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Recently Listed Stockholm Convention POPs: Analytical Methodology, Occurrence in food and Dietary Exposure

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Abstract

In recent years, the Stockholm Convention has listed an additional set of persistent organic pollutants (POPs) for elimination or restricted use/release. Data on the occurrence of these contaminants in food is scarce. Validated analytical methodology was developed to investigate the occurrence of hexachlorobutadiene (HCBD), pentachlorobenzene (PCBz), hexachlorobenzene (HCB) pentachlorophenol (PCP) and polychlorinated naphthalenes (PCNs) in 120 retail foods and 19 total diet study samples. The foods covered the range of commonly consumed dietary items including dairy products, eggs (hen and other species), poultry, meat, fish, vegetables, etc. HCBD showed a low frequency of detection, whereas PCBz, HCB and PCNs occurred in most samples (ranges: <0.01 to 0.19 µg/kg; <0.01 to 3.16 µg/kg and 0.1 to 166 ng ΣPCNs/kg respectively). PCP (<0.01 to 1.9 µg/kg) was detected more frequently in meat products, offal and eggs. Fish, shellfish, eggs from all species, animal fats, meat, offal and meat products showed higher contamination levels, which is normal when investigating lipophilic POPs. These levels of occurrence are similar to more recently reported literature levels but perhaps lower, relative to historic data. This is not unexpected, given the restrictions/limitations on these chemicals within the UK and Western Europe. The estimated human exposure to population groups through dietary intake is correspondingly low and based on current toxicological knowledge, the levels in the examined samples do not suggest a cause for health concern. The data also provide a current baseline for HCBD, PCBz and PCP, and update existing data for PCN and HCB occurrence in foods.

1.0 Introduction

Pentachlorobenzene (PCBz), hexachlorobutadiene (HCBD), polychlorinated naphthalenes (PCNs) and pentachlorophenol (PCP) are all industrial chemical, recognised as environmental contaminants, and potential or confirmed food contaminants. In 2004, the Stockholm Convention (a global treaty on persistent organic pollutant (POP) control) listed an initial set of twelve POPs for elimination or global restrictive action on production and use (Stockholm Convention). Since its fourth meeting in 2009, the POP review committee of the Stockholm Convention has listed a further set off sixteen chemicals (including PCBz, HCBD, PCNs and PCP) in its various annexes. There is increasing evidence that exposure to low levels of POPs can lead to disorders of the nervous, reproductive, immune and other human systems, induce carcinogenicity, and interfere with normal infant and child development (Carpenter, 2011, WHO, 2019).

1.1 Hexachlorobutadiene (HCBD)

Hexachlorobutadiene (HCBD) is listed in Annex A (elimination of production and use) of the Stockholm Convention. It has been used as an intermediate in the manufacture of rubber compounds, in the production of lubricants, as a heat transfer liquid, and in hydraulic fluids (ATSDR, 1994). It has also been used as a vineyard pesticide in a number of countries. Although it is no longer produced in the EU and the US, there is evidence of production in China and Taiwan (Juang et al., 2010). A more significant source of current HCBD emissions may be inadvertent production during the manufacture of perchloroethylene, trichloroethylene and carbon tetrachloride (Lecloux, 2004).

HCBD is a lipophilic compound with a high vapour pressure. It has no hydrolysable functional groups and is therefore likely to be persistent in the environment. In environmental compartments it is adsorbed on organic material, with a reported half-life of up to six months in soils. It also shows a high bio-concentration potential in environmental biota such as fish, molluscs and crustaceans with factors (BCFs) of up to 19,000 L/kg (Environment Canada, 2000). Long range transport has been confirmed by detection in arctic fish, water and in polar bear fat. In animal studies, HCBD has been found to target the kidneys (Boroushaki, 2003: Green et al., 2003: Cristofori et al., 2013) and is reported to be toxic at low levels after repeated and chronic exposure. The main adverse effect reported (Yang et al., 1989) was kidney damage in dosed rodents (dose ranges: 0.2 - 2.0 mg/kg bw/day) and damage to the liver and nervous system (Kociba et al., 1977) at a higher dose of 20 mg/kg bw/day. Retardation of foetal growth in rats (Schwetz et al., 1977) dosed at 20 mg/kg bw/day has also been reported. Information on other species is scarce, but available evidence suggests that HCBD is toxic to birds and very toxic to aquatic organisms (UNEP, 2012).

1.2 Pentachlorobenzene (PCBz)

PCBz was produced as a chlorobenzene mixture and used primarily as a viscosity moderator for commercial polychlorinated biphenyls (PCBs) in dielectric fluids or heat exchange liquids (Van de Plassche, et al., 2002; Environment Canada, 2005) Manufacture has reportedly long since ceased in the EU and North America, but the compound may still occur as an impurity in some pesticides. Inadvertently, PCBz may also occur in incinerator emissions and during magnesium production.

PCBz is lipophilic (Log K_{ow} values 4.9 – 6.1. Mackay et al 2006) and this, together with high BCFs in fish and other aquatic organisms, suggest a high bioaccumulation potential. It is persistent in the environment where it is associated with organic matter in matrices such as soil (environmental half-life – 6 years). The reports of occurrence in remote Arctic regions and in Arctic birds and their eggs (Corsolini et al., 2006; Vorkamp et al., 2004) demonstrate long range transport, confirmed by the detection in Svalbard polar bear fat and serum (Gabrielsen et al., 2004), as well as the ability to bioaccumulate in higher order mammals. PCBz has been detected in human milk from Denmark and Finland (Shen et al., 2008) at concentrations ranging from 0.08 to 1.41 μg/kg lipid. There is no current data available for food, but based on toxicological studies, a tolerable daily intake (TDI) of 0.5 μg/kg bw/day has been used (Health Canada, 1993) for evaluating risk.

Adverse effects following sub-chronic dosing of rodents (Sherman rats) at 500 ppm (UNEP, 2007), include increased liver, kidney and adrenal gland weight, disruption of thyroid function, and reduction in haemoglobin content. PCBz is reported as being moderately toxic to humans (harmful if swallowed with an acute exposure based LD_{50} of 250 mg/kg bw) but very toxic to aquatic organisms with a reported LC_{50} for fish or daphnia/algae of ≤ 1 mg/L (UNEP, 2007). Following the listing in Annex A by the Stockholm convention, production and use of PCBz in the EU was prohibited in 2010 (European Commission, 2010).

1.3 Hexachlorobenzene (HCB)

HCB was a mass produced fungicide initially used from 1945 to treat crop seeds, in particular, wheat. Following toxicological studies on HCB that showed increased incidences of animal carcinogenicity in organs such as the kidney, liver and thyroid, it was designated as an animal carcinogen and was additionally given a Group 2B (possible human carcinogen)

classification by the International Agency for Research on Cancer (IARC, 2001). HCB, like PCNs, has been found to bind to the aryl hydrocarbon receptor (AhR) which is a typical response for dioxin-like compounds but the relative potency value is thought to be relatively low. Hahn et al 1989 and Van Birgelen, 1998, suggest 10,000 fold lower potency than TCDD. It is known to occur in foods such as fish at low (0.2 to 2.2 µg/kg) concentrations (Fernandes et al., 2015). Although not a recent Stockholm convention listing, HCB is chemically similar to PCBz, which allows simultaneous analysis with little additional effort.

1.4 Pentachlorophenol (PCP)

PCP is historically reported to have been produced at a rate of up to 90, 000 tons/year and has seen historical applications as an herbicide, insecticide, disinfectant and an ingredient in antifouling paint (UNEP, 2013). It was later used as a fungicide for outdoor wood such as fencing, posts, etc. It was associated with a major food contamination incident in 2007 when dioxin contamination of yoghurt was traced back to PCP-contaminated guar gum from India used as a food thickening ingredient, leading to the introduction of special measures (European Commission, 2010B).

