SETAC PRESS

Environmental Toxicology and Chemistry, Vol. 18, No. 12, pp. 2804–2816, 1999
Printed in the USA
0730-7268/99 $9.00 + .00

ENVIRONMENTAL CONTAMINANTS IN GREAT BLUE HERONS (*ARDEA HERODIAS*) FROM THE LOWER COLUMBIA AND WILLAMETTE RIVERS, OREGON AND WASHINGTON, USA

CARMEN M. THOMAS*† and ROBERT G. ANTHONY‡
†U.S. Fish and Wildlife Service, 2800 Cottage Way, Room W2605, Sacramento, California 95825
‡Oregon State University, Department of Fisheries and Wildlife, 104 Nash Hall Corvallis, Oregon 97330, USA

(*Received* 25 *June* 1998; *Accepted* 21 *December* 1998)

Abstract—Great blue heron (*Ardea herodias*) eggs and prey items were collected from six colonies in Oregon and Washington, USA, during 1994 to 1995. Contaminant concentrations, reproductive success, and biomagnification factors were determined and effects of residue levels were measured by H4IIE rat hepatoma bioassays. Mean residue concentrations in heron eggs and prey items were generally low. However, elevated concentrations of polychlorinated biphenyls (PCBs) were detected in eggs and prey from Ross Island on the Willamette River. Biomagnification factors varied among sites. Sites were not significantly different in H4IIE tetrachlorodibenzo-*p*-dioxin equivalents (TCDD-EQs), although the TCDD-EQ for Karlson Island was 9 to 20 times greater than that of any other site. Large differences existed between toxic equivalents calculated from egg residue concentrations and TCDD-EQs, which indicated nonadditive interactions among the compounds. Tetrachlorodibenzo-*p*-dioxin equivalents and nest failure were positively correlated with TCDD concentration. Fledging and reproductive rates were similar to those determined for healthy heron populations, however, indicating that any adverse effects were occurring at the individual level and not at the colony level. Our results support the use of great blue herons as a biomonitor for contamination in aquatic ecosystems. Their relatively low sensitivity to organochlorine contaminants and high trophic position allows contaminant accumulation and biomagnification without immediate adverse effects that are often seen in other, more sensitive species.

Keywords—Contaminants Herons Oregon Washington Pesticides

INTRODUCTION

The Columbia and Willamette Rivers provide essential breeding and wintering habitat for migratory birds, anadromous fish, and threatened and endangered species. Both rivers are exposed to a variety of contaminant sources, including municipal and industrial discharges, urban and industrial nonpoint pollution, accidental spills of oil and hazardous materials, and agricultural runoff, all of which may affect the viability of fish and wildlife populations.

Several contaminants, including dichlorodiphenylethylene (DDE), polychlorinated biphenyls (PCBs), and polychlorinated dibenzo-*p*-dioxins (PCDDs), have been found at potentially hazardous concentrations in fish and wildlife from both rivers. Bald eagle productivity along the Columbia River since 1984 has been markedly lower than productivity of pairs elsewhere in Oregon and Washington and has been associated with elevated concentrations of DDE and PCBs in eggs and carcasses [1]. Dichlorodiphenylethylene and PCB residues were detected in all life stages of Columbia River bald eagles in the 1980s [1], which strongly implicated the river as a primary source of these contaminants. Polychlorinated dibenzo-*p*-dioxin concentrations in unhatched bald eagle eggs collected from the Columbia River Estuary [1] were similar to concentrations found in fish-eating birds from Michigan that have had poor reproductive success [2]. The PCDD concentrations in 1994 eagle eggs exceeded the no-effects threshold calculated by Giesy et al. [3], suggesting that dioxins are also contributing to the reduced reproductive success [4].

* To whom correspondence may be addressed (carmen_thomas@fws.gov).

Recent studies by the U.S. Environmental Protection Agency (U.S. EPA) [5] found tetrachlorodibenzo-*p*-dioxin (TCDD) residues in some fish that exceeded guidelines for the protection of human health and resulted in the issuance of a total maximum daily load for dioxin in the Columbia and Willamette Rivers. Fish collected from the Portland Harbor of the Willamette River exhibited strongly induced cytochrome P4501A1, which is indicative of exposure to TCDD [6]. Researchers also found that the organochlorine burdens in Willamette River fish were not acutely toxic but could not determine whether any chronic impacts such as reproductive dysfunction were occurring.

The purpose of this study was to determine whether great blue herons would serve as a good monitoring species for contaminants in piscivorous birds from the Columbia and Willamette River basins. An indicator species should have a wide distribution, high trophic status, nest fidelity [7], and low sensitivity to contaminants [8]. Great blue herons on the lower Columbia and Willamette Rivers meet all four criteria [9].

We tested three hypotheses with this study. First, that environmental contaminants are elevated in great blue herons from the lower Columbia and Willamette Rivers. Second, concentrations of TCDD in heron eggs are related to the distance of the colony from the nearest chlorine-bleaching pulp and paper mill. Third, concentrations of certain contaminants such as DDE, PCBs, and TCDD are adversely affecting heron reproductive success.

METHODS

Sample collection

Study sites (heron colonies) were located in northwest Oregon and Washington, USA (Fig. 1). We selected colonies

Fig. 1. Great blue heron colony locations and distances (miles) from nearest pulp and paper mill (+ if colony is upstream from mill and — if colony is downstream from mill), 1994 to 1995.

based on the following criteria: colony size, proximity to pulp and paper mills, and accessibility. The minimum colony size for consideration was 50 nests. The Bachelor and Fisher Island colonies were located on the lower Columbia River and Karlson Island was in the Columbia River Estuary. The Molalla State Park and Ross Island colonies were located on the lower Willamette River, and Samish Island was in Puget Sound. Karlson and Samish Islands were in transition zones between fresh and saltwater, and the remaining sites were on or near fresh water. We designated Samish Island as our reference site due to a previous study that detected low concentrations of most organochlorines (OCs) in heron eggs from that colony [10,11]. With the exception of the Molalla site, all sites were in tidal areas. Ross Island is within the city limits of Portland, Oregon.

From late March until the end of April in 1994 and 1995, professional tree climbers randomly collected five eggs per colony. All nests from which an egg was collected contained ≥2 eggs. Egg tissues were placed in chemically cleaned glassware and frozen at −20°C [12]. An aliquot from each egg was sent to the Midwest Science Center (U.S. Geological Survey, Columbia, MO) for use in the H4IIE rat hepatoma bioassay. Prey items were collected from colony floors throughout the breeding season. Prey were composited by species and wrapped in aluminum foil. All samples were shipped on dry ice to a laboratory for residue analysis.

Lab methods

Eggshell processing and measurement. Eggshells were rinsed with tap water and air dried for ≥30 days. Eggshell thickness with and without shell membranes was measured at three to five sites along the equator using a rounded-edge micrometer graduated in 0.01-mm units. Measurements were averaged to determine mean thickness for each egg and each site [13]. Length and breadth of each egg was measured using a straight-edge micrometer graduated in 0.01-mm units, and Ratcliffe indices were calculated for each egg and site [14].

Contaminant analyses. Thirty eggs collected in 1994 were sent to the Geochemical and Environmental Research Group, A&M University, College Station, Texas, USA, for analysis of OCs, congener-specific PCBs, PCDDs, polychlorinated dibenzofurans (PCDFs), and trace elements. Capillary gas chromatography with electron capture detection was used for analyses of OCs and PCBs [15]. High resolution gas chromatography and high resolution mass spectrometry were used for analyses of dioxins and furans [16]. Extraction of all trace elements followed U.S. EPA method 245.5 [17]. Cold vapor atomic absorption spectrometry was used in mercury analysis [18]. Graphite furnace or flame atomic absorption spectrometry [19] was used to analyze the remaining trace elements [9]. Detection limits were 20 ng/g for OCs and congener-specific PCBs, 50 ng/g for total PCBs, 10 pg/g for dioxin and furan congeners, and 0.2 μg/g for mercury in heron eggs in 1994. Due to high detection limits for organochlorine compounds in 1994, an alternate lab was selected in 1995.

Thirty eggs collected in 1995 were sent to Midwest Science Center for analysis of OCs, non-*ortho*-chloro-substituted PCBs, dioxins, and furans. Sample remains from six eggs collected in 1994 and analyzed by Geochemical and Environmental Research Group, Texas A&M University were also sent to Midwest Science Center for analyses (Table 1). Capillary gas chromatography with electron capture detection was used for analyses of OCs [20]. High resolution gas chromatography and high resolution mass spectrometry were used for analyses of non-*ortho*-chloro-substituted PCBs [21,22]. Gas chromatography and high resolution mass spectrometry were used for dioxin and furan analyses [23]. Detection limits in 1995 were 0.1 ng/g for OCs, 1 pg/g for non-*ortho*-chloro-substituted PCBs, and 0.1 pg/g for dioxins and furans in heron eggs and all prey samples.

Quality assurance by both labs included analysis of blanks, spiked samples, and replicates. Residue levels were not adjusted for percent recovery. Organic contaminant concentra-

Table 1. Quantitation of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) and 2,3,7,8-tetrachlorodibenzofuran (TCDF) in great blue heron split samples sent to the Geochemical Environmental Research Group (GERG) and the Midwest Science Center (MSC), 1994–1995

		Site					
Analyte	Lab	Bachelor	Fisher	Karlson	Molalla	Ross	Samish
2,3,7,8-TCDD	GERG	7.1	21	3.3	2.1	<1.9[a]	7.4
	MSC	6.0	18	3.1	1.5	7.1	6.7
2,3,7,8-TCDF	GERG	<2.0	<2.0	<2.0	<1.9	<1.9	<2.0
	MSC	0.8	0.8	0.8	0.9	1.0	1.0

[a] Below detection limit.

