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Contamination by chlorinated hydrocarbons and lead in Steller's Sea Eagle and White-tailed Sea Eagle from Hokkaido, Japan

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Abstract. Chronic exposure to man-made chemicals, particularly chlorinated hydrocarbons, in raptors has been associated with reproductive impairment and population declines. In addition, incidents of sub-lethal and lethal lead poisoning in raptors through ingestion of spent gunshot have been reported. However, little information is available on the contaminant levels of Steller's Sea Eagle (SSE: *Haliaeetus pelagicus*) and White-tailed Sea Eagle (WSE: *H. albicilla*) from Hokkaido, Japan. The objective of this study was to determine the levels of toxic contaminants including polychlorinated biphenyls (PCBs), organochlorine pesticides, and lead in sea eagles wintering in Hokkaido, and to evaluate the ecotoxicological risk based on their concentrations. SSEs and WSEs which were found dead or debilitated and subsequently died in Hokkaido from 1986 to 1998 were analysed.

All eagles contained detectable amounts of PCBs, DDTs, hexachlorocyclohexane isomers, chlordane related compounds, and hexachlorobenzene. The highest concentrations of PCBs and DDTs in breast muscles were 18,000 and 17,000 ng/g (wet weight), respectively. Furthermore, non-*ortho* and mono-*ortho* substituted coplanar PCB congeners were also detected, leading to 60-540 pg/g of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin toxic equivalents derived from WHO toxic equivalence factors in the breast muscles. Our previous survey of contaminants in the Far East showed that atmospheric concentrations of PCBs and DDTs in Khabarovsk and Magadan were much higher than those in Hokkaido and North Pacific, suggesting the potential that some east Siberian cities might be a source of contamination found in eagles. Residue levels of PCBs and DDTs in breast muscles of SSEs were comparable to those of raptors collected after the 1980s from North America. Risk assessments based on the residue levels of coplanar PCB congeners and *p,p'*-DDE detected in these eagles indicated their potential of hepatic cytochrome P450 1A induction and eggshell thinning.

High lead concentrations of over 10,000 ng/g (dry weight) were detected in the livers of some SSEs and WSEs. Three of six dead or moribund SSEs (50%) and three of three WSEs (100%) with livers analyzed for lead died from lead poisoning. Five of the six lead poisoned eagles had lead bullet fragments in their gizzards. The hair of deer was found along with a lead fragment in the intestine of

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a SSE. In Hokkaido, whole and partial carcasses of deer shot by hunters are left in the field. These are potential sources of lead fragments that are ingested by scavenging raptors during and after the hunting season. Therefore, secondary poisoning by the lead fragment embedded in the tissue of deer is implicated as the main cause of death. Mercury and cadmium concentrations in the tissue of these species were low, implying that the toxicities of these elements were negligible.

INTRODUCTION

Chlorinated hydrocarbons (CHCs) are ubiquitous contaminants detected in various environmental compartments on a global scale (Iwata *et al*. 1993), and are notably bioaccumulated in higher trophic organisms through the food web (Muir *et al*. 1988, Tanabe 1988). Contamination of marine organisms by CHCs, such as polychlorinated biphenyl (PCB) and DDT, continues to be an issue of growing concern. Concerns are based on the implication that CHCs can cause a variety of adverse effects including teratogenicity, reproductive failure, and endocrine disruption in higher trophic animals such as raptors and fish-eating waterbirds (Giesy *et al*. 1994).

Lead poisoning of waterfowl through the ingestion of spent lead shot has also been reported in various areas of the world (Anderson 1975, Guitart *et al*. 1994). In addition, it is known that raptors have been exposed to high levels of lead through secondary ingestion of lead shot embedded in the tissue of waterfowl and other prey. The widespread mortality of raptors by secondary lead poisoning, especially Bald Eagle *Haliaeetus leucocephalus* and some hawks that feed on dead or wounded waterfowl, has been documented in the United States, Canada and Europe (Reichel *et al*. 1984, Elliott *et al*. 1992, Pain & Amiard-Triquet 1993). In these countries, concern over lead poisoning led, in the 1980's, to the progressive prohibition of the use of lead shot to harvest waterfowl. In Japan, no such restriction has been introduced.

Steller's Sea Eagle (SSE: *H. pelagicus*) and White-tailed Sea Eagle (WSE: *H. albicilla*) are classified as "vulnerable" and "endangered", respectively in the Red Data Book of the Environment Agency of Japan. SSE breeds on large rivers and the coast in northeast Siberia-Kamchatka and winters to northern Japan, Kamchatka and south of Korea (Nakagawa *et al*. 1987). WSE in the Far East is distributed in Kamchatka, Okhotsk, Sakhalin and Hokkaido and winters to Japan and Korea (Knystautas 1993). Hokkaido is a critical habitat area for both species.

