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ORIGINAL ARTICLE

Contaminant exposure in outmigrant juvenile salmon from Pacific Northwest estuaries of the United States[1]

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Abstract To better understand the dynamics of contaminant uptake in outmigrant juvenile salmon in the Pacific Northwest, concentrations of polychlorinated biphenyls (PCBs), DDTs, polycylic aromatic hydrocarbons (PAHs) and organochlorine pesticides were measured in tissues and prey of juvenile chinook and coho salmon from several estuaries and hatcheries in the US Pacific Northwest. PCBs, DDTs, and PAHs were found in tissues (whole bodies or bile) and stomach contents of chinook and coho salmon sampled from all estuaries, as well as in chinook salmon from hatcheries. Organochlorine pesticides were detected less frequently. Of the two species sampled, chinook salmon had the highest whole body contaminant concentrations, typically 2–5 times higher than coho salmon from the same sites. In comparison to estuarine chinook salmon, body burdens of PCBs and DDTs in hatchery chinook were relatively high, in part because of the high lipid content of the hatchery fish. Concentrations of PCBs were highest in chinook salmon from the Duwamish Estuary, the Columbia River and Yaquina Bay, exceeding the NOAA Fisheries' estimated threshold for adverse health effects of 2400 ng/g lipid. Concentrations of DDTs were especially high in juvenile chinook salmon from the Columbia River and Nisqually Estuary; concentrations of PAH metabolites in bile were highest in chinook salmon from the Duwamish Estuary and Grays Harbor. Juvenile chinook salmon are likely absorbing some contaminants during estuarine residence through their prey, as PCBs, PAHs, and DDTs were consistently present in stomach contents, at concentrations significantly correlated with contaminant body burdens in fish from the same sites.

Keywords Chinook salmon · Coho salmon · Contaminants · PAHs · PCBs · DDTs · Pesticides · Washington · Oregon · Estuary

1 Introduction

Estuaries are important habitats for salmon during the juvenile stage of their life cycle, when they make the transition from freshwater to the ocean (Healey, 1982). Estuaries provide outmigrating juvenile salmon with a refuge from predators, a rich food supply that supports rapid growth, and appropriate conditions for the physiological adaptation to saltwater (Dorcey *et al.*, 1978; Simenstad *et al.*, 1982). However, urban and industrial development may impair the quality of estuarine habitats. Estuaries located near urban centers often receive inputs of toxic contaminants from municipal and industrial activities (Brown *et al.*, 1998; USEPA,

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[1] Environmental Monitoring and Assessment

1997), which may be taken up by juvenile salmon and their prey. Because juvenile salmon are in a period of rapid development, and undergoing many physiological changes during their residence in estuarine environments, they may be especially vulnerable to the deleterious effects of toxic chemicals.

The well-documented presence of chemically contaminated sediments in Puget Sound urban estuaries (e.g., Malins *et al.*, 1982) prompted a series of studies to examine the degree to which juvenile salmon were exposed to toxic chemicals during estuarine residence (McCain *et al.*, 1990; Varanasi *et al.*, 1993; Stein *et al.*, 1995; Stehr *et al.*, 2000). Juvenile salmon (primarily chinook and coho, *Onchorhynchus tshawytscha* and *O. kisutch)* were sampled from several urban and non-urban estuaries in Puget Sound including the Green River/Duwamish Estuary system in Seattle, the Puyallup River/Hylebos Waterway system in Tacoma, and the more rural Snohomish River and Nisqually River Estuaries. Juvenile chinook salmon from hatcheries associated with sampled estuaries were also collected and whole bodies and stomach contents were analyzed for chemical concentrations. Results of these surveys showed that outmigrating juvenile chinook salmon from the Duwamish and Hylebos Waterways exhibited consistent evidence of exposure to contaminants. Juvenile chinook salmon from the Snohomish Estuary, which has some urban development, also appeared to be exposed to contaminants, but to a much lesser degree than salmon from the Duwamish and Hylebos Waterways. In addition, when held in tanks with flow-through seawater for a period of several months, juvenile salmon from the Duwamish Estuary exhibited reduced growth and reduced disease resistance when compared to salmon from either the Green River Hatchery (the primary source of salmon for the Duwamish Estuary) or to salmon from the nonurban Nisqually system (Arkoosh *et al.*, 1998; Casillas *et al.*, 1995). Similar effects were observed for juvenile salmon from the Hylebos Waterway (Arkoosh *et al.*, 2001; Casillas *et al.*, 1998). Chemical contaminant exposure in the estuary appeared to place additional stresses on juvenile chinook salmon that could affect their long-term health and survival as they enter the marine environment.

To increase our knowledge of concentrations of chemical contaminants in outmigrant salmon in the Pacific Northwest, we carried out an expanded study from 1996–2001 in which juvenile coho and chinook salmon were collected for contaminant analyses from a number estuaries in Washington and Oregon. Classified by the overall level of development and channel alteration in each estuary (Cortright *et al.*, 1987), the sampling areas included: five deep draft estuaries, with the maximum level channel alteration and urban development (Duwamish Estuary, Columbia River, Grays Harbor, Yaquina Bay, and Coos Bay); two shallow draft estuaries with less extensive channel alteration and some urban and industrial development (Tillamook Bay and Coquille River), four conservation estuaries, where channel alteration is minimal and development is limited (Skokomish Estuary, Nisqually Estuary, Willapa Bay and Alsea Bay); and two natural estuaries, which are largely undeveloped for residential, commercial or industrial uses (Elk River and Salmon River). Predominantly wild fish were collected in the estuaries, although some fish of hatchery origin may have been sampled due to incomplete marking of hatchery fish. Juvenile chinook salmon were also sampled from regional hatcheries to evaluate contaminant uptake during rearing but prior to release. Our results indicate that exposure to chemical contaminants is widespread in outmigrant juvenile chinook and coho salmon, and concentrations in tissues of chinook salmon from several estuaries are high enough to pose a potential threat to their health and survival.

2 Materials and methods

2.1 Collecting juvenile salmon

Juvenile, subyearling chinook salmon were collected from a number of Washington and Oregon estuaries over a 6-year period (1996–2001; Fig. 1; Table 1). The Washington estuaries included: Skokomish and Nisqually Estuaries; Duwamish Estuary, and Grays Harbor and Willapa Bay. The Oregon estuaries included the Columbia, Salmon, Coquille, and Elk Rivers; and Yaquina, Alsea, and Coos Bays. Juvenile coho were also collected from Grays Harbor and Willapa, Yaquina, Alsea, and Coos Bays during 1998 (Fig. 1; Table 1). Due to the pattern of salmon movement in the estuaries, we generally sampled on early morning outgoing tides. Salmon were caught with a beach seine net 36.6 meters in length. The wings of

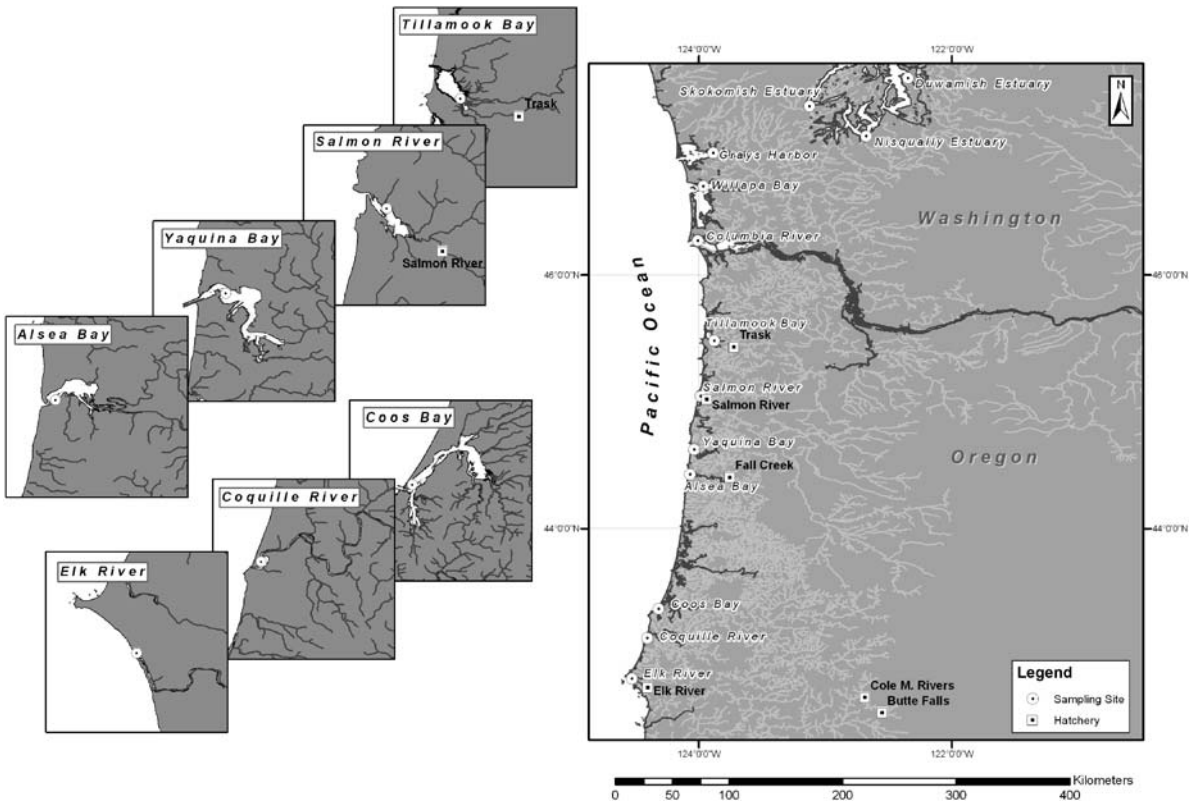

Fig. 1 Locations of hatcheries and estuaries where juvenile coho and chinook salmon were collected

the net were 18 meters long by 2.3 meters deep with 0.6 cm mesh.

Appropriate sampling permits were obtained from the National Marine Fisheries Service (NMFS), and the Oregon and Washington Departments of Fish & Wildlife prior to sampling. To ensure sampling of wild fish instead of hatchery-reared fish we attempted to collect fish from field sites prior to releases from hatcheries or other programs (such as the Salmon and Trout Enhancement Program or STEP). Although a few finclipped hatchery fish were collected and sampled, we did not include these fish in our analyses. Once target salmonids were removed from the net they were placed in insulated aerated tanks and transported live to the nearest laboratory, either the Hatfield Marine Science Center in Newport, Oregon; the University of Oregon's Oregon Institute of Marine Biology in Charleston, Oregon; the U.S. Fish and Wildlife's Olympia Fish Health Center in Olympia, Washington, the Point Adams Field Station in Hammond, Oregon or the Northwest Fisheries Science Center in Seattle, Washington, where they were necropsied within a few hours of collection. Juvenile chinook salmon were also obtained directly from several hatcheries (Fall Creek, Butte Falls, Cole M. Rivers, Elk River, Salmon River, and Trask; see Fig. 1 for locations) to evaluate contaminant uptake during hatchery rearing. Juvenile hatchery coho salmon were not available for sampling at the time of the survey.

Fish to be necropsied were measured (to the nearest mm) and weighed (to the nearest 0.1 g), then sacrificed by a blow to the head. Bile and stomach contents were removed, and composites of 10–15 fish each were generated. Whole gutted bodies from 10 fish were also collected and composited. Bile and stomach contents samples were frozen and stored at −80 °C and whole body samples were frozen and stored at −20 °C until chemical analyses were performed. Sampling sites, dates, and sample types collected are listed in Table 1. Because of limitations associated with fish availability and tissue requirements for analysis, not all samples types could be collected each year from all sites.

Table 1 Sites sampled in Washington and Oregon for juvenile salmonids. Sites were classified by estuary type according to Cortright *et al.* (1987). N = natural estuary; C = conservation estuary; S = shallow draft estuary; D = deep draft estuary NS = not sampled; CH = chinook sampled; CO = coho sampled. wb = whole body sampled; b = bile sampled; s = stomach contents sampled

	1996	1997	1998	1999	2000	2001
WA						
Skokomish Estuary (C)	NS	NS	CH (wb,b)	CH (wb,b)	CH (b)	NS
Duwamish Estuary (D)	NS	NS	CH (wb,b)	CH (wb,b,s)	NS	NS
Nisqually Estuary (C)	NS	NS	CH (wb,b,s)	CH (wb,b,s)	NS	NS
Grays Harbor (D)	NS	NS	CH (wb,b.s) CO (wb,b,s)	CH (wb,b,s)	NS	NS
Willapa Bay (C)	NS	NS	CH (wb,b,s) CO (wb,b,s)	CH (wb,b,s)	NS	NS
Columbia River (D)	NS	NS	CH (wb,s)	CH (wb,b,s)	CH(b,s)	CH (b)
OR						
Salmon River (N)	CH (wb)	NS	CH (b)	CH (wb,s)	CH (wb,s)	CH (wb,s)
Yaquina Bay (D)	NS	NS	CH (wb,b,s) CO (wb,b,s)	CH (wb,b,s)	CH (wb,s)	CH (b) CO (wb,s)
Alsea Bay (C)	CH (wb,b)	NS	CH (wb,s) CO (wb,b,s)	CH (wb,b,s)	CH (wb,b,s)	CH (wb,b,s) CO (wb,s)
Coos Bay (D)	CH (wb)	NS	CH (wb,b,s) CO (wb,b,s)	CH (wb,b,s)	CH (wb,s)	NS
Coquille River (S)	CH (wb)	NS	NS	NS	NS	NS
Elk River (N)	CH (wb)	NS	CH (wb,b.s)	NS	CH (wb,s)	CH (wb,b,s)
Salmon River Hatchery	CH (wb)	NS	NS	NS	NS	NS
Fall Creek Hatchery	CH (wb)	NS	NS	NS	NS	NS
Trask Hatchery	CH (wb)	NS	NS	NS	NS	NS
Butte Falls Hatchery	CH (wb)	NS	NS	NS	NS	NS
Cole M. Rivers Hatchery	CH (wb)	NS	NS	NS	NS	NS
Elk River Hatchery	CH (wb)	NS	CH (wb,s)	NS	NS	NS

2.2 Sample analyses

2.2.1 Organochlorine and aromatic hydrocarbon analyses of composite whole body and stomach content samples

Samples in this study were analyzed using a performance-based measurement system (Telliard, 1999), described in detail by Sloan *et al.* (1993) and updated in Sloan *et al.* (2005). Briefly, after the addition of surrogate standards, samples of up to 3 g were extracted with dichloromethane either by homogenizing in the presence of sodium sulfate (Sloan *et al.*, 1993) or utilizing accelerated solvent extraction (Sloan *et al.*, 2005). For composite whole body samples, a portion of the extract was taken for gravimetric lipid determination. The portion of the extract to be analyzed underwent initial cleanup by filtering through silica gel and neutral alumina, followed by the addition of a recovery standard to determine the fraction of the total extract analyzed. After further sample cleanup using high-performance liquid chromatography with size-exclusion chromatography, the sample fraction containing organochlorines (OCs) and 2–6 ring aromatic hydrocarbons was collected. The fraction was reduced in volume, a GC standard was added, and the sample was analyzed using high-resolution gas chromatography coupled with electron capture detection (samples analyzed for OCs 1996–1998; Sloan *et al.*, 1993) or mass spectrometry with selected-ion monitoring (samples analyzed for OCs 1999–2001; Sloan *et al.*, 2005) with 5–10 levels of calibration standards. Concentrations of aromatic hydrocarbons (stomach contents samples only) were analyzed in all sampling years by high-resolution gas chromatography with mass spectrometry using selected ion monitoring and 5–6 levels of calibration standards. Quality assurance measures included analysis of a certified reference material and a

laboratory blank with each batch of samples. Performance criteria were met for all samples and sample batches.

Analyses for OCs included individual PCB (polychlorinated biphenyl) congeners, DDTs, chlordanes, lindane, aldrin, dieldrin and mirex. PCBs measured over all years included a standard list of 17 congeners (IUPAC numbers 18, 28, 44, 52, 95, 101, 105, 118, 128, 138, 153, 170, 180, 187, 195, 206, and 209). Total PCBs was calculated by summing the concentrations of these individual congeners and multiplying the result by two. This formula provides a good estimate of the total PCBs in a typical environmental sample of sediments or animals feeding on lower trophic levels, where a mixture of Aroclors 1254 and 1260 is the predominant pattern (Lauenstein *et al.*, 1993). Summed DDTs (ΣDDTs) levels were calculated by summing the concentrations of *o,p′*- and *p,p′*-DDD, *o,p′*- and *p,p′*-DDE, and *o,p′*- and *p,p′*-DDT. Summed chlordanes (ΣCHLDs) were calculated by summing the concentrations of heptachlor, heptachlor epoxide, γ-chlordane, α-chlordane, oxychlordane, *cis*-nonachlor, *trans*-nonachlor and nonachlor III. Summed low molecular weight aromatic hydrocarbons (ΣLAHs) were determined by adding the concentrations of biphenyl, naphthalene, 1-methylnaphthalene, 2-methylnaphthalene, 2,6-dimethylnapthalene, acenaphthene, fluorene, phenanthrene; 1-methylphenanthrene, and anthracene. Summed high molecular weight aromatic hydrocarbons (ΣHAHs) were calculated by adding the concentrations of fluoranthene, pyrene, benz[a]anthracene, chrysene, benzo[a]pyrene, benzo[e]pyrene, perylene, dibenz[a,h]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, indenopyrene, and benzo[ghi]perylene. Summed total aromatic hydrocarbons (ΣAHs) were calculated by adding ΣHAHs and ΣLAHs.

