REVIEW

An updated review of the toxicology and ecotoxicology of sodium fluoroacetate (1080) in relation to its use as a pest control tool in New Zealand

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Abstract: Sodium fluoroacetate (1080) is a vertebrate pesticide, originally developed in the 1940s and principally used for the control of unwanted introduced animals in New Zealand and Australia. Fluoroacetate is also a toxic component of poisonous plants found in Australia, Africa, South America, and India. In relation to its use as a pesticide, recent research has focused on further elucidation of its potential sub-lethal effects, on animal welfare issues, on understanding and reducing its risk to non-target species, on its ecotoxicology, and fate in the environment following use in baits. 1080 acts by interfering with cellular energy production through inhibition of the tricarboxylic acid cycle and lethal doses can kill animal pests within 6–48 h of eating baits. Exposure to sub-lethal doses has been shown to have harmful effects on the heart and testes in animal studies, and strict safety precautions are enforced to protect contractors and workers in the pest control industry. Considerable care must be taken when using 1080 for the control of animal pests. Primary poisoning of non-target birds and secondary poisoning of dogs must be minimised to ensure that benefits in terms of conservation outcomes and pest and disease control significantly outweigh the risks associated with its use. Despite over 60 years of research and practical experience, the use of 1080 is still embroiled in controversy, while research efforts continue to improve its target specificity when it is used as a conservation tool or for Tb vector control.

Keywords: animal welfare; compound 1080; non-target effects

Introduction

The toxic nature of fluoroacetate-containing compounds was first noted in the 1930s, leading to their patenting as potential rodenticides prior to the Second World War. US-led research programmes in the early 1940s identified sodium fluoroacetate as the most promising candidate (Atzert 1971). It received the acquisition number 1080 from British scientists at the Patuxent Wildlife Research Center in Maryland, USA, and is now commonly known as compound 1080, or just 1080. The structural formula for 1080 is FCH_2COONa (empirical formula $C_2H_2FNaO_2$) and the molecular weight is 100.3. It forms an odourless, white, non-volatile powder that decomposes at about 200ºC. Sodium fluoroacetate (1080) is very water soluble but has low solubility in organic solvents such as ethanol and oils. The early studies in the 1940s marked the beginning of over 60 years of continuous research on 1080 linked to its use as a vertebrate pesticide.

1080 was first applied in bait to control gophers, ground squirrels, prairie dogs and rodents in the USA, and has been used in New Zealand for pest control since the 1950s. It is the only effective poison that is registered for aerial control of possums (*Trichosurus vulpecula*). Its use is a reflection of unique wildlife pest problems within New Zealand and its effectiveness at reducing the unwanted impacts of introduced species. Possums, introduced from Australia in the late 1800s, and other 'target' pest species in New Zealand, have no natural predators, as New Zealand's only native terrestrial mammals are both bats (*Chalinolobus tuberculatus*, *Mystacina*

tuberculata). In Australia, 1080 is used for protection of native animals from introduced species such as foxes (*Vulpes vulpes*) (McIlroy 1990).

The most extensive use of 1080 occurs in New Zealand, followed by Australia (PCE 1994; JE Cavanagh & P Fisher, unpubl. report 2005). It is also used in Israel and the USA. Even in countries that have very active control programmes targeting introduced mammals, products containing vertebrate pesticides are considered 'minor use products' in terms of the total amounts of pesticides used. For example, in New Zealand, 3500 tonnes of active pesticide ingredient are used annually, most of which are herbicides (Manktelow et al. 2005). In comparison to insecticides, fungicides and herbicides, vertebrate pesticides make up only 0.1–0.2% of our annual use (i.e. 3.5–7 t), although this figure increases slightly to 0.4% (14 t) per annum if household rodenticides are included. Even though New Zealand is the largest user of 1080 in the world, the amount of active ingredient used per year is very small (1.0–3.5 t; Innes & Barker 1999), less than 0.1% of the total active pesticide ingredient used.

In the USA, 1080 use is restricted to a livestock-protection collar to protect sheep (*Ovis aries*) and goats (*Capra hircus*) from coyotes (*Canis latrans*) (Fagerstone et al. 1994). Field use of baits containing 1080 is less appropriate in countries where indigenous non-target mammals may eat bait or poisoned carcasses. In New Zealand there are six poisons currently registered for possum control: 1080 (sodium fluoroacetate), cyanide, cholecalciferol (Vitamin D_3), brodifacoum, pindone, and phosphorus. Their use for killing possums and other

animal pest species raises a variety of concerns including risks to non-target animals, persistence and humaneness, which are different for each. Zinc phosphide and a combination of coumatetralyl and cholecalciferol are currently being developed in New Zealand as additional tools for possum and rodent control (Eason et al. 2008). Currently, the most extensively used poison is 1080, applied in cereal or carrot baits for aerial sowing or use in bait stations. Despite improvements in the use and targeting of 1080 (Eason et al. 2006), its use is still embroiled in controversy (Hansford 2009). Most concerns in New Zealand relate to fears with regard to contamination of water supplies, possible sub-lethal effects on humans, welfare impacts on those species targeted by 1080, non-target species that might be inadvertently exposed to baits, and the potential to kill native birds and other non-target species (ERMA 2007).

Natural toxin

Manufactured 1080 for use in toxic baits has been shown to be chemically and toxicologically identical to the fluoroacetate found in poisonous plants (Eason 2002a). Marais (1944) identified fluoroacetate in the South African plant gifblaar (*Dichapetalum cymosum*). Interestingly, the caterpillar moth, *Sindrus albimaculatus*, which feeds on *D. cymosum*, cannot only detoxify fluoroacetate, but also accumulate it (probably in vacuoles), and uses fluoroacetate as a defence against predation (Meyer & O'Hagan 1992).

As pointed out in earlier work by Eason (2002a), fluoroacetate has been identified as the toxic agent in many other poisonous plants native to Brazil (de Moraes-Moreau et al. 1995) and Africa (Atzert 1971). Fluoroacetate also occurs naturally in some 40 plant species in Australia (Twigg 1994; Twigg et al. 1996a, b, 1999). The highest fluoroacetate concentration so far reported from a living source is 8.0 mg g^{-1} in the seeds of the African plant *Dichapetalum braunii* (O'Hagan et al. 1993). The mechanism of toxicity for naturally occurring fluoroacetate and for 1080 in bait is the same. Both forms are equally poisonous (de Moraes-Moreau et al. 1995). Fluoroacetate appears to be one of the many secondary plant compounds that have evolved at high concentrations as a defence mechanism against browsing animals (King et al. 1981). Most studies assessing fluoroacetate concentrations in plants have focused on those species that are overtly toxic to mammals. However, it would appear that the ability of plants to synthesise fluoroacetate may be more widespread than generally supposed, since fluoroacetate occurs at extremely low concentrations in some Finnish plants (Vartiainen & Kauranen 1980), in tea leaves (Vartiainen & Kauranen 1984), and guar gum (*Cyamopsis tetragonolobus*) (Vartiainen & Gynther 1984; Twigg et al. 1996b). In addition, some plants, when exposed to fluoride ions, can biosynthesise fluoroacetate, albeit at very low levels (Cheng et al. 1968). Fluorocitrate, the toxic metabolite of fluoroacetate, has also been detected in tea leaves (Peters et al. 1972). Fluoroacetate biosynthesis can occur in some bacteria, notably *Streptomyces cattleya* (O'Hagan & Harper 1999). Recent research has shown fluoroacetate to be present at extremely low concentrations in native New Zealand plants such as puha (*Sonchus asper*), both naturally, and by uptake when exposed to 1080 baits in the environment (Ogilvie et al. 2009). Puha is often consumed by humans, raising a health concern; however, puha is boiled prior to eating. As 1080 is highly water soluble (Parfitt et al. 1995), it is considered that such preparation methods render the plant non-hazardous

due to the negligible 1080 concentrations present (Ogilvie et al. 2009).

Mode of action

The primary mode of action of 1080 is mediated via its toxic metabolite, fluorocitrate. Following absorption, intracellular conversion of 1080 within an animal produces fluorocitrate, which inhibits energy production in the tricarboxylic acid (Krebs) cycle (Peters 1952, 1957; Peters et al. 1953; Buffa et al. 1973). Synthesis of fluorocitrate, termed 'lethal synthesis' (Peters 1952), occurs in the mitochondria. When 1080 enters the tricarboxylic acid cycle it condenses with oxaloacetate to form fluorocitrate instead of the usual citrate. Fluorocitrate competitively inhibits tricarboxylic acid cycle enzymes, notably aconitase hydratase (Morrison & Peters 1954; Buffa et al. 1973), which prevents the conversion of citrate to aconitate (Fanshier et al. 1964). It also inhibits citrate transport into and out of mitochondria (Kirsten et al. 1978; Kun 1982). There is also evidence to suggest that fluorocitrate inhibits succinate dehydrogenase, and the high levels of citrate concentration that occur during fluoroacetate intoxication can also have an inhibitory effect on the glycolytic enzyme phosphofructokinase. Enzyme systems linked to glucose transport and lipolysis are also affected (Peters 1952; Peters et al. 1972; Buffa et al. 1973; Kirsten et al. 1978; Kun 1982; Clarke 1991).