Table 2. Contaminant concentrations detected in great blue heron eggs collected from the Columbia and Willamette Rivers and Puget Sound, 1994–1995; results are geometric means and 95% confidence intervals (range); means with different letters were significantly different ($p = 0.05$) using the Tukey–Kramer multiple range test[a]

	Bachelor (+4)[b]		Fisher (−2)		Karlson (−17)	
	1994	1995	1994	1995	1994	1995
N[c]	5	5	5	5	5	5
% Lipid	4.2	5.0	4.7	6.1	4.1	6.4
% Moisture	81.6	—[d]	80.5	—	81.8	—
Organochlorines (ng/g fresh wet mass)						
p,p'-DDE	269XY	416AB	898X	183A	954X	775AB
	83–870	183–944	278–2,902	81–417	295–3,084	336–1,740
	(55–712)	(244–934)	(364–1,024)	(75–340)	(535–1,584)	(442–1,056)
p,p'-DDT	NC[e]	9.1	NC	4.6	NC	8.3
	—	3.2–26	—	1.6–13	—	2.9–24
	(<16–18)	(3.5–29)	(<16–45)	(0.5–31)	(<15–31)	(2.3–19)
trans-nonachlor	18X	12AB	36X	8.4A	63XY	31BC
	ND–42	7.0–22	ND–80	4.7–15	28–141	17–55
	(<16–37)	(8.1–17)	(<17–153)	(3.6–41)	(31–185)	(17–42)
Total PCBs	382X	615AC	1,394XY	423A	1,664XY	1,492BC
	133–1,288	372–1,016	410–4,675	256–699	493–5,625	903–2,464
	(117–849)	(424–863)	(443–6,947)	(233–854)	(91–3,038)	(656–2,665)
PCB congeners (ng/g fresh wet mass)						
77	<18	0.08ABC	<18	0.03A	NC	0.20B
		0.04–0.16		0.02–0.06	—	0.10–0.40
		(0.04–0.22)		(0.02–0.26)	(<15–22)	(0.06–0.32)
126	<18	0.17AC	<18	0.12A	NC	0.49BC
		0.10–0.29		0.07–0.21	—	0.29–0.82
		(0.12–0.34)		(0.06–1.45)	(<15–76)	(0.28–0.92)
169	NA[f]	0.02A	NA	0.02A	NA	0.04AB
		0.01–0.03		0.01–0.03		0.03–0.06
		(0.02–0.06)		(0.01–0.12)		(0.03–0.06)
PCDDs and PCDFs (pg/g fresh wet mass)						
2378-TCDD	2.7	2.5AB	5.2	1.7B	8.3	6.4A
	1.0–6.9	1.6–3.9	2.0–14	1.0–2.6	3.2–22	4.0–10
	(<1.9–7.2)	(1.3–4.0)	(<2.0–23)	(0.7–2.7)	(3.2–16)	(3.7–9.2)
OCDD	33	40	<20	10	NC	24
	17–64	15–104		3.8–26	—	9.0–62
	(9.7–181)	(9.8–292)		(8.4–26)	(<20–51)	(13–40)
2378-TCDF	<2.0	1.3	<2.0	0.6	<2.0	1.2
		0.7–2.2		ND–1.1		0.7–2.0
		(0.8–2.6)		(<0.7–0.9)		(0.8–1.8)
Elements (μg/g dry mass)						
Mercury	0.35	NA	0.46	NA	0.66	NA
	0.24–0.49		0.32–0.65		0.47–0.94	
	(0.23–0.58)		(0.31–0.91)		(0.42–0.94)	

[a] DDE = dichlorodiphenylethylene; TCDD = tetrachlorodibenzo-*p*-dioxin; OCDD = octachlorodibenzo-*p*-dioxin; TCDF = tetrachlorodibenzofuran; PCDD = polychlorinated dibenzo-*p*-dioxins; PCDF = polychlorinated dibenzofurans.
[b] Miles between site and nearest pulp or paper mill (+ if colony is upstream from mill and − if colony is downstream from mill).
[c] Sample size.
[d] Not available.
[e] Not calculated.
[f] Not analyzed.

tions in heron eggs were adjusted for moisture loss [24] and were reported in fresh wet mass. Residue concentrations in prey items were reported as wet mass. Trace element concentrations in heron eggs were reported in dry mass.

H4IIE bioassay. An aliquot of tissue was taken from heron eggs prior to residue analysis for the H4IIE bioassay. The assay was conducted following methods described by Tillitt et al. [25] with minor modifications to miniaturize and automate the procedure [26]. Fluorescence of rat hepatoma cells exposed to sample extracts and 2,3,7,8-TCDD (TCDD) was measured over time. Linear portions of both curves were used to determine relative potencies of egg and prey samples with TCDD-exposed cells as the standard for comparison [26]. Tetrachlorodibenzo-*p*-dioxin equivalents (TCDD-EQs) were calculated by the slope ratio assay [27]. Two samples were analyzed in triplicate, and replication was performed at many points during the bioassay. The detection limit of the bioassay was 0.043 pg TCDD-EQ/g, calculated using wet mass.

Heron reproductive success

Each colony was visited approximately three times each month during the nesting season. Observers located each chick at all visible nests. Herons were considered successfully fledged when they were able to fly to branches away from the nest, which was about 8 weeks of age [28]. Between 40 and 66 nests were followed at each site to determine hatching, fledging, and reproductive success. Blinds were used to min-

Table 2. Extended

Molalla (+10)		Ross (−14)		Samish (−38)	
1994	1995	1994	1995	1994	1995
5	5	5	5	5	5
4.0	5.4	4.2	6.0	4.6	6.4
81.7	—	81.1	—	81.1	—
795XY	353AB	1,616X	1,489B	101Y	150A
246–2,571	155–803	820–8,570	655–3,386	31–328	66–341
(347–2,225)	(68–1,015)	(95–6,414)	(158–7,339)	(59–176)	(65–588)
34	4.5	26	22	<16	5.0
16–69	1.6–13	ND–53	7.7-62		1.7–14
(<15–169)	(2.0–6.2)	(<18–75)	(11–66)		(1.1–62)
60XY	27ABC	199Y	88C	NC	14AB
27–133	15–48	89–446	49–156	—	7.6–24
(21–171)	(7.7–52)	(56–853)	(55–126)	(<16–33)	(4.9–29)
425XY	534AC	3454Y	2,426B	223X	396A
126–1,437	333–910	1,895–21,612	1,469–4,008	66–752	240–654
(271–725)	(185–1,851)	(106–33,168)	(1,501–4,649)	(92–836)	(324–459)
<19	0.03A	<18	0.23BC	<17	0.05A
	0.02–0.07		0.11–0.46		0.03–0.10
	(0.02–0.08)		(0.05–0.59)		(0.02–0.20)
<19	0.17AC	<18	0.65B	<17	0.21AC
	0.10–0.29		0.39–1.08		0.13–0.34
	(0.09–0.48)		(0.32–1.71)		(0.14–0.57)
NA	0.02A	NA	0.06B	NA	0.03AB
	0.01–0.04		0.04–0.10		0.02–0.04
	(0.01–0.05)		(0.03–0.17)		(0.02–0.06)
3.2	3.0AB	4.2	6.3A	NC	3.7AB
1.2–8.4	1.9–4.7	1.6–11	4.0–10	—	2.3–5.8
(2.2–4.7)	(1.8–5.1)	(<1.0–19)	(3.7–11)	(<1.9–7.7)	(2.1–8.9)
NC	12	NC	36	NC	34
—	4.7–33	—	14–93	—	13–89
(<19–21)	(4.9–37)	(<10–30)	(12–164)	(<19–29)	(12–120)
<2.0	0.5	<1.9	1.3	NC	1.3
	ND–0.9		0.8–2.3	—	0.8–2.3
	(<0.8–0.6)		(0.6–3.4)	(<1.9–6.0)	(0.5–4.3)
0.65	NA	0.62	NA	0.53	NA
0.46–0.92		0.44–0.88		0.37–0.75	
(0.45–0.91)		(0.41–1.06)		(0.28–0.99)	

imize disturbance. Observations began just prior to hatching and continued for 9 weeks past the estimated peak of hatching.

Clutch size was estimated by averaging the number of eggs seen in each nest when eggs were collected. A successful nest was defined as a nest in which at least one chick hatched. Hatch success was calculated as the maximum number of chicks seen in a successful nest. Fledge success was computed as the number of chicks present at each successful nest on week 8 (unless an earlier fledge date had been recorded). Nests containing chicks that were not fully feathered by week 9 were censored. Reproductive success was reported as the number of chicks fledged per number of occupied nests, including nests that were abandoned or predated during nesting.

Statistical analyses

Although both labs detected similar contaminant concentrations in aliquots from identical eggs, residues for 1994 and 1995 were analyzed separately due to differences in detection limits. Contaminant concentrations in eggs were converted to fresh wet mass and log transformed to normalize skewed distributions. Means for each contaminant at each site were calculated for compounds with levels above the detection limit in at least half of the samples. Samples with concentrations below the detection limit were assigned a value of one half of the detection limit for computations.