2.2.2 PAH metabolites in bile

Composite samples of bile were analyzed by high-performance liquid chromatography with fluorescence detection (HPLC/uvf) for aromatic hydrocarbon (AH) metabolites as described in Krahn *et al.* (1986). In brief, bile was injected directly onto a C18 reverse-phase column (Phenomenex Synergi Hydro) and eluted with a linear gradient from 100% water (containing a trace amount of acetic acid) to 100% methanol at a flow of 1.0 mL/min. Chromatograms were recorded at the following wavelength pairs: 1) 260/380 nm where several 3–4 ring compounds (e.g., phenanthrene) fluoresce and 2) 380/430 nm where 4–5 ring compounds (e.g., benzo[a]pyrene) fluoresce. Peaks eluting after 5 minutes were integrated and the areas of these peaks were summed. The concentrations of fluorescent AHs in bile were determined using phenanthrene (PHN) and benzo[a]pyrene (BaP) as external standards and converting the fluorescence response of bile to phenanthrene (ng PHN equivalents/g bile), and benzo[a]pyrene (ng BaP equivalents/g bile) equivalents. Bile metabolites fluorescing at phenanthrene wavelengths were considered an indicator of exposure to low molecular weight PAHs, while metabolites fluorescing at benzo[a]pyrene (BaP) wavelengths were considered as an indicator of exposure to high molecular weight PAHs.

2.2.3 Statistical methods

Statistical analyses were conducted with the Statview©statistical software package (SAS Institute, Inc., Cary, NC, USA). Temporal and intersite differences in tissue, stomach contents, and bile contaminant concentrations were determined by ANOVA. Data were log-transformed as necessary to achieve a normal distribution. The significance level for all analyses was set at $\alpha = 0.05$.

3 Results

3.1 Lipid content in whole bodies

Lipid content (as total extractable organics) in bodies of chinook salmon collected from the estuaries varied from 0.8% in fish from Tillamook Bay to 3.5% in fish from Coquille River, with an average concentration of 2.4% (Fig. 2; Table 2). Lipid levels in juvenile coho salmon were slightly lower, with an average concentration of 1.2% (Fig. 2; Table 2), but not significantly different than levels in estuarine chinook salmon (ANOVA, $p = 0.08$). Lipid concentrations in hatchery chinook salmon were significantly higher than in estuary chinook (ANOVA, $p = 0.001$), with an average concentration of 7.9% (Fig. 2; Table 2). The number of samples collected (typically one composite per site or hatchery) was too small for intersite or interhatchery differences to be meaningfully evaluated,

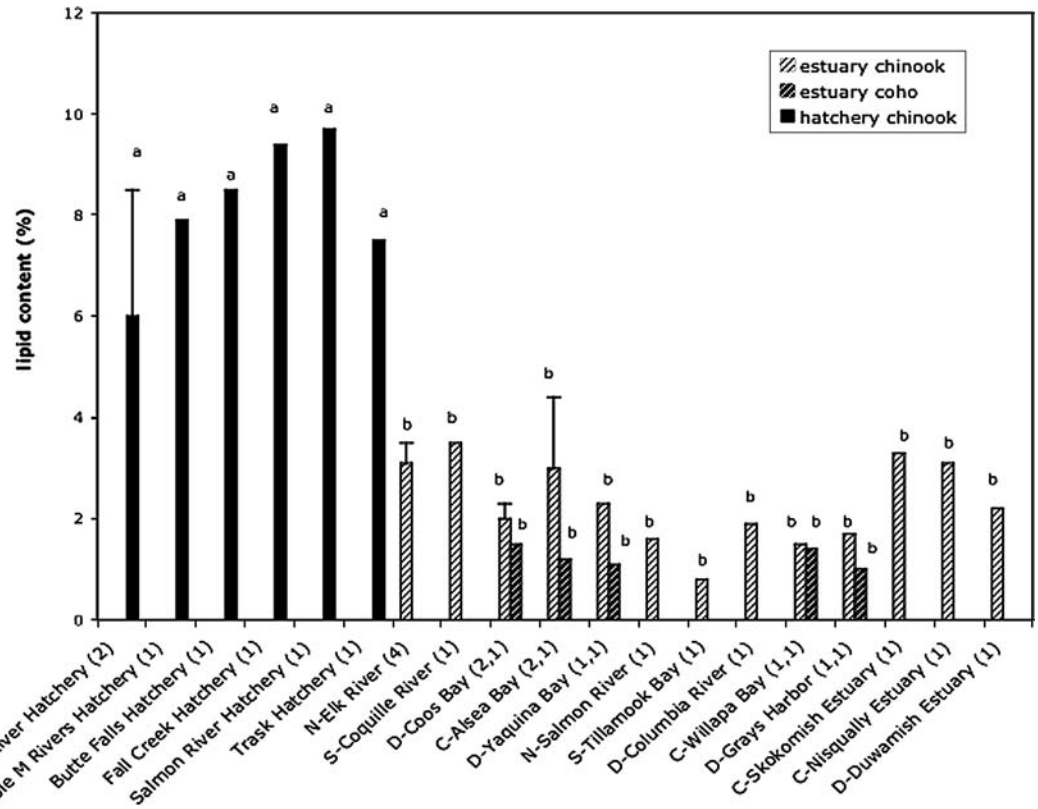

Fig. 2 Mean lipid content (%, as total extractable organics, ± SE) in whole bodies of chinook and coho salmon from Pacific Northwest estuaries and juvenile chinook salmon from associated hatcheries. N = natural estuary; C = conservation estuary; S = shallow draft estuary; D = deep draft estuary. Numbers in parentheses indicate number of composite samples (10–15 fish each) analyzed per site or group. Measurements with different letters are significantly different (ANOVA, $p < 0.05$)

but concentrations tended to be fairly uniform within the sampling groups (i.e, estuarine chinook, estuarine coho, and hatchery chinook).

3.2 Organochlorine contaminants in whole bodies

Concentrations of PCBs in whole bodies of estuarine chinook salmon (Fig. 3, Tables 2 and 3) were quite variable, ranging from ∼500 ng/g lipid weight (lw) in salmon from Elk River and Coquille Estuaries to 3100 ng/g lw in salmon from the Duwamish Estuary in Seattle (or from 3.6 ng/g wet weight (ww) at Salmon River to 103 ng/g ww at Duwamish). The lowest concentrations of PCBs were found in chinook salmon from Elk River Estuary, Coquille River, Alsea Bay Estuary, Salmon River, and Tillamook Bay; wet weight PCB concentrations were less than 20 ng/g ww at all these sites, and lipid weight PCB concentrations were below 600 ng/g lw in chinook from Elk River Estuary, Coquille River, and Tillamook. The highest PCB concentrations (2500–3100 ng/g lw or 45–103 ng/g ww) were found in salmon from Yaquina Bay, the Columbia River, and the Duwamish Estuary.

Concentrations of PCBs in juvenile coho salmon (Fig. 3, Tables 2 and 3) tended to be lower than those in chinook salmon. At sites where both species were collected, the mean PCB concentration overall was significantly lower in coho than in chinook on both a lipid weight and wet weight basis (1030 vs. 1650 ng/g lw, $p = 0.018$; 10 vs. 30 ng/g ww; $p = 0.0026$). No significant differences were observed in PCB concentrations in coho salmon from different sampling sites, but the number of samples was very small.

The mean concentration of PCBs in juvenile chinook salmon from hatcheries (Fig. 3, Tables 2 and 3) was relatively low on a lipid weight basis (620 ng/g lw),

Table 2 Contaminant concentration mean values (± SE), ranges, and sites where high and low values were observed in juvenile chinook and coho salmon from Pacific Northwest estuaries and juvenile chinook salmon from Pacific Northwest hatcheries. Values with different superscripts are significantly different (ANOVA, $p = 0.05$) in estuarine chinook, estuarine coho, and hatchery chinook

	Estuaries		Hatcheries
	Chinook	Coho	Chinook
% lipid	2.4 ± 0.2 (n = 19)^a	1.2 ± 0.1 (n = 5)^a	7.9 ± 0.8 (n = 7)^b
	0.8–3.5.%	1.1–1.5%	6–9.7%[1]
	Tillamook–Coquille	Grays Hbr.-Coos	Elk–Salmon
Body PCBs	27 ± 4 (n = 65)^a	9.7 ± 1.6 (n = 9)^b	46 ± 3 (n = 7)^c
(ng/g wet wt)	3.6–103	6–16	39–59
	Salmon–Duwamish	Alsea–Grays Hbr.	Trask–Salmon
Body PCBs	1650 ± 190 (n = 19)^a	1030 ± 230 (n = 5)^a	620 ± 50 (n = 7)^b
(ng/g lipid)	516–3099	470–1564	521–760
	Elk R.–Duwamish	Willapa-Grays Hbr.	Fall Cr.–Elk
Body DDTs	13 ± 2 (n = 65)^a	1.7 ± 0.3 (9)^b	34 ± 3 (7)^c
(ng/g wet wt)	0.5–41	0.9–3.4	27–45
	Tillamook–Columbia.	Willapa-Grays Hbr.	Trask–Salmon
Body DDTs	550 + 120 (n = 19)	140 + 50 (n = 5)	436 + 234 (n = 7)
(ng/g lipid)	62–2280	66–333	354–507
	Tillamook–Columbia	Willapa-Grays Hbr.	Trask–Elk
Whole body	0.63 ± 0.06 (n = 65)^a	0.21 ± 0.03 (n = 9)^b	0.72 ± 0.03 (n = 7)^a
DDT/PCB ratio	0.10–1.1	0.13–0.26	0.68–0.75
	Tillamook–Salmon	Coos-Alsea	Elk/Trask–Salmon
FACs-BaP	364 ± 96 (n = 47)	218 ± 26 (n = 10)	ND
(ng/g bile)	108–1925	136–298	
	Alsea–Duwamish	Yaquina–Grays Hbr.	
FACs-PHN	44600 ± 15900 (n = 47)	17600 ± 2040 (n = 10)	
(ng/g bile)	9270–359000	12900–25400	ND
	Nisqually-Duwamish	Yaquina–Coos Bay	
Stomach contents	18.6 ± 5.7 (n = 35)	11.6 ± 2.5 (n = 9)	13 (n = 1)
PCBs	4.5–200	5.4–22	
(ng/g wet wt)	Salmon–Duwamish	Alsea–Grays Hbr.	Elk
Stomach contents	8.3 ± 2.9 (n = 35)	1.5 ± 0.4 (n = 9)	4.5 (n = 1)
DDTs	0.6–45	0.9–2.3	
(ng/g wet wt)	Elk.–Grays Hbr.	Alsea–Grays Hbr.	Elk
Stomach contents	415 ± 235 (n = 35)^a	40 ± 19 (n = 9)^b	28 (n = 1)^b
ΣLAHs	12–8000	10–69	
(ng/g wet wt)	Elk-Duwamish	Coos Bay-Alsea Bay	Elk
Stomach contents	594 ± 353 (n = 35)^a	5.4 ± 1.7 (n = 35)^b	5 (n = 1)^b
ΣHAHs	1.3–6300	1.3–10	
(ng/g wet wt)	Elk/Salmon-Willapa	Coos Bay–Grays Hbr.	Elk

comparable to concentrations observed in estuary chinook and coho salmon from rural estuaries (e.g., Elk River, Coquille River, Alsea Bay). On a wet weight basis, however, the mean PCB concentration in hatchery chinook was quite high (47 ng/g ww), comparable to concentrations in moderately to heavily urbanized estuaries (Table 3).

Concentrations of ΣDDTs in estuarine chinook salmon bodies ranged from 62 ng/g lw at Tillamook Bay to 2280 ng/g lw in the Columbia River (or from below 0.5 ng/g ww in fish from Tillamook Bay to 41 ng/g ww in fish from the Columbia River) (Fig. 4, Tables 2 and 3), with a mean concentration of 550 ng/g lw or 13 n/g ww (Fig. 4; Tables 2 and 3). Concentrations

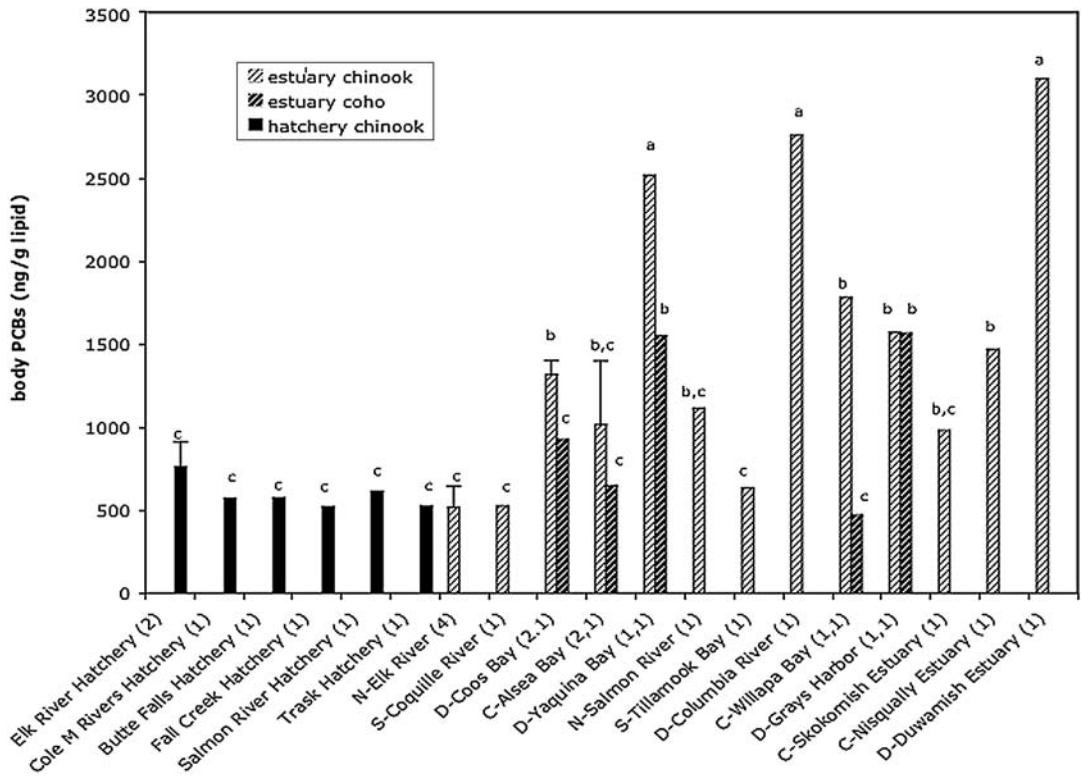

Fig. 3 Mean concentrations of ΣPCBs (ng/g lipid, ± SE) in whole bodies of juvenile chinook and coho salmon from Pacific Northwest Estuaries and juvenile chinook salmon from associated hatcheries. N = natural estuary; C = conservation estuary; S = shallow draft estuary; D = deep draft estuary. Numbers in parentheses indicate number of composite samples (10–15 fish each) analyzed per site or group. Measurements with different letters are significantly different (ANOVA, $p < 0.05$)

of ΣDDTs were low in fish from Tillamook Bay, Alsea Bay, and Elk River on both a wet wt and lipid wt basis (below 250 ng/g lw and 5 ng/g ww); at Coquille River lipid wt DDT concentrations were comparable but wet wt concentrations were higher, while the reverse was true for chinook from Salmon River. Concentrations of ΣDDTs were relatively high (over 1000 ng/g lw or 25 ng/g ww) in fish from the Nisqually, Duwamish, and Columbia River Estuaries. Fish with the highest ΣDDT concentrations were from the Columbia River, where levels were over 2200 ng/g lw or 40 ng/g ww.

In juvenile coho salmon, the maximum ΣDDT concentration was 333 ng/g lw or 3.4 n/g ww in fish from Grays Harbor (Fig. 4; Tables 2 and 3), while the mean concentration was 140 ng/g lw or 1.7 ng/g ww. When coho and chinook salmon collected from the same sites were compared, ΣDDT concentrations were much lower in coho salmon (1.7 ± 0.3 ng/g ww vs. 8.8 ng/g ww, $p = 0.0026$; or 137 ng/g lw vs. 551 ± 95 ng/g lw, $p \leq 0.001$).

On a wet weight basis, concentrations of ΣDDTs in whole bodies of juvenile Chinook collected from the hatcheries were fairly high, with the mean concentrations for all hatcheries significantly above the mean concentrations measured in estuarine chinook and coho (Tables 2 and 3). However, because of the high lipid content of the hatchery fish, their whole body ΣDDT concentrations on a lipid weight basis were more moderate (400–500 ng/g lw), and did not differ significantly from mean concentrations in estuarine salmon (Fig. 4; Tables 2 and 3).

