Organochlorine Toxicity In Tawny Frogmouths

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Introduction

Each year for the last five years, unexplained mortalities and neurological signs have been observed in tawny frogmouths (*Podargus strigoides*) in Sydney. The timing of outbreaks has been consistent each year, occurring over a 4-5 week period commencing in late winter and early spring, with submission of affected birds to wildlife shelters usually ceasing abruptly in mid- to late spring, corresponding with the annual emergence of prey insect species.

Clinical signs observed in live tawny frogmouths have included the following:

- abnormal activity during daylight;
- inability to fly or walk;
- leg extension;
- wings cowled or mantled around the body;
- tail flicking upwards;
- abnormal head carriage, with unilateral tilt or opisthotonus;
- eyes shut or maintained fully open with a glazed expression;
- convulsions, often precipitated by noise, with extreme opisthotonus and accompanied by raucous screeching;
- prior to death, seizure activity with violent body movements and repeated somersaulting;
- death in convulsions or coma.

Members of the public described birds literally falling dead in flight. Other birds were found dazed and in dorsal recumbency.

Seizure activity could be controlled by repeated heavy diazepam sedation, administered every 3-4 hours intramuscularly or intravenously. Despite several birds being maintained sedated in wildlife shelters for up to two weeks after submission, recovery of birds was rare.

Most affected birds were in poor nutritional condition, although some were in good condition. Both sexes were equally represented and the age of affected birds was highly variable.

In the late winter and early spring of 1994, the number of frogmouths with neurological signs submitted to Sydney wildlife shelters was much higher than in previous years. Three Sydney branches of WIRES (the NSW Wildlife Information and Rescue Service) recorded 53 frogmouth deaths within a one week period, the majority of affected birds appearing to be first year adults. Shelters in the North Shore suburbs of Sydney recorded the highest number of submissions (Table 1) but mortalities and neurological signs were also reported during the same period by wildlife workers in Wollongong, Dubbo, Port Macquarie, Taree and Brisbane.

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Limited necropsies and histopathological examinations had been undertaken on affected frogmouths in previous years but no significant lesions had been detected. The unexpectedly high number of affected birds in 1994 encouraged a more systematic investigation, initiated by the North Shore branch of WIRES.

Nineteen fresh carcases of frogmouths from various northern Sydney suburbs, all displaying typical neurological signs prior to death or euthanasia, were necropsied and samples of all organs (including brain, spinal cord, peripheral nerves and ganglia) examined histologically. All examined birds harboured a moderate to high intestinal burden of nematodes and cestodes, and all were in relativley poor nutritional condition, with negligible subcutaneous and visceral adipose depots and often atrophy of the pectoral skeletal muscles. No gross or microscopic central or peripheral nervous system lesions were detected and no bacteria were isolated from meningeal swabs, liver or lung of each bird.

The absence of histological lesions permitted exclusion of infectious causes, vitamin deficiencies, parasitic larval migration through the central nervous system and several neurotoxins from the differential diagnostic list, and implied a functional rather than structural lesion underlying the clinical signs. Samples of liver and brain from nine of the 19 submitted frogmouths were tested for organophosphate and organochlorine compounds by the Residue Laboratory, Animal Health Laboratories, Department of Primary Industries, Yeerongpilly, Queensland. The results are summarised in Table 2.

All livers and brains of tested frogmouths were negative for organophosphates and for the following organochlorine compounds - hexachlorocyclohexanes (BHC), lindane, chlordane, pentachlormethoxybenzene, isodrin, endosulphan, strobane, camphechlor, methoxychlor, endrin, aldrin, hexachorobenzene (HCB), dicofol, polychlorinated biphenyls (PCB) and chlorpropham. Tissues from all tested birds were positive for four organochlorine compounds - oxychlordane, heptachlor epoxide, DDE and dieldrin - all being some of the most environmentally persistent organochlorine compounds. An owlet nightjar (*Aegotheles cristatus*) that died with neurological signs and was submitted for necropsy by WIRES was also tested. It too contained hepatic residues of the same four compounds but at low, probably subclinical, levels. This particular bird had succumbed to a *Pasteurella multocida* bacteraemia following an attack by a cat, but it served as a convenient control for the frogmouth toxicological assays.