We used two-way analysis of variance to test differences between years and among sites in eggshell thickness, Ratcliffe Index, enzymatic activity, clutch size and hatch, fledge, and reproductive rates. We used a one-way analysis of variance to test among-site differences in residue concentration. When a significant difference was detected ($p \leq 0.05$), differences among means were determined using a multiple range test with the Tukey–Kramer adjustment for unequal sample sizes. To test differences among sites in nest failure, we used logistic regression. We calculated toxic equivalents (TEQs) for PCDD and PCDF concentrations using factors from a rodent-based model [29] and a chicken-based model [30]. Toxic equivalents were calculated for PCB concentrations using rodent [29] and chicken-based [30] factors. Total TEQs were calculated for each site by summing dioxin, furan, and PCB TEQs. We used

Table 3. Shell thickness (mm) and Ratcliffe Index in great blue heron eggs collected from the Columbia and Willamette Rivers and Puget Sound, 1994–1995; results are arithmetic means (SE); means with different letters were significantly different ($p = 0.05$) using the Tukey–Kramer multiple range test

	Site						
	Bachelor	Fisher	Karlson	Molalla	Ross	Samish	pre-1947[a]
N	26	20	25	25	20	24	64 (130)[b]
Length (mm)	65.15 (0.40)	64.41 (0.69)	64.23 (0.62)	64.74 (0.57)	64.30 (0.46)	63.90 (0.53)	—
Width (mm)	46.48 (0.26)	45.48 (0.24)	46.31 (0.83)	46.60 (0.77)	46.15 (0.24)	45.01 (0.18)	—
Shell thickness without membrane	0.316A (0.005)	0.313AB (0.007)	0.325A (0.005)	0.308AB (0.007)	0.291B (0.005)	0.307AB (0.006)	—
Shell thickness with membrane	0.362A (0.005)	0.352AB (0.007)	0.370A (0.006)	0.351AB (0.007)	0.333B (0.005)	0.352AB (0.006)	0.389C (0.005)
% Thinning[c]	−6.9	−9.5	−4.9	−9.8	−14.4	−9.5	—
Ratcliffe index	1.92AB (0.03)	1.89AB (0.04)	1.94A (0.04)	1.83AB (0.04)	1.77B (0.03)	1.84AB (0.03)	2.02C (0.02)

[a] For *Ardea herodias fannini* in the Pacific Northwest [33].
[b] Sample size for shell thickness = 130 mm; sample size for Ratcliffe Index = 64.
[c] Compared to pre-1947 mean.

linear regression to determine whether relationships existed between distance of colonies from pulp and paper mills, biological parameters, and residue concentrations.

Apparent biomagnification factors (BMFs) were calculated for each site as the geometric mean of a residue in great blue heron eggs divided by the geometric site mean of that residue in prey items. We collected prey items opportunistically and did not weight site means to account for heron diet composition because that data was not available for the study area. Because the prey we collected was freshly regurgitated or rejecta cast from the nests, the apparent BMF should be a reasonable approximation of the actual BMF. To compare BMFs among piscivorous birds from the Columbia River, we used results from this study, residue concentrations in double-crested cormorant and bald eagle eggs collected from the estuary in 1994 to 1995 [4], and fish collected from the lower Columbia River during 1991 and 1993 [31]. To approximate contaminant concentrations accumulated by bald eagles, data for BMF calculations was restricted to composites containing fish <33 cm in length [32]. Dietary composition was weighted according to proportions of birds and fish in eagle diets [29]. Because the diet composition of bald eagles on the lower Columbia River changes markedly throughout the year, upper and lower bounds for BMFs were calculated. The actual BMF for each compound most likely lies between the two values.

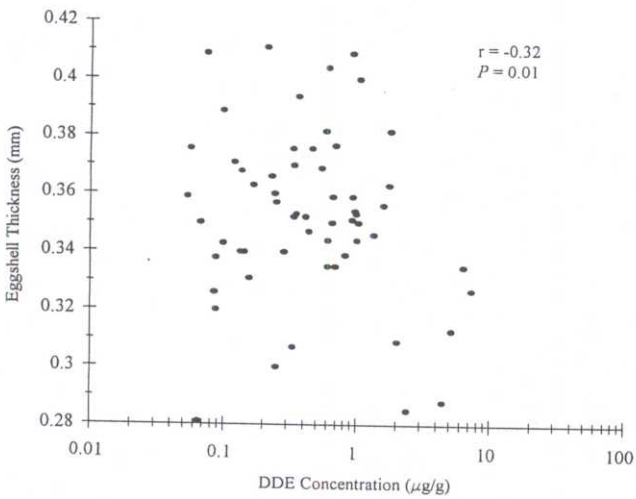

Fig. 2. Inverse relationship between eggshell thickness (mm) and DDE concentration (μg/g) in great blue herons from the Columbia and Willamette Rivers and Puget Sound, 1994 to 1995.

RESULTS

Contaminants in herons

Of the 25 OCs analyzed, dieldrin, oxychlordane, *p,p′*-DDE, *p,p′*-DDT, *cis*- and *trans*-nonachlor, and total PCBs were consistently above detection limits [9]. Of the PCBs, di-*ortho*-chlorinated congeners 138, 153, and 180 were consistently above detection limits in 1994 [9]. Most other congeners were detected at low levels in a few eggs from Fisher, Karlson, and Ross Islands. Only non-*ortho*-chlorinated (non-*ortho*) congeners were analyzed in 1995, and all were above detection limits.

Tetrachlorodibenzo-*p*-dioxin was the only PCDD consistently above detection limits at all sites in both years. Other PCDD and PCDF compounds were either below the 1994 detection limits or present in too few samples to allow calculation of site means. With the exception of the hepta- and octa-chlorinated furans, most PCDDs and PCDFs were above detection limits in 1995. Mercury concentrations were also above 1994 detection limits.

Eggs from Ross had the highest mean concentrations of all measured OCs (Table 2). Concentrations of DDE were significantly greater at Ross than at Fisher and Samish. Although mean DDE concentrations did not differ from any other site, Molalla had an unusually high ratio of DDT to total DDT-related compounds in 1994. This ratio was 7 to 18 times higher than other site ratios and indicates recent exposure to DDT [13]. In 1995, the DDT ratio at Molalla was similar to that of the other sites. Concentrations of *trans*-nonachlor were significantly greater at Ross compared to Bachelor, Fisher, and Samish. Total PCB concentrations were significantly higher at Ross than at most other sites. Although site means for all OCs are relatively low, concentrations of DDE and total PCBs were elevated in some eggs from Ross, with levels up to 7.3 μg/g for DDE and 33 μg/g for PCBs. Mercury concentrations in eggs were low at all sites in 1994 and were not analyzed in 1995.

PCB congeners 81 and 169 were not quantitated in 1994

Table 4. Contaminant concentrations in whole body composite prey samples[a] collected from the Columbia and Willamette Rivers and Puget Sound, 1994–1995; results are geometric means and 95% confidence intervals (range); means with different letters were significantly different using the Tukey–Kramer multiple range test[b]

	Bachelor (n = 6/12)[c]	Fisher (n = 5/6)	Karlson (n = 5/11)	Molalla (n = 4/5)	Ross (n = 2/4)	Samish (n = 4/30)
Organochlorines (ng/g wet mass)						
p,p'-DDE	45.7AB	71.0A	6.00B	22.2AB	63.1AB	3.64B
	13.3–71.9	24.5–206	2.27–15.9	6.75–73.0	11.7–340	1.11–12.0
	(9.63–105)	(1.45–142)	(0.65–46.7)	(6.70–130)	(56.6–70.3)	(1.37–6.93)
p,p'-DDT	0.76	0.85	0.30	2.08	13.4	0.31
	0.16–3.61	0.12–6.15	ND–1.85	0.23–18.9	0.59–304	ND–2.79
	(<0.06–19.1)	(<0.06–2.53)	(<0.06–9.99)	(0.26–26.2)	(9.99–17.9)	(<0.06–0.89)
trans-nonachlor	2.15AB	2.75AB	0.41A	2.44AB	15.3B	0.49AB
	0.79–5.87	0.77–9.77	0.13–1.29	0.59–10.1	2.06–114	0.12–2.01
	(0.84–13.3)	(0.29–6.38)	(<0.06–5.97)	(0.82)	(7.79–30.1)	(0.22–0.87)
Total PCBs	94BC	118BC	20A	40AC	627B	30AC
	50–178	53–265	10–41	16–97	176–2233	12–74
	(35–489)	(33–169)	(3–68)	(23–56)	(447–880)	(22–44)
PCB congeners (ng/g wet mass)						
77	0.05AB	0.15AB	0.03AB	0.02A	0.42B	0.05AB
	0.02–0.12	0.05–0.39	0.01–0.07	0.01–0.06	0.09–2.01	0.02–0.12
	(0.01–0.16)	(0.03–0.32)	(0.004–0.14)	(0.01–0.03)	(0.21–0.85)	(0.03–0.14)
126	0.02	0.02	0.01	0.01	0.05	0.01
	0.01–0.04	0.01–0.04	0.003–0.02	0.003–0.01	0.01–0.17	0.004–0.02
	(0.01–0.05)	(0.01–0.04)	(<0.001–0.02)	(0.01–0.01)	(0.03–0.09)	(0.01–0.01)
169	0.002	0.001	0.003	0.001	0.002	NC[d]
	0.001–0.005	ND–0.002	ND–0.003	ND–0.002	0.002–0.002	—
	(0.001–0.01)	(<0.001–0.003)	(<0.001–0.003)	(<0.001–0.002)	(0.002–0.002)	(<0.001–0.001)
PCDDs and PCDFs (pg/g wet mass)						
2378-TCDD	NC	0.72	0.23	NC	0.75	NC
	—	0.32–1.59	0.10–0.51	—	0.19–2.97	—
	(<0.10–0.50)	(0.40–1.30)	(<0.10–1.0)	(<0.10–0.10)	(0.70–0.80)	(<0.10–0.20)
OCDD	5.78A	3.08AB	1.56B	2.34AB	6.90AB	2.86AB
	3.35–9.97	1.64–5.78	ND–2.93	1.17–4.68	2.31–21	1.43–5.70
	(1.50–14)	(2.00–12)	(<1.5–1.8)	(0.90–7.90)	(5.80–8.20)	(2.20–4.20)
2378-TCDF	0.93AB	3.95A	0.48AB	0.25B	2.19AB	0.69AB
	0.34–2.58	1.22–13	0.15–1.57	ND–0.91	0.28–17	ND–2.51
	(0.30–4.00)	(0.50–16)	(<0.10–7.90)	(<0.10–1.30)	(1.50–3.20)	(<0.50–1.70)

[a] Prey species included brown bullhead, common carp, crappie spp., largescale sucker, pacific lamprey, peamouth chub, warmouth bass, American shad, trout spp., saddleback gunnel, shiner perch, and snake prickleback.
[b] DDE = dichlorodiphenyletheylene; TCDD = tetrachlorodibenzo-*p*-dioxin; OCDD = octachlorodibenzo-*p*-dioxin; TCDF = tetrachlorodibenzofuran; PCDD = polychlorinated dibenzo-*p*-dioxins; PCDF = polychlorinated dibenzofurans.
[c] Number of composite samples/number of individuals per composite.
[d] Not calculated.

