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Investigation into levels of chlorinated and brominated organic pollutants in carcass fat, offal, eggs and milk produced in Ireland

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TABLE OF CONTENTS

SUMMARY

The Food Safety Authority of Ireland FSAI in collaboration with the Department of Agriculture, Fisheries and Food have carried out a study of levels of chlorinated and brominated organic pollutants, some of which are known to be persistent (POPs), in carcass fat, liver, eggs and milk produced in Ireland. The study was undertaken because of awareness about the possible effects on human health of these (biopersistent) environmental contaminants, known to be present in a number of foodstuffs, notably meat, fish, eggs and dairy products and to fulfill EC monitoring requirements in this area. Furthermore the aim of this study also was to proactively monitor the Irish food supply for emerging new contaminants, namely the brominated flame retardants and related compounds, with a view to aid national and international efforts in the management of these contaminants.

The study showed that levels of chlorinated dioxins, furans and dioxin like PCBs in Irish produce were well below existing legal limits with the exception of one sheep liver sample, however, a possible change of legislative limits for animal offal are currently under discussion. Concentrations of upperbound Total WHO TEQs for PCDD/Fs and dl- PCBs expressed on a ng/kg fat basis ranged from 0.2-0.7 in milk, 0.1-0.9 in carcass fat, 0.2-0.7 in eggs and 0.3-16.4 in liver.

Brominated Dioxins and Furans were detected infrequently at very low levels (<1 ng/kg fat), and in general the occurrence of brominated furans was more pronounced than brominated dioxins.

Of the brominated flame retardants covered in this survey, PBDEs and HBCD were detected in the majority of samples, Polybrominated Biphenyls were detected infrequently and Tetrabromobisphenol A, Hexabromobenzene, Bis(2,4,6tribromophenoxy)ethane and Decabromodiphenylethane were not observed at levels above the Limit of Detection.

PBDEs were found in all matrices and the occurrence pattern is consistent with the congeners found in commercial penta-formulations. Liver and egg samples contained the highest upperbound concentrations for the sum of 17BDEs with medium values of 1.1 and 1.3 µg/kg fat respectively whereas milk contained the lowest concentrations with a median concentration of 0.6 µg/kg fat. Carcass fat concentrations ranged from 0.5-1.4µg/kg fat excluding one sample with an extreme value of 25.3µg/kg fat. The latter is currently subject to follow up investigation.

Total HBCD median concentrations observed were 0.7 µg/kg fat in eggs, 1.3 µg/kg fat in milk, 2.4 µg/kg fat in liver and 0.5 µg/kg fat in carcass fat.

Levels observed for chlorinated dioxins, furans and biphenyls and PBDEs are in line with those from previous FSAI studies. Chlorinated dioxins, furans and PCB levels in Irish food are relatively low compared with similar products from more industrialised countries in the European Union and exposure of consumers of Irish food to chlorinated dioxins and furans in food is below the maximum tolerable monthly intake. Exposure to PBDE is within similar range as observed in other EU countries. Although no safe level of exposure has been established for PBDEs so far, exposure to these chemicals is unlikely to cause any adverse effects based on toxicity studies in animals, which showed neuro-developmental effects at concentrations approximately 8,000 times higher than the estimated population intake for consumers of Irish produce.

Exposure to brominated dioxins, furans and some biphenyls, which are likely to incrementally add to the Total TEQ is low and unlikely to significantly increase total dioxin body burden.

Overall, the survey shows that Irish produce contains low amounts of the persistent bio-accumulative toxicants measured in this survey, and levels observed do not raise concern for human health. However, with the ban of all PBDE commercial flame retardant mixtures in the European Union, the use and production of alternative substances is predicted to increase and future monitoring programs should closely monitor any trends in Irish produce.

ABBREVIATIONS

Abbreviation	Full Name
Ah receptor	aryl hydrocarbon (Ah) receptor
b.w.	body weight
BTBPA	Bis(246-tribromophenoxy)ethane
Boxplot	Boxplot showing the median, quartiles, and outlier and extreme values for a scale variable. The interquartile range (IQR) is the difference between the 75th and 25th percentiles and corresponds to the length of the box. ○: Outliers are values between 1.5 IQR's and 3 IQR's from the end of a box *: Values more than 3 IQR's from the end of a box are defined as extreme.
congener	a chemical substance related to another
DAFF	Department of Agriculture, Fisheries and Food
DBDPE	Decabromodi-phenylethane
dl PCB	dioxin-like PCB
EC	European Community
EFSA	European Food Safety Authority
FSAI	Food Safety Authority of Ireland
HBCD	Hexabromocyclododecane
HBH	Hexabromobenzene
HSE	Health Service Executive
JECFA	FAO/WHO Joint Expert Committee Food Additives and Contaminants
K_{ow}	The octanol-water partition coefficient is the ratio of the concentration of a chemical in octanol and in water at equilibrium and at a specified temperature.
LOD	Limit of Detection
LOQ	Limit of Quantification/Quantitation
Lower-bound	Analytical results reported below the LOD set at zero
MI	Marine Institute
ng	nanogram (0.000000001 g)
PBB	polybrominated biphenyl
PBDD	polybrominated dibenzo-*p*-dioxins
PBDEs	polybrominated diphenylethers
PBDF	polybrominated dibenzofurans
PCB	polychlorinated biphenyl
PCDD/F	abbreviation for PCDDs and PCDFs
PCDDs	polychlorinated dibenzo-*p*-dioxins
PCDFs	polychlorinated dibenzofurans
pg	picogram (0.000000000001 g)
ppb	parts per billion (equal to ng/g or µg/kg)
PTMI	Provisional Tolerable Monthly Intake
SCF	Scientific Committee of Food
SFPA	Sea Fisheries Protection Agency
TBBPA	Tetrabromobisphenol A
TDI	Tolerable Daily Intake
TEF	toxic equivalency factor
TEQ	toxicity equivalent
TWI	Tolerable Weekly Intake
Upper-bound	Analytical results reported below the LOD set at the LOD value
w.w.	wet weight or whole weight
µg	microgram (0.000001 g)
Σ	Sum
Σ7PCB	7 marker PCBs

BACKGROUND

The Food Safety Authority of Ireland (FSAI) mission is to ensure the safety of food consumed, distributed, produced and sold on the Irish market. In this respect, the FSAI co-ordinates the collation of food safety surveillance information from laboratories run by the Official Agencies under service contract to the Authority. These include the Health Service Executive (HSE), the Department of Agriculture, Fisheries and Food, the Sea Fisheries Protection Agency, the Marine Institute and the local authorities. The FSAI also conducts targeted food safety surveillance in areas where potential safety issues have been identified. This report provides the results of a targeted surveillance study on levels of chlorinated dioxins (PCDDs) and furans (PCDFs), brominated dioxins (PBDDs) and furans (PBDFs), polychlorinated biphenyls (PCBs), polybrominated biphenyls (PBBs), polybrominated diphenyl esters (PBDEs), hexabromocyclododecane enantiomers (HBCD Enantiomers), Decabromodiphenyl ethane, Hexabromobenzene, Bis(2,4,6-tribromophenoxy)ethane and Tetrabromo-bisphenol A (TBBP-A) in carcass fat, offal, eggs and milk produced in Ireland.

The study builds on previous studies undertaken by FSAI into levels of PCDD/Fs, PCBs and PBDEs in milk, fish/fish oils, meat, eggs and fish[1, 2, 3,,4,,5] and was undertaken against the background of increased awareness in the European Union of the possible health risks posed by these substances in the food chain. It also reflects Ireland's participation in the 2004 – 2006 EC monitoring programme for the background presence of dioxins, furans and dioxin-like PCBs in foodstuffs which has been agreed between the European Commission and the Member States via Commission Recommendation 2004/705/EC. This study further includes compounds recommended by EFSA in 20066 for inclusion in the core group of brominated flame retardants (BFRs), as part of a European monitoring programme for feed and food, due to the production volumes, the occurrence of the chemical compounds in food and feed, their persistence in the environment and their toxicity.

Polychlorinated dibenzodioxins and polychlorinated dibenzofurans

The term 'dioxins' covers a group of 75 polychlorinated dibenzo-p-dioxin (PCDDs) and 135 polychlorinated dibenzofuran (PCDFs) congeners, of which 17 are of toxicological concern. Exposure to dioxins can result in a wide range of toxic responses, including dermal toxicity (chloracne), immunotoxicity, carcinogenicity, reproductive toxicity and possible neurobehavioral (cognitive) effects. Studies on children exposed in utero to dioxins are reported to have shown endocrine and developmental changes, persisting for long periods. The toxicological effects of the dioxins are thought to arise due to binding of the dioxins to a specific receptor protein in the cells, the aryl hydrocarbon (Ah) receptor present in most tissues of animals and humans. The most toxic dioxin congener is 2, 3, 7, 8-tetrachlordibenzo-p-dioxin (TCDD) and is classified by the International Agency for Research on Cancer (IARC) and other international organisations as a known human carcinogen. By analogy other dioxins are therefore considered as presumed carcinogens. The EU Scientific Committee for Food ("SCF"), in line with the World Health Organization ("WHO"), have concluded however that the carcinogenic effect of dioxins does not occur at levels below a certain threshold.

Dioxins and furans are environmental contaminants and have no commercial applications, other than for preparation of analytical standards and research materials. They are formed during combustion processes when the element chlorine is present, for example in the incineration of municipal waste, although natural combustion processes such as forest fires and bonfires also result in dioxin formation. They can also occur as by-products of industrial processes, for example production and use of pentachlorophenol-containing wood preservatives, production and use of certain herbicides and bleaching of paper pulp using chlorine. Dioxins have been identified in almost all environmental compartments in industrialised countries, as a result of these emissions. Emissions to air result in deposition in the terrestrial environment and in aquatic sediments, followed by uptake into the food chain e.g. by ruminants and by fish. Dioxins are highly resistant to degradation processes in the environment and consequently persist in the environmental compartments where they have been deposited. This is due to their lipophilic characteristics, and also results in accumulation in the fatty tissues of the primary intake species, e.g. cows or fish. Approximately 90% of human exposure to these compounds results from the consumption of contaminated food. Exposure by other routes, such as inhalation and ingestion of particles from air, ingestion of

contaminated soil and dermal absorption normally contributes less than 10% of daily intake.

Because humans are the ultimate receivers in the food chain, there is a significant potential for accumulation of dioxins in human tissues as a result of exposure via food. In the case of cows or other lactating species, high levels of dioxins can potentially occur in milk, specifically in milk fat and consequentially also in cream and in milk products such as cheese, in addition to carcass meat. In fish, high levels may be found in fatty tissues such as liver and consequently in fish liver oils. In Europe, the fraction of the dietary intake of dioxins contributed by these foods is: fish and fish products: 2 – 63 %; meat and meat products: 6 – 32 %; milk and dairy products: 16 – 39 %. Fruit and vegetables provide only a minor contribution to human intake[7].

The Belgium dioxin crisis in 1999 triggered an increased awareness in the European Union of the dangers posed by dioxins, furans and polychlorinated biphenyls in the food chain and as a consequence of this crisis, the European Community (EC) established maximum levels for dioxins in furans in foodstuffs.

Polychlorinated Biphenyls

The polychlorinated biphenyls or PCBs are a group of extremely stable aromatic chlorinated compounds which, like the dioxins, are resistant to biological degradation and hence persist and accumulate in the environment and in the food chain. There are 209 possible PCB compounds, with one to ten chlorine atoms per molecule. They have excellent electrical and heat transfer properties, which led to their widespread use in a variety of industrial, commercial and domestic applications. The production and use of PCBs has been discontinued in most countries, due to concern about their toxicity and persistence, but large amounts remain in electrical equipment, plastic products, buildings and the environment. Disposal of such material results in continued release to the environment, adding to existing levels present as a consequence of past releases.

As a class, PCBs are generally regarded as having potentially adverse effects on health, with particular concern being expressed about the 12 so-called dioxin-like PCBs. This group of non-ortho (PCBs 77, 81, 126, 169) and mono-ortho (PCBs 105, 114, 118, 123, 156, 157, 167, 189) PCBs are assumed to have essentially the same toxicity profile as the dioxins and furans, since they also bind to the Ah receptor. Other PCBs (non-dioxin-like PCBs) do not exert their toxicological effects via binding to the Ah receptor but nonetheless are associated with a wide spectrum of toxic responses in toxicological studies, including developmental effects, immuno- and neurotoxicity, endocrine disrupting effects and tumour promotion. They have been evaluated, inter alia, by the International Programme on Chemical Safety (IPCS), who noted that the PCB congener pattern found in food, human tissues and the environment is different from that of commercial PCB mixtures on which the majority of toxicological studies have been carried out. The so-called marker or indicator PCBs (i.e. PCBs 28, 52, 101, 118, 138, 153 and 180) are detected in these media using readily applicable analytical techniques and have been used as indicators of the total PCB content or body burden of environmental biota, food and human tissue.

Toxic equivalence factors and Tolerable Intakes for PCDD/Fs and dioxin-like PCBs

The toxicity of PCDD, PCDF and the dioxin-like PCB congeners are expressed using toxic equivalence factors (TEFs) (see Table 1 and Table 2) representing the relative toxicity of the compound being measured to the most toxic congener, TCDD. This in turn reflects the relative strength of binding to the Ah receptor. It should be noted however that the toxicity of many of these substances, both dioxins and PCBs, has not been extensively evaluated. An arbitrary TEF of 1 is assigned to TCDD, and by multiplying the analytically determined amounts of each congener in a sample by the corresponding TEF and summing the contribution from each congener the total TEQ value of the sample can be obtained using the following equation:

$$TEQ = (PCDDi \times TEFi) + (PCDFi \times TEFi) + (\text{dioxin-like } PCBi \times TEFi)$$

Several different TEF schemes have been proposed. For many years the most widely used schemes were that of NATO/CCMS[8], giving the so-called International TEFs (I-TEFs) for PCDDs and PCDFs and the WHO-ECEH (European Centre for Environment and Health of the World Health Organization) scheme for PCBs. In 1998,

WHO-ECEH proposed a new scheme of WHO-TEFs for PCDDs, PCDFs and dl-PCBs, which to date has been the most commonly used scheme9. Dioxin TEQ values for food and human samples based on WHO-TEFs are approximately 10-20% higher than those obtained by using the I-TEFs of NATO/CCMS. WHO has recently re-evaluated the WHO-TEFs proposed in 199810 and has adjusted the TEFs for a number of compounds. The results provided in this report are however based on the 1998 scheme for WHO-TEFs.