There are no definitively established organochlorine levels that reliably equate with clinical signs of intoxication in mammalian or avian species. However, the tissue levels detected in the frogmouths indicated heavy exposure to organochlorines and subsequent storage of toxic residues, with the levels recorded (particularly in the brain) sufficient to confidently ascribe the clinical signs and mortality to intoxication. In intoxication of dogs by dieldrin, brain levels of 3-5 mg/kg are responsible for neurological signs and death, whilst clinical signs with recovery are associated with brain levels of 2 mg/kg or less in the same species. Clinical signs of intoxication occur in calves dosed at 2.5 mg/kg of heptachlor epoxide. These levels contrast with much higher oral LD_{50} levels set for acute single dose intoxication, eg the LD_{50} for chlordane in domestic animals is 50-150 mg/kg, for dieldrin in most domestic species 25-90 mg/kg and for heptachlor 15-50 mg/kg in most livestock species and dogs[9].

The four componds detected in all tested birds are additive in effect but a simple summation of the concentration of the residues in the liver or brain underestimates the toxic effect in an individual. The chlorinated cyclodienes (oxychlordane, heptachlor epoxide and dieldrin) are more toxic than the chlorinated ethane class of organochlorines (which includes DDE, a metabolite of DDT). Moreover, heptachlor epoxide, the metabolite of heptachlor, is approximately ten times more toxic than the parent molecule.

Organochlorine compounds are highly lipid-soluble and non-water-soluble. Once absorbed, they persist as residues sequestered in lipid-rich tissues, undergoing slow metabolism and excretion. The compounds are also poorly degradable and persistent in the environment. The half-life of heptachlor in sole is approximately 10 months and that of chlordane one year. Biomagnification of the compounds in the food

chain is a characteristic feature, often without provoking overt clinical signs in intermediate prey species, and chronic intoxication is more common than acute[1,9].

Sequestration of organochlorines in adipose tissue acts as a safety mechanism in mammals and birds, so that individuals with substantial fat stores or those on rising planes of nutrition are less susceptible to intoxication than cachectic animals or those on falling nutritional planes[9]. The poor nutritional condition of most of the clinically affected frogmouths, the seasonal increase in mortalities corresponding with a period of poor feed availability and the abrupt cessation of the outbreaks as prey insect populations increase suggest that the plane of nutrition and physical condition of the birds are important epidemiologically.
Hepatic levels of organochlorine compounds are a general indicator of exposure and storage in lipid-rich tissues. Cerebral levels are a more precise indicator of toxic levels, and they may represent potentially both storage residues and mobilisation of residues from extraneural sites as adipose stores atrophy in periods when the diet is suboptimal[9].

Whilst the tabulated hepatic levels in the Sydney frogmouths confirm high level exposure and storage, the brain levels are the more significant in determining a causal association with clinical signs and death. Neither value permits a diagnosis of acute versus chronic intoxication. However, the pattern of annual outbreaks coinciding with a period of relatively poor feed availability in late winter and early spring is suggestive of chronic intoxication, with signs of acute toxicity developing as sequestered stores are mobilised from fatty tissue to reach a critical threshold in the central nervous system. There is also increased activity of frogmouths in the spring with peak breeding activity in September and October; this could accelerate mobilisation of stored residues but, at least in females, could also have a sparing effect as organochlorines become concentrated in yolk[5,11].

Organochlorine poisoning can occur as a result of inhalation, percutaneous absorption or by direct or indirect ingestion[9]. The diet of tawny frogmouths is variable but includes nocturnal insects, centipedes, spiders and cockroaches, snails, frogs and occasionally small mammals. The geographic range of frogmouths is relatively small, apart from young adult males establishing new territories, and often the same nesting site is used for many years[2,5-6,11]. The clustering of clinically affected frogmouths in the northern suburbs of Sydney therefore suggests local exposure to organochlorines.

