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Ecotoxicity and risk to human fish consumers of polychlorinated biphenyls in fish near the Hanford Site (USA)

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HIGHLIGHTS

- Polychlorinated biphenyls (PCB) evaluated in Columbia River fish near Hanford Site
- Three groups of PCB congeners quantified (TEQ, non-dioxin-like PCB, total PCB)
- PCBs in fish varied with species, tissue, and location in river
- PCBs below no effect levels in the literature for fish survival, growth, and reproduction
- PCBs exceeded USEPA tissue screening levels for human fish consumers

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ABSTRACT

The purpose of this study was to quantify three groups of polychlorinated biphenyl (PCB) congeners (i.e., dioxin-like toxic equivalents [TEQ], non-dioxin-like PCBs, total PCBs) in fish in several species, tissues, and locations in the Columbia River near the Hanford Site. For TEQ and total PCBs, fish ecotoxicity and risk to human fish consumers were also evaluated. Non-dioxin-like PCBs were not assessed for toxicity, due to lack of available benchmarks. In sturgeon liver, TEQ was significantly higher ($P<0.05$) within the Hanford Site study areas, relative to upriver. However, this same spatial relationship in sturgeon liver did not attain statistical significance for non-dioxin-like PCBs and total PCBs. Non-dioxin-like PCBs and total PCBs were significantly higher ($P<0.05$) in whitefish fillet than in other species (except carp) and significantly higher ($P<0.05$) in carp fillet, relative to bass. All PCB residues in carcass were significantly elevated ($P<0.005$) in comparison to fillet. In addition to PCB source, many factors (e.g., dietary composition, tissue lipid content, fish mobility and home range, age, toxicokinetic processes, seasonal adaptations) influence patterns in PCB bioaccumulation across species, tissues, and locations. TEQ and total PCB residues in liver, fillet, and carcass, observed in this study, were below corresponding no effect residues for TEQ and Aroclors in the literature for fish survival, growth, and reproduction. In contrast, TEQ and total PCBs in fillet in this study exceeded USEPA tissue screening levels for cancer (1E-6 risk) and noncancer (hazard quotient [HQ] = 1) toxicity for human fish consumers. Key uncertainties in these comparisons to assess toxicity relate to variation in fish species sensitivity to PCBs and use of Aroclor data in the literature to represent total PCBs.

1. Introduction

1.1. Hanford Site

The Hanford Site was acquired by the United States (US) federal government in 1943 in order to produce plutonium for some of the nuclear weapons tested and used in World War II (USDOE, 2012). The site is located in southeastern Washington State and includes a section of the Columbia River which provided a source of clean water to cool nine plutonium production reactors. Historic operations at Hanford resulted in the release of both radiological and non-radiological wastes to the environment. Some of these wastes have entered the Columbia River through groundwater, soil, and air pathways.

1.2. Polychlorinated biphenyls (PCBs) in Columbia River fish

PCBs are persistent organic pollutants (POPs) that have been detected in the Columbia River environment on the Hanford Site (Riley et al., 1986). The source of PCBs in the Columbia River system is likely tied to both Hanford and non-Hanford activities and processes. For example, Hanford PCBs were associated with use of electrical devices (e.g., transformers) and road oiling for dust suppression (Hermann, 2007). In contrast, PCBs can be transported from more

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distant environmental reservoirs (e.g., soils) over long distances via successive volatilization/condensation cycles in the global atmospheric circulation (Simonich and Hites, 1995).

PCBs are present as complex mixtures of 209 possible congeners, exhibiting both dioxin-like and non-dioxin-like toxicity to human and ecological receptors. Twelve PCB congeners display dioxin-like toxicity, act via a common mechanism (binding the aryl hydrocarbon receptor [AHR] as an initial step), and are typically expressed in toxic equivalents (TEQ) of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) (Van den Berg et al., 1998, 2006; USEPA, 2008). Toxic effects due to dioxin-like PCB congeners typically occur at relatively lower concentrations than those due to non-dioxin-like PCBs (Giesy and Kannan, 1998).

Dioxin-like PCBs and non-dioxin-like PCBs exert numerous effects on multiple systems. In particular, dioxin-like toxicity includes cancer, as well as noncancer effects, including dermal, immunological, reproductive, endocrine, and developmental toxicity (Van den Berg et al., 1998). Non-dioxin-like PCBs (comprised of the remaining 197 congeners) act through a series of toxicity pathways (independent of the AHR) and also elicit a wide range of effects, including cancer, neurotoxicity, immunotoxicity, and endocrine disruption (Henry and DeVito, 2003; Fernandes et al., 2010).

Extensive literature exists on human toxicity and ecotoxicity, resulting from PCB exposure. For example, human health effects have been compiled by USEPA (1996) and USDHHS (2000), while ecotoxicity has been reviewed by many investigators (e.g., Eisler, 1986; Niimi, 1996; Tillitt, 1999; Henry and DeVito, 2003; Wenning et al., 2011). Recent challenges of PCB research have been enumerated by Hornbuckle and Robertson (2010). For instance, chirality is emerging as an important property in the interaction of PCBs with biological systems (Lehmler et al., 2010).

