Pesticide toxicity in Australian native birds

Fact sheet

November 2022

Introductory statement

There are increasing reports of toxicities associated with pesticide exposure in Australian wild birds. Although the majority of poisonings are due to exposure to organophosphates, in many cases the source of toxin is either unknown, or unconfirmed (Grillo 2011). Effects of toxins are well documented from studies on birds overseas, however there is limited information on the impacts of these compounds on Australian native birds.

This fact sheet summarises information on commonly reported toxic events in Australian wild birds, with an emphasis on native species. The information presented is based on data available in the National Wildlife Health Information System unless otherwise stated. These data have most recently been collated and reviewed by Grillo (2011) and Cox-Witton et al. (2014). Where available, information on common toxicities in introduced species is also included.

Sources of toxin

Agricultural chemicals, in particular insecticides and rodenticides, are the cause of most avian poisoning cases reported in Australia. Most poisonings are a result of exposure to organophosphate, organochlorine or rodenticide products. Avian taxa vary in their likelihood of exposure to chemical products, and in their susceptibility to toxic effects. Poisoning events in wild birds may result from:

- misuse of chemicals
- accidental or deliberate contamination of food sources
- illegal or inappropriate disposal of chemicals
- inadvertent primary and secondary poisoning of bystander species through pest species control programs.

McLeod and Saunders (2013) reviewed pesticides licensed for use in management of vertebrate pests in Australia (https://www.dpi.nsw.gov.au/biosecurity/vertebrate-pests/publications/pesticides-used-in-the-management-of-vertebrate-pests). Currently, alpha-chloralose and 4-aminopyridine are registered for use in management of pest birds in Australia (APVMA 2017).

Diagnosis

There are challenges in recording and confirming toxicity events in wildlife. Ideally, cases should be confirmed through a synthesis of available information including signalment, history, presenting signs, post mortem pathology and confirmed by the presence and concentration of toxin (Grillo 2011). Not all wildlife poisoning events are reported and, in some cases, there may be no suitable samples or carcasses for investigation. In some cases, testing options may be limited by availability of assays, cost of tests and lack of baseline information for the species in question. Strong circumstantial evidence, including known exposure to toxins and typical presentation, may be considered sufficient in many cases to conclude that an event occurred as a result of exposure to chemical toxins.

Organophosphate (and carbamate) toxicity

Sources

Organophosphate (OP) and carbamate pesticides are neuroactive compounds that act on the enzyme acetylcholinesterase. They are commonly used as miticides and insecticides used for agricultural, household and garden purposes. Commonly used OPs (either currently or in the past) include fenthion, fenamiphos, parathion, diazinon, malathion, famphur, phorate, terbufos and chlorpyrifos. Carbofuran, aldicarb and carbaryl are commonly used carbamates.

Although OPs degrade rapidly once in the environment; they can have significant acute toxic effects, particularly in bees and wildlife. Birds are considered particularly sensitive to the toxic effects of OPs. Exposure occurs either via the inhalation of aerosols; cutaneous absorption via contact with treated plants; or the ingestion of contaminated insects, carcasses or vegetation. In birds, secondary poisoning in carnivorous and insectivorous species occurs through predation or scavenging of contaminated carcasses or consumption of contaminated insects (McLeod and Saunders 2013).

Clinical signs

Exposure to OPs may result in acute signs of toxicity including excessive salivation, ataxia, dyspnoea, tremors, convulsions, paralysis, regurgitation and diarrhoea. High dose OP exposure usually results in death within hours. Poisoning may be unrecognised as birds are often found dead with an absence of signs associated with toxicity (Rose 2005). Birds that survive often recover within 48 hours. Sub-lethal or low concentration exposure to OP and carbamates may result in behaviour changes and chronic, decreased reproductive success including decreased chick survival (Fairbrother 1996).

Events in Australian wild birds

The majority of recently reported avian poisoning events in Australian have been caused by OPs, including fenitrothion, fenthion ethyl, fenamiphos and diazinon (see Appendix 1, tables 2 and 3)

(Grillo and Post 2010; Grillo 2011; Grillo et al. 2014). Reported events include both deliberate and accidental exposures (Du Guesclin et al. 1983).

