Chlorinated hydrocarbon residues and autopsy data from British Columbia bald eagles (*Haliaeetus leucocephalus*), 1989-1993.

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1. Introduction

Bald eagles (*Haliaeetus leucocephalus*) are predatory birds, which feed at the top of marine and estuarine foodchains and are thus exposed to an array of persistent chlorinated hydrocarbon contaminants. Populations of bald eagles declined during the 1950s to the 1970s in many parts of North America, largely due to the reproductive effects of DDE [1]. However, survival of adult bald eagles was also impacted through acute poisoning by chlorinated hydrocarbons, in particular the cyclodiene insecticide, dieldrin[2].

Several thousand bald eagles breed and tens of thousands winter on the British Columbia coast, particularly in the Strait of Georgia region. Previous studies documented high levels of polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) in fish-eating birds which are potential bald eagle prey[3][4]. Sources of PCDDs and PCDFs in the region were dominated by bleached-kraft pulp mill effluents and forest industry usage of chlorophenols, particularly as tainted wood chips in mill feedstock[3]. This study was undertaken to investigate acute poisoning of bald eagles by chlorinated hydrocarbons, including PCDDs and PCDFs, and to consider the threat posed by multiple exposure to environmental contaminants and other stresses. We report here the results of liver analyses of 59 eagles for organochlorines and a subset of 10 for PCDDs and PCDFs.

2. Methods

Samples collected for this study were part of an overall investigation into the health status of bald eagles in British Columbia. Sick, injured and deceased bald eagles were collected from a variety of government and non-government sources. The majority of the eagles were collected in the south-western area of British Columbia.

Postmortem examination of carcasses was performed at the Island Veterinary Hospital, Nanaimo. Tissue samples were stored in chemically-cleaned (acetone/hexane) glass jars, frozen, and shipped to the National Wildlife Research Centre, Hull, Quebec for analysis. Liver samples were analyzed for organochlorines and PCBs according to methods described previously[3]. PCDD/PCDF and non-*ortho* PCBs in bald eagle livers were analyzed by a Hewlett-Packard 5987B machine with a 30 m DB-5 capillary GC column [5]. All results are expressed on a wet weight basis.

3. Results

Liver samples from 59 bald eagles, collected from a variety of sites on the British Columbia coast, were analyzed for organochlorines. Residue levels for PCBs and DDE were <5 mg/kg in the majority of eagle samples (Fig. 1); however, some birds had levels as high as 186 mg/kg, DDE, and 72 mg/kg, PCBs. DDE levels >100 mg/kg are diagnostic of acute poisoning[6]. Eagles with elevated chlorinated hydrocarbons were all from Johnstone Strait (Port Hardy area) and northern Strait of Georgia (Powell River and Campbell River). Levels of other organochlorines were generally <1 mg/kg, except for some chlordane-related compounds which were occasionally >2 mg/kg. Liver residues of oxychlordane >3 mg/kg are associated with acute poisoning[7].

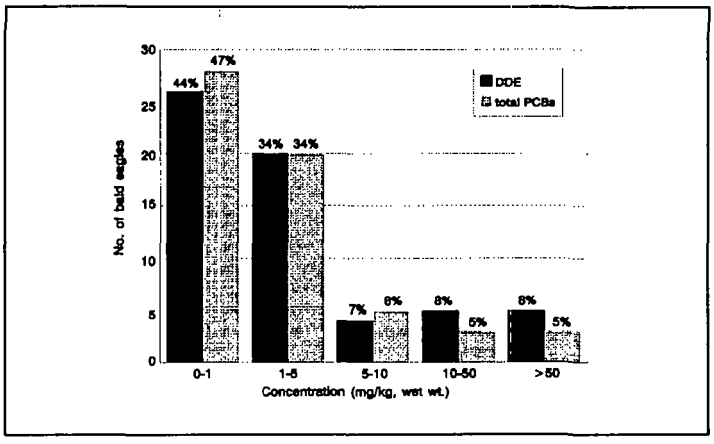

Fig. 1. Distribution of DDE and total PCB residue levels in bald eagle livers (N=59).

