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# 3. Toxins no longer used by the Department of Conservation

## 3.1 PHOSPHORUS

Chemical Name: $P_4$.

Synonym: Phosphorus.

Phosphorus is used as a paste and is generally applied to turf spits on the ground. It is only available to licensed operators.

### 3.1.1 Physical and chemical properties

Phosphorus is a yellow solid with a waxy lustre that has a melting point of 44.1°C. Phosphorus is mixed with water, bentonite, and magnesium oxide to produce an emulsion that is incorporated into a fruit paste for the control of rabbits and possums.

Raw phosphorus is a corrosive dangerous product. Pastes have somewhat different properties.

### 3.1.2 Historical development and use

Phosphorus was first used in rabbit control in New Zealand and Australia in the early 1920s. The initial use of this toxicant was in pollard pellets by dissolving the phosphorus sticks in carbon bi-sulphide or mixing phosphorus in boiled water and then adding pollard to make the pellets. It was also used on oats and wheat.

In the 1950s phosphorus was incorporated into paste for rabbit control. In the 1960s phosphorus pellets were withdrawn from the market because the phosphorus broke down (oxidised) quickly in the bait, and was ineffective.

Phosphorus paste is still used by regional councils and is publicly available. Under the Pesticides (Vertebrate Pest Control) Regulations 1977, the operator must either hold a licensed operators certificate or be working under the supervision of a certificate holder to use phosphorus for pest control.

### 3.1.3 Fate in the environment

Phosphorus is unlikely to be persistent in the environment. Phosphorus is usually added to paste bait for possum control. On exposure to air the phosphorus oxidises to phosphates, which are not poisonous. Accordingly, phosphorus is more stable in paste, which tends to ‘cake’ and protect the phosphorus from oxidation.

### 3.1.4 Toxicology and pathology

#### *Onset of signs*

In the veterinary literature, phosphorus poisoning is usually categorised in three phases:

- An acute initial phase occurring within hours of ingestion characterised by gastrointestinal, abdominal, and circulatory signs. Initial signs generally involve vomiting and diarrhoea. If the dosage is sufficiently large, shock, cyanosis, inco-ordination and coma may develop, with death occurring before the second and third phases appear.
- An interim or latent phase with apparent recovery occurs at lower doses approximately 48 hours to several days after initial clinical signs.
- The third stage is characterised by recurrence of marked clinical signs involving the gastrointestinal tract. Liver failure then occurs.

These literature reports suggest that death may occur in 1–2 days, or there may be improvement for 1–2 days before vomiting, diarrhoea, and other signs return. Death is usually due to liver necrosis and heart failure. There may be a delay of up to 3 weeks after ingestion before convulsions, coma, and death. Recent trials at Landcare Research have shown that possums eating phosphorus-paste baits die within 18 hours and do not experience the prolonged toxicosis commonly attributed to phosphorus in the scientific and veterinary literature (Eason et al. 1997, 1998b; O'Connor et al. 1998).

There is no antidote to phosphorus, but with early diagnosis the poison may be removed by vomiting or gastric lavage, then treated with 0.1% potassium permanganate or 2% hydrogen peroxide (to oxidise the toxicant to harmless phosphates) and mineral oil (which prevents absorption). However, if there is bleeding or ulceration treatment is more difficult.

#### *Mode of action*

The mode of action is unknown. It has not been possible to associate the main clinical or pathological features of intoxication with inhibition of any particular enzyme or class of enzymes. Phosphorus is sometimes referred to as a protoplasmic poison, but it is difficult to distinguish its possible direct effects on the liver, kidney, brain, and heart from the effects of anoxia on those organs. The peripheral vascular dilatation, which is the first and most pervasive systemic effect of phosphorus, contributes to all the disorders that may be seen in various organs. However, the mechanism of this dilatation is not clear.

Phosphorus not only leads to structural damage of vital organs, but also produces serious disruption of their metabolic function, as evidenced by hypoglycemia, azotemia, inhibition of glycogen formation in the liver, and many other disorders.

#### *Pathology and regulatory toxicology*

Pathological changes include gross evidence of fatty degeneration and swollen livers, as well as gastrointestinal irritation, necrosis, and haemorrhage. If death is sufficiently prompt, there is no pathology except irritation of the oesophagus and stomach. Perforation may occur. Following survival for several days, fatty degeneration is striking in the liver, heart, and kidney but may be found in all organs, including the brain. We were unable to locate any material relating to

genotoxicity or teratogenicity, or data from other regulatory toxicology studies on phosphorus.

### *Fate in animals*

Phosphorus is readily absorbed but its persistence in lethally and sub-lethally poisoned possums has not been elucidated.

### *Species variation in response to phosphorus*

There is little species variation in response to phosphorus and most species are at risk if they eat bait (Table 23).

TABLE 23. ACUTE ORAL TOXICITY ($LD_{50}$mg/kg) OF PHOSPHORUS (Hone & Mulligan 1982).

| SPECIES | $LD_{50}$ (mg/kg) |
|---|---|
| Sheep | 1 |
| Pig | 1–6 |
| Rabbit | 4 |
| Dog | 3–6 |
| Cat | 3–6 |
| Possum | 6–10 |
| Poultry (unspecified) | 10 |

### *Aquatic toxicology*

Unknown. It is unlikely that significant amounts of phosphorus baits used for possum control will enter watercourses.

## 3.1.5 Current use

This poison has been in use since the 1920s and is one of the few poisons that is still available to the public as an acute poison for rabbit and possum control. It is also still used in some instances to poison pigs. It is not currently used by the Department of Conservation, but is used around houses and public areas by regional councils where there is a risk to dogs from 1080. However, use of phosphorus is also associated with secondary poisoning of dogs.

| Advantages | Disadvantages |
|---|---|
| Effective (kills of >90% achieved) | Has some animal welfare concerns‡ |
| Less public opposition than with 1080† | Secondary poisoning risk to dogs and birds |
| | Risk of fire |
| | Antidotes of limited value |

† When farmers or the community oppose the use of 1080 they will often accept phosphorus as a replacement.

‡ Studies show that the symptoms of phosphorus poisoning in possums differ from those reported in the veterinary literature for other animals

## 3.2 ARSENIC

Chemical Name: Arsenic trioxide ($As_2 O_3$).

Synonyms: White arsenic, arsenous oxide.

### 3.2.1 Physical and chemical properties

Arsenic is a naturally occurring toxin found in combination with other metals, particularly iron as arsenic pyrite (FeAs), and iron-arsenic sulphide. Two other forms of arsenic sulphide, orpiment and regular white arsenic (arsenic trioxide), are less common.

### 3.2.2 Historical development and use

The first recorded use of arsenic in New Zealand and Australia was in the 1880s where it was used in a variety of baits including oats, wheat, root crops, apples, and pollard pellets.

In the early 1970s arsenic was discontinued from use in rabbit control due to its inability to decompose. In addition to this, a number of people were reported to have been affected by arsenic's cumulative properties.