Contaminants in tissue represent a more integrated and more direct toxicity metric than concentrations in abiotic exposure media (Meador et al., 2011; Sappington et al., 2011). PCBs are lipophilic and readily accumulate in aquatic food chains (Hope, 2008). In particular, several studies have evaluated PCBs in fish tissue in the Columbia River (e.g., USEPA, 2002, 2009). PCB residues in fish present a direct risk to fish, as well as an indirect risk to fish consumers (e.g., humans).

1.3. Purpose

The purpose of this study is to quantify three groups of PCB congeners (i.e., TEQ, non-dioxin-like PCBs, total PCBs) in multiple fish species, tissues, and locations in the Columbia River system near the Hanford Site. For TEQ and total PCBs, tissue-specific residue-effect data from the literature were compiled to assess fish ecotoxicity, while tissue screening levels based on toxicity factors (cancer slope factor [CSF], reference dose [RfD]) and exposure assumptions (e.g., fish consumption rate) were identified to estimate human health risk from consuming fish. Non-dioxin-like PCBs were not evaluated separately for toxicity, given that unique benchmarks for this complete congener group are unavailable and because these congeners are included (to some extent) in total PCB toxicity assessment.

2. Methods

2.1. Study area

The study area includes a section of the Columbia River in southeastern Washington State, based on proximity to the Hanford Site (Fig. 1). This area begins upriver from the Hanford Site above the Wanapum Dam and continues to the McNary Dam, the first dam downriver from the Hanford Site. The study area was divided into four subareas, bounded by river mile (RM) markers. Although non-metric, river miles are shown in Fig. 1, because these are a conventional measure (in the US) of distance upriver from the river mouth. Subareas included Upriver (RM 440-388), 100 Area (RM 387-366), 300 Area (RM 365-340), and Lake Wallula (RM 339–292) (Hulstrom, 2011).

2.2. Fish collection and sample preparation

Six fish species were collected from the study area during 2009–2010, including common carp (*Cyprinus carpio*), mountain whitefish (*Prosopium williamsoni*), walleye (*Stizostedion vitreum*), smallmouth bass (*Micropterus dolomieu*), bridgelip sucker (*Catostomus columbianus*), and white sturgeon (*Acipenser transmontanus*). These species are resident fish and are consumed by the local human population (Hulstrom, 2011). The number of fish collected, including individual and composite samples, is shown for each subarea location (Table S1).

For nonsturgeon species, fish were grouped into composites by species, tissue, and location. Fillets for each composite sample were combined, homogenized in a food grinder, and frozen. Carcass composite samples were processed in a manner similar to fillet composites. Fillet consisted of muscle, skin, and scales, while carcass consisted of bones, head, and fins.

For sturgeon, samples were from individual fish (rather than composites) and grouped by tissue and location. Liver, fillet, and carcass samples were obtained by dissection and frozen. Fillet consisted of muscle, while carcass consisted of bones, head, fins, and skin.

In terms of human fish consumption, definitions of fillet in nonsturgeon vs. sturgeon species are consistent with the recommendation by USEPA (2000) that contaminant concentrations be measured using skin-on fillets for scaled fish species (nonsturgeon fish in this study) and skinless fillets for scaleless fish (sturgeon in this study). Details of fish collection and sample preparation are described elsewhere (Hulstrom and Tiller, 2010).

2.3. PCB congener analysis

PCB congeners were quantified in fish tissues by high resolution gas chromatography/high resolution mass spectrometry (HRGS/HRMS) with USEPA Method 1668A (USEPA, 1999). Three groups of congeners were evaluated, including 12 dioxin-like PCBs, 197 non-dioxin-like PCBs, and 209 total PCBs. This evaluation was performed in the following three steps.

First, dioxin-like PCBs were expressed in terms of TEQ, using toxic equivalency factors (TEFs) for fish (Van den Berg et al., 1998) to assess risk to fish ("fish TEQ") and TEFs for humans (Van den Berg et al., 2006) to evaluate risk to humans consuming fish ("human dietary TEQ"). Because eight of 12 TEFs for dioxin-like PCBs in fish are expressed as "less than values," a minimum and maximum TEQ were computed for fish by setting the TEF equal to zero and the maximum value for these eight TEFs (USEPA, 2008). These two fish TEQ concentrations were denoted as "fish TEQ (minimum)" and "fish TEQ (maximum)," respectively.

Second, means for dioxin-like PCBs (fish TEQ [minimum], fish TEQ [maximum], human dietary TEQ), non-dioxin-like PCBs, and total PCBs were calculated with ProUCL (USEPA, 2012a) or JMP (SAS, 2012) software. Both of these software applications incorporate non-detects with the Kaplan–Meier method (Helsel, 2009). The Kaplan–Meier method is nonparametric and useful in addressing variable reporting limits. In seven cases, fish TEQ (minimum) could not be calculated, because only one distinct data value was detected.