Products containing fenthion include those formerly registered for use in Australia by the APVMA for control of feral birds, specifically rock pigeons (*Columba livia*), house sparrows (*Passer domesticus*), European common starlings (*Sturnus vulgaris*), and Indian mynahs (*Acridotheres tristis*). Such products were restricted for use by authorised personnel and carried label conditions aimed at reducing the impact on non-target species. Following concerns about toxicity, occupational health and safety, residues in food, and environmental and trade aspects, the registration and approvals of fenthion were reviewed by the APVMA, who concluded that "the use of products containing fenthion may, in most situations, pose undue risks to human health (via dietary and occupational exposure) and the environment." Following the review, the registration of all fenthion products, including three products for control of exotic bird species, was cancelled in 2014 (APVMA 2014). Despite this, intoxications continue to be reported (perhaps because stocks of OPs remain on premises), although the rate of reporting appears reduced.

Hundreds of birds may be affected in one poisoning event, in particular if poisoning has been deliberate. OPs may be detected in seed deliberately placed in the area where the birds are known to feed, and OPs may be found in gastrointestinal contents.

Some of the wild Australian bird species involved in confirmed or suspected OP toxicities are listed in Table 3 (those species specifically involved in fenthion poisoning events). Other species include[1]:

- silvereyes (*Zosterops lateralis*)
- eastern rainbow lorikeets (*Trichoglossus haematodus moluccanus*)
- little penguins (*Eudyptula minor novaehollandiae*)
- egrets (*Egretta* sp.)
- spur-winged plover (*Vanellus spinosus*)
- plumed whistling duck (*Dendrocygna eytoni*)
- wood duck *(Chenonetta jubata*)
- Carnaby's black cockatoo (*Calyptorhynchus latirostris*)
- other unidentified duck species
- some species of unidentified insectivorous birds.

Sampling, diagnosis and toxicology

Samples of blood, brain, liver, kidney and ingesta may be collected to assay for OP concentrations by mass spectrometry and/or HPLC. Seed and gastrointestinal samples may also be collected for assay. Tissue and ingesta samples must be stored in aluminium foil prior to freezing and may be

[1] McIlroy (1985); Forshaw (1991); McKenzie (1991); Glastonbury (1992); McKenzie et al. (1996); Gordon and Field (2006); Vaughan-Higgins et al. (2016), Animal Health Surveillance Quarterly Reports 2010-2017.

stored at -80°C for up to six months prior to testing. Contact your state/territory animal health laboratory for advice on sample collection.

There are no gross or microscopic lesions specific to OP toxicity. It may be suspected upon gross examination when birds are found dead in good body condition and with no significant lesions. Secondary observations such as pulmonary oedema, pulmonary haemorrhage, mild haemorrhagic enteritis, gastroenteritis and sometimes degenerated liver and kidneys may indicate OP poisoning (Glastonbury 1992; Michigan Department of Natural Resources 2015). Acetylcholinesterase (AChE) activity can be measured in the blood or brain tissue (Pohanka et al. 2011). These assays may be costly and results may be difficult to interpret in the absence of species- and laboratory-specific reference ranges (Vaughan-Higgins et al. 2016).

Treatment

Survival rates in affected birds may be low. Atropine, pralidoxime (2-PAM) and diazepam may be used to reverse the paralysis of respiratory muscles induced by OPs. Supportive treatment including fluid therapy and nutritional support may lead to a higher survival rate in clinically affected birds (Grillo et al. 2014).

Organochlorine toxicity

Source

Organochlorines (OCs) are a diverse group of chemicals; their toxicity, potential to build up in tissues and environmental persistence varies. OCs were used in Australia from the 1940s until the 1980s in many commercial products, to protect crops, livestock, buildings and households from damaging by insects, including termites and ants. Almost all OCs are now deregistered and listed as scheduled wastes.

Many OCs resist degradation and can remain in the environment for many years. They are toxic to humans and other animals, highly toxic to most aquatic life and can have serious short- and long-term toxic effects, even at low concentrations. Poisoning of birds occurs when they become exposed to the toxin through inhalation, percutaneous absorption or ingestion. Chronic effects of OCs may be significant and include immune system and reproductive damage. OCs accumulate in fat tissue and other organs and may be liberated into the host's body when fat stores are metabolized (Rose 2005). Birds and other animals high up the food chain (e.g. birds of prey and humans) may accumulate higher levels of the pesticides than animals lower in the food chain.

It is expected that poisoning events associated with OCs in Australia will gradually diminish as environmental levels continue to fall slowly over time.