A subset of 10 eagles, mainly those found during the late spring and summer of 1990 (therefore

likely resident birds) from the Strait of Georgia and neighbouring Johnstone Strait were analyzed for PCDDs and PCDFs. The major 2378-substituted PCDDs and PCDFs were detected in all birds analyzed; high levels were measured in several individuals (Table 1).

Table 1. PCDDs/PCDFs in bald eagle livers from the south coast of British Columbia (ng/kg wet wt.).

Location	Yr.	COD[a]	Body Cond.[b]	Age/ Sex	% Fat	2378-TCDD	12378-PnCDD	123678-HxCDD	2378-TCDF	23478/ 13489-PnCDF
Port Hardy	90	Ud	2	J/M	1.7	5	10	9	20	trace[d]
CampbellR.[e]	90	Tr	5	J/F	4.4	18	25	37	33	8
Powell R.[e]	90	El	2	A/M	2.4	392	1415	4360	3	375
Powell R.[e]	90	Pb	3	A/F	4.0	41	83	184	63	27
Comox	90	Co	5	J/M	2.8	21	51	169	60	17
Denman Is.	90	As	3	A/M	2.6	49	92	295	78	30
Bowser	90	Pb	1	A/F	3.9	263	603	2051	15	152
Coombs	90	Pb	1	A/M	3.5	6	9	10	42	6
Nanoose	90	Tr	3	A/F	1.9	23	57	90	28	11
Sechelt	90	El	3	A/F	1.4	29	199	936	24	138

[a] COD - cause of death; As-asphyxiation, Co-collision, El-electrocution, Pb-lead, Tr-trauma, Ud-undetermined
[b] scale: 0-5; 0=emaciated 5=excellent
[c] only 23478-PnCDF congener
[d] 'trace' = 2 ng/kg wet wt; Minimum Detection Limit = 3 ng/kg wet wt.
[e] pulp mill location

4. Discussion

A small proportion of bald eagles tested had DDT- and chlordane-related levels diagnostic of acute poisoning. Most of those cases were adult birds of both sexes, found during the breeding season, in an emaciated condition. This suggests the energetic demands of breeding reduced lipid stores, leading to mobilization of chlorinated hydrocarbons and toxicity. PCDD and PCDF levels were also elevated in those specimens.

Highest levels of PCDDs/PCDFs were in livers of adult eagles of both sexes found, usually in an emaciated state, during the breeding season near the pulp mills at Campbell River and Powell River. There is no data on tissue levels of TCDD-like compounds which could be diagnostic of acute poisoning. LD_{50}s reported for 2378-TCDD are 240 ng/kg in chicken and 1350-2180 in pheasant embryos[7]. Lethal doses in adult birds are one to two orders of magnitude higher. Toxic equivalents, for example in one of the eagles from Powell River, were calculated to be from 2,740 ng/kg $TEQs_{WHO}$[8] to 6,550 $TEQs_{Safe\ 1990}$[9], and estimated using a chick embryo hepatocyte bioassay[10]

in the laboratory of Dr. Sean Kennedy to be 25,000 ng/kg. TEQs in this range would likely be toxic to sensitive species. Electrocution was the proximal cause of death for the above bird, although it was also in an emaciated state. As an adult bird in summer, as discussed above, low body fat may have been a result of the energetic demands of breeding; alternatively, the bird may have been in an anorexic state symptomatic of wasting syndrome.

The principle causes of death or injury to bald eagles were trauma and electrocution, followed by poisoning, mainly by lead from hunter's shot. A number of electrocuted and lead poisoned birds also had elevated chlorinated hydrocarbons, not all of which were associated with poor body condition and thus lipid and contaminant mobilization. The potential for synergism among contaminants and for association between sub-lethal exposure to chlorinated hydrocarbons and accidental death requires further study.

5. References

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