Little information is available on the contaminant levels of CHCs and lead in these sea eagles. The objective of this study was to determine the levels of CHCs and toxic metals including lead, mercury and cadmium in SSE and WSE migrating to Hokkaido, Japan, and to evaluate the ecotoxicological risk based on their concentrations. This paper summarizes data previously reported in several papers by our group (Iwata *et al*. 1997, Kim *et al*. 1999, Yasunaga *et al*. 2000) and includes some additional data.

Table 1. Details of Steller's Sea Eagles (SSEs) and White-tailed Sea Eagles (WSEs) analyzed.

Sample No.	Species	Collection date	Body weight (kg)	sex
1	SSE	Apr. 1995	4.3	♀
2	SSE	Feb. 1994	4.2	♂
3	SSE	Dec. 1986	6.0	♀
4	SSE	Feb. 1997	3.7	♀
5	SSE	Jan. 1998	4.6	♀
6	SSE	Feb. 1998	4.2	?
7	SSE	Mar. 1998	3.8	♂
8	WSE	Jan. 1997	4.7	♀
9	WSE	Dec. 1997	3.7	♀
10	WSE	Feb. 1998	3.8	♀

MATERIALS AND METHODS

Samples

Seven SSEs and three WSEs were collected in eastern Hokkaido, within the main area where deer hunting was permitted from 1986 to 1998 (Table 1). Two SSEs (sample nos. 1 and 3) and one WSE (no. 8) were found dead. One SSE (no. 2), with an external injury on the left wing, was captured, but died on the same day. The other eagles were found in a debilitated condition, captured, but subsequently died. From the intestine or gizzard of two SSEs (nos. 4 and 7) and all WSEs (nos. 8-10), lead bullet fragments were found. Deer hair along with a metallic fragment was also found in the intestine of a SSE (no. 4). In the case of carcass no.8 (WSE), one fragment was collected from the gizzard, two fragments were embedded in bone, one in fat around the abdominal membrane, and seven in muscle. The injuries around the fragments embedded in body tissues had healed, indicating this bird had survived being shot. The birds in whose alimentary canal lead fragments were found showed clinical signs of lead poisoning including hypertrophy of the gall bladder and green feces.

Carcasses were weighed, a clinical diagnosis was made, and liver, kidney and breast muscle were removed and stored at -20°C until analysis.

Chemical analysis

The analysis of CHCs, including hexachlorobenzene (HCB), hexachlorocyclohexane (HCH: α and β isomers), chlordane compounds (CHLs: *cis*-chlordane, *trans*-chlordane, *cis*-nonachlor, trans-nonachlor, and oxychlordane), and DDTs (*p,p'*-DDE, *p,p'*-DDD, and *p,p'*-DDT) was performed following the procedure described by Tanabe *et al*. (1984). Briefly, the analytical method involved Soxhlet extraction of homogenized fat samples (5 g) with a mixture of diethyl-ether and hexane (3 : 1, 400 ml), followed by elution through a florisil packed glass column with 150 ml of 20% hexane-washed water in acetonitorile, for the removal of fat. The eluent was collected in a separation funnel containing 100 ml hexane and 600 ml hexane-washed water. Subsequently, the concentrated hexane layer was cleaned and fractionated

with a 12 g florisil packed glass column. The first fraction eluted with hexane contained HCB, PCBs, *p,p'*-DDE and *trans*-nonachlor, while the second fraction, eluted with 20% dichloromethane in hexane, was comprised of other organochlorine pesticides. Each fraction was concentrated and the extracts were subjected to a further clean-up with a 5% mixture of fuming H_2SO_4 in concentrated H_2SO_4.

The extraction and separation of individual PCB congeners including non-*ortho* PCBs (3,3',4,4'-TeCB, 3,3',4,4',5-PeCB and 3,3',4,4',5,5'-HxCB) and mono-*ortho* PCBs (2,3,3',4,4'-PeCB, 2,3',4,4',5-PeCB and 2,3,3',4,4',5-HxCB) followed the KOH-digestion method by Wakimoto *et al*. (1971), and the activated carbon column method by Tanabe *et al*. (1987), respectively.