Table 1 TEFS FOR DIOXINS

PCDDs and PCDFs	Toxic Equivalency Factor (TEF)		
	I-TEF	WHO-TEF 1998	WHO-TEF 2005
2,3,7,8-TCDD	1	1	1
1,2,3,7,8-PnCDD	0.5	1	1
1,2,3,4,7,8-HxCDD	0.1	0.1	0.1
1,2,3,6,7,8-HxCDD	0.1	0.1	0.1
1,2,3,7,8,9-HxCDD	0.1	0.1	0.1
1,2,3,4,6,7,8-HpCDD	0.01	0.01	0.01
OCDD	0.001	0.0001	0.0003
2,3,7,8-TCDF	0.1	0.1	0.1
1,2,3,7,8-PnCDF	0.05	0.05	0.03
2,3,4,7,8-PnCDF	0.5	0.5	0.3
1,2,3,4,7,8-HxCDF	0.1	0.1	0.1
1,2,3,6,7,8-HxCDF	0.1	0.1	0.1
1,2,3,7,8,9-HxCDF	0.1	0.1	0.1
2,3,4,6,7,8-HxCDF	0.1	0.1	0.1
1,2,3,4,6,7,8-HpCDF	0.01	0.01	0.01
1,2,3,4,7,8,9-HpCDF	0.01	0.01	0.01
OCDF	0.001	0.0001	0.0003

Abbreviations: PnCDD, pentachlorodibenzo-p-dioxin; HxCDD, hexachlorodibenzo-p-dioxin; HpCDD, heptachlorodibenzo-p-dioxin; OCDD, octachlorodibenzo-p-dioxin; PnCDF, pentachlorodibenzofuran; HxCDF, hexachlorodibenzofuran; HpCDF, heptachlorodibenzofuran; OCDF, octachlorodibenzofuran.

Table 2 TEFS FOR DIOXIN-LIKE PCBS

PCBs (IUPAC No. in parenthesis)	Toxic Equivalency Factor (TEF)		
	I-TEF	WHO-TEF 1998	WHO-TEF 2005
Non-*ortho* PCBs			
3,3',4,4'-TCB (77)	0.0005	0.0001	0.0001
3,4,4',5-TCB (81)	-	0.0001	0.0003
3,3',4,4',5-PnCB (126)	0.1	0.1	0.1
3,3',4,4',5,5'-HxCB (169)	0.01	0.01	0.03
Mono-*ortho* PCBs			
2,3,3',4,4'-PnCB (105)	0.0001	0.0001	0.00003
2,3,4,4',5-PnCB (114)	0.0005	0.0005	0.00003
2,3',4,4',5-PnCB (118)	0.0001	0.0001	0.00003
2,3,4,4'5-PnCB (123)	0.0001	0.0001	0.00003
2,3,3',4,4',5-HxCB (156)	0.0005	0.0005	0.00003
2,3,3',4,4',5'-HxCB (157)	0.0005	0.0005	0.00003
2,3',4,4',5,5'-HxCB (167)	0.00001	0.00001	0.00003
2,3,3',4,4',5,5'-HpCB (189)	0.0001	0.0001	0.00003
Di-*ortho* PCBs			
2,2',3,3',4,4',5-HpCB (170)	0.0001	0.0001	-
2,2',3,4,4',5,5'-HpCB (180)	0.00001	0.00001	-

Abbreviations: TCB, tetrachlorobiphenyl; PnCB, pentachlorobiphenyl; HxCB, hexachlorobiphenyl; HpCB, heptachlorobiphenyl.

Risk assessment of PCDD/Fs and PCBs in food

The SCF carried out a risk assessment of dioxins and dioxin-like PCBs in food, and concluded that the Tolerable Weekly Intake (TWI) for PCDDs, PCDFs and dioxin-like PCBs should be no more than 14pg WHO-TEQ/kg body weight (b.w.)[11]This is very similar to the Provisional Tolerable Monthly Intake (PTMI) of 70pg/kg b.w. per month, as calculated by the FAO/WHO Joint Expert Committee on Food Additives and Contaminants (JECFA)[12.] It has been stated11 that the European average dietary intake is 1.2 to 3.0pg WHO-TEQ/kg b.w./day, which translates into a weekly intake of between 8.4 and 21pg WHO-TEQ/kg b.w. The upper end of this range exceeds the TWI established by the SCF.

However, several studies carried out by the Food Safety Authority of Ireland (FSAI) have shown that levels of dioxins in Irish food are relatively low1, 2, 3, 4, 5. Hence, it is likely that the exposure of the Irish population to dioxins in food is less than the European average. This conclusion is supported by the results of a study carried out by FSAI on levels of PCDDs, PCDFs and dioxin-like PCBs in breast milk from Irish mothers. This study was reported as part of an international WHO survey 3.

A risk assessment for the non-dioxin-like PCBs (ndl-PCBs) in food has also been carried out recently at European level by the Scientific Panel on Contaminants of the European Food Safety Authority (EFSA), to include identification of the most relevant/sensitive toxicological endpoints for the PCB-congener patterns usually found in food[13]. The Panel concluded that the current toxicological database on health effects is not suitable for the separate assessment of ndl-PCBs. Also the human data on exposure did not enable a distinction between the effects of ndl-PCB and PCDD/F to be made, due to co-occurrence of PCDDs and PCDFs, and therefore the

assessment was based on individual ndl-PCB congeners. Due to the absence of mutagenicity the establishment of a health-based guidance value for levels of ndl-PCBs in food was considered possible, however, the Panel considered the toxicological database too limited and hence a "Margin of Exposure" (MoE)1 approach was used. This approach, which can be used to assess the risks to human health of exposure to a substance in absence of a tolerable daily intake or similar guidance value, has recently been endorsed by the EFSA Scientific Committee14 and the WHO/FAO Joint Expert Committee on Food Additives and Contaminants[15.] A rather small margin of exposure of 10 was calculated, however, the Panel stressed that the endpoints considered in the evaluation of individual ndl-PCB congeners can also be observed with PCDD/F and dl-PCB. Overall, the Panel concluded that further research and additional data is needed to better evaluate adverse effects from ndl-PCBs and a continuing effort to lower the levels of ndl-PCB in food is warranted.

Legislation on PCDD/Fs and PCBs in food

Given that the weekly average dietary intake of dioxins by at least some of the European population exceeds the TWI established by the SCF, on a European scale it is desirable to reduce the exposure of the population to dioxins. In 2001 the European Commission published its Community strategy for dioxins, furans and polychlorinated biphenyls, aimed at achieving a reduction in human exposure to dioxins and PCBs. Environmental legislation designed to limit dioxin emissions is in the process of discussion at European level. Other source-directed measures have been introduced to reduce the contamination of feedingstuffs for animal nutrition (Commission Directive 2006/13/EC amending Annexes I and II to Directive 2002/32/EC of the European Parliament and of the Council on undesirable substances in animal feed as regards dioxins and dioxin-like PCBs).

In addition, as part of its reduction strategy the E.C. in 2001 introduced Maximum Levels for PCDDs and PCDFs in foodstuffs. Maximum levels for the sum of dioxins and dioxin-like PCBs have been set in 2006 as this is the most appropriate approach from a toxicological point of view. In order to ensure a smooth transition, the levels for dioxins continue to apply for a transitional period in addition to the levels for the sum of dioxins and dioxin-like PCBs. Foodstuffs must comply during that transitional period with the maximum levels for dioxins and with the maximum levels for the sum of dioxins and dioxin-like PCBs. Considerations are currently being given of whether to keep or dispense with the separate maximum levels for dioxins and a decision is likely to be made in 2010.

A recent overhaul of the contaminants legislation led to consolidation of existing contaminant legislation and was published in 2006. Regulation 1881/2006/EC as amended by Regulation 1126/2007/EC contains maximum levels for certain contaminants in foodstuffs. The currently applicable maximum levels for PCDDs, PCDFs and sum of PCDD/Fs and DL-PCBs in food are shown in Table 3.

[1] The margin of exposure is defined as the reference point on the dose-response curve (usually based on animal experiments in the absence of human data) divided by the estimated intake by humans. (EFSA 2005b).

Table 3 Maximum Levels for dioxins, furans and dioxin-like PCBs in food

FOOD	Maximum levels Sum of dioxins and furans (WHO-PCDD/F-TEQ) (1)	Maximum levels Sum of dioxins, furans and dioxin-like PCBs (WHO-PCDD/F-PCB-TEQ) (1)
5.1.1 Meat and meat products (2) - of ruminants (bovine animals, sheep) - of poultry and farmed game - of pigs 5.1.2 Liver and derived products of terrestrial animals	 3 pg/g fat(3) 2 pg/g fat (3) 1 pg/g fat (3) 6 pg/g fat (3)	 4.5 pg/g fat(3) 4 pg/g fat(3) 1.50 pg/g fat(3) 12 pg/g fat(3)
5.2 Muscle meat of fish and fishery products and products thereof with the exception of eel(4) (5) - Muscle meat of eel (*Anguilla anguilla)* and products thereof	 4 pg/g whole weight 4 pg/g whole weight	 8 pg/g whole weight 12 pg/g whole weight
5.3 Milk(6) and milk products, including butter fat	3 pg/g fat(3)	6 pg/g fat(3)
5.4 Hen eggs and egg products (7)	3 pg/g fat(3)	6 pg/g fat(3)
5.5 Oils and fats - Animal fat - of ruminants - of poultry and farmed game - of pigs - mixed animal fats - Vegetable oil and fats - marine oil (fish body oil, fish liver oil and oils of other marine organisms intended for human consumption)	 3 pg/g fat 2 pg/g fat 1 pg/g fat 2 pg/g fat 0.75 pg/g fat 2 pg/g fat	 4.5 pg/g fat 4 pg/g fat 1.5 pg/g fat 3 pg/g fat 1.5 pg/g fat 10 pg/g fat

(1) Upperbound concentrations: Upperbound concentrations are calculated on the assumption that the values of the different congeners below the limit of quantification are equal to the limit of quantification.

(2) Meat of bovine animals, sheep, pig, poultry and farmed game as defined in Annex I to Regulation (EC) No 853/2004 of the European Parliament and of the Council (OJ L 139, 30.4.2004. Corrected version in OJ L 226, 25.6.2004, p. 22) but not including edible offal as defined in that Annex.

(3) The maximum levels are not applicable for food products containing < 1 % fat.

(4) Muscle meat of fish and fishery products as defined in categories (a), (b), (c), (e) and (f) of the list in Article 1 of Council Regulation (EC) No 104/2000 (OJ L 17, 21.1.2000, p. 22. Regulation as amended by the 2003 Act of Accession). The maximum level applies to crustaceans, excluding the brown meat of crab and excluding head and thorax meat of lobster and similar large crustaceans (Nephropidae and Palinuridae) and to cephalopods without viscera.

(5) Where fish are intended to be eaten whole, the maximum level applies to the whole fish.

(6) Milk (raw milk, milk for the manufacture of milk-based products and heat-treated milk as defined in Annex I to Regulation (EC) No 853/2004.

(7) Hen eggs and egg products as defined in Annex I to Regulation (EC) No 853/2004.

Legislation to cover non dioxin-like PCBs is currently being discussed at EC level and is likely to be adopted in 2010.

Brominated Flame Retardants

Brominated flame retardants are a group of chemicals which are added to many household products for the purpose of fire prevention. The types of products containing these chemicals include clothing and household textiles, furniture, computers and TVs. There are 20-25 classes of BFRs in present production, with at least three major classes: tetrabromobisphenol A (TBBPA) and its derivatives, polybrominated diphenyl ethers (PBDEs) and hexabromocyclododecane (HBCD, including three isomers). Polybrominated phenols, decabromodiphenyl ethane and brominated phthalic acid derivatives are another three chemicals or classes of chemicals in use. This survey covers PBDEs, HBCD, TBBPA, Decabromodiphenyl ethane, Hexabromobenzene and Bis(2,4,6-tribromophenoxy)ethane.

Concern has been raised by the occurrence of several chemical compounds from the group of BFRs in the environment, including feed and food, and in human tissues and fluids. This has led to bans on the production and use of two technical products of polybrominated diphenyl ethers (PBDEs), penta-brominated diphenyl ether and octa-brominated diphenyl ether, within the EU.

In recent risk assessments it is acknowledged that the currently available occurrence data on brominated flame retardants in feed and food do not allow a comprehensive assessment of contamination in all feeds and foods6.

Polybrominated diphenyl ethers

The term polybrominated diphenyl ethers (PBDEs) refers to three commercial mixtures of decabromodiphenyl ether (DBDE), octabromodiphenyl ether (Octa, OBDE), and pentabromodiphenyl ether (Penta, pentaBDE). The European Union16 has banned production of both pentaBDE and octaBDE in 2004, however decaBDE (DBDE) is still in use. However, recently, the European Court of Justice (ECJ) on 1st April 2008 annulled the exemption to the EU Directive on the Restriction of the Use of Certain Hazardous Substances in Electrical and Electronic Equipment 2002/95/EC, commonly referred to as the Restriction of Hazardous Substances Directive or RoHS Directive as of 30 June 2008 that was granted in 2005 for Deca-BDE.