The tricarboxylic acid cycle is a metabolic pathway that breaks down food (carbohydrates) to provide energy for normal cell functions. The inhibition of this metabolic pathway results in accumulation of citrate in the tissues and blood. At high concentrations, citrate binds serum calcium, resulting in hypocalcaemia and ultimately heart failure (Roy et al. 1980; Omara & Sisodia 1990; Parton 2001; Goh et al. 2005) when sufficiently high doses of fluoroacetate are ingested (Peters 1957; Buffa et al. 1973). Central nervous system effects also occur in many species (Buffa et al. 1973; Kirsten et al. 1978; Kun 1982). Species differences in elevation of citrate concentration in different organs and tissues may in part explain species variation in response to 1080 (Bosakowski & Levin 1986) and the difference in susceptibility to 1080 described in the next section. Death may result from: (a) gradual cardiac failure or ventricular fibrillation; or (b) progressive depression of the central nervous system with either cardiac or respiratory failure as the terminal event; or (c) respiratory arrest following severe convulsions (Atzert 1971) linked to a combination of energy depletion at a cellular level, accumulation of citrate in affected tissue, and electrolyte disturbances including hypocalcaemia and hypokalaemia (Chi et al. 1996). In early literature it is reported that death in herbivores is the result of cardiac disorders, and in carnivores the result of central nervous system disorders. Death in omnivores tends to result from both cardiac and central nervous system disorders (Chenoweth 1949).

There has been limited recent research on the mode of action of 1080 that focuses on antidotes or treatments (Wickstrom et al. 1998; Norris et al. 2000; Cook et al. 2001; Robinson et al. 2002; Goh et al. 2005; Collicchio-Zuanaze et al. 2006; Goncharov et al. 2006). 1080 is thought to induce seizures in rats (*Rattus* sp.) through alterations to neurotransmitter concentrations within the brain (Wickstrom et al. 1998). The administration of neurotransmitter-modulating agents (e.g. glutamate inhibitors, GABA and serotonergic antagonists) reduced the toxic effects of 1080 (Cook et al. 2001; Goh et

2005). Calcium replacement may also have a role in offsetting fluoroacetate-induced hypocalcaemia (Robinson et al. 2002; Collicchio-Zuanaze et al. 2006). Despite these endeavours, ethanol, if taken immediately with the aim of reducing fluorocitrate synthesis, has remained the accepted antidote for 1080 poisoning in the last six decades (Goncharov et al. 2006). Veterinary treatment of poisoned dogs (*Canis lupis familiaris*) relies on symptomatic and supportive care (Norris et al. 2000; Goh et al. 2005; Parton et al. 2006). For example, poisoned dogs are sedated to reduce the possibility of seizures, and fluid levels are maintained to offset electrolyte imbalances (Goh et al. 2005; Parton et al. 2006). Detailed treatment regimens, provided by Parton et al. (2006), include the use of sodium bicarbonate or acetamide and decontamination to improve chances of survival.

Acute toxicity

In pest species, the LD_{50} of sodium fluoroacetate is usually 1 mg kg^{-1} or less, whereas in humans, LD_{50} doses have been estimated to be 2–5 mg kg^{-1} (Rammell & Fleming 1978). While fluoroacetate is a broad-spectrum toxin, there are some marked differences in susceptibility (Table 1). There is an extensive database on the acute toxicity of 1080 in a diverse spectrum of species, including birds, mammals, and reptiles (Atzert 1971; Harrison 1978; Rammell & Fleming 1978; Eisler 1995). Unlike most other vertebrate pesticides, 1080 also has insecticidal properties (David & Gardiner 1950; Negherbon 1959; Notman 1989; Booth & Wickstrom 1999). As with other poisons, the relative susceptibility and LD_{50} values can be influenced by the method used to deliver the poison, and by environmental conditions (Henderson et al. 1999). Dogs are extremely susceptible, and most other carnivores are highly sensitive to poisoning. Herbivorous mammals are less sensitive, and birds and reptiles less susceptible still (Atzert 1971; Rammell & Fleming 1978; Eisler 1995).

Table 1. LD_{50} values (mg kg^{-1}) for oral toxicity of sodium fluoroacetate to vertebrates (after Rammell & Fleming 1978; Hone & Mulligan 1982; Eisler 1995). These examples represent a very small proportion of the LD_{50} data available in the literature.

Vertebrate species	LD_{50} (mg kg^{-1})
Dog (*Canis lupis familiaris*)	0.07
Cat (*Felis domesticus*)	0.3
Pig (*Sus* sp.)	0.3
Rabbit (*Oryctolagus cuniculus*)	0.4
Sheep (*Ovis aries*)	0.4
Cow (*Bos taurus*)	0.4
Deer family (Cervidae)	0.5
Goat (*Capra hircus*)	0.6
Wallaby family (Macropodidae)	< 1.0
Rat (*Rattus* sp.)	1.2
Possum (*Trichosurus vulpecula*)	1.2
Human (*Homo sapiens*)	2.5
Weka (*Gallirallus australis*)	8.0
Duck family (Anatidae)	9.0
Clawed toad (*Xenopus* sp.)	500

Several studies have revealed that animals that forage in areas where fluoroacetate-producing plants are common have evolved an increasing resistance to fluoroacetate compared with animals from areas where plants containing the toxin are not indigenous. This phenomenon is well-documented in Australia where the effect is most dramatic in herbivores and seed eaters, which are more directly exposed to the toxin than carnivores (Twigg 1994). The emu (*Dromaius novaehollandiae*) is the oldest seed-eating bird species in Australia, and has a very high level of resistance to fluoroacetate, with an LD_{50} of 100–200 mg kg^{-1}. In contrast, seed-eating birds from regions outside the range of fluoroacetate-producing plant species have an LD_{50} in the range of 0.2–20 mg kg^{-1}. Similarly, the brushtail possum of south-western Australia is 150 times less susceptible to fluoroacetate poisoning than the same species in eastern Australia where plant species containing 1080 are not present. The biochemical basis for tolerance and species variation is not clear (Twigg et al. 1988a; Twigg 1994).

Species variation in mammals in response to fluoroacetate poisoning may be due in part to differences in the biochemical response of different organs to the toxin, and may be linked to differences in glutathione level and a glutathione-requiring defluorinating enzyme (Twigg 1994). For example, 1080-induced changes in the citrate content of the heart are more pronounced than in any other organ in sheep. For this reason, citrate concentrations in sheep hearts may have diagnostic value (Annison et al. 1960; Schultz et al. 1982). Guinea pigs (*Cavia porcellus*) and rabbits (*Oryctolagus cuniculus*), like sheep, are sensitive to myocardial damage. In rats there is some short-lived elevation of citrate in the heart, but in dogs elevation of citrate concentrations in heart tissue has been reported to be minimal (Bosakowski & Levin 1986). Furthermore, dogs do not show the electrocardiogram (ECG) changes seen in sheep that are suggestive of cardiac ischaemia (Matsubara et al. 1980). In comparison with dogs, the clinical signs in cats (*Felis domesticus*) are less severe (Eason & Frampton 1991). In birds, damage to wing muscle is a unique feature that occurs at sub-lethal dose levels (Ataria et al. 2000). Most deaths in mammals generally occur 6–48 h after ingestion of a lethal dose.

A recent review paper highlighting the effects of ambient temperature on possum mortality showed that the acute toxicity of 1080 is reduced at low temperatures (Veltman & Pinder 2001). This is important given that control programmes are mostly conducted in months with the coldest average temperatures (Veltman & Pinder 2001). Research in Australia has indicated that resistance can develop after repeated use. Thus, the LD_{50} for rabbits in Western Australia appears to have increased dramatically, from $0.34 - 0.46$ mg kg^{-1} over 30 years ago, to $0.74 - 1.02$ mg kg^{-1} (Twigg et al. 2002).

Toxicokinetics and exposure risks

1080 is absorbed through the gastrointestinal tract, or via the lungs if inhaled. It is not readily absorbed through intact skin, but can be absorbed more easily through cuts and abrasions (Saunders et al. 1949; Pattison 1959). Studies of laboratory animals since the 1950s have shown that sub-lethal amounts of 1080 are excreted both unchanged and as a range of non-toxic metabolites. Rapid conversion to non-toxic metabolites reduces the toxicity of sub-lethal doses but does not mean there will be no toxic effects.

After oral or intravenous dosing of laboratory rodents,

1080 is rapidly absorbed and distributed through the soft tissues and organs (Hagan et al. 1950; Egekeze & Oehme 1979; Sykes et al. 1987). This contrasts with the action of commonly used anticoagulant rodenticides such as brodifacoum, which preferentially bind to liver cells (Bachman & Sullivan 1983). 1080 is excreted as unchanged fluoroacetate and a range of metabolites (Gal et al. 1961; Schaefer & Machleidt 1971). Approximately 30% of a dose of injected 1080 administered to rats was excreted unchanged in the urine over 4 days (Gal et al. 1961). At least seven unidentified metabolites other than fluoroacetate and fluorocitrate, the toxic metabolite of 1080, were also detected in rat urine (Gal et al. 1961).

Administration of ^{14}C-labelled fluoroacetate to rats showed that the toxic metabolite fluorocitrate accounted for only 3% of the radioactivity (Gal et al. 1961); this was confirmed by Schaefer and Machleidt (1971). The major unidentified metabolite, unlike fluorocitrate, did not inhibit the activity of aconitase (Gal et al. 1961). Phillips and Langdon (1955) suggested that the unidentified metabolites include non-saponifiable lipids that probably serve as intermediates for cholesterol, and some radioactivity was found in fatty acids and cholesterol in the liver. Up to 3% of the radioactivity appeared as respiratory CO_2, and cleavage of the C-F bond is a significant detoxification process (Gal et al. 1961).