The quantification of organochlorine pesticides were made on a gas chromatogragh (Hewlett-Packard 5890) equipped with a 63Ni electron capture detector and moving needle-type injection port. Analysis of the PCB congeners was made using the gas chromatogragh coupled with mass spectrometry (Hewlett-Packard 5972). A DB-1 capillary column (30 m x 0.25 mm i.d., film thickness of 0.25 micro meter, J&W Scientific, CA, USA) was employed for the separation of individual compounds.

An equivalent mixture of Kanechlor preparations (KC-300, KC-400, KC-500 and KC-600) with known PCB composition and content was used as a standard. Concentrations of individually resolved peaks of PCB isomers and congeners were summed to obtain total PCB concentration. Organochlorine pesticides and coplanar PCB congeners were quantified from individually resolved peak areas with the corresponding peak areas of authentic standards. Concentrations of CHCs were given on wet weight basis.

For metal analyses, the method has been reported in detail elsewhere (Kim *et al*. 1999; Yasunaga *et al*. 1999). Three subsamples of each tissue were analyzed to avoid local tissue contamination by lead-shot damage. Subsamples were dried to a constant weight in an oven at 80°C for 12hrs. Analyses for lead and cadmium were performed by inductively coupled plasma-mass spectrometer (ICP-MS; Perkin Elmer ELAN 5000), after microwave digestion with nitric acid in a PTFE (Teflon) vessel (Okamoto 1994). A mixed multi-element standard solution was prepared from 10 micro g/g stock solution. NIST SRM 1577b bovine liver was used for the determination of recoveries of these two elements. Recoveries of lead and cadmium were in the range of 97-116% and 95-105% of the certified values, respectively. Total mercury levels were determined by cold vapor technique using Sansou Automatic Mercury Analyzer Model MERCURY-3000 spectra-photometer and Shimadzu AA-680 after microwave digestion with nitric acid (Akagi & Nishimura 1991). Analytical quality assurance was conducted using a standard reference material, NIES No. 1 (Okamoto *et al*. 1978). Recovery of mercury was 98% of the certified value. Concentrations of all the elements were given on dry weight basis.

RESULTS AND DISCUSSION

CHC levels and source allocation

Table 2 shows levels of CHCs in SSE and WSE. All eagles were contaminated by detectable amounts of CHCs. No differences in CHC accumulation were observed between SSE and WSE. Among the CHCs analyzed, the concentrations of PCBs and DDT were relatively higher than those of other compounds. The highest concentrations of PCBs reached 18 and 41 micro g/g (wet weight) in breast muscle and liver, respectively. The highest total DDT concentrations were 17 micro g/g in the breast muscle and 15 micro g/g in the liver. These concentrations were higher than those in most other bird species in Japan such as Common Cormorants *Phalacrocorax carbo*, Black-crowned Night Heron *Nycticorax nycticorax*, and Mountain Hawk-eagle *Spizaetus nipalensis* reported by Hoshi *et al*. (1998). One reason for the elevated residue levels found in these eagles may be the relatively higher trophic level to which they belong. Sea eagles mainly feed on fish, including Walleye Pollock and salmon, and occasionally stranded marine mammals. Some components of the sea eagle's diet are likely to contribute to the high contaminant levels observed.

Furthermore, three non-*ortho* (3,3',4,4'-TeCB, 3,3',4,4',5-PeCB, 3,3',4,4',5,5'-HxCB) and three mono-*ortho* (2,3,3',4,4'-PeCB, 2,3',4,4',5-PeCB, 2,3,3',4,4',5-HxCB) substituted coplanar PCB congeners were also detected in significant amounts (Table 2). Concentrations of mono-*ortho* coplanar PCBs were greater than non-*ortho* congeners in all the samples analyzed. For the non-*ortho* PCBs, the levels of 3,3',4,4'-TeCB and 3,3',4,4',5-PeCB varied widely among samples, while 3,3',4,4',5,5'-HxCB was consistently lowest. As for the mono-*ortho* PCBs, in most samples the pattern was 2,3',4,4',5-PeCB > 2,3,3',4,4'-PeCB > 2,3,3',4,4',5-HxCB. The 2,3,7,8-tetrachlorodibenzo-*p*-dioxin toxic equivalents (TEQs) calculated with WHO toxic equivalence factors (Ahlborg *et al*., 1994) ranged from 60-540 pg TEQ/g (wet weight) in the breast muscle and 36-2100 pgTEQ/g in the liver. In the coplanar PCB congeners, 3,3',4,4',5-PeCB made a greater contribution to total TEQs, and followed by 2,3',4,4',5-PeCB.

