDE in liver among all tissues. A previous study also inferred that different PBDE concentrations in various chicken tissues were largely attributed to tissue-specificity, which is variable with tissue types, depending on tissue stability and storage capacity (Donohue et al., 1997).

For 90-day old chickens in the control group, the average concentrations of $\sum_8$PBDE in different tissues followed the sequence of stomach (23 ng g^{-1}) > liver (18 ng g^{-1}), skin (15 ng g^{-1}), intestine (12 ng g^{-1}), leg meat (10 ng g^{-1}), blood (7.1 ng g^{-1}), and breast meat (2.0 ng g^{-1}) (Table S1). As with the contaminated group, the variability of $\sum_8$PBDE concentrations with tissue types in the control group was also related to tissue-specificity.

3.2. Bioaccumulation kinetics of BDE-209

Bioaccumulation kinetics of BDE-209 showed generally increasing trends in different tissues for the contaminated group (Fig. 2). Gastrointestinal absorption is related to ingestion and excretion of feces (Richter and McLachlan, 1998). The concentration of BDE-209 in intestine continuously increased during the entire

Table 1
Average concentrations (ng g^{-1} wet weight) of PBDEs in 90-day old chickens of the contaminated group.

	Liver	Blood	Skin	Intestine	Stomach	Leg meat	Breast meat
BDE-28	< RL[a]	< RL	< RL	< RL	< RL	< RL	< RL
BDE-47	2.1	6.6	1.2	0.81	0.87	0.49	0.66
BDE-99	91	160	97	67	104	69	51
BDE-100	< RL	< RL	0.21	0.49	0.2	0.09	< RL
BDE-153	68	102	58	55	75	45	49
BDE-154	< RL	< RL	< RL	< RL	< RL	< RL	< RL
BDE-183	0.22	< RL	0.55	< RL	< RL	0.2	0.33
BDE-209	3890	3530	1940	1550	1320	810	355
$\sum_8$PBDE	4050	3800	2100	1670	1500	924	456

[a] Below the reporting limit (0.025 ng g^{-1}).

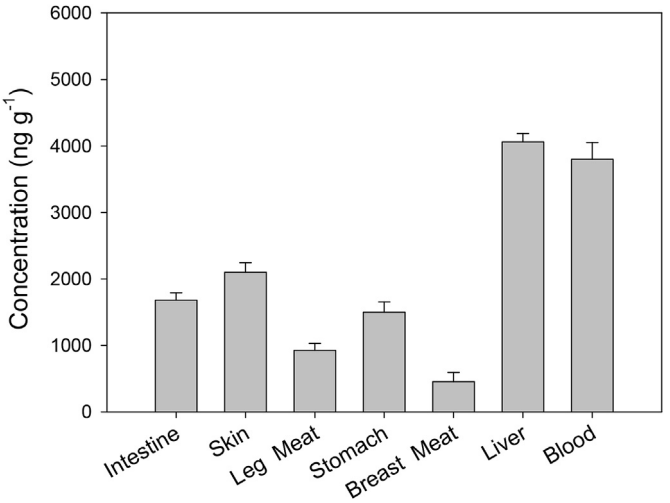

Fig. 1. Occurrence of $\sum_8$PBDE in intestine, skin, leg meat, stomach, breast meat, liver, and blood of chickens in the contaminated group.

exposure period (Fig. 2a), and is largely attributed to absorption of PBDEs in the intestine from chyme (Foster et al., 2011). For the 50-day exposure period, the concentrations of BDE-209 in stomach, breast meat, and blood reached maxima after the first 30 days of exposure, and decreased over the following 20 days (Fig. 2b–d). Different from stomach, breast meat, and blood, the concentrations of BDE-209 in skin, leg meat, and liver gradually increased and decreased alternately in the first 40 days and increased in the last 10 days (Fig. 2e–g). The kinetic patterns (Fig. 2) may have resulted from two competing factors, i.e., the bioaccumulation of BDE-209 in the organs and the growth of the organs themselves (Yang et al., 2010). The continuous growth of the organs offset to some degree the accumulated concentrations of BDE-209, such as stomach and liver (Fig. 2b and g), which was regressed as $\ln m = 3.1 + 0.024\,t$ for stomach and $\ln m = 4.1 + 0.020\,t$ for liver. Breast meat is the largest organ by mass, as a consequence of modern feeding strategies to enlarge chicken muscles rapidly for market (Yang et al., 2010). Hence, breast meat contained the lowest concentrations of BDE-209 among all the organs during the entire exposure of 50 days.