When still in use in the 1970s, arsenic was available to the public. Under the Pesticides (Vertebrate Pest Control) Regulations 1977, the operator had to either hold a licensed operator's certificate or be working under the supervision of a certificate holder. All of these certificates were withdrawn by the Pesticides Board on the advice of the then Agricultural Pest Destruction Council when arsenic was withdrawn from use in New Zealand.

### 3.2.3 Fate in the environment

Arsenic from baits is converted into various inorganic and organic arsenic compounds, most or all of which will be toxic to a varying extent. Arsenic baits were not considered safe to livestock until they had decomposed or had been completely disintegrated by rain.

### 3.2.4 Toxicology and pathology

#### *Onset of signs*

Death from a single dose appears to be a painful process occurring over several hours or days.

#### *Mode of action*

Arsenic causes severe gastro-enteritis, vomiting, copious watery or bloody diarrhoea, with convulsions and coma preceding death. The corrosion of the gastrointestinal tract is thought to lead to shock as well as haemorrhage.

#### *Fate in animals*

Substantial elimination of a sub-lethal dose will occur in 1–6 weeks in most animals. Arsenic is well distributed throughout all tissues and remains for long periods in bone, skin, and hair.

***Species variation in response to arsenic***

One of the main disadvantages of arsenic, in addition to its extremely inhumane mode of action, is its low toxicity to rats and possums as compared to its toxicity to humans (Table 24).

TABLE 24. ACUTE ORAL TOXICITY ($LD_{50}$mg/kg) OF ARSENIC (Hone & Mulligan 1982).

| SPECIES | $LD_{50}$(mg/kg) |
|---|---|
| Human | 1.43 |
| Possum | 8.22 |
| Mouse | 45.00 |
| Rat | 138.00 |

### 3.2.5 Current use

Arsenic is no longer available for use in New Zealand.

## 3.3 STRYCHNINE

Chemical Name: Strychnine alkaloid.

Synonyms: Strychnine.

### 3.3.1 Physical and chemical properties

The empirical formula for strychnine is $C_{21}H_{22}N_2O_2$ and its molecular weight is 334.4. Strychnine is an odourless bitter white powder. Its melting point is 270–290°C. Strychnine has a solubility in water at room temperature of 143 mg/L. Its salts are more soluble in water; for example, the sulphate is soluble in water at 30 g/L at 15°C. Strychnine is soluble in chloroform, slightly soluble in benzene, and less soluble in diethyl ether and petroleum ether.

### 3.3.2 Historical development and use

Strychnine is found in the seeds of the Indian tree *Strychnos nux-vomica* where it is one of a number of different alkaloids. It has a long history as a rodenticide (Schwartze 1922), being used first in Germany in the 16th Century. It affects the central nervous system, leading to paralysis a few minutes after intake and to death within half an hour in rodents (Prakash 1988).

The first recorded use of strychnine in New Zealand and Australia was in the 1880s where it was used in conjunction with a variety of baits including oats, wheat, root crops, thistle roots, apples, and pollard pellets. It was also used in grain to control problem birds.

In the early 1980s strychnine was withdrawn from use in New Zealand on the grounds that the type of death that it caused was inhumane.

When strychnine was used in the early 1970s, an operator was required, under the Pesticides (Vertebrate Pest Control) Regulations 1977, to hold a licensed operators certificate or to be working under the supervision of a certificate holder. All licences were then cancelled by the Pesticides Board on the advice of the then Agricultural Pest Destruction Council on the grounds that the toxin was inhumane and dangerous to staff.

### 3.3.3 Fate in the environment

Strychnine is a stable alkaloid that retains its toxicity indefinitely in the bait and in the carcass of the poisoned animals. Strychnine bait must be completely decomposed or washed out before a poisoned area could be determined as toxin-free.

### 3.3.4 Toxicology and pathology

#### *Onset of signs*

Poisoned animals often die in less than an hour as a result of respiratory failure (asphyxia), but death may take 24 hours or longer if the dose is low. The typical signs of strychnine poisoning are restlessness and muscular twitching that progresses to convulsive seizures continuing for 45 minutes or more before death. Violent muscular spasms extend the limbs and curve the neck upwards and backwards; the jaws fix and the eyes protrude (Osweiler et al. 1985).

Poisoned animals are generally found close to the bait because of the poison's rapid action. Strychnine and its salts (especially strychnine sulphate) are highly toxic to all mammals, less so to birds. The $LD_{50}$ to the Norway rat is 5–6 mg/kg (Prakash 1988). The oral $LD_{90}$ for strychnine in mice is approximately 5 mg/kg (Mutze 1989). Strychnine induces poison shyness in rats and similar shyness is thought to occur in other vertebrate pests (Prakash 1988). The bitter taste is usually masked by a sweetening agent (icing sugar) in baits.

#### *Mode of action*

Strychnine is a fast-acting poison that is readily absorbed into the circulatory system from the intestinal tract. Highest concentrations of strychnine are found in blood, liver, and kidney. Even though it is a neurotoxin, strychnine does not appear to concentrate preferentially in nervous tissues (Hayes 1994).

#### *Fate in animals*

Strychnine is highly persistent in baits and poisoned carcasses.

#### *Species variation in response to strychnine*

Strychnine is highly toxic to most domestic animals and wildlife (Table 25). Studies in the USA have shown a 50% reduction in horned lark populations after using strychnine (Apa et al. 1991). Some individual non-target bird species have been reported killed in mouse control operations in Australia (Anthony et al. 1984; Mutze 1989).

TABLE 25. ACUTE ORAL TOXICITY ($LD_{50}$mg/kg) OF STRYCHNINE (Hone & Mulligan 1982; Osweiler et al. 1985).

| SPECIES | $LD_{50}$(mg/kg) |
|---|---|
| Cow | 0.5 |
| Horse | 0.5 |
| Cat | 0.75 |
| Norway rat | 6.8 |
| Duck | 2.9 |
| Chicken | 5.0 |
| Pigeon | 2.1 |
| House sparrow | 4.0 |

### 3.3.5 Current use

This toxin had been in use since the late 1800s for pest control but has now been banned from use in New Zealand.

It is still used in Australia for mouse plagues and in the USA to control several pests, including skunks, targeted for control in rabies-infected areas, using strychnine-injected eggs. It is also used in Fiji and other islands in the Pacific.

# 4. Comparative risk assessment for commonly used vertebrate pesticides

Here we review some of the information provided in the previous sections on individual poisons used for possum control, for comparative risk-assessment purposes.

## 4.1 WHAT AND WHERE ARE THE EXPOSURE AND NON-TARGET RISKS?

Risks = Hazard × Exposure

Risks to human health and non-target wildlife or livestock are dependent on the inherent toxicity of the pesticide (i.e. hazard) and the potential for exposure to either residues or toxic baits (Eason et al. 1997). Clearly all poisons used for vertebrate pest control are hazardous. Risks can be minimised by preventing exposure of non-target species to these compounds. Some risks will be common for all types of toxic bait, e.g. the risk of a child eating toxic material if it is not stored in a secure location. Other risks, such as the risk of secondary poisoning, will vary dependent on the properties of the pesticide and how it is used Refer to the DOC 'Information on the Animal Pest QCM Module, The Safe Handling of Pesticides', published April 2000.