Third, sums were computed for TEQ, non-dioxin-like PCBs, and total PCBs by multiplying means by the corresponding number of congeners in each group (12, 197, and 209, respectively). These sums were used in subsequent statistical analysis and are reported in units of pg/g wet wt fish tissue. Although these results varied by over six orders of magnitude (TEQ vs. total PCBs), a single concentration unit (pg/g) was selected for ease of comparison. For readability, however, TEQ concentrations were expressed in decimal form, while

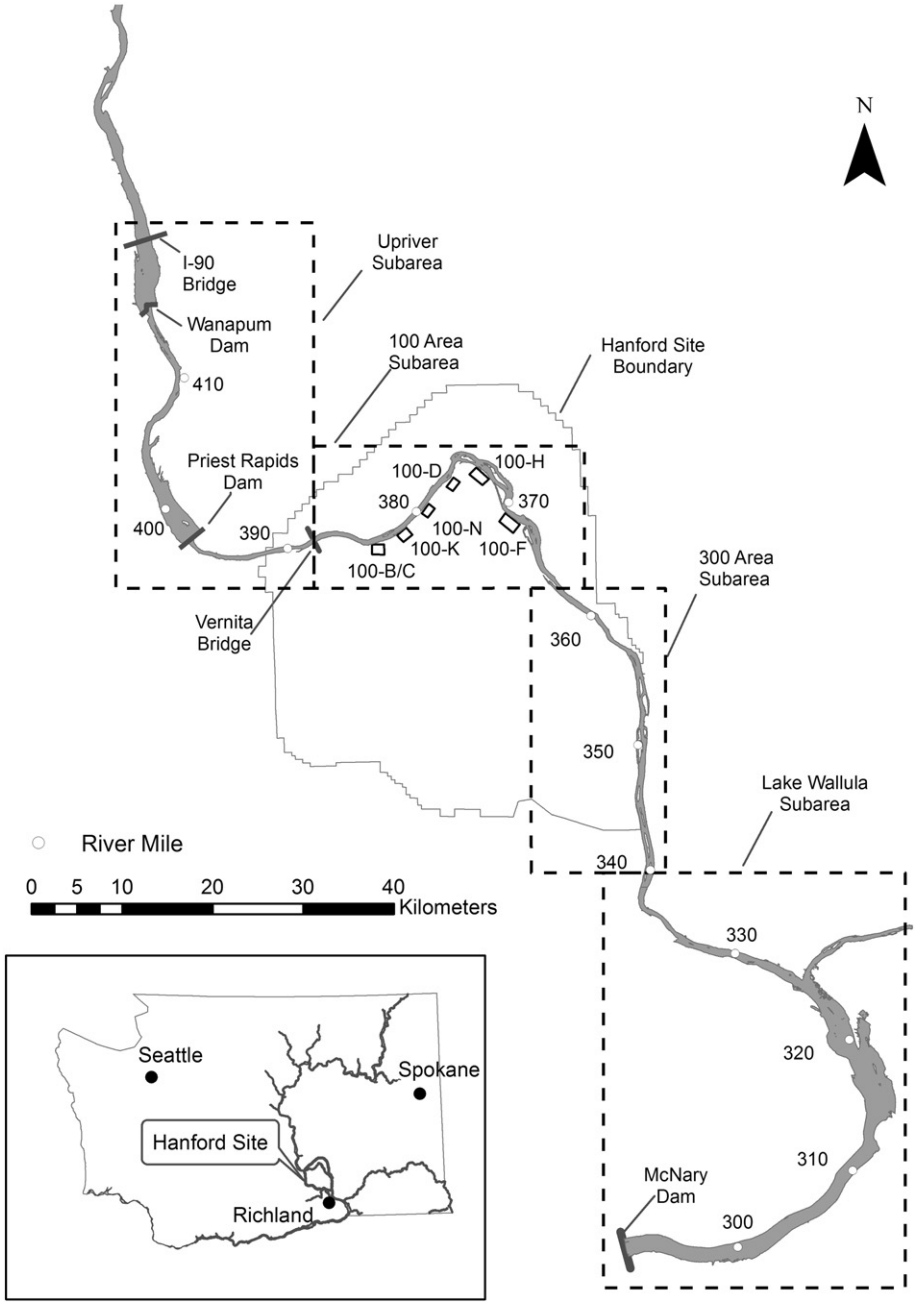

Fig. 1. Columbia River study area near the Hanford Site, divided into four subareas (delineated by dashed rectangles).

non-dioxin-like PCB and total PCB concentrations were expressed in scientific notation.

2.4. Statistical analysis

Descriptive statistics were determined for total length and total weight of each fish species over all locations (Table S2). These data characterize average size and variability in fish species examined in this study. Coefficients of variation (standard deviation/mean) for total length and total weight were below one, indicating relatively low variability in these distributions (Richardson et al., 2011).

Analysis of variance (ANOVA), least significant difference (LSD) tests, and independent *t* tests were performed to evaluate the differences in PCBs among fish species, tissues, and locations. First, a series of single factor ANOVA tests was performed for sturgeon liver to evaluate the effect of location on fish TEQ (minimum and maximum), non-dioxin-like PCBs, and total PCBs. Second, a series of two factor ANOVAs was performed for fish fillet and carcass to evaluate the main effects and interactions of species and location on fish TEQ (minimum and maximum), human dietary TEQ (fillet only), non-dioxin-like PCBs, and total PCBs. Third, LSD tests were applied as a post hoc analysis when ANOVA revealed a significant effect. Fourth, independent *t* tests were used to compare fish TEQ (minimum and maximum), non-dioxin-like PCBs, and total PCBs in fillet vs. carcass. Independent t tests were employed, due to the inclusion of composite (non-paired) samples. An alpha level of 0.05 was considered statistically significant in all tests. Calculations were performed with Statgraphics software (SPTI, 2011).

