Brominated Flame Retardants and Halogenated Phenolic Compounds in North American West Coast Bald Eaglet (*Haliaeetus leucocephalus*) Plasma

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We report on the identity, characterization, and spatial trends of several brominated flame retardants and hydroxylated (OH-) and methoxylated (MeO-) organohalogen contaminants in bald eagle (*Haliaeetus leucocephalus*) nestling plasma collected from sites along the west coast of North America. Samples were from four southwestern British Columbia (BC) locations, a reference site in northern BC (Fort St. James; FSJ), and from Santa Catalina Island, CA (SCI), an area of high DDT and PCB contamination. Mean concentrations of Σpolybrominated diphenyl ether (ΣPBDE (8 congeners monitored); 1.78−8.49 ng/g), ΣOH-polychlorinated biphenyl (ΣOH-PCB (30 congeners monitored); 0.44−0.87 ng/g), and ΣOH-PBDE (14 congeners monitored; 0.31−0.92 ng/g) were similar in eaglets from southwestern BC yet lower than for SCI and significantly higher than for FSJ. Dominant PBDE congeners were BDE47, BDE99, and BDE100, but SCI eaglets also contained low levels of higher brominated congeners. 4-OH-CB187 and 4′-OH-CB202 accounted for 65−100% of ΣOH-PCB in all BC eaglets, with 4′-OH-CB202 as well as 3′-OH-CB138 and 4-OH-CB146 dominating in SCI eaglets. Ostensibly of biogenic origin, 6′-OH-BDE49 and 6-OH-BDE47 were found in BC nestlings. Only 4′-OH-BDE49 (2.10 ng/g) was found in SCI eaglets. MeO-PBDEs and total hexabromocyclododecane (HBCD) were not found in any birds, but the polybrominated biphenyl BB101 was detected in southwestern BC samples. This study demonstrates that west coast North American bald eagles contain previously unreported organohalogens, which have the potential to impact the health and survival of these raptors.

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Introduction

The bald eagle (*Haliaeetus leucocephalus*) is an ideal sentinel species for monitoring the levels and effects of organohalogen contaminant exposure in the North American environment (*1*). As is the case for other predatory birds occupying top trophic positions, many bald eagle populations have exhibited toxicological effects such as eggshell thinning, reproductive and developmental challenges, and mortality associated with contaminant exposure (e.g., 2,2-bis(4-chlorophenyl)-1,1-dichloroethene (DDE) and other pesticides, polychlorinated biphenyls (PCBs), mercury, and lead) (*2*). Linked to high organochlorine contaminant exposures, populations were extirpated from the southern coastal Californian Channel Islands in the early 1960s (*3*) but were reintroduced to the island of Santa Catalina more than 20 years ago. Further up the west coast of North America, the populations sizes of bald eagles in British Columbia (BC) have not decreased to the same extent, but some populations in southwestern coastal BC have exhibited reduced nesting success, which may be related to industrial activities in the region and related contaminant exposures (*4*). Bald eagles from the Fort St. James area in northern BC are geographically separated from large urban and industrial zones and thus can be considered as a low organochlorine exposure site.

Brominated flame retardants (BFRs), including polybrominated biphenyls (PBBs), hexabromocyclododecane (HBCD), and particularly polybrominated diphenyl ethers (PBDEs), are additives in various commercial products for protection from fire ignition. Regulation is increasing with respect to commercial use (e.g., PentaBDE technical mixtures), but there is a large amount of PBDEs available in the technosphere, and they are thus of significant environmental concern (*5*). Similar to PCBs, the physicochemical properties of PBDEs render them persistent and bioaccumulative. Temporal studies in birds have shown exponentially increasing PBDE levels over the last 20 years in the eggs of double-crested cormorant (*Phalacrocorax auritus*) and great blue heron (*Ardea herodias*), two species of fish-eating birds in southwestern BC (*6*). PBDEs have been determined in Great Lakes bald eagle nestling plasma (*7*), but there are no reports of BFRs in any raptor species or populations from the western coastal areas of North America.

In toxicity studies, PBDE exposure was linked to altered neural development, endocrine, liver, and reproductive function in laboratory rodents (*5*). Although knowledge of the effects of PBDE exposure in birds is limited, Fernie et al. (*8*) recently reported decreased plasma thyroxine (T_4) and vitamin A levels as well as indications of oxidative stress in American kestrels (*Falco sparverius*) dosed with a PBDE mixture *in ovo* and post-hatch.

The toxicokinetics of PCBs and particularly PBDEs are not well understood in birds (*9*), but putative metabolites of both organohalogen classes have been reported in the tissues of certain avian species (*10*, *11*). Several OH-PCB congeners were recently quantified in Faroe Island fulmar (*Fulmarus glacialis*) eggs (*12*) and in glaucous gull (*Larus hyperboreus*) eggs and adult plasma from the Norwegian Arctic (*13*). The presence of OH-PCBs in the plasma of birds and other wildlife is more than likely due to oxidative cytochrome P450 (CYP)-mediated PCB biotransformation and subsequent retention of selected congeners in blood via competitive binding to thyroid hormone transport proteins, e.g., transthyretin (TTR) (*14*). It is this binding affinity that has been linked to alterations of thyroid hormone and vitamin A levels in OH-PCB exposed laboratory rats (*15*).