### 4.1.1 Persistence in water, soil, and plants

#### *1080*

Extensive research has demonstrated that 1080 may be degraded in most moist soils over 2 or more weeks by naturally occurring micro-organisms (e.g. *Pseudomonas, Fusarium*), although in climatic extremes (e.g. drought and extreme cold) the breakdown may take several months (Walker & Bong 1981; Wong et al. 1992; King et al. 1994; Parfitt et al. 1994; Walker 1994).

After 1080 is leached from baits into soils, theoretically there may then be an uptake of the toxin into terrestrial plants. In a laboratory study a single bait containing 0.15% 1080 was placed on the soil in 130-mm pots containing perennial ryegrass and broadleaf. Mean 1080 concentrations peaked at 0.08 ppm in ryegrass after 3 days then declined below detection limits after 7 days; and broadleaf concentrations peaked at 0.06 ppm after 10 days and persisted at measurable concentrations (>3 ppb) for a further 28 days (Eason et al. 1998a). Although uptake of 1080 by plants in the field is likely to be much lower, there are currently no data available on 1080 in plants after baits have been aerially broadcast. Extremely low 1080 concentrations in plants for only short periods of time are thought to present an extremely low risk to animal health.

Although it may be leached through some soils, to date no detectable amounts of 1080 have been measured in groundwater following control operations (Parfitt et al. 1994; C. Eason unpubl. data).

Legislation requires that baits not be aerially broadcast within 100 m of streams, yet there have been incidents where measurable amounts of 1080 have been detected in stream water. The amounts recorded to date have usually been less than 9 ppb and were only detected over short periods after aerial application of 1080. Although 1080 is not degraded in distilled water, the concentration of 1080 will decline in stream water (Booth et al. 1999b), and these rates of degradation are about 60% higher when temperatures are increased from 11°C to 21°C (Ogilvie et al. 1996). Immediately following aerial control the Ministry of Health normally requires managers to collect and then send samples of stream water for laboratory analysis of 1080 concentration. Proper use of bait-stations limits contamination of waterways.

### *Cyanide*

Cyanide paste is rapidly degraded by moisture and is therefore likely to remain in the environment for a short period only. Feratox® (pellets) may, however, persist for 2–3 months even when exposed to the weather (Warburton et al. 1996). Cyanide used in bait stations or as a paste is unlikely to contaminate waterways.

### *Cholecalciferol*

Cholecalciferol is used in bait stations and is therefore also unlikely to be found in waterways. It is degraded by sunlight and is also slowly oxidised when exposed to air. There is no published information on the fate of cholecalciferol in soils. However, its physico-chemical characteristics imply minimal leaching is probable.

### *Brodifacoum*

Environmental contamination by brodifacoum can be minimised by using it in well-constructed bait stations. Brodifacoum used in bait stations is unlikely to be detected in waterways, and is persistent in soils.

### *Pindone*

There is little published on the fate of pindone in soils. It is only slowly leached from baits, and no pindone was recovered from the soil under baits that were subjected to 200 mm of rain (Booth et al. 1999a). Rates of microbial degradation may be slow because of the insecticidal and fungicidal properties of pindone (Oliver & Wheeler 1978). Under standard conditions of use there was no pindone found in water samples following an aerial operation to control rabbits (Nelson & Hickling 1994).

### *Phosphorus*

This compound is readily oxidised. Baits that quickly dehydrate in hot, dry weather can ignite by spontaneous combustion and cause fires.

### 4.1.2 Persistence of residues in animal tissues

#### *1080*

Residues of 1080 may persist in sub-lethally poisoned vertebrates for up to about 4 days (Eason et al. 1994c). However, residues of 1080 can persist in the carcasses of dead animals for at least 80 days (Meenken & Booth 1997), and these may be lethal to cats, dogs, rats, stoats, and ferrets (Hegdal et al. 1986; McIlroy & Gifford 1992), mustelids (Alterio 1996, 2000; Heyward & Norbury 1999; Murphy et al. 1999), and possibly some omnivores (e.g. hedgehogs). Furthermore, some insectivorous birds that feed on insects and larvae on carcasses (Hegdal et al. 1986) may be exposed to residues.

#### *Cyanide*

Cyanide is comparatively rapidly metabolised and excreted over several days. The risks to non-target species associated with cyanide control of possums are therefore mainly through primary poisoning. However, animals recently poisoned with cyanide should be regarded as hazardous. (Mouth-to-mouth resuscitation of humans who have ingested cyanide is also dangerous.)

#### *Cholecalciferol*

Cholecalciferol is metabolised in possums to 25-hydroxycholecalciferol which is toxic and will be present in the carcasses of poisoned possums. The risk to dogs and cats by secondary poisoning is low when compared with 1080. Dogs that ate one or two carcasses of possums poisoned with cholecalciferol (Campaign®) exhibited no ill effects, while dogs eating five carcasses over a period of 5 days showed moderate clinical signs of poisoning (Eason & Wickstrom 2000). Cats fed the carcasses of possums that had been killed by cholecalciferol had slightly higher serum calcium levels after eating possum meat for 5 days, but these returned to normal within a few weeks. Human consumption of game meat in areas where cholecalciferol baits have been used is not recommended since carcasses could contain significant amounts of 25-hydroxycholecalciferol for several weeks.

#### *Brodifacoum*

Field use of brodifacoum will kill possums, rats, and mice (Gillies & Pierce 1999; Thomas 1998). Both rodent and possum carcasses will be predated by birds and mammals that eat carrion (Eason et al. 1999c) after eating brodifacoum baits as some die in the open (Cox & Smith 1992; Meenken et al. 1999). This has resulted in secondary poisoning of owls in New Zealand and overseas (Mendenhall & Pank 1980; Hegdal & Blaskiewicz 1984; Hegdal & Colvin 1988; Newton et al. 1990; Ogilvie et al. 1997; Stephenson et al. 1999), and raptors (Radvanyi et al. 1988), domestic cats and dogs (Dodds & Frantz 1984; Marsh 1985; Du Vall et al. 1989; Hoogenboom 1994; Young & De Lai 1997; Stone et al. 1999), and mustelids (Alterio 1996; Shore et al. 1999). In addition to the 33 species of indigenous birds at risk from primary poisoning, and eight species of indigenous birds at risk from secondary poisoning with brodifacoum (Eason & Spurr 1995), residues have recently been identified in dead whiteheads, parakeets, and kokako (Eason & Murphy 2000). Brodifacoum residues have also been identified in wild venison and pork from meat samples taken at processing factories. As brodifacoum will persist in the meat and livers of sub-lethally

poisoned sheep and possums for at least 9 months (Laas et al. 1985; Eason et al. 1996a), there is a theoretical potential for humans to be exposed to brodifacoum residues (Eason et al. 1996a). Brodifacoum use should therefore be limited to bait-station control of rodents and low-density possum populations so that non-target predators or scavengers are placed at less risk of eating animals containing residues. The Department of Conservation has in January 2000 taken steps to reduce the mainland field use of brodifacoum.