A survey of CHC contamination in the Far East including the area used by these eagles for breeding and wintering showed that atmospheric CHC levels in Khabarovsk and Magadan were much higher than those in Hokkaido and the North Pacific Ocean (Iwata *et al*. 1995). In air samples from Khabarovsk where elevated PCB levels were recorded, congeners with four or five chlorines were detected at higher levels. This profile was very similar to that of the PCB product, Sovol, that was manufactured in the former Soviet Union. In contrast, air samples from Chaun exhibited the lowest concentration of PCBs, and contained chlorinated congeners with two or three chlorines that are known to be preferentially transported by air to remote areas. These results suggest a potential for Khabarovsk and Magadan to be CHC contamination sources for eagles that breed in these areas, and others that migrate through but feed during passage. Recent studies of migration routes of SSE and WSE by satellite tracking confirms this risk of exposures. SSE tracked from wintering grounds in Hokkaido migrate to

Table 2. Concentrations of CHCs (ng/g on wet weight basis) and metals (micro g/g on dry weight basis) in tissues of SSEs and WSEs.

Sample Nos.	1			2	3			4		5		6			7			8		9	10		
Species	SSE			SSE	SSE			SSE		SSE		SSE			SSE			WSE		WSE	WSE		
Tissue	BM	L	K	BM	BM	L	K	L	K	L	K	BM	L	K	BM	L	K	L	K	L	BM	L	K
lipid content (%)	8.7	4.5		0.91	6.7	6.5		3.5		3.7		0.94			4.3			5.8		4.4	5.2		
HCB	31	17		85	50	44		110		23		18			29			9.6		9.7	11		
HCHs																							
α-HCH	0.52	< 0.2		2.5	1.8	1.6		1.6		0.47		1.2			0.55			0.21		0.70	0.37		
β-HCH	73	36		450	310	250		400		44		79			64			43		72	25		
DDTs																							
p,p' -DDE	2200	580		17000	3100	2800		15000		390		850			1800			2500		590	1200		
p,p' -DDD	92	31		190	310	230		300		65		84			72			26		38	53		
p,p' -DDT	13	2.5		34	23	9.7		23		2.9		15			12			1.7		3.7	<1.5		
CHLs																							
oxychlordane	110	39		790	100	55		380		18		61			87			26		17	28		
cis -chlordane	29	10		44	72	49		79		16		20			29			7.7		16	17		
trans -chlordane	5.3	< 0.9		7.2	13	6.6		7.3		<0.5		<0.5			<0.5			0.92		<0.5	<0.5		
cis -nonachlor	27	8.8		160	47	34		180		15		22			49			19		20	22		
trans -nonachlor	190	56		2000	310	230		1600		65		130			430			82		82	90		
PCBs																							
non-*ortho* coplanar PCBs																							
3,3',4,4'-TeCB	0.40	0.14		1.0	3.7	3.1		5.0		0.34		0.37			0.71			0.98		0.54	0.68		
3,3',4,4',5-PeCB	0.63	0.23		2.3	1.7	1.5		11		0.23		0.31			1.1			1.1		0.42	0.61		
3,3',4,4',5,5'-HxCB	0.19	0.06		0.55	0.24	0.23		3.8		0.072		0.091			0.37			0.39		0.093	0.20		
mono-*ortho* coplanar PCBs																							
2,3,3',4,4'-PeCB	51	25		420	140	160		1500		24		44			51			130		0.40	49		
2,3',4,4',5-PeCB	210	86		1700	380	470		4700		70		140			150			500		150	170		
2,3,3',4,4',5-HxCB	26	14		180	37	60		710		4.7		18			20			110		15	20		
TEQ (WHO)	0.10	0.042		0.54	0.24	0.25		2.1		0.036		0.060			0.14			0.23		0.066	0.095		
Total PCBs	2100	820		18000	4300	5000		41000		820		2400			1900			6000		1600	1800		
Metals																							
Pb	4.0	75	102	0.04	0.07	0.06	0.06	139	67	0.11	0.11	0.06	0.21	1.1	4.0	232	103	79	58	77	9.3	174	141
Cd	< 0.05	1.3	5.1	0.16	< 0.05	0.18	1.0	0.61	7.9	0.15	1.3	< 0.001	1.0	1.3	0.033	0.61	3.6	0.67	6.5	0.26	0.015	0.26	2.3
Hg	0.68	2.8	5.5	4.6	0.83	4.2	7.6	7.0	15	5.0	13	2.7	16	12	1.8	4.9	8.4	2.6	5.4	2.0	1.4	3.7	19

BM: breast muscle, L: liver, K: kidney

northern Sakhalin and then to nearby continental seaboards (Amur-Shantarskiye Islands) in spring (Ueta *et al*. 2000). Also juvenile SSE fledged in Magadan wintered in Hokkaido (McGrady *et al*. 2000), and WSE satellite tracked in spring migrate from Hokkaido to Sakhalin, then along the coast of the Ohotsk Sea, and spend the summer in northern Kamchatka (Ueta *et al*. 1998).