Events in Australian wild birds

Organochlorine compounds implicated in Australian wild bird poisoning events include methoxychlor, aldrin, dieldrin, chlordane, toxaphene, lindane, DDT, DDE and heptacholor epoxide

and endrin (see Appendix 1). Residues of dieldrin, for example, are known to persist in the environment, although its production, importation and use in Australia have been banned since the 1980s (McKenzie et al. 1982; McKenzie et al. 1996; Gordon and Field 2006; Ladds 2009). Elevated OC levels have been found in a range of native Australian birds species, including little crows (*C. bennetti*) and galahs in the NT and a range of duck species from different areas of NSW (Best 1973; Briggs 1981), although in many cases a link to toxic effects has not been confirmed, and some studies are now several decades old.

Eggshell thinning, with presumed resultant negative effects on chick survival rates, have been reported in a range of raptor species, following OC exposure, across Australia (Olsen and Olsen 1979; Pruett-Jones et al. 1981; Olsen et al. 1993; Falkenberg et al. 1994).

More recently, wild white-bellied sea eagles (*Haliaeetus leucogaster*) from Homebush Bay in Sydney were shown to have 60 times the dioxin levels that are considered harmful to birds in general. It is believed the animals bio-accumulated residual chemicals from the environment, most probably via food sources. Birds were found dead, and a range of potentially toxic chemicals were found in the carcases. The exact cause of death could not be determined (Manning et al. 2008).

OC toxicity, resulting in clinical disease and mortality in tawny frogmouths (*Podargus strigoides*), has been shown to occur in the Sydney region, beginning in late winter or early spring and persisting for an outbreak period of 4-5 weeks (Charles 1995; Rose 2005). The suspected sources of OCs include contaminated soil and domestic and agricultural residues accumulated in mice, invertebrates and insects (which comprise the diet of the tawny frogmouth). It is believed that intoxication occurs when available food supplies diminish seasonally and birds metabolise fat stores, leading to elevation of OC levels in the body.

Australian pelicans (*Pelecanus conspicillatus*) have been adversely affected by OC in southeast Qld (McKenzie et al. 1996; Gordon and Field 2006). In one investigation, eight of 24 pelicans found dead or with clinical signs of poisoning along the south coast of Qld tested positive for dieldrin. Contaminated fish, with elevated levels of dieldrin in their tissues, were confirmed as the source (McKenzie et al. 1982).

Clinical signs

OCs affect the parasympathetic nervous system and acute signs of toxicity in birds are primarily neurological. These include behavioural changes, weakness, inability to fly, head tilt, dilated pupils, impaired vision, leg extension, hyper-excitability, central nervous system depression, drooping wings, flicked-up tails, convulsions in response to external noise stimulation, screeching vocalization, jerky movements and death. Sub-lethal doses of organochlorines have long-term effects on reproduction due to decreased eggshell quality, embryo death and/or chick deformity (Olsen and Olsen 1979; Olsen et al. 1993; Falkenberg et al. 1994; Blus et al. 1996; Ladds 2009).

Sampling, diagnosis and toxicology

The carcase may be emaciated, with general muscle pallor and reduced liver size. Pelicans with dieldrin toxicity displayed poor body condition [along with moderate to high burdens of nematodes and cestodes] (Gordon and Field 2006). Experimental intoxication of birds with OCs resulted in congestion in the lungs, liver, pancreas and kidneys (Ladds 2009).

Toxicological analysis can detect presence or accumulation of OCs in liver, brain, fat and ingesta. Detection of OCs is by either mass spectrometry or high-performance liquid chromatography (HPLC). Samples may be stored in aluminium foil and frozen until testing is performed. Contact your state/territory animal health laboratory for advice on sample collection.

Treatment

There are no products capable of removing OCs from the system of intoxicated animals. Treatment is supportive and fluid therapy and warmth are the recommended treatments for OC toxicity in birds. Birds with seizures can be treated with intravenous or intramuscular diazepam (a central nervous system sedative) (Charles 1995). Over-stimulation of the parasympathetic nervous system may be countered by the administration of small doses of atropine (Blus et al. 1996).

Rodenticide toxicity

Rodenticides used in broadacre agriculture primarily include the non-anticoagulant pesticide zinc phosphide (coated onto grains) and the anticoagulant coumatetralyl (in covered bait stations). **Primary toxicity** can occur in non-target wildlife (e.g. galahs, cockatoos) that consume zinc phosphide baited material[2] . This risk is greatly reduced when the product is used strictly in accordance with the label directions (NRA 2000). **Secondary toxicity** in non-target wildlife can also occur with anticoagulant rodenticides due to accumulation through the food chain, leading to lethal doses ingested by birds of prey and other animals that feed on sick or dead mice (e.g. magpies, kookaburras, quolls and goannas). Information on the use of anticoagulants during mouse plagues is available from the APVMA (APVMA 2021). Monitoring the exposure of wildlife to rodenticides is critical to understanding the impacts of these compounds and assessing the effectiveness of any regulatory changes.