***Pindone***

Pindone, like other first-generation anticoagulants, is less persistent in animal tissues than second-generation anticoagulants such as brodifacoum (Parmar et al. 1987; Huckle et al. 1988). For example, in sheep, residues were detected in the liver and fat of animals dosed with 10 mg/kg for 8 days, but at 2 weeks none was detected (Nelson & Hickling 1994).

***Phosphorus***

Phosphorus residues may persist for some time in the stomach or tissues of carcasses that have been poisoned with phosphorus, and this causes secondary poisoning of birds (Sparling & Federoff 1997) and dogs (Gumbrell & Bentley 1995).

### 4.1.3 Susceptibility and risk reduction for pets and livestock

***1080***

Dogs are highly susceptible to 1080 (Eisler 1995). Residues in possum carcasses may be lethal to dogs for more than 80 days following 1080 poisoning (Meenken & Booth 1997). Dogs should therefore be excluded from control areas where 1080 has been applied for possum control for at least 3 months from the date of application or longer if dry. In areas frequently used by the public it may be necessary for managers to either caution dog owners about the need to fit muzzles to their dogs, or to use an alternative poison that does not present the same residue problems (e.g. cholecalciferol or cyanide). There have also been instances of livestock (e.g. sheep, deer, cattle) being poisoned by baits or by scavenging (e.g. cats, mustelids, and pigs) carcasses of animals that have been killed by 1080 (Annison et al. 1960; Gillies & Pierce 1999; Murphy et al. 1999; Alterio 2000). Most reported livestock deaths are as a result of baits being unintentionally applied in the wrong place, or of inadequate withholding periods before stock are reintroduced into control areas.

Pregnant ewes are more susceptible than non-pregnant sheep; nevertheless, a single sub-lethal dose of 1080 had no long-term effects on the health or productivity of sheep (Wickstrom et al. 1997b; O'Connor et al. 1999).

***Cyanide***

Cyanide is not a persistent toxin, but there have been several reports of sheep, cattle, and dogs (Hughes 1994) ingesting lethal amounts of recently laid baits. To minimise the exposure of non-target species to cyanide, paste baits should be placed sensibly and destroyed after they have been in the field for 2 nights. Uneaten Feratox® capsules should be retrieved.

### *Cholecalciferol*

There is a danger that baits containing cholecalciferol may be used less carefully than other bait types because cholecalciferol is known to be vitamin $D_3$, and perceived to be 'safe'. It is most important, therefore, to re-emphasise that cholecalciferol at the concentrations used in possum or rodent baits is potentially highly toxic to most animals. Cholecalciferol bait may be toxic to pets and domestic stock if they feed on sufficient amounts, and for this reason delivery in bait stations is recommended. The risks of secondary poisoning are low compared to 1080. Nevertheless, pets should be discouraged from eating carcasses as repeated exposure will induce toxicosis.

### *Brodifacoum*

Where bait stations are located along fence lines or in trees within the reach of animals, some livestock (especially cattle) are inclined to rub against bait stations and dislodge bait, which they then eat. Particular care is needed to exclude brodifacoum baits from livestock access. Other domestic animals feeding on carcasses containing brodifacoum residues may ingest lethal amounts of brodifacoum through secondary poisoning (e.g. dogs and cats). Antidotes are available (Vitamin K) for brodifacoum and pindone, but treatment of brodifacoum poisoning is prolonged.

### *Pindone*

Pindone is highly toxic to rabbits ($LD_{50}$ = 6–18 mg/kg), but less toxic to dogs ($LD_{50}$ = 75–100 mg/kg) and sheep ($LD_{50}$ ≈ 100 mg/kg) (Eason 1996). Evaluation of prothrombin times demonstrated that cats were the most susceptible domestic animal to pindone, that cattle may be affected by moderate doses, and horses are the least susceptible (Martin et al. 1991). Sheep administered sub-lethal amounts of pindone eliminated all of the toxicant within 2 weeks of dosing (Nelson & Hickling 1994).

### *Phosphorus*

Phosphorus is lethal to all domestic livestock that feed on paste baits. Cats, dogs, and pigs are also at risk from secondary poisoning. Although only 2–4 tonnes of phosphorus paste were used annually for possum control in New Zealand, between 1960 and 1976 there were 117 confirmed dog deaths by phosphorus compared to 254 deaths with 1080 (Rammell & Fleming 1978).

## 4.1.4 Risk of exposure and toxicity to non-target vertebrates (wildlife)

Non-target animals are principally at risk from eating baits or poisoned carcasses.

### *1080*

The risk to non-target species during aerial control has been extensively studied (Spurr 1993a; Spurr 1994a; Fraser et al. 1995; Spurr & Powlesland 1997; Powlesland et al. 1999; Sherley et al. 1999; Fraser & Sweetapple 2000). Ongoing research is further evaluating species that may be at risk, and options that may further improve the safety of all possum control could include the use of more-potent bird repellents. Although most dead birds found following possum

control have been exotic species (e.g. blackbird, chaffinch), native birds (e.g. whitehead, robin, tomtit, morepork) have also been killed (Spurr 1994a). The numbers of birds killed following aerial application of bait has declined since operators started routinely screening carrot bait to remove highly toxic fragments (Spurr 1994a); adding green dye (Caithness & Williams 1970) and cinnamon (Udy & Pracy 1981) as bird deterrents; and reducing sowing rates (Morgan et al. 1997).

To date aerial control has had no long-term effects on populations of bats (Lloyd & McQueen 1998). The impact of aerial operations on lizard and skink populations has not been well assessed, but it would seem there is some mortality of these vertebrates when exposed to 1080 baits or to insects that have fed on baits (Whitaker & Loh 1991). The impact of aerial control on native frogs has not been assessed, but research conducted in Australia suggests that frogs are not very susceptible to 1080 (McIlroy 1986).

The extent of non-target interference with baits in bait stations has not been well researched. Kaka may eat both plain cereal baits (Spurr 1993b) and baits dyed green and containing 0.1% cinnamon (Hickling 1997) when first exposed to them. However, cinnamon-lured baits are eaten less frequently and in significantly smaller amounts by kaka after they have been exposed to them two or more times (Hickling 1997). Although kiwi may eat cereal baits (Pierce & Montgomery 1992) but not carrot (MacLennan et al. 1992), there have been no kiwi deaths reported following 1080 operations (Spurr 1994a; Robertson et al. 1999b).

Although rats and mice use bait stations (Thomas 1998) it is not known how many other species are at risk by primary poisoning. Possums, mice, rabbits and rats poisoned with 1080 are in themselves a hazard to other animals that eat them. Secondary poisoning of predators is commonly reported (Murphy et al. 1999; Heyward & Norbury 1999) and mortality in deer, usually 30–40% after aerial operations (Fraser et al. 1995) has been higher, i.e. >90% (Fraser & Sweetapple 2000).