In contrast to congeners present in the PentaBDE (mainly BDE 47, BDE 99, BDE 100) and OctaBDE (mainly BDE 183) technical mixtures, it was assumed that BDE-209, the main constituent of decaBDE, is not readily bioavailable, e.g., via dietary exposure as it is not easily absorbed through the gut. Its bioavailability was assumed to be low because of its high molecular weight and hydrophobicity[17]. Based on these characteristics, DecaBDE did not appear to meet traditional criteria used to identify persistent, bioaccumulative and toxic (PBT) substances[18]. However, there is increasing evidence as to its persistence, bioaccumulation and debromination to lower brominated congeners [19, 20, 21, 22, 23, 24]

The PBDEs are similar in structure to the PCBs (polychlorinated biphenyls) and also have some similarities to the dioxin family of chemicals, but they are not dioxin-like. They contain the element bromine rather than the chlorine element found in the PCBs. Like the dioxins and PCBs, the PBDEs break down slowly in the environment and in living organisms including the human body. Continuous exposure to them leads to build-up in the body. Because they have similarities to dioxins and PCBs, they may have some of the same effects on health as these chemicals, although they appear to be less toxic. Recent toxicological studies have shown that some of them are endocrine or hormone disruptors, an effect that is also associated with the dioxins and PCBs, and is thought to be associated with changes in fertility, sexual development and possibly certain types of cancer such as breast, testicular and prostate cancer. It has also been reported that they can have an effect on brain development in mice, slowing the learning process. As with PCBs, exposure to PBDEs may be particularly harmful during a critical window of brain development during pregnancy and early childhood. While the pentabromo compounds appear to be the most toxic, many of these persistent chemicals have not been extensively studied.

PBDEs were first reported in wildlife species, including fish, seals, whales and birds' eggs. In the late 1990's they were reported in the breast milk of mothers in Sweden, and research showed that levels had increased from zero

in 1970 to high levels in the 1990's in parallel with the use of PBDEs.

The EC is currently considering the establishment of maximum limits for these chemicals in food and is encouraging Member States to carry out measurements to assist in this process.

The FSAI has carried out a number of studies looking for the presence of PBDEs[1,2,3,4.]

Levels in Irish eggs were found to be approximately 0.1 parts per billion (ppb) in total egg. The approximate level expressed in ppb per kg egg fat was 1 ppb. In 2004 the Marine Institute in Ireland[25] measured PBDEs in Irish farmed fish and found levels ranging from 2.28 to 4.61 (mean 3.05) ppb w.w. and 0.7 to 1.8 (mean 1.17) ppb w.w. for the sum of the 17 individual PBDEs and for total HBCD respectively. These findings were further confirmed by a survey conducted in 2004. Samples surveyed in 2005 showed a mean level of 0.6 ppb fat in milk, 0.9 ppb fat in liver and levels ranging from 0.3 – 7.5 ppb in food supplements including fish oil supplements, which were at the higher end of the range.

A Tolerable Intake Level (Tolerable Daily or Weekly Intake, TDI/TWI) has not been determined for the PBDEs by expert bodies such as the European Food Safety Authority, because there is as yet insufficient information available on their toxicity and their occurrence in food. Because of the lack of information, FSAI considers that exposure to them should be minimised.

Hexabromocyclododecane

Hexabromocyclododecane (HBCD) has primarily been used to improve flame retardant characteristics of extruded and expanded polystyrene products. Technical HBCD comprises three diasteroisomers (α, β and γ), with γ-HBCD contributing approximately 80% to the technical formulation. However, in biological samples a dominance of the alpha isomer can be observed[26, 27, 28.] In accordance with this observation trophic magnification was observed for the alpha-diastereoisomer as concentrations increased with increasing trophic level in the food web, whereas there was trophic dilution of gamma-HBCD through the food web[29].

As its use may increase, as alternative BFRs such as commercial BDE mixtures are phased out, its detection in a wide range of matrices is a potential environmental and consumer food safety concern.

Studies in laboratory animals have shown that, following oral administration, HBCD can be detected in adipose tissue, liver and muscle and recent studies indicate that HBCD has the potential to bioaccumulate[30, 31].

It has been implicated as a developmental neurotoxicant[32, 33,] enzyme inducer[34], and there is increasing evidence for its endocrine disrupting properties[35, 36, 37, 38, 39] in laboratory animals. A recent study describes the changes in rat hepatic gene profiles and also suggests a sex-specific toxicity with females being more sensitive to HBCD than males[40], which is also observed in other studies[34,35].

Isomer specific HBCD levels were analysed in fish samples landed/farmed in Ireland in 2004 and levels of total HBCD (sum of α-, β- and γ-HBCD) of 1.17 ± 0.26 µg/kg fresh weight in farmed salmon were in agreement with those of a previous Marine Institute survey4,[41]). Also, average levels for total HBCD observed in herring, salmon and mackerel samples are similar to those found in recent Latvian and UK surveys[42,43.]

A Tolerable Intake Level (Tolerable Daily or Weekly Intake, TDI/TWI) has not been determined for the HBCD by expert bodies such as the European Food Safety Authority, because there is as yet insufficient information available on its toxicity.

Polybrominated Biphenyls

The term polybrominated biphenyls (PBBs) refers to a group of halogenated hydrocarbons, formed by substituting hydrogen by bromine in biphenyl. PBBs are not known to occur as natural products. Theoretically, 209 congeners

are possible. Only a few have been synthesized individually and characterized. PBBs, manufactured for commercial use, consist mainly of hexa-, octa-, nona, and decabromobiphenyls, but also contain other homologues. They are additive type flame retardants, and when blended with the dry solid or liquid polymeric material, provide filter-type, flame retardant action with the chemical release of hydrogen bromide if ignited.

PBBs were introduced as flame retardants in the early 1970s and used mainly in small appliance and automotive applications, coatings, lacquers, and polyurethane foam.

The commercial production of FireMaster(R) was started in the USA in 1970 and contained 60-80% of hexabromobiphenyl. After the Michigan disaster, when this compound was inadvertently added to animal feed instead of magnesium oxide and resulted in the destruction of thousands of cattle, pigs, and sheep, and millions of chickens, production was discontinued[44]. Production of decabrominated biphenyl was discontinued in Europe after 2000[45].

In 2003 PBBs were listed as one of six controlled substances under the Restriction of Hazardous Substances Directive (ROHS), which restricts their use in electrical and electronic equipment[46.]

Most of the PBB congeners are persistent, lipophilic and bioaccumulating, which represents a potential threat to both human and environmental life[44].

Most toxicity studies of PBBs in animals have involved oral exposure, and numerous effects have been documented including hepatic, renal, dermal/ocular, immunological, neurological, and developmental effects. Other effects of oral exposure to PBBs include decreased thyroid function, body weight loss, and liver cancer. Adverse hepatic effects, as well as dermal and ocular effects, also have been observed in a limited number of dermal studies in animals. No significant adverse effects were observed in animal inhalation studies of PBBs, but only two studies have been conducted, and the mixtures that were tested (octabromobiphenyl and decabromobiphenyl) are not the lower brominated products (i.e., Firemaster mixtures) expected to be the most toxic based on oral data. A number of PBB effects are dioxin-like and consistent with the Ah receptor-mediated mechanism of action, including altered vitamin A homeostasis, thymic atrophy, dermal and ocular effects (e.g., chloracne and inflammation of eyelids), and body weight changes (wasting syndrome)[47]. PBB occurrence in top predator wildlife species has recently been reported and stresses the need to further identify present human background exposure to PBBs. Thus, inclusion of certain PBB congeners in the TEF scheme is appropriate, but further human exposure analysis is required[9].

Tetrabromo-Bisphenol A

Commercial Tetrabromo-Bisphenol A (TBBPA) is the brominated flame retardant produced in the largest amounts globally. The demand for TBBPA and its derivatives accounts for over 60 000 tonnes per year. TBBPA is used as a reactive (primary use) or additive flame retardant in polymers, such as ABS, epoxy and polycarbonate resins, high impact polystyrene, phenolic resins, adhesives, and others.

Because of its partition coefficient and low water solubility, TBBPA in the environment is expected to adsorb to a large extent onto sediment and organic matter in the soil[48].

TBBPA is relatively less persistent and bio-accumulative than most of the other flame retardants[49] and the risk for the general population from TBBPA exposure has been previously considered insignificant by the WHO in 1995[48] and by the European Commission in 2006[50], however, since then several papers have been published that would increase the level of concern regarding possible risks of TBBPA. Notably the papers of Van der Ven et al.[51] and Lilienthal et al.[52] have demonstrated an interaction with the thyroid hormone system in rats, mediated via its competitive binding to transthyretin (TTR) and resulting in decreased circulating thyroxin (T4) and increased triiodothyronine (T3). The dose at which the most sensitive of these effects was seen was 0.5 mg/kg bw/day, and the authors point out that the Margin of Exposure between this and predicted or actual measured levels for some

human populations is low, additionally indicating concern for human health.

The above studies and other papers published on TBBPA[53, 54, 55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66, 67, 68] since 2006 overall indicate that TBBPA has endocrine disruption potential. It shows developmental and neurological effects, and at high doses is hepatotoxic and nephrotoxic. At the cellular level it disrupts calcium homeostasis and also increases free radicals, resulting in cytotoxicity. Toxicokinetic studies in the rat have shown that it behaves very similarly to BPA.

Decabromodiphenyl ethane, Hexabromobenzene and Bis(2,4,6-tribromophenoxy)ethane

These compounds were included in this survey following a recommendation by EFSA in 2006[6] for inclusion in the core group of brominated flame retardants (BFRs) of a European monitoring programme for feed and food. Very little is known regarding production volumes, occurrence in food and feed, persistence in the environment and toxicity of these compounds.

In 2007 the European Commission commissioned a "review on production processes of Decabromodiphenyl Ether (DecaBDE) used in polymeric applications in electrical and electronic equipment, and assessment of the availability of potential alternatives to DecaBDE"[69], and decabromodiphenyl ethane and Bis(tribromophenoxy)ethane were identified as two of 27 alternative substances.

Decabromodiphenyl ethane

Decabromodiphenyl ethane can be used in a wide range of high performance applications. In particular it finds use in styrenic polymers, engineering resins, wire & cable and elastomers[70] .

The compound is expected not to be an environmental concern because of its large molecular size and low water solubility, however, similar assumptions had been made for deca-BDE, which has shown to be bioavailable and recently, some reports have emerged on the presence of decabromodiphenyl ethane in the environment in Europe[71, 72] and the US[73] .

Very limited toxicological data are available. A report prepared by the German Federal Environment Agency in 2001[74] cites an LD50 of >5000mg/kg in rats and a long term toxicity study with a NOEL of 1000mg/kg. No evidence of mutagenicity or teratogenicity has been reported, but no studies on carcinogenicity were conducted. Based on this information the Federal Environment Agency advocated the application of a safety factor of 1000 in derivation of a potential ADI of 1000µg/kg bw.

Hexabromobenzene

Historically, hexabromobenzene (HBB) was a widely used BFR in Japan as an additive to paper, plastic and electronic goods. It is still in use today albeit in much lower quantities[75]. According to the European chemical substance information system (ESIS), HBB is not reported by the EU industry as a currently produced chemical, however, occurrence in top predator wildlife species has recently been reported and stresses the need to further identify present human background exposure[18, 19, 20, 21, 22, 23].

Bis(2,4,6-tribromophenoxy)ethane (BTBPE)

BTBPE, another alternative to octa or deca PBDE has been detected in the US and Canada in air[76], aquatic species, sediment and water[77, 78], tree bark[79], eggs of herring gulls[80] and in Europe in northern fulmars[81] and Glaucous Gulls[82]. These findings provide evidence on its persistence and possible accumulation in the environment. A recent study further supports that this chemical has a high potential for biomagnification in aquatic food webs[83].

A review by Hakk et. al. in 2003[84] concluded that based on the limited pharmacokinetic and solubility studies, mammalian absorption of BTBPE via ingestion would be minimal. However, there is some indication that

inhalation might quite possibly be the major route for human and wildlife exposure. This is based on a study[85] conducted in Sweden in 2001 where BTBPE levels in an electronics recycling plant rivalled those of much higher production BFRs, e.g. TPPBA and BDE 209. However, more research is needed to clearly identify the main exposure routes.

Legislation on Brominated Flame Retardants

There are currently no EU maximum limits for BFRs in food. Tolerable daily intakes (TDIs) have not been derived, primarily due to limited toxicological data for BFRs and the associated uncertainties with such studies. Considerably work is required internationally on the toxicology and risk assessment of BFRs. Also the use of most flame retardants is not regulated however, Table 4 shows existing application restrictions in the EU for a number of brominated flame retardants.

Table 4 Regulatory overview of EU Legislation on brominated flame retardants

Legal basis:	Content:	Deadline for compliance:	BFR substance concerned
WEEE Directive	Separation of BFR plastics from E&E* equipment prior to recovery and recycling	December 2006	All BFRs used in E&E
RoHS Directive	Ban of use in new E&E applications	1 July 2006	Penta-BDE Octa-BDE PBB
	Exempted from a ban in E&E applications from 15 October 2005 until 1st July 2008**		Deca-BDE
EU Directive establishing the list of priority substances in the field of water policy	Establishment of controls of emissions, discharges and losses in the Environment and water quality standards	Not applicable	Deca-BDE Octa-BDE
	Cessation of emissions in the Environment	2020	Penta-BDE
24th amendment to the marketing and use Directive 76/769/EEC	Ban of use in all applications for the EU Market	15 August 2004	Penta-BDE Octa-BDE
4th amendment to the marketing and use Directive 76/769/EEC	Ban from textile applications in the EU Market	November 1984	PBB (not produced since 2000)

* *E&E – electrical and electronic*

(Table adapted from Bromine and Environmental Science Forum[86])

Polybrominated dibenzodioxins and polybrominated dibenzofurans

Polybrominated dibenzo- p-dioxins (PBDDs) and polybrominated dibenzofurans (PBDFs) are almost planar tricyclic aromatic compounds. Theoretically, seventy five PBDD and one hundred and thirty five PBDF chemical structures are possible. In addition, a large number of mixed halogenated congeners – 1550 brominated/chlorinated dibenzo- p-dioxins (PXDDs) and 3050 brominated/chlorinated dibenzofurans (PXDFs) -- are theoretically possible. The most toxic congeners are those that are substituted at positions 2, 3, 7, and 8. There are seven 2,3,7,8-substituted PBDDs and ten 2,3,7,8-substituted PBDFs, as well as three hundred and thirty seven possible 2,3,7,8-substituted PXDDs and six hundred and forty seven possible 2,3,7,8-substituted PXDFs[87]..

PBDDs/PBDFs so far are not known to occur naturally, however, a recent report[88] suggest natural production of PBDDs by algae and/or cyanobacteria in the Baltic, which accumulate in the marine foodchain. Generally, they are not intentionally produced (except for scientific purposes) but are generated as undesired by-products in various processes. They can be formed by chemical, photochemical, or thermal reactions from precursors and by so-called de novo synthesis[87].