Anticoagulant rodenticides

Background

Anticoagulants are used in Australian as lethal pest control methods for introduced rodents (rats and mice). Anticoagulant poisons are classified as first generation or second-generation products; first-generation poisons are less toxic and require several feeding events over several days to kill a

[2] Primary toxicity due to direct consumption of anticoagulant and non-anticoagulant rodenticide baits is also a risk for some mammals (e.g. possums and native rodents).

rodent, whereas second generation anticoagulant poisons are much more toxic and may kill a rodent after only one feeding event.

Sources and poisoning

Anticoagulant toxicity in wild birds generally occurs as a secondary event in non-target species. Anti-coagulant rodenticides include warfarin, coumatetralyl, diphacinone, brodifacoum and bromadiolone. These are the main ingredients in many domestic, commercial, industrial and agricultural rodent baits (around agricultural buildings, not for use in crops) and are generally not approved for use in crops because they do not meet safety requirements, specifically in relation to residues and the environment. Although all bird species may be affected, toxicity varies with species and the type of anticoagulant involved. Second generation anticoagulant rodenticides containing bromadiolone and brodifacoum have been implicated in the majority of wild Australian bird anticoagulant poisoning events.

Wild raptors such as barn owls (*Tyto alba*) and kestrels (*Falco* sp.) may acquire secondary anticoagulant toxicity via consumption of deliberately poisoned rabbit or rodents. Poisoning of Pacific black ducks (*Anas superciliosa*) has also been reported (Mason 1985).

Clinical signs, diagnosis and toxicology

Toxicity results in blood loss, general pallor of viscera and muscles, and free blood may be present in the abdominal cavity and mouth. Superficial wounds may be seen on the legs and feet (Ladds 2009). Diagnosis of anticoagulant toxicity is dependent on the detection of poison in the ingesta by HPLC or by measurement of clotting parameters such as prothrombin time in blood. However, clotting parameters in birds can be variable and species specific (Hindmarch et al. 2019), with clot formation often delayed compared to mammals (Strindberg et al. 2015). Assessment of packed cell volume (PCV) and timing of whole blood clotting in a serum collection tube have been recommended when assessing raptors admitted to care (Hindmarch et al. 2019).

Treatment

Vitamin K therapy is used to treat anticoagulant poisoning in birds and other animal species. It was administered to an experimentally intoxicated wedge-tailed eagle (*Aquila audax*) 15 days after dosing with pindone (a commonly utilised anticoagulant rodenticide in agricultural settings) with complete recovery (Martin et al. 1994; NRA 2002).

Non-anticoagulant rodenticides - zinc phosphide

Background

Zinc phosphide is generally used in Australia to control mice in and around grain silos, and during mouse plagues it may be applied, under permit, more extensively in crops (NRA 2000; APVMA 2021). It is usually coated onto grains for use. When ingested, zinc phosphide reacts with acidic conditions in the stomach to generate phosphine gas, which distributes rapidly throughout the body and can result in hypoxia and eventual death (USDA 2017).

Sources and poisoning

Avian casualties can occur where baited grain is consumed by birds. The susceptibility to zinc phosphide toxicity varies between bird species [e.g. pheasants are more sensitive than quail (Ramey and Sterner 1995)]. Zinc phosphide is likely to be very highly toxic (LD50 < 10 mg/kg) for sensitive bird species such as geese and galliforms and highly toxic (LD50 < 50 mg/kg) to many other bird species (NRA 2000). However, many bird species can distinguish and avoid zinc phosphide baits or regurgitate the toxicant.

Clinical signs, diagnosis and toxicology

Zinc phosphide is toxic to the heart, liver, and kidneys. After a lethal dose is ingested, death results from heart and kidney failure within 24 hours. Animals may become prostrate with deep slow respiration, terminating in convulsions. Lethal secondary poisoning is less likely as toxic residues are mainly confined to the alimentary tract, but sublethal effects may occur in secondary consumers (NRA 2000). Sublethal impacts in various bird studies include reduced weight, lethargy, and ataxia (USDA 2017). Chickens that died after consuming zinc phosphide had severe pulmonary oedema and congestion of the lung, heart, liver and kidney (Tiwary et al. 2005) and haemorrhage in the lungs and visceral organs (Muraina et al. 2018). Suspect carcasses should not be opened and should be frozen prior to submission for diagnosis.

Treatment

There is no antidote for zinc phosphide toxicity, but some animals may survive with supportive care.

