Published in final edited form as:

J Hazard Mater. 2012 July 15; 223-224C: 72–78. doi:10.1016/j.jhazmat.2012.04.052.

Organic halogenated contaminants in mother-fetus pairs of harbor seals (*Phoca vitulina richardii*) from Alaska, 2000–2002

Dongli Wang^{a,†}, Shannon Atkinson^b, Anne Hoover-Miller^{b,c}, Weilin L. Shelver^d, and Qing X. Li^a

^aDepartment of Molecular Biosciences and Bioengineering, University of Hawaii at Manoa, Honolulu, Hawaii 96822, USA

^bSchool of Fisheries and Ocean Sciences, University of Alaska, Juneau, Alaska 99801, USA

^cAlaska SeaLife Center, 301 Railway Ave, Seward, Alaska 99664, USA

^dUSDA-ARS Biosciences Research Laboratory, 1605 Albrecht Boulevard, Fargo, ND 58102, USA

Abstract

This study measured organochlorine pesticides (OCPs) including hexachlorocyclohexanes (HCHs), hexachlorobenzene (HCB), heptachlor and dichlorodiphenyltrichloroethanes (DDTs), polychlorinated biphenyls (PCBs), polychlorinated naphthalenes (PCNs) and polybrominated diphenyl ethers (PBDEs) in tissues of six mother–fetus pairs of harbor seals that were hunted for subsistence in Alaska waters of the Northern Pacific Ocean. These data suggest that significant amounts of these contaminants were transferred from mother harbor seals to fetuses during pregnancy and distributed among fetal organs. The tissue distribution depended on the chemical groups, the specific compounds in the groups and the target organs. Concentration profiles of Σ OCPs, Σ PCBs, Σ PCNs and Σ PBDEs were remarkably similar among maternal blubber, liver, and placenta, fetal blubber, and liver (except for HCHs), possibly indicating that the placenta did not serve as a barrier for all of the compounds analyzed. DDTs, HCB, HCHs, PCBs and PBDEs could penetrate the placenta and accumulate in the blubber of the fetus in utero, while HCHs, PCBs and PBDEs penetrated the placenta and accumulated more preferentially in the fetal liver than in the fetal brain in comparison with DDTs and HCB. Heptachlor and PCNs penetrated the placenta and accumulated in the fetal liver and brain instead of fetal blubber. Similar maternal transfer trends for OCPs, PCBs, PCNs and PBDEs were shown by fetal to maternal (FM) blubber ratios and FM liver ratios. Prenatal transfer of these toxic contaminants from mothers to fetus presumably through the placenta may pose health risks to the fetus during development.

Keywords

persistent organic pollutants; harbor seals; maternal transfer

© 2012 Elsevier B.V. All rights reserved.

Send correspondence to: Dr. Qing X. Li, Department of Molecular, Biosciences and Bioengineering, University of Hawaii at Manoa, Honolulu, Hawaii 96822, Phone: (808) 956-2011, Fax: (808) 956-3452, qingl@hawaii.edu.

[†]Present Address: California Department of Public Health, Environmental Health Laboratory Branch, 850 Marina Bay Parkway, G365, Richmond, CA 94804-6403

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

1. Introduction

The abundance of harbor seals, *Phoca vitulina richardii* in Alaska has varied regionally in recent decades. Subsequent to a widespread population decline in the Gulf of Alaska, populations in some affected areas have either stabilized or are increasing, yet overall numbers of seals remain substantially reduced [1,2]. Environmental pollution, changes of food resources, disease, and/or predation (human and non-human) have been suspected to have contributed the decline or slow recovery. Persistent, bio-accumulative and toxic pollutants have been one concern, and organochlorine pesticides (OCPs), polychlorinated biphenyls (PCBs) and polychlorinated naphthalenes (PCNs) have been detected in free-ranging Alaskan harbor seals [3–5]. The levels of these contaminants were associated with harbor seal body indices and ages [4,5]. Polybrominated diphenyl ethers (PBDEs), a class of flame retardant chemicals, were also detected in other Alaskan marine mammals including sea otters (*Enhydra lutris kenyoni*) [6], spotted seals (*Phoca largha*) [7,8], bearded seals (*Erignathus barbatus*) [8], ringed seals (*Phoca hispida*) [8], ribbon seals (*Phoca fasciata*) [8] and polar bears (*Ursus maritimus*) [9]. Laboratory and field studies have reported that exposure to these organic halogenated pollutants (OHs) have been associated with developmental abnormalities, immunotoxicity and reproductive impairment through disruption of endocrine processes. Some authors have postulated that environmental exposure is associated with outbreaks of infectious disease in marine mammals by rendering them vulnerable to infection by pathogens such as viruses and bacteria [10].

Concentrations of OHs detected in adult female marine mammals have been generally lower than those in adult males [4,5,11,12]. The reason for this difference was thought to be that adult females transferred contaminants to their offspring during gestation and lactation. Studies have shown that lactation is a major maternal transfer route for OHs because of their assimilation in maternal blubber and transfer into milk [13,14]. However, some studies have shown that prenatal exposure of the mammalian fetus to PCBs and DDTs impairs fetal development [15,16], because small amounts of OHs transferred to the developing fetus could result in a relatively high body burden in developmentally critical stages.

Several studies have demonstrated maternal transfer of OHs in marine mammals. Tissue levels of OHs were reported in a single pregnant striped dolphin (*Stenella coeruleoalba*) and her fetus [17], a single harbor porpoise (*Phocoena phocoena*) fetus [18] and 11 pilot whale (*Globicephala melas*) fetuses [19]. OHs were investigated in matched liver samples from five mother-fetus pairs of gray seals (*Halichoerus grypus*) [20], in blubber samples from 20 female sea lions and their fetuses during late pregnancy [21] and in stranded melon-headed whales in 1982, 2001/2002 and 2006 [22]. These studies demonstrated placental transfer of OHs; however, information on the distribution of OHs in different tissues of mother-fetus pairs is extremely limited. In this study, samples of blubber and liver were collected from six pregnant harbor seals and samples of placenta, blubber, liver, and brain were collected from their corresponding fetuses. These seals were harvested during a legal subsistence hunt conducted by Alaska Natives in the northern and eastern Gulf of Alaska. A suite of OHs including OCPs, PCBs, PCNs and PBDEs were measured in these tissues to understand the prenatal exposure of harbor seals and tissue distribution of OHs in pregnant harbor seals and their fetuses.