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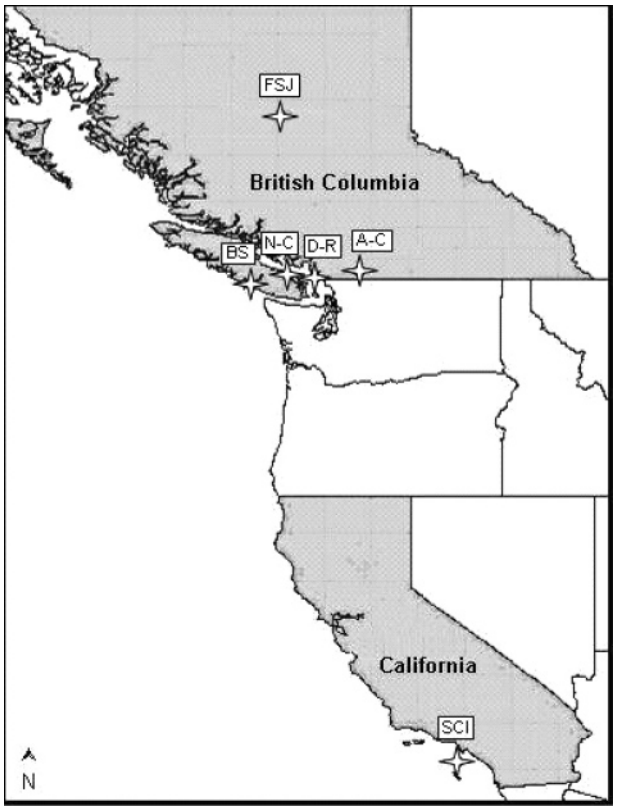

FIGURE 1. Map showing the location of nestling bald eagle sites sampled from British Columbia and southern California. Sites are as follows: FSJ = Fort St. James area ($n = 4$), BS = Barkley Sound ($n = 3$), N−C = Nanaimo/Crofton area ($n = 7$), D−R = Delta−Richmond area ($n = 6$), A−C = Abbotsford−Chilliwack area ($n = 6$), and SCI = Santa Catalina Island ($n = 3$, pooled).

OH-PBDEs as well as methoxylated- (MeO-) PBDEs have now also been quantified in a very limited number of bird species (*10*, *16*). Although the importance remains unclear as a degradation mechanism, oxidative biotransformation of PBDEs and subsequent metabolite formation has been demonstrated in a few studies in rats, certain fish species, and beluga whales (*9*, *17*). MeO-PBDEs and some OH-PBDE congeners can also bioaccumulate in aquatic food webs as natural products produced by marine organisms such as sponges and algae (*18*, *19*). Regardless of origin, estrogenic and thyroidogenic dysfunction have been reported in laboratory rats exposed to OH-PBDEs (*20*, *21*).

The present study identifies and quantifies brominated flame retardants (PBDEs, PBBs and total-HBCD) as well as OH-PCBs, OH-PBDEs, MeO-PBDEs, and 4-OH-heptachlorostyrene (4-OH-HpCS) in the plasma of bald eagle nestlings from the west coast of North America. Spatial differences in the concentrations and congener patterns of these unique and emerging environmental contaminants were assessed among coastal populations of western Canada and the United States.

Experimental Section

Sample Collection. Nestling bald eagle blood samples were collected from five BC sites and one California site (Figure 1), which were chosen to reflect variation in contaminant exposure due to differing current and historic anthropogenic activities. Samples were collected in 2003 from southwestern BC and California sites and in 2001 from the Northern BC site. In BC, samples were obtained from Barkley Sound (BS, southwest Vancouver Island, $n = 3$), Nanaimo−Crofton (N−C, southeast Vancouver Island, $n = 7$), Delta−Richmond (D−R, lower Fraser Valley, $n = 6$), Abbotsford−Chilliwack (A−C, central Fraser Valley, $n = 6$), and Fort St. James (FSJ, northern British Columbia, $n = 4$). In California, samples were taken from Santa Catalina Island (SCI, southern California, $n = 3$, samples pooled prior to analysis). BC eaglets were first located by aerial or boating surveys and then removed from the nest. Body mass and age were determined (see Supporting Information). California nestlings were collected from rocky cliffs as part of a bald eagle reintroduction program carried out by the Institute for Wildlife Studies (Avalon, CA). Up to 24 mL of blood was collected from each nestling and immediately transferred to vacutainers containing sodium heparin. After <6 h on ice, plasma was separated by centrifugation and stored at −20 °C.

Chemicals and Standards. PBDEs, BB101, α-HBCD, and OH-PCB standards were purchased from Wellington Laboratories (Guelph, ON, Canada). OH-PBDE and MeO-PBDE standards were kindly synthesized and provided by Drs. Å. Bergman and G. Marsh (*22*). OH-PCBs were derivatized by diazomethane treatment to generate the corresponding MeO-PCB standards. PCB standards were provided by the Canadian Wildlife Service (Ottawa, ON, Canada). The internal standards used were as follows: BDE30 for PBDE and MeO-PBDE analyses, 2′-OH-BDE28 for OH-PBDE analysis, and a mixture of 12 ^{13}C-labeled OH-PCBs for OH-PCB analysis (Wellington Laboratories) (*16*, *23*, *24*). For separation and cleanup, the chromatographic supports were 1.2% water deactivated Florisil (Fisher Scientific, Ottawa, ON, Canada) and 22% sulfuric acid acidified silica (Aldrich, Milwaukee, WI). All other reagents and solvents were of analytical-grade quality or better.