Table 3 compares levels of CHCs in SSE and WSE with those in eagles from other locations. The concentrations in breast muscle and liver of SSE and WSE appear to be comparable to those of Bald Eagle from Lake Superior and British Columbia, and residue levels in breast muscles of SSE and WSE from Hokkaido were equal to or less than those in European WSE collected from the Baltic Sea and north western Poland.

Toxicological implications by CHCs

Avian egg shell thinning is thought to be influenced mainly by exposure to *p,p'*-DDT metabolites, *p,p'*-DDE. Although we could not measure thickness of SSE and WSE egg shell, degree of egg shell thinning was assessed from *p,p'*-DDE concentrations in breast muscle using the negative relationships between egg *p,p*'-DDE levels and shell thickness observed in three raptors, Cooper's Hawk *Accipiter striatus*, Sharp-shinned Hawk *A. cooperii*, and Goshawk *A. gentilis*, by Elliott *et al*. (1994). On the assumptions that *p,p'*-DDE level in breast muscle reflects the residue level in egg and there is no differences in the regression slopes of eggshell thickness on *p,p'*-DDE exposure between the three hawk species and the sea eagles, reductions in shell thickness of 2.8-20% were anticipated. In addition, *p,p'*-DDE concentrations in breast muscle from eagle no.2 and liver from eagle no.4 were greater than the no-observable-adverse-effect concentrations (3.5 micro g/g) for Bald Eagle proposed by Wiemeyer *et al*. (1984) and Bowerman *et al*. (1995).

Coplanar PCB congeners are structural analogues with polychlorinated dibenzo-*p*-dioxins, which are known to produce a broad spectrum of biological and toxicological responses including hepatic cytochrome P450 1A (CYP 1A) induction in animals. The responses to both compounds are mediated through an aryl hydrocarbon receptor. Consistent structure-activity relationships have been found for such responses among dioxin and coplanar PCB congeners. Therefore, the toxic potencies of each congener have been evaluated as toxic equivalence factor given to the individual compounds, which have been used to calculate TEQ in environmental samples containing a complex mixture of congeners. In the breast muscles of two SSE (nos. 2 and 3) and the livers of two SSE (nos. 3 and 4) and one WSE (no. 8) were 1.1-10 times greater than a lowest-observable-effect level (210 pg TEQ/g) for CYP1A induction in bald eagle chick liver (Elliott *et al*. 1996b). TEQ values suggests possible hepatic CYP1A induction in some eagles in this study, if assumptions are correct that there are no differences in TEQs between adult and chick, and no differences in responses to CYP1A induction among the eagle species.

In a review of the effects of TEQs on birds by Giesy *et al*. (1994), the lowest-observable-

Table 3. Comparison of CHC concentartions (ng/g on wet weight basis) in the tissues of eagles.