PCP is currently manufactured at a production facility in Mexico (6.6 kT/annum) and its main market and uses remain within the North American continent. Additionally, 1.8 kT/per annum of the PCP salt, (Na-PCP) is manufactured in India for use as an anti-fungicide in impregnated wood/particle boards.

Short-term exposure to PCP is reported to cause harmful effects on the liver, kidneys, nervous system, immune system, and gastrointestinal tract (US EPA, 2010; UNEP, 2013).

Chronic low-level exposure can cause damage to the liver, kidneys, blood, and nervous system, and in the US, the EPA has classified PCP as a probable carcinogen. However, the reports on PCP toxicity provide little or no information on the purity of the PCP material used for testing, and it is reasonable to assume that many of these historical epidemiological exposures would arise from technical grade PCP which was commonly contaminated with PCDD/Fs (dioxins). PCP is also listed in Annex A.

1.5 Polychlorinated naphthalenes (PCNs)

Polychlorinated naphthalenes (PCNs) are legacy contaminants that are listed in Annexes A and C of the Stockholm Convention. They were manufactured during the last century for use in electrical equipment but inadvertent contamination in industrial chemicals and formation during incineration processes are also other important sources. The more recent reports of environmental and food occurrence show characterisation of PCNs as individual congeners (Fernandes et al., 2010; 2011; 2017; Hanari et al., 2012). Some of these congeners are known to elicit potent, aryl hydrocarbon receptor (AhR) mediated or dioxin-like responses but other biological effects, e.g. embryotoxicity, fetotoxicity, hepatotoxicity, immunotoxicity, etc. have also been reported (Blankenship et al., 2000; Engwall et al., 1994; Kilanowicz et al., 2010). The potency of the AhR mediated response is configuration-dependent, based on the molecular chlorine number with hexa-chlorinated congeners showing high potency. A few available data indicate that several PCNs are potent inducers of H4IIE-EROD, AHH and H4IIE-luc (Blankenship et al., 2000; Engwall et al., 1994).

Human exposure to PCNs arises generally through the diet, with a number of recent reports that confirm the widespread occurrence of these contaminants in foods (Domingo et al.,

2003; Marti-Cid et al., 2008; Fernandes et al., 2010; 2011; 2017). PCN toxicity is likely to add to the cumulative toxicity of other dioxin-like compounds (Fernandes, 2013).

This study aimed to investigate the occurrence of HCBD, PCBz, HCB, PCP and PCNs in foods that are routinely consumed as part of the diet in order to provide a current baseline level (for HCBD, PCBz, HCB and PCP) and estimate dietary intake in order to allow an assessment of the risk arising through this primary mode of exposure. In the case of PCNs, the new data would provide an update on existing occurrence information and allow refinement of existing risk assessments on exposure through dietary intake.

2.0 Experimental

Two methodologies were used for analyte determination in this study, one for the determination of PCBz, HCB, HCBD, PCP, and the other for PCN congeners. Both methodologies are based on internal standardisation using ^{13}C-labelled analogues of target compounds, and measurement by HRGC-HRMS (high resolution gas chromatography-high resolution mass spectrometry).

2.1 Sample Collection and Preparation

120 retail foods (collected between 2012 and 2015 from different locations in the UK) and 19 total diet study (TDS-2012) samples were analysed, based on a sampling plan for the retail foods that covered the range of commonly consumed dietary items and included milk and dairy products, eggs (hen, duck, goose and 5 other species) and poultry, meat and meat products, fish, shellfish, offal, oils, vegetables and vegetable products, etc. For the TDS, 986

individual food samples were collected in 14 locations across the UK, and prepared (cooked where required) as normal for consumption. They were composited into 19 of the 20 food groups that make up the TDS (Peattie et al., 1982). These were: Bread, Cereals, Carcass meat, Offal, Meat products, Poultry, Fish, Fats & oils, Eggs, Sugar & preserves, Green vegetables, Potatoes, Other vegetables, Canned vegetables, Fresh fruit, Fruit products, Milk, Milk & dairy products and Nuts (group17 – Beverages, was not included).

The samples were stored under appropriate conditions (most food samples were frozen) prior to analysis. Samples with low moisture content (e.g. cereals, potato crisps, oils and fats) were homogenised by blending or grinding as appropriate. Some food samples were processed to isolate edible portions (in the case of shellfish and fish or meat on the bone), and were homogenised by initial grinding followed by blending. Where required, food samples were lyophilised and re-homogenised. Separate aliquots of the homogenised samples were used for analysis of PCNs and the other analytes.

2.2 Analytes

The following contaminant compounds were measured:

Pentachlorobenzene (PCBz)

Hexachlorobenzene (HCB)

1,3-Hexachlorobutadiene (HCBD)

Pentachlorophenol (PCP)

Polychlorinated naphthalene (PCN) congeners: (17 compounds)

CN-42, **CN-52**/60, CN-53, CN-63, **CN-64**/68, CN-65, CN-66/67, CN-69, CN-70, CN71/72, CN-73, CN-74, **CN-75**.

13C labelled surrogates for PCBz, HCB, HCBD, PCP and 4 PCN congeners (marked in bold) were used for internal standardisation.

2.3 Combined analytical procedure for HCBD, PCBz, HCB and PCP.

PCBz, HCBD, HCB and PCP were analysed together using a single method. An aliquot of the homogenised sample along with a method blank and an in-house reference material (IHRM), was fortified with a mixture of ^{13}C labelled HCBD, PCBz, PCP and HCB at a concentration of 5.0 ng each, in a glass sample bottle. The aliquot was dispersed in 50 ml of 60:40, hexane: dichloromethane (DCM) by high-speed blending for 2 min. 30 g of 1:1, H_2SO_4: silica was then added followed by 2 minutes of further blending with the addition of 2 ml of nonane. The blended extract was immediately filtered through a large glass funnel containing 10 g of sodium sulphate with an over-bed of 15 g 1:1 H_2SO_4: silica and 30 g of sodium sulphate. The loaded funnel was slowly washed with ~130 ml of 60:40, hexane:DCM, followed by rinsing to yield approximately 230 ml of filtered extract. 1 ml of n-nonane was added to the extracts which were then gradually concentrated down at ambient temperature to ~ 2 ml.

The concentrate was chromatographed on an activated basic alumina column, sequentially eluted as follows: 35 ml of hexane – Fraction 1 containing PCBz, HCB and HCBD, 60 ml of 60:40 hexane: DCM to waste, followed by 85 ml of ethyl acetate: hexane – Fraction 3 containing PCP. Fractions 1 and 3 were gently concentrated to 5ml and 0.5 ml respectively and sensitivity standardised with 25 µl of ^{13}C PCB-77 (equivalent to 2.5 ng and 250 pg in each fraction respectively) prior to GC-MS analysis.

HRGC-HRMS measurements were performed on a Waters Autospec Ultima high resolution mass spectrometer coupled to a Hewlett Packard 6890N gas chromatograph fitted with a 60m x 0.25mm i.d. J&W DB-5 MS fused silica capillary column (0.25 μm film thickness) and a programmable temperature vaporisation (PTV) injector operated in constant flow (~1 ml/min helium) mode. The mass spectrometer was operated in electron ionisation (EI) mode at a mass resolution of 10,000 (at 10% peak height) with the mass axis calibrated within a window of 250 ppm_{mass} prior to measurement. For all analytes the two most intense ions that did not suffer from chemical interference were targeted. These were: PCBz - 249.8491, 251.8462; HCBD - 224.8413, 222.8443; HCB - 283.8102, 285.8072; PCP - 265.844, 263.847. The identity of chemical interferences was not investigated but it is reasonable to assume that they may be associated with residual lipid in the extracts, as some foods with higher fat content were more affected. An acceleration voltage of 7kV was used in conjunction with an electron energy of 32-37eV and a trap current of 450 μA. The GC-MS interface was set to 280°C.