Of the six DDTs measured in salmon whole bodies, *p,p′*-DDE predominated in whole bodies of both coho and chinook salmon from all estuaries and hatcheries sampled, accounting for 75–100% of DDTs measured (Fig. 5; Table 3). The second most prominent DDT was *p,p′*-DDD; it accounted for 10–20% of DDTs measured in chinook and coho salmon from most sites. Additionally, *p,p′*-DDT was present at several sites, accounting for 3–6% of total DDTs in chinook salmon

Table 3 Mean concentrations (± SE) in ng/g, wet wt of ΣPCBs, ΣDDTs, and DDT isomers in whole bodies of juvenile chinook and coho salmon collected from Pacific Northwest estuaries and juvenile chinook salmon from Pacific Northwest hatcheries. Compounds were measured by GC/ECD in samples collected from 1996–1998 and by GC/MS in samples collected from 1999–2001. Values with different letter superscripts are significantly different (ANOVA, $p \leq 0.05$)

Site	ΣPCBs	ΣDDTs	*o,p′*-DDD	*o,p′*-DDE	*o,p′*-DDT	*p,p′*DDD	*p,p′*-DDE	*p,p′*-DDT
Estuary chinook								
Columbia River (6)	50 ± 14^{b}	41 ± 3^{a}	0.6 ± 0.1^{a}	0.27 ± 0.0^{a}	0.71 ± 0.15^{a}	6.2 ± 0.64^{a}	31 ± 2.3^{a}	2.4 ± 0.6^{a}
Alsea Bay (8)	11 ± 3^{c}	2.4 ± 0.5^{d}	$<DL^{b}$	0.05 ± 0.05^{b}	$<DL^{c}$	0.32 ± 0.25^{b}	2.8 ± 0.8^{c}	0.11 ± 0.09^{b}
Elk River (2)	9.9 ± 3.9^{c}	4.7 ± 2.6^{d}	0.04 ± 0.03^{b}	$<DL^{b}$	0.02 ± 0.03^{c}	0.5 ± 0.4^{b}	4.1 ± 2.1^{c}	0.21 ± 0.15^{b}
Grays Harbor (3)	$27 \pm 8^{b,c}$	11.3 ± 4^{c}	0.07 ± 0.07^{b}	$<DL^{b}$	$<DL^{c}$	1.1 ± 0.6^{b}	9.9 ± 3.3^{b}	0.1 ± 0.1^{b}
Salmon River (11)	3.6 ± 1.6^{c}	1.9 ± 0.5^{d}	$<DL^{b}$	$<DL^{b}$	$<DL^{c}$	0.16 ± 0.09^{b}	1.7 ± 0.4^{c}	0.11 ± 0.06^{b}
Skokomish Estuary (3)	$29 \pm 2^{b,c}$	19.9 ± 1.5^{b}	0.08 ± 0.08^{b}	$<DL^{b}$	0.05 ± 0.05^{c}	1.9 ± 0.15^{b}	17.3 ± 1.2^{b}	0.27 ± 0.18^{b}
Willapa Bay (3)	$24^{b,c}$	12.3 ± 0.4^{c}	<DLb	$<DL^{b}$	$<DL^{c}$	0.62 ± 0.14^{b}	11.2 ± 0.7^{b}	0.14 ± 0.14^{b}
Yaquina Bay (7)	46 ± 1^{b}	7.8 ± 2.2^{d}	$<DL^{b}$	$<DL^{b}$	0.07 ± 0.07^{b}	0.48 ± 0.11^{b}	6.8 ± 1.8^{b}	0.41 ± 0.14^{b}
Coos Bay (3)	$22 \pm 3^{b,c}$	10.8 ± 1.3^{c}	$<DL^{b}$	$<DL^{b}$	0.02 ± 0.02^{c}	0.59 ± 0.09^{b}	9.8 ± 1.1^{b}	0.45 ± 0.12^{b}
Duwamish Estuary (3)	103 ± 29^{a}	27 ± 1^{b}	0.36 ± 0.03	0.18 ± 0.09^{a}	$0.09 \pm .09^{b}$	3.5 ± 0.4^{a}	22 ± 0.6^{a}	0.61 ± 0.14^{b}
Nisqually Esuary (3)	40 ± 4^{b}	30 ± 4^{b}	0.26 ± 0.03	0.09 ± 0.09^{b}	0.04 ± 0.04^{c}	3.4 ± 0.5^{a}	26 ± 3.5^{a}	0.34 ± 0.09^{b}
Coquille River (1)	$18^{b,c}$	$9.2^{c,d}$	$<DL^{b}$	$<DL^{b}$	$<DL^{c}$	1.3^{b}	7.3^{b}	0.58^{b}
Tillamook Bay (1)	5.1^{c}	0.5^{d}	$<DL^{b}$	$<DL^{b}$	$<DL^{c}$	$<DL^{b}$	0.47^{c}	<DL
Hatchery chinook								
Fall Creek (1)	49^{b}	39^{a}	0.51^{a}	$<DL^{b}$	0.03^{c}	5.4^{a}	32^{a}	1.3^{a}
Butte Falls (1)	49^{b}	35^{a}	0.56^{a}	$<DL^{b}$	$<DL^{c}$	4.9^{a}	28^{a}	1.5^{a}
Cole M. Rivers (1)	45^{b}	31^{a}	0.8^{a}	$<DL^{b}$	0.09^{b}	6.1^{a}	22^{a}	2.0^{a}
Elk River (2)	42^{b}	30 ± 10^{b}	0.04^{b}	$<DL^{b}$	0.21^{a}	4.2^{a}	23^{a}	1.7^{a}
Salmon River (1)	59^{b}	45^{a}	0.9^{a}	$<DL^{b}$	0.26^{a}	8.3^{a}	32^{a}	3.0^{a}
Trask (1)	39^{b}	27^{b}	0.67^{a}	$<DL^{b}$	$<DL^{c}$	4.5^{a}	20^{a}	1.3^{a}
Estuary Coho								
Alsea Bay (3)	5.9 ± 1^{c}	1.4 ± 0.2^{d}	$<DL^{b}$	$<DL^{b}$	$<DL^{c}$	0.08 ± 0.04^{b}	1.3 ± 0.2^{c}	$<DL^{b}$
Coos Bay (1)	14^{c}	1.8^{d}	$<DL^{b}$	$<DL^{b}$	$<DL^{c}$	$<DL^{b}$	1.8^{c}	$<DL^{b}$
Grays Harbor (1)	$27^{b,c}$	3.4^{d}	$<DL^{b}$	$<DL^{b}$	$<DL^{c}$	0.26^{b}	3.0^{c}	0.13^{b}
Willapa Bay (1)	6.4^{c}	0.9^{d}	$<DL^{b}$	$<DL^{b}$	$<DL^{c}$	0.13^{b}	0.63^{c}	0.12^{b}
Yaquina Bay (3)	11^{c}	1.7 ± 0.4^{d}	$<DL^{b}$	$<DL^{b}$	$<DL^{c}$	0.13 ± 0.07^{b}	1.6 ± 0.4^{c}	0.4 ± 0.02^{b}

from the Columbia River, Yaquina Bay, Grays Harbor, and Salmon River, 4% of total DDTs in juvenile coho from Grays Harbor, and 13% of total DDTs in coho from Willapa Bay. In hatchery chinook salmon, *p,p′*-DDT accounted for an average of 5% of total DDTs. Concentrations of estrogenic *o,p′*-DDT, *o,p′*-DDD, and *o,p′*-DDE (Fig. 6) were below detection limits in all coho and many chinook salmon sampled, but were present at concentrations above 0.1 ng/g ww or 10 n/g lw in chinook salmon from the Columbia, Nisqually, Duwamish and Yaquina Bay Estuaries. As with ΣDDTs, concentrations of the *o,p′* isomers were highest in chinook from the Columbia River. In hatchery chinook salmon, they averaged 8 ng/g lw.

We calculated the ΣDDTs/ΣPCBs ratios in whole body samples of chinook and coho salmon to identify groups of fish with distinct contaminant profiles (Fig. 7). In coho salmon, the mean ΣDDTs/ΣPCBs ratio was 0.2, and in estuarine chinook salmon, the mean ratio was 0.4. In both coho and chinook salmon from most of the sites we sampled (Nisqually, Skokomish, Coos Bay, Alsea Bay Estuary, Salmon River Estuary, Willapa Bay, Elk River Estuary, Duwamish Estuary, Tillamook Bay, Yaquina Bay), ΣDDT/ΣPCB ratios were 0.5 or lower. This was not true, however, of chinook salmon from the Columbia River, whose ΣDDTs/ΣPCBs ratios were 1.0–1.1. In hatchery chinook, the mean ΣDDTs/ΣPCBs ratio was ∼0.7.

In addition to PCBs and DDTs, chlordanes, hexachlorobenzene, and dieldrin were detected in whole bodies of estuarine chinook and coho salmon from one or more sampling sites, but at much lower concentrations than PCBs or DDTs (mean concentrations ranging from <1 ng/g ww to 4 ng/g ww; Table 4). Of the

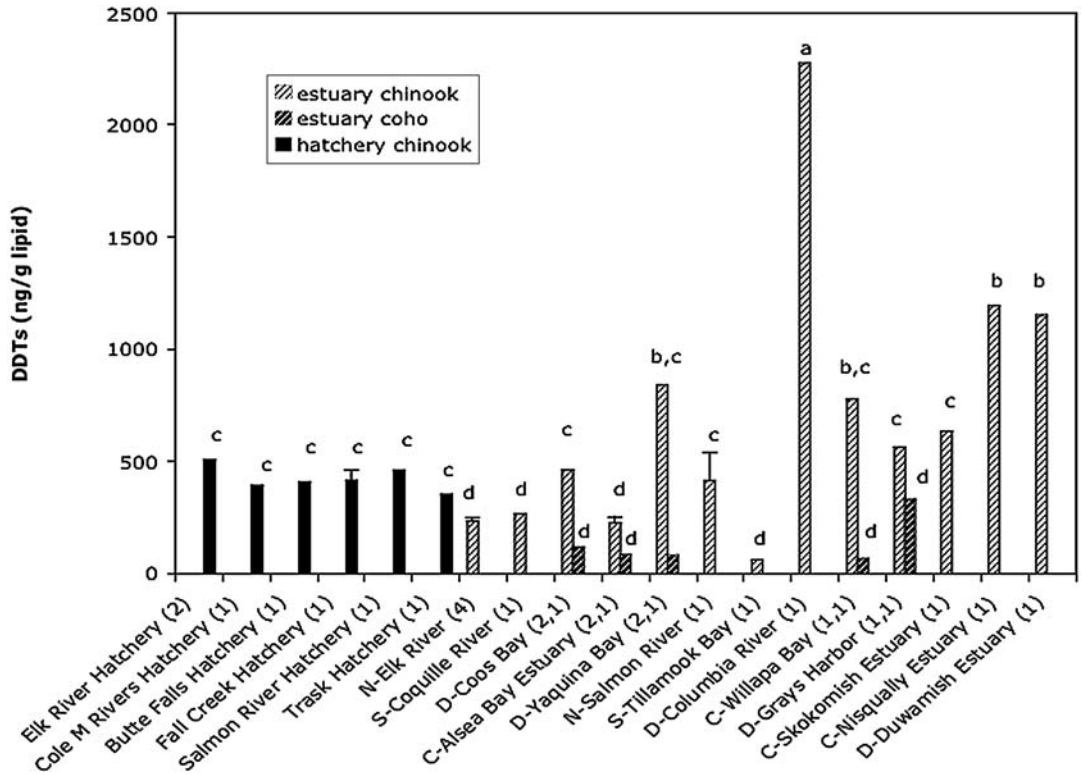

Fig. 4 Mean concentrations of ΣDDTs (ng/g lipid, ± SE) in whole bodies of juvenile chinook and coho salmon from Pacific Northwest estuaries and juvenile chinook salmon from associated hatcheries. N = natural estuary; C = conservation estuary; S = shallow draft estuary; D = deep draft estuary. Numbers in parentheses indicate number of composite samples (10–15 fish each) analyzed per site or group. Measurements with different letters are significantly different (ANOVA, $p < 0.05$)

pesticides detected, chlordanes were generally found at the highest concentrations. Other OC pesticides (i.e., lindane, mirex and aldrin) were below the limits of detection (generally <0.5 ng/g ww) in all samples. Dieldrin, chlordanes, and HCB were detected in whole bodies of juvenile chinook from all sampled hatcheries, typically at concentrations in the 1–5 ng/g ww range. Concentrations were comparable to the highest levels reported in estuarine chinook and coho (Table 4).

3.3 Bile metabolites

Levels of high molecular weight AH metabolites in bile (FACs-BaP) were low to moderate (100–400 ng/g bile) in juvenile fall chinook and coho salmon collected from most of the estuaries sampled along the Washington and Oregon Coast (Fig. 8). Concentrations in chinook salmon from the Duwamish Estuary (∼1930 ng BaP equiv/g bile) were significantly higher than in fish from any other sites. FAC-BaP levels were also somewhat elevated (350–500 ng/g bile) in chinook salmon from the Columbia River, Skokomish Estuary, Grays Harbor, and Willapa Bay, and in coho salmon from Grays Harbor. Lowest concentrations were observed in chinook and coho salmon from Elk River Estuary, Yaquina Bay Estuary, and Alsea Bay Estuary. At 100–200 ng BaP equiv/g bile, concentrations of FACs-BaP in fish at these sites were significantly lower than in chinook salmon from the Columbia, Skokomish, Willapa Bay, and Duwamish sites, and in chinook and coho salmon from Grays Harbor.

Concentrations of metabolites of low molecular weight PAHs (FAC-PHN; Fig. 8) were also significantly higher in chinook salmon from the Duwamish Estuary (359,000 ng PHN equiv/g bile) than in fish from any other sites. Concentrations in chinook salmon from Grays Harbor, Coos Bay, and the Columbia River (60,000–70,000 ng PHN equiv/g bile) were much lower than in the Duwamish chinook, but significantly above levels in either coho or

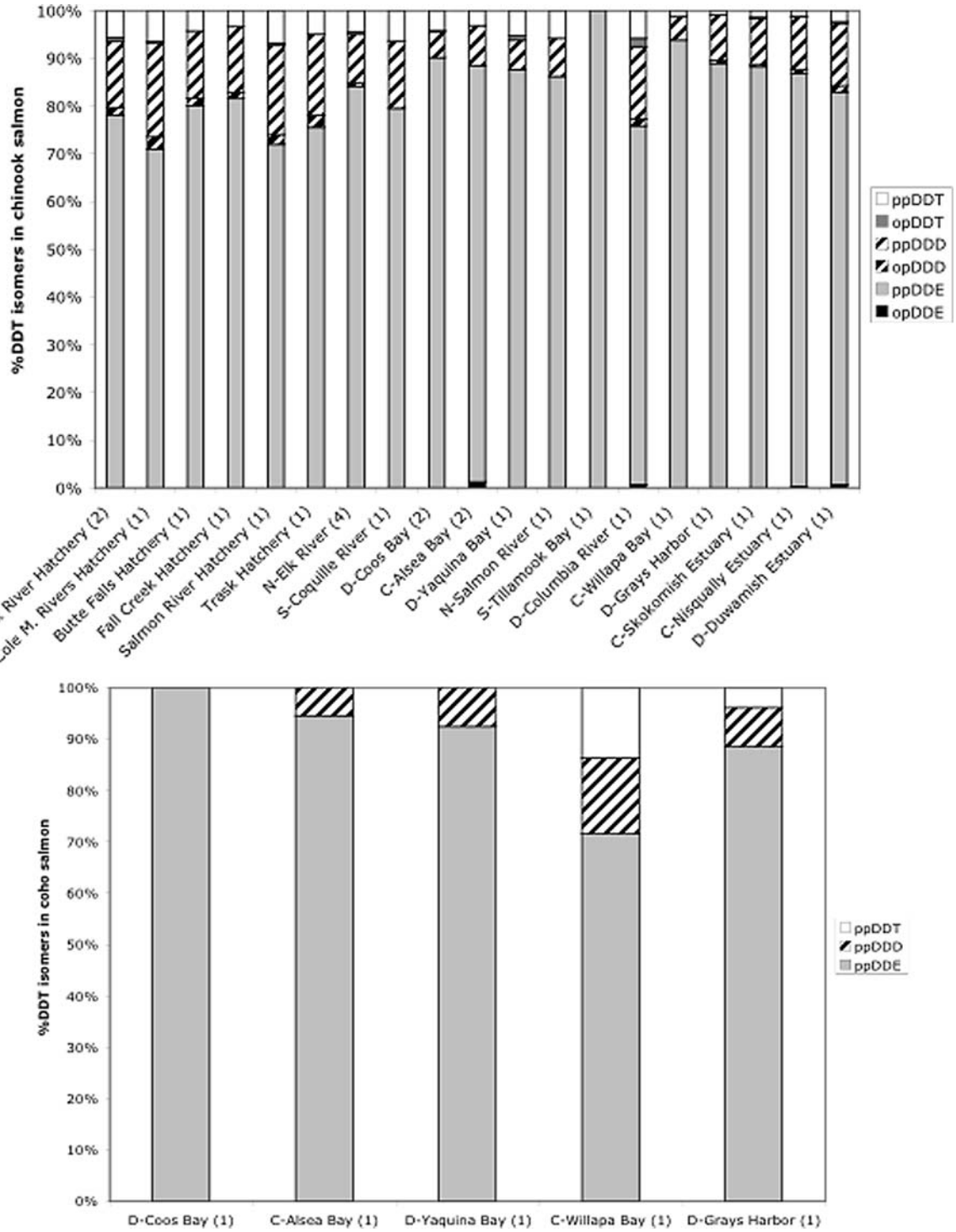

Fig. 5 Proportions of various DDTs in composite whole body samples of juvenile chinook and coho salmon collected from Pacific Northwest estuaries and hatcheries. N = natural estuary; C = conservation estuary; S = shallow draft estuary; D = deep draft estuary. Numbers in parentheses indicate number of composite samples (10–15 fish each) analyzed per site or group

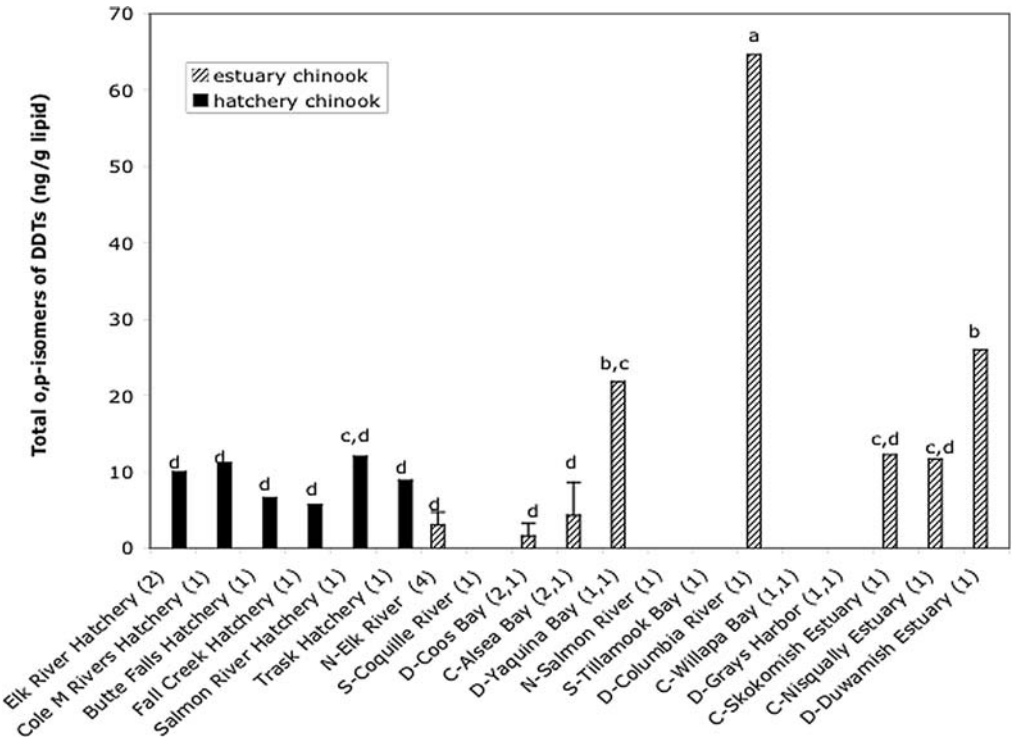

Fig. 6 Mean concentrations of $\Sigma o, p'$-isomers of DDTs (ng/g lipid, ± SE) in whole bodies of juvenile chinook and coho salmon from Pacific Northwest estuaries and juvenile chinook salmon from associated hatcheries. N = natural estuary; C = conservation estuary; S = shallow draft estuary; D = deep draft estuary. Numbers in parentheses indicate number of composite samples (10–15 fish each) analyzed per site or group. Measurements with different letters are significantly different (ANOVA, $p < 0.05$). Values were below detection limits for coho from all sites where they were sampled, and for chinook from Coquille River

chinook salmon from the other sampling sites, whose biliary FACs-PHN concentrations were 30,000 ng PHN equiv/g bile or less. Bile sample could not be collected from chinook salmon at the hatcheries.