### *Cyanide*

There are fewer reports of birds being killed by cyanide than by 1080 or traps (Spurr 1991). However, some ground-dwelling species are at risk, and unfortunately in some regions there have been reports of weka and kiwi being killed where cyanide paste was used. For example, in 1984 some 66 hunters reported 37 kiwi poisoned by cyanide paste, about a quarter of the number caught in traps (Spurr 1991). In comparison no kiwi have been reported poisoned after 1080 operations. The risks that discarded cyanide capsules (i.e. Feratox®) present to non-target species has not been formally assessed. There are anecdotal reports of Feratox® killing hedgehogs, stoats, and cats, but not birds (J. Kerr, pers. comm.).

### *Cholecalciferol*

Cholecalciferol is less toxic to birds than 1080 (Wickstrom et al. 1997a; Eason & Wickstrom 2000), and is therefore likely to kill very few birds by either primary or secondary poisoning. Captive mallard ducks survived very high doses (2000 mg/kg) of cholecalciferol (Marshall 1984; Eason & Wickstrom 2000), but

some canaries and chickens receiving the same dose subsequently died. Risk of exposure of non-target mammals or birds in the field has not yet been quantified.

### *Brodifacoum*

Brodifacoum is a potent second-generation anticoagulant that is toxic to many non-target species by both primary and secondary poisoning (Godfrey 1985; Eason & Spurr 1995). For example, most blackbirds on Red Mercury Island not killed by aerial application of brodifacoum baits were subsequently found to contain brodifacoum residues (Morgan & Wright 1996), and there are now extensive reports of other species of birds and mammals, including feral pigs, showing residues (Ogilvie et al. 1997; Empson & Miskelly 1999; Dowding et al. 1999; Stephenson & Minot 1999; Robertson et al. 1999a; Eason et al. 2000). A wide range of bird species (e.g. saddleback, silvereyes, paradise shelduck, morepork, skua, robin, and weka) have been found dead from poisoning after field use of brodifacoum in New Zealand (e.g. Taylor 1984; Taylor & Thomas 1993; Towns et al. 1993; Williams et al. 1986a, b; Stephenson et al. 1999). The manufacturer of brodifacoum concentrate (Zeneca) recommends that the pesticide should not be aerially broadcast for the control of pests, and that systems for controlled delivery of baits should be used.

Further research in brodifacoum control areas is required to assess the risks to insectivorous birds feeding on invertebrates; the risks to predatory (e.g. New Zealand falcon, morepork) and omnivorous birds; the risk to folivores and seed-eating birds that may eat baits (e.g. Ogilvie et al. 1997); and the impacts of an exposure to brodifacoum on the breeding success of selected bird species. Detrimental effects on some individuals may, however, be counter-balanced by improved survival and breeding success in the absence of possums and rodents. In the short term a research priority is to establish whether or not the extent of brodifacoum transfer through the food web can be contained, for example, by using brodifacoum for rodents alone, or only using brodifacoum in pulses after 1080, cyanide, or cholecalciferol for initial control.

### *Pindone*

Research has demonstrated that pindone presents a risk of primary and secondary poisoning of birds (Martin et al. 1994). Scavengers such as the harrier hawk are likely to be most at risk from secondary poisoning (Calvin & Jackson 1991), and raptors tended to be more susceptible (0.25 mg/kg) than magpies, pigeons, parrots, and ducks (4–5 mg/kg) in dose-ranging experiments (Martin et al. 1994). However, the risks of inducing secondary poisoning are likely to be less pronounced than with brodifacoum, dependent of course on how the toxic baits are applied. In New Zealand there have been anecdotal reports (E.B. Spurr pers. comm.) of extensive bird kills from both primary and secondary poisoning after the use of pindone for rabbit control, but there has been no monitoring to determine whether or not pindone has any long-term effects on the local abundance of bird populations.

### *Phosphorus*

Phosphorus is known to kill birds that feed on carrion. Although the non-target effects on indigenous birds in New Zealand have not been assessed, it is

expected that morepork, New Zealand falcon, black-backed gull, skua, weka, and harriers will be at some risk from secondary poisoning.

### 4.1.5 Risk of exposure and toxicity to invertebrates

Invertebrates are at risk from contact with baits, eating baits or poisoned carcasses, or from exposure to residues in soil or other environmental media.

#### *1080*

There have been no significant changes in the relative abundance of different invertebrate taxa monitored before and after aerial poisoning that can be attributed to insect populations having been exposed to 1080 poison (Spurr 1994b; Spurr & Drew 1999). However, residues of 1080 have been measured in some insects (e.g. weta and cockroach) for up to 3 weeks following aerial application of 1080 baits (Eason et al. 1998a). Invertebrates with 1080 residues may present a risk to insectivorous birds (Hegdal et al. 1986). It has been shown that the addition of cinnamon to baits is a deterrent to some invertebrates (Spurr & Drew 1999). Recent research has confirmed that 1080 is not persistent in invertebrate species (e.g. Booth & Wickstrom 1999). Sherley et al. (1999) have raised concerns over the number of invertebrate species food in contact with 1080 baits, and research is now focusing on the incorporation of an invertebrate repellent into baits to enhance target specificity, and decrease the risk of secondary poisoning via species such as weta, which are known to eat 1080 baits.

#### *Cyanide*

Although the effect of cyanide on invertebrates in New Zealand has not been researched, it is lethal to aquatic invertebrates at relatively low concentrations (i.e. <200 ppb, Hone & Mulligan 1982). Although this indicates that the hazards of cyanide to invertebrates may be high, the exposure of invertebrates to cyanide will be low, particularly if Feratox® pellets are used.

#### *Cholecalciferol*

Few studies have assessed the risk to invertebrates from cholecalciferol baits. One trial on 18 captive weta indicated that weta would eat cholecalciferol baits, but that the baits were not toxic to them (Ogilvie & Eason 1996). When cholecalciferol stock solution was orally administered to weta they survived the highest dose volume (250 µg/g), indicating cholecalciferol lacks insecticidal properties, at least in this species (Eason & Wickstrom 2000).

#### *Brodifacoum*

Field studies have demonstrated that only some invertebrates (e.g. slugs, weta) contained brodifacoum residues after Talon® 20P bait was aerially broadcast for rodent control (Morgan & Wright 1996; Ogilvie et al. 1997), and that there is no measurable change in abundance of invertebrates during such control (Spurr & Powlesland 1997). Brodifacoum had no significant effect on weta when they were orally dosed (Morgan et al. 1996b). No data are available for other invertebrate species. The survival of weta exposed to brodifacoum may in part be due to the fact that they can metabolise and excrete it because they do not have the same blood-clotting systems as vertebrates (Shirer 1992). The role of

invertebrates as a vector in transferring brodifacoum from baits or carcasses to insectivorous birds is, however, unknown.

