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ABSTRACT: During the winter of 1990 in the Fraser Delta of British Columbia, Canada, nine birds of prey were found with symptoms of anticholinesterase poisoning. Immediate surgical removal of crop contents of three birds decreased mortality and recovery time. Chemical analysis was conducted on crop contents, which contained mainly duck parts. A bald eagle (*Haliaeetus leucocephalus*) contained 200 μg/g and a red-tailed hawk (*Buteo jamaicensis*) 2.2 μg/g carbofuran, while the crop of another red-tailed hawk contained 30 μg/g fensulfothion. There was evidence that granular carbofuran and fensulfothion persisted long enough in the wet, low pH conditions of the Fraser Delta to kill waterfowl and cause secondary poisoning of raptors several months after application of the pesticides.

Key words: Raptors, carbofuran, fensulfothion, poisoning.

INTRODUCTION

The effects of persistent organochlorine contaminants on raptor populations have been widely documented (Newton, 1979; Noble and Elliott, 1990). Organochlorines largely have been replaced by organophosphorus and carbamate insecticides that are considered less persistent, less bioaccumlative and therefore of lower risk for secondary poisoning of raptors. There are occasional published reports of anticholinesterase secondary poisoning of raptors by, for example, carbofuran (Balcomb, 1983). Recently, Porter (1993) concluded that secondary poisoning of raptors was a relatively common occurrence.

Large numbers of waterfowl and raptors winter in agricultural fields of the Fraser Delta of British Columbia (Butler, 1992). Many fields are intensively farmed for vegetables and use of toxic, granular insecticides is common (Szeto and Price, 1991). In past wildlife mortality incidents from 1971 to 1989, carbofuran (FMC Corporation, Philadelphia, Pennsylvania, USA) and fensulfothion (Miles, Toronto Ontario, Canada) were implicated in the deaths of thousands of waterfowl and songbirds in the delta (Wilson et al., 1995). We report here on two incidents during the winter of

1990 in the Fraser Delta involving secondary poisoning of raptors resulting from granular insecticides used many months previously.

MATERIALS AND METHODS

This study was begun as an overall investigation into the health status of bald eagles in British Columbia. Toxicological concerns were initially focused on poisoning by lead shot and persistent organochlorines; poisoning by anticholinesterase compounds had not been identified previously as an important mortality factor for eagles. In the fall of 1989, a request for sick, injured, and deceased bald eagles was published in local periodicals and circulated to wildlife agencies. Birds subsequently were received from both government and non-government sources. Postmortem examinations of carcasses were performed at the Island Veterinary Hospital, Nanaimo, British Columbia, Canada. In most cases the entire carcass was available for tissue samples; however, if a carcass was permitted by the Wildlife Branch of the British Columbia Ministry of Environment for taxidermy, only liver and kidney could be removed for chemical residue analysis. Sex of bald eagles was determined on post mortem or by hallux claw and bill depth measurement (Bortolotti, 1984); age was determined from molting sequence (McCollough, 1989).

Between 2 and 12 February 1990, six birds in good to excellent body condition were found in agricultural areas adjacent to a golf course in Richmond or on farmlands in Ladner, British

Columbia, Canada (49°06'N, 123°10'W). Bald eagles 90-48, 90-133, and 90-51 were found alive with symptoms consistent with anticholinesterase exposure, including poor coordination, constricted pupils, and distended crop. Eagle 90-48 was anesthetized by gas anesthesia using isoflurane (tradename: Aerrane, Ohmeda Inc., Mississauga, Ontario, Canada) and crop contents surgically removed. The bird recovered quickly and was released; crop contents were not retained. The other three birds, 90-45, 90-150 and 90-450, were dead on arrival and crop contents were removed and retained for chemical residue analysis. On 13 March 1990, two red-tailed hawks and a bald eagle were found near the Richmond golf course. Hawk 90-452, an adult male bird in good body condition, was brought live into a rehabilitation center, although it died later the same day. Hawk 90-451 was found dead on the same spot with 90-452, but its ingesta was not tested for residues. A bald eagle found dead in the same area also was not tested for residues. Both of these birds had been designated for taxidermy; provisions had not made to obtain either the brain or the crop contents from specimens so designated.