3.4 Contaminants in stomach contents

Several classes of contaminants, including PCBs, DDTs, and low and high molecular weight PAHs, were present at detectable concentrations in stomach contents of outmigrant juvenile chinook and coho salmon. Concentrations of ΣLAHs in stomach contents of estuarine chinook salmon (Fig. 9; Table 2) ranged from 12 ng/g ww at the Elk River Estuary to 8000 ng/g ww at the Duwamish Estuary. Concentrations of ΣLAHs were also fairly high in fish from Willapa Bay, Yaquina Bay, and Grays Harbor in comparison to other sites, ranging from 350 to 1400 ng/g ww. Concentrations of ΣLAHs in stomach contents of chinook and coho salmon from all other sites were <100 ng/g ww (Fig. 9; Table 2). At sites where both species were collected, average ΣLAH concentrations in stomach contents of chinook salmon were higher than in coho salmon (920 ng/g ww vs. 5 ng/g ww). In chinook salmon from Elk River Hatchery, the concentration of ΣLAHs in stomach contents was 28 ng/g ww (Fig. 9; Table 2).

Concentrations of ΣHAHs in stomach contents of juvenile chinook salmon (Fig. 9, Table 2) were highest in fish from the Duwamish Estuary and Willapa Bay (6000–6300 ng/g ww). Concentrations of ΣHAHs at Grays Harbor and Yaquina Bay (330–340 ng/g ww) were also relatively high in comparison to other sites, where concentrations were ∼20 ng/g ww and below. The lowest levels ΣHAHs (1–2 ng/g ww) were observed in chinook from Salmon River and Elk River Estuary sites. In coho salmon (Fig. 9; Table 2) concentrations of ΣHAHs in stomach contents were ∼10 ng/g ww or below in fish from all sites; at sites where both species were collected, ΣHAH concentrations were higher in chinook salmon than in coho salmon (323 ng/g ww vs. 40 ng/g ww). In chinook and coho salmon from most sampling sites, HAHs accounted for

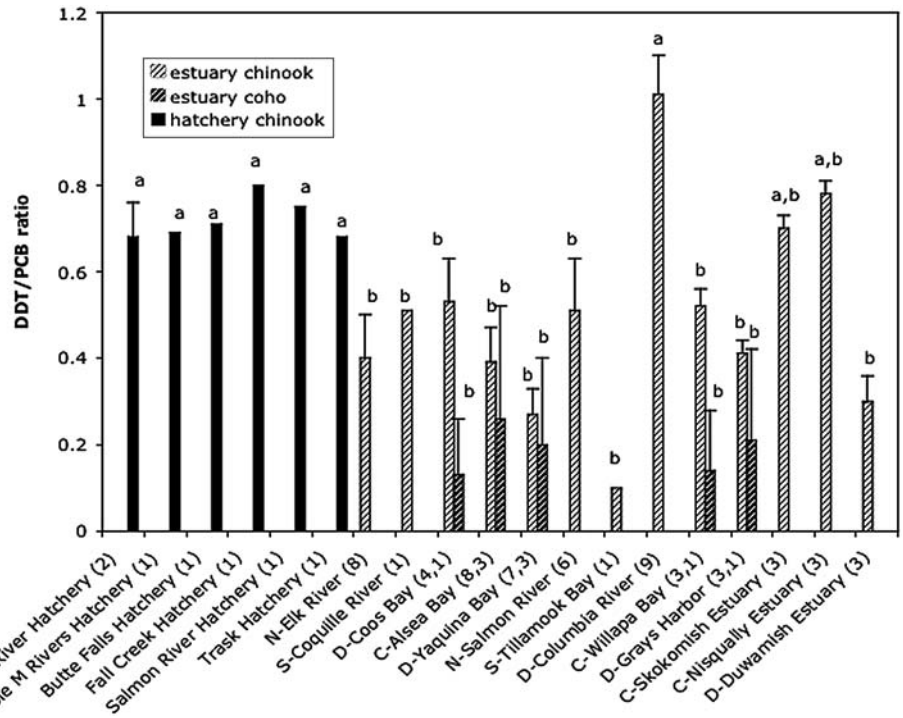

Fig. 7 Mean ΣDDT/ΣPCB ratios (± SE) in whole bodies of juvenile chinook and coho salmon from Pacific Northwest estuaries and juvenile chinook salmon from associated hatcheries. N = natural estuary; C = conservation estuary; S = shallow draft estuary; D = deep draft estuary. Numbers in parentheses indicate number of composite samples (10–15 fish each) analyzed per site or group. Measurements with different letters are significantly different (ANOVA, $p < 0.05$)

10–20% of total AHs. However, in chinook salmon from the Duwamish, Grays Harbor, Yaquina Bay, and Willapa Bay, HAHs were more predominant, accounting for 30–70% of total AHs. In chinook salmon from the Elk River Hatchery (Fig. 9), ΣHAH concentrations were relatively low (5 ng/g ww) and accounted for about 15% of total AHs.

Concentrations of ΣPCBs in stomach contents of estuarine chinook salmon (Fig. 10; Table 2) ranged from 5 ng/g ww in fish from the Salmon River Estuary to 200 ng/g ww in fish from the Duwamish Estuary. Concentrations of PCBs in salmon from the Columbia River and Grays Harbor were about 40 ng/g ww, and concentrations were about 20 ng/g ww or less at all other sampling sites. Lowest levels (5–10 ng/g ww) were observed at Yaquina Bay, Alsea Bay, Coos Bay, Elk River, and Salmon River Estuaries. In coho salmon (Fig. 10, Table 2), PCB concentrations in stomach contents ranged from 5 ng/g ww in fish from Alsea Bay Estuary to 22 ng/g ww in fish from Willapa Bay. At sites where both species were collected, PCB concentrations were similar in stomach contents of chinook salmon and coho salmon, 14 ng/g ww vs. 12 ng/g ww. At the Elk River Hatchery, PCB concentrations in stomach contents were 13 ng/g ww, comparable to levels in estuarine chinook salmon from non-urban sites (Fig. 10; Table 2).

Concentrations of ΣDDTs in stomach contents of estuarine chinook salmon (Fig. 11; Table 2) were highest in fish from Grays Harbor (45 ng/g ww) and the Columbia River (39 ng/g ww), significantly higher than in fish from all other sites. In stomach contents of chinook from all sampling sites except for the Columbia River and Grays Harbor, ΣDDT concentrations were <10 ng/g ww. Concentrations of ΣDDTs in stomach contents of coho salmon (Fig. 11, Table 2) were low (3 ng/g ww) in fish from all sites. At sites where both species were collected, ΣDDT concentrations were higher in chinook salmon than in coho salmon (9 ng/g ww vs. 1.5 ng/g ww). In chinook salmon from the Elk River Hatchery (Fig. 11, Table 2), concentrations of DDTs were also relatively low, 4.5 ng/g ww.

In stomach contents, as in tissues, *p,p′*-DDE was the predominant isomer detected, accounting for about

Table 4 Mean concentrations (± SE) in ng/g, wet wt of selected organochlorine pesticides in bodies of juvenile chinook and coho salmon collected from Pacific Northwest estuaries and hatcheries. Σchlordanes = summed concentrations of heptachlor, heptachlor epoxide, γ-chlordane, α-chlordane, *cis*-nonachlor, *trans*-nonachlor and nonachlor III. DL = detection limit. Pesticides were measured by GC/ECD in samples collected from 1996–1998 and by GC/MS in samples collected from 1999–2001. Values with different letter superscripts are significantly different (ANOVA, $p < 0.05$). Lindane was also measured, but was below DL (generally < 0.5 ng/g ww) in all samples

Site	dieldrin	aldrin	Σchlordanes	HCB	Mirex
Estuary Chinook					
Columbia River (6)	1.9 ± 0.88^{a}	$<DL^{b}$	3.1 ± 0.26^{b}	0.63 ± 0.05^{b}	$<DL^{a}$
Coquille River (1)	0.56^{b}	0.29^{a}	1.5^{c}	$0.65^{a,b}$	0.35^{c}
Alsea Bay (8)	0.69 ± 0.39^{b}	$<DL^{b}$	0.47 ± 0.30^{c}	0.21 ± 0.11^{b}	$<DL^{a}$
Coos Bay (4)	$0.83 \pm 0.83^{a,b}$	$<DL^{b}$	0.73 ± 0.12^{c}	0.33 ± 0.09^{b}	$<DL^{a}$
Duwamish Estuary (3)	$0.97 \pm 0.08^{a,b}$	$<DL^{b}$	4.3 ± 0.18^{a}	0.74 ± 0.09^{b}	$<DL^{a}$
Elk River (2)	0.14 ± 0.11^{b}	$<DL^{b}$	0.64 ± 0.33^{c}	0.21 ± 0.09^{b}	0.06 ± 0.06^{a}
Grays Harbor (3)	0.04 ± 0.04^{b}	$<DL^{b}$	1.53 ± 0.67^{c}	0.26 ± 0.06^{b}	$<DL^{a}$
Nisqually Estuary (3)	$0.71 \pm 0.14^{a,b}$	$<DL^{b}$	3.2 ± 0.46^{b}	0.59 ± 0.12^{b}	0.05 ± 0.05^{a}
Salmon River (11)	$0.78 \pm 0.38^{a,b}$	$<DL^{b}$	0.15 ± 0.09^{c}	0.08 ± 0.04^{c}	$<DL^{a}$
Skokomish Estuary (3)	0.28 ± 0.09^{b}	$<DL^{b}$	2.45 ± 0.51^{b}	0.46 ± 0.15^{b}	0.04 ± 0.04^{a}
Tillamook Bay (1)	$<DL^{b}$	$<DL^{b}$	$<DL^{c}$	$<DL^{c}$	$<DL^{a}$
Yaquina Bay (7)	0.06 ± 0.06^{b}	$<DL^{b}$	1.1 ± 0.6^{c}	0.18 ± 0.08^{b}	$<DL^{a}$
Willapa Bay (3)	$<DL^{b}$	$<DL^{b}$	0.32 ± 0.04^{c}	0.13 ± 0.07^{b}	$<DL^{a}$
Hatchery chinook					
Fall Creek (1)	2.1^{a}	0.22^{a}	4.5^{a}	1.2^{a}	$<DL^{a}$
Butte Falls (1)	1.9^{a}	0.25^{a}	4.7^{a}	1.1^{a}	$<DL^{a}$
Cole M. Rivers (1)	2.3^{a}	$<DL^{b}$	4.2^{a}	$0.88^{a,b}$	$<DL^{a}$
Elk River (2)	1.4 ± 0.9^{a}	$<DL^{b}$	3.7^{a}	$0.65^{a,b}$	0.13 ± 0.13^{b}
Trask (1)	1.7^{a}	$<DL^{b}$	3.6^{a}	$0.87^{a,b}$	$<DL^{a}$
Salmon River (1)	3.7^{a}	$<DL^{b}$	4.4^{a}	1.1^{a}	$<DL^{a}$
Estuary coho					
Alsea Bay (3)	2.5 ± 0.3^{a}	$<DL^{b}$	0.17 ± 0.04^{c}	0.2 ± 0.03b	$<DL^{a}$
Coos Bay (1)	3.3 ± 0.3^{a}	$<DL^{b}$	0.2^{c}	0.16^{b}	0.64^{d}
Grays Harbor (1)	$<DL^{b}$	$<DL^{b}$	0.35^{c}	0.13^{b}	$<DL^{a}$
Willapa Bay (1)	$<DL^{b}$	$<DL^{b}$	0.44 ± 0.26^{c}	0.13 ± 0.0^{b}	$<DL^{a}$
Yaquina Bay (3)	$<DL^{b}$	$<DL^{b}$	0.10^{c}	0.09^{b}	$<DL^{a}$

60–100% of ΣDDTs in stomach contents of both coho and chinook salmon from all sites (Fig. 12; Table 5). Additionally, *p,p′*-DDD and *p,p′*-DDT were found in both chinook and coho salmon stomach contents from several sites, with highest concentrations in juvenile chinook from the Columbia River (5.9 and 2.5 ng/g ww for *p,p′*-DDD and *p,p′*-DDT, respectively). These isomers accounted for 5–25% of total DDTs. In comparison with salmon whole bodies, *p,p′*-DDT was found at higher concentrations in stomach contents. The *o,p'*-DDTs were found only in stomach contents of chinook salmon from the Columbia River, which had measurable concentrations (0.6–1.1 ng/g ww) of both *o,p′*-DDT and *o,p′*-DDD. In stomach contents of juvenile chinook from the Elk River Hatchery, the only DDT isomer found was *p,p′*-DDE, which was present at a concentration of 4.5 ng/g ww.

In addition to PCBs, DDTs, and PAHs, chlordanes HCBs, HCHs, dieldrin, and mirex were detected in stomach contents of estuarine chinook or coho from one or more sampling sites (Table 6). In stomach contents of chinook from the Elk River Hatchery, chlordanes, HCB, and mirex were detected, all at relatively low levels (0.7–1.4 ng/g ww). Aldrin was below the limits of detection in all samples.

3.5 Relationship between contaminants in stomach contents and in salmon bodies

In chinook salmon, concentrations of PCBs and DDTs in stomach contents were significantly and positively

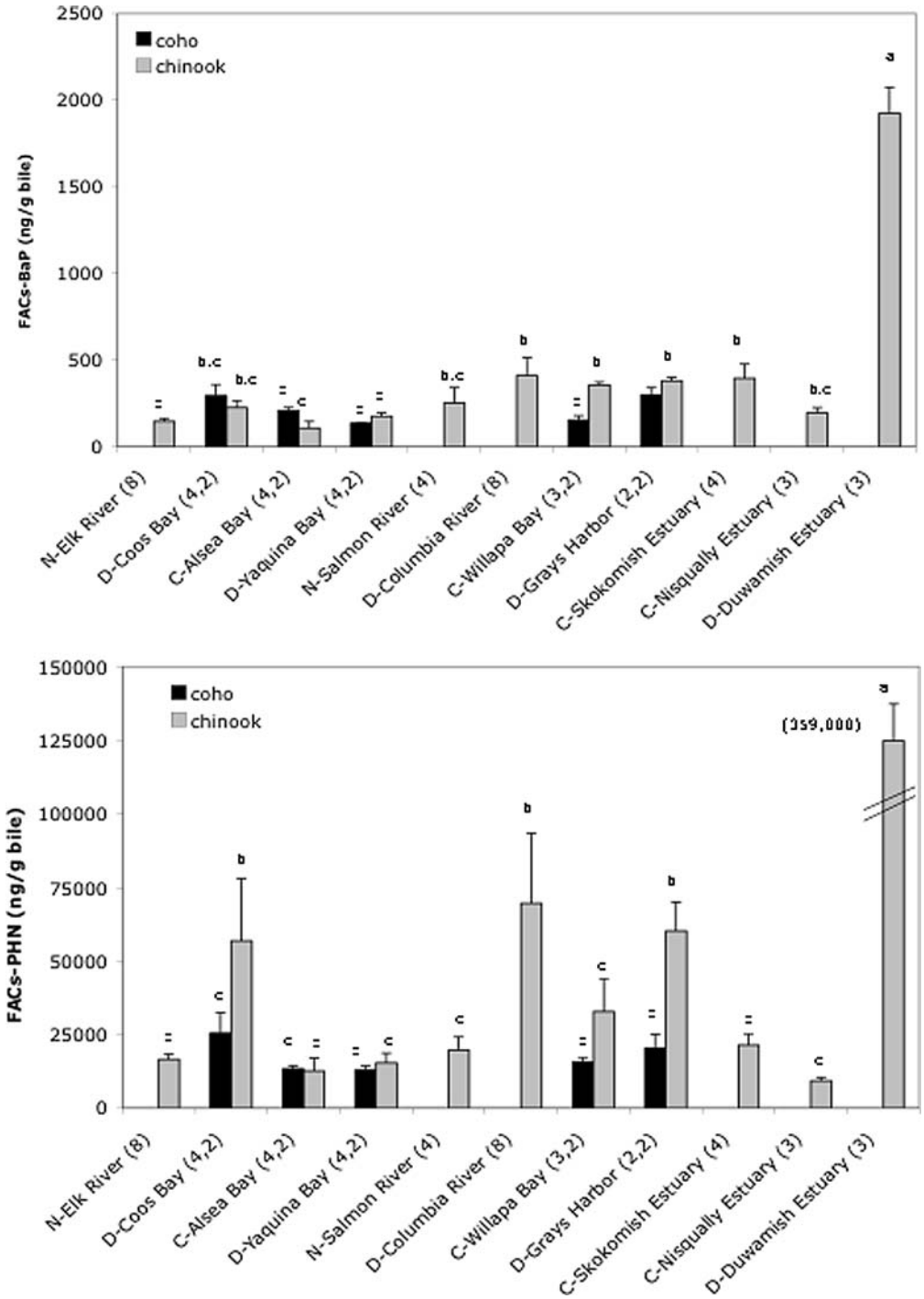

Fig. 8 Mean concentrations of fluorescent aromatic compounds (± SE) measured at phenanthrene wavelengths (FACs-PHN) and benzo[a]pyrene wavelengths (BaP-FACs) in bile of juvenile chinook and coho almon from Pacific Northwest estuaries. N = natural estuary; C = conservation estuary; S = shallow draft estuary; D = deep draft estuary. Bile metabolites measured at PHN and BaP wavelengths are representative of metabolites of low and high molecular weight PAHs, respectively. Numbers in parentheses indicate number of composite samples (10–15 fish each) analyzed per site or group. Measurements with different letters are significantly different (ANOVA, $p < 0.05$)