Sample Extraction. Around 2.5 g of each plasma sample were accurately weighed, and neutral and phenolic fractions were extracted and cleaned up using established methodologies (e.g., refs *16*, *23*, and *24*). In brief, samples were spiked with the internal standards, acidified, denatured, and liquid−liquid extracted with 50:50 methyl *tert*-butyl ether (MtBE):hexane. Phenolic contaminants were extracted with 1 M potassium hydroxide, acidified, back-extracted with 50:50 MtBE:hexane, and converted to MeO analogues. The original organic phase containing neutral contaminants was separated and cleaned up on a Florisil column. Three fractions, 38 mL of hexane, 34 mL of 15:85 DCM:hexane, and 54 mL of 50:50 DCM:hexane, were collected and combined for PBDE, PBB, and HBCD analysis. Detailed PCB extraction, analysis, and quantification procedures for these bald eagle samples have been described and performed previously (*25*). Lipid content was determined colorimetrically using olive oil as the calibration standard (*26*).

Chemical Analysis and Quantification. Analytes were separated and quantified on an Agilent 6890N series GC with a fused silica DB-5 column ((5% phenyl)methylpolysiloxane; length, 30 m; inner diameter, 250 μm; film thickness, 0.25 μm; J&W Scientific, Folsom, CA) with a 5973N series quadrupole mass spectrometer (MS) (Agilent Technologies, Palo Alto, CA) in the electron capture, negative ionization (ECNI), and selected ion monitoring (SIM) modes. An Agilent 7683 series injector and autosampler were used. Helium and methane were used as the carrier and reagent gases, respectively. The GC ramping programs for all analyte classes have been detailed elsewhere (e.g., refs *16*, *23*, and *24*). PBDE, PBB, α-HBCD, derivatized OH-BDE, and MeO-PBDE congeners were monitored using the bromine anions of m/z 79 and 81 (*27*). Although the commercial product is dominated by γ-HBCD, the diastereomers of HBCD thermally rearrange at temperatures above 160 °C, i.e. under the GC conditions (*28*). GC analysis results in a broad peak of unresolved isomers representing the total-HBCD, although mainly composed of α-HBCD (*29*). α-HBCD is the dominant isomer, whereas β-HBCD is very minor, in wildlife samples (*5*). The meth-

TABLE 1. Arithmetic Mean ± * SE and Range of %Lipid of PBDE, PBB, Total (α) HBCD, PCB, and Octachlorostyrene (OCS) Concentrations (ng/g Wet Weight) in Nestling Bald Eagle Plasma Samples from British Columbia and Southern California Sites

	Fort St. James ($n = 4$)		Barkley Sound ($n = 3$)		Nanaimo–Crofton ($n = 7$)		Delta–Richmond ($n = 6$)		Abbotsford–Chilliwack ($n = 6$)		Santa Catalina Island ($n = 3$)[a]	
analyte	mean ± SE (range)	%n > MLOQ	mean ± SE (range)	%n > MLOQ	mean ± SE (range)	%n > MLOQ	mean ± SE (range)	%n > MLOQ	mean ± SE (range)	%n > MLOQ	mean ± SE (range)	%n > MLOQ
% lipid	0.70 ± 0.11 (0.53–1.02)	100	1.00 ± 0.12 (0.79–1.21)	100	1.06 ± 0.06 (0.81–1.29)	100	1.10 ± 0.12 (0.82–1.60)	100	0.87 ± 0.11 (0.47–1.10)	100	0.65 ± 0.07 (0.53–0.78)	100
BDE47	(<0.01–0.42)	25	1.64 ± 0.85 (0.27–3.20)	100	4.51 ± 1.10 (2.15–10.73)	100	1.60 ± 0.50 (0.47–3.54)	100	0.86 ± 0.18 (0.26–1.52)	100	17.45	100
BDE99	0.22 ± 0.11 (<0.01–0.53)	75	1.20 ± 0.80 (0.14–2.78)	100	1.72 ± 0.46 (0.35–3.43)	100	0.86 ± 0.20 (0.23–1.67)	100	0.35 ± 0.11 (0.10–0.85)	100	4.15	100
BDE100	(<0.01–0.30)	25	0.71 ± 0.35 (0.13–1.33)	100	1.94 ± 0.48 (1.02–4.78)	100	0.77 ± 0.18 (0.34–1.48)	100	0.44 ± 0.09 (0.25–0.83)	100	3.69	100
BDE138	<0.01	0	<0.01	0	<0.01	0	<0.01	0	<0.01	0	2.85	100
BDE153	<0.01	0	0.22 ± 0.11 (0.08–0.43)	100	0.31 ± 0.08 (0.02–0.53)	100	0.64 ± 0.38 (<0.01–2.28)	83	0.13 ± 0.02 (0.07–0.21)	100	0.74	100
BDE154/BB153	<0.01	0	<0.01	0	<0.01	0	<0.01	0	<0.01	0	0.30	100
BDE183	<0.01	0	<0.01	0	<0.01	0	<0.01	0	<0.01	0	1.73	100
BDE209	<0.01	0	<0.01	0	<0.01	0	<0.01	0	<0.01	0	<0.01	0
ΣPBDE	0.40 ± 0.28 (NQ–1.25)	75	3.77 ± 2.10 (0.62–7.74)	100	8.49 ± 1.93 (4.27–18.87)	100	3.76 ± 0.91 (1.23–7.08)	100	1.78 ± 0.35 (1.14–3.40)	100	30.91	100
total (α) HBCD	<0.01	0	<0.01	0	<0.01	0	<0.01	0	<0.01	0	<0.01	0
BB101	<0.01	0	0.73 ± 0.15 (0.45–0.92)	100	0.57 ± 0.09 (0.31–1.05)	100	0.48 ± 0.16 (<0.01–1.01)	83	0.36 ± 0.13 (<0.01–0.97)	83	<0.01	0
ΣPCB	2.7 ± 0.8 (0.9–4.2)	100	22.3 ± 9.8 (3.6–36.6)	100	39.6 ± 9.8 (21.3–97.0)	100	10.3 ± 2.3 (3.0–18.6)	100	5.7 ± 1.6 (1.5–12.7)	100	12.3 ± 4.8 (6.5–21.9)	100
OCS	<0.1	0	<0.1	0	<0.1	0	<0.1	0	<0.1	0	<0.1	0