Savarie (1984) noted that 1080 is readily distributed through the body and that synthesis of fluorocitrate occurs in the mitochondria. Defluorination of 1080 or its metabolites,

including fluorocitrate, has been demonstrated in several animal species and other living organisms (Kirk & Goldman 1970; Smith et al. 1977; Egekeze & Oehme 1979; Soiefer & Kostyniak 1983, 1984; Twigg et al. 1986; Tecle & Casida 1989). Although fluoride is also extensively excreted, primarily in urine, some deposition occurs in bone (Sykes et al. 1987). Tissue and blood levels of fluoride are briefly elevated following exposure to 1080 and then return to normal levels (Gooneratne et al. 1994). Current understanding with regard to the metabolites of 1080 is summarised in Table 2.

The earliest reports on rats suggested that some 1080 is retained for 1–4 days (Hagan et al. 1950; Gal et al. 1961). However, in a study of mice (*Mus musculus*), 1080 concentrations in plasma, muscle, and liver decreased by half in less than 2 h (Sykes et al. 1987). Prolonged persistence of 1080 in animals after sub-lethal exposure therefore seems unlikely, and has been confirmed in more recent studies of larger animals such as rabbits, goats, possums, and sheep (Gooneratne et al. 1994; Eason et al. 1994a), in which 1080 was readily absorbed and excreted. Highest concentrations occurred in the blood, with moderate levels in the muscle and kidneys, and the lowest concentration in the liver. In sheep, the highest concentrations in blood occurred 2.5 h after dosing, with negligible amounts of 1080 seen in tissue and plasma 4 days after dosing. The elimination half-life in blood is 11 h or less in sheep (11.0 h), goat (5.5 h), rabbit (1.1 h), mouse (2.0 h) and possum (9.0 h) (Eason et al. 1994a), and contrasts with

Table 2. Summary of 1080, its metabolites and their toxicity.

Compound	Toxicity
1080	Not toxic until metabolised to fluorocitrate
Fluorocitrate	Highly toxic
Fluoride	Potentially toxic but levels in poisoned animals not high. Toxicity overshadowed by effects of fluorocitrate
Fatty acids	Not toxic[1]
Cholesterol	Not toxic[1]
CO_2	Not toxic[1]
Other unidentified metabolites	Do not inhibit aconitase; thought to be non-toxic

[1] = Natural biochemical compounds

Table 3. Elimination half-lives of 1080 in blood plasma and muscle for possum, rabbit and mouse, and for brodifacoum in blood plasma and liver of possums.

Species	Compound	Sample	Route of administration	Dose ($mg\ kg^{-1}$)	Elimination half-life (h, unless specified)
Sheep	1080	Plasma	Oral	0.1	11.0 [a, b]
		Muscle			12.0
Goat	1080	Plasma	Oral	0.1	5.5 [a]
		Muscle			n.d.
Possum	1080	Plasma	Oral	0.1	9.0[a]
		Muscle			n.d.
Rabbit	1080	Plasma	Oral	0.1	1.1[c]
		Muscle		0.1	0.4[c]
Mouse	1080	Plasma	Intravenous injection	0.4	2.0[d]
		Muscle		0.4	1.7[d]
Possum	Brodifacoum	Plasma	Oral	0.1	Approx 8 days [e]
		Liver			> 252 days [e]

n.d. = not determined
[a] Eason et al. 1994b; [b] Rammell 1993; [c] Gooneratne et al. 1994, 1995; [d] Sykes et al. 1987; [e] Eason et al. 1996.

longer elimination half-lives in blood and liver for brodifacoum in possums (Eason et al. 2006), as shown in Table 3.

To further examine the persistence of 1080 and its potential to contaminate milk and meat, two groups of lactating ewes were fed 1080 baits ('high dose', 0.25 mg kg^{-1} body weight; 'low dose', 0.01 mg kg^{-1} body weight) (Milne et al. 2002). 1080 concentrations in blood and milk were examined 4–96 h after dosing. Four hours after dosing, 1080 concentration in blood (0.56 ± 0.04 μg ml^{-1}) was 30 times greater than in milk (0.017 ± 0.001 μg ml^{-1}) in the high-dose group. Lower concentrations of 1080 were detected in the blood after 72 h (0.020 ± 0.002 μg ml^{-1}), and in milk, concentrations were near the analytical level of detection (0.0006 μg ml^{-1}). The low-dose ewes had no detectable 1080 in blood or milk 72 h after dosing (Milne et al. 2002). These recent findings are consistent with earlier research by Gooneratne et al. (1994) and Eason et al. (1994a).

Limited research has been conducted on the pharmacokinetics of fluoroacetate in invertebrates. In a laboratory study, tree weta (*Hemideina* sp.) and cave weta (Rhaphidophoridae) were dosed with 1080 and the persistence of 1080 residues at specified times after dosing was determined. In this experiment most of a 1080 dose was eliminated from weta 6–10 days after exposure, and all weta survived dose levels of 15 mg kg^{-1} (Eason et al. 1993). Similar results were obtained for a native ant (*Huberia brouni*) (Booth & Wickstrom 1999). Insects have been monitored in forests for 1080 residues after aerial sowing of toxic baits for possum control. No 1080 was found in living earthworms (Oligochaeta), spiders, beetles, millipedes, or centipedes. Although 1080 was found in some cockroaches (Blattidae), bush weta, and cave weta during the period the baits were on the ground, after 3–4 weeks (when baits could have been eaten at any time) all invertebrate samples were free from 1080 residues (Eason et al. 1993). Persistence of 1080 in invertebrates appears to be short-lived, so the risk to insectivorous birds or other predators will also be confined to a short period after sowing baits. However, large invertebrates frequently eat bait (Spurr & Drew 1999) and, since species like weta can contain large amounts of bait, secondary poisoning via this route is possible. In an unusual recent study, the toxicity and persistence of 1080 was assessed in a land crab (*Gecarcinus lagostoma*) prior to the eradication of feral cats on Ascension Island to help protect breeding seabird populations. The authors reported most 1080 residues were eliminated from crab tissue within 9–11 days (Pain et al. 2008).

As illustrated above, toxicokinetics in different species has been used to help add some clarity to exposure risk. Eason et al. (2008) recently classified compounds used for animal pest control into four groups based on their persistence in sub-lethally exposed animals, to help distinguish between the risks associated with different vertebrate pesticides. The criteria for the four groups, and the allocation of different compounds to them, are described below and in Table 4.

Group 1. Sub-lethal doses of these poisons are likely to be substantially excreted within 24 h (e.g. cyanide, zinc phosphide, and 1080). In the case of 1080, complete excretion of all residues may take up to 4–7 days. Like other compounds in this group, 1080 will not readily bioaccumulate.

Group 2. Residues resulting from sub-lethal doses of these poisons are likely to be substantially cleared from the body within 2–4 weeks (e.g. pindone).

Group 3. Residues resulting from sub-lethal doses of these poisons are likely to be cleared from the body within 2–4 months (e.g. cholecalciferol and coumatetralyl).

Group 4. Residues resulting from sub-lethal doses of these poisons may never be completely cleared from the body (e.g. brodifacoum), and therefore bioaccumulate.

If recommended practices are followed in pest control operations, 1080 is unlikely to be present in meat for human consumption because of its comparatively rapid elimination as illustrated in Table 4. Where any contact of livestock (farm animals or animals intended for slaughter) with 1080 is suspected, researchers have suggested that an adequate margin of safety should be achieved by imposing a minimum withholding period of 5 days, during which animals should not be sent to an abattoir. Should a death in a flock or herd be attributed to 1080, the withholding period for the surviving stock should be doubled to 10 days and animals should be removed to a 1080-free pasture (Rammell 1993; Eason et al. 1994a) to allow for complete metabolism and excretion of any 1080 residues. The New Zealand Food Safety Authority has a caution period for hunting wild animals for procurement

Table 4. Comparative toxicokinetics and expectation for persistence of residues in sub-lethally exposed target or non-target species (adapted from Eason et al. 2008). For definition of groups, see text.

Group	Compound	Half-life values	Likely persistence of residues after sub-lethal exposure*
1	Cyanide	+	12–24 h
	Zinc phosphide	+	12–24 h
	1080	<11 h	7 days
2	Pindone	2.1 days	4 weeks
3	Cholecalciferol	10–68 days	3 months
	Coumatetralyl	50–70 days	4 months
4	Brodifacoum	130–350 days	24 months or longer

+ = No published value but likely to be <12 h.

* Values are mostly based on data from rats as surrogates for non-target species.

of meat of 2 months and 100 mm rainfall, or 4 months. While 1080 is eliminated comparatively rapidly from living animals, it can persist in carcasses for many months and will pose a risk to scavenging dogs until the carcasses are broken down (Meenken & Booth 1997). In possums that were sub-lethally poisoned, 1080 had a half-life of 9 h, suggesting no significant amount of 1080 would be expected in the tissues of live possums 4 days after exposure. In contrast, possum carcasses collected from the field were shown to pose a serious risk to dogs even up to 75 days after the poisoning operation (Meenken & Booth 1997).

