Supplementary materials

Materials and methods:

Composition and design of the mixtures – selection of chemicals

In the cases where there were many different congeners of a substance found at measurable levels in serum, breast milk or food, we chose the congeners present at the highest levels for inclusion in the mixtures. As we in the present design focused on compounds found at high levels, we deliberately excluded chlorinated compounds such as polychlorinated dibenzodioxins/polychlorinated dibenzofurans (PCDD/PCDF) as well as most dioxin-like polychlorinated biphenyls (PCBs).

The levels of the different compounds to be included in feed/the *in vivo* mixture were based on published estimated daily intake (EDI) levels from Scandinavian studies (tables S1, S4 and S7). Where median and mean values were both presented in a study, mean values were used for the mixture design. For the various substances, where EDI obtained from the different studies were given in mg/kg/day or mg/day values, these were converted to mg/kg/day values for a 70 kg person. From the studies of Tornkvist et al. (2011) and Fromberg et al. (2011) an assumed average weight of an adult of 73 kg was used for the conversions, based on the relationship between the reported mg/kg/day and mg/day levels of ΣDDTs. Kvalem et al. (2009) reported an EDI of POPs for both representative and high consumers of seafood and game. For the design of the *in vivo* mixture the EDI from representative consumers was used, thus further discussions on EDI levels of PCBs refers to this group. The EDIs from high consumers in table S1 are included for comparison. The full *in vivo* mixture/EDI values used are presented in table 1 (main article).

For the studies of blood values used for the design of the *in vitro* mixture, an average value from several Scandinavian studies was calculated for each congener/substance where these were available (table S2, S5 and S8). In general, only the articles reporting mean values for the substances were used, as these were the values presented in most of the studies, as compared to median values. As a result, the levels of PCBs from the study by Kvalem et al. (2009) were not included in the calculations. The values in the Kvalem study were also somewhat higher than the values reported in the other studies, but these as well as other median values are included in table S2 for completeness. In the tables presenting levels of chlorinated compounds in breast milk from the Scandinavian population, a mean value from Polder et al. (2008) was calculated and presented, as this study reported levels from three different Norwegian locations.

Selection of chlorinated compounds:

Choice of polychlorinated biphenyls (PCBs):

The occurrence of mono- and di­-*ortho*-chlorinated PCBs in food is often presented as the values of six or seven indicator PCBs (PCB 28, 52, 101, 138, 153 and 180 +/- PCB 118). These are the PCB congeners occurring at the highest levels in food (EFSA, 2010). In Kvalem et al. (2009), the highest estimated daily intake (EDI) levels in Norwegian consumers are found for PCB 138 and 153, followed by PCB 101, 180, 52 and 28 (table S1). In regards to serum levels and breast milk levels of the same congeners, PCB 153 is the most prominent, followed by PCB 180 and PCB 138. Levels of PCB 101, 52 and 28 are all much lower (tables S2 and S3).

Kvalem et al. (2009) reported a dietary EDI value for PCB 118 as 0.02 toxic equivalents (TEQ)/kg body weight/day. Fromberg et al. (2011) presented an EDI value for PCB 118 in ng/day, which is equivalent to 70 % of the EDIs for PCB 153 and 138 in the same study. The two latter had the highest EDIs of the PCBs in both studies (96 ng/day for both in the Fromberg study as compared to 97 ng/day in the Kvalem study). Correspondingly, for the *in vivo* mixture, an EDI for PCB 118 that was 70 % of the EDI for PCB 153 and 180 reported in the Kvalem study was used, which equalled 68 ng/day. The value used for PCB 118 is thus lower than the values for PCB 153 and 138, but higher than the values for PCB 101, 180, 52 and 28. For the *in vitro* mixture, a mean value for the blood levels of PCB 118 reported by Van Oostdam et al. (2004) in Norwegian and Swedish women, and by Glynn et al. (2007) in Swedish women was used. PCB 118 had both in blood and breast milk samples a value that was lower than the values for PCB 153, 138 and 180, but higher than PCB 28, 52 and 101. As all the seven mentioned indicator PCBs are present at quite significant levels in both food, blood and breast milk, compared to the levels of other organochlorines, they were all included in the mixture design.

Choice of DDTs:

Tornkvist et al. (2011) and Fromberg et al. (2011) reported EDI levels of dichlorodiphenyltrichloroethanes (DDTs) in Swedish and Danish consumers, respectively. In both studies, the sum of DDTs were fairly similar (table S1). Tornkvist et al. (2011) did also report the mean intake of *p,p’-* dichlorodiphenyldichloroethylene (DDE), which was used for the *in vivo* mixture. The *p,p’-*DDE value from Tornkvist et al. (2011) contributed 72 percent to the sum of DDTs. Van Oostdam et al. (2004) presented the sum of DDT and DDE from blood samples from Norwegian and Swedish women (table S2). *p,p’-*DDE accounted here for 95 percent of the sum of DDT and DDE. Several studies of breast milk samples report values of *p,p’-*DDE and the sum of DDTs (table S3). In a Norwegian study by Polder et al. (2008), *p,p’-*DDE corresponds to 93 percent of the sum of DDTs, whereas *p,p’-* (dichlorodiphenyldichloroethane) DDD and *p,p’-*DDT contribute only 0.3 and 8 percent, respectively. In a Danish study by Damgaard et al. (2006) the contribution from *p,p’-*DDE was approximately 70 percent for both cases and controls. Based on the great contribution of *p,p’-*DDE to the sum of DDTs in both food, blood and breast milk samples, this congener was chosen to represent DDTs in the mixture design. Of all the chlorinated compounds included in the mixture design, *p,p’-*DDE was the one present at the highest concentrations in both food, blood and breast milk.

Choice of HCB:

Tornkvist et al. (2011) and Fromberg et al. (2011) reported EDI values for hexachlorobenzene (HCB) from Swedish and Danish consumers, respectively (table S1), and for the *in vivo* mixture an average of the values from the two studies was used. The EDI for HCB was slightly lower than that for the two most prominent PCB congeners 138 and 153. Van Oostdam et al. (2004) measured serum levels of HCB in Norwegian and Swedish women, whereas Glynn et al. (2007) reported levels from Swedish women (table S2). An average of these values was used for the *in vitro* mixture. HCB was one of the dominating chlorinated substances also in blood, in addition to the PCBs and *p,p’-* DDE. Damgaard et al. (2006), Shen et al. (2008) Polder et al. (2008) and Polder et al. (2009) reported Danish and Norwegian breast milk levels of HCB, which were similar to the levels of β-hexachlorocyclohexane (HCH) as well as PCB 118, and higher than many of the other chlorinated compounds (table S3).