[a] The three Santa Catalina Island samples were pooled for all except lipid and PCB analyses.

oxylated analogues of OH-PCBs were monitored as $[M]^-$, $[M + 2]^-$, and $[M - 15]^-$. The monitored ions were as follows: m/z 322 and 324 for 4-MeO-HpCS; m/z 322, 324, and 307 for MeO-tetra-CBs; m/z 356, 358, and 341 for MeO-penta-CBs; m/z 390, 392, and 375 for MeO-hexa-CBs; and m/z 424, 426, and 410 for MeO-hepta-CBs. Analytes were identified by comparison of retention times and ECNI mass spectra to those of the authentic standards.

ΣPBDE consisted of the monitored congeners BDE47, BDE100, BDE99, BDE154/BB153, BDE153, BDE138, BDE183, and BDE209. ΣOH-PCB (as MeO-derivatives) consisted of the monitored congeners 4-OH-CB146, 4-OH-CB163, 4-OH-CB187, 4-OH-CB162, 4′-OH-CB172, 4-OH-CB193, 4′-OH-CB199, 4,4′-diOH-CB202, 4′-OH-CB208, 4-OH-CB178, 4′-OH-CB202, 4′-OH-CB201, 2′-OH-CB114, 4-OH-CB107/4′-OH-CB108, 4′-OH-CB127, 4′-OH-CB130, 3′-OH-CB182, 4-OH-CB97, 4′-OH-CB177, 3′-OH-CB180, 3′-OH-CB203, 4′-OH-CB200, 4′-OH-CB79, 4′-OH-CB120, 4-OH-CB134, 3′-OH-CB184, 3′-OH-CB183, 4′-OH-CB198, and 4′-OH-CB159. ΣOH-PBDE (as MeO-derivatives) and ΣMeO-PBDE consisted of the monitored congeners 6′-MeO-BDE49, 6-MeO-BDE47, 5-MeO-BDE47, 6′-MeO-BDE17, 4′-MeO-BDE17, 2′-MeO-BDE68, 3-MeO-BDE47, 4′-MeO-BDE49, 4-MeO-BDE42, 6-MeO-BDE90, 6-MeO-BDE99, 2-MeO-BDE123, 6-MeO-BDE85, and 6-MeO-BDE137. ΣPCB corresponded to a suite of 67 congeners (*33*).

Quality Control. Mean internal standard recoveries for PBDEs (and MeO-PBDEs), OH-PCBs, and OH-PBDEs were 90 ± 7%, 84 ± 10%, and 53 ± 10%, respectively. Analytes were quantified using an internal standard approach, thus all reported values were inherently recovery-corrected. The method limits of quantification (MLOQ), based on a signal-to-noise ratio of 10, were about 0.01 ng/g wet weight (w.w.) for all analyte classes, except for the previously analyzed PCBs (around 0.1 ng/g w.w.)

Method blanks ($n = 4$) for each sample batch, to assess background interference and possible contamination, demonstrated no significant responses. Duplicate analysis of the samples was not possible as all plasma was consumed to ensure quantifiable analyte levels.

Data Analysis. Summary statistics (Statistica 6.0, Statsoft, Tulsa, OK) and interpopulation comparisons of analyte concentrations were computed only if more than 50% of the samples in a population had levels above the MLOQ. Levels below the MLOQ were set to a random value between zero and 1/2MLOQ. Differences in contaminant concentrations between BC populations were tested using ANOVA, followed by Tukey's HSD test for unequal sample sizes to determine significance. Data were log-transformed to approximate normal distribution prior to statistical analysis. The Brown-Forsythe test demonstrated that the data did not violate the assumption of homogeneity of variance, validating the use of ANOVA. The SCI samples were pooled, and thus SCI contaminant values were not statistically compared with the BC populations. Possible correlations between contaminant groups and metabolite groups were assessed via linear regression analysis. All tests were considered significant at $p < 0.05$.

No significant relationship ($p < 0.05$) was observed between any of the contaminant concentrations and either body mass or age. Thus, these variables were not used to correct or to further subclassify data, and all subsequent analyses considered only interpopulation comparisons. Plasma lipid concentrations (0.96 ± 0.05% for BC nestlings and 0.65 ± 0.07% for California nestlings, Table 1) were not predictive of neutral or phenolic contaminant levels; therefore, all contaminant concentrations were reported on a wet weight basis.