Standard solutions and sample extracts were introduced by 10 μl injections into the PTV injector at 40°C using a CTC Analytics PAL GC autosampler. Analyte transfer to the GC column was performed using a PTV injector programme which consisted of a 1.5 minute isothermal period at 40°C followed by heating at 12°C/sec to 320°C, for 3 min, then at 12°C/sec to 350°C to the end of the run. Chromatographic separation was achieved using a GC oven temperature programme consisting of a 1.5 minute isothermal period at 40°C followed by heating at 20°C/min to 175°C for 1 minute, then at 1.5°C/min to 205°C followed by 10°C/min to 215°C for 1min., then 1.5°C/min to 230°C, then 40°C/min to 325°C for 5 min. Data reduction for the GC-MS analyses, and processing to calculate the mass of each

compound present was performed using Masslynx software supplied by Waters. These data were transcribed to Microsoft Excel for collation and quantitation of concentration data.

2.4 Analysis of PCN congeners

A full description of the reagents, reference standards and procedures used for the extraction and analysis of PCNs has been reported earlier (Fernandes et al. 2010). In brief, samples were fortified with ^{13}C-labelled PCN congeners and extracted using mixed organic solvents. PCNs were chromatographically fractionated from potential interferents such as PCBs, using activated carbon and further purified using adsorption chromatography on alumina. Analytical measurement was carried out using HRGC-HRMS. Additional control was provided by the inclusion of methods blanks and an in-house reference material. As additional PCN congener standards of suitable purity have recently become available, these (CN-42, CN-63, CN-65, CN-70) were included in the methodology.

3.0 Results and discussion

3.1 Method Development and Validation

The analytes under investigation are all chemically stable, chlorinated organic compounds. The aromaticity of some of these (PCBz, HCB and PCP) provides a similarity of structure to other persistent environmental contaminants such as PCDD/Fs, PBDEs, PCBs etc. and the hexane:dichloromethane solvent mixture routinely used for these contaminants (Fernandes et al., 2004) proved equally effective here. Other studies on these compounds (Lacorte et al., 2006; Majoros et al., 2013), also report using similar extraction solvents. The first stage of

the procedure described here allows for simultaneous extraction and initial purification of the extracts by exploiting the hydrolytic effect of suspended H_2SO_4 on the food matrices, which either breaks down nutrient molecules or produces polar derivatives that are strongly retained on silica under the elution conditions used. Purification of crude extracts investigated using Florisil^{TM} and alumina revealed that HCBD, PCBz and HCB show low retention on both chromatographic adsorbents, but PCP is far more strongly retained. A series of elution experiments yielded a scheme where HCBD, PCBz and HCB were eluted off activated alumina with relatively non-polar hexane allowing PCP to be eluted with a more polar ethylacetate:hexane mix. The elution profiles were roughly similar on Florisil^{TM}, but alumina was preferred as it yielded more compact and consistent fractions.

All compounds were amenable to the highly sensitive and selective measurement by HRGC-HRMS which complemented by the specificity provided in earlier stages by alumina purification and acid treatment, effectively excluded similar co-extracted contaminants such as PCBs. Method limits of detection are typically of the order of 0.01 µg/kg to 0.03 µg/kg for HCBD, PCBz and HCB, and 0.02 to 0.05 µg/kg for PCP, but could rise to 0.1 µg/kg for high lipid content samples. The method limits of quantitation (LOQs) were calculated by integrating the accompanying procedural blank concentrations (typically ranging from <0.01 µg/kg for PCBz and HCB to 0.02 µg/kg for PCP and 0.03 µg/kg for HCBD) as well as instrument performance at the time of measurement. The achieved LOQs were generally lower than those quoted in the small amount of literature that is available. The linearity of measurement was confirmed for all analytes over the range of concentrations detected in this study.

The use of ^{13}Carbon labelled surrogates used as internal standards allows more robust measurement, and replicate analysis on the same matrix has shown an average precision (as defined by the co-efficient of variation) of up to 10% for PCBz and HCB, and of the order of 15% for HCBD and PCP. The reliability of this approach has been confirmed by the successful analysis of fortified food matrices, yielding concentrations that were consistent with the level of fortification. Analytical recovery typically ranges from 30-35% to 80% for the most volatile (HCBD) and labile (PCP) compounds and 45% to 90% for PCBz and HCB. Measurement uncertainty was estimated using the most recently recommended hybrid approach (European Commission, 2017) that combines historical quality control elements such as precision and accuracy with real-time analysis parameters such as the LOQ at the time of measurement. The method validation performance parameters are summarised in Table 3.1.

There are no available food-based reference materials for the contaminants investigated here, but the IHRM that is routinely used for PCDD/F, PCB and PCN analysis, (#19680 - an unrefined fish oil matrix) was analysed during the course of this work as it contained appreciable amounts of PCBz, PCN and HCB, and lesser amounts of HCBD and PCP.

The quality control criteria used for evaluating data for PCNs, including method performance parameters have been reported before (Fernandes et al., 2010). CN-42, a tetra-chlorinated PCN congener was found to be relatively more volatile and thus showed lower recoveries. Data for this congener is therefore indicative.

3.2 Occurrence in Foods

The volume of data generated from this study is considerable, and was therefore summarized with descriptive statistics in Table 3.2. Concentration units reflect current convention as reported in available literature and data for HCBD, PCBz, HCB and PCP are reported in µg/kg, while PCN data are reported in ng/kg, on a whole weight basis. The distribution of the occurrence of these contaminants among different food types is shown in Figure 1.

3.2.1 HCBD

HCBD was not detectable in the vast majority of samples, but levels of 0.01 µg/kg to 0.03 µg/kg were found in some of the oily fish samples, and in two samples of duck eggs (0.05 µg/kg). The sugars and preserves group of the 2012 TDS samples also contained low levels as did a sample of strawberry jam, but the highest level (0.14 µg/kg) was found in a sample of suet. Comparative data for HCBD occurrence in foods are scarce and largely historical. McConnell et al., 1975, reported concentrations of non-detected to 3.7 µg/kg in foods (butter, cheese, eggs, meats, milk, fruits, etc.) in the UK. Highest levels were detected in grapes 3.7 µg/kg. In the same year, Kotzias et al., 1975, reported levels ranging from not detected to 42 µg/kg (in egg yolk) for a similar range of foods (chicken, eggs, fish, margarine, meat and milk) from Germany.

There have been fewer studies in recent times, mostly limited to aquatic fish species. Roose et al., 2003, reported positive detections in approximately half of the 20 eel samples with a maximum of 12 µg/kg from a pond in Wevelgem (industrial surroundings) Belgium. Another study on eels in Scotland (Macgregor et al, 2010), reported mostly undetectable levels of HCBD in 150 samples (detection limits of 1 to 3 µg/kg). Two studies (Lacorte et al., 2006; Majoros et al., 2013) which reported analysis of river fish from Spain for HCBD did not

detect the compound, although the reporting limits were around 0.5 µg/kg. A recent French study (Miege et al., 2012) reported that HCBD concentrations in 32 pooled fish samples were all below the LOQ (2 to 3 µg/kg ww). In a recent study investigating priority pollutants in four English rivers, HCBD was detectable in only 12 of the 38 samples examined and all values were below 0.2 µg/kg (Jurgens et al., 2013).