Toxicodynamics and welfare

The latent period between the time fluoroacetate is ingested and the appearance of clinical signs in mammals is between 0.5 and 3 h (Chenoweth 1949; McIlroy 1981a). Peak plasma concentrations of fluoroacetate occurred in possums and rabbits 0.5 h after ingestion, in 0.75 h in goats, and in 2.5 h in sheep (Eason et al. 1994a). These values correlate with the latent period between ingestion and clinical signs, and reflect the time taken for absorption and distribution of fluoroacetate, and the conversion of fluoroacetate to fluorocitrate. Animals receiving small sub-lethal doses of 1080 show mild signs of poisoning, metabolise and excrete 1080 within 1–4 days, and then recover (Egekeze & Oehme 1979; Eason et al. 1997). Animals receiving a lethal dose usually show more severe signs of poisoning in addition to non-specific clinical signs such as nausea and vomiting. Specific signs include cyanosis, drowsiness, tremors, staggering, and death from ventricular fibrillation or respiratory failure (Dreisbach 1983). The clinical signs of 1080 poisoning in birds vary according to the species. Common signs may be lack of balance, slowness, ruffled feathers, and salivation. Vomiting occurs in some species such as raptors. In the terminal phase of poisoning, birds and mammals may exhibit convulsions and coma.

While the ecological impacts of 1080 baiting have been the subject of much research, the animal welfare implications have received comparatively less attention. In the past it has been suggested that 1080 is a humane toxin in herbivores but less so in carnivores (Batcheler 1978; Morgan 1990). Authors that reported 1080 to be a humane poison in species such as possums did so in part based on observation of subdued behaviour in herbivores and death from cardiac failure (Batcheler 1978; Morgan 1990). In carnivores, and notably in dogs, central nervous system disturbances are marked, and poisoned dogs run uncontrollably, retch and vomit, and appear distressed and agitated with prolonged involuntary muscle contractions exacerbated by convulsions and seizures prior to death from respiratory failure (Egekeze & Oehme 1979; Sherley 2007). Authors of articles that conclude that 1080 is a humane poison in carnivores base their conclusions largely on the argument that convulsive seizures seen in the final stages of 1080 toxicosis indicate that affected animals are in an unconscious state and are unable to perceive pain (Gregory 1996; Williams 1996; Twigg & Parker 2010). Other publications describe awareness during seizures or periodic lucidity that suggests central nervous system disruption cannot be assumed to produce a constant, pain-free state and conclude that 1080 should not be considered a humane poison (Sherley 2004, 2007; Cooper et al. 2007). People who have ingested 1080 by accident or in suicide attempts report abdominal pain, agitation and vomiting (Chi et al. 1996). The finding of Chenoweth and St. John

(1947) (reviewed by Sherley 2007) that electroencephalogram (EEG) disturbances similar to the grand mal convulsions of epilepsy were common in poisoned dogs could be supportive of periods of unconsciousness during fitting. However, Foss (1948) indicated that a dog poisoned experimentally with fluoroacetate was conscious during a period of convulsion. Twigg and Parker (2010) disagree, and argue that 1080 will impair the functioning of pain receptors and suggest that when animals are showing disturbing behaviour, they may in fact be incapable of feeling pain.

In addition to challenging the view that animals poisoned with 1080 are unconscious during seizures, Sherley (2004, 2007) also challenged the generalisation that herbivores die of cardiac failure, that carnivores show more extensive central nervous system distress, and that omnivores exhibit a combination of signs. Potentially painful or distressing signs of fluoroacetate poisoning in herbivores have been described in the literature and include tremors, vomiting and retching, muscle weakness, partial paralysis and respiratory distress (Chenoweth & Gilman 1946; McIlroy 1981b; Schultz et al. 1982; Sherley 2004, 2007). While the argument that 1080 induces a humane death in target species has been challenged (Sherley 2004, 2007), it is clear that the symptoms observed in possums (Morgan 1990; Littin et al. 2009) indicate considerably less severe distress than those reported in species such as dogs (Chenoweth & Gilman 1946). Possum bait formulations in New Zealand contain 0.15% 1080 and are optimised to induce as swift a death as possible (Henderson et al. 1999).

A summary of behavioural and pathological observations made on caged possums fed a lethal dose of 1080 are given in Table 5 and comparisons between 1080 and three other toxins for which published data on welfare exists are made in Table 6. Information on the time to onset of the first sign of poisoning, the time of onset and duration of each effect, and the time until death is provided. Effects specific to the poison (e.g. spasms, vomiting) are also described. These provide information on the intensity and duration of each effect, and the mean overall duration of effects for each poison.

The humaneness of a poison depends 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. 1998a). These stages have been described in possums for cyanide (Gregory et al. 1998), phosphorus (O'Connor et al. 2007), brodifacoum (Littin et al. 2002), and 1080 (Littin et al. 2009).

Table 5. Time after dosing at which clinical signs of poisoning appeared in caged possums ($n = 17$) fed a lethal dose of 1080. Times are the mean ± SEM (adapted from Littin et al. 2009).

Clinical sign	Time (h: min)
Onset of change in appearance	1:50 ± 0:09
Onset of retching and vomiting	2:53 ± 0:13
Onset of uncoordination	3:37 ± 0:32
Onset of spasms, tremors and seizures	4:05 ± 0:40
Time to death	11:26 ± 1:55

Table 6. Summary of mean times to onset of clinical signs of toxicosis, duration of key symptoms during sickness behaviour, and time to death in possums following ingestion of poison baits in four studies.

Toxin	Mean time until onset of sickness	Sickness behaviour	Mean duration	Mean time until death	Study
Cyanide	3 min	Ataxia, impaired co-ordination, breathlessness, muscular spasms, unconscious after 6.5 min	15 min	18 min	Gregory et al. 1998
1080	~ 2 h	Ataxia, occasional retching, spasms, breathlessness, laboured breathing	9 h	11.5 h	Littin et al. 2009
Phosphorus paste	6 h	Retching, vomiting, hunched posture, intermittent repositioning, ataxia	19 h	25 h	O'Connor et al. 2007
Brodifacoum	14 days	Anaemia, haemorrhage, loss of appetite, hunched posture, anorexia	7 days	21 days	Littin et al. 2002

Cyanide is the fastest acting vertebrate pesticide used in New Zealand and causes the least distress. It is therefore regarded as the most humane of the toxicants evaluated. In contrast, brodifacoum is slow-acting, and possums sometimes take a long time to die, with an average of 21 days until death after eating baits. Phosphorus causes behavioural responses associated with inflammation of the stomach lining and duodenum (e.g. a crouched/hunched posture). The times to death after possums eat 1080 baits (or other poisons) are on average less than those recorded for phosphorus, and possums dosed with 1080 exhibit few overt signs of pain.

While 1080 is not as humane as cyanide, it would appear to be preferable to compounds like brodifacoum for possum control (Littin et al. 2002, 2009; Twigg & Parker 2010). In recognition of the potential to improve the humaneness of 1080, which is particularly important in control of carnivores such as foxes in Australia, recent research has focused on co-administration of analgesics with 1080, and some improved animal welfare outcomes have been reported (Marks et al. 2009).

Sub-lethal effects and exposure risk in humans and animals

Risk assessment is improved when fundamental research provides information into the fate, effects, and risk of exposure to 1080. Hence, toxicokinetics and the mechanism of toxicity are important in understanding the response of an animal or humans to a foreign compound.

1080 clearly has the potential to kill and cause sub-lethal effects in humans, but exposure of individuals or communities to amounts of 1080 that would cause such effects is most unlikely. The acute toxicity of 1080 is well understood, and the toxic manifestations of sub-lethal aconitate hydratase inhibition have been extensively studied as described above (in Mode of Action). They are explored further below.

As noted above, with the disruption of mitochondrial function and the tricarboxylic acid cycle, energy production in the form of ATP decreases, citrate and other breakdown products of fats accumulate, and cell function is impaired. This results in accumulation of intracellular citrate in the tissues and plasma, leading to energy deprivation and death when sufficiently high doses are ingested. Sub-lethal toxicological effects associated with chronic exposure are also likely to be linked to inhibition of aconitate hydratase and the mitochondrial dysfunction caused by 1080.

Known target organs in animals include the heart, lungs, liver, kidney, testes and foetus. Although 1080 itself is not cumulative (Rammell 1993; Eason et al. 1994a), animal studies demonstrate that cumulative damage to the heart or other organs from repeated exposure to large sub-lethal doses of 1080 can occur (Annison et al. 1960; Whittem & Murray 1963; Schultz et al. 1982; Gooneratne et al. 2008). That single or repeated sub-lethal exposures to fluoroacetate can have toxic effects on the heart has been known for some time (Steyn 1934; Quin & Clark 1947; Hicks et al. 1952). The target organs vary to some extent in different species, and may relate to differences in the citrate response in different species or the metabolic activity of different tissues. Accordingly, it is likely that a number of biochemical mechanisms are responsible for fluoroacetate toxicity in tissues.