2. Material and Methods

2.1. Sample Collection and Process

Reproductive tracts (including fetuses), blubber and liver were obtained from six pregnant harbor seals between January 2000 and May 2002 as part of the Alaska Native Harbor Seal Commission Biosampling Program (Table 1). Five seals were harvested in Prince William

Sound; and another seal (47271) was harvested near Ketchikan, Alaska (Table 1). Tissue sampling and assessment of reproductive status were accomplished by gross dissection. Ages of seals were determined by sectioning and counting growth layer groups of canine teeth at Matson's Laboratory in Milltown, MT as described by Blundell and Pendleton [23].

Ovaries were removed and measured for weight, length, width and depth. Both uterine horns were opened longitudinally for evidence of current and previous pregnancies. Fetuses were removed from ovaries, sexed and measured for weight, standard and curvilinear length and axillary, maximum and pelvic girths. A sample was collected from each placenta. Brain, blubber and liver were collected from each fetus. All samples were placed in Teflon sheets, wrapped with aluminum foil and frozen at -25°C . Scalpels were rinsed with acetone between tissues.

2.2. Sample Preparation and Instrumental Analysis

The samples were prepared following the previously described procedure [24–26]. Briefly, individual tissues were homogenized, lyophilized and extracted with a mixture of hexane and methylene chloride (1:1, v/v) on a Dionex ASE 200 system. The extract was fractionated with a sulfuric acid-modified silica gel column and a Florisil column subsequently. All samples were analyzed on a Varian 3800 GC/Saturn 2000 ion trap mass spectrometer (ITMS, Varian, Walnut Creek, CA, USA). The GC was equipped with a 60 m ZB-1 column (0.25 mm i.d., 0.25 μm film thickness) with a constant flow rate of (2 ml/min) of carrier gas (helium). The MS/MS methods were developed to analyze OCPs, PCBs, PCNs [24, 26] and PBDEs [25], and expanded to analyze 145 PCB congeners (Table 1 in Supplementary Content). $^{13}\text{C}_{12}$ -PCBs 28, 123, 169 and 170 were used as internal standards for the quantitation of PCBs. $^{13}\text{C}_{12}$ -PBDEs 3, 15, 28, 47, 99, 153, 154, 139 and 183 were used to quantify PBDEs. ^{13}C -*p,p'*-DDE was used to determine all OCPs.

Tissue distribution of OHs is generally related to tissue lipid content [28]. The concentrations of OHs are often normalized to lipid weight (lw) [21,26]. However, due to the small amounts of tissue available from fetuses, we could not lipid-normalize and, therefore, concentrations of all OHs are reported as ng/g wet weight (ww). The maternal blubber was analyzed in duplicate or triplicate for all individuals, but sample amounts allowed only single or duplicate analysis for the placenta, maternal liver and fetal blubber, liver and brain tissues.

2.3 Quality Assurance and Quality Control

Standard operating procedures [24–26] were followed for the analysis of 20 samples in a batch. Each batch included one solvent blank, one method blank and three alternative matrices (lard, chicken liver and heart) spiked at a level of 10 ng/g ww. The solvent and method blank showed no detectable concentrations for all analytes. The mean recoveries from the analysis of spiked matrices were in a range of 40% to 120% with relative standard deviations (RSDs) of less than 20%. Each sample was analyzed in duplicate or triplicate as the amount of the samples allowed (Table 1). The method detection limits (MDLs) were 0.00004–0.0017 ng/g for OCPs and 0.00011–0.057 ng/g for PCBs and PCNs, and 0.00018–0.120 ng/g ww for PBDEs. In harbor seal tissue samples, positive detection of the target compounds by GC/MS/MS was based on a match for the retention time, quantification ions and the appropriate ratio ($\pm 20\%$ deviation from the values determined from the measurement of standards) of the qualification ions. Target compounds were quantified based on the integration of the extracted ion chromatograms of the quantification ions relative to the integration of the extracted ion chromatogram of the internal standard.

2.4. Statistics and Calculations

The total OCPs concentrations (Σ OCPs) included 12 OCPs (α , β , γ , and δ -HCH isomers, HCB, heptachlor, *o,p'*-DDE, *p,p'*-DDE, *o,p'*-DDD, *p,p'*-DDD, *o,p'*-DDT and *p,p'*-DDT). The total PCB concentrations (Σ PCBs) were reported as the sum of 145 PCB congeners [26]. The concentrations of marker PCBs were the sum of concentrations of PCBs 28, 52, 101, 118, 138, 153 and 180. The total PCN levels (Σ PCNs) were reported as the sum of the concentrations of 37 individual congeners in Halowax 1014, in which PCN congener compositions were referred to in the study by Harner and Bidleman [27]. The total PBDE levels (Σ PBDEs) were reported as the sum of 27 congeners [25]. Percentage contribution of PCBs, PCNs and PBDEs congeners and homologues were calculated by dividing concentrations of individual congeners and homologues by Σ PCBs, Σ PCNs and Σ PBDEs, respectively. Percentage contribution of HCH and DDT isomers were calculated by dividing concentrations of individual HCH and DDT isomers by Σ HCHs (sum of α , β , γ , and δ -HCH isomers) and Σ DDTs (sum of *o,p'*-DDE, *p,p'*-DDE, *o,p'*-DDD, *p,p'*-DDD, *o,p'*-DDT and *p,p'*-DDT), respectively. Percentage distribution of HCB and heptachlor were calculated by dividing the concentrations of HCB and heptachlor by Σ OCPs.

Concentrations of contaminants in ng/g ww were used in all calculations and statistics. Tissue partition coefficients in pregnant harbor seals were calculated as the ratio of other tissue concentrations to those in maternal blubber: Tissues/maternal blubber partition coefficient = $\text{OHs}_{\text{tissue}} \text{ (ng/g ww)} / \text{OHs}_{\text{maternal blubber}} \text{ (ng/g ww)}$. Placental transfer ratios were calculated as the ratio of OHs concentrations in fetal blubber or fetal liver to those in maternal blubber or maternal liver: fetal blubber /maternal blubber ratio (FM blubber ratio) = $\text{OHs}_{\text{fetal blubber}} \text{ (ng/g ww)} / \text{OHs}_{\text{maternal blubber}} \text{ (ng/g ww)}$; or fetal liver /maternal liver ratio (FM liver ratio) = $\text{OHs}_{\text{fetal liver}} \text{ (ng/g ww)} / \text{OHs}_{\text{maternal liver}} \text{ (ng/g ww)}$. Concentration ratios of Σ HCHs, Σ DDTs, Σ PCBs, Σ PBDEs and Σ PCNs between tissues in six-paired maternal and fetal seals were averages of the individual ratios not the ratios of the average concentrations. It is noteworthy the two methods of calculation give similar but not identical results. The ratios we utilized are more pertinent for our study since the ratios are computed for the individual seals before averaging.