A trend in these more recent studies is the lack of positive detection, although this may in part be attributed to the quantitation limits achieved. The other possible reason is that environmental concentrations have decreased in recent years following the reported reduction in emissions in Europe since 2001(Lecloux, 2004).

3.2.2 PCBz

Most of the analysed samples contained detectable levels of PCBz, with some foods such as eggs (hen and other species), poultry, meat, offal and fish showing occurrence in all or nearly all samples. All meat, meat products, offal and poultry samples showed the presence of PCBz, as well as most samples of fish and eggs. However, it was not detected in most vegetable-based samples, milks and shellfish. The highest concentrations were found in samples of gull eggs, goose fat and nuts (0.15 µg/kg to 0.19 µg/kg). There is very little data in the literature for comparative purposes. Falandysz et al., 2000, reported concentrations of 0.09 to 0.75 µg/kg in a range of fish species (herring, cod, eelpout, round goby, flounder, perch, lamprey, pikeperch, etc.) caught in the Gulf of Gdansk. Lacorte et al., 2006, reported higher concentrations from river fish (0.3 to 3.3 µg/kg) in Northern Spain, although the sampling location was strongly influenced by industrial and urban activity. The occurrence range for fish in this study was <0.01 to 0.09 µg/kg.

Notwithstanding the lack of contemporary data on foods, human exposure which is thought to occur primarily as a result of dietary intake, has been confirmed by detection in human fat (Smeds and Sauko, 2001) from a set of 27 Finnish adults, as well as in human breast milk (Shen et al., 2008).

3.2.3 HCB

Given the more widespread global use of HCB as a pesticide, it is unsurprising that concentrations detected in this study are higher than those observed for PCBz. The frequency of detection was similar to PCBz but concentrations were higher ranging from <0.01 µg/kg to 3.2 µg/kg in a sample of suet. Meat, meat products, fish, shellfish, duck eggs and animal fats showed the highest concentrations, but it was not detected in vegetable samples. These concentrations are of a similar magnitude to those reported in a recent study (Perello et al., 2012) on HCB occurrence in common foodstuffs in Spain. The highest values were reported for oils and fats (0.297 µg/kg of fresh weight), dairy products (0.225 µg/kg), and fish and seafood (0.170 µg/kg), with the highest occurrence in butter at 0.86 µg/kg. Although marginally lower than the levels reported in this study, it should be noted that the Spanish study analysed composite rather than individual samples. Thus, there is closer agreement with concentrations measured in the TDS (composite) samples in this study, where the levels of occurrence ranged from <0.01 to 0.3 µg/kg. In an earlier study on HCB in fish from the Gulf of Gdansk, Falandysz et al., 2000 reported levels of 0.36 to 3.7 µg/kg wet weight, and Lacorte et al., 2006, reported a mean value of 15.9 µg/kg for river fish although, as mentioned above, the Ebro river Basin in Spain from which the samples were sourced was known to be influenced by industrial and agricultural (pesticides) activity.

3.2.4 PCP

PCP was detected more frequently in some foods such as offal, meat products and eggs. Concentrations ranged from <0.01 µg/kg up to 1.9 µg/kg for a sample of hen eggs. PCP was detected infrequently, or not at all, in poultry, fish and shellfish, milk and dairy products. Most reports on PCP levels in food are historical. The US EPA reported a range of 1-5 mg/kg ww for PCP in fish during the 1970s, followed by a later report (Farrington and Munday, 1976) of 1.8-62 mg/kg PCP in peanut butter and 6-12 µg/kg in chicken, in the US in 1979. Estimated average daily dietary intake of the compound was 16 µg/day from ingestion of contaminated food, primarily from root vegetables. It was detected in 15% of the foods collected in eight market basket surveys from different regions of the United States during the period of April 1982 to April 1984 (Gunderson, 1988). PCP in fish and freshwater mussel tissues from remote lakes in New Zealand were below the detection limits of 2 µg/kg (Gifford et al., 1995). In the Danish National Pesticide Monitoring Program from 1995 to 1996, PCP was not detected in the samples of fruits, vegetables, cereals, bran, fish, or animal products such as meats, butter, cheese, fat, and eggs.

More recently, reported PCP concentrations (Ge et al., 2007) in various aquatic species collected from aquaculture farms in China, ranged from < 0.5 µg/kg to 61 µg/kg (common carp). The mean PCP concentration was 5.2 µg/kg ww. Concentrations of PCP were less than 1.0 µg/kg for 54.5% of the samples, with 36.4% showing concentrations between 1.0 µg/kg and 10 µg/kg. PCP was detected in 2 out of 100 samples of swine liver and fish muscle from China, at levels of 0.5–2.9 µg/kg (Zhao, 2013), although it was not specified in which matrix, the residues were detected. Historically, maximum residue limits (MRL) for PCP in some

foods have been set in some European countries and these range from 10 µg/kg for food of plant origin, in Germany, to 50 µg/kg in the Netherlands and Switzerland for mushrooms and milk. A default limit of 10 µg/kg (0.01 mg/kg) now applies throughout Europe to PCP in all foods (European Commission, 2005).

3.2.5 PCNs

The vast majority of samples showed PCN occurrence with concentrations ranging from 0.1 ng/kg up to 38.3 ng/kg for fish (sum of measured congeners, upper-bound) although a sample of goose fat showed a concentration of 166 ng/kg. Across the food groups, shellfish, fish, fats of animal origin and eggs (all species) generally showed the highest levels, while milk and vegetables showed low levels of occurrence. The most frequently detected congeners were CN-66/67, CN-73, CN-52 and CN-42. Of these CN-66/67 and CN-73 are among the most toxicologically significant congeners and accounted for approximately 80% of the dioxin-like toxicity in samples of meat, meat products and offals.

Of all the POPs investigated here, PCNs have received more recent attention probably due to their dioxin-like properties. The concentrations reported here are in good agreement with earlier reported UK data (Fernandes et al., 2010; 2017; 2018; 2019) which showed fish species containing the highest concentrations of PCNs (0.73 to 37.3 ng/kg). Similarly, PCN data for a variety of foods from the Republic of Ireland showed a range of 0.1 ng/kg up to 54.3 ng/kg for a sample of farmed salmon (Fernandes et al 2011). The data are also in good agreement with observations on fish samples from Spain, (Marti-Cid et al 2008) which showed highest concentrations in salmon, although the data are not directly comparable as homologue totals, rather than congeners, were measured in that study.

3.3 Dietary Exposure

In common with many other lipophilic contaminants, one of the principal pathways to human exposure to these POPs is from dietary intake. The contaminant concentrations obtained from the TDS samples that were analysed in this study were therefore used to provide an indication of the potential human exposure. Dietary intake was estimated for adults and young children aged from 4-7 years using bodyweights of 70 kg and 20 kg respectively. The estimated upper bound (the limit of detection value is used for concentrations below this limit) intake for average levels of consumption and high levels (97.5^{th} percentile) are given for both population groups in Table 3.3. For HCBD, PCBz, HCB and PCP, the adult intakes expressed in ng/kg bw/day ranged from 0.4 to 1.26 ng/kg bw/day for mean intake and 0.73 to 2.52 for 97.5 percentile consumers. For PCNs, the estimated intake was 13.3 and 30 pg/kg bw/day for mean and 97.5 percentile consumers respectively. As would be expected, because of lower body weight, HCBD, PCBz, HCB and PCP intakes, estimated for 4-7 year olds ranged from 1.2 to 3.9 ng/kg bw/day for mean, and 2 to 7.9 ng/kg bw/day for 97.5 percentile consumption. The corresponding PCN intakes for 4-7 year olds were estimated at 33 and 66 pg/kg bw/day for mean and 97.5 percentile consumption respectively.