Results of developmental toxicity studies in rats showed that a single sub-lethal dose had no effect (Spielmann et al. 1973). However, when female rats were exposed to relatively high doses (0.33 and 0.75 mg kg^{-1}) for about 30% of their gestation period, mild skeletal effects were detected (Eason et al. 1999). The 'no observable effects level' (NOEL) for developmental effects was 0.1 mg kg^{-1} day^{-1} based on observations of bent ribs at 0.33 mg kg^{-1} day^{-1}. The NOEL for rats administered 1080 via oral gavage for 90 days was 0.075 mg kg^{-1} day^{-1}. Microscopic changes in the testes and heart were seen in males dosed with 1080 at 0.25 mg kg^{-1} day^{-1} (Eason & Turck 2002).

The cancer-inducing potential of 1080 has been evaluated by in vitro and in vivo testing. Results of the Ames assay, the mouse lymphoma assay and the mouse micronucleus assay (bone marrow assay to detect chromosome anomalies) indicate that 1080 is not mutagenic (Eason et al. 1999). In the mouse micronucleus assay, mice were given oral 1080 doses of 0.75, 1.5, 3.0, 6.0 and 7.5 mg kg^{-1}, and no mutagenicity was observed at any dose level.

In addition to these target organs and other effects of 1080, the potential endocrine disrupting activity of 1080 has been accessed at the receptor level. Endocrine disrupting chemicals (EDCs) are defined as synthetic chemicals that, once absorbed, have oestrogenic or anti-androgenic activity, which is mediated by their ability to bind or block oestrogen or androgen receptors (Colborn et al. 1996; Tremblay et al. 2004, 2005). The reproductive studies described above (Eason et al. 1999) followed OECD (Organisation for Economic Co-operation and Development) protocols to determine teratogenicity. The potential effects of 1080 on endocrine systems have been raised as a particular concern (Weaver 2003). EDC effects cannot be determined from conventional regulatory toxicology, which focuses on different end-points, and requires specialised cell-culture test systems. The ability of fluoroacetate and its toxic metabolite, fluorocitrate, to act as EDCs has therefore been investigated in mammals and fish.

1080 and fluorocitrate were first tested to determine whether they can mimic natural oestrogen binding in vitro to mammalian oestrogen receptors. Preparations were isolated from sheep uterine tissue and used in a competitive receptor-binding assay to determine their binding affinity. In that initial study the known oestrogenic pesticides o,p'-DDT and tamoxifen, evaluated as positive controls, showed significant displacement of natural oestrogen, whereas neither 1080 nor its active metabolite (fluorocitrate) had any effect on oestrogen receptor binding. Therefore, they are unlikely to promote oestrogenic or anti-oestrogenic effects by binding to a mammalian oestrogen receptor (Tremblay et al. 2002, 2004) or adversely affect the breeding performance of native animals (Twigg & Parker 2010).

More recently, the potential of 1080 and fluorocitrate to interact with androgen and oestrogen receptors was assessed in animal and human test systems. Neither 1080 nor fluorocitrate showed androgenic activity in a rainbow trout (*Oncorhynchus mykiss*) androgen receptor assay. Furthermore, neither compound showed androgenic or anti-androgenic activity in a reporter gene assay using human androgenic receptors, and in a human oestrogen receptor assay no oestrogenic or anti-oestrogenic activity occurred (Tremblay et al. 2004, 2005). The researchers concluded that 1080 and fluorocitrate lack the properties of the classical EDCs. Neither compound has oestrogenic or anti-androgenic properties. Unlike well-known environmental EDCs like DDT or dioxins, 1080 and fluorocitrate are simple molecules, which are water soluble and do not persist for long in the environment.

While 1080 does not have the properties of a typical EDC, it is a male reproductive toxicant with effects on testes of mammals (Mazzanti et al. 1965; Sullivan et al. 1979; Hornshaw et al. 1986; Wolfe 1998; Shinoda et al. 2000; Eason & Turck 2002) and other species (Balcomb et al. 1983; Twigg et al. 1988b). In the most recently completed 90-day toxicology study of 1080 in rats (Eason & Turck 2002), similar effects were observed. Microscopic changes in males dosed with 1080 at $0.25\,\mathrm{mg\,kg^{-1}\,day^{-1}}$ included hypospermia in the epididymides, degeneration of the seminiferous tubules of the testes, and cardiomyopathy. The NOEL for rats administered 1080 via oral gavage for 90 days was $0.075\,\mathrm{mg\,kg^{-1}\,day^{-1}}$. The lowest observable effects level (LOEL) dose was $0.25\,\mathrm{mg\,kg^{-1}\,day^{-1}}$. Given that 1080 lacks EDC-like activity, these sub-lethal effects on testes are therefore distinct from those mediated by 'classical' endocrine disruptors and are likely to occur through a direct intracellular effect on the mitochondria.

The effects described above have mostly been observed in animal studies. There are also a number of cases of inadvertent human poisoning or exposure described in the literature (Hayes & Laws 1991). In general, these cases are not relevant to the effects of low level exposure to 1080. In several such cases symptoms included vomiting, arrhythmia, tachycardia, cardiac dysfunction, convulsions and coma, sometimes associated with temporary or more prolonged brain damage in survivors. This information from poisoning cases is therefore not particularly helpful when assessing the potential of 1080 to have sub-lethal effects within the context of its use in New Zealand for pest control, and potential exposure patterns.

To summarise, the known target organs for sub-lethal effects following 1080 exposure include the heart, lungs, liver, kidney, testes and foetus, with sub-lethal effects having been observed at doses in the range 0.055 to $0.25\,\mathrm{mg\,kg^{-1}}$ (Annison et al. 1960; McTaggart 1970; Buffa et al. 1977; Sullivan et al. 1979; Schultz et al. 1982; Trabes et al. 1983; Chung 1984; Savarie 1984; Twigg et al. 1988b; Chi et al. 1996; Gregg et al. 1998; Eason et al. 1999; Eason & Turck 2002). Concern regarding whether or not these effects are likely in humans can be explored by examining the likelihood of exposure.

Beasley (1996) pointed out that theoretical routes of human exposure to 1080 might be 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 1080 solution. Bait consumption is unlikely and 1080 does not persist for long in sub-lethally exposed livestock; ingestion in food is therefore unlikely (Rammell 1993; Eason et al. 1994b). Instances of contamination of meat and milk with 1080 are considered to be rare incidents. Furthermore, environmental contamination from dust following aerial possum control has been shown to be minimal and short-lived (Wright et al. 2002).

The most significant source of general public exposure to 1080 is perceived to be contamination of surface water in public water supply catchments by aerially sown baits. Consequently, 1080 water monitoring programmes have been undertaken following numerous pest control operations using aerially sown 1080 baits since 1990. There has been no evidence of significant or prolonged 1080 contamination of surface waters (see below). The risks associated with 1080 to human health are determined by the toxicity of 1080 and the potential for exposure: risk = hazard × exposure. The innate toxicity of 1080 is not in question, as there are clearly identified lethal and sub-lethal effects as illustrated above. However, exposure risk to humans is very low with the exception of the small group of workers in the pest control industry, a group that has been closely monitored to try to ensure minimal exposure (Beasley et al. 2009).

To address the potential for 1080 to exert effects on human health following its use for pest control in New Zealand, we have focused on the known sub-lethal effects outlined above, the mode of action of 1080 and the potential for exposure to 1080, and hence the likelihood of sub-lethal effects. Our conclusions in this section are based on our understanding of the effects described in animals receiving low doses coupled to different exposure scenarios.

It is apparent that 1080 exposure could have a multitude of effects resulting from aconitate hydratase inhibition and secondary biochemical perturbations. Chronic toxicology studies in animals as human substitutes, and mutagenicity tests, enable elucidation of potential toxicological effects in humans if they are exposed. Pathological changes observed in chronic animal studies are consequences of primary and

secondary effects of 1080 on the body. Animal toxicology studies by Eason and Turck (2002) have been used to define conservative no-effect levels, and more recently, tolerable daily intake (TDI) values of $0.03 \, \mu g \, kg^{-1} \, day^{-1}$ (Foronda et al. 2007a, b) have been proposed based on a NOEL of $0.075 \, mg \, kg^{-1} \, day^{-1}$ and a LOEL of $0.25 \, mg \, kg^{-1} \, day^{-1}$ (Eason & Turck 2002). The TDI proposed by Foronda et al. (2007a, b) is of relevance to workers at the bait manufacturing plants but not to the broader community, which should not be exposed to 1080 under normal baiting practices. Weaver (2003) suggested additional toxicology studies would be valuable. Equally, or probably more importantly in a practical sense, the use of 1080 must continue to include safeguards that focus on those individually handling 1080 or 1080 baits to ensure they do not ingest, inhale or absorb 1080.

Fate in the environment, toxicity to aquatic organisms and risk of exposure from contaminated water supplies

Persistence in soil and plants

After leaching from baits, 1080 can be metabolised (broken down) by soil microorganisms such as *Pseudomonas* and *Fusarium* species (Bong et al. 1979; Walker & Bong 1981; King et al. 1994). Under favourable conditions, such as 11–20ºC and 8–15% moisture, 1080 may be significantly defluorinated in 1–2 weeks. In less favourable conditions, breakdown might take several weeks, and in extreme cold and drought, 1080 residues might persist in baits or in the soil for several months (King et al. 1994).