High activity levels of microflora are favorable for detoxification by transforming PBDEs in liver (Morck et al., 2003). The zig-zag pattern of BDE-209 accumulation kinetics in liver (Fig. 2g) was perhaps reflective of the inconsistent offset between rates of metabolism, growth dilution, and bioaccumulation, but more investigation is needed given the lack of current knowledge about metabolism rates. The concentration of BDE-209 in feces reached a maximum after the first 20 days of exposure and decreased in the later 30 days (Fig. 2h). This was probably due to the initial accumulation of abundant BDE-209 in chicken body, resulting in net excretion of BDE-209 in the feed before they could be absorbed. After a while, the chemicals had a chance to distribute themselves, and the chicken body started excreting the parent compounds that were absorbed. In both cases, concentrations of BDE-209 decreased in the feces. Our results were consistent with the study of Pirard and De Pauw (2005), who observed that the concentrations of polychlorinated dibenzo-*p*-dioxin and dibenzofuran in chicken feces reached a maximum after the first 5 weeks of exposure and decreased in the subsequent weeks.

3.3. Metabolism and mass balance of BDE-209 in chicken

The 90-day old chickens of the contaminated group were selected to evaluate the mass balance of BDE-209 in the chicken body. The absorption efficiency of BDE-209 in various tissues and feces was calculated by multiplying the concentration of BDE-209 in a specific tissue and the corresponding weights of these samples and dividing by the amount of BDE-209 in the feed. Only 9.3 ± 1.7% of BDE-209 on average was excreted out of the chicken based on this mass balance calculation. The absorption efficiencies of BDE-209 in chicken tissues followed the sequence of liver (0.15 ± 0.032%) > skin (0.14 ± 0.038%) > intestine (0.071 ± 0.021%) > breast meat (0.062 ± 0.020%) > leg meat (0.059 ± 0.016%) > stomach (0.021 ± 0.0095%) (Fig. 3). This pattern is probably due to facilitated absorption of BDE-209 by transport proteins (P-glycoproteins) (Charman, 2000; Tsuji and Tamai, 1996), at least in part. The absorption efficiencies of BDE-209 in the present study were much lower than those in rats dosed by gavage (26%) (Sandholm et al., 2003), but comparable to those in rainbow trout (0.02–0.13%) (Kierkegaard et al., 1999), and zebrafish (0.62%) (Nyholm et al., 2009), although different feeding modes may have played certain role in the outcome.