Control and prevention

Agricultural chemicals are registered by the Australian Pesticides and Veterinary Medicines Authority (APVMA, http://apvma.gov.au). These chemicals may also be used under a minor use permit. Information aimed at reducing or eliminating risks of adverse effects in wildlife is included in both the product labels and in the necessary permits for use of products.

The risk of primary and secondary toxicity to wildlife due to commonly available rodenticide chemicals can be reduced by following the label instructions. Information on rodenticides approved for use in crop situations is available from the APVMA (NRA 2000; APVMA 2021). For example, bromadiolone bait labels state that they must not be placed in the open, but rather must be placed in and around buildings (within 2 metres) or enclosed spaces (e.g. drains), and carcasses of affected rodents must be collected and disposed of appropriately.

The Adverse Experience Reporting Program (AERP), administered by the APVMA, assesses reports of adverse experiences associated with the registered use of agricultural chemicals. State and territory regulators also enforce appropriate use of agricultural chemicals in Australia and report any adverse events directly to the APVMA. Reports mostly concern production or domestic animals, however some may involve wildlife; the most common of these are poisonings.

Surveillance

Wildlife disease surveillance in Australia is coordinated by the Wildlife Health Australia. The National Wildlife Health Information System (eWHIS) captures information from a variety of sources including Australian government agencies, zoo and wildlife parks, wildlife carers, universities and members of the public. Coordinators in each of Australia's States and Territories report monthly on significant wildlife cases identified in their jurisdictions. NOTE: access to information contained within the National Wildlife Health Information System dataset is by application. See the WHA website for more information: https://wildlifehealthaustralia.com.au/ProgramsProjects/eWHIS-WildlifeHealthInformationSystem.aspx.

As part of Australia's general surveillance system, cases involving intoxications of wildlife may also be reported directly to WHA. In these instances, WHA will collect these reports (for both confirmed and suspected cases) and liaise with the AERP regarding investigation of the incidents. The reporting of intoxications in wildlife by WHA does not replace the processes already in place within each state and territory for management of adverse reactions; it provides an additional level of confidence that all relevant events are captured by the AERP database. The AERP identifies any duplicate records.

There are numerous reports of wild bird toxicities in the national database. Appendix 1 provides an example of the variety of wild bird species and chemical products linked to poisoning events reported to WHA for one year (2010). We encourage those with information on wild bird poisoning events and laboratory confirmed cases of this condition to submit this information to the national system for consideration for inclusion in the national database.

Research

Investigation and reporting of mortality events associated with poisoning in birds can provide useful data to better understand the circumstances and susceptibilities of affected native wildlife.

Work is required to better understand:

- which species and groups of native birds are most susceptible to chemical exposure in Australia, and why (e.g. species-specific susceptibility to toxins or increased exposure)
- exposure pathways
- diagnostic tests
- treatment options
- options for use of alternative chemicals with lowered risk of avian toxicity.

Human health implications

Many of the compounds causing toxicity in birds can also result in effects in humans, however in most cases humans are unlikely to be exposed to the chemicals through the same avenues as birds.

Proper use of pesticides is required to prevent the risk of transmission through the food chain, which can present a risk to both human health and trade.

There are serious occupational health and safety considerations when conducting post-mortem examinations on wildlife that are suspected to have died from zinc phosphide toxicity, as phosphine gas (toxic on inhalation) can be released from the carcass upon opening the stomach. Advice about how to manage this risk is available from the NSW EPA (EPA 2021).

Conclusion

There are increasing reports of toxic events in Australian wild birds, following exposure to agricultural chemicals. Exposure may be malicious or accidental and is under recognised. Widespread environmental use of pesticides due to situations such as mouse plagues can greatly increase the risk of wildlife exposure and toxicity events. Further work is required to better understand and manage the risks to Australia's native fauna of exposure to agricultural chemicals. Agricultural chemicals should only be used for the purposes for which they are approved, and operators should follow guidance and requirements provided by APVMA. Reporting of confirmed or suspected poisoning events in native fauna is always recommended and is often a stated requirement under the permit system.