Crop contents were removed and placed onto solvent-rinsed (acetone and hexane) petri dishes, visually examined and then transferred to solvent-rinsed glass jars. Identifications of duck and gull remains in gut ingesta were made by feathers, feet, and bill. Crop contents were analyzed for organophosphorus and carbamate insecticides at Zenon Laboratories, Burnaby, British Columbia, Canada, according to the methods of British Columbia Ministry of Environment and Parks (1989). Samples were extracted with a mixture of acetone and methylene chloride, concentrated and purified on a charcoal column. Extracts were screened for organophosphorus compounds by gas chromatography and mass spectrometry (GC/MS) (Hewlett Packard Model 5890 Series II gas chromatograph, Hewlett Packard, Cupertino California, USA; Hewlett Packard mass spectrometer Model 5970, Hewlett Packard) and for carbamates by high pressure liquid chromatography (HPLC) (Pickering Laboratories HPLC Model 1090 and post column reaction module PCX 5000, Pickering Laboratories, Pickering Ontario, Canada). Ingesta from two bald eagles, 90-45 (stomach) and 90-150 (crop), and two red-tailed hawks, 90-450 (crop) and 90-452 (stomach), were analyzed by the Agriculture Canada Special Projects Laboratory, Calgary Alberta according to the methods of Agriculture and Agri-Food Canada (1990). Ingesta were homogenized with methanol and extracted with benzene and hexane. Separation and identification of organophosphorus and

carbamate peaks were by GC/MS (Hewlett Packard 5890 Series II gas chromatograph, Hewlett Packard; VG AutoSpec Q mass spectrometer, VG Analytical, Beverly, Massachusetts, USA). Organochlorines in liver were analyzed according to the methods of Norstrom et al. (1988).

Brain acetylcholinesterase (AChE) activity was determined at the Western College of Veterinary Medicine, Saskatoon, Saskatchewan, Canada, according to the method of Hill and Fleming (1982). Comparison of AChE results for normal birds with those reported by Hill (1988) was assured by previous analysis of a number of eagles and hawks which had not been exposed to anticholinesterase agents. Lead exposure was determined by evaluating delta-aminolevulinic acid dehydratase (ALA-d) activity ratios in blood; ALA-d activity was determined as described in Scheuhammer (1987). Normal ALA-d activity can vary among individuals; therefore, the results were converted to the ratio of activated:non-activated enzyme activity to reduce variability. Lead poisoning was considered for bald eagle 90-51 because its blood ALA-d activity was evidence for sub-lethal lead exposure.

RESULTS

Based on the GC/MS analysis of the stomach contents of eagle 90-45, there were no pesticide residues (detection limit 0.001 μg/g) (Table 1). The crop contents of bald eagle 90-150 contained 200 μg/g of carbofuran, and a brain AChE activity of 16 μmol/min/g. Crop contents of red-tailed hawk 90-450 contained 30 μg/g fensulfothion. Organochlorines in livers of eagle 90-150 and hawk 90-450 were: dichlorodiphenyldichloroethylene (DDE) and polychlorinated biphenyls (PCBs), <1 μg/g (wet weight); hexachlorobenzene, trans-nonachlor and dieldrin, <0.1 μg/g. The contents of the distended crop of red-tailed hawk, 90-452, contained 2.2 μg/g of carbofuran; brain AChE activity was 9.9 μmol/min/g for this bird.

DISCUSSION

For the nine raptors collected, diagnosis of pesticide poisoning was confined to cases where organophosphorus or carbamate residues were positively identified and quantified in ingesta. Three of the

TABLE 1. Nine birds of prey confirmed or suspected to have been pesticide poisoned in February and March 1990 in the lower Fraser valley, British Columbia. Birds from Group 1 were collected 2 to 12 February 1990; birds in Group 2 were collected 13 March 1990.

Sample number	Species[a]	Sex and age[b]	Location	Signs	Gut ingesta	Pesticide residue	Comments
Group 1							
90-48	BAEA	M/J	Richmond Golf Course	Poor coordination; constricted pupils DOA[d]	Duck and gull	NA[c]	Released 28 Feb. 1990
90-45	BAEA	M/J	Ladner Dyke		Duck	ND[e]	
90-133	BAEA	U/J	Burns Bog	Poor coordination; crop distended	NC[f]	NA	Released 1 July 1990
90-51	BAEA	M/A	Richmond Golf course	Poor coordination; constricted pupils	Duck	NA	Released 1 March 1990; lead exposed[g]
90-150	BAEA	M/A	Steveston Highway	DOA	Duck	Carbofuran 200 µg/g	
90-450	RTHA	F/A	Steveston Highway	Crop distended, DOA	Duck	Fensulfothion 30 µg/g	Died 13 Feb. 1990
Group 2							
90-451	RTHA	F/A	Richmond Golf Course	DOA	NC	NA	
90-452	RTHA	M/A	Richmond Golf Course	Crop distended; oily exudate from mouth	Duck	Carbofuran 2.2 µg/g	Died same day
None	BAEA	U/J	Richmond Golf Course	DOA	NC	NA	

a BAEA—Bald eagle; RTHA—Red-tailed Hawk.
b M = male, F = female, A = adult, J = juvenile, U = unknown.
c NA = not analyzed.
d DOA = dead on arrival.
e ND = not detected.
f NC = not collected.
g delta aminolevulinic acid dehydratase (ALA-d) ratio = 2.91.

four birds tested contained organophosphorus or carbamate residues. However, based on coincidence in time and place and presence of known symptoms of anticholinesterase poisoning (Grue et al., 1991), and finding of waterfowl remains in ingesta (evidence of feeding in farmland), the six other raptors described here are also strong poisoning suspects. In one of those cases, although GC/MS analyses were undertaken, no pesticides were identified from the crop contents. However, even in cases of extreme AChE inhibition, Greig-Smith (1991) reported that pesticide residues were not detected in gut contents of 25% of a large sample of birds tested. Organochlorine levels in one eagle and one hawk analyzed were lower than those associated with acute toxicity in raptors (Noble and Elliott, 1990).