Results and Discussion

Brominated Flame Retardants. Plasma mean ΣPBDE concentrations were similar among the four southwestern BC bald eaglet populations (Table 1). Comparable mean ΣPBDE levels were reported previously in bald eaglet plasma samples from Lake Superior (7.9 ng/g w.w. (*7*)). Significant variation in the mean ΣPBDE levels among all BC nestlings (ANOVA: $F_{4,21} = 0.402$, $p < 0.001$) was attributed to higher concentrations in the four southwestern BC populations than in eaglets from the FSJ reference site (Figure 2). Higher concentrations of PBDEs (as well as greater proportion of highly brominated

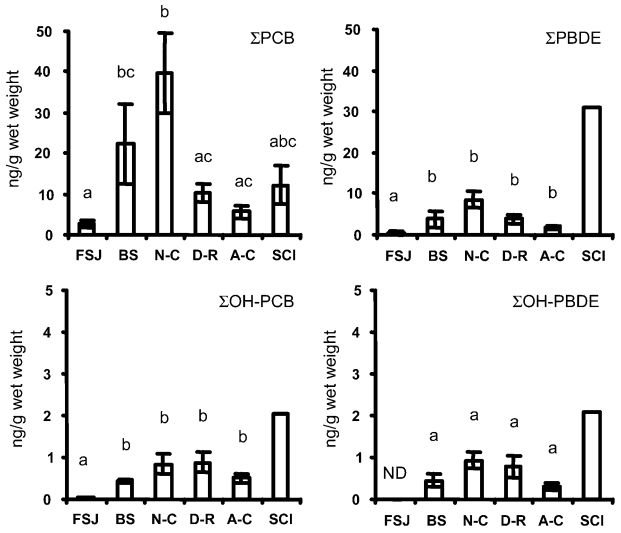

FIGURE 2. Arithmetic mean concentrations (±SE) of organohalogen contaminants in nestling bald eagles from British Columbia and southern California sites. Means that do not share the same lower case letter were significantly different ($p < 0.05$). *Y*-axes are not drawn to the same scale for all panels. Sites are as follows: FSJ = Fort St. James area ($n = 4$), BS = Barkley Sound ($n = 3$), N−C = Nanaimo/Crofton area ($n = 7$), D−R = Delta−Richmond area ($n = 6$), A−C = Abbotsford−Chilliwack area ($n = 6$), and SCI = Santa Catalina Island ($n = 3$, pooled for all but ΣPCB).

congeners, e.g., BDE153) in southwestern BC nestlings compared to those from FSJ may be related to the proximity of the former populations to urban and industrial regions. However, this spatial variability is more likely related to dietary differences (and thus trophic level) between populations, as is suggested to be the case for PCBs. Elliott et al. (*30*) proposed that more piscivorous birds in the diets of some BC bald eagle populations may result in higher levels of PCBs in comparison to populations that have more fish-based diets. This was also supported by the elevated proportion of lower chlorinated PCB congeners in populations with more fish in their diets, as fish have a lower metabolic potential toward PCBs than birds. Significant differences in ΣPCB concentrations were also found in plasma from different BC eaglet populations in the current study (Figure 2). If the spatial differences in PBDE contamination are related mainly to diet as for PCBs, it would be expected that the levels of ΣPBDE would vary similarly with ΣPCB; indeed, ΣPBDE levels in BC eaglets were strongly positively correlated with ΣPCB ($r^2 = 0.83$, $p < 0.001$) but were between 3- and 7-fold lower than ΣPCB.

In SCI eaglets, ΣPBDE levels were 4- (N−C) to 77-fold (FSJ) higher than in BC nestlings, indicating elevated levels of more recently released contaminants in the California population. In contrast to BC eaglets and unlike most current wildlife findings (*5*), the ΣPBDE concentration in the pooled SCI sample was higher (2.5-fold) than the ΣPCB concentration (Figure 2). Despite historically high organochlorine releases near SCI, the mean ΣPCB concentration in SCI nestlings was not measurably different than the mean ΣPCB levels in any of the BC populations. In SCI eaglets, the higher ΣPBDE level was comparable to the 60 ng/g body weight single dose of BDE99 given to adult female rats in a recent study, which resulted in neurobehavioural and reproductive alterations in offspring (*31*).

Congener profiles were similar among BC locations; BDE47 accounted for around half of the ΣPBDE, with lesser amounts of BDE99, BDE100, and BDE153. This PBDE congener profile is consistent with other North American fauna, except for the absence of BDE154, which (along with possible BB153 coelution in GC analysis) is often found at similar levels to BDE153 in humans and in wildlife (*5*). The PBDE congener pattern in SCI eaglets was comparable, but low levels of BDE138, BDE154, and BDE183 were also detected (Table 1). This finding may be related to source pattern differences from BC populations or to the closer proximity of SCI to sources of release (e.g., the industrial and urbanized areas of Los Angeles). Yet, the fully brominated BDE209, which comprises more than 97% of the most highly used commercial BFR product in North America (*5*), was not detected in SCI (nor in BC) nestlings. This congener may not be highly bioavailable as a function of its molecular size relative to other BDE congeners (*5*). However, the assumption of low bioavailability of BDE209 may be confounded by other factors, such as debromination, possibly in conjunction with hydroxylation/methoxylation (*9*). BDE209 also has a relatively short half-life in humans (11−18 d (*32*)) and in seals (8.5−13 d (*33*)) and has also been shown to be photolytically labile (*5*).