The literature on dietary intake for these compounds is scarce but some estimates (including older data) for adult intakes have been included in Table 3.3. Although generally similar, the estimated intake for PCP reported for Canada (Coad and Newhook, 1992) and the US were considerable higher (CDC, 2001) at 50 and 150 ng/kg bw/day compared to the 1.5 ng/kg bw/day estimated in this study. However, there is a considerable difference in time between these two studies accompanied by a decline in the use of PCP in Europe (it is still produced and used in North America) which could account for the difference in estimated intakes.

Risk evaluations for these POPs are similarly scarce and relevant information is only available for some contaminants. For HCBD, based on an animal study (renal toxicity in rats; NOAEL of 0.2 mg/kg bodyweight/day) the WHO developed a tolerable daily intake (TDI) of 0.2 µg/kg of bodyweight (UNEP, 2012). The lowest NOELs for PCBz based on renal and hepatic toxicity in mice and rats ranged from 2.4 and 24 mg/kg bw/ day (UNEP, 2007). In a much earlier assessment, a tolerable daily intake (TDI) of 0.5 µg/kg bw/day was used (Health Canada, 1993) for evaluating risk. For PCP, a proposed acceptable daily intake (ADI) of 6 µg/kg bw/day was used to compare estimations of dietary intake (Health Canada, 2013). Thus, at the current state of knowledge on toxicology and biological effects, it is expected that exposure at the contaminant levels reported in this study are unlikely to be a cause for health concerns.

4.0 Conclusions

All of the Stockholm POPs investigated in this study occurred in commonly consumed foods. Literature data is scarce, but occurrence levels were similar to other studies, albeit a little lower relative to the more historic data. Within the UK and Western Europe, this is perhaps to be expected, given the limited and reduced usage of these chemicals, pollution control legislation and similar declines in food occurrence of other contaminants such as PCDD/Fs, PBDEs and PCBs. However, the rate of decline is likely to be dependent on environmental factors such as long-range transport, as well as factors such as the globalised movement of food and animal feed. A good example of this is the guar gum incident in 2007 where the product, imported from India to European Countries, was found to contain high levels of PCP. As countries in many parts of the world still continue to manufacture and use some of

these POPs, continued monitoring of food would be prudent. The dietary exposure estimates reflect the relatively low levels of food occurrence for these contaminants and it is unlikely that exposure at these levels would be a cause for health concerns. The data provides a baseline for future studies on HCBD, PCBz and PCP in foods and updates current PCN and HCB occurrence.

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References:

ATSDR (Agency for Toxic Substances & Disease Registry), 1994. Public Health Statement for Hexachlorobutadiene. Available at http://www.atsdr.cdc.gov/phs/phs.asp?id=863&tid=168

Boroushaki, M., 2003. Development of resistance against hexachlorobutadiene in the proximal tubules of young male rat. Comp. Biochem. Physiol. C Toxicol. Pharmacol. 136, 367-375.

Carpenter, D. 2011. Health effects of persistent organic pollutants: the challenge for the Pacific Basin and for the world. Rev Environ Health. 26(1), 61-9.

CDC, 2001. Public Health Statement for Pentachlorophenol. Available at: https://www.atsdr.cdc.gov/phs/phs.asp?id=400&tid=70

Coad, S., Newhook, R. 1992. PCP exposure for the Canadian general population: a multimedia analysis. J. Exp. Anal. Environ. Epidemiol. 2, 391-413.

Corsolini, S., Covaci, A., Ademollo, N., Focardi, S., Schepens, P. 2006. Occurrence of organochlorine pesticides and their enantiomeric signatures, and concentrations of polybrominated diphenyl ethers in the Adélie penguin food web. Antarctica. Environ. Poll. 140(2), 371-382.

Blankenship, A., Kannan, K., Villalobos, S., Villeneuve, D., Falandysz, J. et al. 2000. Relative potencies of Halowax mixtures and individual polychlorinated naphthalenes (PCNs) to induce Ah receptor-mediated responses in the rat hepatoma H4IIE-Luc cell bioassay. Environ. Sci. Technol. 34(15), 3153-3158.

Cristofori, P., Defazio, R., Chiusolo, A., Mongillo, M., Bartolucci, G.B., Chiara, F., et al., 2013. Hyaline droplet accumulation in kidney of rats treated with hexachloro-1:3-butadiene: influence of age, dose and time-course. J. Appl. Toxicol. 33, 183-189.

Domingo, J., Falco, G., Llobet, J., Casas, C., Teixido, A., Muller, L. 2003. PCNs in foods: estimated dietary intake by the population of Catalonia, Spain. Environ. Sci. Technol. 37(11), 2332-5.

Engwall, M., Brundstrom, B., Jakobsson, E. 1994. Ethoxyresorufin O-deethylase (EROD) and arylhydrocarbon hydroxylase (AHH) - inducing potency and lethality of chlorinated naphthalenes in chicken and eider duck embryos. Arch. Toxicol. 68, 37-42.

Environment Canada and Health Canada, 2000. Canadian Environmental Protection Act, 1999. Priority Substances List Assessment Report: Hexachlorobutadiene.

Environment Canada, 2005. Risk management strategy for Pentachlorobenzene (QCB) and tetrachlorobenzenes (TeCBs). Available at: http://www.ec.gc.ca/Toxics/docs/substances/certToxics/rms/EN/CBz_RMS_E_05-01-05.pdf

European Commission, 2005. Regulation (EC) No 396/2005 of the European Parliament and of the Council of 23 February 2005 on maximum residue levels of pesticides in or on food and feed of plant and animal origin and amending Council Directive 91/414/EEC.

European Commission, 2010. Commission regulation (EU) No 756/2010 of 24 August 2010 amending Regulation (EC) No 850/2004 of the European Parliament and of the Council on persistent organic pollutants as regards Annexes IV and V. Official Journal of the European Union, L 223/20, 25.8.2010

European Commission, 2010B. Commission Regulation (EU) No 258/2010 of 25 March 2010 imposing special conditions on the imports of guar gum originating in or consigned from India due to contamination risks by pentachlorophenol and dioxins, and repealing Decision 2008/352/EC.

European Commission, 2017. Guidance Document on Measurement Uncertainty for Laboratories performing PCDD/F and PCB Analysis using Isotope Dilution Mass Spectrometry. Available at: https://ec.europa.eu/food/sites/food/files/safety/docs/animal-feed-guidance_document_pcdd-f_pcb_en.pdf

Falandysz, J., Strandberg, L., Strandberg, B., Bergqvist, P-A., Rappe C. 2000. Pentachlorobenzene and hexachlorobenzene in fish in the Gulf of Gdańsk. Polish J. Environ. Stud. 9, 129-132.

Farrington, D., Munday, J. 1976. Determination of trace amounts of chlorophenols by gas-liquid chromatography. Analyst 101, 639-643.

Fernandes, A., White, S., D'Silva, K., Rose, M. 2004. Simultaneous Determination of PCDDs, PCDFs, PCBs and PBDEs in Food. Talanta, 63, 1147-1155.

Fernandes, A., Mortimer, D., Gem, M., Smith, F., Rose, M., Panton, S., Carr, M. 2010. Polychlorinated Naphthalenes (PCNs): Congener specific analysis, occurrence in food and dietary exposure in the UK. Environ. Sci. Technol. 44, 3533.