2.5. Ecotoxicity and human risk assessment

Ecotoxicity and risk were assessed for fish and human receptors, respectively. Typically, toxicity due to PCB exposure is assessed,

based on TEQ and total PCB concentrations (Field, 1998; Giesy and Kannan, 1998). In this analysis, TEQ corresponds to CAS registry number 1746-01-6 (2,3,7,8-TCDD), and total PCBs correspond to CAS registry number 1336-36-3, unless otherwise noted. In particular, Aroclors were used as a surrogate for total PCBs in the absence of total PCB data in the literature. Aroclors are commercial mixtures of PCBs, whose congener composition differs in number and degree of chlorination among specific Aroclor formulations (USDHHS, 2000).

For ecotoxicity assessment of PCBs, tissue residues (liver, muscle, carcass) for TEQ and Aroclors in fish were compiled from two tissue residue databases (Jarvinen and Ankley, 1999; USACE, 2011). Consistent with Jarvinen and Ankley (1999), the effects were limited to survival, growth, and reproduction. These endpoints can be related to population level effects which are ecologically relevant (Suter, 1993).

For human risk assessment of PCBs, resulting from fish consumption, fish tissue screening levels for TEQ and total PCBs were derived by USEPA (2012b). Tissue screening levels were identified for both cancer and noncancer endpoints. Noncancer toxicity for total PCBs is based on Aroclor 1254, because there is no oral RfD established for total PCBs, and bioaccumulative PCBs in fish resemble higher chlorinated mixtures such as Aroclor 1254 (Harris and Jones, 2008). It is assumed that tissue screening levels pertain to fillet, since USEPA (2000) recommends using fillets as the standard sample type for analyzing chemical contaminants for use in fish advisories (in the absence of specific data on fish preparation methods).

3. Results

3.1. Species, tissues, and locations

3.1.1. Sturgeon liver

TEQ data in sturgeon liver are presented in Table 1. Fish TEQ (minimum) did not differ significantly across locations, although data from the Upriver subarea were insufficient ($n<3$) for inclusion in the analysis. In contrast, fish TEQ (maximum) was significantly higher ($P<0.05$) in 100 and 300 Area subareas, relative to Upriver and Lake Wallula locations (Fig. 1).

Non-dioxin-like PCB and total PCB data in sturgeon liver are also shown in Table 1. Both non-dioxin-like PCBs and total PCBs were significantly higher ($P<0.05$) in 100 and 300 Area subareas, relative to Lake Wallula. In general, non-dioxin-like PCB and total PCB results in sturgeon liver mirrored TEQ in terms of relative distribution across locations.

3.1.2. All fish species fillet and carcass

TEQ data for fillet in all fish species are presented in Table 2. No significant differences ($P>0.05$) were observed in TEQ for species, location, or their interaction. Non-dioxin-like PCB and total PCB data for fillet in all fish species are also shown in Table 2. Non-dioxin-like PCBs and total PCBs were significantly higher ($P<0.05$) in whitefish (relative to other species, except carp) and significantly higher ($P<0.05$) in carp (relative to bass). No significant effects were observed in non-dioxin-like PCBs and total PCBs for location or the interaction of species by location.

TEQ data for carcass in all fish species are presented in Table 3, along with non-dioxin-like PCBs and total PCBs. No significant differences were observed in any of these PCB results in carcass for species, location, or their interaction. TEQ, non-dioxin-like PCB, and total PCB concentrations in fillet and carcass tissues are presented in Table 4. All PCB measures were significantly higher ($P<0.005$) in carcass, relative to fillet.

3.2. Ecotoxicity and human risk

3.2.1. Fish ecotoxicity

Selected TEQ and Aroclor residues in fish liver, muscle, and carcass with associated effects in survival, growth, and reproduction, as reported in the literature, are listed in Table 5. Tissue residues for TEQ and total PCBs in this study (included for comparison in Table 5) fall below TEQ and Aroclor residues in the literature, respectively, for corresponding tissues for both effect and no effect data. Regarding total PCB residues, however, it should be noted that literature values are Aroclor data (not total PCBs per se).

3.2.2. Risk to human fish consumers

Fish tissue screening levels for TEQ and total PCBs, along with toxicity factors and exposure assumptions, are listed in Table 6. These tissue screening levels, derived by USEPA (2012b), represent concentrations in fish fillet, associated with a 1E-6 cancer risk and a threshold for noncancer toxicity (hazard quotient [HQ] = 1). Both TEQ and total PCB fillet concentrations, observed in this study, exceed corresponding tissue screening levels, indicating risk to human fish consumers above USEPA criteria (USEPA, 2012b). Similar to fish ecotoxicity data for total PCBs (Table 5), however, it should be noted that the resulting tissue screening level for noncancer toxicity (HQ = 1) for total PCBs corresponds to Aroclor 1254 (rather than total PCBs per se).