Nation	Japan	Japan	USA	Poland	Finland	Finland	Finland	Japan	Japan	Canada	USA	Canada	Finland	Finland	Finland
Location	Hokkaido	Hokkaido	L. Superior	North Western	Baltic Sea	Baltic Sea	Baltic Sea	Hokkaido	Hokkaido	BC	Columbia R.	BC	Baltic Sea	Lapland	Baltic Sea
Species	SSE	WSE	BE	WSE	WSE	WSE	WSE	SSE	WSE	BE	BE	BE	WSE	WSE	WSE
Tissue	BM	BM	BM	BM	BM	BM	BM	L	L	L	Egg	Egg	Egg	Egg	Egg
HCB	18-85	11	20-40		29-210	57-1350	38-596	17-110	9.6-9.7	6-89	<10-500	40-220	311	70	51-166
HCHs															
α-HCH	0.52-2.5	0.37			<5.1	<3		<0.2-1.6	0.21-0.70				<3	<3	
β-HCH	64-450	25						36-400	43-72	1-308		66-330			
DDTs															
p,p' -DDE	850-17000	1200	120-6600		10000-67000	470-32100	3518-27500	390-15000	590-2500	56-26800	4000-20000	6900-36000	8800	910	5210-9000
p,p' -DDD	72-310	53			530-1800	58-1880	48-721	31-300	26-38		300-2600		312	53	60-175
p,p' -DDT	12-34	<1.5			<10	<3		2.5-23	1.7				<3	<3	
CHLs															
oxychlordane	61-790	28			310-1200	43-1440	76-1500	18-380	17-26	4-303		71-380	169	24	63-313
cis -chlordane	20-72	17	200		79-310	<3-126		10-79	7.7-16				138	11	
trans -chlordane	<0.5-13	<0.5			11-24	8-407		<0.9-7.3	0.92				6	<3	
cis -nonachlor	22-160	22	70					8.8-180	19-20						
trans -nonachlor	130-2000	90			430-990	19-1130	255-1285	56-1600	82	23-1610		390-3500	331	19	108-416
PCBs															
non-*ortho* coplanar PCBs															
3,3',4,4'-TeCB	0.37-3.7	0.68		2.9-140	1.7-2.2	9.8-71	0.49-3.8	0.14-5.0	0.54-0.98	0.248-2.3		2.0-12	21	9.8	0.82-1.1
3,3',4,4',5-PeCB	0.31-2.3	0.61		1.2-160	4.9-44	0.97-51	3.3-32	0.23-11	0.42-1.1	0.349-9.96		3.3-13	20.6	0.95	5.2-8.9
3,3',4,4',5,5'-HxCB	0.091-0.55	0.20		0.15-38	1.4-9.3	<0.2-20	0.77-6.4	0.06-3.8	0.093-0.39	0.056-2.64		0.55-2.1	6	<0.2	1.1-2.2
mono-*ortho* coplanar PCBs															
2,3,3',4,4'-PeCB	44-420	49			510-2140		110-1210	24-1500	0.40-130	3-1289					126-230
2,3',4,4',5-PeCB	140-1700	170			1530-5660		388-3990	70-4700	150-500	37-4539					387-775
2,3,3',4,4',5-HxCB	18-180	20			337-2140		68-1030	4.7-710	15-110						124-184
Total PCBs	1900-18000	1800	270-14000	4600-480000	14000-60000	3020-245000		820-41000	1600-6000	265-15100	4800-26700	11000-170000	48500	5900	
References	This study	This study	Kozie *et al.* 1991	Falandysz *et al* . 1994	Koistinen *et al.* 1995#	Tarhanen *et al.* 1989	Koistinen *et al* . 1997	This study	This study	Elliott *et al.* 1996a	Anthony *et al* . 1993	Elliott *et al.* 1996b#	Tarhanen *et al.* 1989	Tarhanen *et al.* 1989	Koistinen *et al.* 1997

Recalculated from the concentrations on fat weight basis. BC: British Columbia, BE: Bald Eagle, BM: breast muscle, L: liver

adverse-effect concentration values were about 10 pgTEQ/g in avian eggs and tissues. Koistinen *et al*. (1997) proposed a value of 7 pgTEQ/g in WSE egg as the lowest-observable-adverse-effect concentration. All the TEQs calculated in the tissues of SSEs and WSEs in this study exceeded the lowest-observable-adverse-effect concentration.

It should be noted that the TEQ values we report do not include the contributions of dioxins and furans. Thus, it is likely that our TEQ values are underestimates. In Baltic WSE muscle and eggs, PCB congeners accounted for 56 to 74 % of total TEQs (Koistinen *et al*. 1997).

Another indirect indication of CYP1A induction may be inferred from examination of 3,3',4,4'-TeCB/3,3',4,4',5-PeCB ratios, which varied greatly between eagles with coplanar PCB exposure. In the two eagles with more than 500 pgTEQ/g (nos. 2 and 4), the ratios were much lower. The ratios for nos. 2 and 4 eagles were 0.43 and 0.45, respectively, while the ratio for other eagles with lower TEQs was 1.21 ± 0.56 (mean ± SD). The declining ratios may indicate that hepatic CYP1A is induced in the eagles exposed to the elevated coplanar PCBs, and consequently more metabolizable 3,3',4,4'-TeCB was preferentially degraded by the enzyme. These indirect indications in some eagles may also imply the possibility of other aryl hydrocarbon receptor mediated responses such as immunological and reproductive effects, which have been reported to correlate with hepatic CYP1A induction (Safe 1990, Fernandez-Salguerro *et al*. 1995).