Fernandes, A., Tlustos, C., Rose, M., Smith, F., Carr, M., Panton, S. 2011. Polychlorinated Naphthalenes (PCNs) in Irish Foods: Occurrence and Human Exposure. Chemosphere, 85, 322-328.

Fernandes, A. 2013. in "Persistent organic pollutants and toxic metals in foods" M Rose and A Fernandes, Eds. Woodhead Publishing Ltd. p367, ISBN 978-0-85709-245-8.

Fernandes, A., Rose, M., Smith, F., Panton, S. 2015. Geographical Investigation for chemical contaminants in fish collected from UK and proximate marine waters. Report FD 15/04 to the UK Food Standards Agency. London, 2015.

Fernandes, A., Rose, M., Falandysz J. 2017. PCNs in Food and Humans. Environ Int. 104, 1-13

Fernandes, A., Mortimer, D., Holmes, M., Rose, M., Zhihua, L., et al. 2018. Occurrence and Spatial Distribution of Chemical Contaminants in Edible Fish Species Collected from UK and Proximate Marine Waters. Env. Int. 114, 219-230

Fernandes, A., Lake, I., Dowding, A., Rose, M., Jones, N., Petch, R., Smith, F., Panton, S. 2019. The Potential of Recycled Materials used in Agriculture to Contaminate Food through Uptake by Livestock. Sci. Tot. Environ. 667, 359–370.

Gabrielsen, G., Knuden, L., Verreault, J., Pusk, K., Muir, D.C., Letcher, R., 2004. Halogenated organics contaminants and metabolites in blood and adipose tissue of polar bears from Svalbard. SFT project 6003080. Norsk Polar Institut. SPFO report 915/2004.

Ge, J., Pan, J., Fei, Z., Wu, G., Giesy, J.2007. Concentrations of pentachlorophenol (PCP) in fish and shrimp in Jiangsu Province, China. Chemosphere, 69, 164–169.

Gifford, J., Judd, M., McFarlane, P., Anderson, S. 1995. Pentachlorophenol in the New Zealand Environment: Assessment near contaminated sites and remote freshwater lakes. Toxicol. Environ. Chem. 48, 1-2.

Green, T., Lee, R., Farrar, D., Hill J. 2003. Assessing the health risks following environmental exposure to hexachlorobutadiene. Toxicol. Lett. 138(1–2), 63-73

Gunderson, E. 1988. Using pesticide data for exposure monitoring: FDA pesticide residue monitoring programme. Available at:https://www.nutrientdataconf.org/PastConf/NDBC15/4-2_Gunderson.pdf

Gunderson, E. 1995. Dietary intakes of pesticides, selected elements, and other chemicals: FDA Total Diet Study, June 1984-April 1986. J AOAC Int. 78(4), 910-21.

Hahn, M., Goldstein, A., Linko, P., Gasiewicz, A. 1989. Interaction of hexachlorobenzene with the receptor for 2,3,7,8-tetrachlorodibenzo-p-dioxin in vitro and in vivo. Evidence that hexachlorobenzene is a weak Ah receptor agonist. Arch. Biochem. Biophys. 270(1), 344-55.

Hanari, N., Falandysz, J., Petrick, G., Yamashita, N. 2012. Determination of congener-specific chloronaphthalenes using comprehensive two-dimensional GC/QMS. Organohalogen Compounds, 74, 32-35.

Health Canada, 1993. Priority Substances List Assessment Report - Pentachlorobenzene. ISBN 0-662-21064-6

Health Canada, 2013. Second Report on Human Biomonitoring of Environmental Chemicals in Canada. Results of the Canadian Health Measures Survey Cycle 2 (2009-2011). 424 pp.

IARC, 2001. Summaries & Evaluations on Hexachlorobenzene. International Agency for Research on Cancer. Vol. 79, p. 493. Available at: http://www.inchem.org/documents/iarc/vol79/79-13.html

Juang, D-F., Lee, C-H., Chen, W-C., Yuan, C-S. 2010. Do the VOCs that evaporate from a heavily polluted river threaten the health of riparian residents? Sci. Tot. Environ. 408(20), 4524–4531.

Jurgens, M., Johnson, A., Jones, K., Hughes, D., Lawlor, A. 2013. The presence of EU priority substances mercury, hexachlorobenzene, hexachlorobutadiene and PBDEs in wild fish from four English rivers. Sci. Tot. Environ. 461–462, 441–452,

Kilanowicz, A., Skrzypinska-Gawrysiak, N. 2010. Toxicity of hexachloronaphthalene (HxCN) and induction of CYP 1A in rats. Ecotoxicol. Environ. Saf. 73, 196-205

Kociba, R., Keyes, D., Jersey, G., Ballard, J., Dittenbov, D., et al. 1977. Results of a 2 year chronic toxicity study with HCBD in rats. Am. Ind. Hyg. Assoc. J. 38, 589-602.

Kotzias, D., Klein, W., Korte, F., 1975. Analysis of residues of hexachlorobutadiene in foodstuff and poultry. Chemosphere, 4, 247–250.

Lacorte, S., Raldua, D., Martinez, E., Navarro, A., Diez, S., Bayona, J.M., Barcelo, D. 2006 Pilot survey of a broad range of priority pollutants in sediment and fish from the Ebro river basin (NE Spain). Environ. Poll. 140, 471–482.

Lecloux, A. 2004. Hexachlorbutadiene – Sources, environmental fate and risk characterization, Science Dossier, Euro Chlor, p43. Available at www.eurochlor.org

Mackay, D., Shiu, W-Y., Ma, K-C., Lee, S. 2006. Physical-chemical properties and environmental fate for organic chemicals. 2nd ed. Boca Raton, FL, U.S.A.: CRC Press, p4182.

Majoros, L., Lava, R., Ricci, M., Binici, B., Sandor, F., Held, A., Emons, H. 2013. Full method validation for the determination of hexachlorobenzene and hexachlorobutadiene in fish tissue by GC-IDMS. Talanta, 116, 251-258.

Marti-Cid, R., Llobet, J., Castell, V., Domingo, J. 2008. Human exposure to PCNs and PCDEs from foods in Catalonia, Spain: Temporal trends. Environ. Sci. Technol. 42, 4195-4201.

Marti-Cid, R., Llobet, J., Castell, V., Domingo, J. 2008B, Human Dietary Exposure to Hexachlorobenzene in Catalonia, Spain, J. Food Protection, 71(10), 2148.

McConnell, G., Ferguson, D.M., Pearson, C. 1975. Chlorinated hydrocarbons and the environment. Endeavor, 34, 13-18.

Macgregor, K., Oliver, I., Harris, L., Ridgway, I. 2010. Persistent organic pollutants (PCB, DDT, HCH, HCB & BDE) in eels (Anguilla anguilla) in Scotland: Current levels and temporal trends. Environ. Poll. 158, 2402-2411.

Miege, C., Peretti, A., Labadie, P., Budzinski, H., Le Bizec, B., Vorkamp, K., et al. 2012. Occurrence of priority and emerging organic compounds in fishes from the Rhone River (France). Anal. Bioanal. Chem. 404(9), 2721-35.

Peattie, M., Buss, D., Lindsay, D., Smart, G. 1982. Reorganization of the British total diet study for monitoring food. Constituents from 1981. Food. Chem. Toxicol. 21(4) 503-507.

Perelló, G., Gómez-Catalán, J., Castell, V., Llobet, J., & Domingo, J. L. 2012. Estimation of the dietary intake of hexachlorobenzene from food consumption by the population of Catalonia, Spain-Health risks. Food Control, 23, 198–202.