BB101 was found at similar mean concentrations in all southwestern BC eaglets (Table 1), at levels from 5- to 15-fold lower than the ΣPBDE levels. However, this congener was not detected in SCI or FSJ nestlings. The presence of BB101 in southwestern BC eaglets is likely due to the historic release of PBBs and their persistent nature, as their usage was discontinued in the early 1970s (*5*). BB101 was detectable in Great Lakes fish and had the second highest PBB congener concentration, aside from BB153 (*34*). BB101 was also recently reported in Norwegian glaucous gulls, though at low levels (*16*). Measurable levels of BB101 in eaglets may also be related to current release; PBBs were recently reported as contaminants in the commercial PBDE mixtures DE-71, DE79, and DE-83 (*35*). Regardless, the PBB levels found in these southwestern BC nestlings were well below the levels known to adversely affect laboratory animals (*36*). The coeluted BB153/BDE154 concentration was only found in SCI and at <1% of ΣPBDE. The currently used flame retardant, HBCD, was not found in any North American eaglet plasma analyzed (Table 1). This is consistent with total-HBCD levels recently reported in the plasma of another high trophic level raptor, Svalbard glaucous gulls, which were at the MLOQ (0.03 ng/g w.w.) (*16*).

Chlorinated Phenolic Contaminants. Up to 14 OH-PCB congeners were identified and quantifiable and are likely metabolites of PCBs transformed by the bald eaglets via CYP enzyme mediation (*14*). OH-PCBs were recently reported in unhatched eggs of Svalbard glaucous gulls and fulmars from the Faroe Islands (*12*, *13*), indicating that OH-PCBs are also maternally transferred during ovogenesis in birds. Low levels of OH-PCBs have been reported in fish blood (*37*, *38*). However, OH-PCB accumulation in the present eaglets from the diet is unlikely since there are no published reports to support OH-PCB accumulation from fish prey to bird predator.

The ΣOH-PCB concentrations were similar among nestlings from the southwestern BC sites (Table 2) but were significantly higher than the levels observed in FSJ nestlings (Figure 2). SCI eaglets contained higher ΣOH-PCB levels than all BC populations, despite comparable levels of ΣPCB (Figure 2), suggesting exposure-induced biotransformation capacity toward PCBs in SCI eaglets. An indicator of metabolic potential toward PCBs, the ΣOH-PCB/ΣPCB ratio varied among populations with the highest in SCI birds (0.24) and the lowest in FSJ birds (0.02). The higher ratio in SCI eaglets supports CYP-induced PCB metabolism in these more highly organochlorine-contaminated eaglets. In bald eagles, significant variation in the ratios of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD), 2,3,7,8-tetrachlorodibenzofuran (2,3,7,8-TCDF), and possibly PCB patterns found in livers

TABLE 2. Arithmetic Mean ± SE and Range of OH-PBDE, MeO-PBDE, OH-PCB, and 4-OH-HpCS Concentrations (ng/g Wet Weight) in Nestling Bald Eagle Plasma Samples from British Columbia and Southern California Sites

	Fort St. James ($n = 4$)		Barkley Sound ($n = 3$)		Nanaimo–Crofton ($n = 7$)		Delta–Richmond ($n = 6$)		Abbotsford–Chilliwack ($n = 6$)		Santa Catalina Island ($n = 3$)[a]	
analyte	mean ± SE (range)	%n > MLOQ	mean ± SE (range)	%n > MLOQ	mean ± SE (range)	%n > MLOQ	mean ± SE (range)	%n > MLOQ	mean ± SE (range)	%n > MLOQ	mean ± SE (range)	%n > MLOQ
6′-OH-BDE49	<0.01	0	(<0.01–0.40)	33	0.54 ± 0.17 (<0.01–1.17)	71	0.46 ± 0.20 (<0.01–1.24)	66	<0.01	0	<0.01	0
6-OH-BDE47	(<0.01–0.47)	25	0.32 ± 0.01 (0.30–0.35)	100	0.38 ± 0.13 (<0.01–1.03)	71	0.31 ± 0.11 (<0.01–0.61)	66	0.31 ± 0.10 (0.14–0.64)	83	<0.01	0
4′-OH-BDE49	<0.01	0	<0.01	0	<0.01	0	<0.01	0	<0.01	0	2.10	100
ΣOH-PBDE	(NQ–0.47)	25	0.46 ± 0.15 (0.30–0.75)	100	0.92 ± 0.20 (NQ–1.51)	86	0.77 ± 0.27 (0.18–1.85)	100	0.31 ± 0.10 (NQ–0.64)	83	2.10	100
ΣOH-PBDE/ ΣPBDE	(NQ–0.38)		0.45 ± 0.38 (0.04–1.20)		0.14 ± 0.04 (NQ–0.32)		0.22 ± 0.07 (0.08–0.48)		0.17 ± 0.05 (NQ–0.35)		0.07	
ΣMeO-PBDE	NQ	0	NQ	0	NQ	0	NQ	0	NQ	0	NQ	0
ΣOH-PCB	0.04 ± 0.02 (0.01–0.09)	100	0.44 ± 0.06 (0.37–0.56)	100	0.83 ± 0.24 (0.47–2.28)	100	0.87 ± 0.24 (0.35–1.98)	100	0.51 ± 0.12 (0.37–1.02)	100	2.03	100
ΣOH-PCB/ ΣPCB	0.02 ± 0.01 (0.003–0.03)		0.04 ± 0.03 (0.01–0.10)		0.03 ± 0.01 (0.005–0.07)		0.11 ± 0.03 (0.03–0.21)		0.16 ± 0.06 (0.01–0.42)		0.24	
4-OH-HpCS	0.01 ± 0.01 (0.01–0.02)	100	0.03 ± 0.01 (0.02–0.03)	100	0.03 ± 0.01 (0.01–0.05)	100	0.06 ± 0.01 (0.02–0.08)	100	0.05 ± 0.01 (0.01–0.09)	100	0.67	100
4-OH-HpCS/ OCS	NQ		NQ		NQ		NQ		NQ		NQ	

[a] The three Santa Catalina Island samples were pooled prior to analysis.

and deviations from the patterns observed in eggs were suggestive of corresponding variation in metabolic capacity as represented by induction of CYP1A-type enzymes (*39*). Variations in the catalytic activity of CYP1A may suggest activity of other CYP enzyme forms that mediate the metabolism of more non-dioxin-like PCB congeners, which constitute the precursors of retained OH-PCB metabolites. Yet, no significant differences in the ratios were found among birds from the BC populations, suggesting that despite significant differences in PCB exposure, the capacity to metabolically transform PCBs to their OH-PCB analogues may be comparable among all BC eaglet populations.