Choice of chlordanes:

Darnerud et al. (2006) and Fromberg et al. (2011) measured chlordanes in Swedish and Danish food, and reported similar EDI values for the sum of chlordanes (table S1). In the study by Fromberg et al. (2011) α-chlordane, γ-chlordane, oxychlordane, and *trans*-nonachlor were measured. However, very few samples contained γ-chlordane. As a rough estimate, the EDI level of oxychlordane is one third of the level of α-chlordane, and the levels of oxychlordane and *trans*-nonachlor are normally similar. However, this will depend on the consumption of the food items included in the EDI calculations, as concentrations of the compounds vary between different food items (Arvid Fromberg, 2012, personal communication). Based on this, for the *in vivo* mixture, the reported value for the sum of chlordanes in Fromberg et al. (2011) were divided between the three compounds as presented in table S1. The EDI for the sum of chlordanes was close to the levels of the most prominent PCB congeners. In the Norwegian and Swedish studies on blood levels by Van Oostdam et al. (2004) and Glynn et al. (2007) (table S2) as well as the Norwegian study on breast milk samples by Polder et al. (2008) (table S3) *trans*-nonachlor was the dominating chlordane, followed by oxychlordane. Blood and breast milk levels of the individual chlordanes were in the range of some of the less prominent PCB congeners. Based on the relatively high contribution of α-chlordane to the sum of chlordanes in food, and the more prominent contribution by oxychlordane and *trans*-nonachlor in breast milk and blood, all the three congeners were included in the mixture design. As none of the included studies on blood levels reported levels of α-chlordane, the level used for the *in vitro* mixture was obtained by using the relationship between α-chlordane and *p,p’-*DDE (the most prominent POP) in breast milk (0.022), and then calculating the value for α-chlordane in relation to the blood level of *p,p’-*DDE.

Choice of HCHs:

Tornkvist et al. (2011) and Fromberg et al. (2011) presented EDI levels of HCH in Swedish and Danish consumers. Tornkvist et al. (2011) reported the sum of HCHs, where the relative contribution was roughly 44 percent α-HCH, 23 percent β-HCH and 33 percent γ-HCH (Per Ola Darnerud, 2012, personal communication). The corresponding values in ng/day and ng/kg/day for the three compounds are presented in (table S1). Fromberg et al. (2011) presented values for the three compounds, and levels for γ-HCH were slightly higher than the values for α-HCH and β-HCH. The levels of the individual HCHs are in the range of PCB 101 and 180. For the design of the *in vivo* mixture, an average value from the Swedish and Danish studies was used. In the study on Swedish blood levels by Glynn et al. (2007) β-HCH was the dominating congener in comparison to α-HCH and γ-HCH (table S2). Van Oostdam et al. (2004) reported similar levels of β-HCH in Norwegian and Swedish women to the level presented in Glynn et al. (2007). In the Danish studies by Damgaard et al. (2006) and Shen et al. (2008), as well as the Norwegian studies by Polder et al. (2008) and Polder et al. (2009) levels of β-HCH in breast milk were reported, and in the studies that also included values of α-HCH and γ-HCH, these were much lower (table S3). In comparison to other chlorinated compounds, the level of β-HCH is similar to the level of PCB 118 and *trans*-nanochlor in blood, as well as HCB and PCB 118 in breast milk, whereas levels of α-HCH and γ-HCH are closer to the values for the less prominent PCB congeners 28, 52 and 101. In the mixture design, due to the presence of α-HCH, β-HCH and γ-HCH in significant amounts in food, all the three congeners were included.

Choice of aldrins:

In the Danish study by Fromberg et al. (2011), an EDI for dieldrin was presented, which was used for the *in vivo* mixture. This EDI exceeds that for HCB, the sum of chlordanes, the sum of HCHs as well as the values for the individual PCB congeners (table S1). Dieldrin has also been measured in breast milk from Denmark, as reported in Damgaard et al. (2006) and Shen et al. (2008). The levels of dieldrin in these studies were similar to the levels of oxychlordane (table S3). Due to its high levels in food and detectable levels in breast milk, dieldrin was included in the mixture design. As none of the included studies on blood levels reported levels of dieldrin, the level of dieldrin was estimated for the *in vitro* mixture using the relationship between dieldrin and *p,p’-*DDE (the most prominent POP) in breast milk (0.048), and calculating the value for dieldrin in relation to the blood level of *p,p’-*DDE. As aldrin is rapidly converted to dieldrin in the environment as well as in the human body, only dieldrin was included in the mixture design.

Selection of brominated compounds:

In a Norwegian study on EDI of polybrominated diphenyl ethers (PBDEs) by Knutsen et al. (2008) the highest dietary intake was found for PBDE 209, followed by PBDE 47, 99, 100, 154 and 153. The level of PBDE 209 alone exceeded the sum of the five other congeners (table S4). In the same study, serum levels were highest for PBDE 47 followed by PBDE 153, 99, 154 and 100 (table S5). Serum levels of PBDE 209 were not analysed in this study. Frederiksen et al. (2010), reported in a study from Denmark PBDE 153 to be the dominating congener, followed by PBDE 209, 47, 99 and 100. PBDE 154 was in the same study present at quite low levels compared to the rest of the PBDEs. Polder et al. (2008), Lignell et al. (2009) and Thomsen et al. (2010) measured the same congeners in Norwegian and Swedish breast milk samples (table S6). Also here, PBDE 47 dominated, followed by PBDE 153, 99, 100 and 154. Polder et al. (2008) and Thomsen et al. (2010) did also measure the level of PBDE 209. In Polder et al. (2008) the mean value of PBDE 209 only exceeded the level of PBDE 154, whereas in Thomsen et al. (2010) the value was higher, and followed that of PBDE 47 in prominence.

Knutsen et al. (2008) did also predict the EDI of hexabromocyclododecane (HBCD), which exceeded the levels of the PBDEs except for PBDE 47 and 209 (table S4). In a Norwegian study on blood levels, Thomsen et al. (2007) detected quite high levels of HBCD in occupationally exposed workers (table S5), whereas no HBCD was detected in the reference group included. In another Norwegian study by Thomsen et al. (2008) on high consumers of fish, lower levels of HBCD were detected. In Polder et al. (2008), only one milk sample had detectable levels of HBCD (table S6), whereas in Lignell et al. (2009), the levels were below the limit of quantification.

If one compares EDI levels of the brominated compounds to the levels of chlorinated compounds these are in the same range. For the blood and breast milk values, the levels of the PBDEs were similar to the levels of the less prominent PCB congeners or even lower. In the mixture design all the mentioned PBDE congeners, as well as HBCD were included.

Selection of perfluorinated compounds:

Haug et al. (2010b) estimated in a study from Norway on dietary intake of perfluoroalkyl acids (PFAAs), the highest EDI for perfluorooctanoic acid (PFOA), followed by perfluorooctane sulfonic acid (PFOS), perfluorodecanoic acid (PFDA), perfluorononanoic acid (PFNA), perfluoroundecanoic acid (PFUnDA) and perfluorohexane sulfonic acid (PFHxS) (table S7). In another Norwegian study by Haug et al. (2010a), as well as Karrman et al. (2007) from Sweden, and Halldorsson et al. (2008) and Fei et al. (2009) from Denmark on serum levels of PFASs, PFOS was the dominating congener in comparison to PFOA, which was also measured (table S8). Karrman et al. (2007) and Haug et al. (2010a) did also measure levels of PFHxS, PFNA, PFDA and PFUnDA. The levels of PFHxS were similar to the levels of PFOA, but the levels of the three other PFAAs were somewhat lower. Breast milk levels of the same PFAAs were also measured in the study by Karrman et al. (2007), but only PFOS and PFHxS were detected in all samples, whereas PFNA was detected in two out of twelve samples. Based on this study Karrman et al. (2007) postulated that the level of perfluorinated compounds in milk is about 1 % of the level in blood. Due to the high concentration of the PFAAs in blood in comparison to the levels of the brominated and chlorinated compounds (when converted to ng/ml – see table 2, main article), as well as due to the presence of the compounds in food, all the above mentioned PFAAs were included in the mixture design.