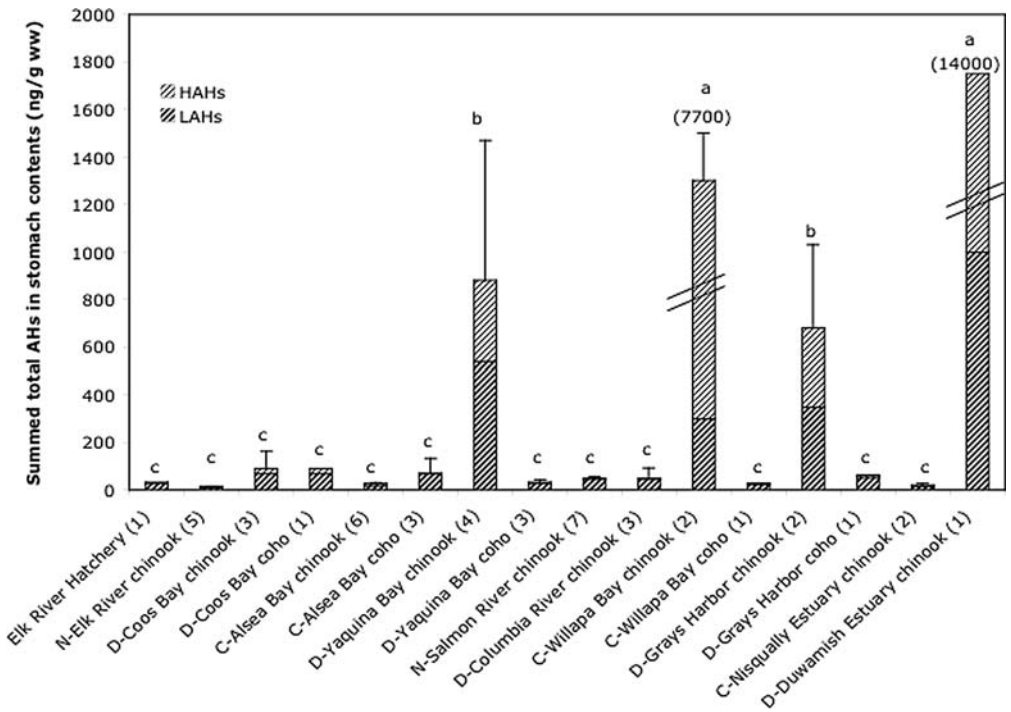

Fig. 9 Mean concentrations of total aromatic hydrocarbons (ΣAHs) (ng/g wet wt, ± SE) in stomach contents of juvenile chinook and coho salmon from Pacific Northwest estuaries and juvenile chinook salmon from Elk River hatchery. N = natural estuary; C = conservation estuary; S = shallow draft estuary; D = deep draft estuary. Contributions of low molecular weight and high molecular weight AHs (LAHs and HAHs) to totals are indicated. Numbers in parentheses indicate number of composite samples (10–15 fish each) analyzed per site or group. Measurements with different letters are significantly different (ANOVA, $p < 0.05$)

correlated with body burdens of the same contaminants. For PCBs ($n = 46$), $r^2 = 0.32$, $p = 0.0001$; while for DDTs ($n = 40$), $r^2 = 0.38$, $p = 0.0001$. In coho salmon, concentrations of contaminant in bodies and stomach contents were also positively correlated, but relationships were marginally significant ($0.06 \leq p \leq 0.08$), in part because of smaller sample size. For body DDTs vs. stomach DDTs ($n = 9$), $r^2 = 0.34$, $p = 0.06$. For body PCBs vs. stomach PCBs ($n = 9$), $r^2 = 0.29$, $p = 0.08$.

In estuarine chinook salmon, concentrations of PCBs and DDTs (ng/g ww) in whole bodies were 3–4 times as high as in stomach contents on average, while in coho salmon, concentrations of PCBs and DDTs in whole bodies and stomach contents were about the same or only slightly higher (1–1.3 times). For chinook salmon from the Elk River Hatchery (the only hatchery where stomach contents data were available), concentrations of PCBs (ng/g ww) were 4.7 times as high in bodies as in stomach contents, while concentrations of DDTs (ng/g ww) were 25 times as high in bodies as in stomach contents.

In chinook salmon, concentrations of PAH metabolites in bile and PAHs in stomach contents were significantly, positively correlated. For ΣLAHs vs. FACs-PHN, $n = 35$, $p = 0.0001$, $r^2 = 0.56$, and for ΣHAHs vs. FACs-BaP, $n = 35$, $p = 0.0006$, $r^2 = 0.28$. In coho salmon, on the other hand, there was no significant correlation between concentrations of either ΣHAHs or ΣLAHs in stomach contents and concentrations of PAH metabolites in bile. For ΣHAHs, $n = 5$, $r^2 = 0.07$, $p = 0.33$. For ΣLAHs, $n = 5$, $r^2 = 0.18$, $p = 0.26$.

4 Discussion

Estuarine and nearshore ecosystems provide a vital role as juvenile rearing habitat for salmonid species (Levy and Northcote, 1982; Gray *et al.*, 2002; Rice *et al.*, 2005), and can be particularly important in the recovery of species at risk (Feist *et al.*, 2003; Fresh *et al.*, 2005). Unfortunately, estuarine and coastal ecosystems are also among the environments that are most heavily

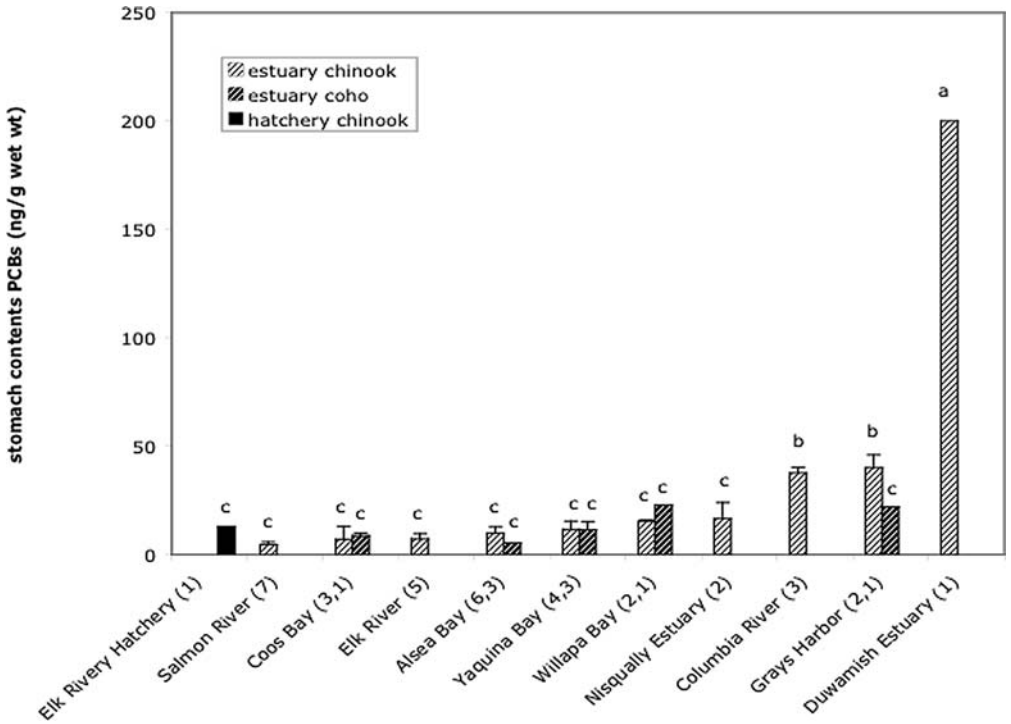

Fig. 10 Mean concentrations of ΣPCBs (ng/g wet wt. ± SE) in stomach contents of juvenile chinook and coho salmon from Pacific Northwest estuaries and juvenile chinook salmon from Elk River hatchery. N = natural estuary; C = conservation estuary; S = shallow draft estuary; D = deep draft estuary. Numbers in parentheses indicate number of composite samples (10–15 fish each) analyzed per site or group. Measurements with different letters are significantly different (ANOVA, $p < 0.05$)

impacted by anthropogenic activities (Shreffler *et al.*, 1990; Beck *et al.*, 2001; Rice *et al.*, 2005). Analyses of risks to salmon populations in estuarine environments have focused largely on alterations to or loss of physical habitat attributes (Bottom *et al.*, 2005; Gray *et al.*, 2002; Fresh *et al.*, 2005), but it is increasingly recognized that habitat degradation associated with chemical contaminants may also pose a significant risk to salmon populations (Spromberg and Meador, 2005; Fresh *et al.*, 2005; Loge *et al.*, 2005).

The importance of estuarine contamination in terms of the health of salmonid species depends in part on the life history strategy of the species in question. In general, ocean-type stocks, such as fall chinook, which spend an extended period during their first year of life in the estuary, are more vulnerable to the impacts of contaminants in this environment than stream-type stocks, such as coho salmon, which pass through the estuary relatively quickly (Fresh *et al.*, 2005). The same may be true of chum salmon, which have a long estuarine residence time (Dorcey *et al.*, 1978; Healey, 1982). Juvenile chum have shown relatively high contaminant body burdens at urban sites in previous surveys in Puget Sound, WA (Stehr *et al.*, 2000).

The results of the current study confirm that chemical contaminants are present in the prey and tissues of outmigrant juvenile salmon from a number of estuaries in the Pacific Northwest. The most widespread contaminants were PCBs, DDTs, and PAHs, which were observed in both tissues and stomach contents of chinook and coho salmon from all estuarine sampling sites, as well as in chinook salmon from local hatcheries. Although additional organochlorine pesticides (chlordanes, lindane, hexachlorobenzene, dieldrin, aldrin and mirex) were also detected in salmon tissues or stomach contents, the measured concentrations were relatively low. Like earlier studies in Puget Sound, the present study highlights the importance of the estuary as a source of exposure to chemical contaminants, especially for juvenile chinook salmon. The observation of elevated contaminant concentrations in stomach contents of salmon from sites in several estuaries indicates that fish are being exposed to these contaminants during estuarine residence through their

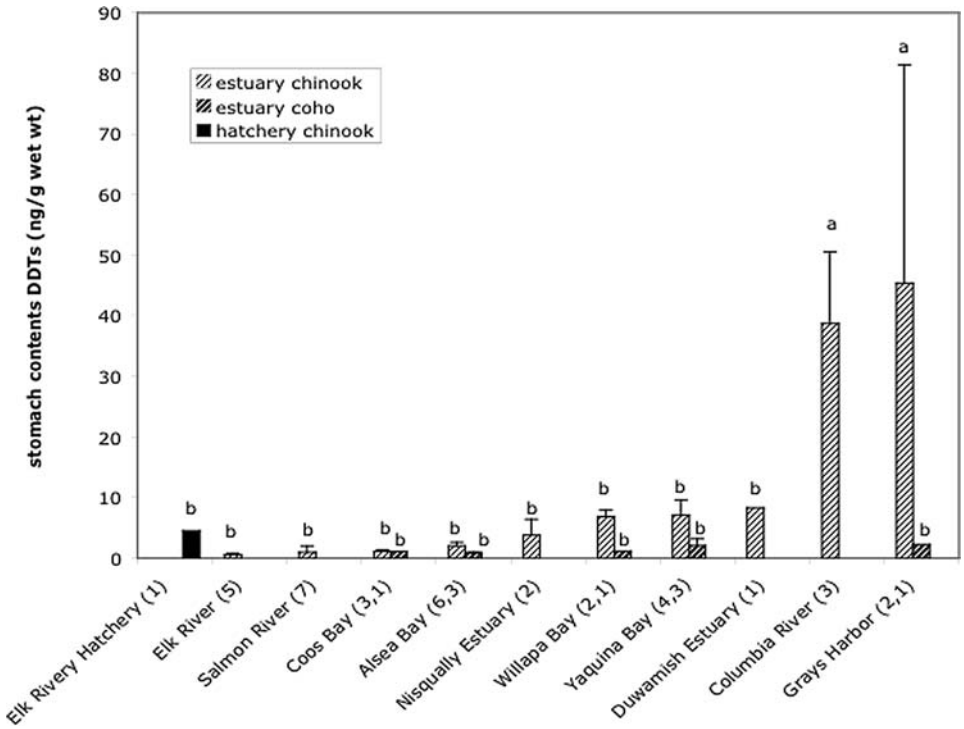

Fig. 11 Mean concentrations of ΣDDTs (ng/g ww, ± SE) in stomach contents of juvenile chinook and coho salmon from Pacific Northwest estuaries and juvenile chinook salmon from Elk River hatchery. N = natural estuary; C = conservation estuary; S = shallow draft estuary; D = deep draft estuary. Numbers in parentheses indicate number of composite samples (10–15 fish each) analyzed per site or group. Measurements with different letters are significantly different (ANOVA, $p < 0.05$)

prey. The hypothesis that this could be an important source of uptake is further supported by the significant correlations between concentrations of PCBs and DDTs in stomach contents and whole bodies of juvenile chinook salmon, and between PAHs in stomach contents and PAH metabolites in bile. Contaminants in the water column, and in suspended particulate material, are also potential sources of exposure, although they were not measured in this study. Depending on their origin, chinook and coho salmon from some populations could also be taking up certain contaminants through the water column or the diet in freshwater before entering the estuary. This is especially true if they are passing through urbanized watersheds. However, the potential contribution of contaminants in freshwater habitats to juvenile salmon body burdens cannot be evaluated based on the samples collected in the present study.

4.1 Species differences in contaminant uptake

Of the two species we examined, chinook salmon exhibited the highest degree of uptake and accumulation of contaminants. On both a lipid weight and a wet weight basis, contaminant concentrations in whole bodies of chinook salmon were significantly higher than in coho salmon sampled from the same sites, with levels typically 2–5 times as great in chinook than in coho salmon collected at the same sites. Concentrations of contaminants in chinook salmon stomach contents tended to be higher as well, although the difference was less marked. Additionally, correlations between contaminant body burdens and contaminant concentrations in stomach contents were stronger in chinook than in coho salmon.

These findings are consistent with results of other studies on chinook and coho salmon in the Great Lakes (Manchester-Neesvig *et al.*, 2001; Jackson *et al.*, 2001; Rohrer *et al.*, 1982), and are likely related to differences in life history and habitat use, as well as diet and metabolism. Assuming that the estuary is an important source of contaminants for outmigrant salmonids, these differences are consistent with the more prolonged period of estuarine residence in chinook salmon. Of the five species of Pacific salmon, chinook salmon