Retained OH-PCBs in biota tend to be more highly chlorinated congeners with no *ortho-meta* or *meta-para* Cl unsubstituted sites or only *ortho-meta* Cl unsubstituted sites (*14*). Although tetra- through nonachlorinated congeners were investigated, only highly halogenated (hexa- to nonachlorinated) congeners were detected for all bald eaglet populations (Figure 3). The major congeners found in BC nestlings were the heptachlorinated 4-OH-CB187 followed by the octachlorinated 4′-OH-CB202, which combined accounted for between 65% and 100% of the ΣOH-PCB. The latter congener was also dominant in SCI plasma along with two hexachlorinated congeners, 3′-OH-CB138 and 4-OH-CB146 (Figure 3). In the blood or plasma of four other high trophic feeding bird species, 4-OH-CB187 and 4-OH-CB146 were identified as the major congeners (*10*, *11*, *13*). The concentrations of 4-OH-CB187 and 4-OH-CB146 determined in Swedish white-tailed eagle nestlings (whole blood (*10*)) and in Norwegian glaucous gulls (plasma (*13*)) were higher than those found in North American bald eagle nestlings (this study), including SCI eaglets. The dominant congener, 4-OH-CB187, may be a metabolite of CB187 (direct OH insertion) or of CB183 (1,2-shift of chlorine and hydrogen atoms). Concentrations of 4-OH-CB187 were weakly correlated with CB187 concentrations ($r^2 = 0.25$, $p < 0.01$) in BC birds but not significantly ($r^2 = 0.20$, $p = 0.07$) with CB183 concentrations. A putative metabolite of CB138 and/or CB153 (two of the major PCB congeners detected), 4-OH-CB146, had concentrations that were similarly but weakly correlated (positive) with CB138 ($r^2 = 0.36$, $p < 0.01$) and with CB153 ($r^2 = 0.36$, $p < 0.01$) concentrations. These results suggest that 4-OH-CB187 may be derived solely from CB187, whereas 4-OH-CB146 may be a metabolite of CB138 and CB153 in these eaglets, in agreement with previous research in other species (*14*).

An ostensible metabolite of octachlorostyrene (OCS), 4-OH-HpCS, was a detected in all bald eaglet populations (Table 2). Although the MLOQ for OCS was higher (0.1 ng/g w.w.) than for 4-OH-HpCS, OCS was not detected in any nestlings. Verreault et al. (*13*) recently reported 4-OH-HpCS in Norwegian glaucous gulls (<0.04–0.43 ng/g w.w). The ratio of 4-OH-HpCS to the slowly metabolized CB153 may suggest the relative capacity to biotransform OCS and/or retain 4-OH-HpCS. The ratio was higher in SCI eagle nestlings (0.29) than in BC nestlings (0.005–0.070). The variation in OCS sources and the possibility of actual 4-OH-HpCS accumulation may also contribute to these interpopulation differences. Regardless of the nature of origin, 4-OH-HpCS and other chlorinated phenolic contaminant concentrations contributed considerably to the overall level of plasma organohalogen contamination in west coast North American bald eaglets.

Hydroxylated- and Methoxylated PBDEs. The levels of ΣOH-PBDE were similar to the ΣOH-PCB concentrations found for all populations (Figure 2). There was variability in ΣOH-PBDE concentrations among BC locations, e.g., higher concentrations of 6′-OH-BDE49 in N-C and D-R nestlings, but the levels were not significantly different among BC sites with the exception of the FSJ reference site (>50% of samples below the MLOQ) (Figure 2). Unlike OH-PCBs, the OH-PBDE congener patterns were simple and distinctly different between the BC and SCI nestlings. Although 14 congeners were assessed, just two tetrabrominated congeners, 6′-OH-BDE49 and 6-OH-BDE47, were detected in BC samples (Table 2). Only 4′-OH-BDE49 was found in the pooled SCI sample. The level of this congener was at least 2-fold higher than the sum of 6′-OH-BDE49 and 6-OH-BDE47 in all BC populations. The concentration of 4′-OH-BDE49 was slightly higher than ΣOH-PCB in SCI birds and was about 7% of the ΣPBDE level. The total concentration of the two congeners found in BC birds was between 14% and 45% of the ΣPBDE levels.