For the fluorinated and brominated compounds, as well as most of the PCBs included in the *in vivo* mixture, the values used were based on Norwegian studies. Values for the PCBs from Swedish and Danish studies, are however also included in table S1 for the benefit of the reader. For the rest of the chlorinated compounds, values from Swedish and Danish studies were used as these had not been measured in any recent Norwegian studies prior to 2012.

Analyses of chemical concentration levels:

For the extraction of POPs from the lipophilic groups of chemicals at the Norwegian University of Life Sciences (NMBU), the samples were weighed, and added internal standards (PCB 29, 112 and 207 (Ultra Scientific, RI, USA); PBDE 77, 119 and 181 and 13C12-BDE 209 (Cambridge Isotope Laboratories, Inc., MA, USA)) and solvents (cyclohexane/acetone/water), and homogenized using a T25 Ika Ultra-Turrax®. The removal of lipids for the determination of dieldrin was performed using a gel permeation column, filled with Bio-Beads S-X3, 200–400 mesh (Bio-Rad Laboratories, Inc., CA, USA) installed on a Gilson Model 233 combined injector and fractionating system (Gilson, Inc., WI, USA). The removal of lipids for the determination of the rest of the pesticides, PCBs, PBDEs and HBCD was performed using ≥ 97.5% H2SO4 (Fluka Analytical®).

Separation and detection of the pesticides and PCBs were performed on a gas chromatography (GC) coupled to Electron Capture Detector (ECD) and low resolution mass spectrometry (LRMS) (Agilent 6890 Series; Agilent Technologies). PCB 28, 52 and 101, and dieldrin were quantified using a 63Ni micro μ-ECD (Agilent 6890 μ-ECD). The rest of the PCBs and pesticides were quantified, using a MS detector (Agilent 5975C; Agilent Technologies), which was operated by negative chemical ionization (NCI) in selected ion monitoring (SIM) mode. The target ions used were at m/z 71 (HCHs), 284 (HCB), 359 (oxychlordane), 410 (*α*-chlordane), 444 (*trans*-nonachlor), 318 (*p,p’*- DDE), 326 (PCB 118), 360 (PCB 138 and 153), 396 (PCB 180). Detection of PBDEs and HBCD was performed on a HRGC–LRMS (Agilent 6890 Series; Agilent Technologies), equipped with an autosampler (Agilent 7683 Series; Agilent Technologies) and coupled to a MS detector (Agilent 5973 Network; Agilent Technologies) (Polder et al. 2014). The PBDEs and HBCD were monitored using negative chemical ionization (NCI) in selected ion monitoring (SIM) mode at m/z 79/81. PBDE 209 was monitored at m/z 484/486 and 13C12-BDE-209 at m/z 495/497.

 The samples were analysed for perfluorinated compounds by separation with high-performance liquid chromatography (HPLC) using a Discovery C18 column, connected to a C18 pre-column (Supelco, Sigma-Aldrich). Detection was achieved by LC tandem mass spectrometry (MS-MS) (API 3000, LCMS/MS System).

Quality Assurance:

Briefly, every analytical series included three procedural blanks (solvents), one blind (non-spiked clean feed), two spiked samples of clean feed for recoveries and the laboratory's own reference materials (LRMs) of blubber of harp seal (*Pagophilus groenlandicus*). The lowest levels of detection (LODs) for individual compounds were defined as three times the noise level. The LODs (ng/g wet weight (ww)) and relative recoveries (%) were for HCB 0.03 (97 %), HCHs 0.02 (87-103%), *p,p’*- DDE 0.05 (106%), dieldrin 0.5 (99%), PCBs 0.03–0.1 (89-109%), chlordanes 0.03 (87-93%), PBDEs 0.03-0.2 (90-122%), HBCD 0.03 (108%) and perfluorinated compounds 0.06-0.28 (83-90%). Positive consistent blanks were found for dieldrin, and some PCBs (153,138 and 180) and PBDEs (47 and 209), and results were corrected for these blanks. The quality control parameters were within the accepted ranges for the methodology applied. In addition to the LRM, analytical quality was successfully approved by routinely analysing relevant Certified Reference Materials (CRM) such as mackerel oil (CRM 350) and by participation in relevant intercalibration tests such as the 2011 MOE Interlaboratory study for the Northern Contaminants Program (NCP) III — phase 6 on lake trout (*Salvelinus namaycush*) and brown trout (*Salmo trutta)* organized by the Ontario Ministry of the Environment, Laboratory Services Branch.

Discussion:

Criteria for inclusion of food items and gathering of information on intake levels - used in the studies providing the basis for the mixture design:

EDI estimates may be affected by several factors including sampling year, how information on diet is obtained, what the criteria are for inclusion of food items, how the food samples are collected and how the analytical procedures are performed. In the two Norwegian studies reporting EDI levels for the PCBs as well as the PBDEs and HBCD, values from the Norwegian Fish and Game study were used. This study aimed to include food items containing the highest concentration of environmental contaminants in the intake estimations, which is food not usually included in normal surveys (Kvalem et al., 2009). Establishment of EDI levels was based on a food frequency questionnaire, and a database on concentrations in food. In the Swedish studies by Darnerud et al. (2006) and Tornkvist et al. (2011) reporting EDI values for several of the other chlorinated compounds included in the mixtures, market basket analysis were conducted. Here the food included was based on per *capita* – consumption data, and food items consumed at a minimum of 0.5 kg per person and year were selected. Their data on intake levels used for EDI calculations were taken from Swedish producers and trade statistics from the year of food purchase (Tornkvist et al., 2011; Darnerud et al., 2006). For the EDI estimations conducted by Fromberg et al. (2011) for the chlorinated compounds, food items selected for the study included fatty food items with either a high content of contaminant residues or a high level of consumption. The calculation of intake levels were based on a dietary survey. In the study by Haug et al. (2010b), a few food items were purchased and used for chemical measurements of PFAAs, and the dietary intake calculations were based on data from a consumption survey. The reported time of food sampling in the studies used for the *in vivo* mixture varied from 1998 to 2006 for the chlorinated compounds, from 2003 to 2006 for the brominated compounds and from 2008 to 2009 for the perfluorinated compounds (table S1, S4 and S7). Also for blood and breast milk levels, sampling year for the selected studies could be of importance, in addition to other factors such as sex and parity of the donors. In the studies used for the construction of the *in vitro* mixture, blood sampling year was reported to be between 1995 and 2003 for the chlorinated compounds, between 2003 and 2007 for the brominated compounds, and between 1996 and 2004 for the perfluorinated compounds (table S2, S5 and S8).