Roose, P., Van Thuyne, G., Belpaire, C., Raemaekers, M., Brinkman, U. 2003. Determination of VOCs in yellow eel from various inland water bodies in Flanders (Belgium). J. Environ. Monit. 5, 876-884.

Schwetz, B., Smith, F., Humiston, C., Quast, J., Kociba, R. 1977. Results of a reproductive toxicity study in rats fed diets containing HCBD. Toxicol. Appl. Pharmacol. 42, 382-398.

Shen, H., Main, K., Andersson, A., Damgaard, I., Virtanen ,H., Sjajjebaek, N., Toppari, J., Schramm, K. 2008 Concentrations of persistent organochlorine compounds in human milk and placenta are higher in Denmark than in Finland. Human Reprod. 23(1), 201-210.

Smeds, A., Saukko, P. 2001. Identification and quantification of polychlorinated biphenyls and some endocrine disrupting pesticides in human adipose tissue form Finland. Chemosphere, 44(6), 1463-71.

Stockholm Convention. The listing of POPs in the Stockholm Convention. Available at: http://chm.pops.int/TheConvention/ThePOPs/ListingofPOPs/tabid/2509/Default.aspx

UNEP, 2007. Draft risk profile for Pentachlorobenzene. Persistent Organic Pollutants Review Committee (POPRC). An introduction to the chemical added to the Stockholm Convention at the fifth meeting of the Conference of the Parties. http://www.pops.int/documents/meetings/poprc/drprofile/drp/DraftRiskProfile_PCBz.pdf

UNEP, 2012. Risk profile on hexachlorobutadiene. Available at: http://chm.pops.int/Implementation/Alternatives/AlternativestoPOPs/ChemicalslistedinAnnexA/HCBD/tabid/5863/Default.aspx

UNEP, 2013. Risk profile on pentachlorophenol and its salts and esters. Available at: http://chm.pops.int/TheConvention/POPsReviewCommittee/Reports/tabid/2301/Default.aspx

U.S. EPA, 2010. Toxicological review of pentachlorophenol. Integrated Risk Information System (IRIS) database. EPA/635/R-09/004F.

Van Birgelen, A. 1998. Hexachlorobenzene as a possible major contributor to the dioxin-like activity of human milk. Environ. Health Perspect. 106(11) 683-688.

Van de Plassche, E., Schwegler, A., Rasenberg, M. Schouten, A. 2002. Pentachlorobenzene. Dossier prepared for the third meeting of the UN-ECE Ad hoc Expert Group on POPs. Royal Haskoning report L0002.A0/R0010/EVDP/TL.

Vorkamp, K., Riget, F., Glasius, M., Pecseli, M., Lebeuf, M., Muir, D. 2004. Chlorobenzenes, chlorinated pesticides, coplanar chlorobiphenyls and other organochlorine compounds in Greenland biota. Sci. Total Environ. 331, 157-175.

WHO (World Health Organisation). 2019. Persistent organic pollutants (POPs). Available at: https://www.who.int/foodsafety/areas_work/chemical-risks/pops/en/index1.html

Yang, R., Abdo, K., Elwell, M., Levy, A., Brendnecke, L. 1989. Subchronic toxicology studies of hexachloro-1,3-butadiene (HCBD) in B6C3F1 mice by dietary incorporation. J. Environ. Pathol. Toxicol. Oncol. 9, 323-332.

Zhao, D. 2013. Determination of Pentachlorophenol Residue in Meat and Fish by Gas Chromatography Electron Capture Detection and Gas Chromatography Mass Spectrometry with Accelerated Solvent Extraction. J. Chromatogr. Sci. 52(9), 429 – 435.

Figure 1: Mean *Concentrations in different foods - **A**: HCBD, PCBz, HCB & PCP and **B**: PCNs

Tables 3.1 to 3.3

Table 3.1 Summary of method performance parameters

ANALYTE	Method Precision	Typical Recovery	Method Limit of Detection	Linearity of Measurement	Measurement Uncertainty**
	%	%	w.w.		%
PCNs	10	40-90*	0.01-0.1ng/kg	<0.0004 -5 ng	30 - 126%
HCBD	16	35 - 80	0.01-0.03 µg/kg	<0.004 -1 ng	[a]100 - 200%
PCBz	11	45 -90	0.01-0.03 µg/kg	<0.004 -1 ng	23 - 110%
HCB	7	50-90	0.01-0.03 µg/kg	<0.004 -1 ng	21 - 100%
PCP	17	30-80	0.02-0.05 µg/kg	<0.004 -1 ng	40 - 200%

***** Except PCN-42 (20-45%) ****** Expanded Uncertainty range for samples

[a] higher measurement uncertainty observed for HCBD as most reported concentrations were near or below the LOQ.

Table 3.2 Statistical summary of occurrence levels in food (whole weight basis)

Food Group	HCBD	PCBz	HCB	PCP	*ΣPCN-lwr	*ΣPCN- upr
	µg/kg				ng/kg	
Meat = 8						
Minimum	<0.01	0.01	0.04	<0.01	<0.01	0.20
Median		0.02	0.15		0.97	1.09
Mean		0.02	0.31		0.89	1.04
Maximum	<0.04	0.04	1.07	0.20	2.19	2.24
Meat products = 6						
Minimum	<0.02	0.01	0.02	<0.01	<0.01	0.62
Median		0.02	0.06	0.04	0.09	1.02
Mean		0.02	0.05	0.07	0.37	0.93
Maximum	<0.05	0.04	0.09	0.12	1.02	1.14
Offal = 10						
Minimum	<0.03	0.01	0.02	<0.03	0.22	0.54
Median		0.01	0.13	0.09	0.55	0.98
Mean		0.02	0.15	0.30	0.88	1.28
Maximum	<0.09	0.08	0.35	0.95	3.09	3.66
Poultry = 6						
Minimum	<0.02	0.01	0.02	<0.02	0.02	0.45
Median		0.03	0.04		1.21	1.29
Mean		0.03	0.07		2.92	3.11
Maximum	<0.03	0.09	0.19	0.09	10.97	11.05
Hen Eggs = 9						
Minimum	<0.02	<0.01	0.05	<0.01	<0.01	0.18
Median		0.03	0.24	0.15	1.30	1.36
Mean		0.04	0.21	0.54	4.71	4.82
Maximum	<0.04	0.06	0.35	1.90	23.0	23.0
Fish = 12						
Minimum	<0.01	<0.01	0.04	<0.01	<0.01	0.13
Median	0.02	0.04	0.17		10.11	10.17
Mean	0.02	0.04	0.36		13.46	13.52
Maximum	0.03	0.09	0.97	0.03	38.2	38.3
shellfish = 14						
Minimum	<0.01	<0.01	0.01	<0.01	0.04	0.56

Median		0.06	0.02		13.79	13.84
Mean		0.05	0.22		13.91	14.00
Maximum	<0.03	0.09	0.97	<0.15	31.5	31.7

Table 3.2 (cont'd) Statistical summary of occurrence levels in food (whole weight basis)