Table 5. Mean biomagnification factors (SE) for contaminants detected in great blue heron eggs in 1995 and prey from the Columbia and Willamette Rivers and Puget Sound, 1994–1995[a]

	Bachelor	Fisher	Karlson	Molalla	Ross	Samish
Organochlorines						
p,p'-DDE	9 ± 0.9	3 ± 0.9	143 ± 0.9	16 ± 0.8	24 ± 0.7	41 ± 0.8
trans-nonachlor	4 ± 0.8	3 ± 0.7	89 ± 0.7	11 ± 0.7	6 ± 0.5	28 ± 0.7
Total PCBs	5 ± 0.9	4 ± 0.9	73 ± 0.9	13 ± 0.8	4 ± 0.7	13 ± 0.8
PCB congeners						
77	2 ± 0.8	0.2 ± 0.8	18 ± 0.8	2 ± 0.7	1 ± 0.5	1 ± 0.8
126	9 ± 0.8	7 ± 0.7	150 ± 0.7	28 ± 0.7	13 ± 0.5	31 ± 0.7
169	9 ± 0.7	18 ± 0.6	12 ± 0.6	32 ± 0.6	30 ± 0.4	14 ± 0.6
Dioxins						
2,3,7,8-TCDD	NC[b]	2 ± 0.8	23 ± 0.8	NC	8 ± 0.6	NC
OCDD	9 ± 1.3	5 ± 1.2	20 ± 1.2	7 ± 1.2	5 ± 1.1	10 ± 1.3
Furans						
2,3,7,8-TCDF	1 ± 0.7	0.2 ± 0.7	3 ± 0.7	2 ± 0.6	1 ± 0.5	2 ± 0.7

[a] DDE = dichlorodiphenyletheylene; TCDD = tetrachlorodibenzo-*p*-dioxin; OCDD = octachlorodibenzo-*p*-dioxin; TCDF = tetrachlorodibenzofuran.
[b] Not calculated.

Table 6. Calculated toxic equivalents (TEQs) and H4IIE bioassay tetrachlorodibenzo-*p*-dioxin equivalents (TCDD-EQs) of great blue heron eggs from the Columbia and Willamette Rivers and Puget Sound, 1994–1995 (pg/g); results are geometric means and 95% confidence intervals (range); means with different letters were significantly different ($p = 0.05$) using the Tukey–Kramer multiple range test

	Bachelor		Fisher		Karlson	
	1994	1995	1994	1995	1994	1995
N[a]	5	5	5	5	5	5
Dioxin TEQs						
Rodent-based[b]	13	6.2AB	19	4.9A	25	12B
	7.8–21	4.0–9.6	11–31	3.2–7.6	15–41	8.0–19
	(9.9–17)	(3.7–13)	(11–45)	(2.5–9.5)	(12–75)	(8.3–18)
Chicken-based[c]	20	11AC	29	8.2A	38	19ABC
	13–32	6.9–16	19–47	5.4–12	24–60	13–29
	(17–24)	(6.6–22)	(19–64)	(3.8–17)	(19–119)	(14–30)
PCB TEQs						
Rodent-based[b]	14X	19A	63XZ	14A	76YZ	53C
	6.6–28	12–31	30–129	8.4–22	37–157	33–86
	(12–18)	(13–24)	(16–285)	(6.9–26)	(44–136)	(32–98)
Chicken-based[c]	1.0X	19A	3.7XY	13A	4.2YZ	54C
	0.5–2.0	12–31	1.9–7.2	8.1–21	2.1–8.1	33–87
	(0.9–1.2)	(13–23)	(1.2–16)	(6.5–26)	(2.4–7.4)	(33–98)
Total TEQs						
Rodent-based[b]	27	26	125	21	114	70
	23–30	20–32	15–235	11–31	60–168	45–95
	(23–32)	(17–34)	(27–329)	(10–36)	(59–211)	(40–115)
Chicken-based[c]	21	31	39	25	52	77
	19–24	23–39	17–61	13–37	15–90	50–105
	(18–25)	(20–44)	(20–80)	(12–43)	(22–126)	(47–127)
H4IIE bioassay TCDD-EQs	2.0	<1.7	1.5	<0.6	30	NC[c]
	0.2–22		0.1–18		2.6–344	—[d]
	(<0.1–11)		(<0.1–6.2)		(4.9–78)	(<1.7–30)

[a] Sample size.
[b] [29].
[c] [30].
[d] Not calculated.

(Table 2). Only non-*ortho* congeners were analyzed in 1995, and all four congeners were detected in eggs from each site. Of the non-*ortho* PCBs, congener 126 was present in the highest concentrations. Concentrations of 126 were significantly greater in eggs from Ross (0.65 ng/g) compared to eggs from Bachelor, Fisher, Molalla, and Samish. For many congeners, eggs from Ross had the highest mean concentration.

Significant among-site differences were detected in mean concentrations of 2,3,7,8-TCDD (Table 2). In 1995, Fisher had the lowest concentration of 2,3,7,8-TCDD and Karlson and Ross had mean concentrations over three times higher. Concentrations of octachlorodibenzo-*p*-dioxin (OCDD) in eggs from Bachelor, Karlson, Ross, and Samish were elevated. No significant correlation was detected between distance of the colonies from pulp and paper mills and mean TCDD or tetrachlorodibenzofuran (TCDF) concentration ($r = 0.27$, $p = 0.43$ and $r = 0.52$, $p = 0.29$, respectively). The TCDF–distance correlation coefficient is relatively large; the lack of significance may be due to a small sample size and low statistical power (power < 0.40).

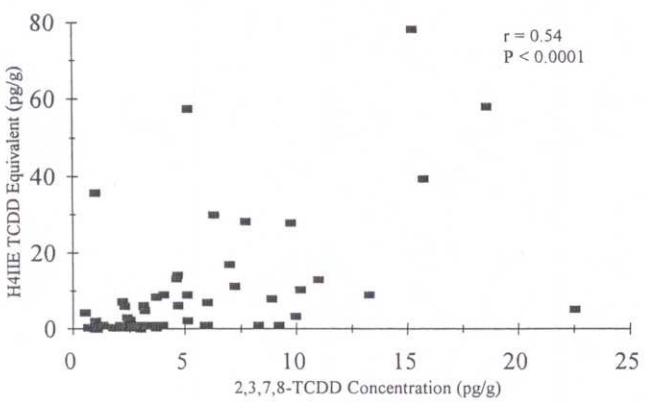

Fig. 3. Positive correlation between H4IIE TCDD equivalents (pg/g) and 2,3,7,8-TCDD concentration (pg/g) in great blue herons from the Columbia and Willamette Rivers and Puget Sound, 1994 to 1995.

Eggshell thickness. Shell thinning at Ross was two and three times greater than at Bachelor and Karlson, respectively (Table 3). All sites had significantly reduced shell thickness (0.333–0.370 mm) and Ratcliffe index compared to the pre-DDT mean (0.389 mm) measured by Anderson and Hickey [33]. Shell thickness and DDE concentration in individual eggs were negatively correlated ($r = -0.32$, $p = 0.01$) (Fig. 2).

Contaminants in heron prey

With the exception of *p,p'*-DDE, OC concentrations were highest in heron prey from Ross (Table 4). Concentrations of *trans*-nonachlor were significantly greater at Ross compared to Karlson. The DDE concentrations were significantly greater in prey from Fisher compared to prey from Karlson and Samish. Total PCB concentrations were 4.8 to 31 times greater at Ross compared to all other sites and were highest in largescale suckers (*Catostomus macrocheilus*), carp (*Cyprinus carpio*), peamouth chub (*Mylocheilus caurinus*), and trout (*Salmo* spp.) from nonreference sites [9].

Concentrations of PCB congeners in prey items were low

Table 6. Extended

Molalla (n = 5)		Ross (n = 5)		Samish (n = 5)	
1994	1995	1994	1995	1994	1995
5	5	5	5	5	5
14	7.3AB	18	15B	13	12AB
8.7–11	4.7–11.3	11–30	9.9–24	7.8–21	7.6–18
(11–22)	(4.3–17)	(5.4–36)	(9.9–22)	(10–22)	(8.0–24)
23	10AC	29	25B	22	20BC
14–36	6.8–15	19–47	17–38	14–35	14–31
(19–35)	(6.6–17)	(9.4–54)	(16–35)	(18–34)	(14–37)
17XZ	19A	103Y	71BC	13X	19A
8.2–35	12–30	50–213	44–114	6.2–26	12–31
(13–30)	(9.6–51)	(15–338)	(34–127)	(12–14)	(14–32)
1.2XZ	18A	7.1Y	70BC	1.0X	19A
0.6–2.2	11–29	3.6–14	43–113	0.5–1.9	12–30
(1.0–1.7)	(9.3–49)	(1.1–23)	(33–129)	(0.9–1.0)	(13–31)
33	31	206	97	26	33
23–42	12–51	20–361	54–140	22–31	25–42
(25–52)	(14–68)	(68–344)	(45–149)	(22–35)	(23–47)
24	33	47	106	24	42
18–31	15–51	27–66	61–152	17–30	31–52
(19–37)	(16–66)	(11–71)	(53–163)	(19–35)	(28–54)
2.3	NC	3.5	NC	1.5	2.5
0.2–27	—	0.3–40	—	0.1–17	1.2–3.8
(<0.1–14)	(<0.6–9.0)	(<0.1–58)	(<1.7–17)	(<0.1–36)	(<1.7–8.0)

(Table 4). At all sites, mean concentrations in prey were highest for congener 77 and ranged up to 847 pg/g in trout from Ross Island. Concentrations of congener 77 were significantly greater in prey samples from Ross compared to prey from Karlson. Trout from Ross had the highest concentrations of three of the four congeners.