Pb, Cd and Hg levels and their toxicological implications

The concentrations of lead, cadmium and mercury in the liver, muscle and kidney of SSEs and WSEs are shown in Table 2. Lead concentrations in all tissues of four SSEs (nos. 2, 3, 5 and 6) were less than 0.5 micro g/g dry weight. However, high lead concentrations (over 50 micro g/g dry weight) were found in the liver and kidney of the other SSEs (nos. 1, 4 and 7) and all WSEs (nos. 8, 9 and 10).

Lead concentrations in the tissues of birds that do not have any contact with sources of lead contamination (e.g. lead mining and lead shot) are generally very low. Hepatic lead concentrations found in raptors which were not diagnosed as having died of lead poisoning have been reported to be less than 2 micro g/g dry weight (Pain *et al*. 1995). Some authors have suggested that concentrations of 8-10 micro g/g wet weight indicate acute exposure and sufficient residues to cause mortality (Pattee *et al*. 1981, Craig *et al*. 1990). In the present study, 10 micro g/g Pb wet weight (approximately 35 micro g/g dry weight based on a water content of liver and kidney tissues of ca. 65%) was used to indicate acute exposure. Hepatic and renal lead concentrations of three SSEs and three WSEs exceeded this level. Comparison of our data to the concentrations in lead-poisoned raptors previously reported (Table 4), suggested that eagles analyzed in this study were suffering from lead poisoning. This conclusion was also supported by presence of lead fragments in the intestine or gizzard and clinical signs of lead poisoning, such as hypertrophy of the gall bladder and green feces.

Table 4. Comparison of hepatic Hg and Pb concentrations (micro g/g on dry weight basis) in acute poisoned raptor species with those in Steller's Sea Eagles and White-tailed Sea Eagles.

	No.	Hg concentration*	Reference		No.	Pb concentration*	Reference
Steller's Sea Eagle	6	2.8-16	This study	Steller's sea eagle	6	0.06-232	This study
White-tailed Sea Eagle	3	2.0-3.7	This study	White-tailed sea eagle	3	77-174	This study
Hg-poisoned species				Pb-poisoned species			
White-tailed Sea Eagle		16.1-94.9	Henriksson *et al.* (1966)	Bald Eagle	36	17.5-214	Franson (1996)
White-tailed Sea Eagle		169	Koeman *et al.* (1972)	Bald Eagle	2	80.5, 52.5	Jacobson *et al.* (1977)
							Redig *et al.* (1980)
White-tailed Sea Eagle	3	105, 38.5, 116	Falandysz (1984)	Bald Eagle	4	59.5	Pattee *et al.* (1981)
			Falandysz (1986)				
			Falandysz *et al.* (1988)	California Condor	3	21, 80.5, 123	Wiemeyer *et al.* (1988)
				Andean Condor	1	133	Locke *et al.* (1969)
				KingVulture	2	221, 24.5	Decker *et al.* (1979)

* Dry weight based on a water content of liver and kidney tissue of ca. 65%.

In autumn, the most important food resource for SSE and WSE is spawning salmon (Ueta *et al.* 1999). In winter, eagles feed in limited areas where active fisheries are, mainly upon fish that have fallen from nets (WGWS 1996) and upon the carcasses of beached marine mammals (Nakagawa 1998). Since the mid 1990s, the distribution of wintering eagles has shifted into mountain areas, and this has been linked to changes in the availability of winter prey. It is thought that due to a recent decline in fish abundance in areas where eagles traditionally congregated and fed, wintering eagles have shifted their diet toward the abundant deer carcasses resulting from human hunting activity in mountain areas (WGWS 1996, Nakagawa 1998). In Hokkaido, deer hunting is used in an attempt to control the size of the deer population. Whole and partial carcasses of deer shot by hunters are left in the field. Lead fragments embedded in the tissue of deer are ingested by scavenging raptors during and after the hunting season.

In the present study, the hair of deer was found along with a lead fragment in the intestine of a SSE suffering from lead poisoning (no. 4). This suggests that lead from the deer carcasses is a main cause of death of eagles in which lead fragments were found. On the other hand, the origin of lead in the SSE which exhibited high lead concentration in the tissues, but had no deer hair in the digestive tract (no.1) was unclear.