The statistical analyses were performed with SPSS Statistics (Version 17.0) software. Analysis of variance (ANOVA) and Student's t-test were used to compare mean concentrations of contaminants among various tissues in fetuses and their corresponding mothers. The level of significance used for all statistical tests was $p < 0.05$. All values below MDLs were treated as half of the MDL.

3. Results and Discussion

3.1 Concentrations of OHs in Maternal and Fetal Tissues

Mean concentrations (ng/g ww) of OHs in six maternal and fetal tissues are shown in Table 2. OHs concentration profiles in fetal tissues resembled those in mother tissues. Σ PCBs and Σ DDTs were dominant contaminants among the chemical groups of the target analytes in the maternal blubber and liver and in the fetal blubber, liver, brain and placenta (Table 2). In maternal and fetal blubber, Σ PCBs were highest in concentration followed by Σ DDTs, Σ PBDEs, Σ HCHs, HCB, heptachlor and Σ PCNs (Table 2). Σ OCPs is the sum of Σ HCHs, Σ DDTs, HCB and heptachlor. Σ PCBs, Σ OCPs and Σ PBDEs were higher in the maternal blubber than those in the maternal liver and placental tissues. This is confirmed by the less than 1.0 ratios of maternal liver to maternal blubber for PCBs, DDTs, PBDEs, HCHs and HCB (Figure 1). The maternal liver/maternal blubber ratios of heptachlor and PCNs, however, were significantly higher than those of PCBs, DDTs, PBDEs, HCHs and HCB ($p < 0.048$, Figure 1). This suggests that heptachlor and PCNs were preferentially deposited in

liver tissues relative to PCBs, DDTs, PBDEs, HCHs and HCB, which were readily deposited in maternal blubber tissue. Asplund *et al.* [28] similarly observed that the two PCN isomers 1,2,3,5,6,7- and 1,2,3,4,6,7- hexachloronaphthalene were selectively retained in the liver of rats orally dosed with the commercial PCN product Halowax 1014 and the concentration ratios in liver/adipose tissues were remarkably high. Weistrand and Norén [29] reported that the concentrations of 1,2,3,5,7/1,2,4,6,7-penta-CN and 1,2,3,4,6,7/1,2,3,5,6,7-hexa-CN were much higher in the liver than in adipose tissue in one human male subject.

Mean concentrations of HCB, heptachlor and PCBs were in the range of 0.07–0.85, 0.09–0.26 and 7.0–19.6 ng/g ww, respectively in the fetal blubber, liver and brain tissues (Table 2), again with Σ PCBs, Σ DDTs, Σ HCHs, HCB and Σ PBDEs being numerically higher in the blubber than in the liver and brain tissues. Σ PCBs in the fetal blubber was higher than those in fetal liver and brain (Table 2), but the difference was not significant ($p = 0.119$ and 0.130 , respectively). In contrast, Σ OCPs and Σ PBDEs in the fetal blubber were significantly higher or nearly so than those in the fetus liver ($p = 0.054$ and 0.011 , respectively) and brain ($p = 0.027$ and 0.036 , respectively) (Table 2). It is interesting that Σ PCNs in the fetal blubber were slightly lower, but Σ PCNs were similar among the maternal blubber, maternal liver, and fetal liver and fetal brain (Table 2). It showed an equilibration through the placenta barrier.

Σ PCBs, Σ DDTs, Σ HCHs, HCB and Σ PBDEs are more readily accumulated in the mother and fetal seal blubber (Table 2). It has been reported that blubber contains more than 90% of the whole body burden of contaminants [30] and plays important roles in the mobilization of lipids and lipophilic contaminants inside the animal body [31]. Lipid movement between body compartments may facilitate the distribution of lipophilic contaminants among blood and tissues [32,33]. Therefore, blubber tissue is often used to assess exposure of free ranging marine mammals to these contaminants. However, PCNs and heptachlor showed relatively low accumulation potential in maternal and fetal blubber. It suggested that tissue distributions and toxicokinetics of OHs depend upon the chemical structures and properties [9].

In the placenta, mean concentrations of OHs were not significantly different (Table 2). Although no significant difference was observed ($p > 0.05$), Σ PCNs in placenta was slightly higher than in the fetal blubber, liver and brain and then in the maternal blubber and liver. Levels of heptachlor in placenta were higher in the fetal blubber, liver and brain than in the maternal liver. This is also demonstrated by values of far greater than 1.0 for placenta to maternal blubber ratios for heptachlor (3.2) and PCNs (5.0), and less than 1.0 of placenta to maternal blubber ratios for PCBs, DDTs, PBDEs, HCHs and HCB (Figure 1). Higher levels of PCNs in placenta are primarily due to the higher contributions of tri-, tetra-, penta- and hepta-CN congeners (Figure 2C), indicating that the placenta can serve as a barrier to prevent transfer of contaminants from mothers to fetuses [19,21]. The preferential accumulation of PCNs and heptachlor in the placenta might result from preferential binding of heptachlor and PCNs to proteins or other components in the tissue. However, in humans, Polishuk *et al.* [34,35] reported 0.28 ppm of heptachlor in the maternal blood, 0.5 ppm in the placenta and 1.0 ppm in the fetal blood. These results suggested that heptachlor can readily cross the placenta to become more concentrated in the fetus.

Similar concentrations of OHs were observed in maternal and fetal liver (Table 2). OH concentrations for fetal liver in decreasing order were Σ PCBs $>$ Σ DDTs $>$ Σ PCNs $>$ Σ PBDEs $>$ Σ HCHs $>$ heptachlor $>$ HCB. It is to note that Σ OCPs is the sum of Σ HCHs, Σ DDTs, HCB and heptachlor. OH concentrations for maternal liver were similar, only heptachlor and HCB were reversed. The ratios of the fetal liver to maternal blubber were 1.8 for heptachlor and 1.2 for PCNs (Figure 1), possibly indicating preferential accumulation for

these contaminants in tissues of less lipid content. Hepatic CYP450 isozymes are involved in xenobiotic detoxification and steroid metabolism [36]. Mixed function oxidase (MFO) activity in newborn harbor seal pups and fetuses were significantly lower than that in adult females [37]. The lower MFO activity suggests that fetal liver tissues have lower potential to sufficiently detoxify these organic contaminants. With little possibility for fetal livers to metabolize OHs, harbor seal fetuses may be at an increased risk for toxicity as compared to their mothers. Effects of these contaminants are mostly related to disturbances of hormonal and endocrine systems as they can bind to and interact with several hormone receptors and transport proteins [38,39].