The overall mean OCDD concentration in prey from Bachelor was significantly higher than the mean OCDD concentration at Karlson. The OCDD levels were generally highest in crayfish (*Pacifastacus leniusculus*), with concentrations ranging from 6.0 pg/g at Karlson up to 14.0 pg/g at Bachelor. Largescale suckers from Bachelor and Ross had elevated OCDD concentrations of 9.9 and 8.2 pg/g, respectively. The mean TCDF concentration at Fisher was significantly higher than that at Molalla and almost six times greater than the level detected at the reference site. Tetrachlorodibenzo-*p*-dioxin equivalents were present at trace levels in most species from Fisher, Karlson, and Ross.

Biomagnification factors

Compounds with the highest BMFs differed among sites (Table 5); Karlson had the greatest BMFs for all contaminants except PCB 169. Of the PCBs, congeners 126 and 169 had the highest. Biomagnification factors for congener 77 at Fisher, Ross, and Samish were ≤1.0, suggesting no biomagnification. Biomagnification factors were calculated for TCDD at three sites and all were >1.0, indicating biomagnification. Biomagnification of TCDF did not occur (BMFs ≥1.0) at three of our six sites, and TCDF BMFs at the remaining sites were low.

Bioassays

The TCDD-EQs were positively correlated ($r = 0.54$; $p < 0.0001$) with 2,3,7,8-TCDD concentrations in heron eggs (Fig. 3). The TCDD equivalents (TCDD-EQs) from the 1994 H4IIE bioassay were not statistically different among sites (Table 6); however, the TCDD-EQ for Karlson was 9 to 20 times greater than that for any other site. An among-site comparison was not conducted for the 1995 results because only one site had a sufficient number of detections to allow calculation of a site mean. H4IIE bioassays were also conducted with extracts from prey items, and activity in all samples was below the detection limit.

Toxic equivalents

Toxic equivalents (TEQs) for Karlson and Ross were consistently greater than those for most other sites and were independent of year or model (rodent vs chicken) used (Table 6). Total TEQs differed between years largely due to differences in PCB TEQs. Mono-*ortho*-PCB concentrations were included in the 1994 TEQ calculations, and comparable data for 1995 was not available. The rodent-based and international TEQs [29,34] were similar for most residues. The TEQs calculated using the chicken-based model [30] were consistently lower due to greater weight given to PCDD/F compounds. The PCBs contributed ≤54% to the chicken-based TEQ, whereas PCBs contributed 57 to 84% of the rodent-based TEQ. Total TEQs calculated from egg residue concentrations were up to 24 times greater than those derived from the H4IIE bioassay.

Table 7. Reproductive parameters (SE) and embryo deformities of great blue herons from the Columbia and Willamette Rivers and Puget Sound, 1994–1995; means with different letters were significantly different ($p = 0.05$) using the Tukey–Kramer multiple range test

	Site											
	Bachelor		Fisher		Karlson		Molalla		Ross		Samish	
	1994	1995	1994	1995	1994	1995	1994	1995	1994	1995	1994	1995
Colony Size[a]	563	563	225	256	107	79	96	77	55	71	207	205
Nests observed	43	46	43	52	42	34	43	45	36	39	48	51
Failed nests	2	2	1	2	13	11	1	2	3	3	6	5
Clutch size[b]	3.7	3.2	3.6	3.7	3.1	3.5	3.9	3.4	4.2	3.3	3.6	3.5
	(0.3)	(0.2)	(0.3)	(0.2)	(0.3)	(0.2)	(0.3)	(0.2)	(0.3)	(0.3)	(0.3)	(0.3)
Hatch rate[c]	2.6AB	2.1	3.0AB	1.9	3.2B	1.8	2.5A	1.9	2.7AB	2.2	2.4A	2.1
	(0.1)	(0.1)	(0.1)	(0.1)	(0.2)	(0.2)	(0.1)	(0.1)	(0.2)	(0.1)	(0.1)	(0.1)
Fledge rate[d]	2.3AB	2.4	2.6BC	2.3	3.0BC	2.6	2.1AB	2.2	2.0A	2.5	1.9A	2.4
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.2)	(0.1)	(0.1)	(0.1)
Reproductive rate[e]	2.2AB	2.3	2.4A	2.3	2.2AB	2.4	2.0AB	2.1	1.8AB	2.4	1.7B	2.4
	(0.2)	(0.1)	(0.2)	(0.1)	(0.2)	(0.17)	(0.2)	(0.1)	(0.2)	(0.2)	(0.1)	(0.1)
% Deformed embryos[f]	0	0	20	0	40	0	40	10	0	20	0	0

[a] Total number of nests.
[b] Average number of eggs/observed nests.
[c] Maximum number of chicks observed/successful nests.
[d] Number of chicks fledged/successful nests.
[e] Number of chicks fledged/observed nests, abandoned and predated nests included.
[f] Number of deformed embryos/embryos harvested for bioassay.

Heron reproductive success

Clutch size ranged from 3.1 to 4.2 eggs per nest (Table 7) and was not different among sites. Hatching success ranged from 1.8 to 3.2 chicks per nest and was significantly greater at Karlson compared to Molalla and Samish in 1994. Nests at Karlson had the highest fledging success during both years. In 1994, fledging success at Ross and Samish (2.0 and 1.9, respectively) was significantly lower than at Fisher and Karlson (2.6 and 3.0, respectively). Reproductive success ranged from 1.7 to 2.4 young fledged per occupied nest and was significantly greater at Fisher in 1994 compared to Samish. Fledging and reproductive success were not correlated with concentrations of DDE ($r \leq -0.47$, $p \geq 0.68$), total PCBs ($r \leq -0.57$, $p \geq 0.54$), or TCDD ($r \leq -0.86$, $p \geq 0.06$). However, the TCDD correlations were suggestive, and a larger sample size (i.e., higher statistical power) would clarify any relationships. Deformities were found in pipping embryos from four of the six sites. Gross physical deformities such as misshapen heads, crossed bills, and club feet were found at Karlson in 1994 and Molalla in both years. Neck edema was observed in embryos from Molalla and Ross in 1995.

Nest failure differed markedly among sites. Nest failures for 1994 and 1995 were combined because differences between years were not significant. Fisher and Molalla had the lowest (4%) and Karlson had the highest (30%) percentage of failed nests (Fig. 4). The odds of a nest failure at Karlson ranged from 4 to 13 times greater than at any other site and was significantly higher compared to all other sites ($p \leq 0.003$). This apparent contradiction between rates of hatching, fledging, and nest failure at Karlson may be explained by a bimodal pattern in nest success. Most nests at all other sites hatched and fledged between one to three eggs and chicks. In contrast, most nests at Karlson either fledged three to four chicks or completely failed. Site means for nest failure were positively correlated with mean TCDD concentration ($r = 0.82$, $p = 0.04$). In addition, individual fledge success was negatively correlated ($r = -0.35$, $p < 0.001$) with TCDD concentrations detected in eggs from the same nests.

DISCUSSION

Contaminant concentrations in herons

The results of this study supported the hypothesis that environmental contaminants were elevated in great blue herons from the lower Columbia and Willamette Rivers. Concentrations of DDE detected in heron eggs during this study were generally below levels previously detected in other piscivorous birds from the Columbia River [1,12,35,36] but above those detected in recent studies on herons in other areas of the Pacific Northwest [13]. Elevated DDE concentrations present in black-crowned night heron and bald eagle eggs from the Columbia River were associated with reduced reproductive success [1,35]. The DDE concentrations at Molalla were similar to, and at Ross exceeded, concentrations detected in two heron eggs collected from colonies on the Willamette River in 1975 [37]. With the exception of eggs collected from Bachelor, mean DDE concentrations from all sites in 1995 exceeded elevated concentrations in heron eggs from an agricultural area in Brit-

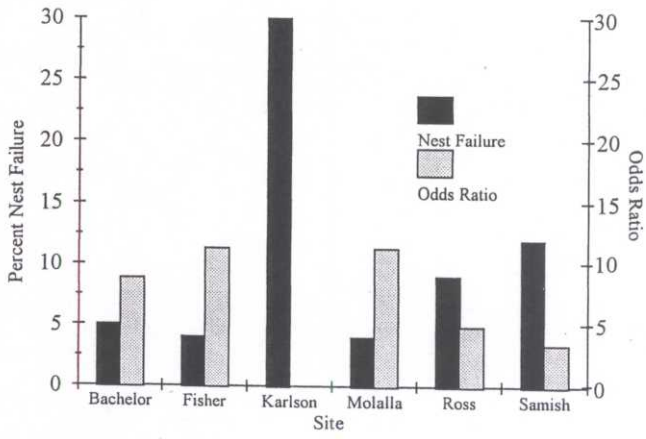

Fig. 4. Odds ratios for nest failure (relative to Karlson) of six great blue heron colonies along the Columbia and Willamette Rivers and Puget Sound, 1994 to 1995.

ish Columbia. Eggs from Bachelor and Samish had DDE levels that were similar to those at less contaminated sites [13]. Adverse effects of DDE on hatchability or reproductive success of great blue herons are unlikely at the concentrations observed during this study. Great blue herons are relatively insensitive to DDE contamination and eggs have successfully pipped while carrying a load of 78 μg/g [38]. The remaining OCs were either not detected or were present in concentrations below levels associated with adverse effects in predatory birds [12,35,36,39].