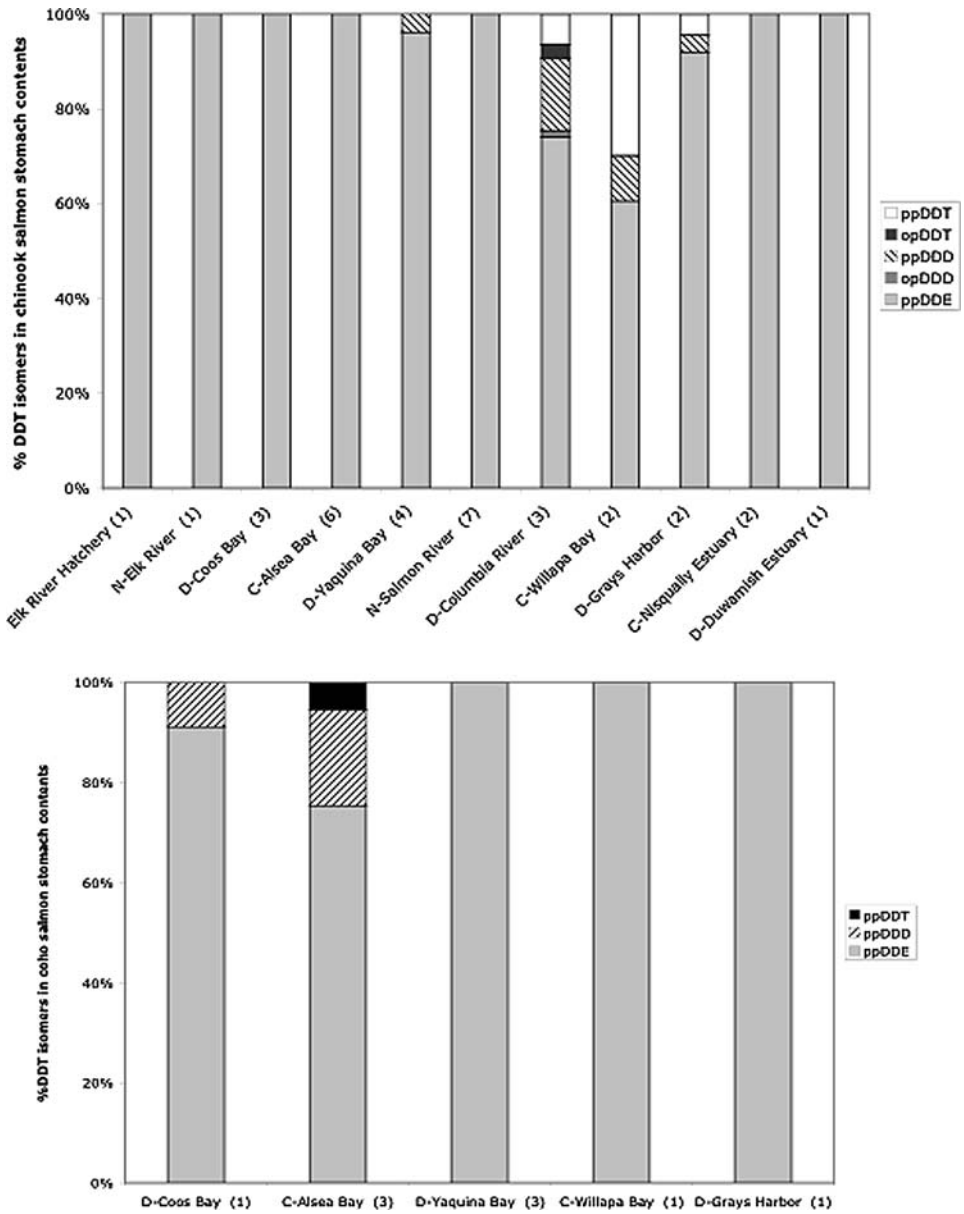

Fig. 12 Proportions of different DDTs in composite stomach contents samples of juvenile chinook and coho salmon collected from Pacific Northwest Estuaries. N = natural estuary; C = conservation estuary; S = shallow draft estuary; D = deep draft estuary. Numbers in parentheses indicate number of composite samples (10–15 fish each) analyzed per site or group

are most dependent upon estuaries during the early stages of their life cycle (Healey, 1982; 1991; Healey and Prince, 1995), typically residing in estuaries for one to two months (Simenstead *et al.*, 1982), but in some cases for up to 6 months (Healey, 1982; Reimers, 1973; Levy and Northcote, 1982; Simenstad *et al.*, 1982). Outmigrant juvenile coho, on the other hand, are much less estuarine-dependent, typically passing through the estuary within a few days (Moser *et al.*, 1991; McMahon and Holtby, 1992; Magnusson, 2003; Duffy *et al.*, 2005). Increased bioaccumulation in chinook salmon may also indicate that they are feeding at a higher trophic level than coho salmon, which would be supported by the generally higher concentrations of PCBs and DDTs in stomach contents of chinook salmon in comparison with levels in stomach contents of coho salmon collected from the same sites. This is consistent with dietary studies showing that,

Table 5 Mean concentrations (± SE) in ng/g wet wt of DDT isomers in stomach contents composites of juvenile chinook and coho salmon from Pacific Northwest estuaries, and juvenile chinook salmon from Elk River Hatchery. DDTs were measured by GC/ECD in samples collected from 1996–1998 and by GC/MS in samples collected from 1999–2001. Composites contain stomach contents from 10–15 fish. Values with different letter superscripts are significantly different (ANOVA, $p \leq 0.05$)

Site	*o,p′*-DDD	*o,p′*-DDT	*p,p′*-DDE	*p,p′*-DDD	*p,p′*-DDT
Hatchery chinook					
Elk River (1)	<DL^b	<DL^b	4.5^b	<DL^b	<DL^b
Estuary chinook					
Alsea Bay (6)	<DL^b	<DL^b	2.0 ± 0.6^b	<DL^b	<DL^b
Columbia River (3)	0.6 ± 0.6^a	1.1 ± 0.6^a	28.7 ± 9.1^a	5.9 ± 0.7^a	2.5 ± 1.4^a
Coos Bay (3)	<DL^b	<DL^b	1.1 ± 0.3^b	<DL^b	<DL^b
Duwamish Estuary (1)	<DL^b	<DL^b	5.8^b	<DL^b	2.5^a
Elk River (5)	<DL^b	<DL^b	0.6 ± 0.2^b	<DL^b	<DL^b
Grays Harbor (2)	<DL^b	<DL^b	41.7 ± 32.3^a	1.6 ± 1.6^b	2.1 ± 2.1^a
Nisqually Estuary (2)	<DL^b	<DL^b	3.5 ± 2.3^b	0.3 ± 0.3^b	<DL^b
Salmon River (7)	<DL^b	<DL^b	1.0 ± 1.0^b	<DL^b	<DL^b
Willapa Bay (2)	<DL^b	<DL^b	4.2 ± 0.4^b	0.7 ± 0.7^b	2.1 ± 2.1^a
Yaquina Bay (3)	<DL^b	<DL^b	6.9 ± 2.2^b	0.3 ± 0.3^b	<DL^b
Estuary coho					
Alsea Bay (3)	<DL^b	<DL^b	0.8 ± 0.1^b	0.11 ± 0.1^b	<DL^b
Coos Bay (1)	<DL^b	<DL^b	1.1^b	<DL^b	<DL^b
Grays Harbor (1)	<DL^b	<DL^b	2.3^b	<DL^b	<DL^b
Willapa Bay (1)	<DL^b	<DL^b	1.2^b	<DL^b	2.5^a
Yaquina Bay (3)	<DL^b	<DL^b	1.9 ± 0.9^b	0.2 ± 0.1^b	0.1 ± 0.1^b

Table 6 Mean concentrations (± SE) in ng/g, wet wt of selected organochlorine pesticides measured in stomach contents of juvenile chinook and coho salmon collected from the Pacific Northwest estuaries and hatcheries. Σchlordanes = summed concentrations of heptachlor, heptachlor epoxide, γ-chlordane, α-chlordane, *cis*-nonachlor, *trans*-nonachlor and nonachlor III. DL = detection limit. Pesticides were measured by GC/ECD in samples collected from 1996–1998 and by GC/MS in samples collected from 1999–2001. Values with different letter superscripts are significantly different (ANOVA, $p \leq 0.05$)

Site	lindane	dieldrin	Σchlordanes	HCB	mirex
Hatchery chinook					
Elk River (1)	<DL^b	<DL^b	1.4^c	0.7^b	0.7^b
Estuary chinook					
Alsea Bay (6)	<DL^b	<DL^b	<DL^c	0.6 ± 0.3^{b,c}	0.2 ± 0.2^b
Columbia River (3)	<DL^b	6.0 ± 6.0^a	0.8 ± 0.5^c	1.5 ± 0.8^{a,b}	0.3 ± 0.3^b
Coos Bay (3)	<DL^b	<DL^b	<DL^c	0.3 ± 0.2^c	0.6 ± 0.6^b
Duwamish Estuary (1)	<DL^b	<DL^b	12^a	<DL^c	2.5^b
Elk River (5)	<DL^b	<DL^b	1.4^c	0.3 ± 0.2^c	0.24 ± 0.25^b
Grays Harbor (2)	1.8 + 1.8^a	1.5 ± 1.5^{a,b}	6.1 ± 0.6^b	1.9 ± 1.9^a	2.7 ± 2.7^b
Nisqually Estuary (2)	<DL^b	0.9^b	0.5 ± 0.5^c	0.17 ± 0.17^c	<DL^b
Salmon River (7)	<DL^b	<DL^b	<DL^c	<DL^c	<DL^b
Willapa Bay (2)	<DL^b	6.5 ± 6.5^a	<DL^c	<DL^c	6 ± 6^a
Yaquina Bay (3)	0.6 + 0.6^a	<DL^b	1.8 ± 1.8^c	0.24 ± 0.24^c	0.4 ± 0.4^b
Estuary coho					
Alsea Bay (3)	<DL^b	<DL^b	0.17 ± 0.06^c	0.72 ± 0.22^b	<DL^b
Coos Bay (1)	<DL^b	4.0 ± 4.0^b	0.31^c	0.25^c	<DL^b
Grays Harbor (1)	<DL^b	<DL^b	<DL^c	<DL^c	<DL^b
Willapa Bay (1)	<DL^b	<DL^b	0.65^c	0.65^b	<DL^b
Yaquina Bay (3)	<DL^b	<DL^b	0.69 ± 0.36^c	0.12 ± 0.07^c	<DL^b

while there is considerable overlap in the diet of juvenile coho and chinook salmon, coho tend to consume a lower proportion of juvenile and larval fish and a higher proportion of invertebrates than chinook (Schabetsberger *et al.*, 2003; Brodeur and Pearcy, 1990).

4.2 Site-related differences in contaminant body burdens

Although contaminant concentrations in coho salmon showed no strong spatial trends, in chinook salmon there were marked intersite differences in contaminant concentrations in tissues and stomach contents, with highest exposure levels in the industrial and urbanized estuaries. Concentrations of PCBs were highest in samples from the Duwamish Estuary, and were similar to or somewhat lower than concentrations reported in earlier Puget Sound studies at this location (Stein *et al.*, 1995; Varanasi *et al.*, 1993; Meador *et al.*, 2002). Total PCB concentrations 2 to 3 times higher than those reported in this study have been measured in juvenile chinook collected from heavily contaminated Duwamish Estuary sites (Varanasi *et al.*, 1993; Meador *et al.*, 2002). The somewhat lower concentrations of PCBs observed in juvenile salmon sampled in the present study may be due to differences in sampling location, or because sampling occurred early in the season, when juvenile salmon may have only recently entered the estuary (Bottom *et al.*, 2005). The lower concentrations may also be reflective of a low proportion of hatchery fish in this sample. Such differences in contaminant concentrations between wild and hatchery-released fish have been noted in other studies (Meador *et al.*, 2002). In addition to Duwamish chinook, concentrations of PCBs were also relatively high in chinook salmon from the Columbia River and Yaquina Bay.

Interestingly, PCB concentrations in the juvenile chinook salmon we sampled were quite similar to concentrations reported in returning adult chinook salmon from Washington State (Missildine *et al.*, 2005). Mean concentrations of PCBs in adult chinook ranged from 48–50 ng/g ww in salmon returning to Puget Sound hatcheries (Deschutes and Issaquah), and from 15–29 ng/g ww in salmon returning to coastal hatcheries (Makah and Quinault). Although it is unlikely that exposures occurring in the juvenile stage make a major contribution to adult contaminant body burdens (O'Neill *et al.*, 1998), these data do suggest consistent exposure at multiple life stages for salmon from urban estuaries.

Concentrations of DDTs were especially high in juvenile chinook salmon from the Lower Columbia River and in the Nisqually Estuary in Puget Sound. The high DDT concentrations in Columbia River chinook are consistent with elevated DDT concentrations observed in other resident marine and freshwater fish from the Columbia River in earlier studies by EPA, NOAA, and USGS, and the States of Washington and Oregon (USEPA, 2000; Tetra-Tech Inc., 1993, 1994, 1996; LCREP, 1999; Brown *et al.*, 1998; Foster *et al.*, 2001a,b). As in most environmental samples, DDT breakdown products, especially *p,p′*-DDE, predominated in coho and chinook salmon body and stomach contents samples. However, *p,p′*-DDT and *o,p′*-DDT were also detected in samples from some sites, particularly chinook salmon from the Columbia River and Yaquina Bay, and coho salmon from Willapa Bay. The presence of these parent compounds suggests that there may be fresher sources of DDT in these areas, although the half-lives of *p,p′*- and *o,p′*-DDT in soils can be quite variable (ATSDR, 2002).

Concentrations of PAHs were especially high in stomach contents of fish from the Duwamish Estuary, Willapa Bay, Grays Harbor and Yaquina Bay, although very high concentrations of PAH metabolites in bile (i.e., >1000 ng/g bile for FACs-BaP and >200,000 ng/g bile for FACs-PHN) were observed only in fish from the Duwamish Estuary. In fish from more pristine estuaries such as Alsea Bay, Salmon River, Elk River, and Tillamook, PAH concentrations were lower than any of those previously reported in Puget Sound (Stein *et al.*, 1995; Varanasi *et al.*, 1993; McCain *et al.*, 1990). High molecular weight AHs, which originate primarily from combustion products (Varanasi *et al.*, 1992; MacDonald and Crecelius, 1994), accounted for a higher proportion of total AHs in stomach contents of fish from the Duwamish Estuary, Willapa Bay, Grays Harbor and Yaquina Bay, than in fish from other estuaries. This suggests that atmospheric emissions from incineration and automobile emissions may be major contamination sources in these areas, as well as releases from industries that generate high molecular weight PAHs (e.g., aluminum smelters, oil refineries, creosote plants; Varanasi *et al.*, 1992; MacDonald and Crecelius, 1994). The predominance of LAHs, which are primarily associated with petroleum products (Varanasi *et al.*, 1992; MacDonald and Crecelius,

1994), in stomach contents of salmon from Alsea Bay, Coos Bay, Nisqually, Salmon River, the Columbia River, and Elk River, suggests that PAHs in these areas come mainly from releases of fuel oil, crude oil, and related materials into the environment.

Ratios of ΣDDT/ΣPCB varied from site to site, indicating differences in contaminant profiles among different groups of fish. For example, the ΣDDT/ΣPCB ratio in bodies of salmon from the Columbia Estuary site (∼1.1) was higher than in juvenile chinook salmon the other estuarine sites, suggesting particularly high uptake of DDTs from the environment at this site. Fish from the Duwamish Estuary, the other hand, had one of the lowest DDT/PCB ratios, reflecting the very high concentrations of PCBs in fish from this site.

4.3 Contaminants in hatchery salmon

Measurable concentrations of PCBs and DDTs were also present in bodies of juvenile chinook salmon sampled directly from Pacific Northwest hatcheries. On a wet weight basis, concentrations of both PCBs and DDTs in hatchery chinook were relatively high, comparable to those in juvenile chinook from the more contaminated estuarine sites. However, as the lipid content of hatchery fish was also quite high (8% as compared to 1–3% in estuarine fish), when PCB and DDT body burdens were calculated on a lipid weight basis, concentrations in hatchery chinook were relatively low in comparison to levels in chinook from urban and industrialized estuaries. In stomach contents of juvenile hatchery chinook, levels of PAHs, PCBs, DDTs, were also relatively low, similar to concentrations in rural estuaries such as Elk River and Alsea Bay. This suggests that elevated contaminant concentrations in the hatchery fish we sampled are due not so much to high concentrations of contaminants in feed, but to the high body fat levels in hatchery reared juveniles that facilitate the uptake of lipid soluble contaminants. It is uncertain, though, whether the Elk River Hatchery sample is representative of feed from other sampled hatcheries, or of feeds in current use.

Chemical contaminants, especially PCBs, have been detected in hatchery fish and feed and in farmed fish in several other studies (Easton *et al.*, 2002; Parkins, 2003; Karl *et al.*, 2003; Hites *et al.*, 2004). Available data suggest that the problem is widespread, and also that contaminant concentrations in different lots of feed and in fish from different hatcheries are highly variable. Concentrations of PCBs in juvenile salmon from the Pacific Northwest hatcheries sampled in this study were similar to mean levels (∼50 ng/g ww) reported by Easton *et al.* (2002) and Hites *et al.* (2004) in farmed salmon. However, PCB concentrations in commercial feed analyzed by Easton *et al.* (2002) and Hites *et al.* (2004) were generally higher than PCB concentrations in stomach contents of Elk River Hatchery salmon, with a number of samples in the 30–90 ng/g ww range.

In the hatchery chinook we analyzed, the DDT isomers *p,p′*-DDT and *o,p′*-DDT made up a substantial proportion of DDTs present. This appears to be common in farmed and hatchery fish, and may indicate use of oils or fish meals from sources where there was relatively recent usage of DDTs (Jacobs *et al.*, 2002).

The observation of chemical contaminants in prerelease hatchery fish is likely to be a concern for the management of these animals. If contaminant body burdens are already moderate to high when fish leave the hatchery, they have an increased risk of reaching exposure concentrations during estuarine residence that could significantly reduce their likelihood of survival. Moreover, contaminated salmon may be a significant source of toxicants in the environment and in the food chain (Kreummel *et al.*, 2003). This represents a hazard for birds and other piscivorous wildlife. More comprehensive sampling of fish and feed from hatcheries is needed to determine the extent of this problem in the Pacific Northwest.

4.4 Potential health effects of contaminants on salmon

For some contaminants, exposure levels in juvenile salmon from selected sites are approaching concentrations that could affect their health and survival. Indeed, adverse health effects have been observed in juvenile salmon from the Duwamish Estuary, which is contaminated with PAHs and PCBs. Fish from this area showed immunosuppression, reduced disease resistance and decreased growth rates (Arkoosh *et al.*, 1991, 1994, 1998, 2001; Varanasi *et al.*, 1993; Casillas *et al.*, 1995, 1998), as well as biochemical alterations such as DNA damage (i.e., PAH-DNA adducts in liver) and induction of cytochrome P4501A (CYP1A), an enzyme that metabolizes selected contaminants including PAHs, dioxins and furans, and dioxin-like PCB congeners (Stein *et al.*, 1995; McCain *et al.*, 1990; Varanasi *et al.*, 1993; Collier *et al.*, 1998; Stehr *et al.*,

2000). These biochemical alterations are not necessarily indicative of adverse health effects in themselves, but are associated with disease conditions including reproductive and developmental abnormalities and liver disease (Williams *et al.*, 1998; Whyte *et al.*, 2000; Myers *et al.*, 2003). Fish from several sites sampled in the present study (Grays Harbor, Yaquina Bay, the Columbia River) had concentrations of PCBs, PAHs or both in tissues or stomach contents that were comparable to those found in Duwamish Estuary fish, suggesting that they may also be at risk for the types of adverse health effects documented in fish from that Puget Sound site. The possibility of increased disease-induced mortality is increased by recent finding of widespread occurrence of potentially lethal parasites and pathogens in juvenile chinook and coho salmon from the estuaries sampled in this study (Arkoosh *et al.*, 2004).