Concentrations of any OH ranged from 0.07–7.0 ng/g ww in the brain tissues of harbor seal fetuses, which were comparable to those in the maternal liver and placental tissues and had an order of Σ PCBs > Σ PBDEs > Σ PCNs > Σ DDTs > Σ HCHs > heptachlor > HCB (Table 2). The ratios of the fetal brain to maternal blubber were 1.2 for heptachlor and 0.7 for PCNs (Figure 1), possibly indicating greater accumulation potential for these contaminants in tissues of less lipid content. Lower levels of persistent organic contaminants were reported in brain tissues of adult marine mammals [40,41]. Movement of OHs into brain may be controlled by the blood-brain barrier, specific transporters and chemical composition of the brain matter [40]. However, the relatively high levels of some contaminants, e.g., Σ PBDEs and Σ PCNs in fetal brains may suggest that the blood brain barrier was ineffective for blocking these OHs entering into the fetal brains.

3.2 Distribution of Homologues and Congeners in Maternal and Fetal Tissues

OCPs—Among the OCPs, *p,p'*-DDE was dominant in all maternal and fetal tissues (Figure 2A). Mean percentage contributions (*p,p'*-DDE/ Σ DDTs) ranged from 21.4 % in the fetal brain to 72.0 % in the maternal blubber. The percentage contribution of *p,p'*-DDE to Σ OCPs was the highest in the maternal blubber and decreased from the maternal blubber, liver to the placenta. Similar trends were found for *p,p'*-DDE in the fetal blubber, liver and brain. However, contributions of other OCP compounds varied a great deal. The α -, β -, γ -HCHs showed higher contributions in the fetal blubber, liver and brain tissues than the maternal tissues, implying a preferential maternal transfer of HCH isomers into the fetus (Figure 2A). Heptachlor (heptachlor/ Σ OCPs) showed high contributions in the placenta, fetal liver and brain (Figure 2A). HCB concentrations were similar among all maternal and fetal tissues. Difference in OH distribution among tissues suggest that pathways for accumulation are chemical and tissue specific (Figure 2A).

PCBs—Tri-, tetra-, penta-, hexa- and hepta-CB homologues were dominant in all maternal and fetal tissues (Figure 2B). Contributions of hexa-CBs, hepta-CBs and octa-CBs were in a decreasing order: maternal blubber > liver > placenta and fetal blubber > liver > brain. Contributions of di-CBs, tri-CBs and tetra-CBs increased in an order of maternal blubber > liver > placenta and in an order of fetal blubber > liver > brain. An approximately equal contribution of penta-CBs was found among the maternal and fetal tissues (Figure 2B). These results demonstrated that the lower chlorinated PCBs are transferred and accumulated in the fetal tissues more readily than higher chlorinated PCBs. The higher lipophilicity of the higher chlorinated PCBs could inhibit their maternal transfer and thus maintain their predominance in the maternal side (21). The marker PCBs covered 45.2–52.6%, 32.5–41.7% and 27.8–38.1% of Σ PCBs in the maternal blubber and liver and placenta, respectively, and 28.2–50.7%, 28.2–34.4% and 27.1–31.6% of Σ PCBs in the fetal blubber, liver and brain, respectively. The relatively high percentages demonstrated the usefulness of the marker PCBs to evaluate exposure.

Homologue contributions increased from mono-CBs (0%) to hexa-CBs and then decreased to deca-CBs, with hexa-CBs being the most dominant in the maternal blubber and liver tissues (Figure 2B). In the placenta, PCB homologue contribution increased from mono-CBs (0%) to tetra-CBs and then decreased to deca-CBs, with tetra-CBs being the most dominant (Figure 2B). Distributions of PCB homologues in the fetal blubber, liver and brain were very similar to those in the placenta. PCB homologue contributions increased from mono-CBs to tetra-CBs and then decreased to deca-CBs, with tetra-CBs being dominant (Figure 2B). This unique distribution further suggested mono- to tetra-CBs transferred preferentially to the fetal bodies than hexa-, hepta- and octa-CBs via placenta. After maternal transfer, a different distribution of PCB homologues was observed in the fetal blubber, liver and brain. The differential distribution of PCB homologues in pregnant female harbor seals and their fetuses suggests that hexa-CBs be a dominant homologue in the maternal blubber and liver tissues, while tetra-CBs were dominant homologues in the placental and fetal tissues. This different preferential transfer of PCB homologues via placenta might cause differential PCB homologue distributions between the adult males and females.

PCNs—Tetra-, penta- and hexa-CN homologues were major homologues in the maternal blubber and covered 24.5%, 32.1%, and 29.5 %, respectively. Tri-, tetra-, penta-, hepta-CN turned to be major homologues in the maternal liver, while tetra-CN was dominant (37.3% – 53.5%) in the fetal brain, liver and placenta. Hepta-CN (CN 73/74 as major congeners) contributed 12.3%–18.0% in the maternal liver and in the fetal blubber, liver, brain and placenta (Figure 2C), which were comparable to tri-CN in these tissues (8.4% – 14.4%). CN 73 contributed 17.5% to Σ PCNs in human serum, which the relatively high CN 73 value in the human serum samples might be due to the contribution from combustion sources [42]. Combustion could also be the source of CN 73 in the harbor seals.

PBDEs—Although 27 PBDE congeners were analyzed, only PBDEs 28, 47 and 99 were dominant congeners, but no distinct pattern of tissue distribution was noted (data not shown).