In Japan, lead poisoning in large waterfowl such as swans has been known since 1980. Honda *et al.* (1990) reported that lead poisoning occurred in 7 cases of Whooper Swan *Cygnus cygnus* and Bewick's Swan *C. columbianus* collected in Hokkaido and northeastern Japan during 1984-1987. Lead poisoning in 120 individual Whooper Swans and White-fronted Geese *Anser albifrons* migrating to Hokkaido was also reported in 1989 and 1990 (Ochiai *et al.* 1992, Murase *et al*. 1992). Ochiai *et al*. (1993) reported the occurrence of lead shot in stomach contents of 56 ducks of 9 species collected from three areas of Hokkaido in 1991. The percentages of ducks from these areas with lead were 19-27%. These results suggest that a large number of waterfowl in Japan may be exposed to lead shot by ingestion, although the sample size is not large enough for statistical analysis. The occurrence and frequency of lead poisoning and secondary poisoning in other birds are unclear.

In Japan, the total amount of lead used for hunting game birds and deer amount to about 75 tons every year (Pain 1992). No regulation of lead bullet use has been introduced by the Japanese government (On Jan. 21, 2000, Environment Agency of Japan announced ban of lead bullet use in Hokkaido from the next hunting season), and it is feared that if lead bullets are not restricted, lead poisoning could be a significant cause of eagle mortality in the future. During 1997 to 1999, a total of 55 dead or debilitated SSEs and WSEs in Hokkaido were examined for lead poisoning. Fourty six eagles were found to have died from lead poisoning (Kurosawa 2000). While the loss of habitat, and persecution of eagles have been recognized as primary sources of mortality, lead poisoning in the wintering areas could be another important cause of mortality and may contribute to population decrease.

Mercury concentrations ranged from 5.5-15 in kidney and 2.8-16 micro g/g dry weight in

liver (Table 2). Ranges of cadmium concentrations in the kidney and liver of SSEs were 1.0-7.9 and 0.15-1.3 micro g/g dry weight, respectively. Only trace levels of these elements were detected in muscle. Cadmium and mercury concentrations in WSEs were similar to those in SSEs.

Published values of mercury concentrations in raptors found dead and considered to have been exposed directly to toxic levels of mercury are summarized in Table 4. Although it would be difficult to suggest critical residue levels of mercury that are applicable to all raptors, concentrations in the liver and kidney in excess of 30 mercury mg/kg wet weight (approximately 105 mg/kg dry weight) would appear to be lethal to a range of species of raptors (Thompson 1996). Mercury concentrations in the liver and kidney of SSEs and WSEs in the present study were less than 16 micro g/g dry weight, and unlikely to be a direct cause of death or debilitation, even though slight differences in sensitivity to mercury may exist between species.

Garcia-Fernandez *et al.* (1995) reported that concentrations of cadmium in the kidney of diurnal raptors ranged from 0.35-6.14 micro g/g dry weight (based on a water content of liver and kidney tissues of ca. 65%). The range of cadmium concentration in the kidney of SSEs and WSEs the present study was 1.0-7.9 and 2.3-6.5 micro g/g dry weight, respectively. This range of cadmium concentrations was similar to those in other healthy raptors.

The distribution patterns of mercury and cadmium concentrations in tissues agreed with those found in many other bird species (Osborn *et al.* 1979, Honda *et al.* 1986, Kim *et al.* 1996). Cadmium concentrations in birds are typically highest in the kidney, lower in the liver, and at trace levels in muscle. For healthy, adult birds in wild populations, the concentration of cadmium in the liver is usually between one half and one tenth of the concentration in the kidney of the same bird (Honda *et al.* 1990, Osborn *et al.* 1979, Kim *et al.* 1996). Scheuhammer (1987) suggested that comparing liver and kidney concentrations of cadmium may indicate acute exposure to cadmium. Under acute exposure, birds in such a situation would be expected to have hepatic cadmium concentrations as high as or higher than those in the kidney. In the present study, the ratios of cadmium concentration of liver to kidney in both eagles ranged from 0.08 to 0.25, implying that the cadmium exposures for these birds were not toxic.

CONCLUSIONS

CHC analysis showed coplanar PCBs and *p,p'*-DDE to have accumulated in the tissues of SSEs and WSEs wintering in Japan. The CHC residue levels in some eagles appeared to be greater than the no- or lowest-observable-adverse-effect concentrations that are responsible for CYP1A induction and egg shell thinning. From a long term view, the current concentrations of these CHCs may cause chronic adverse effects in the Far East populations of SSEs and WSEs. However, acute poisoning through the ingestion of lead fragments is likely to have a negative effect on the eagle populations in the short term. Further epidemiological studies of the

contaminant exposure and toxicological effects including sampling of eagle eggs and juveniles in their breeding areas would be necessary to evaluate more accurately the threat to populations.

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