***Pindone***

Pindone was initially evaluated as an insecticide (Kilgare et al. 1942), and was demonstrated to be effective at controlling body lice (Eddy & Bushland 1948). It may, therefore, be lethal to invertebrates feeding on baits or pindone residues in carcasses. The effects on invertebrate populations in New Zealand have not been assessed. Prior to assessing populations, the insecticidal properties reported in the 1940s should be verified if there are concerns that pindone may be having an effect on invertebrates.

***Phosphorus***

The effects on invertebrates are not known. However, there are anecdotal reports of bees being found dead on paste baits.

When new bait formulations are produced, part of the research and development process should include an assessment of their attractiveness to invertebrates. Contamination of honey bees presents a human health risk, and all baits must be proven to be unattractive to bees.

### 4.1.6 Risk of exposure to humans

Human exposure might arise from drinking contaminated water, ingestion of toxic baits, consumption of food contaminated by contact with bait, or by inhalation of bait dust or contact with the active ingredient or stock solutions by pest control operators and bait manufacturers. Potentially the most significant source of general public exposure was considered to be contamination of surface water in public-water-supply catchments by aerially sown 1080 baits.

***1080***

Ministry of Health Model Permit Conditions place strict controls on the broadcasting of 1080 baits, which is not permitted within 20 m of any camping ground, formed public road currently in use, picnic areas, lake, pond, water supply intake or stream/river specified by the Medical Officer of Health. Bait applied within 60 m of these areas must be applied by helicopter, either with a competent observer in the aircraft, or by an aircraft fitted with and using a recording GPS. The bait must be laid by hand if it is to be applied closer than 20 m to any such areas.

After 1153 samples of stream water collected during 1990–2000 shortly after the aerial application of 1080 were analysed, residues of 1080 above 2 ppm were rarely found (Eason et al. 1999b; G.R.G. Wright pers. comm.). Monitoring of water has shown the detection of 1080 in stream water to be of a short duration. (Parfitt et al. 1994; Ogilvie et al. 1996). (See section 4.1.2 for further details.)

The toxin 1080 is metabolised and largely excreted within 4 days of a single exposure (Rammell 1993; Eason et al. 1994b). This limits the risk of meat being consumed that contains 1080 residues. Withholding milk for a minimum of 1 week would be appropriate should inadvertent poisoning of cows be suspected.

Withholding periods of 5–10 days are recommended when incidents of livestock poisoning have occurred. It would be unwise to take game from areas that have been poisoned, until all poisoned carcasses have degraded. Pesticide regulations require that hunting permits must warn hunters of areas where 1080 poisoning is planned, and toxic baits not in use must be secured in safe storage. Game meat can now only be sold to packhouses by approved suppliers, and this meat must contain no 1080 residues. The Ministry of Agriculture and Forestry regulations regarding the procurement of game meat have recently been reviewed.

To prevent both bee mortality and 1080 contamination of honey, the original paste that contained sugar was withdrawn from the market (Morgan & Goodwin 1995) and legislation precludes control with pastes within 4 km of beehives (Ministry of Health 1995). Formulations that are unattractive to bees have since been introduced for possum control (Morgan & Goodwin 1995).

To date only one person has been reportedly killed (a suicide) in New Zealand from 1080 poisoning (Hughes 1994).

### *Cyanide*

Cyanide is degraded in the environment, is unlikely to contaminate waterways, and does not persist in animals. Cyanide pastes and pellets are potentially extremely hazardous to pest control operators and the public. However, if used properly and stored securely, pastes or Feratox® pellets are unlikely to pose a significant health risk.

### *Cholecalciferol*

Cholecalciferol is unlikely to contaminate surface water because it is used in bait placed in bait stations, and is unlikely to be mobile in soil. The risks that residues will occur in meat are low compared with brodifacoum because cholecalciferol is rapidly absorbed and converted to toxic metabolites (e.g. 25-hydroxycholecalciferol; 1,25 dihydroxycholecalciferol), which are in themselves subsequently metabolised and excreted over a period of days or weeks, depending on the dose ingested (Bahri 1990; Eason & Wickstrom 2000). These hydroxylated metabolites are retained mainly in the plasma, liver, heart, and adipose tissue of poisoned animals (Eason et al. 1996c; Wickstrom et al. 1997a; Eason & Wickstrom 2000). These toxic metabolites are present in poisoned possums at high enough concentrations to cause toxicosis (e.g. hypercalcaemia, kidney damage, and loss of appetite) in dogs that were repeatedly fed poisoned possum carcasses (Eason & Wickstrom 2000). Whilst these dogs recovered, a precautionary approach would be to limit the procurement of game meat from areas where cholecalciferol had been used. The greatest hazard to humans would be from game species that had recently gained direct access to bait.

### *Brodifacoum*

Brodifacoum is unlikely to contaminate surface water since it is principally used in baits enclosed in bait stations, and it does not migrate through soil because it is not readily soluble in water (Jackson et al. 1991).

Exposure to brodifacoum residues by eating the meat (especially livers or kidneys) of animals that have ingested the poison is theoretically possible. Animals may become contaminated with brodifacoum residues by eating baits, or by eating the carcasses of other animals poisoned with brodifacoum (e.g. wild pigs that have fed on possum carcasses and baits: Eason et al. 1999c, 2000). Brodifacoum therefore represents a potential health risk to hunters who repeatedly eat the livers of wild pigs and/or wild pork from pigs shot in areas where brodifacoum has been extensively used for pest control (Eason et al. 1999c, 2000).

***Pindone***

Pindone is unlikely to occur in surface water. Livestock (e.g. sheep) contaminated with pindone are likely to metabolise and excrete it within approximately 2 weeks (Nelson & Hickling 1994), and it is therefore unlikely to be present in meat for human consumption unless there has been fairly recent exposure to baits. An adequate withholding period of at least 1 month would be sensible if exposure to pindone was suspected.

***Phosphorus***

Phosphorus is unlikely to occur in surface water. Dogs may be at risk of secondary poisoning if they consume carcasses of possums poisoned with phosphorus (Gumbrell & Bentley 1995). This suggests that wild pigs eating baits or the carcasses of possums poisoned with phosphorus may also contain phosphorus residues. The duration of phosphorus residues in game or other wildlife after sub-lethal exposure is unknown.

***Risks to pest controllers***

Personnel regularly handling toxic baits or who are exposed to toxic concentrates must comply with label instructions and information supplied in material safety data sheets (MSDS). Those persons most at risk from exposure are those frequently handling materials that are very hazardous. Poisons may be ingested accidentally, resulting in a single sub-lethal exposure, or by careless use, which may result in repeated sub-lethal exposures. The known effects of substantial but sub-lethal exposures on animals and humans are summarised below (Section 4.1.6). The effects or lack of sub-lethal effects from single or multiple exposures will be dose-dependent. Not surprisingly animals or humans surviving substantial doses of a poison may be permanently affected through brain damage or effects on some other target organs. It has, in the past, been mistakenly assumed (in the New Zealand pest control industry) that rapidly eliminated poisons only have short-term effects. This is a misunderstanding of the basic principles of toxicology, confusing rates of metabolism or excretion with the lack or presence of toxic effects.

Monitoring of blood and urine of workers in the pest control industry is important to ensure safety procedures minimise exposure.