3.3. Effectiveness of placenta as OH barrier

The placenta is an important tissue for comparing maternal transfer ratios of OHs. Table 3 lists the ratios of OH concentrations in the fetal blubber, liver and brain to those in placenta. For Σ HCHs, HCB, Σ DDTs, Σ OCPs, Σ PCBs and Σ PBDEs, the mean ratios of the fetal blubber to placenta were in the range of 3.5 to 11.3, while the mean ratios of the fetal liver (0.76–1.6) and fetal brain (0.4–2.7) to placenta were much lower, indicating that Σ HCHs, HCB, Σ DDTs, Σ OCPs, Σ PCBs and Σ PBDEs readily penetrate the placenta and accumulate in the blubber of the fetus in utero (Table 3). For HCHs, PCBs and PBDEs, the mean ratios of the fetal liver to placenta were greater than 1 (Table 3), indicating that HCHs, PCBs and PBDEs cross the placenta and preferentially accumulate in the fetal liver. Only for PCNs and heptachlor were the mean ratios of fetal blubber to placenta lower than fetal liver to placenta and fetal brain to placenta (Table 3), indicating that PCNs and heptachlor cross the placenta to accumulate in fetal tissues.

Similar concentration profiles were found between the fetal blubber and the maternal blubber, and between the fetal liver and the maternal liver, therefore, it would be reasonable to express the maternal transfers by using fetal to maternal (FM) blubber OH concentration ratios (FM blubber ratios) (Figure 1) or FM liver OH concentration ratios (FM liver ratios) (Table 4). Σ DDTs, Σ OCPs and Σ PCBs had similar FM blubber ratios (0.10, 0.16, and 0.13, respectively) (Figure 1 and Table 4), which were lower than mean FM blubber ratios (0.45 by ww and 0.97 by lw) for PCBs and mean FM blubber ratios (0.5 by ww and 1.1 by lw) for DDTs in 20 mother-fetus pairs of California sea lions [21]. The FM blubber ratios observed

in the present study were also lower than mean Σ PCB ratios (0.8) and mean Σ DDT ratios (1.1) in long-finned pilot whales from the Faroe Islands [19] and in pregnant striped dolphins [17]. For HCHs, HCB, heptachlor, PBDEs and PCNs, the FM blubber ratios ranged from 0.25 to 0.43, which is much greater than those of PCBs and DDTs we analyzed (Table 4). The FM blubber ratios of PBDEs in the harbor seals were comparable to the FM ratios of BDE 47 (0.47) and 99 (0.25) at the steady state in a human placenta perfusion system [43]. It suggests that HCHs, HCB, heptachlor, PBDEs and PCNs would be more transferable from the harbor seal mothers to their fetuses via placenta compared with PCBs and DDTs (Figure 1, and Table 4). Interestingly, although not identical to those FM blubber ratios, the FM liver ratios of HCB, DDTs, OCPs and PCBs were all less than 1, while the FM liver ratios of HCHs, heptachlor, PBDEs and PCNs were all greater than 1 (Table 4). These results further demonstrated that HCHs, heptachlor, PBDEs and PCNs are more transferable than DDTs, OCPs and PCBs. As estimated by body burdens of two pairs of melon headed whale mothers and fetuses obtained from Japanese coasts, maternal transfer rates of HCHs (5.6–6.0%) and HCB (3.7–5.0%) through gestation were also higher than those of PBDEs (2.6–3.5%) and PCBs (3.5–3.6%), indicating that HCHs and HCB are more transferrable than PCBs and PBDEs [44]. These conclusions concur with those from the harbor seals in the present study. However, results of PBDEs from the whales [44] differed from the harbor seals in the present study, suggesting that there exists a species and chemical specific differences in maternal transfer. Molecular size, shape and electronic properties of contaminants [43,45] and differences in affinity for carrier proteins (e.g., albumin) may control the transport of various congeners [46].

4. Conclusions

A significant amount of OHs were transferred from mother harbor seals to fetuses during pregnancy and distributed into at least three fetal organs. Concentration profiles of PCBs, OCPs, PBDEs and PCNs in fetal tissues resembled those in their mothers; however, maternal and fetal distribution of these contaminants was tissue and chemical specific. Of the three fetal tissues analyzed, OH concentrations were the highest in the fetal blubber. Concentrations of PCBs, OCPs and PBDEs were remarkably similar among the maternal liver, placenta, and fetal liver (except for HCHs), possibly indicating that the placenta was neutral for all of the compounds analyzed. HCH, HCB and low halogenated congeners of PCBs, PCNs and PBDEs were preferentially transferred from mothers to fetuses, while *p,p'*-DDE and highly halogenated congeners of PCBs, PCNs and PBDEs were relatively retained in the maternal tissues. Both FM blubber ratios and FM liver ratios showed that PCNs and PBDEs are more transferrable than OCPs and PCBs, while HCHs and heptachlor are more transferable than HCB and DDTs. Prenatal transfer of OHs even at a low concentration may pose a threat to the critical early development of the fetuses. The retention of some toxic congeners in the mother seals may serve as protection to the developing fetus.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Harbor seal tissue samples were generously donated by the Alaska Native Harbor Seal Commission Biosampling Program. Dr. Gail Blundell of Alaska Department of Fish and Game kindly provided the tooth aging data. This study was supported in part by the Alaska SeaLife Center, with funds from the National Marine Fisheries Service (NMFS research permit numbers: 881–1724) and by the National Institute on Minority Health and Health Disparities (G12 RR003061 and G12 MD007601). The authors sincerely thank Lori Quakenbush and an anonymous reviewer for their constructive and throughout comments and suggestions to improve this paper.