4. Discussion

4.1. Species, tissues, and locations

Several observations in PCB residues were common to all species, tissues, and locations examined in this study (Tables 1–4). The difference between fish TEQ (minimum) vs. fish TEQ (maximum) is relatively minor, since the difference in fish TEFs for the bounding conditions is relatively small (0 vs. 0.000005) (Van den Berg et al., 1998). In addition, it can be seen that human dietary TEQ (typically calculated for fillet) is greater than fish TEQ, owing to generally larger TEFs in humans vs. fish (Van den Berg et al., 1998, 2006). More dramatically, it is apparent that all TEQ concentrations are a small portion of corresponding non-dioxin-like PCB or total PCB concentrations, due to the smaller number of congeners and fractional TEF values that are

Table 1
Polychlorinated biphenyl (PCB) concentrations (pg/g wet wt) in sturgeon liver.

Location	Fish TEQ (minimum)[a]				Fish TEQ (maximum)				Non-dioxin-like PCBs				Total PCBs			
	n[b]	Mean	SE[c]	P[d]	n	Mean	SE	P	n	Mean	SE	P	n	Mean	SE	P
Upriver	ID[e]			0.071	4	0.30^{A}	0.30	0.0099	4	2.8E5^{AB}	1.5E5	0.044	4	2.9E5^{AB}	1.6E5	0.041
100 Area	8	1.2	0.19		8	1.4^{B}	0.21		8	6.4E5^{B}	1.1E5		8	6.8E5^{B}	1.1E5	
300 Area	9	0.92	0.18		9	1.1^{B}	0.20		9	5.5E5^{B}	1.0E5		9	5.8E5^{B}	1.1E5	
Lake Wallula	5	0.40	0.24		6	0.40^{A}	0.24		6	1.9E5^{A}	1.3E5		6	2.0E5^{A}	1.3E5	

[a] TEQ = toxic equivalent.
[b] All sturgeon livers were not analyzed for PCBs (see Table S1). In addition, fish TEQ (minimum) could not be quantified for sturgeon liver samples in four cases.
[c] SE = standard error.
[d] P value for a single factor (location) ANOVA for each dependent variable. If $P<0.05$, then a post hoc analysis was performed. Mean values with different uppercase letter superscripts across location differ significantly by LSD test ($P<0.05$).
[e] ID = insufficient data ($n<3$) for statistical analysis.

Table 2
Polychlorinated biphenyl (PCB) concentrations (pg/g wet wt) in fish fillet.

Factor	Level	Fish TEQ (minimum)[a]				Fish TEQ (maximum)				Human dietary TEQ				Non-dioxin-like PCBs				Total PCBs			
		n[b]	Mean	SE[c]	P[d]	n	Mean	SE	P	n	Mean	SE	P	n	Mean	SE	P	n	Mean	SE	P
Species	Bass	20	0.14	0.20	0.10	20	0.17	0.24	0.087	20	3.0	4.2	0.088	20	6.7E4^{A}	6.6E4	0.024	20	7.3E4^{A}	7.4E4	0.028
	Carp	19	0.60	0.21		19	0.69	0.25		19	13	4.3		19	2.8E5^{BC}	6.8E4		19	3.0E5^{BC}	7.6E4	
	Sturgeon	29	0.30	0.18		30	0.33	0.20		30	6.1	3.6		30	1.6E5^{AB}	5.6E4		30	1.7E5^{AB}	6.3E4	
	Sucker	20	0.26	0.20		20	0.30	0.24		20	5.3	4.2		20	1.3E5^{AB}	6.6E4		20	1.4E5^{AB}	7.4E4	
	Walleye	19	0.29	0.21		20	0.33	0.24		20	6.0	4.2		20	1.4E5^{AB}	6.6E4		20	1.4E5^{AB}	7.4E4	
	Whitefish	20	0.88	0.20		20	1.1	0.24		20	19	4.2		20	3.6E5^{C}	6.6E4		20	4.0E5^{C}	7.4E4	
Location	Upriver	28	0.60	0.17	0.46	29	0.71	0.20	0.46	29	12	3.5	0.46	29	2.5E5	5.5E4	0.36	29	2.7E5	6.2E4	0.38
	100 Area	34	0.46	0.16		34	0.54	0.19		34	9.9	3.3		34	2.1E5	5.2E4		34	2.3E5	5.8E4	
	300 Area	35	0.35	0.16		35	0.42	0.19		35	7.5	3.3		35	1.8E5	5.2E4		35	1.9E5	5.8E4	
	Lake Wallula	30	0.23	0.16		31	0.27	0.19		31	4.9	3.4		31	1.2E5	5.3E4		31	1.3E5	6.0E4	

[a] TEQ = toxic equivalent.
[b] Fish TEQ (minimum) could not be quantified for fillet in two cases (one walleye and one sturgeon).
[c] SE = standard error.
[d] P value for each factor (species and location) in a two factor ANOVA for each dependent variable. If $P<0.05$, then a post hoc analysis was performed. Mean values with different uppercase letter superscripts (within a factor) differ significantly by LSD test ($P<0.05$). The two way interaction (species by location) was not significant for each dependent variable.

used to calculate TEQ. Consequently, non-dioxin-like PCBs dominate total PCB residues in fish tissue on a mass basis.