DDT, dieldrin and other organochlorines were extensively employed in Australian agriculture in the 1950s and 1960s. Use of organochlorines has been legally restricted since 1987 to virtually sole use in termite eradication campaigns, although use of heptachlor was still permitted in 1993 on sugarcane crops and dieldrin use against cane grub was still allowed in 1991. DDT use for agricultural crops continued until late 1987 in South Australia although such use had been generally outlawed elsewhere in Australia in the 1970s. Heptachlor, aldrin and dieldrin use has continued for domestic termite control (and indirectly control of cockroaches, fleas and other arthropods), with legislation to phase out all such use from June 1995 in all Australian states except the Northern Territory[10]. The prolonged and widespread domestic use of organochlorines in Sydney affords a source of toxins for such species as frogmouths with limited territorial ranges and positioned high in the feed chain. Some frogmouths may well have succumbed to acute intoxication via inhalation, percutaneous absorption (especially relevant for the chlorinated cyclodienes)[9],
ingestion of contaminated soil or of poisoned prey species such as cockroaches soon after domestic application of the compounds. More gradual accumulation of residues by the majority of affected frogmouths is, however, more likely.

Neurological signs and mortality are dramatic manifestations of organochlorine toxicity. More subtle pathophysiological effects occur at lower levels of exposure. Organochlorines can enhance hepatic microsomal mixed-function enzymatic oxidation and hence affect the detoxification capacity of the liver for other compounds and the organ's ability to metabolise endogenous steroid hormones. Excessive inactivation of oestrogens by hyperplastic smooth endoplasmic reticulum of hepatocytes can disturb or

interrupt nesting and egg-laying cycles of birds. Organochlorines can also interfere with energy-dependent calcium transport and hence eggshell formation. Increased fragility of eggs contributes to decreased hatching rates and organochlorines may also have more direct effects on embryo survival. Breakage of thin-shelled eggs due to organochlorine toxicity has been incriminated in the decline of peregrine falcon populations in Victoria and South Australia, paralleling the well-documented effects of chronic DDT intoxication in the American bald eagle and other predatory species. Moreover, many avian species sequester and effectively excrete organochlorines in the yolk of eggs. Newly hatched chicks, rapidly absorbing yolk contents into the intestinal tract, are exposed to high organochlorine levels, and acute toxicity and mortality can occur within 2 to 3 days of hatching. Skeletal and other deformities are recorded in some exposed hatchlings that survive[1,3,6-7,9]. The critical DDE level for significant eggshell thinning in peregrine falcons is established at 15-20 mg/kg. The critical level of dieldrin for deleterious effects on the reprooductive system of the same species is 1.0 mg/kg[8].

The mechanism by which organochlorines produce neurological signs is still incompletely understood. DDT is known to alter plasma membrane permeability by impairment of the stabilising function of membrane phospholipids, thereby inhibiting ATPase membrane pumps. By retarding influx of sodium ions and inhibiting egress of potassium ions across the neural axolemma, the nerve membranes become partially or completely depolarised, resulting in generalised muscle tremors. Sensory nerves appear more susceptible than motor nerves. Intention tremors may be associated with voluntary movements and may progress to coarse and sustained body tremors. Inactivation of calcium-dependent pumps in skeletal muscle membranes probably contributes to neuromuscular signs. The cause of convulsions is less well-understood, although elevation of cerebral ammonia levels due to impaired glutamine synthesis has been documented. Central nervous depression rather than excitation can, however, be a characteristic of intoxication by some organochlorine compounds at particular dosage rates[1,6,9].

In experimentally intoxicated mammals, clinical signs become progressively more severe from onset. Initial apprehension and hypersensitivity with facial and cervical muscle fasciculations progress to tonic-clonic muscle contractions, apparent blindness, abnormal posturing, ptyalism and continuous chewing movements and vocalisation. Acute intoxication of mammals is also associated with marked terminal pyrexia, largely attributable to hypothalamic dysfunction. Death can occur during severe seizure activity or in a comatose state. Intoxicated birds may show, in addition, erratic flight, ataxia, anorexia and listlessness, abnormal eyelid blinking and side-to-side head movements, wing drop and excessive swallowing[6,9].

It is anticipated that mortalities associated with neurological disease will again be reported in tawny frogmouths in late winter/early spring in 1995 and ensuing years. Future investigative projects are required to establish whether similar clinical syndromes reported in the species in rural NSW and Brisbane are also attributable to organochlorine toxicity, and to quantitate the level of toxic residues in unhatched embryos and hatchlings. Australian Geographic has donated funds in 1995-1996 to attempt to quantify the population of tawny frogmouths and map the major breeding sites along the eastern Australian seaboard, information crucial to determining the true impact of chemical toxicity on this species.