Appendix 1: Summary of avian poisoning events reported to WHA

Table 1. Wild bird poisoning events in Australia reported to WHA in 2010 [from Grillo (2011)]

Avian order	Number of events*	Species
Psittaciformes	7	Long-billed corella (*Cacatua tenuirostris*) Galah (*Cacatua roseicapilla*) Red-rumped parrot (*Psephotus haematonotus*)
Passeriformes	4	Australian magpie (*Gymnorhina tibicen*) Torresian crow (*Corvus orru*)
Columbiformes	3	Rock (feral) pigeon (*Columba livia*) Topknot pigeon (*Lopholaimus antarcticus*) Crested pigeon (*Ocyphaps lophotes*)
Anseriformes	2	Wood duck (*Aix sponsa*) Pacific black duck (*Anas superciliosa*) Mallard duck - wild form (*Anas platyrhynchos*)
Charadriiformes	1	Kelp gull (*Larus dominicanus*) Skua (family *Stercorariidae*)
Procellariiformes	1	Northern giant petrel (*Macronectes halli*) Southern giant petrel (*Macronectes giganteus*)

* Four events involved more than one order.

Table 2. Toxins implicated in 2010 wild bird poisoning events [from (Grillo 2011)]

Toxin class	Toxin	Confirmed events	Suspected events
Organophosphate	Fenthion ethyl	3*	-
	Fenitrothion	2	-
	Chlorpyrifos	1	-
	Diazinon	-	1
	Undetermined	-	1
Organophosphate/ Organochlorine	Undetermined (not tested)	-	2
Carbamate	Methomyl/ methomyl oxime	2	-
Anticoagulant	Brodifacoum	1	-
Not known	Undetermined (mouse baits)	-	1

– = not reported

* In one event, DDE, dieldrin and heptachlor epoxide were also detected (background levels).

Table 3. Bird species involved in fenthion poisoning events in Australia, April 2009 to March 2014 [from (Cox-Witton et al. 2014)]

Order	**Species**
Anseriformes	Magpie goose (*Anseranas semipalmata*)
Accipitriformes	Whistling kite (*Haliastur sphenurus*)
Charadriformes	Silver gull (*Larus novaehollandiae*)
Columbiformes	Pigeon (*Columbidae*) Spotted turtle-dove (*Streptopelia chinensis*)* Topknot pigeon (*Lopholaimus antarcticus)*
Coraciiformes	Laughing kookaburra (*Dacelo novaeguineae*)
Ciconiiformes	Australian white ibis (*Threskiornis molucca*)
Passeriformes	Apostle bird (*Struthidea cinerea*) Australian magpie (*Gymnorhina tibicen)* Black-faced cuckoo-shrike (*Coracina novaehollandiae*) Butcherbird (*Cracticus* sp.) Chestnut-breasted mannikin (*Lonchura castaneothora*) Honeyeater (*Meliphagidae*) House sparrow (*Passer domesticus*)* Magpie lark (*Grallina cyanoleuca*) Noisy miner (*Manorina melanocephala*) Olive-backed sunbird (*Nectarinia jugularis*) Pied currawong (*Strepera versicolor*) Raven (*Corvus* sp.) Red-browed finch (*Aegintha temporalis*) Torresian crow (*Corvus orru)*
Pelecaniformes	Pelican (*Pelecanus* sp.)
Psittaciformes	Corella (*Cacatua* sp.) Galah (*Cacatua roseicapilla*) King parrot (*Alisterus scapularis*) Little corella (*Cacatua sanguinea*) Long-billed corella (*Cacatua tenuirostris)* Rainbow lorikeet (*Trichoglossus haematodus*) Sulphur-crested cockatoo (*Cacatua galerita)*
Strigiformes	Southern boobook (*Nino novaeseelandiae*)

*Non-native species

Note: This table includes individual and mixed-species events. In some mixed-species events, the species listed may not have been individually tested, but were reported as part of a mortality event where other submitted birds tested positive for fenthion.

References

APVMA, (2014) APVMA Gazette. Australian Government, Canberra, ACT. 16 October 2014

APVMA (2017) 'Australian Pesticides and Veterinary Medicines Authority.' Available at https://apvma.gov.au/ [Accessed 5 May 2017].

APVMA (2021) 'Bromadiolone and the current mouse plague.' Available at https://apvma.gov.au/node/87226 [Accessed 25 July 2021].

Best SM (1973) Some organochlorine pesticide residues in wildlife of the Northern Territory, Australia, 1970-71. *Australian Journal of Biological Sciences* **26**, 1161-1170.

Blus L, Weimyer S, Henny C (1996) Organochloride pesticides. In 'Non-infectious diseases of wildlife.' (Eds A Fairbrother, L Locke, G Hogg.) pp. 61-70. (Iowa State University Press: Ames, Iowa).

Briggs S (1981) Possible medical impacts of waterbirds on humans – three examples. In '4th International Conference of the Wildlife Disease Association. Sydney, Australia'. (Ed. ME Fowler) pp. 25-32. (Wildlife Disease Association).