PBDDs/PBDFs have been found as contaminants in brominated organic chemicals (e.g. bromophenols) and, in particular, in flame retardants, such as polybrominated diphenyl ethers (PBDEs), decabromobiphenyl (decaBB or DBB), 1,2-bis(tribromophenoxy)ethane, tetrabromobisphenol A (TBBPA), and others. They have been detected in distillation residues of some bromophenols and bromoanilines and in wastes from chemical laboratories[87].

PBDFs and, to a lesser extent, PBDDs have been detected as photochemical degradation products of brominated organic chemicals, such as PBDEs and bromophenols[87].

In comparison to their chlorinated homologs, the PBDDs/PBDFs have higher molecular weights, higher melting points, lower vapour pressures, lower water solubility and a higher octanol-water partition coefficient (log K0W values). Although the PBDDs/PBDFs are more lipophilic and less water soluble than their chlorinated counterparts, they appear to be less environmentally persistent and more sensitive to UV degradation, possibly because bromine is a better leaving group than chlorine. The biochemical properties of the dioxins and furans are also altered by the bromine atom, since the larger size of the bromine atom alters susceptibility to enzymatic attack, and the carbon-bromine bond has lower strength than the carbon-chlorine bond. Little is known about their pharmacokinetic and pharmacodymatic properties, and inferences are mostly drawn from the chlorinated analogs [89]. Essentially all of the classic effects demonstrated for TCDD and the other chlorinated dioxins and furans – lethality, wasting, thymic atrophy, teratogenesis, reproductive effects, chlor-acne, immunotoxicity, enzyme induction, decreases in T4 and Vitamin A, and increased hepatic porphyrins – have been observed in the limited toxicological studies available[87]. Overall, the limited data base on the health effects of PBDDs and PBDFs supports the hypothesis that these brominated congeners have similar biological properties to their chlorinated relatives. With the increasing use and the environmental presence of brominated flame retardants, it is likely that human, as well as wildlife exposure to these chemicals will increase. Given the common mechanisms of action (binding to the Ah receptor) and effects, it is reasonable to predict that their presence will incrementally add to the total dioxin body burden[89].

Polybrominated dibenzo-p-dioxins and dibenzofurans and certain other individual and groups of compounds were identified for possible future inclusion in the TEF concept, including 3,4,4'-TCB (PCB 37), mixed polyhalogenated dibenzo-p-dioxins and dibenzofurans, polyhalogenated naphthalenes, and polybrominated biphenyls. However, more relative effect potency (REP) studies are needed, before a TEF system for PBDD/Fs can be established[9].

MATERIALS AND METHODS

Study outline

The present study was undertaken to investigate the current levels of dioxins, furans, PCBs and a number of BFRs in carcass fat, offal, eggs and milk and thereby increase the available data pool on the occurrence of these contaminants in these foodstuffs.

For this survey the following types of food samples were collected (Table 5)

Table 5 Food Samples included in this survey

Species	Matrix	Number of Samples	Number of sub-samples per sample
Avian	Fat	13	10-40
	Liver	3	10-40
	Eggs	20	24
Bovine	Fat	9	10
	Liver	2	10
	Milk	32	1
Ovine	Fat	10	10
	Liver	3	10
Porcine	Fat	6	10-20
	Liver	2	10
Equine	Liver	2	10

With the exception of 2 lamb's liver samples, all samples were supplied by officers of the Department of Agriculture, Fisheries and Food at production level (slaughterhouse: fat and liver, farm/dairy tanker: milk, packing station: eggs) and the remainder taken by officers of the Food Safety Authority of Ireland at retail level.

Analysis of the samples was undertaken by the Central Science Laboratory, York, UK, during 2006-2008 under contract to FSAI.

Analytes included in the survey

Chlorinated Compounds

17 PCDD/PCDF congeners

2,3,7,8-TCDD	2,3,7,8-TCDF
1,2,3,7,8-PeCDD	1,2,3,7,8-PeCDF
1,2,3,4,7,8-HxCDD	2,3,4,7,8-PeCDF
1,2,3,6,7,8-HxCDD	1,2,3,4,7,8-HxCDF
1,2,3,7,8,9-HxCDD	1,2,3,6,7,8-HxCDF
1,2,3,4,6,7,8-HpCDD	1,2,3,7,8,9-HxCDF
OCDD	2,3,4,6,7,8-HxCDF
	1,2,3,4,6,7,8-HpCDF
	1,2,3,4,7,8,9-HpCDF
	OCDF

PCB congeners

PCB 77 (dl-PCB)	PCB 153 (Marker PCB)	PCB 110
PCB 81 (dl-PCB)	PCB 180 (Marker PCB)	PCB 141
PCB 126 (dl-PCB)	PCB 18	PCB 151
PCB 169 (dl-PCB)	PCB 31	PCB 167
PCB 105 (dl-PCB)	PCB 33	PCB 183
PCB 114 (dl-PCB)	PCB 37	PCB 185
PCB 123 (dl-PCB)	PCB 41	PCB 187
PCB 156 (dl-PCB)	PCB 44	PCB 189
PCB 157 (dl-PCB)	PCB 47	PCB 191
PCB 167 (dl-PCB)	PCB 49	PCB 193
PCB 189 (dl-PCB)	PCB 51	PCB 194
PCB 118 (dl-PCB/ Marker PCB)	PCB 60	PCB 201
PCB 28 (Marker PCB)	PCB 66	PCB 203
PCB 52 (Marker PCB)	PCB 74	PCB 206
PCB 101 (Marker PCB)	PCB 87	PCB 209
PCB 138 (Marker PCB)	PCB 99	

Brominated Compounds

PBDE congeners

BDE-17	BDE-77	BDE153
BDE-28	BDE-85	BDE138
BDE-47	BDE-99	BDE 154
BDE-49	BDE-100	BDE-183
BDE-66	BDE-119	BDE-209
BDE-71	BDE-126	

HBCD Enantiomers

Alpha-HBCD
Beta-HBCD
Gamma-HBCD

PBB congeners

PBB 77 (3,3',4,4')	PBB 52 (2,2',5,5')
PBB 126 (3,3',4,4',5)	PBB 80 (3,3',5,5')
PBB 169 (3,3',4,4',5,5')	PBB 101 (2,2',4,5,5')
PBB 15 (4,4')	PBB 153 (2,2',4,4',5,5')
PBB 49 (2,2',4,5')	PBB 209 (2,2',3,3',4,4',5,5',6,6')

PBD/F congeners

2,3,7-TriBDD	2,3,8-TriBDF
2,3,7,8-TetraBDD	2,3,7,8-TetraBDF
1,2,3,7,8-PentaBDD	1,2,3,7,8-PentaBDF
1,2,3,4,7,8-/1,2,3,6,7,8-HexaBDD	2,3,4,7,8-PentaBDF
1,2,3,7,8,9-HexaBDD	1,2,3,4,7,8-HexaBDF
	1,2,3,4,6,7,8-HeptaBDF

Other BFRs

Decabromodiphenyl ethane
Hexabromobenzene
Bis(2,4,6-tribromophenoxy)ethane
Tetrabromo-bisphenol A (TBBP-A)

Analytical methods

Sample preparation

The pooled samples were provided frozen by the Food Safety Authority of Ireland and sent to CSL. The homogenates of the samples were freeze-dried by the laboratory and further homogenized by means of grinding.

Sample Analysis

The analytical methodology for dioxin and PCB analysis at CSL followed EU Directive 2002/69/EC laying down the methods of analysis for the determination of dioxin-like PCBs in foodstuffs, as amended by Commission Directive 2004/44/EC. All analyses were carried out using 13Carbon labelled internal standards and measurement was made using both, high-resolution GC-MS and low-resolution GC-MS as appropriate. Brominated dioxins (PBDD/Fs) PBDEs and brominated biphenyl (PBB) analysis was carried out using similar methodology as the chlorinated dioxins and PCBs – ie HRGC-HRMS and 13C-labelled internal standardisation[90]. HBCD enantiomers and tetrabromo-bisphenol Analysis was by LC-MS/MS using 13C-labelled internal standards[91].

Methodology for other 3 brominated compounds specified by EFSA – Hexabromobenzene, Decabromodiphenylethane, and Bis(246 tribromophenoxy) ethane was similar to that for chlorinated dioxins and PCBs.

The samples were fortified with either 13C hexabromobenzene, 13C bis-(2,4,6-tribromophenoxy) ethane or 13C decabromodiphenylethane, homogenised and extracted on a bed of modified silicas using mixed solvents. The extracts were concentrated, treated with acid and purified using florisil, before concentration to final volume and addition of syringe standard. Analytes were then measured using high resolution gas chromatography/high resolution mass spectrometry and quantified using the 13C-labelled internal standards.

RESULTS

PCDD/Fs and PCBs

Table 6 presents summary information on the levels of PCDD/Fs, dioxin-like PCBs and indicator PCBs measured during this study.

Results are expressed as total WHO-TEQs in ng/kg fat weight for PCDD/Fs and dioxin-like PCBs separately and for the sum of PCDD/Fs and dioxin-like PCBs together, and as the sum total in µg/kg fat weight for the sum of 7 and sum of 6 indicator PCBs[2]. In each case results are presented as upper-bound values, substituting values below the analytical limit of quantification with the limit of quantification (<LOQ=LOQ).

Table 6 Upper-bound levels (<LOQ = LOQ) of PCDD/Fs, dioxin-like PCBS and TOTAL TEQS, and sum of 7 and sum of 6 Indicator PCBs

Product	Category	Statistics	*PCDD/F*	*dl- PCBs*	*Σdl PCBs & PCDD/F*	*Σ*PCB 6	*ΣPCB s7*
			WHO TEQs ng/kg fat			µg/kg fat	
Eggs	Barn	Mean	0.17	0.11	0.27	7.81	7.94
		Med	0.18	0.12	0.29	2.99	3.14
		Min	0.13	0.07	0.20	0.62	0.75
		Max	0.19	0.15	0.29	30.62	30.78
		P97.5	0.19	0.14	0.29	27.93	28.09
	Cage	Mean	0.20	0.12	0.30	0.85	1.00
		Med	0.18	0.10	0.28	0.53	0.64
		Min	0.17	0.08	0.25	0.37	0.48
		Max	0.24	0.23	0.39	2.38	2.62
		P97.5	0.24	0.22	0.38	2.20	2.42
	Free Range	Mean	0.23	0.14	0.35	1.08	1.25
		Med	0.22	0.14	0.33	0.90	1.02
		Min	0.20	0.10	0.29	0.82	0.95
		Max	0.29	0.22	0.42	1.72	2.02
		P97.5	0.29	0.21	0.42	1.66	1.94
	Organic	Mean	0.34	0.22	0.55	0.93	1.10
		Med	0.25	0.23	0.49	0.73	0.86
		Min	0.19	0.19	0.36	0.70	0.82
		Max	0.52	0.25	0.72	1.32	1.58
		P97.5	0.52	0.25	0.72	1.31	1.56
Milk	Dairy	Mean	0.16	0.20	0.37	0.48	0.57
		Med	0.16	0.18	0.34	0.39	0.47
		Min	0.11	0.07	0.22	0.26	0.31
		Max	0.27	0.53	0.66	1.95	1.99
		P97.5	0.26	0.42	0.65	1.25	1.40

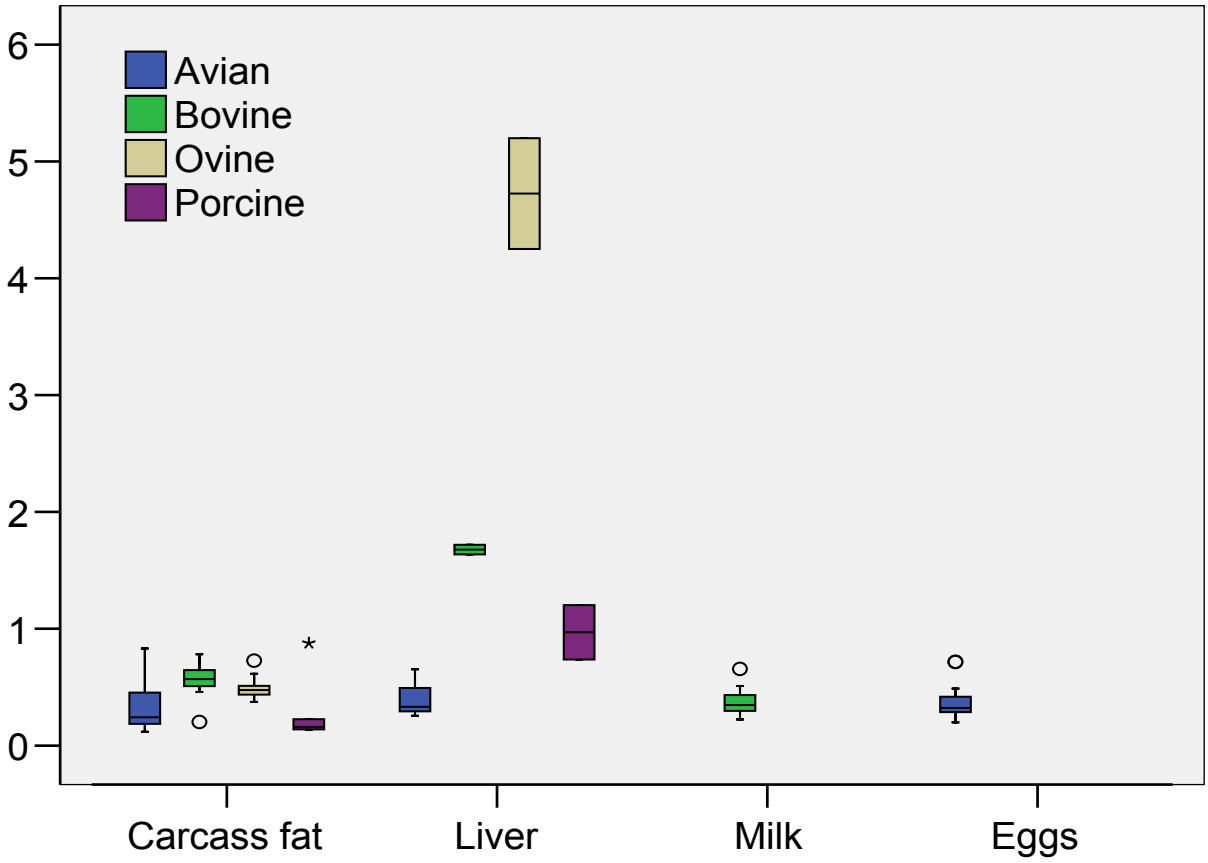
[2] Sum of 6 non-dioxin like Marker PCBs: PCBs 28, 52, 101, 138, 153 and 180. Sum of 7 Marker PCBs: Sum of 6 plus PCB 118 (dioxin-like)