Tables:

**Table S1 Estimated daily intake for chlorinated compounds from Scandinavian studies**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| Compound | Estimated daily intake ng/day70 kg person | Daily intake ng/kg/day | Year of food sampling # | Year of intake evaluation ## | Reference, Country |
| PCBs |  |  |  |  |  |
| PCB 28 | 14 | 0.2 | 2000-2006 | 2003 | Kvalem et al. (2009), Norway a |
|  | 10 | 0.14 | 2000-2006 | 2003 | Kvalem et al. (2009), Norway b |
|  | 86 | 1.23 | 1998-2003 | 2000-2002 | Fromberg et al. (2011), Denmark |
| PCB 52 | 33 | 0.47 | 2000-2006 | 2003 | Kvalem et al. (2009), Norway a |
|  | 23 | 0.33 | 2000-2006 | 2003 | Kvalem et al. (2009), Norway b |
|  | 96 | 1.37 | 1998-2003 | 2000-2002 | Fromberg et al. (2011), Denmark |
| PCB 101 | 57 | 0.82 | 2000-2006 | 2003 | Kvalem et al. (2009), Norway a |
|  | 39 | 0.56 | 2000-2006 | 2003 | Kvalem et al. (2009), Norway b |
|  | 77 | 1.1 | 1998-2003 | 2000-2002 | Fromberg et al. (2011), Denmark  |
| PCB 118 (TEQ) | (2.1)\* | 0.03\* | 2000-2006 | 2003 | Kvalem et al. (2009), Norway a |
|  | (1.4)\* | 0.02\* | 2000-2006 | 2003 | Kvalem et al. (2009), Norway b |
|  | 67 | 0.96 | 1998-2003 | 2000-2002 | Fromberg et al. (2011), Denmark |
| PCB 138 | 136 | 1.94 | 2000-2006 | 2003 | Kvalem et al. (2009), Norway a |
|  | 97 | 1.38 | 2000-2006 | 2003 | Kvalem et al. (2009), Norway b |
|  | 96 | 1.37 | 1998-2003 | 2000-2002 | Fromberg et al. (2011), Denmark |
| PCB 153 | 149 | 2.13 | 2000-2006 | 2003 | Kvalem et al. (2009), Norway a |
|  | 97 | 1.38 | 2000-2006 | 2003 | Kvalem et al. (2009), Norway b |
|  | 96 | 1.37 | 1998-2003 | 2000-2002 | Fromberg et al. (2011), Denmark |
|  | 82 | 1.17 | 2005 | 2005 | Törnkvist et al. (2011), Sweden |
| PCB 180 | 39 | 0.55 | 2000-2006 | 2003 | Kvalem et al. (2009), Norway a |
|  | 26 | 0.37 | 2000-2006 | 2003 | Kvalem et al. (2009), Norway b |
|  | 58 | 0.82 | 1998-2003 | 2000-2002 | Fromberg et al. (2011), Denmark |
| DDTs |  |  |  |  |  |
| Σ DDTs | 259 | 3.7 | 1998-2003 | 2000-2002 | Fromberg et al. (2011), Denmark |
|  | 280 | 4 | 2005 | 2005 | Törnkvist et al. (2011), Sweden |
| *p,p'*-DDE | 201 | 2.87 | 2005 | 2005 | Törnkvist et al. (2011), Sweden |
| HCB |  |  |  |  |  |
|  | 91 | 1.3 | 1998-2003 | 2000-2002 | Fromberg et al. (2011), Denmark |
|  | 77 | 1.1 | 2005 | 2005 | Törnkvist et al. (2011), Sweden |
| Chlordanes |  |  |  |  |  |
| Σ Chlordanes | 105 | 1.5 | 1998-2003 | 2000-2002 | Fromberg et al. (2011), Denmark c |
|  | 112 | 1.6 | 1999 | 1999 | Darnerud et al. (2006), Sweden d |
| α-Chlordane | 63 | 0.9 | 1998-2003 | 2000-2002 | Fromberg et al. (2011), Denmark e |
| Oxychlordane | 21 | 0.3 | 1998-2003 | 2000-2002 | Fromberg et al. (2011), Denmark e |
| *trans*-Nonachlor | 21 | 0.3 | 1998-2003 | 2000-2002 | Fromberg et al. (2011), Denmark e |
| HCHs |  |  |  |  |  |
| Σ HCHs | 70 | 1.0 | 2005 | 2005 | Törnkvist et al. (2011), Sweden f |
| α-HCH | 42 | 0.6 | 1998-2003 | 2000-2002 | Fromberg et al. (2011), Denmark |
|  | 31 | 0.44 | 2005 | 2005 | Törnkvist et al. (2011), Sweden f |
| β-HCH | 42 | 0.6 | 1998-2003 | 2000-2002 | Fromberg et al. (2011), Denmark |
|  | 16 | 0.23 | 2005 | 2005 | Törnkvist et al. (2011), Sweden f |
| γ-HCH (Lindane) | 56 | 0.8 | 1998-2003 | 2000-2002 | Fromberg et al. (2011), Denmark |
|  | 23 | 0.33 | 2005 | 2005 | Törnkvist et al. (2011), Sweden f |
| Aldrins |  |  |  |  |  |
| Dieldrin | 126 | 1.8 | 1998-2003 | 2000-2002 | Fromberg et al. (2011), Denmark |
| a High consumers of fish and game |  |  |  |  |
| b Representative consumers of fish and game |  |  |  |
| c Sum of chlordanes (oxy-, α-chlordane, trans-nonachlor) |  |  |
| d Sum of chlordanes (oxy-,α-,γ-chlordane, trans-nonachlor) |  |  |
| e Sum of chlordanes was divided in 60 % α-chlordane, 20 % oxy-chlordane, and 20 % trans-nonachlor |
| after personal communication with Arvid Fromberg, 2012 |  |  |
| f The sum of HCHs from Törnkvist et al., 2011, was divided in 44 % α-HCH, 23 % β-HCH and 33 % γ-HCH (Lindane) |
| after personal communication with Per Ola Darnerud, 2012 |  |  |
| ()\* Values in pg TEQ/day and pg TEQ/kg/day |  |  |  |
| # Indicates the year the food used for intake calculations in the study were collected |
| ## Indicates the year the intake evaluations used for calculations in the study were conducted |