Charles J (1995) Organochlorine toxicity in tawny frogmouths, Proceedings of the Australian Committee of the Association of Avian Veterinarians. Dubbo, NSW. (Association of Avian Veterinarians)

Cox-Witton K, Grillo T, Post L (2014) Wild bird poisoning events — fenthion detected. *Animal Health Surveillance Quarterly Report* **19**, 7-8.

Du Guesclin P, Emison W, Temby I (1983) Deliberate misuse of organophosphorus pesticide, fenthion-ethyl, to poison birds in Victoria. *Corella*

EPA (2021) 'Guidance on handling native wildlife in areas where zinc phosphide has been deployed.' Available at https://www.epa.nsw.gov.au/-/media/epa/corporate-site/resources/news/21p3074-handling-wildlife-where-zinc-phosphide-used.pdf

Fairbrother A (1996) Cholinesterase inhibiting pesticides. In 'Non-infectious diseases of wildlife.' (Eds A Fairbrother, L Locke, G Hogg.) pp. 52-60. (Iowa State University Press: Ames).

Falkenberg I, Dennis T, Williams B (1994) Organochlorine Pesticide Contamination in Three Species of Raptor and Their Prey in South Australia. *Wildlife Research* **21**, 163-173.

Forshaw D (1991) Organophosphate poisoning and gastric helminthiasis in ravens and magpies. Animal Health Laboratory, Perth, WA.

Glastonbury J (1992) Ethyl fenthion toxicity in birds. Australian Society for Veterinary Pathology.

Gordon A, Field H (2006) A Cross-Jurisdiction Model for Targeted Surveillance of Wild Bird Species. DAFF, Canberra. Available at https://www.agriculture.gov.au/sites/default/files/sitecollectiondocuments/animal-plant/emergency/wildlifeexoticdiseaseprogram/08-09/cross-jur-wild-bird-surveillance.pdf.

Grillo T (2011) Poisoning events in wildlife. *Animal Health Surveillance Quarterly Report* **16, 2**, 8-9.

Grillo T, Cox-Witton K, Post L (2014) Australian Wildlife Health Network Report. *Animal Health Surveillance Quarterly Report* **18, 3**, 5-7.

Grillo T, Post L (2010) Australian Wildlife Health Network Report. *Animal Health Surveillance Quarterly Report* **15**, 5-8.

Hindmarch S, Rattner BA, Elliott JE (2019) Use of blood clotting assays to assess potential anticoagulant rodenticide exposure and effects in free-ranging birds of prey. *Science of the Total Environment* **657**, 1205-1216.

Ladds P (2009) 'Pathology of Australian Native Wildlife.' (CSIRO Publishing: Melbourne).

Manning T, Ross GA, Symons R (2008) Environmental contaminants in white-bellied sea-eagles (*Haliaeetus leucogaster*) found in Sydney, Australia. *Australasian Journal of Ecotoxicology* **14**, 21.

Martin G, Kirkpatrick W, King D, Robertson I, Hood P *et al.* (1994) Assessment of the potential toxicity of an anticoagulant, pindone (2-Pivalyl-1, 3-Indandione), to some Australian birds. *Wildlife Research* **21**, 85-93.

Mason R (1985) Suspect rodentidife poisoning in ducks. Australian Society for Veterinary Pathology.

McIlroy J (1985) Observations on the sensitivity of some Australian birds and the feral pig to the organophosphorus insecticide, fenthion ethyl. *Wildlife Research* **12**, 331-335.

McKenzie R (1991) Diazinon poisoning in sacred ibis (*Threskiornis molucca*). Australian Society for Veterinary Pathology.

McKenzie R, Freudigmann C, Mawhinney H, Eaves L, Green P *et al.* (1982) Dieldrin poisoning and botulism in Australian pelicans (*Pelecanus conspicillatus*). *Australian Veterinary Journal* **58**, 148-152.

McKenzie R, Lanham A, Taylor J, Gibson J, Pierce R (1996) Fenthion poisoning of native birds in southern Queensland. *Australian Veterinary Journal* **74**, 321-323.

McLeod L, Saunders G (2013) Pesticides used in the management of vertebrate pests in Australia: A review. (Ed. DoP Industries.) (Department of Primary Industries: Orange, NSW). Available at https://www.dpi.nsw.gov.au/biosecurity/vertebrate-pests/publications/pesticides-used-in-the-management-of-vertebrate-pests [Accessed 5 May 2017].