4.1.1. Sturgeon liver

Although higher levels of fish TEQ in sturgeon liver in 100 and 300 Area subareas (relative to Upriver and Lake Wallula locations) (Table 1) may be linked to the Hanford Site (Fig. 1), this relationship is uncertain, due in part to mobility of sturgeon. With respect to the Upriver subarea, however, all sturgeon were collected upriver of Wanapum Dam (Hulstrom and Tiller, 2010), restricting their mobility downriver (Warren and Beckman, 1993). Alternatively, non-Hanford sources of PCBs also contribute to sturgeon liver TEQ in the 100 and 300 Area subareas.

Non-dioxin-like PCB and total PCB residues in sturgeon liver exhibit a spatial profile similar to TEQ residues (Table 1). Although the pattern of non-dioxin-like PCBs and total PCBs in sturgeon liver was similar to TEQ in terms of location, statistical significance was not attained for non-dioxin-like PCBs and total PCBs between Upriver vs. 100 and 300 Area subareas, weakening a potential Hanford link.

4.1.2. All fish species fillet and carcass

Although significant differences were not observed in TEQ in fillet across species, non-dioxin like PCBs and total PCBs exhibited significant species differences (Table 2). PCB differences across species may relate to variation in dietary composition (Johnson et al., 2007), lipid content of fillet (Mendez et al., 1996), mobility and home range (Monosson et al., 2003), or fish age and seasonal adaptations (Volta et al., 2009). All of these factors can influence PCB bioaccumulation.

Differences in PCB residues in carcass across species and locations failed to reach statistical significance (Table 3). Relative to fillet, however, corresponding PCB concentrations in carcass were significantly elevated (Table 4). This reflects toxicokinetic processes influencing PCB disposition in tissues (Lehman-McKeeman, 2008). In particular, carcass included fish heads which likely increased lipid content (Narayan et al., 2012), resulting in higher PCB residues in carcass vs. fillet. Because fillet and carcass were defined slightly differently for sturgeon vs. nonsturgeon fish species, the comparison of PCBs in fillet vs. carcass was repeated with sturgeon data removed. Essentially, the same result was achieved for PCB residues (carcass > fillet, $P<0.05$), demonstrating that the slight difference in tissue definitions between sturgeon and nonsturgeon species had little effect.

4.2. Ecotoxicity and human risk

One observation with PCB fish residues in the literature was common to both ecotoxicity and human risk. That is, adverse effects due to TEQ occur at much lower concentrations than those due to total PCBs (or Aroclors as a surrogate) (Tables 5 and 6). This observation is also consistent with the comparison of toxicity for TEQ vs. non-dioxin-like PCBs (Giesy and Kannan, 1998).

4.2.1. Fish ecotoxicity

Fish tissue residues of TEQ and total PCBs in this study were below concentrations, reported in the literature, associated with reduced survival, growth, and reproduction (Table 5). However, differences in total PCB vs. Aroclor, as well as variation in species sensitivity,

Table 3
Polychlorinated biphenyl (PCB) concentrations (pg/g wet wt) in fish carcass.

Factor	Level	Fish TEQ (minimum)[a]				Fish TEQ (maximum)				Non-dioxin-like PCBs				Total PCBs			
		n[b]	Mean	SE[c]	P[d]	n	Mean	SE	P	n	Mean	SE	P	n	Mean	SE	P
Species	Bass	20	0.68	0.29	0.42	20	0.80	0.35	0.35	20	3.0E5	1.0E5	0.22	20	3.2E5	1.1E5	0.20
	Carp	19	1.1	0.30		19	1.2	0.36		19	4.2E5	1.0E5		19	4.5E5	1.2E5	
	Sturgeon	29	0.68	0.25		30	0.77	0.30		30	3.5E5	8.6E4		30	3.7E5	9.6E4	
	Sucker	20	0.58	0.29		20	0.68	0.35		20	2.8E5	1.0E5		20	3.0E5	1.1E5	
	Walleye	20	0.61	0.29		20	0.71	0.35		20	2.9E5	1.0E5		20	3.1E5	1.1E5	
	Whitefish	20	1.3	0.29		20	1.6	0.35		20	6.0E5	1.0E5		20	6.6E5	1.1E5	
Location	Upriver	29	0.98	0.24	0.69	29	1.2	0.29	0.65	29	4.4E5	8.4E4	0.34	29	4.8E5	9.4E4	0.37
	100 Area	34	0.91	0.23		34	1.1	0.28		34	4.1E5	7.9E4		34	4.4E5	8.9E4	
	300 Area	34	0.75	0.23		35	0.88	0.27		35	4.0E5	7.9E4		35	4.3E5	8.9E4	
	Lake Wallula	31	0.62	0.23		31	0.71	0.28		31	2.5E5	8.1E4		31	2.6E5	9.1E4	

[a] TEQ = toxic equivalent.
[b] Fish TEQ (minimum) could not be quantified for carcass in one case (one sturgeon).
[c] SE = standard error.
[d] P value for each factor (species and location) in a two factor ANOVA for each dependent variable. Because $P>0.05$, post hoc analysis was not performed. In addition, the two way interaction (species by location) was not significant for each dependent variable.