Food Group	**HCBD**	**PCBz**	**HCB**	**PCP**	***ΣPCN-lwr**	***ΣPCN- upr**
	µg/kg				ng/kg	
Milk = 8						
Minimum	<0.01	<0.01	<0.01	<0.01	<0.01	0.17
Median			0.04		0.03	0.33
Mean			0.04		0.16	0.55
Maximum	<0.09	<0.02	0.05	0.05	0.75	1.56
Dairy Products = 8						
Minimum	<0.01	<0.01	0.01	<0.02	0.07	0.22
Median		0.03	0.15		0.32	1.02
Mean		0.03	0.49		1.95	2.57
Maximum	<0.12	0.06	2.00	0.22	10.5	10.6
Vegetables = 7						
Minimum	<0.01	<0.01	<0.01	<0.01	<0.01	0.20
Median					0.08	0.34
Mean					0.13	0.73
Maximum	<0.1	0.01	<0.07	0.02	0.31	1.95
Oils and fats = 5						
Minimum	<0.09	<0.02	<0.05	<0.13	0.02	2.10
Median		0.10	0.44		0.43	2.44
Mean		0.10	1.04		35.4	36.7
Maximum	0.14	0.17	3.16	<0.13	166	166
Other foods = 7						
Minimum	<0.01	<0.01	<0.01	<0.01	<0.01	0.28
Median					0.23	1.25
Mean					0.31	1.45
Maximum	<0.06	0.19	0.14	0.16	1.89	3.29
Other eggs = 20						
Minimum	<0.03	0.02	0.03	<0.02	1.67	1.83
Median	0.05	0.05	0.19	0.11	5.57	5.72
Mean	0.05	0.06	0.33	0.11	8.08	8.28
Maximum	0.05	0.15	1.42	0.27	24.8	24.9
TDS = 19						
Minimum	<0.01	<0.01	<0.01	<0.01	0.01	0.11
Median		0.03	0.11	0.05	0.44	0.65
Mean		0.03	0.13	0.06	1.11	1.28

Maximum	<0.06	0.05	0.30	0.12	9.17	9.19

*ΣPCN-lwr – sum of PCN congeners excludes values below LOQ. *ΣPCN-upr – sum including LOQ, of all measured PCN congeners

Table 3.3 Estimated dietary intakes for adults and children for HCB, HBCD, PCBz, PCP and PCNs.

Stockholm Convention Listed POP	**Mean**	**97.5th percentile**	**Literature data**	
Adult *Exposure	ng/kg bw/day			Reference
Hexachlorobenzene (HCB)	1.26	2.52	2.4 (2000) and 1.0 (2008)	Marti-cid et al., 2008B
Hexachlorobutadiene (HCBD)	0.4	0.73	-	
Pentachlorobenzene (PCBz)	0.39	0.73	**0.2 - 0.4 ng/kg per day	Gunderson, 1995
Pentachlorophenol (PCP)	0.75	1.37	150 & 50	CDC, 2001 & Coad and Newhook, 1992
	pg/kg bw/day			
Polychlorinated naphthalenes (PCN) sum	13.3	30.3	100	Marti-cid et al., 2008
Children (4-6 yrs) *Exposure	ng/kg bw/day			
Hexachlorobenzene (HCB)	3.92	7.85	-	
Hexachlorobutadiene (HCBD)	1.23	2.04	-	
Pentachlorobenzene (PCBz)	1.21	2.1	**0.3 - 3 ng/kg per day	Gunderson, 1995
Pentachlorophenol (PCP)	2.28	4.17	-	
	pg/kg bw/day			
Polychlorinated naphthalenes (PCN) sum	33.1	66	-	

* Upper bound exposure estimates
** ng/kg per day depending on age group

Highlights

First structured study investigating HCBD, PCBz, HCB, PCP and PCN collectively in food

PCBz, HCB & PCNs occurred in most samples; HCBD & PCP occurred less frequently

Based on current toxicology, dietary intake from UK food unlikely to cause health concern

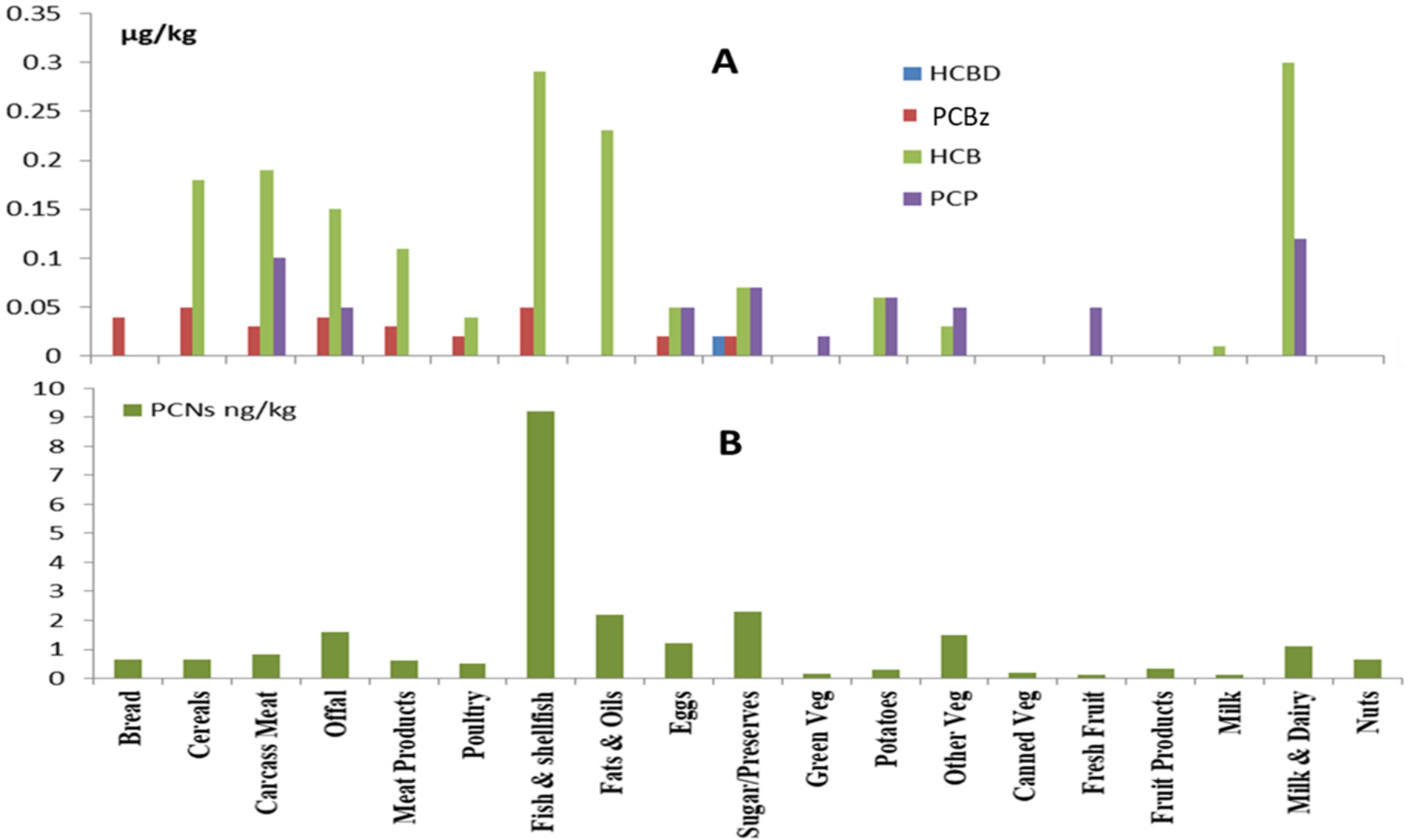
µg/kg
A
HCBD
PCBz
HCB
PCP
PCNs ng/kg
B
Bread
Cereals
Carcass Meat
Offal
Meat Products
Poultry
Fish & shellfish
Fats & Oils
Eggs
Sugar/Preserves
Green Veg
Potatoes
Other Veg
Canned Veg
Fresh Fruit
Fruit Products
Milk
Milk & Dairy
Nuts

Figure 1