Michigan Department of Natural Resources (2015) 'Organophosphate Toxicity.' Available at http://www.michigan.gov/dnr/0,1607,7-153-10370_12150_12220-27249--,00.html [Accessed 29 Sept 2015].

Muraina IA, Oladipo OO, Akanbi OB, Shallmizhili JJ, Gyang MD *et al.* (2018) Phosphine poisoning in free-range local chickens: a case report. *Veterinary Medicine and Science* **4**, 263-268.

NRA (2000) Public Release Summary on Evaluation of the new active zinc phosphide in the product Mouseoff zinc phosphide bait. National Registration Authority for Agricultural and Veterinary Chemicals, Canberra. Available at https://apvma.gov.au/sites/default/files/publication/14101-prs-zinc-phosphide.pdf.

NRA (2002) The NRA Review of Pindone. Available at https://apvma.gov.au/sites/default/files/publication/14856-pindone-review-final-report.pdf.

Olsen P, Fuller P, Marples T (1993) Pesticide-related eggshell thinning in Australian raptors. *Emu* **93**, 1-11.

Olsen P, Olsen J (1979) Eggshell Thinning in the Peregrine, *Falco peregrinus* (Aves: Falconidae), in Australia. *Wildlife Research* **6**, 217-226.

Pohanka M, Hrabinova M, Kuca K, Simonato J-P (2011) Assessment of acetylcholinesterase activity using indoxylacetate and comparison with the standard Ellman's method. *International Journal of Molecular Sciences* **12**, 2631-2640.

Pruett-Jones S, White C, Emison W (1981) Eggshell thinning and organochlorine residues in eggs and prey of peregrine falcons from Victoria, Australia. *Emu* **80**, 281-287.

Ramey C, Sterner R (1995) Mortality of gallinaceous birds associated with 2% zinc phosphide baits for control of voles in alfalfa. *International biodeterioration & biodegradation* **36**, 51-64.

Rose K (2005) Common Diseases of Urban Wildlife: Birds. (Australian Registry of Wildlife Health: Sydney). Available at https://arwh.org/wp-content/uploads/2021/06/COMMON-DISEASES-OF-BIRDS-2021-FINAL.pdf [Accessed 4 Nov 2022].

Strindberg S, Nielsen TW, Ribeiro ÂM, Wiinberg B, Kristensen AT *et al.* (2015) Thromboelastography in selected avian species. *Journal of Avian Medicine and Surgery* 282-289.

Tiwary AK, Puschner B, Charlton BR, Filigenzi MS (2005) Diagnosis of zinc phosphide poisoning in chickens using a new analytical approach. *Avian Diseases* **49**, 288-291.

USDA (2017) Human Health and Ecological Risk Assessment for the Use of Wildlife Damage Management Methods by USDA-APHIS-Wildlife Services In 'The Use of Zinc Phosphide in Wildlife Damage Management.' (Ed. USDA.) (USDA:

Vaughan-Higgins R, Vitali S, Reiss A, Besier S, Hollingsworth T *et al.* (2016) Development of reference ranges for plasma total cholinesterase and brain acetylcholinesterase activity in free-ranging Carnaby's black-bockatoos (*Calyptorhynchus latirostris*). *Journal of Wildlife Diseases* **52**, 592-598.

Acknowledgements

We are extremely grateful to the many people who had input into this fact sheet. Without their ongoing support production of these fact sheets would not be possible.

Updated: November 2022

To provide feedback on this fact sheet

Wildlife Health Australia would be very grateful for any feedback on this fact sheet. Please provide detailed comments or suggestions to admin@wildlifehealthaustralia.com.au. We would also like to hear from you if you have a particular area of expertise and would like to produce a fact sheet (or sheets) for the network (or update current sheets). A small amount of funding is available to facilitate this.

Disclaimer

This fact sheet is managed by Wildlife Health Australia for information purposes only. Information contained in it is drawn from a variety of sources external to Wildlife Health Australia. Although reasonable care was taken in its preparation, Wildlife Health Australia does not guarantee or warrant the accuracy, reliability, completeness or currency of the information or its usefulness in achieving any purpose. It should not be relied on in place of professional veterinary or medical consultation. To the fullest extent permitted by law, Wildlife Health Australia will not be liable for any loss, damage, cost or expense incurred in or arising by reason of any person relying on information in this fact sheet. Persons should accordingly make and rely on their own assessments and enquiries to verify the accuracy of the information provided.

Wildlife Health AUSTRALIA

Find out more at www.wildlifehealthaustralia.com.au

Email admin@wildlifehealthaustralia.com.au

Or call +61 2 9960 6333