

FIRST RECORDS OF CHLORDANE-RELATED MORTALITY IN WILD BIRDS

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Technical chlordane is a mixture of chlorinated hydrocarbons that has been used as an insecticide since the late 1940's. Its primary current use is for control of soil pests through surface or subsurface applications. Despite its widespread use, persistence, and lipophilic (and therefore bioaccumulative) properties, there is no firm evidence of either lethal or sublethal effects of chlordane on wild vertebrates when used in terrestrial systems (Natl. Res. Council, Canada 1974, Stickel et al. 1979). Lowered reproductive success and limited mortality of young of several avian species were associated with applications of chlordane to a marsh in North Dakota; however, these problems probably were partially attributable to depletion of invertebrate prey in the treated marsh (Hanson 1952).

Production and use of chlordane in the United States has decreased greatly over the past several years; the only registered use for chlordane after 1980 was for termite control through subterranean applications (Sittig 1980). Because changes in use regulations are possible and because chlordane use continues in other parts of the world, it is important to document adverse effects that may occur in the field. This note provides the first records of chlordane-related mortality of wild birds—2 adult male red-shouldered hawks

(*Buteo lineatus*) and an adult female great horned owl (*Bubo virginianus*).

One hawk was found freshly dead on 25 April 1978 in a wooded area just south of Greenbelt National Park, Greenbelt, Maryland. The other hawk was found dead near a large industrial complex in Birmingham, Alabama in February 1981. The hawks were frozen and later necropsied at the Patuxent Wildlife Research Center in Laurel, Maryland. The adult female owl was found alive on the ground on 6 June 1980 about 6 km south of Corvallis, Oregon at the Corvallis Airport. It was listless and offered no resistance when picked up. It was placed in a pen where it was found dead the next morning. The carcass was frozen and subsequently shipped to the National Wildlife Health Laboratory in Madison, Wisconsin where it was necropsied. Residue analyses were performed at the Patuxent Wildlife Research Center. Brains and carcasses of the 3 birds were extracted in Soxhlet with hexane and the extracts cleaned on a Florisil column. The clean extracts were separated into 3 fractions in a silicic acid column and analyzed for organochlorine pesticides, their metabolites, and PCB's (Cromartie et al. 1975).

Residues of heptachlor epoxide and oxychlordane in the brains of the 3 raptors (Table 1) were within the critical lethal range established in experimental studies. Although technical chlordane is a mixture of compounds, 2 metabolites—heptachlor epoxide and oxychlordane—have been identified as causing mortality in experimental birds given diets containing technical chlordane (Stickel et al. 1979). Res-

Table 1. Residues of organochlorine pollutants in tissues of 3 raptors.

Species	Tissue	Residue, ^a $\mu\text{g/g}$ (wet weight)										
		DDE	DDD	Dieldrin	HCB	HE	OXY	CCH	TNCH	CNCH	Mirex	PCB's
Red-shouldered hawk	Brain ^b	4.8	0.9	3.4		3.4	5.2	2.4	6.4	1.0	0.1	17.0
	Carcass	2.3		1.2		1.1	1.5	1.1	3.0	0.4		11.0
	Brain ^c	1.0		4.7	NA ^d	4.0	1.9	1.1	2.6	0.9	NA	3.2
	Carcass	1.4		4.5	NA	4.5	2.3	2.0	4.5	1.4	NA	3.5
Great horned owl	Brain	28.0		0.8	5.1	5.8	3.7	0.1	0.1			8.2
	Carcass	4.5		0.1	0.7	0.7	0.4					0.8

^a HCB = hexachlorobenzene, HE = heptachlor epoxide, OXY = oxychlordan, CCH = *cis*-chlordan, TNCH = *trans*-nonachlor, CNCH = *cis*-nonachlor, PCB's = polychlorinated biphenyls.

^b Found dead in Maryland.

^c Found dead in Alabama.

^d NA = Pollutant not analyzed.

idues of the 2 critical compounds in brains of experimental birds dying from chlordan ranged from 3.4 to 8.3 $\mu\text{g/g}$ for heptachlor epoxide and 1.1 to 5.0 $\mu\text{g/g}$ for oxychlordan. The 2 critical compounds originate from biological and physical breakdown in the environment or from metabolism after ingestion. Heptachlor is introduced into the environment primarily through its use as an insecticide and to a small extent from breakdown of *cis*- and *trans*-chlordan. Heptachlor is rarely found in environmental samples because of its extreme propensity for epoxidation. Oxychlordan residues in the environment originate from breakdown of components or contaminants of heptachlor and technical chlordan including *trans*- and *cis*-chlordan and *trans*-nonachlor (Tashiro and Matsumara 1978, Stickel et al. 1979). Residues of other compounds related to technical chlordan were present in the brains of the 3 birds. Levels of these other compounds in brains of both hawks were similar to those in brains of experimental birds dying on chlordan dosage, but those in the owl were much lower. Although both oxychlordan and heptachlor epoxide were also present in brains of Canada geese (*Branta canadensis*), raptors, and other birds that died from

ingestion of heptachlor, heptachlor epoxide was strongly predominant, $\geq 8:1$ (Blus et al. 1979, Blus et al. in prep.). The proportion of heptachlor epoxide relative to oxychlordan in the brain of the owl (0.6:1), the hawk from Alabama (2.1:1), and that of the hawk from Maryland (1.5:1) corresponds well with the proportion in brains of experimental birds killed by chlordan.

Dieldrin levels of 3.4 and 4.7 $\mu\text{g/g}$ in the brains of the hawks were within or near the lower lethal range of 4–5 $\mu\text{g/g}$ in the brains of experimental birds (Stickel et al. 1969). More recently, the lower lethal limit for dieldrin was amended upward to 5 $\mu\text{g/g}$ (W. H. Stickel, pers. commun.). Thus, it is likely that chlordan was primarily responsible for deaths of both hawks; but there is also a possibility that mortality of the 2 birds was related to interaction between dieldrin, heptachlor epoxide, and chlordan. None of the other organochlorines was present in concentrations that are known to cause mortality individually. The necropsy data were inconclusive as to cause of death; but, body condition, including severe emaciation and other factors, was suggestive of mortality from organochlorine insecticide poisoning (Stickel et al. 1969).