The potential for health risks in Pacific Northwest salmon can also be evaluated by comparing measured tissue contaminant concentrations against established effects thresholds. For PCBs, Meador *et al.* (2002) estimated a critical body residue of 2400 ng/g lipid for protection against 95% of effects ranging from enzyme induction to mortality, based on a range of sublethal effects observed in salmonids in peer-reviewed studies conducted by NMFS and other researchers. Mean PCB body burdens in juvenile salmon analyzed in this study were near or above 2400 ng/g lw in fish from three sampling sites, the Columbia River, the Duwamish Estuary, and Willapa Bay. These findings suggest that a significant portion of outmigrant juvenile chinook salmon from these sites may be at risk of some type of health impairment due to PCB exposure.

A threshold concentration for the impact of DDTs on listed salmon has not been systematically determined, unlike the PCBs (Meador *et al.*, 2002). Most reported effects in salmonids are associated with whole body tissue total DDT concentrations at or above 500 ng/g ww (Allison *et al.*, 1963; Burdick *et al.*, 1964; Buhler *et al.*, 1969; Johnson and Pecor, 1969; Peterson, 1976; Poels *et al.*, 1980), or about 5000 ng/g lipid, assuming that the test fish had a lipid content of around 10%, which is typical of laboratory-reared salmonids (Meador *et al.*, 2002). A number of recent studies suggest that certain DDT isomers, such as *o,p′*-DDT and *o,p′*-DDE, have estrogenic activity, and may have endocrine-disrupting or immunotoxic effects (Donohoe and Curtis, 1996; Arukwe *et al.*, 1998; Celius and Walther, 1998; Khan and Thomas, 1998; Christiansen *et al.*, 2000; Zaroogian *et al.*, 2001; Milston *et al.*, 2003; Papoulias *et al.*, 2003). However, measured or estimated body burdens associated with these effects are typically in the 10–20 ng/g ww or 100–200 ng/g lipid range or above. Lipid-adjusted concentrations of total DDTs and *o,p′*-isomers of DDTs approached these concentrations in some fish from the Columbia River, but DDT body burdens typically found in estuarine chinook and coho salmon were substantially lower. This suggests that, by themselves, body burdens of DDTs would be unlikely to cause adverse health effects in most Pacific Northwest juvenile salmon. However, DDTs do not occur in isolation in Pacific Northwest estuaries, but are present with a variety of other contaminants. Estrogenic DDT metabolites, for example, even at low concentrations, could act in concert with other estrogenic contaminants (e.g., plasticizers, pharmaceuticals, and surfactants) to alter reproductive processes or other physiological functions. In fact, some field studies have reported effect thresholds for DDTs lower than those observed in laboratory exposure studies [e.g., maternal muscle concentrations of 25–30 ng/g ww for increased yolk sac fry mortality in Baltic salmon; Vuorinen *et al.* (1997)], possibly because of the presence of other contaminants, as well as lower lipid concentrations in wild fish. More work is needed to understand the potential cumulative effects of DDTs and other contaminants present in salmon habitats.

Exposure to PAHs may also contribute to health risks in juvenile chinook salmon from some of the sampling sites. In juvenile chinook salmon from Puget Sound sites where immunosuppression and other health effects have been observed (Arkoosh *et al.*, 1991, 1994, 1998, 2001; Varanasi *et al.*, 1993; Stein *et al.*, 1995; Casillas *et al.*, 1995, 1998; Stehr *et al.*, 2000), concentrations of total PAHs in stomach contents of these fish were in the 1,200 to 8,000 ng/g ww range for ΣLAHs and in the 2,000 to 6,000 ng/g ww range for ΣHAHs, or 4,000 to 15,000 ng/g ww for total PAHs (Stein *et al.*, 1995; Varanasi *et al.*, 1993; Stehr *et al.*, 2000). In the present study, PAH concentrations in this range were detected once again in chinook salmon from the Duwamish Estuary, suggesting a potential for health risks to fish from this site. Concentrations of ΣHAHs were also surprisingly high in stomach contents of chinook salmon from Willapa Bay, but this was not reflected in bile metabolite levels of fish from this site. Additional sampling may be needed to determine if

there is consistent exposure to PAHs in Willapa Bay salmon.

In laboratory feeding studies where fish were exposed to PAHs alone, reported effect concentrations are somewhat higher than levels of PAHs measured in stomach contents of salmon from sites in where biological effects have been reported in the field, or PAH levels measured in the present study. Meador *et al*. (2005) found physiological changes in juvenile chinook exposed to 120 ppm total PAHs dry wt, or about 25,000 ng/g ww, while Bravo *et al.* (2005) observed immunosuppression, CYP1A induction and DNA damage in rainbow trout exposed to concentrations of 40,000 ng/g ww PAH in diet. Reported no effect doses for immunosuppressive and other physiological effects are in the 8,000–16,000 ng/g ww range (Palm *et al.*, 2004; Meador *et al.*, 2005). Total PAH concentrations in stomach contents of juvenile chinook collected from the Duwamish Estuary and Willapa Bay as part of this study are similar, and thus might be considered as being close to a threshold effect level. Moreover, PAHs may contribute to immunosuppressive or growth-altering impacts of other contaminants in environmental mixtures, even if they are below toxicity thresholds when considered alone (e.g., see Loge *et al*. (2005).

4.5 Trophic transfer and health effects on wildlife

Even if levels of bioaccumulative compounds such as DDTs and PCBs are not sufficient to cause direct effects on juvenile salmonids, they may represent a hazard to fish-eating predators through bioaccumulation and bioconcentration. The U.S. Fish and Wildlife Service (2004) estimated a no-observable adverse effects level (NOAEL) for impacts of fish prey on bald eagles of 60 ng/g ww for PCBs and 40 ng/g ww for DDTs, while Nendza *et al.* (1997) estimated a ΣDDTs NOAEL of 22–50 ng/g ww in fish tissue for impacts of related to bioaccumulation and bioconcentration of DDTs in estuarine systems. Juvenile chinook salmon sampled in this study from the Columbia River, the Duwamish Estuary, and the Nisqually Estuary had whole body DDT concentrations in the 20–50 ng/g ww range, and chinook salmon from the Duwamish Estuary had PCB concentrations above 60 ng/g ww, suggesting these fish may pose a hazard to fish-eating wildlife. Indeed, there is considerable evidence of bioconcentration of DDTs in birds and other wildlife that use the Columbia River, resulting in body burdens high enough to cause reproductive problems (Anthony *et al.*, 1993; USFWS, 1999, 2004; Thomas and Anthony, 2003; Henny *et al.*, 2003; Buck *et al.*, 2005).

4.6 Summary

Overall, the results of this study indicate significant exposure to PCBs, DDTs, and PAHs in outmigrant juvenile chinook salmon from several Pacific Northwest estuaries. Contaminant concentrations were generally highest in stomach contents and tissues of salmon from the deep draft estuaries, with the highest levels of urban and industrial development (i.e., the Duwamish Estuary, the Columbia River, Yaquina Bay, Coos Bay and Grays Harbor), and lowest in the natural estuaries (Elk River and Salmon River), which are largely undeveloped. However, relatively high concentrations of contaminants were detected in juvenile chinook from some of the conservation estuaries (Nisqually Estuary, Skokomish Estuary, Willapa Bay, and Alsea Bay), where land use is primarily agricultural. For example, concentrations of DDTs in salmon from the Nisqually Estuary were among the highest observed in this survey. For juvenile chinook salmon from the Duwamish Estuary, the Columbia River, and Yaquina Bay, whole body PCBs were within the range where they could potentially affect fish health and survival. In juvenile coho salmon, on the other hand, contaminant concentrations were relatively low, below estimated biological effects thresholds, and showed minimal variation from site to site. Juvenile chinook salmon are likely absorbing some contamination during estuarine residence through their prey, as PCBs, PAHs, and DDTs were consistently present in stomach contents, and PCBs and DDTs were significantly correlated with contaminant body burdens in fish from the same sites. Hatchery chinook also showed evidence of contaminant uptake. Although contaminant concentrations were not especially high in stomach contents of fish from the hatchery we tested, body burdens were elevated, in part because of the high lipid content of the fish. More research is needed to document exposure and associated effects of chemical contaminants on endangered Pacific Northwest salmon, but the available data show clearly that tissue burdens of some classes of contaminants are within the range where they could potentially affect survival and productivity of listed stocks or have adverse effects on the ecosystem of which salmon are a part.

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References

Allison, D., Kallman, B.J., Cope, O.B., & Van Valin C. (1963). Insecticides: Effects on cutthroat trout of repeated exposure to DDT. *Science*, *142*, 958–961.

Anthony, R.G., Garrett, M.G., & Schuler, C. (1993). Environmental contaminants in bald eagles in the Columbia River Estuary. *Journal of Wildlife Management, 57*, 10–19.

Arkoosh, M.R., Casillas, E., Clemons, E., McCain, B.B., & Varanasi, U. (1991). Suppression of immunological memory in juvenile chinook salmon (*Oncorhynchus tshawytscha*) from an urban estuary. *Fish & Shellfish Immunology*, *1*, 261–277.

Arkoosh, M.R., Clemons, E., Myers, M., & Casillas, E. (1994). Suppression of B-cell mediated immunity in juvenile chinook *salmon* (*Oncorhynchus tshawytscha*) after exposure to either a polycyclic aromatic hydrocarbon or to polychlorinated biphenyls'. *Immunopharmacology and Immunotoxicology*, *16*, 293–314.

Arkoosh, M.R, Casillas, E., Huffman, P., Clemons, E., Evered, J. Stein, J.E., & Varanasi., U. (1998). Increased susceptibility of juvenile chinook salmon from a contaminated estuary to the pathogen *Vibrio anguillarum*. *Transactions of the American Fisheries Society, 127*, 360–374.

Arkoosh, M.R., Casillas, E., Clemons, E., Huffman, P., Kagley, A.N., Collier, T.K., & Stein, J.E. (2001). Increased susceptibility of juvenile chinook salmon (*Oncorhynchus tshawytscha*) to vibriosis after exposure to chlorinated and aromatic compounds found in contaminated urban estuaries. *Journal of Aquatic Animal Health*, *13*, 257–268.

Arkoosh, M.R, Clemons, E., Kagley, A.N., Stafford, C., Glass, A.C., Jacobson, K., Reno, P., Myers, M.S., Casillas, E., Johnson, L.L., & Collier.T.K. (2004). Survey of pathogens in juvenile (*Onchorhynchus* spp.) migrating through Pacific Northwest estuaries. *Journal of Aquatic Animal Health, 16*, 186–196.

Arukwe, A.T., Celius, B., Walther, T., & Goksoyr, A. (1998). Plasma levels of vitellogenin and eggshell Zona radiata proteins in 4-nonyphenol and *o,p′*-DDT treated juvenile Atlantic salmon (*Salmo salar*). *Marine Environmental Research, 46*, 133–136.

ATSDR (Agency for Toxic Substances and Disease Registry): (2002). *Toxicological profile for DDT, DDE, DDD*, U.S. Department of Health and Human Services, Public Health Service. Atlanta, Georgia.

Beck, M.W., Heck, K.L., Jr., Able, K.W., Childers, D.L., Eggleston, D.B., Gillanders, B.M., Halpern, B., Hays, C.G., Hoshino, K., Minello, T.J., Orth, R.J., Sheridan, P.F., & Weinstein, M.P. (2001). The identification, conservation, and management of estuarine and marine nurseries for fish and invertebrates. *Bioscience*, *51*, 633–641.

Bottom, D.L., Simenstad, C.A., Burke, J., Baptista, A.M., Jay, D.A., Jones, K.K., Casillas, E., & Schiewe, M.H. (2005). Salmon at River's End: The Role of the Estuary in the Decline and Recovery of Columbia River Salmon. NOAA Technical Memorandum, NMFS-NWFSC-68, Northwest Fisheries Science Center, National Marine Fisheries Service, U.S. Department of Commerce, Seattle, Washington.

Bravo, C., Curtis, L., Bayne, C., Gerwick, L., Arkoosh, M., Lambertini, E., Loge, F., & Collier, T. (2005). Increased disease susceptibility in *Oncorhynchus mykiss* associated with exposure to environmentally relevant concentrations of PAH. In: Proceedings of SETAC 26th Annual Meeting in North America, 'Environmental Science in a Global Society: SETAC's Role in the Next 25 Years', November 13–17, 2005, Baltimore, Maryland, USA. Paper BRA-1117-846804.

Brodeur, R.D. & Pearcy, W.G. (1990). Trophic relations of juvenile Pacific salmon off the Oregon and Washington coast. *Fisheries Bulletin, 88*, 617–636.

Brown, D.W., McCain, B.B., Horness, B.H., Sloan, C.A., Tilbury, K.L., S.M.Pierce, S.M., Burrows, D., Chan, S-L., Landahl, J.T., & Krahn, M.M. (1998). Status, correlations, and temporal trends of chemical contaminants in fish and sediment from selected sites on the Pacific Coast of the USA. *Marine Pollution Bulletin, 37*, 67–85.

Buck, J.A., Anthony, R.G., Schuler, C.A., Isaacs, F.B., & Tillitt, D.E. (2005). Changes in productivity and contaminants in bald eagles nesting along the Lower Columbia River, USA. *Environmental Toxicology and Chemistry, 24*, 1779–1792.

Buhler, D.R., Rasmusson, M.E., & Shanks, W.E. (1969). Chronic oral DDT toxicity in juvenile coho and chinook salmon. *Toxicology and Applied Pharmacology, 14*, 535–555.

Burdick, G.E., Harris, E.J., Dean, H.J., Walker, T.M., Skea, J., & Colby, D. (1964). The accumulation of DDT in lake trout and the effect on reproduction. *Transactions of the American Fisheries Society, 93*, 127–136.

Casillas E., Arkoosh, M.R., Clemons, E., Hom, T., Misitano, D., Collier, T.K., Stein, J.E., & Varanasi, U. (1995). Chemical contaminant exposure and physiological effects in outmigrant juvenile chinook salmon from selected urban estuaries of Puget Sound, Washington. In: M. Keefe (ed), *Salmon Ecosystem Restoration: Myth and Reality: Proceedings of the 1994 Northeast Pacific chinook and Coho Salmon Workshop*, Oregon Chapter, Corvallis, OR: American Fisheries Society, pp. 85–102.

Casillas, E., Eberhart, B-T.L. Sommers, F.C., Collier, T.K., Krahn, M.M., & Stein, J.E. (1998). Effects of Chemical contaminants from the Hylebos Waterway on growth of juvenile chinook salmon. *Interpretive Report*, prepared for NOAA Damage Assessment Center by the Northwest Fisheries Science Center, Naional Marine Fisheries Service, Seattle, Washington.

Celius, T., & Walther, B.T. (1998). Differential sensitivity of zonagenesis and vitellogenesis in Atlantic salmon (*Salmo salar* L) to DDT pesticides. *Journal of Experimental Zoology, 281*, 346–353.

Christiansen, L.B., Pedersen, K.L., Pedersen, S.N., Korsgaard, B., & Bjerregaard, P. (2000). In vivo comparison of xenoestrogens using rainbow trout vitellogenin induction as a screening system. *Environmental Toxicology and Chemistry, 19*, 1867–1874.

Collier, T.K., Johnson, L.L., Stehr, C.M., Myers, M.S., Krahn, M.M., & Stein, J.E. (1998). Fish Injury in the Hylebos Waterway of Commencement Bay, Washington. NOAA Technical Memorandum, NMFS-NWFSC-36, Northwest Fisheries Science Center, National Marine Fisheries Service, Seattle, Washington.

Cortright, R., Weber, J., & Bailey, R. (1987). The Oregon Estuary Plan Book. Oregon Department of Land Conservation and Development, State of Oregon, Salem, Oregon.

Dorcey, A.H.J, Northcote, R.G., & Ward, D.V. (1978). Are the Fraser marshes essential to salmon? Technical Report, Univ. British Columbia Westwater Res. Cent. Tech. Rep. 1, University of British Columbia, Vancouver, B.C., Canada.

Donohoe, R.M., & Curtis, L.R. (1996). Estrogenic activity of chlordecone, *o,p′*-DDT and *o,p′*-DDE in juvenile rainbow trout: Induction of vitellogenesis and interaction with hepatic estrogen binding sites. *Aquatic Toxicology, 36*, 31–52.

Duffy, E.J., Beauchamp, D.A., & Buckley, R.M. (2005). Early marine life history of juvenile Pacific salmon in two regions of Puget Sound. *Estuarine*, *Coastal and Shelf Science, 64*, 94–107.

Easton, M.D.K., Luszniak, D., & Von der Geest, E. (2002). Preliminary examination of contaminant loadings in farmed salmon, wild salmon and commercial salmon feed. *Chemosphere*, *46*, 1053–1074.

Feist, B.D., Steel, E.A., Pess, G.R., & Bilby, R.E. (2003). The influence of scale on salmon habitat restoration priorities. *Animal Conservation, 6*, 271–282.

Foster, E.P, Fitzpatrick, M.S., Feist, G.W., Schreck, C.B., Yates, J., Spitsbergen, J.M., & J.R. Heidel, J.R. (2001a). Plasma androgen correlation, EROD Induction, reduced condition factor, and the occurrence of organochlorine pollutants in reproductively immature white sturgeon (*Acipenser transmontanus*) from the Columbia River, USA. *Archives of Environmental Contamination and Toxicology, 41*, 182–191.

Foster, E.P., Fitzpatrick, M.S., Feist, G.W., Schreck, C.B., & Yates, J. (2001b). Gonad organochlorine concentrations and plasma steroid levels in white sturgeon (*Acipenser transmontanus*) from the Columbia River, USA. *Bulletin of Environmental of Contamination and Toxicology*, *67*, 239–245.

Fresh, K.L., Casillas, E., Johnson, L.L., & Bottom, D.L. (2005). Role of the Estuary in the Recovery of Columbia River Basin Salmon and Steelhead: An Evaluation of the Effects of Selected Factors on Salmonid Population Viability. NOAA Technical Memorandum, NMFS-NWFSC-69, Northwest Fisheries Science Center, NOAA Fisheries, Seattle, Washington.

Gray, A., Simenstad, C.A., D.L. Bottom, D.L., & Cornwell, T.J. (2002). Contrasting functional performance of juvenile salmon habitat in recovering wetlands of the Salmon River Estuary, Oregon, U.S.A. *Restoration Ecology*, *10*, 514–526.