**Table S2 Levels of chlorinated compounds in blood from Scandinavian studies**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| Compound | Blood levelsng/g lipid | Sampling period | n | % positive samples # | Reference, Country |
| PCBs |  |  |  |  |  |
| PCB 28 | 2.9 | 1995-1996 | 60 | 85 | Van Oostdam et al. (2004), Norway a,g |
|  | 2.5 | 1995-1996 | 40 | 88 | Van Oostdam et al. (2004), Sweden a,g |
|  | 1 | 1996-1999 | 323 | N/A | Glynn et al. (2007), Sweden b,h |
|  | N/A | N/A | N/A | N/A | Kvalem et al. (2009), Norway c,d,e,h |
| PCB 52 | 1.8 | 1995-1996 | 60 | 58 | Van Oostdam et al. (2004), Norway a,g |
|  | 2.0 | 1995-1996 | 40 | 85 | Van Oostdam et al. (2004), Sweden a,g |
|  | 1 | 1996-1999 | 323 | N/A | Glynn et al. (2007), Sweden b,h |
|  | N/A | N/A | N/A | N/A | Kvalem et al. (2009), Norway c,d,e,h |
| PCB 101 | 1.4 | 1995-1996 | 60 | 15 | Van Oostdam et al. (2004), Norway a,g |
|  | 1.5 | 1995-1996 | 40 | 65 | Van Oostdam et al. (2004), Sweden a,g |
|  | 1 | 1996-1999 | 323 | N/A | Glynn et al. (2007), Sweden b,h |
|  | (4.32) | 2003 | 85 | N/A | Kvalem et al. (2009), Norway c,d,h |
|  | (3.74) | 2003 | 34 | N/A | Kvalem et al. (2009), Norway c,e,h |
| PCB 118 | 10 | 1995-1996 | 60 | 100 | Van Oostdam et al. (2004), Norway a,g |
|  | 11 | 1995-1996 | 40 | 100 | Van Oostdam et al. (2004), Sweden a,g |
|  | 11 | 1996-1999 | 323 | N/A | Glynn et al. (2007), Sweden b,h |
|  | (0.92) \* | 2003 | 85 | N/A | Kvalem et al. (2009), Norway c,d,i |
|  | (1.20)\* | 2003 | 34 | N/A | Kvalem et al. (2009), Norway c,e,i |
| PCB 138 | 35 | 1995-1996 | 60 | 100 | Van Oostdam et al. (2004), Norway a,g |
|  | 47 | 1995-1996 | 40 | 100 | Van Oostdam et al. (2004), Sweden a,g |
|  | 29 | 1996-1999 | 323 | N/A | Glynn et al. (2007), Sweden b,h |
|  | (67.2) | 2003 | 85 | N/A | Kvalem et al. (2009), Norway c,d,h |
|  | (84.1) | 2003 | 34 | N/A | Kvalem et al. (2009), Norway c,e,h |
| PCB 153 | 53 | 1995-1996 | 60 | 100 | Van Oostdam et al. (2004), Norway a,g |
|  | 69 | 1995-1996 | 40 | 100 | Van Oostdam et al. (2004), Sweden a,g |
|  | 59 | 1996-1999 | 323 | N/A | Glynn et al. (2007), Sweden b,h |
|  | (90.1) | 2003 | 85 | N/A | Kvalem et al. (2009), Norway c,d,h |
|  | (108) | 2003 | 34 | N/A | Kvalem et al. (2009), Norway c,e,h |
| PCB 180 | 25 | 1995-1996 | 60 | 100 | Van Oostdam et al. (2004), Norway a,g |
|  | 34 | 1995-1996 | 40 | 100 | Van Oostdam et al. (2004), Sweden a,g |
|  | 38 | 1996-1999 | 323 | N/A | Glynn et al. (2007), Sweden b,h |
|  | (80.5) | 2003 | 85 | N/A | Kvalem et al. (2009), Norway c,d,h |
|  | (96.99) | 2003 | 34 | N/A | Kvalem et al. (2009), Norway c,e,h |
| DDTs |  |  |  |  |  |
| Σ DDT + DDE | 83 | 1995-1996 | 60 | N/A | Van Oostdam et al. (2004), Norway a,g |
|  | 87 | 1995-1996 | 40 | N/A | Van Oostdam et al. (2004), Sweden a,g |
| *p,p'-*DDE | 79 | 1995-1996 | 60 | 100 | Van Oostdam et al. (2004), Norway a,g |
|  | 84 | 1995-1996 | 40 | 100 | Van Oostdam et al. (2004), Sweden a,g |
|  | 88 | 1996-1999 | 323 | N/A | Glynn et al. (2007), Sweden b,h |
| HCB |  |  |  |  |  |
|  | 23 | 1995-1996 | 60 | 100 | Van Oostdam et al. (2004), Norway a,g |
|  | 16 | 1995-1996 | 40 | 100 | Van Oostdam et al. (2004), Sweden a,g |
|  | 23 | 1996-1999 | 323 | N/A | Glynn et al. (2007), Sweden b,h |
| Chlordanes |  |  |  |  |  |
| α-Chlordane | 1.8 | N/A f | N/A f | N/A f | N/A f |
| Oxychlordane | 3.7 | 1995-1996 | 60 | 92 | Van Oostdam et al. (2004), Norway a,g |
|  | 1.9 | 1995-1996 | 40 | 70 | Van Oostdam et al. (2004), Sweden a,g |
|  | 3 | 1996-1999 | 323 | N/A | Glynn et al. (2007), Sweden b,h |
| *trans*-Nonachlor | 6.8 | 1995-1996 | 60 | 100 | Van Oostdam et al. (2004), Norway a,g |
|  | 3.8 | 1995-1996 | 40 | 100 | Van Oostdam et al. (2004), Sweden a,g |
|  | 5 | 1996-1999 | 323 | N/A | Glynn et al. (2007), Sweden b,h |
| HCHs |  |  |  |  |  |
| α-HCH | 1 | 1996-1999 | 323 | N/A | Glynn et al. (2007), Sweden b,h |
| β-HCH | 8.1 | 1995-1996 | 60 | 97 | Van Oostdam et al. (2004), Norway a,g |
|  | 9.2 | 1995-1996 | 40 | 95 | Van Oostdam et al. (2004), Sweden a,g |
|  | 9 | 1996-1999 | 323 | N/A | Glynn et al. (2007), Sweden b,h |
| γ-HCH (Lindane) | 1 | 1996-1999 | 323 | N/A | Glynn et al. (2007), Sweden b,h |
| Aldrins |  |  |  |  |  |
| Dieldrin | 4 | N/A f | N/A f | N/A f | N/A f |
| n = total number of samples analysed |
| # Frequency of samples above the limit of quantification presented in study |
| Values in () represent median values, whereas values without () represent mean values |
| \* Values in pg TEQ/g lipid |
| a Women, all parities |
| b Women, primiparous |
| c Men and women |
| d High consumers of fish and game |
| e Representative consumers of fish and game |
| f Calculated as % of p,p'-DDE (84 ng/g lipid) - based on the relationship between alpha- chlordane and p,p'-DDE (0.022) and |
| between dieldrin and p,p'-DDE (0.048) in breast milk  |
| g Plasma levels |
| h Serum levels  |
| i Whole blood levels |
| N/A - Not available |