Table 4
Polychlorinated biphenyl (PCB) concentrations (pg/g wet wt) in fillet vs. carcass for all fish species and locations.

Tissue	Fish TEQ (minimum)[a]			Fish TEQ (maximum)			Non-dioxin-like PCBs			Total PCBs		
	n[b]	Mean[c]	SE[d]	n	Mean	SE	n	Mean	SE	n	Mean	SE
Fillet	127	0.41[A]	0.08	129	0.48[A]	0.10	129	1.9E5[A]	2.7E4	129	2.0E5[A]	3.0E4
Carcass	128	0.82[B]	0.11	129	0.96[B]	0.14	129	3.8E5[B]	4.0E4	129	4.0E5[B]	4.5E4

[a] TEQ = toxic equivalent.
[b] Fish TEQ (minimum) could not be quantified for fillet in two cases (one walleye and one sturgeon) nor for carcass in one case (one sturgeon).
[c] Mean values with different uppercase letter superscripts (between tissues) differ significantly by independent *t* test ($P<0.005$).
[d] SE = standard error.

introduce uncertainty into this comparison. This may be especially relevant when comparative residues exhibit similar concentrations. For example, the high end of the range for total PCB residues in sturgeon liver in this study (6.8E5 pg/g) is relatively similar to the corresponding Aroclor 1254 residue in coho salmon liver for reduced survival (1.05E6 pg/g) (Table 5). In addition, lack of ecotoxicity benchmarks for non-dioxin-like PCBs represents a notable data gap. Finally, other environmental factors (e.g., exposure from additional contaminants, dams impeding fish mobility) may interact with PCB effects to adversely impact fish in the Columbia River system (USEPA, 2002, 2009).

4.2.2. Risk to human fish consumers

Fish tissue residues of TEQ and total PCBs in this study exceeded corresponding tissue screening levels for human fish consumers, derived by USEPA (2012b) (Table 6). In particular, tissue screening levels for cancer risk were exceeded by a larger margin than tissue screening levels for noncancer toxicity. Similar to ecotoxicity tissue residues for total PCBs, however, the tissue screening level for human noncancer toxicity for total PCBs was also based on Aroclor toxicity (Aroclor 1254). This surrogate approach introduces uncertainty into this evaluation, along with other uncertainties in toxicity and exposure factors. In addition, tissue screening levels for non-dioxin-like PCBs for human fish consumers are unavailable, despite adverse effects reported for these congeners (e.g., Mukerjee, 1997, 1998; Giesy and Kannan, 1998; Fernandes et al., 2010). Given the screening nature of this analysis and attendant uncertainties, these human risk results suggest that further study be performed to reduce uncertainty and more fully characterize risk to human fish consumers in the Columbia River system.

5. Conclusions

TEQ residues in sturgeon liver in study areas within the Hanford Site were significantly higher ($P<0.05$), compared to upriver. Although non-dioxin-like PCBs and total PCBs displayed this same pattern, these congener groups did not attain statistical significance with this comparison. Non-dioxin-like PCBs and total PCBs were significantly higher ($P<0.05$) in whitefish fillet than in other species (except carp) and significantly higher ($P<0.05$) in carp fillet, relative to bass. All PCB residues in carcass were significantly higher ($P<0.005$) than residues in fillet. This variation in PCB residues in fish species, tissues, and locations relates to spatial and temporal components of PCB sources, seasonal effects, as well as specific fish characteristics (e.g., dietary

Table 5
Selected tissue residues (pg/g wet wt) and effects in fish for toxic equivalent (TEQ) and Aroclor data in the literature, along with corresponding tissue residues (pg/g wet wt) in this study.

TEQ or Aroclor[a]	Tissue	Tissue residue	Effect[b]	Fish species	Reference[c]	Tissue residue in this study[d]
TEQ	Liver	72	No effect S, G	Rainbow trout	Kleeman et al. (1986a)	0.30–1.4
TEQ	Liver	270	No effect S	Lake trout	Walker et al. (1994)	
TEQ	Liver	466	No effect S, G	Yellow perch	Kleeman et al. (1986b)	
TEQ	Liver	1500	No effect S, G	Rainbow trout	Parrot et al. (1995)	
TEQ	Liver	3710	Reduced G	Rainbow trout	Branson et al. (1985)	
TEQ	Muscle	9	No effect S, G	Yellow perch	Kleeman et al. (1986b)	0.14–1.1
TEQ	Muscle	29	No effect S, G	Rainbow trout	Kleeman et al. (1986a)	
TEQ	Muscle	260	Reduced S, G	Rainbow trout	Branson et al. (1985)	
TEQ	Muscle	530	No effect S	Lake trout	Walker et al. (1994)	
TEQ	Carcass	129	No effect S, G	Yellow perch	Kleeman et al. (1986b)	0.58–1.6
TEQ	Carcass	315	No effect S, G	Rainbow trout	Kleeman et al. (1986a)	
Aroclor 1254	Liver	1.05E6	Reduced S	Coho salmon	Folmar et al. (1982)	2.0E5–6.8E5
Aroclor 1254	Liver	2.3E6	No effect S, G	Rainbow trout	Lieb et al. (1974)	
Aroclor 1254	Liver	6.03E6	No effect R	Atlantic cod	Sangalang et al. (1981)	
Aroclor 1254	Liver	7.88E6	Reduced R	Atlantic cod	Sangalang et al. (1981)	
Aroclor 1254	Liver	1.01E8	No effect S	Atlantic cod	Sangalang et al. (1981)	
Aroclor 1254	Liver	1.56E8	Reduced S	Atlantic cod	Sangalang et al. (1981)	
Aroclor 1254	Muscle	2.8E6	No effect S, G	Rainbow trout	Lieb et al. (1974)	7.3E4–4.0E5
Aroclor 1254	Muscle	8.36E6	No effect G	Arctic char	Maule et al. (2005)	
Aroclor 1016	Muscle	3.0E7	Reduced S	Pinfish	Hansen et al. (1974)	
Aroclor 1254	Muscle	3.28E7	Reduced R	Brook trout	Freeman and Idler (1975)	
Aroclor 1254	Muscle	3.9E7	No effect G	Brook trout	Addison et al. (1978)	
Aroclor 1242	Carcass	2.3E6	No effect S	Catfish	Hansen et al. (1976)	3.0E5–6.6E5
Aroclors 1242/1254	Carcass	1.3E7	No effect G	Coho salmon	Leatherland et al. (1979)	
Aroclors 1242/1254	Carcass	4.03E7	No effect S	Rainbow trout	Leatherland and Sonstegard (1980)	
Aroclors 1242/1254	Carcass	4.3E7	Reduced G	Coho salmon	Leatherland et al. (1979)	