Although bald eagle 90-150 had 200 µg/g carbofuran in its ingesta, the brain cholinesterase activity was within the range (12 to 20 µmol/min/g) considered normal by Hill (1988). Greig-Smith (1991) reported no relationship between carbamate residues and brain AChE depression in a large sample of wild birds found dead and submitted for pesticide investigation in Great Britain. Eagle 90-150 was found dead and therefore post-mortem reactivation of brain cholinesterase likely occurred. Red-tailed hawk 90-452 was found alive and died later the same day. Its ingesta contained 2.2 µg/g carbofuran and brain cholinesterase activity was depressed 50% below the normal red-tailed hawk mean of 19 µmol/min/g (range 12 to 26 µmol/min/g; Hill, 1988). Thus, we believe that freezing of the brain soon after death may have reduced post-mortem reactivation of AChE activity.

Based on our experience, secondary pesticide poisoning investigations require close cooperation among agencies and individuals that receive carcasses. In jurisdictions such as British Columbia that issue taxidermy permits to private individuals for provincially managed species such as birds of prey, extra efforts may be nec-essary to obtain tissues in a properly preserved state. Moratoriums on issuance of taxidermy permits are only partially successful as some individuals stop submitting carcasses. A more promising approach involves close cooperation with reputable taxidermists who will prepare specimens while partially frozen, and thus remove the brain, internal organs and gastrointestinal tract in a condition suitable for analysis.

Granular formulations of carbofuran and fensulfothion, applied in the Fraser Delta in spring and early summer from 1971 to 1976 and 1986 to 1989 for control of soil pests, caused waterfowl and songbird mortalities (Mineau, 1993). However, most mortality occurred during the fall and winter, when flooded fields attracted waterfowl. Granular insecticides are a hazard to songbirds which may mistake them for grit or food (Best and Fisher, 1992). Given their bill morphology, it is unlikely that waterfowl selectively pick up single granules from the soil surface. It is more likely that ducks ingest the granules while sifting sediments. At issue is granular persistence following normal use. Base-catalyzed hydrolysis is the most important chemical degradation pathway for carbofuran (National Research Council of Canada, 1979). The half-life of carbofuran in water due to hydrolysis ranged from 0.2 days at pH 9.5 to 1,700 days at pH 5.2. Fensulfothion is primarily metabolized by soil microbes (Miles et al., 1979). Shella and Vasantharajan (1977) reported significant fensulfothion residues in soils up to 900 days after application, and postulated that microbial breakdown of organophosphorus pesticides is likely to be highly pH-dependent. The pH of lower Fraser Delta soils was between 5.0 and 5.9 (Mineau, 1993); degradation of both carbofuran and fensulfothion is expected to be very slow at such low pH values. Williams et al. (1976) observed a build-up of carbofuran residues in Fraser valley soils after 2 yr of use. Recently, Szeto and Price (1991) reported persistence of carbofuran and fensulfothion at concentrations of 78 and 92

μg/g, respectively, in Fraser Delta silt loam soils, almost a year after labelled granular application.

Waterfowl mortality in puddled fields treated with granular carbofuran is not restricted to the acidic organic soils of the Fraser Delta of British Columbia. In California (USA), between 1984 and 1988, 22 wildlife kill incidents, resulting from the use of a 5% carbofuran granule on rice, were documented (Littrell, 1988). Approximately 525 birds, mostly waterfowl and some raptors were recovered in those incidents. Avian gastro-intestinal tracts had residues ranging from non-detectable to 640 μg/g carbofuran (median 6.3 μg/g). The granules were applied in spring and the fields flooded and seeded with rice. Although most mortality occurred during spring, there also were fall kills on record. As carbofuran was not registered in California for fall use, those kills were due either to product misuse or long-term granular persistence. Littrell (1988) favored misuse, assuming that granules did not persist; however, based on the preceeding evidence, fall kills are possible from spring carbofuran applications.

Because of its repeated association with wildlife kills, carbofuran was withdrawn from the Fraser Valley market in 1976, but subsequently was returned in 1986 at the request of the agricultural community. Based on our data, spring-applied granular insecticides can cause both primary and secondary bird kills as late as the following winter. On this evidence, granular carbofuran again was withdrawn from the lower Fraser Valley market in 1990. Coincidentally, production of fensulfothion was halted in 1991 by the manufacturer, due to concerns about its environmental toxicity.

ACKNOWLEDGMENTS

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