**Table S3 Levels of chlorinated compounds in breast milk from Scandinavian studies**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **Compound** | **Breastmilk levels** | **Sampling period** | **n** | **% positive samples #** | **Reference, Country** |
|  | **ng/g lipid** |  |  |  |  |
| **PCBs** |  |  |  |  |  |
| **PCB 28**  | 2.9 (2.6) | 2000-2002 | 29 | N/A | Polder et al. (2008), Norway a |
|  | 2.8 (1.8) | 1996-2006 | 325 | 90 | Lignell et al. (2009), Sweden a |
| **PCB 52** | 0.3 (0.3) | 2000-2002 | 29 | N/A | Polder et al. (2008), Norway a |
|  | < LOQ | 1996-2006 | 325 | < 40 | Lignell et al. (2009), Sweden a |
| **PCB 101** | 0.8 (0.8) | 2000-2002 | 29 | N/A | Polder et al. (2008), Norway a |
|  | < LOQ | 1996-2006 | 325 | < 40 | Lignell et al. (2009), Sweden a |
| **PCB 118** | 13\* (12)\* | 2000-2002 | 29 | N/A | Polder et al. (2008), Norway a |
|  | 11 (9.5) | 1996-2006 | 325 | 100 | Lignell et al. (2009), Sweden a |
| **PCB 138** | 36 (37) | 2000-2002 | 29 | N/A | Polder et al. (2008), Norway a |
|  | 29 (26) | 1996-2006 | 325 | 100 | Lignell et al. (2009), Sweden a |
| **PCB 153** | 50 (51) | 2000-2002 | 29 | N/A | Polder et al. (2008), Norway a |
|  | 58 (52) | 1996-2006 | 325 | 100 | Lignell et al. (2009), Sweden a |
| **PCB 180** | 23 (23) | 2000-2002 | 29 | N/A | Polder et al. (2008), Norway a |
|  | 28 (25) | 1996-2006 | 325 | 100 | Lignell et al. (2009), Sweden a |
| **DDTs** |  |  |  |  |  |
| **Σ DDTs** | (117) | 1997-2001 | 68 | N/A | Damgaard et al. (2006), Denmark b |
|  | (140) | 1997-2001 | 62 | N/A | Damgaard et al. (2006), Denmark c |
|  | 110 (109) | 2000-2002 | 29 | N/A | Polder et al. (2008), Norway a |
| ***p,p'-*DDE** | (84) | 1997-2001 | 68 | 100 | Damgaard et al. (2006), Denmark b |
|  | (97) | 1997-2001 | 62 | 100 | Damgaard et al. (2006), Denmark c |
|  | 102 (99) | 2000-2002 | 29 | N/A | Polder et al. (2008), Norway a |
|  | (134) | 1997-2001 | 65 | 100 | Shen et al. (2008), Denmark d |
|  | 53 (41) | 2002-2006 | 337 | N/A | Polder et al. (2009), Norway e |
| **HCB** |  |  |  |  |  |
|  | (8.8) | 1997-2001 | 68 | 100 | Damgaard et al. (2006), Denmark b |
|  | (10.6) | 1997-2001 | 62 | 100 | Damgaard et al. (2006), Denmark c |
|  | 18 (19) | 2000-2002 | 29 | N/A | Polder et al. (2008), Norway a |
|  | (12.4) | 1997-2001 | 65 | 100 | Shen et al. (2008), Denmark d |
|  | 11 (11) | 2002-2006 | 337 | N/A | Polder et al. (2009), Norway e |
| **Chlordanes** |  |  |  |  |  |
| **α-Chlordane** | 2.0 (2.0) | 2000-2002 | 29 | N/A | Polder et al. (2008), Norway a |
|  | (0.03) | 1997-2001 | 65 | 49.2 | Shen et al. (2008), Denmark d |
| **Oxychlordane** | (4.1) | 1997-2001 | 68 | 100 | Damgaard et al. (2006), Denmark b |
|  | (4.5) | 1997-2001 | 62 | 100 | Damgaard et al. (2006), Denmark c |
|  | 4.4 (4.5) | 2000-2002 | 29 | N/A | Polder et al. (2008), Norway a |
|  | (4.7) | 1997-2001 | 65 | 100 | Shen et al. (2008), Denmark d |
|  | 3.0 (2.8) | 2002-2006 | 337 | N/A | Polder et al. (2009), Norway e |
| ***trans*-Nonachlor** | 7.9 (8.0) | 2000-2002 | 29 | N/A | Polder et al. (2008), Norway a |
| **HCHs** |  |  |  |  |  |
| **α-HCH** | (0.19) | 1997-2001 | 68 | 69.2 | Damgaard et al. (2006), Denmark b |
|  | (0.2) | 1997-2001 | 62 | 69.2 | Damgaard et al. (2006), Denmark c |
|  | 0.2 (0.2) | 2000-2002 | 29 | N/A | Polder et al. (2008), Norway a |
|  | (0.26) | 1997-2001 | 65 | 100 | Shen et al. (2008), Denmark d |
| **β-HCH** | (12.3) | 1997-2001 | 68 | 100 | Damgaard et al. (2006), Denmark b |
|  | (13.6) | 1997-2001 | 62 | 100 | Damgaard et al. (2006), Denmark c |
|  | 13 (12) | 2000-2002 | 29 | N/A | Polder et al. (2008), Norway a |
|  | (16.9) | 1997-2001 | 65 | 100 | Shen et al. (2008), Denmark d |
|  | 5.4 (4.7) | 2002-2006 | 337 | N/A | Polder et al. (2009), Norway e |
| **γ-HCH (Lindane)** | 0.5 (0.4) | 2000-2002 | 29 | N/A | Polder et al. (2008), Norway a |
|  | (0.65) | 1997-2001 | 65 | 100 | Shen et al. (2008), Denmark d |
| **Aldrins** |  |  |  |  |  |
| **Dieldrin** | (3.11) | 1997-2001 | 68 | 100 | Damgaard et al. (2006), Denmark b |
|  | (4.06) | 1997-2001 | 62 | 100 | Damgaard et al. (2006), Denmark c |
|  | (4.88) | 1997-2001 | 65 | 100 | Shen et al. (2008), Denmark d |
| **n = total number of samples analysed** |
| **Values in () represent median values, whereas values without () represent mean values** |
| **\* Values in pg TEQ/g lipid** |
| **# % of positive samples as presented, or calculated ((100 % – % of samples < LOQ) or (n positive samples detected/n analysed)) –**  |
| **depending on the study** |
| **LOQ = Limit of quantification** |
| **a Women primiparous - A mean value for three different Norwegian locations has been used** |
| **b Control mothers** |
| **c Mothers with cryptorchid boys** |
| **d All parities**  |
| **e All, parities, mothers with Norwegian background** |

**Table S4 Estimated daily intake for brominated compounds from Scandinavian studies**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| Compound | Estimated daily intake ng/day70 kg person | Daily intake ng/kg/day  | Year of food sampling # | Year of intake evaluation ## | Reference, Country |
|   |  |
| PBDEs |  |  |  |  |  |
| Σ PBDEs\* | 98 | 1.4 | 2003-2006 | 2003 | Knutsen et al. (2008) Norway a |
| PBDE 47 | 68 | 0.97 | 2003-2006 | 2003 | Knutsen et al. (2008) Norway a |
| PBDE 99 | 13 | 0.19 | 2003-2006 | 2003 | Knutsen et al. (2008) Norway a |
| PBDE 100 | 11 | 0.15 | 2003-2006 | 2003 | Knutsen et al. (2008) Norway a |
| PBDE 153 | 2 | 0.03 | 2003-2006 | 2003 | Knutsen et al. (2008), Norway a |
| PBDE 154 | 4 | 0.06 | 2003-2006 | 2003 | Knutsen et al. (2008), Norway a |
| PBDE 209 | 105 | 1.5 | 2003-2006 | 2003 | Knutsen et al. (2008), Norway a |
| HBCD |  |  |  |  |  |
| HBCD | 21 | 0.3 | 2003-2006 | 2003 | Knutsen et al. (2008), Norway a |
| \* Sum of PBDE 47,99,100,153 and 154 |
| a Values from all the 184 study participants have been used including the reference group |
| # Indicates the year the food used for intake calculations in the study were collected |
| ## Indicates the year the intake evaluations used for calculations in the study were conducted |