[a] Because fish tissue residues for total PCBs were unavailable in a literature search, Aroclor data are presented as a surrogate for total PCBs.
[b] S = survival, G = growth, R = reproduction.
[c] These tissue residue data are reported in Jarvinen and Ankley (1999) or USACE (2011).
[d] Range of mean concentrations in corresponding tissue for fish TEQ and total PCB (see Tables 1–4). Muscle is assumed to be comparable with fillet.

Table 6
Fish tissue screening level (pg/g wet wt) for toxic equivalent (TEQ) and total polychlorinated biphenyl (total PCB) for cancer (1E-6 risk) and noncancer (hazard quotient [HQ] = 1) toxicity in humans from fish ingestion, along with fillet residue (pg/g wet wt) in this study.

TEQ or total PCB	Oral CSF[a] (mg/kg-d)$^{-1}$	Oral RfD[b] (mg/kg-d)	Tissue screening level[c]		Reference	Fillet residue in this study[d]
			1E-6 risk	HQ = 1		
TEQ	1.3E5	7E-10	0.024	0.95	USEPA (2012b)	3.0–19
Total PCB	2.0E0	2E-5	1.6E3	2.7E4	USEPA (2012b)	7.3E4–4.0E5

[a] Oral CSF = oral cancer slope factor. Oral CSF for TEQ corresponds to 2,3,7,8-TCDD. Oral CSF for total PCB corresponds to PCB (high risk).
[b] Oral RfD = oral reference dose. Oral RfD for TEQ corresponds to 2,3,7,8-TCDD. Oral RfD for total PCB corresponds to Aroclor 1254 (Harris and Jones, 2008).
[c] Exposure assumptions used to derive tissue screening levels include fish consumption rate (FCR) = 54 g/d, body weight (BW) = 70 kg, averaging time (AT) = 365 d/y, exposure frequency (EF) = 350 d/y, exposure duration (ED) = 30 y, and lifetime (LT) = 70 y. Tissue screening levels are assumed to refer to fillet (USEPA, 2000).
[d] Range of mean fillet concentration for human dietary TEQ or total PCB (see Tables 2 and 4).

composition, tissue lipid content, toxicokinetics, mobility and home range, age).

Fish tissue residues in this study were below corresponding no effect residues for TEQ and Aroclors, reported in the literature, in liver, muscle, and carcass for fish survival, growth, and reproduction endpoints. Due to lack of residue-effect data for total PCBs in fish in the literature, Aroclor residues served as a surrogate for comparison. In contrast to fish ecotoxicity results, TEQ and total PCB residues in fillet in this study exceeded USEPA tissue screening levels for human fish consumers for both cancer (1E-6 risk) and noncancer (HQ = 1) toxicity endpoints. Again, Aroclor data served as a surrogate to assess noncancer toxicity for total PCBs.

In addition to the use of Aroclors as a surrogate for total PCBs, other sources of uncertainty impact the assessment of fish ecotoxicity and risk to human fish consumers. In particular, variation in species sensitivity to PCBs introduces uncertainty into the evaluation of fish ecotoxicity. Furthermore, definitions of fillet and carcass in this study may differ somewhat from definitions in the literature. With respect to human risk, toxicity factors (CSF, RfD), exposure assumptions (e.g., fish consumption rate), as well as risk and hazard targets (1E-6 cancer risk, noncancer HQ = 1) influence tissue screening levels for human fish consumers. Finally, non-dioxin-like PCBs could not be assessed for toxicity, due to lack of published benchmarks for this PCB congener group. Nonetheless, it is hoped that this analysis of PCB residues in fish in the Columbia River will effectively contribute to ongoing risk assessment activities at the Hanford Site.

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