References

1. Boveng PL, Bengtson JL, Withrow DE, Cesarone JC, Simpkins MA, Frost KJ, Burns JJ. The abundance of harbor seals in the Gulf of Alaska. *Mar. Mam. Sci.* 2003; 19:111–127.
2. Small RJ, Boveng PL, Vernon Byrd G, Withrow DE. Harbor seal population decline in the Aleutian Archipelago. *Mar. Mam. Sci.* 2008; 24:845–863.
3. Krahn MM, Becker PR, Tilbury KL, Stein JE. Organochlorine contaminants in blubber of four seal species: integrating biomonitoring and specimen banking. *Chemosphere.* 1997; 34:2109–2121. [PubMed: 9159908]
4. Wang D, Atkinson S, Hoover-Miller A, Lee SE, Li QX. Organochlorines in harbor seal (*Phoca vitulina*) tissues from the northern Gulf of Alaska. *Environ. Pollut.* 2007; 146:268–480. [PubMed: 16938369]
5. Wang D, Atkinson S, Hoover-Miller A, Li QX. Polychlorinated naphthalenes and coplanar polychlorinated biphenyls in tissues of harbor seals (*Phoca vitulina*) from the northern Gulf of Alaska. *Chemosphere.* 2007; 67:2044–2057. [PubMed: 17223166]
6. Kannan K, Moon HB, Yun SH, Agusa T, Thomas NJ, Tanabe S. Chlorinated, brominated, and perfluorinated compounds, polycyclic aromatic hydrocarbons and trace elements in livers of sea otters from California, Washington, and Alaska (USA), and Kamchatka (Russia). *J. Environ. Monit.* 2008; 10:552–558. [PubMed: 18385877]
7. Neale JCC, Small RJ, Schmelzer KR, Tjeerdema RS. Blood concentrations of some persistent organohalogenes in free-ranging spotted seals (*Phoca largha*) from Bristol Bay, Alaska. *J. Toxicol. Environ. Health A.* 2007; 70:1776–1778. [PubMed: 17885935]
8. Quakenbush LT. Polybrominated diphenyl ether compounds in ringed, bearded, spotted, and ribbon seals from the Alaskan Bering Sea. *Mar. Pollut. Bull.* 2007; 54:232–236. [PubMed: 17169381]
9. Muir DC, Backus S, Derocher AE, Dietz R, Evans TJ, Gabrielsen GW, Nagy J, Norstrom RJ, Sonne C, Stirling I, Taylor MK, Letcher RJ. Brominated flame retardants in polar bears (*Ursus maritimus*) from Alaska, the Canadian Arctic, East Greenland, and Svalbard. *Environ. Sci. Technol.* 2006; 40:449–455. [PubMed: 16468388]
10. Mos L, Morsey B, Jeffries SJ, Yunker MB, Raverty S, de Guise S, Ross PS. Chemical and biological pollution contribute to the immunological profiles of free-ranging harbor seals. *Environ. Toxicol. Chem.* 2006; 25:3110–3117. [PubMed: 17220078]
11. Aguilar A, Borrell A. Reproductive transfer and variation of body load of organochlorine pollutants with age in fin whales (*Balaenoptera physalus*). *Arch. Environ. Contam. Toxicol.* 1994; 27:546–554. [PubMed: 7811111]
12. Ross PS, Ellis GM, Ikonomou MG, Barrett-Lennard LG, Addison RF. High PCB concentrations in free-ranging Pacific killer whales, *Orcinus orca*: Effects of age, sex and dietary preference. *Mar. Pollut. Bull.* 2000; 40:504–515.
13. Beckmen KB, Ylitalo GM, Towell RG, Krahn MM, O'Hara TM, Blake JE. Factors affecting organochlorine contaminant concentrations in milk and blood of northern fur seal (*Callorhinus ursinus*) dams and pups from St. George Island, Alaska. *Sci. Tot. Environ.* 1999; 231:183–200.
14. Debier C, Pomeroy PP, Dupont C, Joiris C, Comblin V, Le Boulenger E, Larondelle Y, Thome J-P. Quantitative dynamics of PCB transfer from mother to pup during lactation in UK gray seals (*Halichoerus grypus*). *Mar. Ecol. Prog. Ser.* 2003; 247:237–248.
15. Jacobson JL, Jacobson SW. Association of prenatal exposure to an environmental contaminant with intellectual function in childhood. *J. Toxicol. Clin. Toxicol.* 2002; 40:467–475. [PubMed: 12216999]
16. Gauger KJ, Kato Y, Haraguchi K, Lehmler H, Robertson LW, Bansal R, Zoeller RT. Polychlorinated biphenyls (PCBs) exert thyroid hormone-like effects in the fetal brain but do not bind to thyroid hormone receptors. *Environ. Health. Persp.* 2004; 112:516–523.
17. Tanabe S, Tatsukawa R, Maruyama K, Miyazaki N. Transplacental transfer of PCBs and chlorinated hydrocarbon pesticides from the pregnant striped dolphin (*Stenella coeruleoalba*) to her fetus. *Agric. Bio. Chem.* 1982; 46:1249–1254.

18. Duinker JC, Hillebrand MTJ. Mobilization of organochlorines from female lipid tissue and transplacental transfer to fetus in a harbor porpoise (*Phocoena phocoena*) in a contaminated area. *Bull. Environ. Contam. Toxicol.* 1979; 23:728–732. [PubMed: 117861]
19. Borrell A, Bloch D, Desportes G. Age trends and reproductive transfer of organochlorine compounds in long-finned pilot whales from the Faroe Islands. *Environ. Pollut.* 1995; 88:283–292. [PubMed: 15091540]
20. Donkin P, Mann SV, Hamilton EI. Polychlorinated biphenyl, DDT, and dieldrin residues in gray seal (*Halichoerus grypus*) males, females, and mother—fetus pairs sampled at the Farne Islands, England, during the breeding season. *Sci. Tot. Environ.* 1981; 19:121–142.
21. Greig DJ, Ylitalo GM, Hall AJ, Fauquier DA, Gulland F. Transplacental transfer of organochlorines in California sea lions (*Zalophus californianus*). *Environ. Toxicol. Chem.* 2007; 26:37–44. [PubMed: 17269457]
22. Hart K, Kannan K, Isobe T, Takahashi S, Yamada TK, Miyazaki N, Tanabe S. Time trends and transplacental transfer of perfluorinated compounds in melon-headed whales stranded along the Japanese coast in 1982, 2001/2002, and 2006. *Environ. Sci. Technol.* 2008; 42:7132–7137. [PubMed: 18939537]
23. Blundell GM, Pendleton GW. Estimating age of harbor seals (*Phoca vitulina*) with incisor teeth and morphometrics. *Mar. Mam. Sci.* 2008; 24:577–590.
24. Wang D, Atkinson S, Hoover-Miller A, Li QX. Analysis of organochlorines in harbor seal (*Phoca vitulina*) tissue samples from Alaska using gas chromatography/ion trap mass spectrometry by an isotopic dilution technique. *Rapid Commun. Mass Spectrom.* 2005; 19:1815–1821. [PubMed: 15945031]
25. Wang D, Atkinson S, Hoover-Miller A, Shelver WL, Li QX. Simultaneous use of gas chromatography/ion trap mass spectrometry - electron capture detection to improve the analysis of bromodiphenyl ethers in biological and environmental samples. *Rapid Commun. Mass Spectrom.* 2008; 22:647–656. [PubMed: 18265429]
26. Wang D, Shelver WL, Atkinson S, Mellish JA, Li QX. Tissue distribution of polychlorinated biphenyls and organochlorine pesticides and potential toxicity to Alaskan northern fur seals assessed using PCBs congener specific mode of action schemes. *Arch. Environ. Contam. Toxicol.* 2010; 58:478–488. [PubMed: 19789908]
27. Harner T, Bidleman TF. Polychlorinated naphthalenes in urban air. *Atmos. Environ.* 1997; 31:4009–4016.
28. Asplund L, Jakobsson E, Haglund P, Bergman Å. 1,2,3,5,6,7-Hexachloronaphthalene and 1,2,3,4,6,7-hexachloronaphthalene -selective retention in rat liver and appearance in wildlife. *Chemosphere.* 1994; 28:2075–2086.
29. Wiestrand C, Noren K. Polychlorinated naphthalenes and other organochlorine contaminants in human adipose and liver tissue. *J. Toxicol. Environ. Health.* 1998; 53:293–311.
30. Yordy JE, Pabst DA, McLellan WA, Wells RS, Rowles TK, Kucklick JR. Tissue-specific distribution and whole-body burden estimates of persistent organic pollutants in the bottlenose dolphin (*Tursiops truncatus*). *Environ. Toxicol. Chem.* 2010; 29:1263–1273. [PubMed: 20821568]
31. Dunkin RC, McLellan WA, Blum JE, Pabst DA. The ontogenetic changes in the thermal properties of blubber from Atlantic bottlenose dolphin (*Tursiops truncatus*). *J. Exp. Bio.* 2005; 208:1469–1480. [PubMed: 15802671]
32. Boon JP, Oostingh I, van der Meer J, Hillebrand MTJ. A model for the bioaccumulation of chlorobiphenyl congeners in marine mammals. *Eu. J. Pharm. Environ. Toxicol.* 1994; 270:237–251.
33. Bentzen TW, Muir DC, Amstrup SC, O'Hara TM. Organohalogen concentrations in blood and adipose tissue of Southern Beaufort Sea polar bears. *Sci. Tot. Environ.* 2008; 406:352–367.
34. Polishuk ZW, Ron M, Wassermann M, Cucos S, Wassermann D, Lemesch C. Pesticides in people. Organochlorine compounds in human blood plasma and milk. *Pestic. Monit. J.* 1977; 10:121–129. [PubMed: 857240]
35. Polishuk ZW, Wassermann D, Wassermann D, Cucos S, Ron M. Organochlorine compounds in mother and fetus during labor. *Environ. Res.* 1977; 13:278–294. [PubMed: 405211]