PBDE analogues with OH- or MeO-substitution in the *ortho* position are known to be formed by some marine sponges and algae. Teuten et al. (*19*) demonstrated by ^{14}C analysis that 6-MeO-BDE47 and 2′-MeO-BDE68 found in True's beaked whale (*Mesoplodon mirus*) from the North Atlantic Ocean were of biogenic origin. MeO-PBDEs were

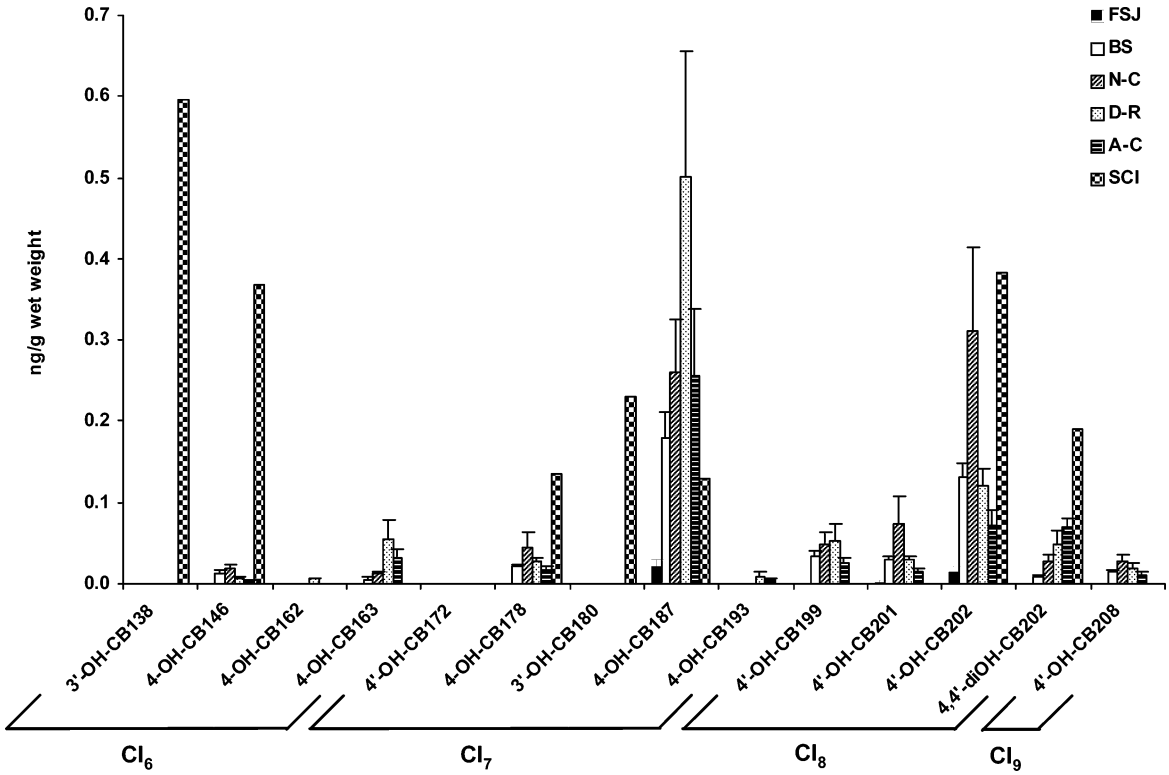

FIGURE 3. Arithmetic mean concentrations (+SE) of individual OH-PCB congeners in nestling bald eagles from British Columbia and southern California sites. Sites are as follows: FSJ = Fort St. James area (n = 4), BS = Barkley Sound (n =3), N−C = Nanaimo/Crofton area (n = 7), D−R = Delta−Richmond area (n = 6), A−C = Abbotsford−Chilliwack area (n = 6), and SCI = Santa Catalina Island (n = 3, pooled).

not detected in the plasma of BC or SCI eaglets; however, the two OH-PBDE congeners found in BC nestlings are *ortho* substituted and would appear to be natural products accumulated by the eaglets from their diet. 6′-OH-BDE49 and 6-OH-BDE47 are also possible metabolites of anthropogenic PBDEs (*9*). 6-OH-BDE47 is a potential metabolite of the biologically predominant BDE47 congener. Tetra- and tribrominated OH-PBDE metabolites were detected in rodents dosed with BDE47 (*9*). However, 6-OH-BDE47 concentrations were not correlated (r^2 = 0.10, p < 0.20) with BDE47 concentrations in BC eaglets. 6′-OH-BDE49 could be a biotransformation product of BDE49, although exposure to BDE49 cannot be confirmed as it was not detected in any of the plasma samples. Unlike the OH-PBDE congeners found in BC samples, the *para* substituted 4′-OH-BDE49 found in SCI eaglets is more likely to be a metabolite of anthropogenic BDE47 via a 1,2-shift analogous to OH-PCB formation from PCBs (*9*, *14*). *Meta* and *para* substituted OH-PBDEs are not known to be natural products (*40*), and for OH-PCB congeners retained in biota, the OH group is located only at a *meta* or *para* position on the biphenyl ring system.

The spatial trend of mean ΣOH-PBDE concentrations was similar to that of the mean ΣPCB, ΣPBDE, and ΣOH-PCB concentrations (Figure 2). ΣOH-PBDE concentrations in BC eaglets were marginally positively correlated with ΣPCB (r^2 = 0.17, p = 0.065) and ΣPBDE (r^2 = 0.18, p = 0.059). ΣOH-PCB levels were associated with ΣPCB levels (r^2 = 0.36, p < 0.001) and ΣPBDE levels (r^2 = 0.54, p < 0.001). Correlations between the contaminant classes may be due to dietary exposure or possibly to similar binding affinities for circulating blood transport proteins, e.g., TTR (*14*). Further studies examining the relationships between plasma organohalogen concentrations and thyroid hormone and vitamin A levels are warranted and may elucidate potential biochemical effects of such contaminants on these bald eagle populations.

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Supporting Information Available

Body mass and age data for individual bald eagle nestlings and PBDE congener profiles for all populations. This material is available free of charge via the Internet at http://pubs.acs.org.

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