**Table S5 Levels of brominated compounds in blood from Scandinavian studies**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **Compound** | **Blood levelsng/g lipid** | **Sampling period** | **n** | **% positive samples**  | **Reference, Country** |
| **PBDEs** |  |  |  |  |  |
| **PBDE 47** | 2.00 (1.44) | 2003 | 125 | 99.2 # | Knutsen et al. (2008), Norway a |
|  | 0.859 (0.381) | 2007 | 51 | 80 ## | Frederiksen et al. (2010), Denmark b |
| **PBDE 99** | 0.65 (0.43) | 2003 | 125 | 98.4 # | Knutsen et al. (2008), Norway a |
|  | 0.552 (< 0.105) | 2007 | 51 | 37 ## | Frederiksen et al. (2010), Denmark b |
| **PBDE 100** | 0.43 (0.34) | 2003 | 125 | 92 # | Knutsen et al. (2008), Norway a |
|  | 0.290 (< 0.104) | 2007 | 51 | 27 ## | Frederiksen et al. (2010), Denmark b |
| **PBDE 153** | 1.36 (1.10) | 2003 | 125 | 99.2 # | Knutsen et al. (2008), Norway a |
|  | 1.916 (1.126) | 2007 | 51 | 98 ## | Frederiksen et al. (2010), Denmark b |
| **PBDE 154** | 0.52 (0.39 ) | 2003 | 125 | 94.4 # | Knutsen et al. (2008), Norway a |
|  | 0.069 (< 0.018) | 2007 | 51 | 45 ## | Frederiksen et al. (2010), Denmark b |
| **PBDE 209** | 1.805 (1.709 ) | 2007 | 17 | 94 ##  | Frederiksen et al. (2010), Denmark b |
| **HBCD** |  |  |  |  |  |
| **HBCD** | 190 (101) | 2005 | 20 | 100 # | Thomsen et al. (2007), Norway c |
|  | 9.6 (4.1) | 2004-2005 | 41 | 76 # | Thomsen et al. (2008), Norway d |
|  | 3.7 (2.6) | 2004-2005 | 25 | 72 # | Thomsen et al. (2008), Norway e |
| **n = total number of samples analysed** |
| **Values in () represent median values, whereas values without () represent mean values** |
| **# % of positive samples calculated (n positive samples detected/n analysed, as presented in the studies)** |
| **## Frequency of samples above the limit of quantification presented in study** |
| **a All the presented values from the 125 study participants, men and women, have been used including the reference group** |
| **b Healthy women admitted for caesarean section** |
| **c Occupationally exposed workers** |
| **d High consumers of fish, men** |
| **e High consumers of fish, women, all parities** |

**Table S6 Levels of brominated compounds in breast milk from Scandinavian studies**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **Compound** | **Breastmilk levels** | **Sampling period** | **n** | **% positive samples a** | **Reference, Country** |
|  | **ng/g lipid** |  |  |  |  |
| **PBDEs** |  |  |  |  |  |
| **PBDE 47** | 1.7 (1.26 ) | 2000-2002 | 10 | 100 | Polder et al. (2008), Norway b |
|  | 1.90 (1.50) | 1996-2006 | 276 | 99 | Lignell et al. (2009), Sweden b |
|  | 1.7 | 2003-2005 | 393 | 100 | Thomsen et al. (2010), Norway c |
| **PBDE 99** | 0.49 (0.41) | 2000-2002 | 10 | 100 | Polder et al. (2008), Norway b |
|  | 0.45 (0.32) | 1996-2006 | 276 | 84 | Lignell et al. (2009), Sweden b |
|  | 0.49 | 2003-2005 | 393 | 100 | Thomsen et al. (2010), Norway c |
| **PBDE 100** | 0.38 (0.40 ) | 2000-2002 | 10 | 100 | Polder et al. (2008), Norway b |
|  | 0.36 (0.29 ) | 1996-2006 | 276 | 91 | Lignell et al. (2009), Sweden b |
|  | 0.40 | 2003-2005 | 393 | 99.7 | Thomsen et al. (2010), Norway c |
| **PBDE 153** | 0.77 (0.68 ) | 2000-2002 | 10 | 100 | Polder et al. (2008), Norway b |
|  | 0.64 (0.57) | 1996-2006 | 276 | 98 | Lignell et al. (2009), Sweden b |
|  | 0.56 | 2003-2005 | 393 | 99.5 | Thomsen et al. (2010), Norway c |
| **PBDE 154** | 0.07 (0.06 ) | 2000-2002 | 10 | 60 | Polder et al. (2008), Norway b |
|  | < (LOQ) | 1996-2006 | 276 | < 40 | Lignell et al. (2009), Sweden b |
|  | 0.062 | 2003-2005 | 393 | 63.4 | Thomsen et al. (2010), Norway c |
| **PBDE 209**  | 0.22 (0.13) | 2000-2002 | 10 | 100 | Polder et al. (2008), Norway b |
|  | 0.61 (0.32) | 2003-2005 | 46 | 76 | Thomsen et al. (2010), Norway c |
| **HBCD** |  |  |  |  |  |
| **HBCD** | 0.13 | 2000-2002 | 10 | 10 | Polder et al. (2008), Norway b |
|  | < (LOQ) | 1996-2006 | 276 | < 40 | Lignell et al. (2009), Sweden b |
|  | 1.7 (0.86) | 2003-2005 | 310 | 56.8 | Thomsen et al. (2010), Norway c |
| **n = total number of samples analysed** |
| **Values in () represent median values, whereas values without () represent mean values** |
| **a % of positive samples (100 % – % of samples < LOQ) – depending on the study** |  |
| **b Women primiparous** |  |  |  |  |
| **c Women all parities** |  |  |  |  |
| **LOQ = Limit of quantification** |  |  |  |  |
| **N/A = Information not available** |  |  |  |  |

**Table S7 Estimated daily intake for perfluorinated compounds from Scandinavian studies**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **Compound** | **Estimated daily intake ng/day****70 kg person** | **Daily intake ng/kg/day** | **Year of food sampling #** | **Year of intake evaluation ##** | **Reference, Country** |
|  |  |
| **PFAAs** |  |  |  |  |  |
| **PFHxS** | 1.2 | 0.017  | 2008-2009 | 1997 | Haug et al. (2010b), Norway |
| **PFOS** | 18  | 0.26  | 2008-2009 | 1997 | Haug et al. (2010b), Norway |
| **PFOA** | 31  | 0.44  | 2008-2009 | 1997 | Haug et al. (2010b), Norway |
| **PFNA** | 9.5  | 0.14  | 2008-2009 | 1997 | Haug et al. (2010b), Norway |
| **PFDA** | 13  | 0.19  | 2008-2009 | 1997 | Haug et al. (2010b), Norway |
| **PFUnDA** | 6.7  | 0.096  | 2008-2009 | 1997 | Haug et al. (2010b), Norway |
| **# Indicates the year the food used for intake calculations in the study were collected** |
| **## Indicates the year the intake evaluations used for calculations in the study were conducted** |