Total PCB concentrations at Ross in 1994 exceeded levels detected in previous piscivorous bird studies on the Columbia River and in Washington, California, and British Columbia [12,13,36,40]. They were similar to levels found in concurrently collected double-crested cormorant and bald eagle eggs from the Columbia River [4] but below the mean PCB concentration in bald eagles in 1985 to 1987 [1]. One egg from Ross had 33 ppm total PCBs, exceeding the critical values associated with impaired embryo health in chickens [41,42] and reduced reproductive success in Forster's terns [2]. This nest fledged two chicks, thus demonstrating the lower sensitivity of great blue herons to dietary PCBs. Total PCB concentrations at all other sites were either below or similar to concentrations previously detected in bird eggs from healthy populations along the Columbia between 1978 and 1982 [1,12,35,36].

Concentrations of the most toxic PCBs, the non-*ortho* congeners, were low in heron eggs in this study. Mean concentrations were below levels that were detected in Columbia River bald eagles and associated with reduced productivity [1]. Of the non-*ortho* PCB congeners, 126 was present in the highest concentrations in heron eggs in this study. In contrast, congener 77 was the most elevated non-*ortho* PCB detected in recently collected bald eagle eggs [4]. Differences in species-specific metabolism of PCB congeners may be partially responsible for this discrepancy.

With the exception of OCDD, mean PCDD and PCDF concentrations in great blue heron eggs from the Columbia and Willamette Rivers were low in 1994 and 1995. Compared to concentrations in heron eggs in this study, bald eagle [1] and double-crested cormorant eggs recently collected from the Columbia River contained much greater TCDD and TCDF concentrations [4]. The TCDD concentrations in heron eggs in this study were generally below the 10 pg/g background level suggested for eggs of piscivorous birds in the Great Lakes [2] and were similar to concentrations found at reference sites in British Columbia, Canada [13,43–45]. The TCDF concentrations in eagle eggs collected from Bachelor, Fisher, and Karlson Islands were five to eight times greater than concentrations detected in heron eggs from the same areas [4]. This difference is likely indicative of dietary differences between these two species; eagles partially feed one trophic level above herons, with a significant portion of fish-eating birds in their diet [32]. Highly chlorinated TCDD congeners were present in greater amounts relative to tetra- and penta-chlorinated congeners in heron eggs from this study, as is typical in environmental samples [34]. Although OCDD concentrations were elevated at all sites, concentrations are probably not biologically significant due to the low toxicity of OCDD relative to tetra- through hexachloro-2,3,7,8-substituted congeners [29].

Mercury concentrations in our heron eggs were less than residues previously detected in eggs from most other fish-eating birds from the Columbia River [1,46]. Mean mercury concentrations in eggs from Karlson, Molalla, and Ross were higher than levels detected in British Columbia [13] but below concentrations associated with reduced hatch, fledge, and reproductive success in common terns and common loons from the Great Lakes [47].

Toxic equivalents

Although PCB concentrations in heron eggs were not high in terms of potential to cause direct toxicity, they contributed substantially to the calculated TEQs. In 1994, PCB TEQs from the rodent-based model were comparable to 1995 PCB TEQs, which were based solely on non-*ortho* congeners. This is due to a considerable contribution to the 1994 TEQ by one mono-*ortho* and three di-*ortho* congeners, namely 118, 180, 153, and 138, respectively. These four congeners constituted 48 to 100% of the total PCBs quantified in 1994. Therefore, the PCB TEQ that we report is probably low for both years because the contribution of both groups was not measured simultaneously.

Regardless of which model (rodent or chicken based) we used to calculate total TEQs, our calculated values were much higher than the values obtained via the H4IIE bioassay. The bioassay TCDD-EQs are more representative of the actual effects of PCDDs, PCDFs, and PCBs on great blue herons because they are not restricted by the primary assumption of additive effects inherent in the other TEQ models [34,48]. Because the rat hepatoma cells in the bioassay are exposed to the mixture of contaminants present in the heron eggs, the results integrate both the individual toxicities of the compounds and their interactions for a more accurate result [25,49]. Our results suggest that the PCDDs, PCDFs, and PCBs are not acting in an additive manner in great blue herons.

Contaminant concentrations in prey

Organochlorine concentrations in Columbia and Willamette River fish have been monitored for the last three decades. Concentrations of DDE and total PCBs detected in prey were similar to or below concentrations detected in fish from previous studies on both rivers [9]. The DDE concentrations at all our sites were below both the carcinogenic and noncarcinogenic levels of concern established by New York State [50]. Similar guidelines for PCB levels, 0.1 μg/g [51] and 0.11 μg/g [50] were exceeded by prey from Bachelor, Fisher, and Karlson Islands. The TCDD concentrations in fish in this study were below guidelines for piscivorous wildlife [50] but exceed the consumption guideline for human health [52]. The TCDD exceedence demonstrates the need for continued regulation on dioxin loads in both rivers and suggests that the current limits need to be lowered.

Biomagnification factors

Care must be taken to look at identical tissues when comparing BMFs among studies. The BMFs calculated between prey and whole bodies of fish-eating birds will be uniformly larger than those calculated between prey and bird eggs. Biomagnification factor profiles of different tissues may also differ within a species due to preferential retention or elimination of a compound from a tissue [53].

Biomagnification factors varied greatly among colonies for most compounds. These among-site differences may partly reflect differences in dietary composition of herons at various locations along the river. Biomagnification factors were greatest at Karlson for all compounds, which was largely due to

markedly low residue concentrations detected in prey. With the exception of Bachelor and Fisher, BMFs for DDE were much higher than those for DDT. This is likely due to the rapid metabolism of DDT to DDE in most tissues. Most of our BMFs for OCs were below those reported for other fish-eating birds in the Great Lakes, Canada, and Great Britain [53–56]. These differences may result from dietary differences or species differences in tissue distribution and elimination [53]. Our results are similar to those of Norstrom et al. [57], who reported BMFs for DDE, HCB, oxychlordane, and heptachlor epoxide in herring gulls (*Larus argentatus*) ranging from 11 to 55. In contrast to previous studies [53], TCDD was not the most highly biomagnified PCDD or PCDF. Little biomagnification occurred with TCDF, which is in agreement with an earlier study on Forster's terns in the Great Lakes [2]. The PCBs 126 and 169 were magnified more than congener 77, which is also consistent with Great Lakes data [2].

Biomagnification of TCDF was negligible in both double-crested cormorants and bald eagles, as it was in Forster's terns in the Great Lakes [2]. Bald eagle BMFs varied somewhat and depended on which estimates of dietary composition were used. The mean BMFs for DDE, PCBs, and TCDD in cormorants were greater than comparable values calculated for bald eagles. All BMFs calculated for bald eagles and double-crested cormorants in the Columbia River Estuary fall within the range of values calculated for herons from the lower Columbia River. We expected the greatest biomagnification to occur in bald eagles; however, this was not observed and may be partially due to species differences in metabolism of DDE, PCBs, and TCDD.

Effects of contaminants

Eggshell thinning at Bachelor, Fisher, Molalla, and Samish was comparable to that found at sites upriver on the Columbia during 1978 to 1982. Thinning at Ross (14.4%) was greater than that found at any site in the Columbia River in 1978 to 1982 [12,36] and is similar to thinning of heron eggshells (17%) that were broken during incubation [58]. Thinning at Ross also approaches levels (15–20%) associated with long-term population declines of the species [33].

Positive correlations between H4IIE TEQs and TCDD concentrations ($r = 0.87$, $p = 0.02$), the presence of embryo deformities, and positive correlations between nest failure and TCDD concentrations ($r = 0.85$, $p = 0.03$) demonstrated that contaminants were impacting individual herons on the Columbia and Willamette Rivers. The H4IIE bioassay assesses exposure of an organism to 2,3,7,8-TCDD and structurally similar compounds via induction of cytochrome P450-dependent enzymes [25,43]. Deformities were found at four of the six sites at rates similar to or above those reported for double-crested cormorants from Green Bay, Wisconsin, USA, in 1988 [59]. These hard tissue deformities have typically been linked to toxicity of TCDD-like compounds [60,61]. Although many compounds were measured in this study, our list was not exhaustive. Other contaminants such as phthalates may be present in the system and may be partially responsible for the embryo deformities and nest failure we observed.

The colony at Karlson Island invites further study. The bimodal nest success and elevated deformity rate suggest that a source/sink dynamic may be occurring. Birds may be accumulating contaminants from the area annually until they experience low nest success and embryo deformities. Younger breeders from outside the area may be responsible for the nests with higher success. At present, hatch and fledge rates at all of our sites were similar to those calculated for most other colonies in which reproduction was not impaired [12,13,37,62–64]. Therefore, contaminants do not appear to be significantly impairing great blue heron reproduction at the colony level on the Columbia and Willamette Rivers where our study was conducted.

In summary, results from this study supported our first hypothesis that environmental contaminants were elevated in great blue herons from the lower Columbia and Willamette Rivers. Elevated concentrations of DDE and total PCBs were detected at several sites, and contaminant concentrations in some eggs exceeded levels associated with impaired reproduction in other species. Significant among-site differences were detected in concentrations of DDE, total PCBs, and TCDD. The results from this study did not support our second hypothesis, that TCDD concentrations in heron eggs were inversely related to distance of the colony from pulp and paper mills. Our final hypothesis, that concentrations of certain residues were adversely affecting reproductive success of herons, was supported only at the individual level and not at the colony level. Elevated deformity rates and positive correlations between H4IIE TEQs and TCDD concentrations and between nest failure and TCDD concentration demonstrated that contaminants were impacting individual herons on both rivers. However, hatch and fledge rates at the colony level were comparable to rates calculated for other heron colonies in which reproduction was not impaired. This demonstrated a lack of effect of residues on great blue heron colonies from which we sampled. Despite the apparent lack of colony-level effects on herons, elevated concentrations of DDE and total PCBs are present in wildlife from both rivers and may be adversely affecting individual herons and/or more sensitive species.