Table 6 continued

Product	Category	Statistics	PCDD/F	dl- PCBs	Σdl PCBs&PCDD/F	ΣPCB 6	ΣPCB s7
			WHO TEQs ng/kg fat			µg/kg fat	
Liver	Avian	Mean	0.25	0.16	0.41	1.13	1.18
		Med	0.27	0.08	0.33	1.07	1.13
		Min	0.18	0.07	0.25	0.38	0.41
		Max	0.31	0.35	0.65	1.95	1.99
		P97.5	0.31	0.33	0.64	1.91	1.95
	Bovine	Mean	1.03	0.65	1.68	3.06	3.29
		Med	1.03	0.65	1.68	3.06	3.29
		Min	0.99	0.65	1.64	2.94	3.17
		Max	1.07	0.65	1.72	3.18	3.41
		P97.5	1.07	0.65	1.72	3.17	3.40
	Equine	Mean	11.58	2.68	14.25	4.33	4.75
		Med	11.58	2.68	14.25	4.33	4.75
		Min	4.77	1.31	6.08	2.90	3.24
		Max	18.39	4.04	22.43	5.75	6.26
		P97.5	18.04	3.97	22.02	5.68	6.18
	Ovine	Mean	7.03	1.58	8.61	3.15	3.41
		Med	3.93	1.27	5.20	2.46	2.56
		Min	3.52	0.73	4.25	2.13	2.22
		Max	13.64	2.75	16.39	4.85	5.45
		P97.5	13.16	2.67	15.83	4.73	5.31
	Porcine	Mean	0.78	0.19	0.97	0.52	0.55
		Med	0.78	0.19	0.97	0.52	0.55
		Min	0.60	0.14	0.74	0.46	0.49
		Max	0.96	0.24	1.20	0.57	0.61
		P97.5	0.95	0.24	1.19	0.57	0.61
Carcass fat	Avian	Mean	0.16	0.19	0.35	1.47	1.69
		Med	0.14	0.11	0.24	0.69	0.77
		Min	0.07	0.04	0.12	0.25	0.28
		Max	0.27	0.58	0.83	4.25	5.01
		P97.5	0.26	0.56	0.81	4.12	4.85
	Bovine	Mean	0.27	0.29	0.56	0.92	1.11
		Med	0.27	0.29	0.57	0.85	1.04
		Min	0.11	0.09	0.20	0.68	0.82
		Max	0.39	0.41	0.78	1.34	1.59
		P97.5	0.38	0.41	0.77	1.30	1.55
	Ovine	Mean	0.30	0.20	0.50	0.94	1.02
		Med	0.28	0.19	0.48	0.96	1.04
		Min	0.20	0.17	0.37	0.71	0.81
		Max	0.46	0.27	0.73	1.13	1.25
		P97.5	0.44	0.26	0.70	1.12	1.23
	Porcine	Mean	0.15	0.13	0.28	0.62	0.67
		Med	0.10	0.06	0.16	0.64	0.70
		Min	0.07	0.05	0.13	0.34	0.37
		Max	0.41	0.46	0.88	0.95	1.01
		P97.5	0.38	0.42	0.80	0.93	0.99

Figure 1 provides a graphic overview of total WHO TEQ concentrations (ng/kg fat) found in the different matrices and shows that liver, particularly liver of ruminants contains dioxins in higher concentrations than carcass fat, the other tissue analysed. Horse liver contained the highest concentrations of total WHO TEQ of 22 pg/g fat of any sample tested.

Figure 1 Upperbound total WHO TEQ concentration range in ng/kg fat

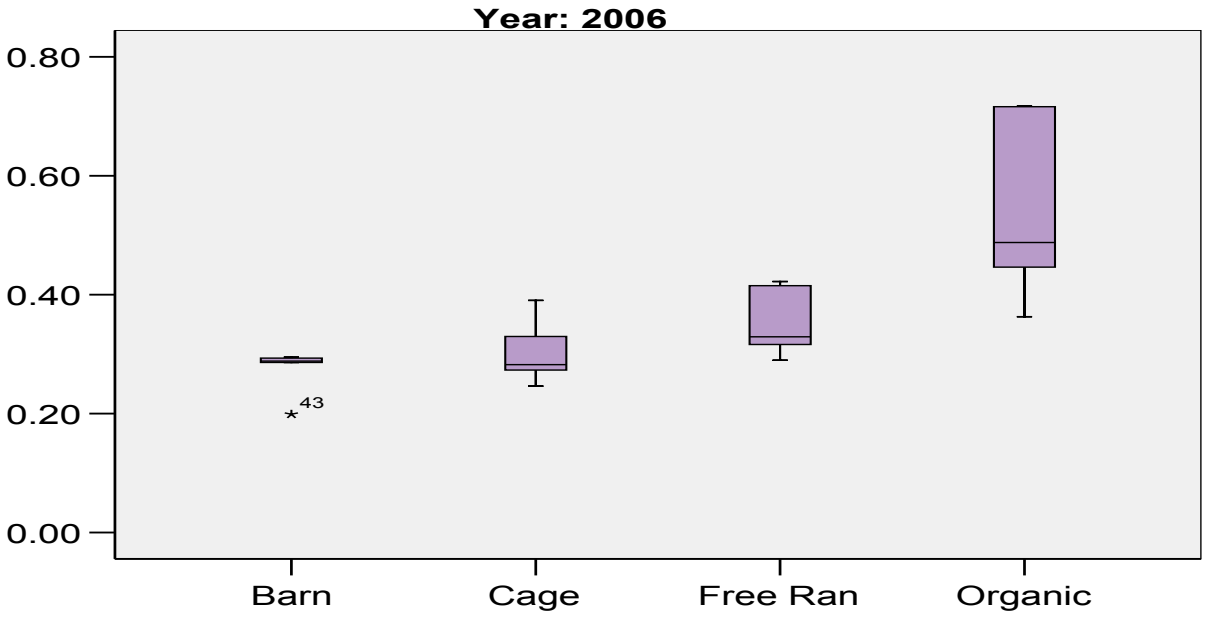


*Note: Boxplot showing the median, quartiles, and outlier and extreme values for a scale variable. The interquartile range (IQR) is the difference between the 75th and 25th percentiles and corresponds to the length of the box. ○: Outliers are values between 1.5 IQR's and 3 IQR's from the end of a box *: Values more than 3 IQR's from the end of a box are defined as extreme.*

Eggs

Figure 2 provides an overview of occurrence levels of total TEQ in eggs according to the relevant production type.

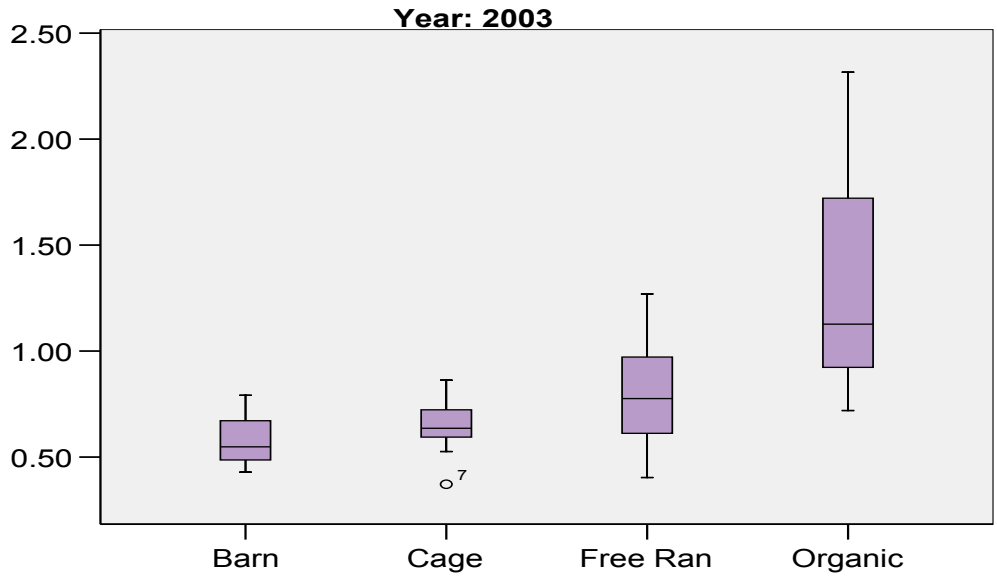
Figure 2 Distribution of total TEQ (ng/kg fat) in 5 samples each of Barn, Cage, Free Range and Organic Eggs observed in this survey (2006)



The lowest levels were observed in barn eggs and the highest levels were observed in organic eggs, however, as these production methods are uncommon in Ireland, each category only represents 3 producers, whereas cage and free range eggs represent 5 different producers. Organic eggs typically contain higher levels than eggs from cage/indoor production as the birds are likely to pick up soil during feeding. The overall range of upperbound total TEQ in eggs ranged from 0.20 – 0.72 ng/kg fat, which is well below the existing legislative limit of 6 ng/kg fat. The levels for sum7 Marker PCBs typically ranged from 0.62 – 3.74 with the exception of one barn egg sample with a total sum7 Marker PCBs of 30.62 µg/kg fat weight. The latter result is atypical and attributable to one congener (PCB 28) and another sample from the same producer taken from a different house showed levels within the norm (< 4 µg/kg fat). There were also no irregularities found in samples from the same producer taken in 2003. The irregularity therefore appears to be isolated to one particular house and follow up investigations with the producer pointed to the use of a particular paint in the egg lobby of the house as source of the isolated high level.

Figure 3 displays levels of total TEQ observed in eggs in a similar study conducted in 2003. The overall pattern of total TEQ levels was comparable for eggs from the different methods of production. Overall a highly significant reduction in total TEQ (P<0.001) can be observed in barn, cage and free range eggs from 2003 to 2006 with an average reduction of 50%. No significant difference (p=0.22) can be observed for organic eggs, however, the sample size in both years was very small, due to the overall small production of organic eggs in Ireland.

Figure 3 Distribution of total TEQ (ng/kg fat) samples of Barn, Cage, Free Range and Organic Eggs observed in a previous survey (2003)



Similar observations were made for the sum of 7 Marker PCBs. Very significant reductions (P<0.005) could be seen for free range and cage eggs, but not for barn and organic eggs. Again this observation has to be considered carefully in the light of the small sample sizes mentioned earlier.

Milk

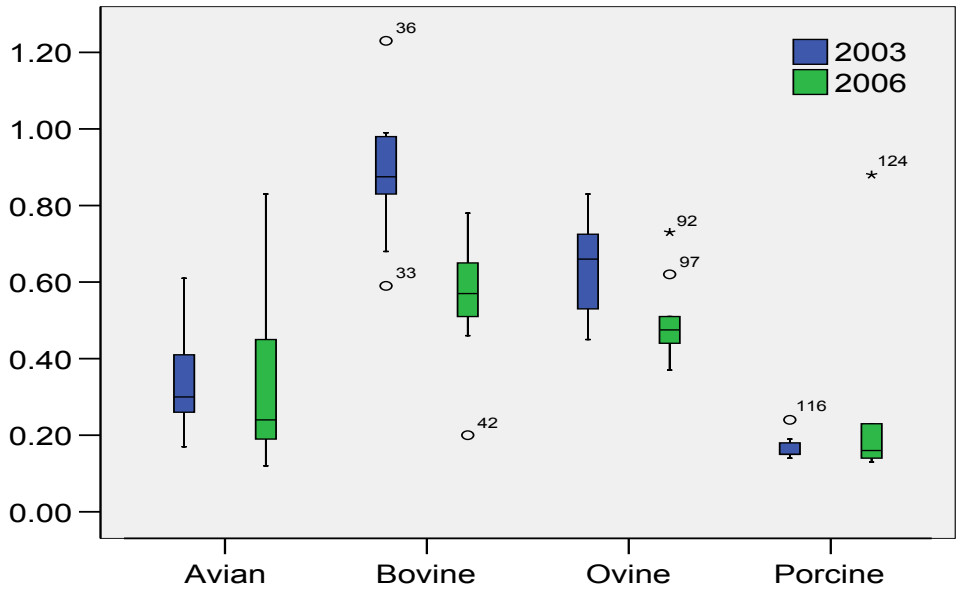
Total TEQ occurrence levels in farm milk ranged from 0.37 – 0.66 pg/g fat, which is well below existing legislative limits of 6 pg/g fat. A survey conducted by FSAI5 in 2005 showed comparable levels ranging from 0.41 – 0.88 ng/kg fat. Another survey conducted by the EPA[92] in 2006 also shows comparable levels ranging from 0.24 – 1 pg/g fat in background samples. Levels of sum7 Marker PCBs ranged from 0.31 – 1.99 µg/kg fat and are also well below currently discussed maximum levels for non-dioxin like PCBs.

Carcass fat

Total TEQ occurrence levels in carcass fat differ according to species. The lowest levels were observed in chicken fat, ranging from 0.12 – 0.83, followed by pork fat, ranging from 0.13 – 0.88, followed by sheep fat, ranging from 0.37 – 0.7 pg/g fat and beef fat, ranging from 0.2 – 0.78 pg/g fat. All observed levels are well below existing legislative limits of 4.5 (bovine, ovine), 4 (avian) and 1.5 pg/g fat (porcine), respectively. Sum of 7 Marker PCBs ranged from 0.28 – 45.01 µg/kg fat in avian fat. The wide range of occurrence of total TEQ as well as Marker PCBs in avian fat is unusual and was only found in samples of two producers and is likely to be attributable to the feed used. Marker PCBs in pork fat ranged from 0.37 – 1, in sheep fat from 0.81 – 1.25 and cattle fat 0.82 – 1.59 µg/kg fat.