36. Troisi GM, Mason CF. PCB-associated alteration of hepatic steroid metabolism in harbor seals (*Phoca vitulina*). *J. Toxicol. Environ. Health A*. 2000; 61:649–655. [PubMed: 11132695]
37. Addison RF, Brodie PF, Edwards A, Sadler MC. Mixed function oxidase activity in the harbour seal (*Phoca vitulina*) from Sable Is., N.S. *Comp. Biochem. Physiol. C*. 1986; 85:121–124. [PubMed: 2877778]
38. Birnbaum LS, Staskal DF. Brominated flame retardants: cause for concern? *Environ. Health Persp.* 2004; 112:9–17.
39. Hamers T, Kamstra JH, Sonneveld E, Murk AJ, Kester MHA, Andersson PL, Legler J, Brouwer A. *In vitro* profiling of the endocrine-disrupting potency of brominated flame retardants. *Toxicol. Sci.* 2006; 92:157–173. [PubMed: 16601080]
40. Tilbury KL, Stein JE, Meador JP, Krone CA, Chan S-L. Chemical contaminants in harbour porpoise (*Phocoena phocoena*) from the north Atlantic coast: Tissue concentrations and intra- and inter-organ distribution. *Chemosphere*. 1997; 34:2159–2181. [PubMed: 9159912]
41. Duinker JC, Hillebrand MTJ, Zeinstra T, Boon JP. Individual chlorinated biphenyls and pesticides in tissues of some cetacean species from the North Sea and the Atlantic Ocean: Tissue distribution and biotransformation. *Aquat. Mam.* 1989; 15:95–124.
42. Park H, Kang JH, Baek SY, Chang YS. Relative importance of polychlorinated naphthalenes compared to dioxins and polychlorinated biphenyls in human serum from Korea: contribution to TEQs and potential sources. *Environ. Pollut.* 2010; 158:1420–1427. [PubMed: 20089337]
43. Frederiksen M, Vorkamp K, Mathiesen L, Mose T, Knudsen LE. Placental transfer of the polybrominated diphenyl ethers BDE-47, BDE-99 and BDE-209 in a human placenta perfusion system: an experimental study. *Environ. Health.* 2010; 5:9–32.
44. Kajiwarra N, Kamikawa S, Amano M, Hayano A, Yamada TK, Miyazaki N, Tanabe S. Polybrominated diphenyl ethers (PBDEs) and organochlorines in melon-headed whales *Peponocephala electra*, mass stranded along the Japanese coasts: Maternal transfer and temporal trend. *Environ. Pollut.* 2008; 156:106–114. [PubMed: 18272274]
45. Guvenius DM, Aronsson A, Ekman-Ordeberg G, Bergman A, Norén K. Human prenatal and postnatal exposure to polybrominated diphenyl ethers, polychlorinated biphenyls, polychlorobiphenyls, and pentachlorophenol. *Environ. Health Persp.* 2003; 111:1235–1141.
46. Syme MR, Paxton JW, Keelan JA. Drug transfer and metabolism by the human placenta. *Clin. Pharmacokinet.* 2004; 43:487–514. [PubMed: 15170365]

Highlights

► Significant amounts of organic halogenated contaminants (OHs) were transferred from maternal harbor seals to their fetuses. ► HCH, HCB, PCNs, PBDEs and low chlorinated PCBs were preferentially transferred from mothers to fetuses. ► *p,p'*-DDE and highly chlorinated PCBs were retained in the mother to a greater extent. ► The retention of toxic congeners in the mother seals could threaten future reproductive success. ► Prenatal transfer of OHs could pose a threat to the critical early development of the fetuses.

251658240

Figure 1.

Mean ratios of OHs (ng/g ww) of maternal liver, placenta, fetal blubber, liver and brain versus maternal blubber. ΣPCBs included 145 congeners [26]; ΣOCPs include ΣHCH, HCB, heptachlor and ΣDDTs; ΣDDTs is the total concentration of *o,p'*-DDE, *p,p'*-DDE, *o,p'*-DDD; *p,p'*-DDD; *o,p'*-DDT, and *p,p'*-DDT; ΣPBDEs is the total concentration of 27 congeners [25]; ΣHCH is the total concentration of α, β, γ, and δ-HCH isomers; ΣPCNs is the sum of the concentrations of individual congeners identified in Halowax 1014 [27]. The ratios in Figure 1 were calculated by averaging the ratios of the two-compared tissues of the 6-paired maternal and fetal seals rather than by using the data in Table 2.