River hydraulics and contaminant bioavailability

We expected contaminant concentrations to be most similar for two pairs of sites, for Bachelor and Fisher and for Molalla and Ross. These two pairs of sites are located in physically similar areas of the Columbia and Willamette Rivers, respectively, and thus would be subject to similar hydrological conditions. Residues in eggs from Karlson were typically higher than those detected in eggs from Bachelor and Fisher. This difference may be due to the fact that Karlson Island is in the estuary, which has a salinity gradient that affects both the available prey base and contaminant flux through the system. Salinity is an important factor in determining contaminant bioavailability because it influences settling times of suspended sediment particles to which organic compounds typically adsorb. Bachelor and Fisher Islands are located in the fluvial reach of the Columbia, which has low particle retention times and little settling of suspended sediments due to rapid flow velocities and low bed roughness [65]. Suspended sediments from the fluvial reach settle rapidly upon reaching the estuary because it is a transition zone between fresh and saline water [66] and has greater bed roughness [65]. Suspended sediments in the Columbia River settle primarily in the estuary and backwaters [67,68], which are areas in which herons typically feed. Release of organochlorine contaminants from suspended sediments has been documented in both fresh and saline systems in other areas [69–72], and resultant bioaccumulation has been reported in several species of fish [71,72].

This has important implications for dredging operations. Flow velocities and short suspended sediment retention times

in the main channel of the Columbia River above the estuary suggest that dredging in these areas will not increase bioavailability of contaminants to organisms living in the immediate vicinity. However, dredging activities conducted upriver may impact organisms in the upper end of the estuary due to longer retention times of suspended sediments and greater deposition in these areas. Dredging activities within the estuary may increase contaminant availability to organisms in a large area due to complex flows and mixing patterns.

Great blue herons as indicator species

The results of our study and others [12,36] have demonstrated the utility of herons as an indicator species of environmental contamination. Although juvenile herons may feed in grasslands along the west coast, adult females typically feed in estuarine marshes and intertidal beaches [73]. Chemical residues in eggs are therefore most representative of contamination in aquatic ecosystems.

Current contaminant concentrations in the Columbia and Willamette Rivers do not seem to be adversely affecting reproductive success at most of our sites. However, they are affecting individuals, and this is evident in the deformities and TCDD-EQs observed from some eggs. Adverse effects may be expected in species with similar exposure and higher sensitivity. Modeling is necessary to define more precise relationships between residue concentrations in herons and other species for which they would serve as an indicator. Continued monitoring of residue levels in great blue heron eggs would provide valuable information for other piscivorous species such as bald eagles, whose productivity has been depressed in association with high DDE and PCB levels in eggs [1].

Acknowledgement—Many people helped with this project. We thank C. Barclay and E. Sproul for their field assistance. J. Fairchild, R. Frenzel, S. Foster, and K. Hillary climbed most of the trees for egg collection. R. Anthony and F. Ramsey provided statistical advice and D. Buehler provided lab space. This research was supported by the U.S. Fish and Wildlife Service and the Oregon Cooperative Wildlife Research Unit, Oregon State University.

REFERENCES

1. Anthony RG, Garrett MG, Schuler C. 1993. Environmental contaminants in bald eagles in the Columbia River Estuary. *J Wildl Manage* 57:10–19.
2. Kubiak TJ, et al. 1989. Microcontaminants and reproductive impairment of the Forster's tern on Green Bay, Lake Michigan—1983. *Arch Environ Contam Toxicol* 18:706–727.
3. Giesy JP, et al. 1995. Contaminants in fishes from the Great Lakes—Influenced sections and above dams of three Michigan rivers: III. Implications for health of bald eagles. *Arch Environ Contam Toxicol* 29:309–321.
4. U.S. Fish and Wildlife Service. 1998. Changes in productivity and environmental contaminants in bald eagles nesting along the lower Columbia River. Final Draft, July. Portland, OR.
5. U.S. Environmental Protection Agency. 1992. National study of chemical residues in fish. EPA 823-R-92-008. Washington, DC.
6. Curtis LR, et al. 1993. Sensitivity of cytochrome P450-1A1 induction in fish as a biomarker for distribution of TCDD and TCDF in the Willamette River, Oregon. *Environ Sci Technol* 27:2149–2157.
7. Custer TW, Rattner BA, Ohlendorf HM, Melancon MJ. 1991. Herons and egrets as proposed indicators of estuarine contamination in the United States. *Acta XX Congressus Internationalis Ornithologici* 20:2474–2479.
8. Moore NW. 1966. A pesticide monitoring system with special reference to the selection of indicator species. *J Appl Ecol* 3:261–270.
9. Thomas CM. 1997. Environmental contaminants and breeding biology of great blue herons in the Columbia River Basin. MS thesis. Oregon State University, Corvallis, OR, USA.
10. Cobb GP, Norman DM, Kendall RJ. 1994. Organochlorine contaminant assessment in great blue herons using traditional and nonlethal monitoring techniques. *Environ Pollut* 83:299–309.
11. Cobb GP, Norman DM, MW Miller DM, Brewer LW. 1995. Chlorinated contaminants in chorio-allantoic membranes from great blue heron eggs at Whidbey Island naval air station. *Chemosphere* 30:151–164.
12. Blus LJ, Henny CJ, Kaiser TE. 1980. Pollution ecology of breeding great blue herons in the Columbia Basin, Oregon and Washington. *Murrelet* 61:63–71.
13. Elliott JE, Butler RW, Norstrom RJ, Whitehead PE. 1989. Environmental contaminants and reproductive success of great blue herons *Ardea herodias* in British Columbia, 1986–87. *Environ Pollut* 59:91–114.
14. Ratcliffe DA. 1970. Changes attributable to pesticides in egg breakage frequency and eggshell thickness in some British birds. *J Appl Ecol* 7:67–113.
15. Wade TL, Atlas EL, Brooks JM, Kennicutt MC II, Fox RG, Sericano J, Garcia B, DeFreitas D. 1988. NOAA Gulf of Mexico Status and Trends Program: Trace organic contaminant distribution in sediments and oyster. *Estuaries* 11:171–179.
16. U.S. Environmental Protection Agency. 1990. Tetra- through octa-chlorinated dioxins and furans by isotope dilution HRGC/HRMS. Revised April 1990. Method 1613. Office of Water Regulation and Standards, Washington, DC.
17. U.S. Environmental Protection Agency. 1983. Interim method for the sampling and analysis of priority pollutants in sediments and fish tissue. In *Methods for Chemical Analysis of Water and Wastes*. EPA/600/4-79-020. Environmental Monitoring and Support Laboratory, Cincinnati, OH.
18. Hatch WR, Ott WL. 1968. Determination of sub-microgram quantities of mercury in solution by a flameless atomic absorption technique. *Anal Chem* 40:2085–2087.
19. Texas A&M University. 1994. Digestion of biological materials for trace metals analysis. Standard Operating Procedure 9408. Geochemical and Environmental Research, College Station, TX, USA.
20. Schmitt CJ, Zajicek JL, Peterman PH. 1990. National contaminant biomonitoring program: Residue of organochlorine chemicals in freshwater fish, 1976–1984. *Arch Environ Contam Toxicol* 19: 748–781.
21. Schwartz TR, Tillitt DE, Feltz KP, Peterman PH. 1993. Determination of mono- and non-*o,o'*-chlorine substituted polychlorinated biphenyls in arochlors and environmental samples. *Chemosphere* 26:1443–1460.
22. Peterman PH, Gale RW, Tillitt DE, Feltz KP. 1996. Analysis of non-*ortho*-PCBs in fish, bird eggs, sediments, soils, and SPMD samples by gas chromatography/high resolution mass spectrometry. In Ostrander GK, ed, *Techniques in Aquatic Toxicology*. CRC, Boca Raton, FL, USA, pp 517–553.
23. Peterman PH. 1995. Gas chromatography/high resolution mass spectrometry for the determination of tetra- through octa-substituted polychlorinated dibenzo-*p*-dioxins and dibenzofurans. Standard Operating Procedure C5.183. U.S. Geological Survey, Columbia, MO.
24. Stonehouse B. 1963. Egg volumes from linear dimensions. *Emu* 65:227–228.
25. Tillitt DE, Giesy JP, Ankley GT. 1991a. Characterization of the H4IIE rat hepatoma cell bioassay as a tool for assessing toxic potency of planar halogenated hydrocarbons (PHHs) in environmental samples. *Environ Sci Technol* 25:87–92.
26. Tysklind M, Tillitt D, Eriksson L, Lundgren K, Rappe C. 1994. A toxic equivalency factor scale for polychlorinated dibenzofurans. *Fundam Appl Toxicol* 22:277–285.
27. Ankley GT, Tillitt DE, Giesy JP, Jones PD, Verbrugge DA. 1991. Bioassay-derived 2,3,7,8-tetrachlorodibenzo-*p*-dioxin equivalents in the flesh and eggs of Lake Michigan chinook salmon and possible implications for reproduction. *Can J Fish Aquat Sci* 48: 1685–1690.
28. Pratt HM, Winkler DW. 1985. Clutch size, timing of laying, and reproductive success in a colony of great blue herons and great egrets. *Auk* 102:49–63.
29. Safe S. 1990. Plychlorinated biphenyls (PCBs), dibenzo-*p*-dioxins (PCDDs), dibenzofurans (PDCFs), and related compounds: Environmental and mechanistic considerations which support the development of toxic equivalency factors. *Crit Rev Toxicol* 21: 51–88.