### 4.1.7 Toxic effects and humaneness in non-target species

Figure 1 illustrates the range of levels at which ecotoxicological and toxicological evaluations, including effects studies on non-target species, can be undertaken. Toxicological risk assessment integrates information from different levels of organisation.

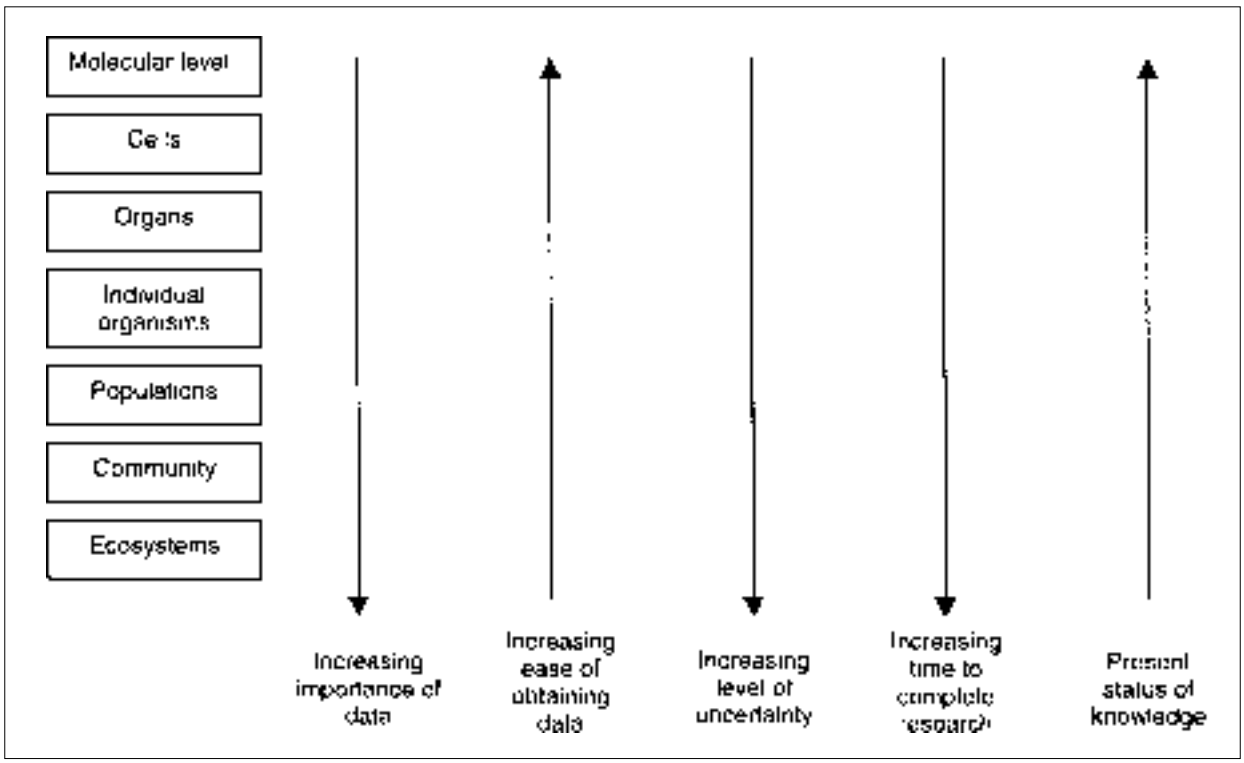


Figure 1. Relationships of aspects of the science of ecotoxicology and toxicology and the different levels of biological organisation.

### *1080*

1080 is a broad-spectrum poison capable of readily inducing death in most species ingesting sufficient bait. A single sub-lethal exposure may cause a variety of effects, including 1080-induced myocardial lesions (Buffa et al. 1977), as observed in the cardiac muscle of sheep (Annison et al. 1960; Schultz et al. 1982; Wickstrom et al. 1997b; O'Connor et al. 1999). Large single doses (100 and 200 mg/kg) caused a reduction in plasma testosterone concentrations and degeneration of seminiferous tubules in skinks (Twigg et al. 1988). Single doses of 15 and 60 mg/kg resulted in damage to the kidneys of rats (Savarie 1984). Humans sub-lethally poisoned have on occasions suffered chronic cardiac dysfunctions or renal problems (Chung 1984; Chi et al. 1996), and neurophysiological effects (Trabes et al. 1983). A very large sub-lethal exposure caused permanent brain damage to a child (McTaggart 1970). Neuro-physiological effects are likely to be linked to glial cell dysfunction. Glial cells in the brain are implicated in brain extracellular fluid ion and acid base homeostasis. Glial cells are very sensitive to fluorocitrate which is described as a glial toxin (Erlichman et al. 1998).

Repeated sub-lethal exposure to 1080 has resulted in lower levels of spermatogenesis in rats (Sullivan et al. 1979; Miller & Philips 1955) and mink (Hornshaw et al. 1986), and foetal abnormalities in rats (Eason et al. 1999b). In a 90-day rodent exposure study no effect levels have been established for effects on spermatogenesis. The testes are the most sensitive target organ in rats. Partial recovery to normal sperm production has been shown following the cessation of treatment. Sheep receiving repeated sub-lethal doses of 1080 had myocardial degeneration as well as necrosis of individual or small groups of myocardial fibres (Schultz et al. 1982).

### *Cyanide*

Cyanide is an extremely hazardous substance due to the release of HCN gas when it is exposed to air. Ingestion of cyanide pellets or paste or inhalation of the gas produces adverse reactions within seconds. Signs of sub-lethal poisoning in humans include hyperventilation, headache, nausea, vomiting, and generalised weakness. Both manufacturers and pest controllers must therefore avoid inhaling cyanide fumes by ensuring they operate in a well-ventilated area and/or wear breathing apparatus (especially during bait manufacture).

The scientific literature suggests that repeated exposure to substantial sub-lethal amounts of cyanide could potentially cause lasting neurological effects (Kanthasamy et al. 1994).

### *Cholecalciferol*

No known accidental poisoning with cholecalciferol rodenticides have been lethal (Pelfrene 1991). However, it should be noted that cholecalciferol has been used world-wide as a rodenticide at 0.075%. In possum baits the concentration is 0.8% and these baits therefore present a greater risk. A single sub-lethal poisoning may result in soft tissue calcification (Toda et al. 1985; Pelfrene 1991).

Long-term exposure to sub-lethal doses of cholecalciferol can also result in hypervitaminosis D whereby calcium mobilised from the skeleton may be deposited in many soft tissues (renal arteries and tubules, heart and coronary arteries, lungs, bronchi, and stomach). Clinical signs include fatigue and weight loss. High repeat doses of cholecalciferol may also cause foetal abnormalities and embryo toxicity (Pelfrene 1991).

### *Brodifacoum*

Brodifacoum is potent (as well as persistent), hence a single exposure of sufficient magnitude could alter clotting factors, resulting in haemorrhage. The effects or lack of effects range from no effect on blood clotting, to death and, as with other anticoagulants, will be dose-dependent.