Presumably, naturally occurring fluoroacetate is diluted by rainwater and breaks down in soil after leaves and seeds drop to the ground or when plants die. Not all microorganisms can readily defluorinate fluoroacetate and the rate of metabolism differs with different species of soil bacteria and fungi (King et al. 1994). Enzymes capable of defluorinating fluoroacetate have been isolated from several soil microorganisms. The fluoride–carbon bond is cleaved and, ultimately, enzyme-bound intermediates form non-toxic metabolites such as glycolate (O'Hagan & Harper 1999). The gene encoding for the bacterial enzyme capable of defluorinating fluoroacetate in soil has been cloned and expressed in the rumen bacterium *Butyrivibros fibriscolens*. This organism has then been employed, in an experimental setting, to infect the guts of sheep in an attempt to protect against dietary poisoning by fluoroacetate-containing plants. Early indications are that the genetically modified organisms become established sufficiently well to give the sheep a degree of resistance to fluoroacetate (Annison & Bryden 1998; Gregg et al. 1998). This line of research has not been pursued further and there are no plans to introduce genetically modified organisms into New Zealand livestock to protect them from 1080 baits. However, recent research has continued to clarify the enzymes involved in cleavage of the carbon-fluoride bond and their gene coding in bacteria (Kurihara et al. 2003)

1080 derived from baits will also be dispersed by water since it is highly water soluble and mobile (Parfitt et al. 1995). If heavy rainfall follows the use of 1080 baits, dilution to immeasurable concentrations (<0.0001 ppm) may precede biodegradation. In comparison with cereal bait, 1080 is retained in carrot baits and will only leach slowly from carrots into the soil (Bowen et al. 1995). However, control operations are planned to coincide with periods of dry weather, and some defluorination by microorganisms on the decaying baits and in the soil around baits is probable, particularly if baits become moist.

Recent studies have focused on determining the actual concentrations of 1080 in soil and leaf litter and whether or not these concentrations would be likely to affect soil organisms. The highest concentration of 1080 recorded in soil or leaf litter samples after aerial baiting was $0.19 \, mg \, kg^{-1}$ (Wright et al. 2002), but where other samples have contained 1080 with detectable residues, they were below $0.01 \, mg \, kg^{-1}$. The toxicity of 1080 to earthworms has been evaluated in separate laboratory studies. A lowest observable effect concentration (LOEC) of $90 \, mg \, kg^{-1}$ was determined based on reduced earthworm cocoon production (O'Halloran et al. 2005), which represents a very high concentration compared with that likely to occur in practice. No mortality was observed in worms exposed to a 1080 concentration of $865 \, mg \, kg^{-1}$, and concentrations of $1000 \, mg \, kg^{-1}$ in soil did not affect mineralisation of nitrogen by soil microorganisms (O'Halloran et al. 2005). Very recent research on the aerobic transformation of 1080 in soils has confirmed that breakdown varies significantly depending on temperature, and that the key transformation products are hydroxyacetic acid, also known as glycolate, and carbon dioxide (Fisher & Northcott 2010).

1080 that has leached into soil may be absorbed by plants (Atzert 1971; Rammell & Fleming 1978). An experiment by David and Gardiner (1950) showed that broad beans (*Vicia faba*) were able to systematically accumulate 1080 through the roots to the detriment of aphids (*Aphis fabae*) introduced to the test plants. Work on cabbage (*Brassica oleracea capitata*) also showed systematic accumulation of 1080 through the roots and subsequent toxicity to aphids, and was reviewed by Negherbon (1959). Recent research on plants has focused on determining whether 1080 residues might have an adverse effect on plant growth and whether or not uptake of 1080 by food plants could adversely affect animal or human health. Emergence of lettuce seedlings was adversely affected at 1080 concentrations in soil of $7 \, mg \, kg^{-1}$, a significantly greater concentration than that found in soil after the aerial application of bait (O'Halloran et al. 2005). To investigate whether herbivores may be at risk of secondary poisoning if they consume plants that have taken up 1080 leached from bait, concentrations of 1080 were assessed in broadleaf (*Griselinia littoralis*) and perennial ryegrass (*Lolium perenne*) following simulated baiting (Ogilvie et al. 1998). The observation that both species absorbed 1080, which reached a peak and then declined to near the limits of detection, supported previous findings that plants can degrade 1080 (Preuss & Weinstein 1969; Ward & Huskisson 1969). The concentration achieved in broadleaf and ryegrass would be most unlikely to cause poisoning of herbivores (Ogilvie et al. 1998).

Recently, research has been undertaken to determine whether plants used by Māori (the indigenous people of New Zealand) for food and medicine can take up 1080. Cereal baits containing 0.15% 1080 were placed at the base of naturally growing individual plants of two species, pikopiko (*Asplenium bulbiferum*) and karamuramu (*Coprosma robusta*). Plants were sampled at various times up to 56 days and analysed for 1080 content. No 1080 was detected in any of the pikopiko samples, but it was detected in karamuramu at a maximum concentration of 5 ppb ($\mu g \, L^{-1}$) after 7 days, and 2.5 ppb after 14 days. Concentrations declined to zero at 28 days (Ogilvie et al. 2006). A similar study was carried out

on two commonly eaten plant species, puha (*Sonchus asper*) and watercress (*Nasturtium microphyllum/officinale*), using methods comparable to those of Ogilvie et al. (2006). Samples of plant tissue were taken at various times up to 38 days for puha, and up to 17 days for watercress. 1080 was detected at a maximum concentration of 15 ppb in a single puha sample after 3 days, and at a maximum of 63 ppb from a single watercress sample on day 8. By day 38, all 1080 concentrations were below the method's detection level for puha; 1080 was not detected in watercress after day 8 (Ogilvie et al. 2009).

Water and 1080

Beasley (1996) pointed out the theoretical routes of human exposure to 1080, as mentioned above. Of these routes, the one generally perceived to be most significant is contamination of surface water in public water-supply catchments by aerially sown 1080 baits.

In the Drinking Water Standards for New Zealand, issued in 2000 by the Ministry of Health (MoH), the provisional maximum acceptable value (PMAV) for 1080 in water is 3.5 ppb (μg L^{-1}). As a precautionary measure, MoH also recommended that water taken from catchments sown with 1080 baits should not be used for human supply until tests show that the concentration of 1080 is below 2 ppb. In this section we summarise research on the fate of 1080 after aerial application of 1080 bait, with a focus on water.

1080 is highly water-soluble (Parfitt et al. 1995). Some baits will fall into streams following aerial application, even when major watercourses are avoided. Laboratory studies have shown that 1080 is biodegraded by aquatic plants and microorganisms. Parfitt et al. (1994) showed that 1080 was degraded in biologically active water in 2–6 days, and Eason et al. (1993) showed that 1080 declined by approximately 70% in 1 day and to below detectable limits in 4 days in aquaria containing plants and invertebrates. Research has also indicated that residues of 1080 do not persist in aquatic plants. Ogilvie et al. (1996, 1998) showed that warmer temperatures significantly increased the rate of 1080 degradation, and was enhanced in the presence of aquatic plants and microorganisms. Their laboratory studies also demonstrated that concentrations of 1080 in aquatic plants decreased in parallel with the decline in concentration of 1080 in water (Ogilvie et al. 1995, 1996; Booth et al. 1999), indicating degradation of 1080 within the plants. Some of these studies of 1080 breakdown in water and aquatic plants (Ogilvie et al. 1996; Booth et al. 1999) deliberately used solutions containing much higher concentrations of 1080 (0.12 – 5 ppm) than have been detected in streams during field monitoring (0.1 – 9 ppb) to simulate worst-case scenarios.

Water monitoring of streams and waterways after aerial applications of 1080 baits was initiated by research teams studying the fate of 1080 in soil, invertebrates and water (Eason et al. 1992, 1993). It was continued in order to provide community reassurance and evolved into routine practice by the mid-1990s (MoH 1995). Results of the initial research and subsequent monitoring demonstrated that there has been no evidence of 1080 presence in reticulated water and no evidence of significant or prolonged 1080 contamination in surface waters (Parfitt et al. 1994; Eason et al. 1992, 1993; Hamilton & Eason 1994; Meenken & Eason 1995; Booth et al. 1997; Eason et al. 1999; Eason & Wright 2001; Wright et al. 2002; Eason & Temple 2008). Between 1990 and 2006, Landcare Research analysed 1973 water samples taken following 298 pest control operations. Of the samples taken, 96.3% were free of detectable 1080, including all 136 samples from reticulated town water supplies for household consumption (Booth et al. 2007).

Of the 3.7% (72) of water samples that contained detectable 1080 residues, most (65) had 1080 concentrations of less than 1 μg L^{-1} (ppb). When 1080 was found in samples in monitoring programmes, it was at very low concentrations, typically in small streams in remote locations. Often baits were seen near where samples were taken. The contamination was transient and not picked up in repeat samples.

Suren (2006) inspected small streams during aerial distribution of cereal baits containing 0.15% 1080, to quantify the number of baits falling into waterways. The number of baits found in streams was related only to bait size, with more small baits being found in streams. The leaching rates of 1080 baits were also examined under laboratory conditions. Baits leached 50% of the 1080 present in 5 h, and 90% in 24 h, consistent with results of water monitoring that have shown some contamination in streams but not in repeat samples taken later. Suren (2006) obtained no evidence of 1080 being washed into streams after heavy rainfall, perhaps not surprisingly, given the massive dilution that would occur after heavy rain. 1080 has not been detected in over 300 samples of reticulated water and there is no evidence of significant or prolonged contamination in surface waters (Eason & Temple 2008). Given the amounts of 1080 used in ground and aerial applications, exposure of individuals living near possum control areas is unlikely, and it is even more unlikely to occur if safety procedures are adhered to.