30. Bosveld BATC, Gradener J, Murk AJ, Brouwer A, van Kampen M, Evers EHG, van den Berg M. 1995. Effects of PCDDs, PCDFs, and PCBs in common tern (*Sterna hirundo*) breeding in estuarine and coastal colonies in The Netherlands and Belgium. *Environ Toxicol Chem* 14:99–115.
31. Tetra Tech. 1996. Assessing human health risks from chemically contaminated fish in the lower Columbia River. Final Report. TC 9968-05. Tetra Tech, Redmond, WA, USA.
32. Watson JW, Garrett MG, Anthony RG. 1991. Foraging ecology of bald eagles in the Columbia River Estuary. *J Wildl Manage* 55:492–499.
33. Anderson DW, Hickey JJ. 1972. Eggshell changes in certain North American birds. *Proc Int Ornithol Cong* 15:514–540.
34. Alborg U, et al. 1992. Impact of polychlorinated dibenzo-*p*-dioxins, dibenzofurans, and biphenyls on human and environmental health, with special emphasis on application of the toxic equivalency factor concept. *Eur J Pharmacol* 228:179–199.
35. Henny CJ, Blus LJ, Krynitsky AJ, Bunck CM. 1984. Current impact of DDE on black-crowned night herons in the intermountain west. *J Wildl Manage* 48:1–13.
36. Fitzner RE, Blus LJ, Henny CJ, Carlile DW. 1988. Organochlorine residues in great blue herons in the northwestern United States. *Colon Waterbirds* 11:293–300.
37. English SM. 1978. Distribution and ecology of great blue heron colonies in the Willamette River, Oregon. In Sprunt A, Winckler O, eds, *Wading Birds*. National Audubon Society, New York, NY, USA, pp 235–244.
38. Vermeer K, Reynolds LM. 1970. Organochlorine residues in aquatic birds in the Canadian Prairie Provinces. *Can Field Nat* 84:117–129.
39. Henny DJ, Blus LJ, Stafford DJ. 1983. Effects of heptachlor on American kestrels in the Columbia Basin, Oregon. *J Wildl Manage* 47:1080–1087.
40. Hoffman DJ, Rattner BA, Bunck CM, Krynitsky A, Ohlendorf HM, Lowe RW. 1986. Association between PCBs and lower embryonic weight in black-crowned night herons in San Francisco Bay. *J Toxicol Environ Health* 19:383–391.
41. Tumasonis CF, Bush B, Baker FD. 1973. PCB levels in egg yolks associated with embryonic mortality and deformity of hatched chicks. *Arch Environ Contam Toxicol* 1:312–324.
42. Cecil HC, Harris SJ, Bitman J, Fries GF. 1973. Polychlorinated biphenyl-induced decrease in liver vitamin A in Japanese quail and rats. *Bull Environ Contam Toxicol* 9:179–185.
43. Bellward GD, et al. 1990. Comparison of polychlorinated dibenzodioxin levels with hepatic mixed-function oxidase induction in great blue herons. *J Toxicol Environ Health* 30:33–52.
44. Hart LE, et al. 1991. Dioxin contamination and growth and development in great blue heron embryos. *J Toxicol Environ Health* 32:331–344.
45. Sanderson JT, Elliott JE, Norstrom RJ, Whitehead PE, Hart LE, Cheng KM, Bellward GD. 1994. Monitoring biological effects of polychlorinated dibenzo-*p*-dioxins, dibenzofurans, and biphenyls in great blue heron chicks (*Ardea herodias*) in British Columbia. *J Toxicol Environ Health* 41:435–450.
46. Blus LJ, Henny CJ, Anderson A, Fitzner RE. 1985. Reproduction, mortality, and heavy metal concentrations in great blue herons from three colonies in Washington and Idaho. *Colon Waterbirds* 8:110–116.
47. Eisler R. 1987. Mercury hazards to fish, wildlife, and invertebrates: A synoptic review. Biological Report 85(1.10). U.S. Fish and Wildlife Service, Washington, DC.
48. Bosveld BATC, van den Berg M, Theelen RMC. 1992. Assessment of the EROD inducing potency of eleven 2,3,7,8-substituted PCDD/Fs and three coplanar PCBs in the chick embryo. *Chemosphere* 25:911–916.
49. Tillitt DE, Ankley GT, Verbrugge DA, Giesy JP, Ludwig JP, Kubiak TJ. 1991. H4IIE rat hepatoma cell bioassay-derived 2,3,7,8-tetrachlorodibenzo-*p*-dioxin equivalents in colonial fish-eating waterbird eggs from the Great Lakes. *Arch Environ Contam Toxicol* 21:91–101.
50. Newell AJ, Johnson DW, Allen LK. 1987. Niagara River biota contamination project: Fish flesh criteria for piscivorous wildlife. Technical Report 87-3. New York State Department of Environmental Conservation, Albany, NY, USA.
51. Nagpal NK. 1992. Water quality criteria for polychlorinated biphenyls (PCBs). Ministry of the Environment, Lands and Parks, Victoria, BC, Canada.
52. U.S. Environmental Protection Agency. 1986. Quality criteria for water 1986. EPA 440/586001. Washington, DC.
53. Braune BM, Norstrom RJ. 1989. Dynamics of organochlorine compounds in herring gulls: III. Tissue distribution and bioaccumulation in Lake Ontario gulls. *Environ Toxicol Chem* 8:957–968.
54. Zitko V, Choi PMK. 1972. PCB and *p,p'*-DDE in eggs of cormorants, gulls, and ducks from the Bay of Fundy, Canada. *Bull Environ Contam Toxicol* 7:63–4.
55. Walker CH, Craven ACC, Kurukgy M. 1975. The metabolism of organochlorine compounds by microsomal enzymes of the shag (*Phalacrocorax aristotelis*). *Environ Physiol Biochem* 5:58–64.
56. Anderson DW, Hickey JJ. 1976. Dynamics of storage of organochlorine pollutants in herring gulls. *Environ Pollut* 10:183–200.
57. Norstrom RJ, Hallett DJ, Sonstegard RA. 1978. Coho salmon (*Oncorhynchus kisutch*) and herring gulls (*Larus argentatus*) as indicators of organochlorine contamination in Lake Ontario. *J Fish Res Board Can* 35:217–228.
58. Faber RA, Risebrough RW, Pratt HM. 1972. Organochlorines and mercury in common egrets and great blue herons. *Environ Pollut* 3:111–122.
59. Yamashita N, Tanabe S, Ludwig JP, Kurita H, Ludwig ME, Tatsukawa R. 1993. Embryonic abnormalities and organochlorine contamination in double-crested cormorants (*Phalcrocorax auritus*) and caspian terns (*Hydroprogne caspia*) from the upper Great Lakes in 1988. *Environ Pollut* 79:163–173.
60. Brunstrom B, Andersson L. 1988. Toxicity and 7-ethoxyresorufin-*O*-deethylase-inducing potency of coplanar polychlorinated biphenyls (PCBs) in chick embryos. *Arch Toxicol* 62:263–266.
61. Brunstrom B. 1990. Mono-*ortho*-chlorinated chlorobiphenyls: Toxicity and induction of 7-ethoxyresorufin-*O*-deethylase (EROD) activity in chick embryos. *Arch Toxicol Chem* 64:188–192.
62. Henny CJ, Bethers MR. 1971. Population ecology of the great blue heron with special reference to western Oregon. *Can Field Nat* 85:205–209.
63. Werschkul D, McMahon E, Leitschuh M, English S, Skibinski C, Williamson G. 1977. Observations on the reproductive ecology of the great blue heron (*Ardea herodias*) in western Oregon. *Murrelet* 58:7–12.
64. Sullivan JP, Payne SM. 1988. Aspects of history and nestling mortality at a great blue heron, *Ardea herodias*, colony, Quetico Provincial Park, Ontario. *Can Field Nat* 102:237–241.
65. Tetra Tech. 1992. Reconnaissance survey of the lower Columbia River. Task 3: Review of hydraulic, hydrologic, sediment transport, and geomorphic characteristics of the lower Columbia River. Final Report. TC-8526-03. Tetra Tech, Redmond, WA, USA.
66. DiGiano FA, Miller CT, Yoon J. 1993. Predicting release of PCBs at point of dredging. *J Environ Eng* 119:72–89.
67. Hubbell DW, Glenn JL. 1973. Distribution of radionuclides in bottom sediments of the Columbia River Estuary. Professional Paper 433-L. U.S. Geological Survey, Washington, DC.
68. Simenstad D, Jay D, McIntire CD, Nehlsen W, Sherwood C, Small L. 1984. The dynamics of the Columbia River estuarine ecosystem. Columbia River Estuary Data Development Program, Portland, OR, USA.
69. Hafferty AJ, Pavlou SP, Hom W. 1977. Release of polychlorinated biphenyls (PCBs) in a salt-wedge estuary as induced by dredging of contaminated sediments. *Sci Total Environ* 8:229–239.
70. Halter MT, Johnson HE. 1977. A model system to study the desorption and biological availability of PCB in hydrosoils. In Mayer FL, Hamelink JL, eds, *Aquatic Toxicology and Hazard Evaluation*. STP 634. American Society for Testing and Materials, Philadelphia, PA, pp 178–195.
71. Seelye JG, Hesselberg RJ, Mac MJ. 1982. Accumulation by fish of contaminants released from dredged sediments. *Environ Sci Technol* 16:459–464.
72. Rice CP, White DS. 1986. PCB availability assessment of river dredging using caged clams and fish. *Environ Toxicol Chem* 6: 259–274.
73. Butler RW. 1991. Habitat selection and time of breeding in the great blue heron (*Ardea herodias*). PhD thesis. University of British Columbia, Vancouver, Canada.