A similar study undertaken in carcass fat in 20033 also shows that porcine and avian fat have lower occurrence levels compared to bovine and ovine fat (see Figure 4).

Figure 4 Boxplot showing Total WHO TEQ (upperbound) concentrations in ng/kg fat found in carcass fat, displayed by species for the years 2003 and 2006



Statistical analysis by means of a T-Test of the concentration ranges available for 2003 and 2006 revealed no significant differences in concentrations in total TEQ or sum of marker PCBs for avian and porcine fat, however, statistically significant reductions in concentrations could be observed for bovine and ovine fat, (Total TEQ: p 0.001, p 0.02 respectively, Indicator PCBs: p<0.01 in both cases). The nature of this observation is not clear and could be due to a number of factors, such as inter- and intra-species variation, seasonal/climatic conditions preceding the sampling period or different feeding regimes (poultry and pigs being reared almost entirely on feed, whereas cows are grazing outside during summer season and sheep are grazing outside all year round).

Offal

Total TEQ occurrence levels in liver differ according to species. Total TEQ concentrations in avian liver ranged from 0.25 – 0.65 pg/g fat, in bovine liver from 1.64 – 1.72 pg/g fat, in ovine liver from 4.25 – 16.39 and porcine liver from 0.74 – 1.2 pg/g fat. Two samples of equine liver showed very high levels at 6 and 22 pg/g fat respectively, however, equine liver is not consumed in Ireland and was only included in the survey from a research point of view, in particular to collect information on dioxin concentration patterns in liver of different species.

A maximum legislative limit of 12pg/g fat currently exists for liver of animals of terrestrial origin. Avian, bovine and porcine liver samples readily comply with this limit, however, repeated exceedances have been observed for ovine liver in this and previous surveys and surveys conducted in the UK[93] and Denmark[94]. PCDD/F is the major contributor to this total TEQ and shows that dioxins and furans accumulate in the ovine liver and the wide distribution therefore appears to be age- and origin-related. The same phenomenon has not been observed in cows, which show higher body fat burden and appears to be due to particular metabolism occurring in the sheep. Dioxins are known to bind to aryl hydrocarbon receptor sites and these are largely present in the liver. It is also known that dioxins tend to bind more strongly than PCBs. Ruminants in particular, which include sheep and deer, do not produce large amounts of fat in the liver, which means that the system for transporting dioxins and PCBs from the liver is limited and is likely to favour the less strongly bound PCBs. This would account for the higher dioxin to PCB ratio found in the liver and also why higher levels would be expected in older animals. It also

explains the lack of a relationship between fat content and dioxin content[95].

Due to the latter reason, discussions have commenced in the EC to regulate dioxins and PCBs on a whole weight basis rather than a fat weight basis in ovine liver. With an average 5% fat content, ovine liver contains between 0.2 - 0.8 total TEQ pg/g liver.

Sum of PCB6 ranged from 0.41 – 1.99 in avian liver, 3.17 – 3.41 in bovine liver, 2.22 – 5.45 in ovine liver and 0.49 – 0.61 µg/kg fat in porcine liver. These levels are well below currently discussed maximum limits for non-dioxin like PCBs.

Brominated Flame Retardants

Polybrominated Diphenyl Ethers

Eggs

Of the 17 PBDE congeners analysed, BDEs 100, 183 and 209 could be quantified in all samples. Table 7 shows the percentage of samples in which the respective BDE congeners occurred. No sample showed levels of BDE 17, 71, 77, 119 and 126 above the LOQ.

Table 7 Congener specific occurrence of PBDEs in egg samples (%)

BDE Congener	% quantified Occurrence
100, 183, 209	100
47	90
49, 99, 153	90
66, 154	65
28	15
85, 119, 138	5
17, 71, 77, 126	0

Table 8 presents a data overview of the sum of 16 PBDEs and sum of 17 PBDEs found in the egg samples.

Table 8 Data overview of upperbound concentrations of Sum of 16 PBDE congeners (excluding BDE 209) and Sum of 17 PBDE congeners (including BDE 209) in µg/kg fat weight in eggs

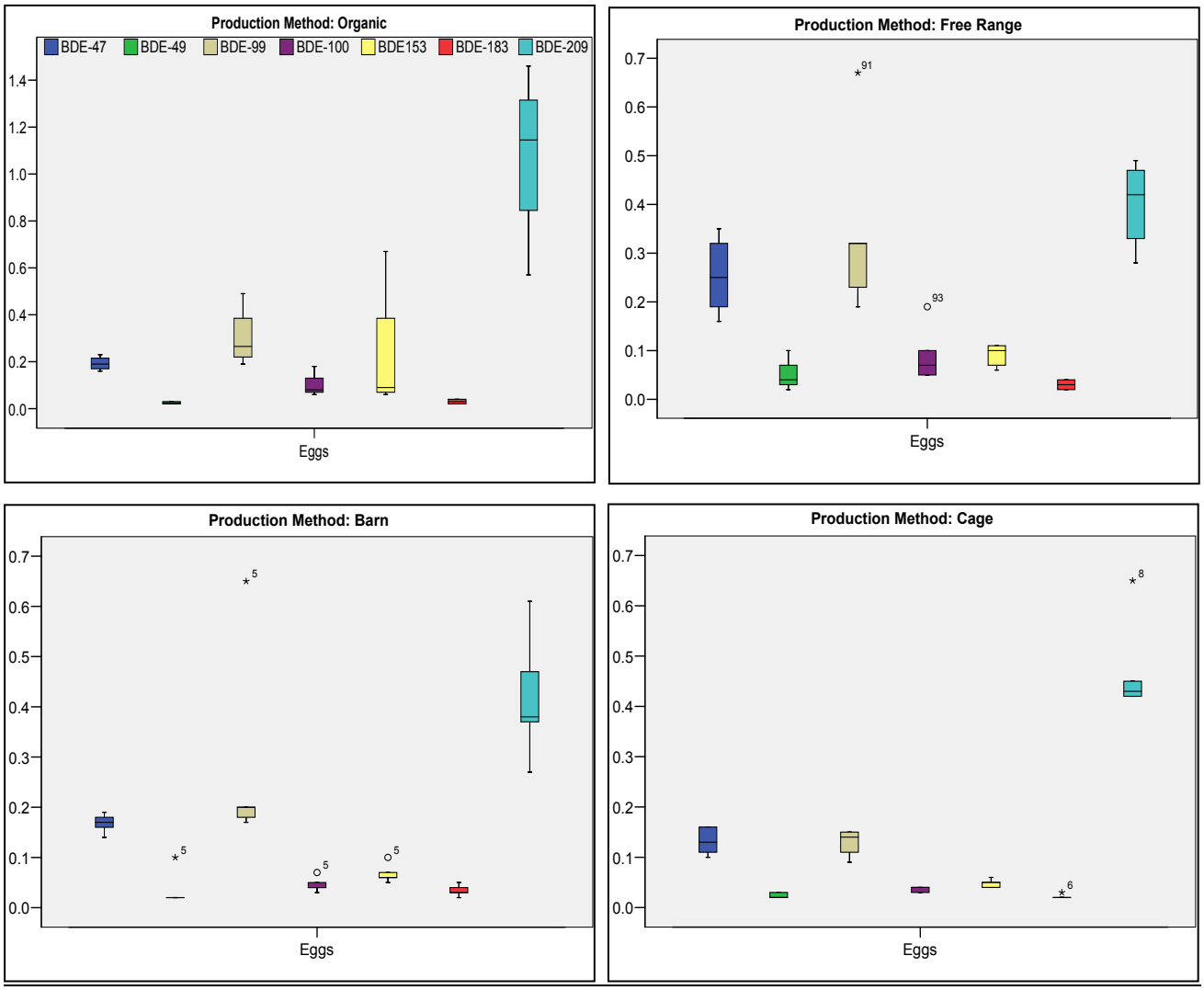
		Sum BDE16	Sum BDE17
Eggs (Barn)	Mean	0.79	1.21
	Med	0.67	1.10
	Min	0.57	0.95
	Max	1.41	1.68
	P97.5	1.337	1.641
Eggs (Cage)	Mean	0.51	0.94
	Med	0.54	0.99
	Min	0.43	0.66
	Max	0.57	1.19
	P97.5	0.54	1.17
Eggs (Free Range)	Mean	1.07	1.47
	Med	0.99	1.46
	Min	0.65	0.98
	Max	1.63	2.12
	P97.5	1.604	2.073
Eggs (Organic)	Mean	0.95	2.59
	Med	0.77	2.23
	Min	0.58	1.46
	Max	1.85	4.43
	P97.5	1.754	4.289

Figure 5 provides an overview of the most commonly detected congeners per egg production type. All egg production categories showed similar occurrence of BDEs, with BDE 209 being the predominant congener in all

production types. Whereas BDE 47, 49, 99, 100 and 183 were within comparable ranges for each production type, BDEs 209 and 153 occurred at much higher concentrations in organic eggs.

As expected, cage/battery eggs showed the lowest concentrations of BDEs overall, which is due to the restricted environment in which the hens are kept.

Figure 5 Boxplot showing upperbound concentration ranges of BDE congeners 47, 49, 99, 100, 153, 183 and 209 in µg/kg fat found in Organic, Free Range, Barn and Cage Eggs



Carcass Fat

Of the 17 PBDE congeners analysed, BDEs 99, 153, 47 and 100 could be quantified in all samples. Table 9 shows the percentage of samples in which the respective BDE congeners occurred. No sample showed levels of BDE 17, 71, 77, 119 and 126 above the LOQ.

Table 9 Congener specific occurrence of PBDEs in carcass fat samples (%)

BDE Congener	% quantified Occurrence
99, 153	100
47, 100	97
154	92
183	74
49	61
66	50
209	32
138	18
85	13
28	3
17, 71, 77, 119, 126	0

Table 10 presents a data overview of the sum of 16 PBDEs and sum of 17 PBDEs found in the carcass fat samples.

Table 10 Data overview of upperbound concentrations of Sum of 16 PBDE congeners (excl BDE 209) and Sum of 17 PBDE congeners (incl BDE 209) in µg/kg fat weight in carcass fat

		Sum BDE16	Sum BDE17
Avian	Mean	0.64	1.16
	Median	0.55	1.23
	Min	0.35	0.53
	Max	1.19	1.61
	P97.5	1.13	1.56
Bovine	Mean	0.74	1.07
	Median	0.48	0.80
	Min	0.28	0.46
	Max	2.87	3.33
	P97.5	2.46	2.92
Ovine	Mean	0.66	1.06
	Median	0.48	0.89
	Min	0.36	0.67
	Max	1.53	1.97
	P97.5	1.41	1.90
Porcine	Mean	0.75	1.19
	Median	0.75	1.20
	Min	0.66	0.92
	Max	0.83	1.35
	P97.5	0.83	1.35

Unlike PCDD/F concentrations, all four species tested showed PBDE occurrence in comparable ranges. Beef was showing the widest range and the highest maximum level of 3.33 µg/kg Sum of 17 BDE, whereas pork was found to have a very narrow range, with the lowest maximum level of 1.35 µg/kg Sum of 17 BDE.

Direct comparison of Sum of 17 BDE with a previous survey conducted in 2003 is not possible due to the advancement in analytical sensitivity in-between the surveys. Limit of detection in this survey improved by a factor of 5-10 for most congeners. However, as BDE congeners 47 and 99 were detected in all samples in 2006 and all but two samples in 2003, comparisons can be made for these two congeners. No statistically significant difference, using a T-test, could be observed between means for the two congeners measured in carcass fat in 2003 and 2006. This is expected, as the EU wide ban of penta- and octaBDE coming into effect in 2006 will take time to result in a decline of occurrence in food.

Offal

Of 17 congeners included in the survey 13 were detectable. Table 11 shows the percentage of liver samples in which the congeners were detected.

Table 11 Congener specific occurrence of PBDEs in liver samples (%)

BDE Congener	% detected Occurrence
47, 99, 183	100
153, 209	83
100	75
66	67
154	58
28	50
49	25
17, 85, 138	17

Table 12 presents a data overview of the sum of 16 PBDEs and sum of 17PBDEs in liver.

Table 12 Data overview of upperbound concentrations of Sum of 16 PBDE congeners (excluding BDE 209) and Sum of 17 PBDE congeners (including BDE 209) in µg/kg fat weight in liver

Product	N	Statistics	Sum BDE UB (excl 209)	Sum BDE UB (incl 209)
Avian	3	Mean	0.67	1.15
		Minimum	0.47	0.85
		Maximum	0.84	1.72
Bovine	2	Mean	0.61	2.44
		Minimum	0.5	1.03
		Maximum	0.72	3.84
Ovine	3	Mean	1.47	2.42
		Minimum	0.51	0.79
		Maximum	2.9	4.34
Porcine	2	Mean	0.64	0.96
		Minimum	0.61	0.75
		Maximum	0.67	1.17

Similarly to carcass fat, concentrations in offal appear to be within similar range irrespective of species, however,

due to the small sample size no firm conclusions can be drawn.

A survey conducted on retail samples in 2005 showed levels ranging from 0.39 – 2.13 µg/kg fat in ovine liver samples, which compares well with the findings of this survey.

Milk

Of 17 congeners only 8 were detectable. Table 13 shows the percentage of milk samples in which the congeners were detected.

Table 13 Congener specific occurrence of PBDEs in milk samples (%)

BDE Congener	% detected Occurrence
47	37
99	30
100, 153	27
154	17
66	7
49, 85	3

Table 14 provides a data overview of occurrence of PBDE congeners in milk taken at farm level. Two out of 30 samples presented considerably higher concentrations and values calculated excluding these two outliers are presented in brackets. Excluding the outlier values, levels in milk for Sum BDE 17 range from 0.31 - 1.06 µg/kg fat. A survey conducted in 2003 found levels ranging from 0.3 – 0.77 µg/kg fat weight for Sum BDE 16, which compares well with values for sum BDE 16 found in this survey (0.22 – 0.73 µg/kg fat).