In other research by the National Institute of Water & Atmospheric Research (NIWA), scientists deliberately spiked small streams with 1080 baits. They examined the ecological consequences of 1080 baiting in waterways, found none, and at the same time monitored the fate of 1080 (Suren & Lambert 2004 unpubl., 2006; Suren 2006; Suren & Bonnett 2006). In these field experiments, quantities of baits were deliberately placed in small (< 3 m wide) streams. 1080 was only detected in water samples for a short period (< 24 h) thereafter. 1080 concentrations 10 m downstream from the site where baits were added were higher than those 100 m downstream, illustrating the influence of dilution. Observed concentrations were always below the MoH guidelines of 2 μg L^{-1} (ppb). It is noteworthy that the conclusions of research groups involved in over a decade of monitoring of actual aerial control operations, and the conclusions of the NIWA research team conducting a simulated exposure, were very similar. Suren (2006) recommended water samples be taken within 4–8 h of streams being potentially contaminated with bait, and suggested that later monitoring was likely to be of limited value. All the research groups have reported that when contamination does occur, only low concentrations are detected and only for a short period. Even in small streams and under warm conditions, dilution of 1080 will be more important than biodegradation, since dilution occurs immediately.

In practice, Medical Officers of Health in New Zealand are faced with a complex array of legislation, guidelines and challenges when exercising their responsibilities in regard to authorisation of 1080 aerial operations and applying conditions for monitoring water quality. In this regard, the conclusions of the NIWA researchers on the duration of 1080 in water after deliberate spiking of streams are helpful, in particular Suren's (2006) recommendation that water samples should preferably be taken within 4–8 h of potential contamination with 1080 from baits. It would seem to be sensible to extend

this recommendation to sampling close to reticulated water supplies. In conclusion, there is a case for rationalisation of water monitoring based on the results of the recent NIWA research, limiting samples to within or at 24 h after an operation.

In summary, research in the 1990s has demonstrated that biodegradation could play an important role in the elimination of 1080 derived from baits and residues in waterways. There are two means by which any 1080 present in water will be reduced to undetectable and toxicologically insignificant amounts: (1) dilution, and (2) biodegradation. Even though substantial biodegradation can occur over the first 24 h of 1080 entering water, the effect of dilution will be immediate. There have been recent suggestions that further research on 1080 degradation in water would be useful (Weaver 2003). However, since dilution to undetectable concentrations is likely to occur before significant biodegradation, a focus firstly on more information about the fate of baits in streams and operational improvements to avoid watercourses might be more fruitful. Water monitoring since 1990 has shown that significant water contamination is unlikely when safety procedures are adhered to. In the amounts used either in ground or aerial application, exposure of individuals living near possum control areas is most unlikely to occur. Research teams involved in water monitoring in the 1990s initially looked for 1080 in streams, sometimes for several days after rainfall and on occasion for some weeks after the application of baits. None of the monitoring work picked up 1080 washed into streams after heavy rainfall. Research on bait degradation and the effects of soil microorganisms indicate that 1080 from baits that are not eaten by pests is unlikely to migrate to streams in measurable quantities, particularly when dilution is combined with biodegradation. As indicated above, if or when small amounts of 1080 enter streams, biodegradation is likely to be overshadowed by dilution, even when conditions for biodegradation are favourable. If 1080 monitoring of small streams is to be undertaken, then water samples should be taken within 4–8 h after the streams are potentially contaminated if the purpose is to detect transient contamination. However, when testing public water supply sources, water analyses need to demonstrate absence of 1080; hence sampling within and at 24 h will be important.

In terms of effects on aquatic organisms, the concentrations described above in 'real world' operational settings are consistently below those that will impact on aquatic organisms. This conclusion is most clearly demonstrated by assessing aquatic toxicology data. Historical data indicate that fish are relatively resistant to 1080. Fingerling bream and bass (species unidentified) survived indefinitely, without any signs of toxicity, in water containing 370 mg of 1080 per litre (King & Penfound 1946). In New Zealand, fingerling trout (species unspecified) were subjected to 1080 concentrations of 500 mg L^{-1} and 1000 mg L^{-1} without any visible effect on the fish. Force-feeding pellets containing a total of about 4 mg of 1080 (two fingerling trout and five adult trout) or about 8 mg of 1080 (two adult trout) also had no visible effect (Rammell & Fleming 1978). Fluoroacetamide (a compound related to 1080) at a concentration of 3500 mg L^{-1} killed only 50% of a harlequin fish (*Rasbara heteromorpha*) population in 48 h. The LD_{50} of 1080 after intraperitoneal injection was approximately 500 mg kg^{-1} (Bauermeister et al. 1977). No explanation of the high resistance of fish to 1080 has been published, but it is presumably associated with differences in metabolic pathways or the relative importance of the Krebs

cycle in fish metabolism (Rammell & Fleming 1978).

During 1993, three aquatic toxicity tests were completed in the USA. The first estimated the acute toxicity of 1080 to bluegill sunfish (*Lepomis macrochirus*). No mortality or sublethal effects were observed at any concentration tested, with a highest NOEC (no observed effect concentration) of 970 mg L^{-1}. Based on the results of that study and criteria established by the US Environmental Protection Agency (EPA), 1080 would be classified as practically non-toxic to bluegill sunfish. The second test, on rainbow trout, used the same test conditions as the bluegill sunfish studies. The NOEC was 13 mg L^{-1}, which the US EPA classifies as slightly toxic to rainbow trout. The third test estimated the acute toxicity (EC_{50}) of 1080 to the small freshwater invertebrate *Daphnia magna*. The 48-h EC_{50} value for daphniids exposed to 1080 was 350 mg L^{-1} and the NOEC was 130 mg L^{-1}, making 1080 practically non-toxic to *Daphnia magna* by US EPA classification standards (Fagerstone et al. 1994). An early experiment reported that mosquito larvae (*Anopheles quadrimaculatus*) were comparatively sensitive to 1080 (Deonier et al. 1946), with 1080 concentrations as low as 0.025 mg L^{-1} being fatal to 15% of a test population. Since the concentrations of 1080 described above are many times higher than residue concentrations associated with 1080 use (<0.001 mg L^{-1}), adverse effects on aquatic animals are unlikely. In practice, these data would only be of value in risk assessment relating to a large amount of 1080 bait or stock solutions being deliberately or accidentally tipped into a waterway.

In their field research on 1080 residue analyses in waterways, Suren and Lambert (2006) not only studied the responses of an invertebrate community to 1080 contamination, but also monitored caged longfin eels (*Anguilla dieffenbachii*), koaro (*Galaxias brevipinnis*) and upland bullies (*Gobiomorphus breviceps*) after deliberately spiking small streams. Despite large numbers of toxic baits being placed in the study streams (maximum 80 baits in the stream with greatest water discharge), 1080 was detected for less than 12 h, and only at very low concentrations (c. 0.2 µg L^{-1}). No effects of 1080 were detected on any of the invertebrate species, including caddisflies (*Helicopsyche* sp., *Pycnocentrodes* sp., *Pycnocentria* sp.), orthoclad midges and mayflies (*Deleatidium* sp.), or the three caged fish species. These findings are consistent with the concentrations present being below those having toxic effects on these species. Further work on eels (*Anguilla dieffenbachii*) and freshwater crayfish (*Paranephrops planifrons*), when the animals were fed 1080 baits under experimental conditions designed to mimic real-site situations, also showed no effects (Lyver et al. 2005; Suren & Bonnett 2006).

Non-target responses

Controlling pests has enhanced and restored New Zealand ecosystems and protected endangered native species. Careful monitoring of the use of 1080 has been essential to ensure that non-target effects are minimised and the benefits of pest control outweigh adverse effects (Eason 2002a). Non-target effects of 1080 used for possum control have been studied extensively during the last 20–30 years in New Zealand (Harrison 1978; Spurr 1991, 1994; Eason et al. 1998b; Innes & Barker 1999; Powlesland et al. 1999; Lloyd & McQueen 2000).

Effects on birds

Bird deaths have been reported during 1080 pest control

operations almost from when they were first conducted in New Zealand. However, the first formal monitoring of bird deaths occurred in 1976–77. During that monitoring exercise, it was discovered that a number of birds from a range of species were dying during control operations in which non-dyed, raspberry-lured carrot bait that had a high percentage of small fragments ('chaff') was being used (Harrison 1978). This led to significant changes in baits, with carrot baits being screened to remove small fragments, raspberry lure being banned, baits being dyed green, and cinnamon oil added as a deterrent. At about the same time, bait application rates started to be reduced and there was an increased use of cereal-based baits. Fewer birds and fewer species have been reported dead since these changes were made (Spurr 1994).

The significance of any loss of individuals as a result of 1080 poisoning to the long-term viability of a species depends on its resilience. Therefore, much recent monitoring has focused on the impacts 1080 has on native bird species at a population level. Those species with large populations and a high reproductive rate can compensate for small losses. North Island robins (*Petroica australis longipes*) suffered c. 50% mortality during a 1996 poison operation using 1080 carrots. However, a reduction in predators resulted in improved breeding of robins. A year later the robin population contained more birds and a greater proportion of females than just prior to the operation (Powlesland et al. 1999). Similarly, during a 1080 carrot operation in 1997, mortality of tomtits (*Petroica macrocephala*) was estimated to be 79% (Powlesland et al. 2000). However, the tomtits had enhanced nesting success following the 1080 operation, with pairs rearing two, and in some cases, three broods the following season. More recent research indicates that cereal bait operations using low sowing rates and large baits have little if any impact on tomtit populations (Westbrooke & Powlesland 2005).