Healey, M.C. (1982). Juvenile Pacific salmon in estuaries: the life support system. In: V.S. Kennedy (ed), *Estuarine Comparisons*. New York: Academic Press pp. 315–341.

Healey, M.C. (1991). Life-history of chinook salmon (*Oncorhynchus tshawytscha*). In: C. Groot and L. Margolis (eds), *Pacific Salmon Life Histories*. Vancouver, British Columbia: UBC Press, pp. 311–393.

Healey, M.C., & Prince, A. (1995). Scales of variation in life history tactics of Pacific salmon and the conservation of phenotype and genotype. *American Fisheries Society Symposium*, *17*, 176–184.

Henny, C.J., Kaiser, J.L., Grove, R.A., Bentley, V.R., & Elliott, J.E. (2003). Biomagnification factors (fish to Osprey eggs from Willamette River, Oregon, U.S.A.) for PCDDs, PCDFs, PCBs and OC pesticides. *Environmental Monitoring and Assessment, 84*, 275–315.

Hites, R.A., Foran, J.A., Carpenter, D.O., Hamilton, M.C., Knuth, B.A., & Schwager, S.J. (2004). Global assessment of organic contaminants in farmed salmon. *Science*, *303*, 226–229.

Jackson, L.J., Carpenter, S.R., Manchester-Neesvig, J., & Stow, C.A. (2001). PCB congeners in Lake Michigan coho (*Oncorhynchus kisutch*) and chinook (*Oncorhynchus tshawytscha*) salmon. *Environmental Science and Technology, 35*, 856–62.

Jacobs, M.N, Covaci, A., & Schepens, P. (2002). Investigation of selected persistent organic pollutants in farmed Atlantic salmon (*Salmo salar*), salmon aquaculture feed, and fish oil components of feed. *Environmental Science and Technology, 36*, 2797–2805.

Johnson, H.E., & Pecor, C. (1969). Coho salmon mortality and DDT in Lake Michigan. *Transactions of the North American Wildlife and Natural Resources Conference*, *34*, 159.

Karl, H., Khulmann, H., & Ruhoff, U. (2003). Transfer of PCDDs and PCDFs into the edible parts of farmed rainbow trout, *Oncorhynchus mykiss* (Walbaum), via feed. *Aquaculture Research*, *34*, 1009–1014.

Khan, A. & Thomas, P. (1998). Estradiol-17 beta and *o,p′*-DDT stimulate gonadotropin release in Atlantic croaker. *Marine Environmental Research, 46*, 149–152.

Krahn, M.M., Moore, L.K., & MacLeod, J.W.D. (1986). Standard Analytical Procedures of the NOAA National Analytical Facility, 1986. Metabolites of Aromatic Compounds in Fish Bile. NOAA Technical Memorandum, NMFS-F/NWC-102, Northwest Fisheries Science Center, NOAA Fisheries, Seattle, Washington.

Kreummel, E., MacDonald, R.W., Kimpe, L.E., Gregory-Eaves, I., Demers, M.J., Smol, J.P., Finney, B., & Blais, J.M. (2003). Aquatic ecology – Delivery of pollutants by spawning salmon. *Nature*, *425*, 255–256.

Lauenstein, G.G., Cantillo A.Y., & Dolvin, S.S. (1993). NOAA National Status and Trends Program Development and Methods. In: Lauenstein, G.G., Cantillo, A.Y., (eds), *Sampling and Analytical Methods of the National Status and Trends Programs National Benthic Surveillance and Mussel Watch Projects 1984–1992,* Vol 1—Overview and Summary Methods. NOAA Technical Memorandum NOS ORCA 71. National Oceanic and Atmospheric Administration, Silver Spring, MD, USA.

LCREP (Lower Columbia River Estuary Partnership): (1999). Lower Columbia River Estuary Program Comprehensive Conservation and Management Plan. Technical Report. LCREP, Portland, Oregon, USA.

Levy, D.A. & Northcote, T.G. (1982). Juvenile salmon residency in a marsh area of the Fraser River Estuary. *Canadian Journal of Fisheries and Aquatic Sciences, 39*, 270–276.

Loge, F.J., Arkoosh, M.R., Ginn, T.R., Johnson, L.L., & Collier, T.K. (2005). Impact of environmental stressors on dynamics of disease transmission. *Environmental Science and Technology, 39*, 7329–7336. .

MacDonald, R.W., & Crecelius, E.A. (1994). Marine sediments in the Strait of Georgia, Juan de Fuca Strait, and Puget Sound: What can they tell us about contamination? *Canadian Technical Report of Fisheries and Aquatic Sciences*, *1948*, 101–134.

Magnusson, A. (2003). Estuarine influence on survival rates of coho (*Oncorhynchus kisutch*) and chinook salmon (*Oncorhynchus tshawytscha*) released from hatcheries on the U. S. Pacific Coast. Estuaries, *26*, 1094–1103.

Malins, D.C., McCain, B.B., Brown, D.W., Sparks, A.K., Hodgins, H.O., & Chan, S.-L. (1982). Chemical contaminants and abnormalities in fish and invertebrates from Puget Sound. NOAA Technical Memorandum OMPA 19. National Oceanic and Atmospheric Administration, Silver Spring, MD, USA.

Manchester-Neesvig, J.B., Valters, K., & Sonzogni, W.C. (2001). Comparison of polybrominated diphenyl ethers (PBDEs) and polychlorinated biphenyls (PCBs) in Lake Michigan salmonids. *Environmental Science and Technology, 35*, 1072–1077.

McCain, B.B., Malins, D.C., Krahn, M.M., Brown, D.W., Gronlund, W.D., Moore, L.K., & Chan. S.-L. (1990). Uptake or aromatic and chlorinated hydrocarbons by juvenile chinook salmon (*Oncorynchus tshawytscha*) in an urban estuary. *Arch Environ Contam Toxicol*, *19*, 10–16.

McMahon, T.E. & Holtby, L.B. (1992). Behavior, habitat use, and movements of coho salmon (*Oncorhynchus kisutch*) smolts during seaward migration. *Canadian Journal of Fisheries and Aquatic Sciences, 49*, 1478–1485.

Meador, J.P., Collier, T.K., & J.E. Stein, J.E. (2002). Use of tissue and sediment-based threshold concentrations of polychlorinated biphenyls (PCBs) to protect juvenile salmonids listed under the US Endangered Species Act. *Aquatic Conservation Marine and Freshwater Ecosystems*, *12*, 493–516.

Meador, J.P., Sommers, F.C., Ylitalo, G.M., & Brown, D.W. (2005). Biological responses in juvenile chinook salmon from dietary exposure to polycyclic aromatic hydrocarbons (PAHs)', in: Proceedings of SETAC 26th Annual Meeting in North America, 'Environmental Science in a Global Society: SETAC's Role in the Next 25 Years', November 13–17, 2005, Baltimore, Maryland, USA. Paper MEA-1117-84663.

Milston, R.H., Fitzpatrick, M.S., Vella, A.T., Clements, S., Gunderson, D., Feist, G., Crippe, T.L., Leong, J., & Schreck, C.B. (2003). Short-term exposure of chinook salmon (*Onchorhynchus tschawytstcha*) to *o,p′*-DDE or DMSO during early life-history stages causes long-term humoral immunosuppression. *Environmental Health Perspectives, 111*, 1601–1607.

Missildine, B.R., Peters, R.J., Chin-Leo, G., & Houck, D. (2005). Polychlorinated biphenyl concentrations in adult chinook salmon (*Oncorhynchus tshawytscha*) returning to coastal and Puget Sound hatcheries of Washington State. *Environmental Science and Technology, 39*, 6944–6951.

Moser, M.L., Olson, A.F., & Quinn, T.P. (1991). Riverine and estuarine migratory behavior of coho salmon (*Oncorhynchus kisutch*) smolts. *Canadian Journal of Fisheries and Aquatic Sciences, 48*, 1670–1678.

Myers, M.M., L.L., & Collier, T.K. (2003). Establishing the causal relationship between polycyclic aromatic hydrocarbon (PAH) exposure and hepatic neoplasms and neoplasia-related liver lesions in English sole (*Pleuronectes vetulus*). *Human and Ecological Risk Assessment, 9*, 67–94.

Nendza, M., Herbst, T., Kussatz, C., & Gies, A. (1997). Potential for secondary poisoning and biomagnification in marine organisms. *Chemosphere*, *35*, 1875–1885.

O'Neill, S.M., West, J.E., & Hoeman, J.C. (1998). Spatial trends in the concentrations of polychlorinated biphenyls (PCBs) in chinook (*Oncorhynchus tshawytscha*) and coho (*Oncorhynchus kisutch*) in Puget Sound and factors affecting PCB accumulation: results from the Puget Sound Ambient Monitoring Program. Puget Sound Research Proceedings, Olympia, WA.

Palm, R.C. Jr., Powell, D.B., Skillman, A., & Godtfredsen, K. (2004). Immunocompetence of juvenile chinook salmon against *Listonella anguillarum* following dietary exposure to polycyclic aromatic hydrocarbons. *Environmental Toxicology and Chemistry, 22*, 2986–2994.

Papoulias, D.M., Villalobos, S.A., Meadows, J., Noltie, D.B., Giesy, J.P., & Tillitt, D.E. (2003). In ovo exposure to o,p′-DDE affects sexual development but not sexual differentiation in Japanese medaka (*Oryzias latipes*). *Environmental Health Perspectives, 111*, 29–32

Parkins, C. (2003). The potential of Polychlorinated Biphenyls contamination of aquaculture products through feed. *Journal of Shellfish Research, 22*, 298–299.

Peterson, R.H. (1976). Temperature selection of Atlantic salmon (*Salmo salar*) as influenced by various toxic substances. *Journal of the Fisheries Research Board of Canada*, *33*, 1722–1730.

Poels, C.L.M., van Der Gaag, M.A., & van de Kerkhoff, J.F.J. (1980). An investigation into the long-term effect of Rhine water on rainbow trout. *Water Research, 14*, 1029–1033.

Reimers, P.E. (1973). The length of residence of juvenile fall chinook salmon in Sixes River. Oregon.Res. Rep. Fish Comm. Oreg, *4*, 3–43.

Rice, C.A. Johnson, L.L., P. Roni, P., Feist, B.E., Hood, W.G., Tear, L.A., Simenstad, C.A., & Williams G.D. (2005). Monitoring rehabilitation in temperate North American estuaries. In: Roni, P., (ed), *Monitoring Stream and Watershed Restoration*. Alpharetta, GA: American Fisheries Society, pp. 165–204.

Roher, T.K., Forney, J.C., & Hartig, J.H. (1982). Organochlorine and heavy metal residues in standard fillets of coho and chinook salmon of the Great Lakes – 1980. *Journal of Great Lakes Research, 8*, 623–634.

Schabetsberger, R., Morgan, C.A., Brodeur, R.D., Potts, C.L., Peterson, W.T., & Emmett, R.L. (2003). Prey selectivity and diel feeding chronology of juvenile chinook (*Oncorhynchus*

tshawytscha) and coho (*O. kisutch*) salmon in the Columbia River plume. *Fisheries Oceanography, 12*, 523–540.

Shreffler, D.K., Simenstead, C.A., & Thom, R.M. (1990). Temporary residence by juvenile salmon in a restored estuarine wetland. *Canadian Journal of Fisheries and Aquatic Sciences, 47*, 2079–2084.

Simenstad, C.A., Fresh, K.L., & Salo E.O. (1982). The role of Puget Sound and Washington coastal estuaries in the life history of Pacific salmon: an unappreciated function. In: V.S. Kennedy (ed), *Estuarine Comparisons*. New York: *Academic Press*, pp. 343–364.

Sloan, C.A., N.G. Adams, N.G., Pearce, R.W., Brown, D.W., & Chan, S-L. (1993). Northwest Fisheries Science Center Organic Analytical Procedures. In: Lauenstein, G.G. and Cantillo, A.Y., (eds.) *Sampling and Analytical Methods of the National Status and Trends Program, National Benthic Surveillance and Mussel Watch Projects 1984–1992*. Volume IV. Comprehensive Descriptions of Trace Organic Analytical Methods. U.S. Dept. Commerce, NOAA Technical. Memorandum. NOS ORCA 71, National Oceanic and Atmospheric Administration, Maryland, USA: Silver Spring, pp. 53–97

Sloan, C.A., Brown, D.W., Pearce, R.W., Boyer, R.H., Bolton, J.L., Burrows, D.G., Herman, D.P., & Krahn, M.M. (2005). Determining aromatic hydrocarbons and chlorinated hydrocarbons in sediments and tissues using accelerated solvent extraction and gas chromatography/mass spectrometry. In: Ostrander, G.K., (ed), *Techniques in Aquatic Toxicology – Volume 2*, Boca Raton, FL: CRC Press.

Spromberg, J.A., & Meador, J.P. (2005). Population-level effects on chinook salmon from chronic toxicity test measurement endpoints. *Integrated Environmental Monitoring and Assessment*, *1*, 9–21.

Stehr, C.M., Brown, D.W., Hom, T., Anulacion, B.F., Reichert, W.L., & Collier, T.K. (2000). Exposure of juvenile chinook and chum salmon to chemical contaminants in the Hylebos Waterway of Commencement Bay, Tacoma, Washington. *Journal of Aquatic Ecosystem Stress and Recovery*, *7*, 215–227.

Stein, J.E., Hom, T., Collier, T.K., Brown, D.W., & Varanasi, U. (1995). Contaminant exposure and biochemical effects in outmigrant juvenile chinook salmon from urban and nonurban estuaries of Puget Sound, WA. *Environmental Toxicology and Chemistry, 14*, 1019–1029.

Telliard, W.A. (1999). EPA analytical methods for the determination of pollutants in the environment. *Critical Reviews in Analytical Chemistry, 29*, 249–257.

TetraTech Inc. (1993). Reconnaissance survey of the Lower Columbia River. *Final Reconnaissance Report* TC-8526-06. Prepared for Lower Columbia River Bi- State Water Quality Program by Tetra Tech, Inc., Redmond, Washington, USA.

TetraTech Inc. (1994). Lower Columbia River backwater reconnaissance survey. *Reconnaissance Report* TC 9405-01 Prepared for Lower Columbia River Bi-State Water Quality Program by Tetra Tech, Inc., Redmond, Washington, USA.

TetraTech Inc. (1996). Lower Columbia River Bi-State Program-The Health of the River, 1990–1996. *Integrated Technical Report* 0253-01, prepared for Oregon Department of Environmental Quality and Washington Department of Ecology by Tetra Tech, Inc., Redmond, Washington, USA.

Thomas, C.M. & Anthony, R.G. (2003). Environmental contaminants in great blue herons (*Ardea herodias*) from the Lower Columbia and Willamette Rivers, Oregon and Washington, USA. *Environmental Toxicology and Chemistry, 18*, 2804–2816.

USEPA (U.S. Environmental Protection Agency). (1997). The Incidence and Severity of Sediment Contamination in Surface Waters of the United States. Volume 1: National Sediment Quality Survey. *EPA Report* No. EPA 823-R-97-006. Washington, D.C.: Office of Science and Technology.

USEPA. (2000). Columbia River Basin Fish Contaminant Survey1996–1998. *EPA Report* No. EPA 910-R-02-006. U.S. Environmental Protection Agency. Region 10. Seattle, Washington 98101.

USFWS (U.S. Fish and Wildlife Service): (1999). Organochlorine contaminants in double-crested cormorants from Lewis and Clark National Wildlife Refuge in the Columbia River Estuary. *Technical Report*, U.S. Fish and Wildlife Service. Portland, OR: Oregon Fish and Wildlife Office.

UWFWS. (2004). Environmental contaminants in aquatic resources from the Columbia River. U.S. Fish and Wildlife Service, *Technical Report*, Portland, Oregon, USA: Oregon Fish and Wildlife Office.

Varanasi, U., Stein, J.E., Reichert, W.L., Tilbury, K.L., Krahn, M.M., & Chan, S.-L. (1992). Chlorinated and aromatic hydrocarbons in bottom sediments, fish and marine mammals in US coastal waters: Laboratory and field studies of metabolism and accumulation. In: Walker, C., and D.R. Livingstone (eds), *Persistent Pollutants in the Marine Environment.* New York, New York, USA: Permagon Press.

Varanasi, U., Casillas, E., Arkoosh, M.R., Hom, T., Misitano, D.A., Brown, D.W., Chan, S.-L,, Collier, T.K., McCain, B.B., & Stein, J.E. (1993). Contaminant exposure and associated biological effects in juvenile chinook salmon (*Oncorhynchus tshawytscha*) from urban and nonurban estuaries of Puget Sound, *NOAA Technical Memorandum*. NMFS-NWFSC-8, Northwest Fisheries Science Center, Seattle, Washington.

Vuorinen, P.J., Paasivirta, J., Keinaenen, M., Koistinen, J., Rantio, T., Hyoetylaeinen, T., & Welling, L. (1997). The M74 syndrome of Baltic salmon (*Salmo salar*) and organochlorine concentrations in the muscle of female salmon. *Chemosphere*, *34*, 1151–1166.

Whyte J.J., Jung R.E., Schmitt C.J., & Tillitt D.E. (2000). Ethoxyresorufin-O-deethylase (EROD) activity in fish as a biomarker of chemical exposure. *Critical Reviews in Toxicology, 30*, 347–570

Williams, D.E., Lech, J.J., & Buhler, D.R. (1998). Xenobiotics and xenoestrogens in fish: modulation of cytochrome P450 and carcinogenesis. *Mutation Research, 399*, 179–92.

Zaroogian, G. Gardner, D., Borsay Horowitz, B., Gutjahr-Gobell, R., Haebler, R., & Mills, L. (2001). Effect of 17beta-estradiol, o,p-DDT, octylphenol and p,p-DDE on gonadal development and liver and kidney pathology in juvenile male summer flounder (*Paralichthys dentatus*). *Aquatic Toxicology, 54*, 101–112.