Table 14 Data overview of upperbound concentrations of Sum of 16 PBDE congeners (excluding BDE 209) and Sum of 17 PBDE congeners (including BDE 209) in µg/kg fat weight in farm milk

Product	N	Statistics	Sum BDE 16	Sum BDE 17
Farm Milk	30	Mean	0.49 (0.34)	0.71 (0.55)
		Median	0.31 (0.28)	0.58 (0.54)
		Std. Deviation	0.6 (0.13)	0.64 (0.17)
		Minimum	0.22 (0.22)	0.31 (0.31)
		Maximum	3.03 (0.73)	3.12 (1.06)
		P97.5	3.03 (0.73)	3.12 (1.06)

Numbers in brackets exclude two outliers

Polybrominated Biphenyls

Table 15 Congener specific occurrence of PBBs (%)

	Carcass Fat	Eggs	Liver	Milk
PBB-153	0	0	8	0
PBB-209	13	100	75	0
PBB-15, 49, 52, 80, 101, 169	0	0	0	0
PBB77	13	21	8	0
PBB126	0	0	58	0

PBBs were only detected in carcass fat, liver and eggs (see Table 15). The predominant congener was PBB-209, which occurred in all egg samples (0.1-2.84 µg/kg fat), half of all avian fat samples (0.03-0.26 µg/kg fat) and in all avian, ovine and porcine liver samples (0.15-0.30 µg/kg fat). PBB-126 occurred in bovine and ovine liver only (0.16-0.78 µg/kg fat). PBB-77 was detected in 5 samples close to the LOD (0.05-0.08 µg/kg fat) and PBB-153 was detected in one ovine liver. All remaining PBB congeners were not detected.

HBCD and TBBPA

With the exception of bovine and porcine Liver, all samples tested contained HBCD, whereas TBBPA was not detected in any sample (<LOD). Table 16 provides an overview of detected occurrence of HBCD isomers and TBBPA as percent of total samples tested.

Table 16 % of Samples in which alpha, beta, gamma, total HBCD or TBBPA was determined above the Limit of Detection

	aHBCD	bHBCD	gHBCD	Sum HBCD	TBBPA
			%		
Milk	13	3	0	13	0
Eggs	30	0	10	35	0
Avian Fat	21	0	7	21	0
Bovine Fat	75	25	38	75	0
Ovine Fat	70	20	10	70	0
Porcine Fat	83	33	33	83	0
Avian liver	33	33	0	33	0
Bovine Liver	0	0	0	0	0
Ovine liver	33	0	0	33	0
Porcine Liver	0	0	0	0	0

Overall, alpha HBCD was the dominant congener, which is in line with other reports in the literature. With the exception of avian fat, carcass fat showed the highest occurrence of Sum HBCD, followed by eggs. Only a third of avian and ovine liver contained HBCD and the lowest percentage of occurrence was found in milk samples at 13%. No sample contained detectable levels of TBBPA.

Detailed results in the following are reported as upperbound values (=LOD) and therefore present a worst-case scenario.

Milk

Table 17 provides an overview of isomer specific HBCD concentrations and TBPPA concentration expressed on a µg/kg fat weight basis. Concentrations in milk ranged from 1.06 – 2.32 µg/kg fat total HBCD, and 0.77 – 7.2 µg/kg of TBBPA, respectively.

Table 17 Data overview of upperbound concentrations of alpha, beta and gamma HBCD and total HBCD and TBBPA expressed as µg/kg fat weight

		aHBCD	bHBCD	gHBCD	Sum HBCD	TBBPA*
		µg/kg fat weight				
Milk (N =30)	mean	0.411	0.311	0.629	1.352	1.419
	med	0.370	0.290	0.600	1.265	0.845
	min	0.310	0.240	0.510	1.060	0.770
	max	0.820	0.530	1.100	2.320	7.200
	P97.5	0.726	0.523	0.955	2.001	6.053

**All results for TBBPA were <LOD but are calculated as upperbound values (=LOD) and reflect the worst case scenario*

Eggs

Table 18 provides an overview of isomer specific HBCD concentrations and TBPPA concentration expressed on a µg/kg fat weight basis. Concentrations in eggs ranged from 0.48 – 11.38 µg/kg fat Total HBCD, and 0.16 – 0.82 µg/kg of TBBPA, respectively. Concentrations in eggs of all production types (Barn, Free Range, Cage, Organic) showed comparable concentrations, with the exception of one Barn Egg, which showed elevated levels of HBCD.

Table 18 Data overview of upperbound concentrations of alpha, beta and gamma HBCD and total HBCD and TBBPA expressed as µg/kg fat weight

		aHBCD	bHBCD	gHBCD	Sum HBCD	TBBPA*
		µg/kg fat weight				
Eggs (N = 20)	mean	0.617	0.238	0.441	1.295	0.287
	med	0.225	0.190	0.190	0.675	0.180
	min	0.130	0.180	0.170	0.480	0.160
	max	6.600	0.480	4.600	11.380	0.820
	P97.5	3.864	0.461	2.624	6.687	0.768

**All results for TBBPA were <LOD but are calculated as upperbound values (=LOD) and reflect the worst case scenario*

Carcass Fat

Table 19 provides an overview of isomer specific HBCD concentrations and TBPPA concentration expressed on a µg/kg fat weight basis. Concentrations in carcass fat ranged from 0.25 - 4.69 µg/kg fat Total HBCD, and 0.087- 6.1 µg/kg of TBBPA, respectively.

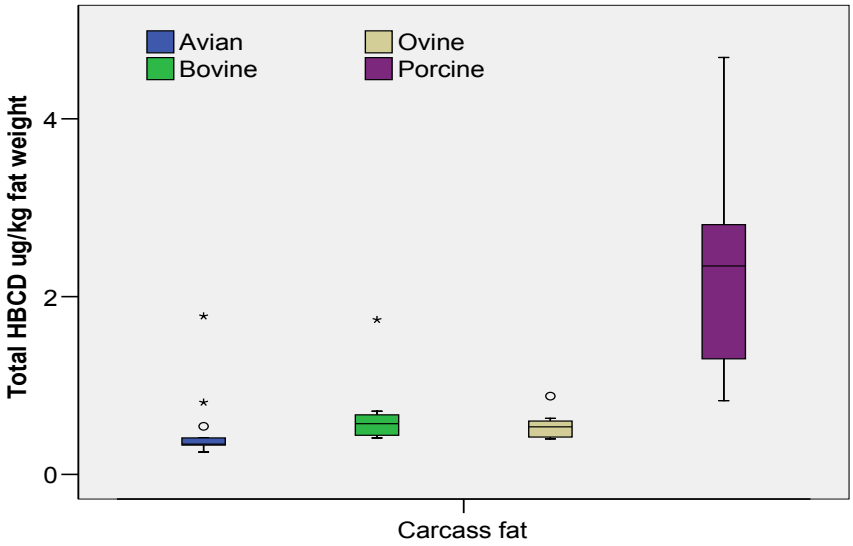
Table 19 Data overview of upperbound concentrations of alpha, beta and gamma HBCD and total HBCD and TBBPA expressed as µg/kg fat weight

		aHBCD	bHBCD	gHBCD	Sum HBCD	TBBPA*
		µg/kg fat weight				
Avian Fat	mean	0.212	0.147	0.140	0.499	0.099
	med	0.110	0.130	0.105	0.370	0.095
	min	0.010	0.120	0.097	0.250	0.087
	max	1.200	0.240	0.420	1.780	0.130
	P97.5	0.901	0.234	0.378	1.465	0.127
Bovine Fat	mean	0.409	0.208	0.146	0.711	1.000
	med	0.230	0.210	0.150	0.600	0.185
	min	0.140	0.120	0.110	0.410	0.096
	max	1.500	0.280	0.180	1.740	6.100
	P97.5	1.322	0.278	0.178	1.560	5.208
Ovine Fat	mean	0.233	0.152	0.156	0.541	0.354
	med	0.215	0.135	0.125	0.535	0.095
	min	0.120	0.120	0.100	0.400	0.086
	max	0.390	0.210	0.450	0.880	1.700
	P97.5	0.372	0.210	0.385	0.824	1.565
Porcine Fat	mean	1.923	0.195	0.268	2.387	0.645
	med	1.850	0.165	0.255	2.345	0.150
	min	0.550	0.150	0.130	0.830	0.100
	max	4.400	0.310	0.440	4.690	3.100
	P97.5	4.125	0.300	0.435	4.455	2.744

**All results for TBBPA were <LOD but are calculated as upperbound values (=LOD) and reflect the worst case scenario*

Porcine fat showed a much wider range of HBCD occurrence levels than the fat samples taken from other species (see Figure 6). This finding suggests either an inter-species variation in HBCD metabolism or higher exposure to HBCD due to a different feed and/or housing scheme used in pig rearing.

Figure 6 Total upperbound HBCD concentration in carcass fat expressed as µg/kg fat



Offal

Table 20 provides an overview of isomer specific HBCD concentrations and TBPPA concentration expressed on a µg/kg fat weight basis. Concentrations in liver ranged from 1.4 – 6.7 µg/kg fat Total HBCD, and 0.49 – 3.8 µg/kg of TBBPA, respectively. Sum of HBCD was comparable for all species with the exception of one slightly elevated bovine liver sample.

Table 20 Data overview of upperbound concentrations of alpha, beta and gamma HBCD and total HBCD and TBBPA expressed as µg/kg fat weight

		aHBCD	bHBCD	gHBCD	Sum HBCD	TBBPA*
		µg/kg fat weight				
Avian liver	mean	0.847	0.810	0.430	2.087	0.603
	med	0.740	0.680	0.370	1.920	0.590
	min	0.700	0.650	0.350	1.790	0.590
	max	1.100	1.100	0.570	2.550	0.630
	P97.5	1.082	1.079	0.560	2.519	0.628
Bovine Liver	mean	1.150	1.280	2.160	4.590	0.990
	med	1.150	1.280	2.160	4.590	0.990
	min	1.000	0.960	0.520	2.480	0.880
	max	1.300	1.600	3.800	6.700	1.100
	P97.5	1.293	1.584	3.718	6.595	1.095
Ovine liver	mean	0.760	0.710	0.383	1.853	0.650
	med	0.780	0.750	0.400	1.930	0.680
	min	0.580	0.530	0.290	1.400	0.490
	max	0.920	0.850	0.460	2.230	0.780
	P97.5	1.082	1.079	0.560	2.519	0.628
Porcine Liver	mean	0.935	1.720	0.770	3.425	2.270
	med	0.935	1.720	0.770	3.425	2.270
	min	0.870	0.940	0.440	3.040	0.740
	max	1.000	2.500	1.100	3.810	3.800
	P97.5	0.997	2.461	1.084	3.791	3.724

**All results for TBBPA were <LOD but are calculated as upperbound values (=LOD) and reflect the worst case scenario*

This is the first time milk, eggs, fat and liver samples have been tested for HBCD and TBBPA in Ireland, therefore no direct comparisons to previous surveys can be made. However, levels for HBCD compare well with background levels established for fish and fishery products in 2004[4].

Hexabromobenzene, Bis(2,4,6-tribromophenoxy)ethane and Decabromodi-phenylethane

This is the first time milk, eggs, fat and liver samples have been tested for HBH, BTBPA and DBDPE in Ireland, and no sample contained levels above the limit of detection (see Table 21).

Table 21 Range of Limit of Detection of HBH, BTPBA and DBDPE in milk, eggs, fat and liver samples expressed as µg/kg fat weight

	Limit of Detection µg/kg fat weight			
	Milk	Carcass Fat	Liver	Eggs
Hexabromobenzene	0.2-0.3	0.01	0.2-0.31	0.1-0.11
Bis(246-tribromophenoxy)ethane	0.2-0.8	0.2-0.9	0.2-2.76	0.2-1.38
Decabromodiphenylethane	0.9-3	1.2-2.7	1.42-7.97	ND-6.01

The LODs, which are comparably higher than those for other analytes covered in this survey, are mainly governed by the levels of ambient concentrations in the laboratories and reagents rather than limited by the methodology applied. This is due to the extended use of these flame retardants in current electric and electronic appliances.

PBBD/Fs

Table 22 provides an overview of the percentage of samples in which PBDD/Fs were detected above the Limit of Detection.

Table 22 Congener specific occurrence of PBDD/Fs (%)

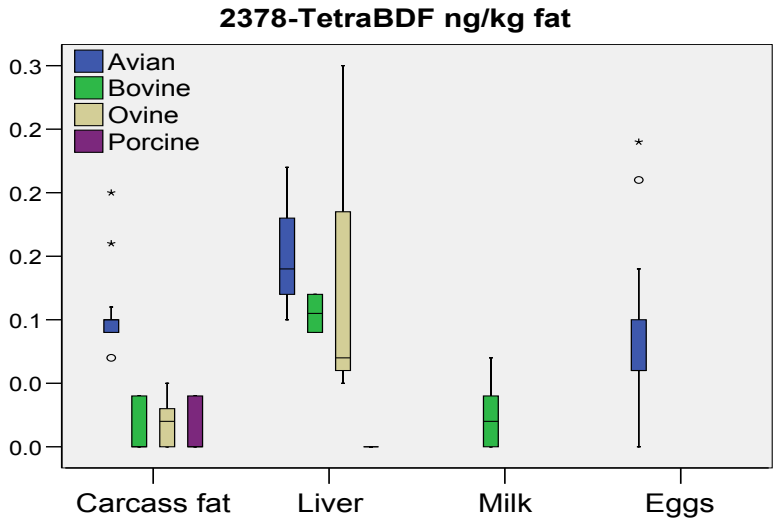
	Carcass Fat	Eggs	Liver	Milk
237-TriBDD	18	0	17	0
2378-TetraBDD	18	5	17	0
12378-PentaBDD	0	0	0	0
123478/123678-HexaBDD	0	0	0	0
123789-HexaBDD	0	0	0	0
238-TriBDF	18	5	58	20
2378-TetraBDF	66	95	83	60
12378-PentaBDF	0	16	17	7
23478-PentaBDF	32	37	75	57
123478-HexaBDF	0	0	42	3
1234678-HeptabromoBDF	26	53	33	10

With the exception of penta- and hexa-brominated compounds PBDD/Fs were detectable in the majority of samples and overall brominated furans occurred at a higher frequency than brominated dioxins.

Similar to BDE occurrence, PBDD/F occurrence was matrix-dependant with liver showing the highest levels, which is expected due to the metabolic function of this organ (Figure 7). Whereas Tetra-BDF was the most frequently occurring congener, Hepta and 2,3,4,7,8-Penta BDF showed the highest concentrations.