**Table S8 Levels of perfluorinated compounds in blood from Scandinavian studies**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **Compound** | **Blood levels****ng/ml** | **Sampling period** | **n** | **% positive** **samples #** | **Reference, Country** |
|  |  |  |  |
| **PFAAs** |  |  |  |  |  |
| **PFHxS** | 4.7 (4.0) | 2004 | 12 | 100 | Kärrman et al. (2007), Sweden a |
|  | 2.2 | 2003 | 175 | 100 | Haug et al. (2010a), Norway b |
| **PFOS** | 20.7 (18.7) | 2004 | 12 | 100 | Kärrman et al. (2007), Sweden a |
|  | 35.1 | 1996-2002 | 1076 | 100 | Halldorsson et al. (2008), Denmark c |
|  | 29.9 | 1996-2002 | 154 | 100 | Halldorsson et al. (2008), Denmark d |
|  | (33.7) | 1996-2002 | 1240 | 100 | Fei et al. (2009), Denmark e |
|  | 32 | 2003 | 175 | 100 | Haug et al. (2010a), Norway b |
| **PFOA** | 3.8 (3.8) | 2004 | 12 | 100 | Kärrman et al. (2007), Sweden a |
|  | 5.6 | 1996-2002 | 1076 | ~ 100\* | Halldorsson et al. (2008), Denmark c |
|  | 4.5 | 1996-2002 | 154 | ~ 100\* | Halldorsson et al. (2008), Denmark d |
|  | (5.3) | 1996-2002 | 1240 | 99.9 | Fei et al. (2009), Denmark e |
|  | 4.1 | 2003 | 175 | 100 | Haug et al. (2010a), Norway b |
| **PFNA** | 0.80 (0.63) | 2004 | 12 | 100 | Kärrman et al. (2007), Sweden a |
|  | 1.1 | 2003 | 175 | 100 | Haug et al. (2010a), Norway b |
| **PFDA** | 0.53 (0.43 ) | 2004 | 12 | 100 | Kärrman et al. (2007), Sweden a |
|  | 0.46 | 2003 | 175 | 100 | Haug et al. (2010a), Norway b |
| **PFUnDA** | 0.40 (0.28 ) | 2004 | 12 | 100 | Kärrman et al. (2007), Sweden a |
|  | 0.72 | 2003 | 175 | 100 | Haug et al. (2010a), Norway b |
| **n = total number of samples analysed** |
| **Values in () represent median values, whereas values without () represent mean values** |
| **# % of positive samples calculated (n positive samples detected/n analysed, as presented in the studies)** |
| **a Women, primiparous** |
| **b Men and women** |
| **c Women all parities, week 8 of gestation** |
| **d Women all parities, week 24 of gestation** |
| **\* Only 2 samples of PFOA included in the study was below the lower limit of quantification, but information on which of the two**  |
| **groups this referred to was not available** |
| **e Women all parities, week 4-14 of gestation** |

**Table S9 Mixture stock concentrations in the seven different *in vitro* mixtures**

|  |  |
| --- | --- |
| **Compound** | **Mixture stock concentration in mg/ml (1000000x serum levels)** |
|  | **Nominal** | **Measured** |
|  | **Total mixture**  | **Total mixture** | **PFAA mixture** | **Br mixture** | **Cl mixture** | **Cl+Br mixture** | **Cl+PFAA mixture** | **Br+PFAA mixture** |
| **PCBs** |  |  |  |  |  |  |  |  |
| **PCB 28**  | 0.013 | 0.008 |  |  | 0.008 | 0.008 | 0.008 |  |
| **PCB 52**  | 0.010 | 0.006 |  |  | 0.007 | 0.006 | 0.006 |  |
| **PCB 101** | 0.008 | 0.008 |  |  | 0.008 | 0.007 | 0.008 |  |
| **PCB 118** | 0.064 | 0.045 |  |  | 0.043 | 0.040 | 0.041 |  |
| **PCB 138** | 0.222 | 0.155 |  |  | 0.162 | 0.141 | 0.150 |  |
| **PCB 153** | 0.362 | 0.252 |  |  | 0.263 | 0.227 | 0.242 |  |
| **PCB 180** | 0.194 | 0.134 |  |  | 0.149 | 0.129 | 0.138 |  |
| **OCPs** |  |  |  |  |  |  |  |  |
| ***p,p'*-DDE** | 0.502 | 0.339 |  |  | 0.346 | 0.303 | 0,320 |  |
| **HCB** | 0.117 | 0.065 |  |  | 0.067 | 0.059 | 0.063 |  |
| **α-Chlordane** | 0.011 | 0.010 |  |  | 0.012 | 0.013 | 0.013 |  |
| **Oxychlordane** | 0.022 | 0.014 |  |  | 0.014 | 0.012 | 0.012 |  |
| ***trans*-Nonachlor** | 0.041 | 0.044 |  |  | 0.044 | 0.045 | 0.045 |  |
| **α-HCH** | 0.006 | 0.005 |  |  | 0.005 | 0.005 | 0.005 |  |
| **β-HCH** | 0.053 | 0.022 |  |  | 0.022 | 0.020 | 0.020 |  |
| **γ-HCH (Lindane)** | 0.006 | 0.005 |  |  | 0.006 | 0.005 | 0.005 |  |
| **Dieldrin** | 0.024 | 0.021 |  |  | 0.024 | 0.023 | 0.022 |  |
| **BFRs** |  |  |  |  |  |  |  |  |
| **BDE 47** | 0.009 | 0.009 |  | 0.006 |  | 0.008 |  | 0.007 |
| **BDE 99** | 0.004 | 0.004 |  | 0.003 |  | 0.004 |  | 0.004 |
| **BDE 100** | 0.002 | 0.002 |  | 0.002 |  | 0.002 |  | 0.002 |
| **BDE 153** | 0.001 | 0.001 |  | 0.001 |  | 0.001 |  | 0.001 |
| **BDE 154** | 0.002 | 0.002 |  | 0.002 |  | 0.002 |  | 0.001 |
| **BDE 209** | 0.011 | 0.009 |  | 0.010 |  | 0.009 |  | 0.009 |
| **HBCD** | 0.025 | 0.035 |  | 0.035 |  | 0.021 |  | 0.042 |
| **PFAAs** |  |  |  |  |  |  |  |  |
| **PFHxS**  | 3.450 | 3.422 | 2.942 |  |  |  | 3.249 | 3.028 |
| **PFOS**  | 29.425 | 22.348 | 9.788 |  |  |  | 21.978 | 8.809 |
| **PFOA** | 4.523 | 1.743 | 2.999 |  |  |  | 1.831 | 2.770 |
| **PFNA** | 0.800 | 0.507 | 0.817 |  |  |  | 0.481 | 0.807 |
| **PFDA** | 0.495 | 0.193 | 0.369 |  |  |  | 0.228 | 0.341 |
| **PFUnDA**  | 0.560 | 0.190 | 0.182 |  |  |  | 0.095 | 0.156 |
| **Abbreviations: PCBs (polychlorinated biphenyls); OCPs (organochlorine pesticides); BFRs (brominated flame retardants); PFAAs (perfluoroalkyl acids)** |

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