The results for the contaminated group showed that a small

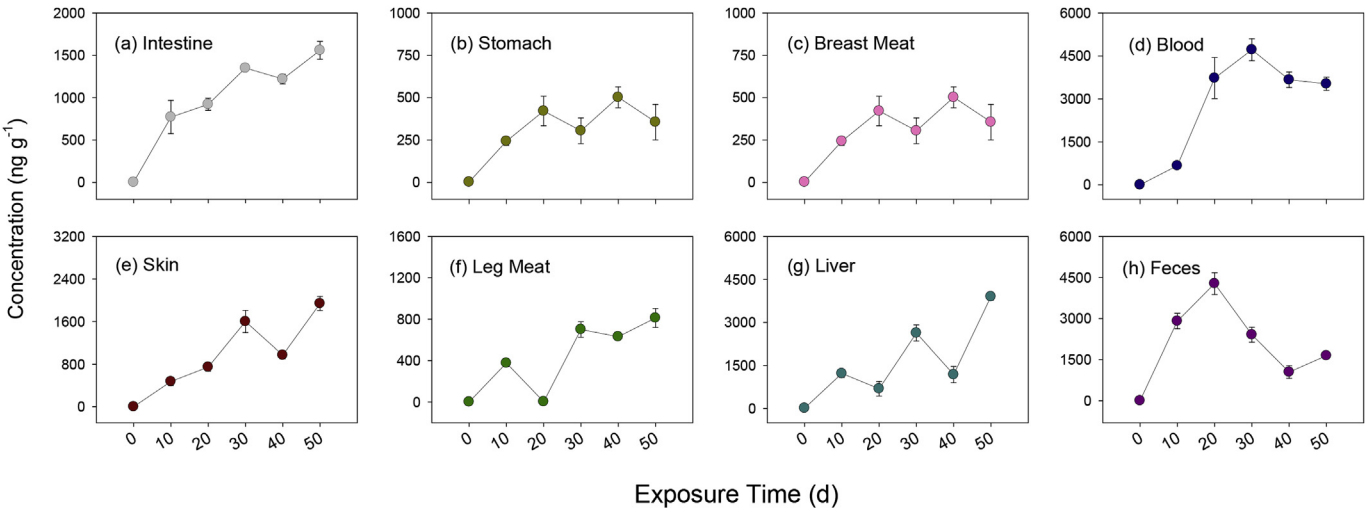

Fig. 2. Bioaccumulation kinetics of BDE-209 in various tissues and feces of chickens in the contaminated group: (a) intestine; (b) stomach; (c) breast meat; (d) blood; (e) skin; (f) leg meat; (g) liver; and (h) feces.

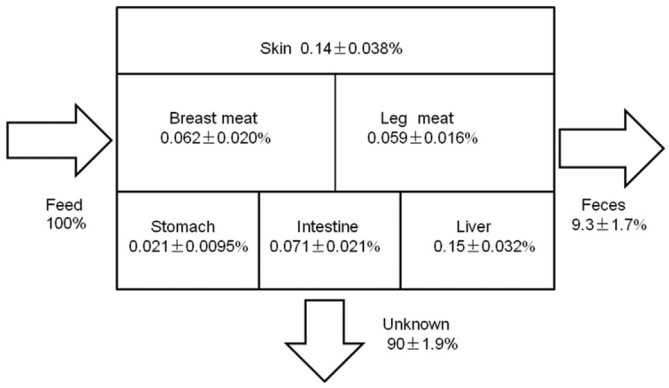

Fig. 3. Schematic showing mass balance of BDE-209 in the 90-day old chickens of the contaminated group.

portion of BDE-209 was reductively debrominated to BDE-47 (0.49–6.6 ng g^{-1}), BDE-99 (51–160 ng g^{-1}), BDE-100 (ND (<reporting limit)–0.49 ng g^{-1}), and BDE-183 (ND–0.55 ng g^{-1}) in chickens. BDE-47 and BDE-99 were perhaps derived from BDE-209 in contaminated feed and subsequent debromination. Similarly, European starlings (Van den Steen et al., 2007) and earthworms (Zhang et al., 2014) were also found to degrade BDE-209 to lower brominated congeners, such as BDE-47, 99, 153, 206, and 208. Several studies showed that BDE-209 can be accumulated and metabolized in fish, rats, and seals (Kierkegaard et al., 1999; Morck et al., 2003; Thomas et al., 2005). In fish, BDE-209 can be debrominated to lower brominated congeners such as penta-to nona-BDEs, which may be more toxic than BDE-209 (Stapleton et al., 2004, 2006). A small portion of BDE-209 can also be metabolized to hydroxymethoxy-substituted or hydroxyl-substituted diphenyl ethers in rats (Morck et al., 2003). Because none of the hydroxymethoxy-substituted or hydroxyl-substituted diphenyl ethers were targeted in the present study, these compounds were not included in subsequent risk assessment.

3.4. Human consumption risk assessment

Chicken meat is a major source of proteins for the general population and liver is also consumed in some countries such as Korea, France, and China (Hoffmeister et al., 2007; Xing et al., 2010). Consumption of intestine, stomach, skin, and blood, on the other hand, is relatively uncommon in the rest of the world. We thus selected chicken meat and liver for assessed daily intake exposure. The intakes of $\sum_8$PBDE by children and adults via consumption of leg meat, breast meat, and liver of 90-day old chickens in the contaminated group were estimated (Table 2). Estimated average daily intake of $\sum_8$PBDE for adults from chicken consumption decreased in the sequence of liver (319 ng day^{-1}) > leg meat (211 ng day^{-1}) > breast meat (104 ng day^{-1}). The same trend was also observed for children, i.e., liver (1380 ng day^{-1}) > leg meat (632 ng day^{-1}) > breast meat (311 ng day^{-1}). Consumption of these foodstuffs contributes to daily $\sum_8$PBDE intakes more substantially for children than for adults. Breast meat poses lower exposure risk for humans than does liver and leg meat.