Unfortunately, the legacy of chemical contamination of the environment can be anticipated to be long-lived and many wild mammalian and avian species are likely to be affected in the future as a result of decades of organochlorine use in Australia. As long ago as 1981, a keen amateur naturalist recorded the now familiar clinical signs of organochlorine toxicity in a Victorian tawny frogmouth. Toxic levels of hepatchlor, lindane and alpha-benzene-hexachloride were detected in its brain[4]. From 1983 to 1985, the NSW National Parks and Wildlife Service and the Department of Agriculture tested 400 native mammals and birds from north coast NSW to determine the extent of environmental contamination by organochlorines. The investigation was prompted by consistent detection of residues in meat and milk samples from the area since late 1969. Kookaburras, cormorants and magpies were found to contain DDT tissue residues

in excess of 90 mg/kg, and one magpie had adipose dieldrin stores of 375 mg/kg. Bandicoots from the area contained up to 50 mg/kg DDT in adipose tissue and in excess of 115 mg/kg dieldrin. Birds of prey from the area were tested in 1989 and 1990. A sea eagle from Coff's Creek had dieldrin levels of 26 mg/kg, DDT at 17 mg/kg and hepatchlor epoxide at 7.3 mg/kg. In 1990, fat from a Grafton osprey revealed DDT levels of 14 mg/kg[10].

These data suggest pockets of significant environmental contamination in Australia. Given the prolonged persistence of organochlorines in the soil, toxic effects on native fauna should be anticipated for many years to come.

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TABLE 1 - Reported mortalities in tawny frogmouths in North Shore suburbs of Sydney in July to October 1994 (WIRES data)

Suburb	Number of dead frogmouths
Avalon	2
Berowra	5
Brooklyn	2
Cammeray	2
Castle Hill	5
Cherrybrook	4
Cremorne	4
Danger Island	1
Epping	4
Frenchs Forest	2
Galston	4
Hornsby	6
Killara	6
Ku-ring-gai	1
Lane Cove	5
Lindfield	3
Neutral Bay	2
Normanhurst	2
Palm Beach	1
Pennant Hills	4
Pymble	7
Ryde	2
St Ives	6
Terrey Hills	3
Thornleigh	2
Turramurra	8
Wahroonga	6
Waitara	6
Westleigh	3
TOTAL	**108**

TABLE 2 - Organochlorine residues detected in brain and liver of tawny frogmouths and an owlet nightjar (Assays performed by the Residue Laboratory, Animal Health Laboratories, Department of Primary Industries, Yeerongpilly, Queensland)

	OXYCHLORDANE (mg/kg)	HEPTACHLOREPOXIDE (mg/kg)	DDE (mg/kg)	DIELDRIN (mg/kg)	TOTAL OC (mg/kg)
BIRD A					
S413042					
Liver	0.9	7.2	0.7	4.4	13.20
Brain	0.61	5.5	0.34	4.4	10.85
BIRD B					
S413039					
Liver	1.4	14.0	1.1	11.0	27.50
Brain	0.35	9.4	0.48	4.2	14.43
BIRD C					
S413041					
Liver	0.3	7.1	0.1	3.4	10.90
Brain	0.44	13.0	0.14	7.1	20.68
BIRD D					
S412672					
Liver	2.3	39.0	1.5	23.0	65.80
BIRD E					
S413037		14.0	1.0		
Liver	0.9	21.0	0.5	4.1	20.00
Brain	0.9			5.2	27.60
BIRD F					
S413035					
Liver	0.9	19.0	1.3	5.5	26.70
Brain	0.81	7.6	0.41	4.4	13.22
BIRD G					
S413040					
Liver	1.1	13.0	4.8	1.1	20.00
Brain	0.96	11.0	3.0	1.0	15.96
BIRD H					
S413038					
Liver	0.6	8.1	0.5	3.9	13.10
Brain	0.56	7.9	0.18	3.8	12.44
BIRD I					
S413036					
Brain	1.4	13.0	0.57	14.0	28.97
BIRD J					
S412671					
Nightjar		0.42			
Liver	0.13		0.03	0.09	0.67