Similar observations in shellfish are reported in a recent paper by Fernandes et al[96].

Figure 7 TBDF (ng/kg fat weight) per Food Category and Species



EXPOSURE ESTIMATES

Exposure estimates provided are for dioxins, PCBs and PBDEs in the Irish population, based on the occurrence data shown in this report

Dioxins and PCBs

Exposure of the Irish population to PCDD/Fs and dioxin like PCBs has been calculated using Crème probabilistic modelling software[97]. Estimated intake of these contaminants from consumption of eggs, dairy products, meat, offal, vegetable oil and fish produced in Ireland has been calculated using data from this and previous surveys.

Table 23 Estimated exposure of the Irish adult population to Total WHO TEQ in pg/kg bw per month for PCDD/Fs & DL-PCBs from intake of fish, meat, offal, dairy products, vegetable oils, bivalve molluscs and eggs produced in Ireland

	Average intake		P97.5 intake	
	LB	UB	LB	UB
	pg/kg bw/month			
Total WHO TEQ (1998)	10	12	47	49
Total WHO TEQ (2005)	9	10	39	41

Note: 98-Total WHO TEQ calculated using the 1998 WHO TEF scheme, whereas 05-Total WHO TEQ calculated using the 2005 WHO TEF scheme.

Exposure of the average adult consumer to upperbound Total WHO TEQ PCDD/Fs&DL-PCBs is estimated at 12pg/kg bw, which compared to the WHO PMTI of 70pg/kg bw falls within 17% of the tolerable monthly intake, and exposure of the above average consumer (P97.5) is estimated at 49 pg/kg bw per month, falling within 70% of the tolerable monthly intake.

PBDEs

Exposure of the Irish population to PBDEs has been calculated using Crème probabilistic modelling software[97]. Studies on the commercial PBDEs indicate that pentaBDE is the most toxic and exposure has been estimated excluding BDEs 183 and 209, which are main components of octa and deca formulations.

Estimated upperbound intake of these contaminants from consumption of eggs, dairy products, meat, offal and fish produced in Ireland has been calculated using data from this and previous surveys.

Estimated Exposure to PBDEs

	Average intake	P97.5 intake
	ng/kg bw/d	
Sum BDE 15 (excl BDE 183 and 209)	0.6 - 0.7	3.1 - 3.3

PBBs

PBBs were measured in foodstuffs for the first time in Ireland and were only detected in carcass fat, liver and eggs. The predominant congener was PBB-209, which occurred in all egg samples (0.1-2.84 µg/kg fat), half of all avian fat samples (0.03-0.26 µg/kg fat) and in all avian, ovine and porcine liver samples (0.15-0.30 µg/kg fat). Daily intake of PBB 209 from these sources was estimated at 0.04 and 0.13 ng/kg bw for the average and P97.5% consumer respectively.

HBCD

Exposure to HBCD from the foodstuffs covered in this survey and including HBCD from fish determined in a

previous survey4 was calculated using probabilistic modelling. Lowerbound to upperbound levels were estimated at between 0.43-0.92 ng/kg bw for the average consumer and between 1.55-2.30 ng/kg bw for the P97.5 consumer respectively.

Tetrabromobisphenol A

Exposure to TBBPA from the foodstuffs covered in this survey was calculated using probabilistic modelling. Lowerbound to upperbound levels were estimated at between 0-0.9 ng/kg bw for the average consumer and between 0-2.70 ng/kg bw for the P97.5 consumer respectively.

Hexabromobenzene, Bis(246-tribromophenoxy)ethane and Decabromodi-phenylethane

None of the samples tested contained levels above the respective Limits of Detection, which due to ambient interferences were reasonably higher than for other compounds included in this survey. Therefore, at this time, any intake estimates would be imprecise.

DISCUSSION

The results of this study, undertaken to investigate levels of the persistent organic pollutants PCDDs, PCDFs, PCBs, PBDEs, HBCD, TBBPA, BCDDs, PBDFs and PBBs in carcass fat, milk, eggs and liver produced in Ireland, have demonstrated that levels in the food commodities analysed were generally low, and were well below the maximum limits laid down for PCDDs/PCDFs and dioxin-like PCBs in Council Regulation 1881/2006 with the exception of one ovine liver sample. Levels of the indicator PCBs 28, 52, 101, 118, 138, 153, and 180 were similarly low and are below proposed legislative limits. Levels of brominated flame retardants measured in the study are also low but in comparable ranges to other European countries, which reflect their widespread international use.

Overall, liver was found to be the most contaminated matrix versus milk which on average contains the least amount of contaminants. This observation is not surprising, given that liver is a target organ for persistent organic pollutants, and particularly ruminants tend to store these contaminants in the liver.

A number of atypical results found in this survey include one barn egg sample which was found to contain high levels of PCB 128; this finding was traced back to the use of paint in the egg lobby. Follow up investigation of a finding of elevated PBDE 183 in a pooled porcine fat sample (20 subsamples) could not identify the source, as only 10 of 20 herds could be identified. None of the 10 follow up samples taken was found to contain elevated PBDE levels. The highest single level of Total WHO TEQ PCDD/F&DL-PCB of 22pg/g fat was found in an equine liver sample which was included in this survey for research purposes. The age of the horses that comprised the sub-samples in this sample are unknown but age could be a contributing factor in this finding.

The intake estimates made in this study for dioxins and PCBs confirm that exposure of the Irish adult population to these pollutants, at 17% and 70% of the PTWI for average and above average Irish consumers, respectively, are below the European average, however, this estimate is based solely on consumption of Irish produce and does not take into account intake from imported foodstuffs or other non-food sources. Intake estimates will therefore have to be refined in the future. Furthermore, other substances (i.e PBDD/Fs, some PBBs, PXDD/Fs, PCNs, etc) which are also known to exert dioxin-like effects and are consistent with the Ah receptor mediated mechanism of action will have to be included in these estimates. At the time of writing no TEFs are available for these substances, but it is reasonable to predict that their presence in food will incrementally add to the total dioxin body burden.

In an attempt to estimate theoretical additive burden from 2,3,7,8-TBDF and 2,3,4,7,8-PentaBDF, the most frequently detected congeners of brominated Dioxins and Furans in this survey, relative effect potencies of these congeners in relation to TCDD reported in the literature89 were used instead of TEFs to estimate the additional toxic load. Estimated additional exposure was calculated at 0.6 and 1.5 pg/kg bw per month for average and above average Irish consumers respectively.

Differences between chlorinated and brominated compounds render TEFs developed for chlorinated compounds unsuitable for brominated compounds and more research is needed before robust TEFs can be developed for calculation of overall additive burden from these contaminants to the total TEQ. In particular, the contribution from the tri-brominated compounds needs consideration, as they show a higher level of AhR activity compared to their chlorinated analogues.

Exposure of the Irish population to PBDEs based on the data generated in this study appears to be low, average intake being estimated to be between 0.6 – 0.7 ng/kg bw/day for the average consumer and 3.1 – 3.3 ng/kg bw/day for the P97.5% consumer. Uncertainties and deficiencies in the toxicological databases for PBDEs prevent establishment of tolerable daily intakes. In 2005 JECFA[98] stated that limited toxicity data suggested that for the more toxic PBDE congeners, adverse effects would be unlikely to occur in rodents at doses of less than approximately 100 µg/kg bw per day. JECFA used average dietary intake estimates of 4 ng/kg bw/day, which are based on the North American region and which are considerably higher than estimates calculated here, and

concluded that there appeared to be a large margin of exposure for a non-genotoxic compound which, despite the inadequacy of the data on toxicity and intake, gave reassurance that intakes of PBDEs are not likely to be a significant health concern. Intake estimates calculated by JECFA for the European region were 2.2 ng/kg bw, which are based on the GEMS/Cluster Diet for Europe, which contains much higher intake estimates for Fish, Meat and Fats and Oils compared to the intake estimates derived from the Irish Adult Food Consumption survey.

PBBs were measured in foodstuffs for the first time in Ireland and were only detected in carcass fat, liver and eggs, the predominant congener being PBB-209. Daily intake of PBB 209 from these sources was estimated at 0.04 and 0.13 ng/kg bw for the average and P97.5% consumer respectively.

Individual PBB congeners vary in their pattern of toxicity. PBBs have been categorised on a similar structural basis as the PCBs, with non-ortho coplanar PCBs being dioxin-like with regards to their toxicity and are included in the toxicity equivalency factor (TEF) concept. PBB-169, a coplanar PBB, has been found to be the most toxic congener in several systems, but this compound is present in low concentrations in commercial PBB mixtures[99].

PBB congeners that exhibit AhR-mediated responses constitute only a fraction of the components in commercial PBB mixtures. Therefore, it is presumed that congeners that act by other mechanisms also contribute to the toxicity of PBB mixtures. The mechanism(s) of toxicity for non-dioxin-like PBB congeners is less clearly elucidated, but also may involve receptors (e.g. the estrogen receptor), or the involvement of reactive intermediates (e.g., arene oxides) that can form potentially toxic covalently bound substrate-macromolecular adducts[100].

Due to the Michigan accident in 1973-1974, many toxicity studies on PBBs are available. The critical experimental effects are those on reproduction and carcinogenicity indicating a NOAEL of 0.15 mg/kg body weight/day based on the cancer effects. Based on the carcinogenic effects in animals, a human TDI of 0.15 µg/kg body weight was suggested by WHO in 1994[101]

Assays for mutagenicity and genotoxicity have not shown positive effects with commercial mixtures or individual PBB congeners[102,103]. It was concluded that PBBs probably induce cancer by a non-genotoxic mechanism, and an uncertainty factor of 1000 was applied to the NOAEL to obtain a TDI of 0.15 µg/kg bw/day.

However, analogy to the discussion of non-dioxin-like PCBs indicates that the derivation of this proposed TDI may not be appropriate since simultaneous exposure to dioxin-like PBBs cannot be excluded[100].

Exposure to HBCD from the foodstuffs covered in this survey were estimated at between 0.43-0.92 ng/kg bw for the average consumer and between 1.55-2.30 ng/kg bw for the P97.5 consumer respectively. HBCD is currently undergoing an extensive EU risk assessment. The human health effect assessment was put on hold, while awaiting further information with regard to a developmental neurotoxicity study, however confirmed endpoints of concern for human exposure identified at final draft level[104] were:

Table 24 Summary of effects of HBCD exposure at endpoints of concern

Endpoint Study	NOAEL (mg/kg/day)	Effects observed
Repeated dose toxicity	22.9	Liver weight increase, thyroid weight increase (and decreased serum T4 levels), and increased pituitary weight
Reproductive toxicity/fertility	10	Dose-dependent decrease in fertility index and a reduced number of primordial follicles

The estimated upperbound exposure of 2.3 ng/kg bw for the above average consumer can therefore be compared with a NOAEL of 22.9 mg/kg/day for repeated dose toxicity, giving a MOS of 1×10^7. When compared with a NOAEL of 10 mg/kg/day for reproductive toxicity/fertility, a MOS of 4×10^6 can be calculated. These MOEs do not

indicate concern for human health.

Exposure to TBBPA from the foodstuffs covered in this survey was estimated at between 0-0.9 ng/kg bw for the average consumer and between 0-2.70 ng/kg bw for the P97.5 consumer respectively.

The risk for the general population from TBBPA exposure has been considered insignificant by the WHO in 1998[48] and by the European Commission in 2006[50], however, more recent studies have indicated that TBBPA has endocrine disruption potential. It shows developmental and neurological effects, and at high doses is hepatotoxic and nephrotoxic. At the cellular level it disrupts calcium homeostasis and also increases free radicals, resulting in cytotoxicity. Toxicokinetic studies in the rat have shown that it behaves very similarly to BPA[51-68].

Table 25 Estimated exposure to TBBPA

	Average intake	P97.5 intake
	ng/kg bw/d	
TBBPA Chemical Intake ng/kg bw (LB-UB)	0 - 0.9	0 - 2.7

Compared with an exposure level of 0.5 mg/kg bw/d at which the most sensitive effect was detected in the study conducted by Van der Ven et al, a MoE of 1.85×10^5 can be calculated. In general an MoE of 10,000 or higher, is considered by the EFSA Scientific Committee to be of low concern from a public health point of view and might be considered as a low priority for risk management actions[14].

Very limited toxicological data is available for Hexabromobenzene, Bis(246-tribromophenoxy)ethane and Decabromodi-phenylethane. For decabromodiphenylethane the German Federal Environment Agency in 2000, based on a long term toxicity study with a NOEL of 1000mg/kg suggested a potential ADI of 1000µg/kg bw [74]. No conclusion can be reached on risk for human health based on the limited data available on exposure and toxicity.

CONCLUSIONS

Levels observed for chlorinated dioxins, furans and biphenyls and PBDEs are in line with those from previous FSAI studies. Chlorinated dioxins, furans and PCB levels in Irish food are relatively low compared with similar products from more industrialised countries in the European Union and exposure of Irish consumers of Irish food to chlorinated dioxins and furans in food is below the maximum tolerable monthly intake. Exposure to PBDE is similar to that observed in other EU countries. Although no safe level of exposure has been established for PBDEs so far, exposure to these chemicals is unlikely to cause any adverse effects based on toxicity studies in animals, which showed neuro-developmental effects at concentrations approximately 8,000 times higher than the estimated population intake for consumers of Irish produce.

Exposure to brominated dioxins, furans and some biphenyls, which are likely to incrementally add to the Total TEQ is low and unlikely to significantly increase total dioxin body burden.

Exposure to HBCD, TBBPA, Hexabromobenzene, Bis(246-tribromophenoxy)ethane and Decabromodiphenylethane is similarly low and based on current toxicological data is unlikely to be of concern.

Exposure to flame retardants is not restricted to intake via food alone, however, and exposure to these substances from dust or inhalation has not been taken into account in the estimates reported here.

Overall, the survey shows that Irish meat contains low amounts of the persistent bio-accumulative toxicants measured in this survey, and levels observed do not raise concern for human health. However, with the ban of all PBDE commercial flame retardant mixtures in the European Union, the use and production of alternative substances is predicted to increase and future monitoring programs should closely monitor any trends in Irish food.

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