Brodifacoum is highly toxic if it is orally ingested or inhaled (Pelfrene 1991), and it may be absorbed through the skin. Brodifacoum persists in the liver and muscle of sub-lethally poisoned animals and may be equally persistent in pest controllers exposed to it. If sufficient brodifacoum was ingested by humans, toxicosis can readily be detected by alteration in blood-clotting parameters. The effects of long-term exposure to low doses of brodifacoum are largely unknown. However, there are reports of an adverse effect on bone metabolism and the development of osteoporosis (Szulc et al. 1993). Repeated exposure to another anticoagulant warfarin (as a therapeutic agent) has also been linked with developmental malformations in pregnant women (Tasheva 1995). In laboratory studies there was no evidence that brodifacoum is either a teratogen or mutagen, but care should be taken to ensure that operators are not unnecessarily exposed to the poison.

### *Pindone*

Pindone is not as potent as brodifacoum, and therefore larger doses would be needed to result in alterations in blood clotting parameters, bruising, or haemorrhaging. Clinical signs may include anaemia and weakness.

### *Phosphorus*

In humans the initial signs of phosphorus poisoning are severe epigastric pain, nausea, vomiting, dizziness, headache, and a garlic odour on the breath. A large sub-lethal dose may cause liver damage (e.g. cirrhosis: Moeschlin 1965; kidney failure: Cushman & Alexander 1966; myocardial damage, hypoglycaemia: Clarkson 1991; hypocalcaemia: Cushman & Alexander 1966; and abortions: Piribauer & Wallenko 1961).

Chronic poisoning in humans leads to toothache followed by swelling of the jaw and then necrosis of the mandible (colloquially known as 'phossy jaw'). This condition may be the only clinical sign from mild exposures to phosphorus, although higher repeat doses also cause liver and kidney damage (Clarkson 1991). Signs of chronic high exposures to phosphorus are weakness, weight loss, anaemia, loss of appetite, and spontaneous fractures.

## 4.1.8 Humaneness in target species

The RSPCA New Zealand is opposed to the use of any toxicant that causes any animal to suffer and is particularly opposed to the use of arsenic and strychnine (Loague 1994). The humaneness of poisons is dependent on the duration and severity of distress or pain that animals experience during three stages of toxicosis described as: an initial lag phase until the onset of clinical signs; a period of sickness behaviour when animals are most likely to experience pain; and, a final phase preceding death when animals may be unconscious (Eason et al. 1998b). These stages have been described in possums for cyanide (Gregory et al. 1998; O'Connor et al. 1998), 1080 (Eason et al. 1998b) phosphorus (O'Connor et al. 1998), and brodifacoum (Littin et al. 1999). Ongoing research is evaluating the behavioural, physiological, and pathological effects of brodifacoum and cholecalciferol on the welfare of possums (pers. comm. C. O'Connor). The amount of distress and times to death are dose-dependent (O'Connor et al. 1998), and for some poisons the individual responses during toxicosis are extremely variable (e.g. cholecalciferol, brodifacoum); a general overview of welfare for the five commonly used possum toxicants is summarised in Table 26.

TABLE 26. SUMMARY OF MEAN TIMES TO ONSET OF CLINICAL SIGNS OF TOXICOSIS, DURATION OF KEY SYMPTOMS DURING SICKNESS BEHAVIOUR, AND TIMES TO DEATH IN POSSUMS FOLLOWING INGESTION OF POISON BAITS (Eason et al. 1998b; O'Connor & Littin unpubl. data).

| TOXIN | MEAN TIME UNTIL ONSET OF SICKNESS | SICKNESS BEHAVIOUR | MEAN DURATION | MEAN TIME UNTIL DEATH |
|---|---|---|---|---|
| Cyanide | 2 min | Ataxia, impaired co-ordination, breathlessness, muscular spasms | 12 min | 14 min |
| 1080 | 3 hours | Anorexia, ataxia, occasional retching, spasms, breathlessness, laboured breathing | 8.5 hours | 11.5 hours |
| Phosphorus paste | 5 hours | Retching, vomiting, hunched posture, intermittent repositioning, ataxia | 13 hours | 18 hours |
| Cholecalciferol | 5 days | Loss of appetite, lethargy, breathlessness | 3.5 days | 8.5 days |
| Brodifacoum | 16 days | Anaemia, haemorrhage, loss of appetite, hunched posture, anorexia | 3 days | 19 days |

Cyanide is the fastest acting vertebrate pesticide used in New Zealand and causes least distress. Cyanide is therefore regarded as the most humane of the toxicants evaluated. In comparison brodifacoum is slow-acting, and possum sometimes take a long time to die, an average of 21 days after eating baits. Phosphorus causes behavioural responses associated with inflammation of the stomach lining and duodenum (e.g. a crouched/hunched posture), but the clinical signs of phosphorus poisoning in possum given paste are less severe and less prolonged than has been previously reported for other species. The times to death after possums eat 1080 baits (or other poisons) are dose-dependent, but are on average considerably less than that recorded for phosphorus, and possums dosed with 1080 exhibit few overt signs of pain. Cholecalciferol causes anorexia, with possums on average losing 20% of their body weight before death. There have been no studies on the humaneness of pindone.

### 4.1.9 Summary of characteristics of poisons used for possum control in New Zealand

***Key advantages, disadvantages, and risk factors***

**1080**

- Moderately rapid and humane
- Essential for aerial control
- Very effective
- Low environmental persistence
- Secondary poison risks
- No antidote

**Cyanide**

- Rapid action, most humane
- Cyanide aversion influences effectiveness
- Low environmental persistence
- Low secondary poison risk
- Effective antidotes lacking

**Cholecalciferol**

- Effective
- Lower toxicity to birds than 1080
- Low risk of secondary poisoning
- Expensive compared with 1080 or cyanide

**Brodifacoum**

- Possums take 2–4 weeks to die
- Effective against low-density, poison/bait-shy possums
- Very persistent
- High secondary poisoning risk
- Widespread contamination of wildlife and game possible
- Antidote available
- Expensive compared with 1080 or cyanide

**Pindone**

- Possums take 2–3 weeks to die
- Low effectiveness
- Moderate persistence

- Low secondary poison risk
- Antidote available

**Phosphorus**

- Causes longer periods of pain and sickness than cyanide and 1080
- Effective
- Causes secondary poisoning
- Effective antidotes lacking

# 5. Acknowledgements

Staff from the Department of Conservation provided useful comments on earlier draft versions of the manual. The first edition was produced in 1997 with editorial assistance from DOC staff, in particular Nicola Haydock. Many staff members of the Department of Conservation and regional councils responded with useful comments and the provision of both unpublished and published material. Information on the treatment and diagnosis of toxic poisoning was peer reviewed by toxicologists of the National Toxicology Group. Kate Littin reviewed the draft manuscript, and various anonymous referees suggested further improvements.

This work was funded from the Unplanned Science Advice Fund, Department of Conservation investigation no. 2118.

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