Figure 2. Percentage distribution of OCP congeners (A), PCB homologues (B), and PCN homologues, (C) in maternal and fetal tissues. α , β , γ , and δ -HCH isomers were calculated on Σ HCH. o,p' -DDE, p,p' -DDE, o,p' -DDD, p,p' -DDD, o,p' -DDT, and p,p' -DDT were based on Σ DDTs; HCB, and heptachlor were calculated based on Σ OCPs include Σ DDTs, Σ HCH, HCB and heptachlor.

Table 1

Specimen information for maternal harbor seals and their fetuses

Identification No.	Age (y)	Collection date M/D/Y	Length (cm)	Mass (kg)	Blubber thickness (mm)	Fetus mass (g)
18877	3	02/09/2000	131	59.9	60	2100
18858	7	01/14/2000	144	91.7	45	1672
47050	10	02/02/2001	141	84.0	35	2151
47109	4	11/24/2001	122	63.1	33	111
47116	4	11/18/2001	126	50.8	40	232
47271	7	01/31/2002	135	ND ^a	41	1553

^aND, not determined.

Overall mean concentrations (ng/g ww) $\pm$ standard deviations of each chemical group in different tissues of the six-paired maternal and fetal harbor seals

Table 2

Chemical groups ^a	Maternal tissues		Fetal tissues			
	blubber (n=6)	liver (n=5)	placenta (n=6)	blubber (n=6)	liver (n=6)	brain (n=6)
Σ HCHs	8.4 $\pm$ 3.8	0.23 $\pm$ 0.09	0.43 $\pm$ 0.24	3.3 $\pm$ 2.4	0.56 $\pm$ 0.33	0.21 $\pm$ 0.21
HCB	3.6 $\pm$ 0.63	0.16 $\pm$ 0.03	0.22 $\pm$ 0.25	0.11 $\pm$ 0.040	0.11 $\pm$ 0.04	0.07 $\pm$ 0.05
Heptachlor	0.32 $\pm$ 0.38	0.15 $\pm$ 0.09	0.62 $\pm$ 0.58	0.09 $\pm$ 0.02	0.26 $\pm$ 0.18	0.20 $\pm$ 0.15
Σ DDTs	121.6 $\pm$ 67.2	2.6 $\pm$ 1.4	1.5 $\pm$ 1.3	8.0 $\pm$ 6.6	1.1 $\pm$ 1.0	0.44 $\pm$ 0.38
Σ PCBs	148.1 $\pm$ 37.8	6.9 $\pm$ 2.3	8.4 $\pm$ 4.4	16.9 $\pm$ 8.3	7.2 $\pm$ 4.9	7.0 $\pm$ 6.4
Σ PBDEs	14.6 $\pm$ 7.7	0.60 $\pm$ 0.52	0.59 $\pm$ 0.31	5.1 $\pm$ 2.7	0.71 $\pm$ 1.2	2.1 $\pm$ 4.9
Σ PCNs	0.96 $\pm$ 0.38	0.74 $\pm$ 0.31	2.8 $\pm$ 3.2	0.32 $\pm$ 0.18	0.87 $\pm$ 0.50	0.76 $\pm$ 0.73
Σ OCPs	144.3 $\pm$ 65.0	3.4 $\pm$ 1.4	3.2 $\pm$ 1.4	15.5 $\pm$ 11.2	2.6 $\pm$ 1.6	1.1 $\pm$ 0.69

Σ HCHs is the sum of the concentrations of α , β , γ , and δ -HCH isomers; Σ DDTs is the sum of the concentrations of o,p' -DDE, p,p' -DDE, o,p' -DDD, p,p' -DDD, and p,p' -DDT; Σ OCPs is the sum of Σ HCHs, Σ DDTs, HCB and heptachlor. Σ PCBs is the sum of the concentrations of 145 PCB congeners [26]; Σ PBDEs is the sum of the concentrations of 27 congeners [25]; Σ PCNs is the sum of the concentrations of 37 congeners identified in Halowax 1014 [27].

Table 3Concentration ratios of fetal tissues (blubber, liver and brain) to placenta^a

Chemical groups	fetal blubber/placenta	fetal liver/placenta	fetal brain/placenta
ΣHCHs	10.2±7.6	1.4±0.65	0.58±0.76
HCB	8.9±9.8	0.93±0.80	0.66±0.45
Heptachlor	0.26±0.20	0.93±1.4	0.73±0.93
ΣDDTs	9.7±8.2	0.76±0.52	0.40±0.40
ΣOCPs	6.9±5.8	0.87±0.53	0.45±0.43
ΣPCBs	3.5±3.6	1.4±1.8	0.94±0.68
ΣPBDEs	11.3±7.0	1.6±3.1	2.7±6.2
ΣPCNs	0.17±0.065	0.58±0.60	0.57±0.80

^aThe ratios were calculated by averaging the ratios of the two-compared tissues of the 6-paired maternal and fetal seals rather than by using the overall mean concentrations provided in Table 2.

Table 4Concentration ratios of fetal blubber to maternal blubber and fetal liver to maternal liver^a

Chemical groups	Concentration ratios $\pm$ standard deviations	
	fetal blubber /maternal blubber	fetal liver /maternal liver
Σ HCHs	0.37 $\pm$ 0.28	2.3 $\pm$ 1.4
HCB	0.25 $\pm$ 0.17	0.59 $\pm$ 0.18
Heptachlor	0.58 $\pm$ 0.42	2.8 $\pm$ 3.0
Σ DDTs	0.10 $\pm$ 0.084	0.33 $\pm$ 0.23
Σ OCPs	0.16 $\pm$ 0.13	0.69 $\pm$ 0.43
Σ PCBs	0.13 $\pm$ 0.087	0.89 $\pm$ 0.64
Σ PBDEs	0.43 $\pm$ 0.26	1.1 $\pm$ 0.95
Σ PCNs	0.42 $\pm$ 0.36	1.5 $\pm$ 1.4

^aThe ratios were calculated by averaging the ratios of the two-compared tissues of the 6-paired maternal and fetal seals rather than by using the data in Table 2.