Labunska et al. (2014) found that the intake of PBDEs for children and adults was greater via consumption of duck meat than via consumption of duck liver. Chan et al. (2013) also indicated that chicken meat contributed more to the intake of PBDEs for adults than chicken offal (e.g., liver and intestines). These previous findings suggest that the difference in accumulation of PBDEs may be species-dependent, which is also corroborated by the present study. Estimated average daily $\sum_8$PBDE intakes for adults (319 ng day^{-1}) and children (1380 ng day^{-1}) via consumption of chicken liver contributed the most to the total intakes (adults: 634 ng day^{-1}; children: 2320 ng day^{-1}) in the present study, highlighting the importance of chicken liver for human dietary exposure to PBDEs. This is particularly significant for the general population in countries such as Korea, France, and China, where liver is widely consumed (Hoffmeister et al., 2007; Xing et al., 2010).

The levels of exposure to individual PBDE congeners (BDE-28, 47, 99, 100, 153, 154, 183, and 209) in both the contaminated and control groups for adults and children (Tables 2 and S2) are below the reference dose values promulgated by the United States Environmental Protection Agency (100, 100, 100, 100, 200, 200, 200, and 7000 ng kg^{-1} bw day^{-1}, respectively) (United States Environmental Protection Agency, 2014). The estimated mean level of exposure to BDE-99, the most toxic BDE congener with reproductive and neurodevelopmental impairment, is 0.03–0.15 ng kg^{-1} bw^{-1} day^{-1} for children and 0.01–0.05 ng kg^{-1} bw^{-1} day^{-1} for adults in the control group, below the no adverse effect level (0.23–0.30 ng kg^{-1} bw^{-1} day^{-1}) for impaired spermatogenesis proposed by Netherlands researchers (Bakker et al., 2008). However, the same exposure level for the contaminated group, 31–47 ng kg^{-1} bw^{-1} day^{-1} for children and 7.2–16 ng kg^{-1} bw^{-1} day^{-1} for adults, well exceeds the no adverse effect level. This underscores the potential adverse effects on human health arising from exposure to PBDEs via consumption of chicken if fed with contaminated feed in farms.

4. Conclusions

The present study showed that BDE-209 was the most abundant congener in all chicken tissues among the target analytes, and liver contained the highest PBDE concentrations during the entire exposure period. Tissue concentrations of $\sum_8$PBDE followed the order: liver > blood > skin > intestine > stomach > leg meat > breast meat. The occurrence and relative abundances of BDE-28, 47, 99, 100, 153, 154, 183, and 209 in chicken tissues may be dictated by the tissue partition coefficients and perfusion rates of

Table 2
The estimated daily intake (EDI; ng kg^{-1} bw day^{-1}) for children and adults via the consumption of chicken meat and liver in the contaminated group.

EDI	BDE-28	BDE-47	BDE-99	BDE-100	BDE-153	BDE-154	BDE-183	BDE-209	$\sum_8$PBDE
Children									
leg meat	NA [a]	0.33	47	0.06	31	NA	0.14	553	632
breast meat	NA	0.45	35	NA	33	NA	0.23	242	311
liver	NA	0.72	31	NA	23	NA	0.08	1330	1380
Adult									
leg meat	NA	0.11	16	0.02	10	NA	0.05	185	211
breast meat	NA	0.15	12	NA	11	NA	0.08	81	104
liver	NA	0.17	7.2	NA	5.3	NA	0.02	306	319

[a] Not available, because their corresponding concentrations were lower than the reporting limits (0.025 ng g^{-1}).

PBDEs in the blood. Only a small portion of BDE-209 was reductively debrominated to BDE-47, 99, 100, and 183. The absorption efficiency of BDE-209 in chicken tissues was in the order of liver > skin > intestine > breast meat > leg meat > stomach. In the contaminated group, estimated average daily intake of $\sum_8$PBDE via consumption of chicken for adults and children followed the sequence of liver > leg meat > breast meat. The $\sum_8$PBDE intakes via consumption of chicken liver, leg meat, and breast meat were greater for children than for adults. Liver poses higher exposure risk for humans than does leg meat and breast meat. The estimated intakes of BDE-28, 47, 99, 100, 153, 154, 183, and 209 for adults and children are below the reference dose values established by the United States Environmental Protection Agency, but the mean estimated intake of BDE-99, most toxic congener among all, for the contaminated group well exceed the no adverse effect level. Therefore, the potential human health via consumption of chicken fed by PBDEs-contaminated feeds should not be overlooked.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.envpol.2017.08.084.

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