In contrast, threatened species usually have a limited ability to recover from additional mortality, making the consequences of potential losses from poisoning of more concern. Therefore, a number of studies using individually marked birds have been undertaken on threatened native non-target populations to determine the effects of aerial poisoning, using carrots and cereal baits. Most intensive monitoring has been of kōkako (*Callaeas cinerea wilsoni*), with four of 366 birds monitored through 1080 operations disappearing, giving a calculated mortality rate of 1.4% deaths per operation (Flux & Innes 2001). Kiwi have been closely studied with radio-tagged adult kiwi monitored throughout 1080 operations; no deaths were recorded. In total, 269 kiwi have been monitored throughout fourteen 1080 operations (10 aerial, 3 handlaid and 1 bait station operation). Species monitored include the North Island brown kiwi (*Apteryx mantelli*), great spotted kiwi (*A. haastii*), rowi (*A. rowi*) and Haast Tokoeka (*A. australis*) (H. Robertson, pers. comm.). All 73 kākā (*Nestor meridionalis*), 19 blue ducks (*Hymenolaimus malacorhynchus*) and 15 kererū (*Hemiphaga novaeseelandiae*) monitored through aerial poisoning operations using radio transmitters have survived. During monitoring, one of 40 weka (*Gallirallus australis*) died, and four of 23 fernbirds (*Bowdleria punctata*) disappeared. None of these studies have identified population-level mortality that threatens the viability of a species (Broome et al. 2009). Recent monitoring of kea (*Nestor notabilis*) suggests that some populations may be affected by aerial 1080 operations. Research is ongoing to identify those populations at risk and whether the risks can be mitigated (Broome et al. 2009).

Seaton et al. (2009) collected productivity data from 87 New Zealand falcon nests (*Falco novaeseelandiae*) in the Kaingaroa pine plantation during three breeding seasons, 2003–2006. During this time, 1080 pellets and carrots were ground laid or aerially applied in forest compartments where falcon breed. During the study, the breeding falcon population increased from 20 to 36 pairs, leading the authors to conclude that 1080 did not have a negative impact on falcon, and in fact probably had a positive impact by reducing predation pressure on them.

Very recently, the collation and analysis of 48 surveys of individually banded or radio-tracked birds during aerial 1080 operations from 1986 to 2009 was completed by Veltman and Westbrooke (2011). Their paper is a very important contribution to the field. They confirmed that kiwi, kākā and kōkako have a small risk of mortality. However, they also noted that many of the surveys in which zero bird mortality has been observed had small sample sizes, creating a false sense of security. Furthermore, operations in which bird survival had been tracked often did not follow best baiting practices, or have adequate monitoring methodology. Eleven native species reported dead after 1080 operations have not been studied systematically by radio-collaring individuals to monitor survival (Veltman & Westbrooke 2011). The authors highlighted a further need to obtain robust information on bird mortality during 1080 operations, including long-term monitoring and the judicious use of intensive methods for data-poor species, and when baiting procedures change. Baiting practice has continued to evolve (Morgan 1994, 2004; Morgan et al. 1997) and now routinely includes prefeeding (Warburton et al. 2009), raising the possibility it could similarly affect bird behaviour and increase the poisoning risk to birds (Veltman & Westbrooke 2011).

Considerable effort has been invested in research on ways to further reduce the negative effects of 1080 on non-target species through the use of bait stations, bait matrices that are less palatable to non-target species, and by identifying and incorporating potential repellents (e.g. see Hickling 1997: Morgan et al. 1997 unpubl. report; Spurr & Porter 1998; Morgan 1999; Day et al. 2003; Ross & Henderson 2003 unpubl. report). While the first two lines of research have been successful and adopted during 1080 operations, research into repellents has been less successful. Despite improvements, Veltman and Westbrooke (2011) recommend continued diligence and vigilance to improve monitoring.

Effects on invertebrates

In surveys conducted in the 1990s, no negative impacts were detected on populations of weta in Waipoua Forest, a range of invertebrate species on Rangitoto Island, predatory insects in Mapara Reserve, or ground-dwelling invertebrates in Puketi Forest and Titirangi Reserve (Spurr 1994). Observations of the number of species and number of individual invertebrates found feeding on 1080 baits has led to the prediction that vertebrate pest control operations are unlikely to have long-term deleterious impacts on invertebrate populations (Sherley et al. 1999; Spurr & Drew 1999). This conclusion is consistent with the monitoring of Aspen et al. (1999), which showed no impacts on the number of ground-dwelling insects up to 1 year after aerial 1080 application. Similar results were obtained by Spurr and Berben (2004) using artificial refuges and mark-recapture techniques. No impacts on weta, slugs, cockroaches and spiders were identified. Powlesland et al. (2005) have since confirmed that aerial 1080 did not have a detrimental effect on forest invertebrates.

Effects on deer

The percentage of red deer (*Cervus elaphus scoticus*) killed during aerial 1080 poisoning operations has been formally monitored in several different areas, and was found to range from 5% to 93% after an aerial carrot operation in Pureora in 1997 (Fraser & Sweetapple 2000). Two-thirds to three-quarters of the fallow deer (*Dama dama*) population in the Blue Mountains were killed during an aerial pellet operation (Nugent & Yockney 2004), the kill appearing to be greatest for the youngest (smallest) deer in areas with the highest deer density and the most open understorey. There appears to be no consistent pattern in the number of deer deaths based on bait type, sowing rate or toxic loading, suggesting that other factors such as deer density are important (Nugent et al. 2001). Deer by-kill can now be avoided by including a deer repellent in bait that does not deter possums (Morriss 2007).

Risks to domestic animals

Dogs are extremely susceptible to 1080 and must be kept away from toxic baits and possum carcasses. Records of dog poisoning incidents have not been maintained during the years that 1080 has been used; however, 254 dogs were reported to have been killed by 1080 during 1960–1976 (Rammell & Fleming 1978). The results of a persistence study by Meenken and Booth (1997) coupled with knowledge of the LD_{50} for dogs, is useful for getting a better understanding of secondary poisoning risks to dogs. Based on an LD_{50} for dogs of 0.07 mg kg^{-1}, a 20-kg dog would be seriously at risk if it consumed 200 g of toxic offal containing 7 mg kg^{-1} of 1080. In Meenken and Booth's (1997) study, 5 out of 6 possums on day 25, and 4 out of 10 on day 75, were reported to exceed this threshold. One possum carcass out of 6 on day 25 contained 70 mg kg^{-1}, 10 times the amount needed to kill a 20-kg dog. These analyses confirm that dogs are a special case because of their unique sensitivity to 1080 (Meenken & Booth 1997). Concentrations of 1080 in rat and rabbit carcasses ranged from 1.9 to 14.4 mg kg^{-1} (rats) and from <0.02 to 0.78 mg kg^{-1} (rabbits) (Twigg et al. 2003), showing they also present risks to dogs. Cats, being predators, are also susceptible to secondary poisoning from 1080 (Eason 2002b). While showing a high degree of sensitivity to 1080 (LD_{50} of 0.28 mg kg^{-1}) (Eason & Frampton 1991), it is lower than that for dogs (LD_{50} of 0.07 mg kg^{-1}), and clinical signs in cats are less severe (Eason & Frampton 1991).

Livestock can also succumb to 1080 poisoning (ERMA 2008) and must be kept well away from baits. Even partially degraded baits should be regarded as hazardous to sheep and cattle (*Bos* sp.). Where livestock deaths have been reported, it has generally been due to animals being returned too early to areas where 1080 application has occurred (Eason 2002b). Sheep exposed to single sub-lethal doses of 1080 have not been reported to experience any long-term adverse affects (Eason 2002b).

Improved methods of bait application

There has been a sustained effort to increase target specificity and reduce bait application rates when using 1080. Bait application rates of 3 kg ha^{-1} or less are now used (Morgan 1994, 2004; Morgan et al. 1997a). Pest control operators guard against the careless use of 1080, poor-quality control operations, and the use of poor-quality baits. Non-target mortality can be minimised by well-planned operations using high-quality baits and by the increased use of bait stations (Eason 2002a). However, as aerial 1080 methods continue to evolve, e.g. the use of prefeeding to enhance bait uptake by possums and rodents, further monitoring is required to ensure that the new methods do not impact on native species (Veltman & Westbrooke 2011).

Conclusions

Fluoroacetate, the active ingredient of 1080, occurs naturally in toxic plants in Australia, Africa, and South America. Manufactured 1080 is used to control introduced mammals in New Zealand and Australia to protect native species from the impacts of introduced pests. Some microorganisms in the soil, such as *Pseudomonas* spp., will defluorinate 1080. Water-monitoring surveys, conducted during the 1990s, have confirmed that significant contamination of waterways is unlikely following aerial application of 1080 bait (Eason 2002a).

1080 is a broad-spectrum poison that acts by interfering with the energy-producing tricarboxylic acid cycle in the mitochondria. Possums die from heart failure, often within 6–18 h of eating baits. Dogs are extremely susceptible to poisoning. If livestock become exposed to 1080 bait, a minimum withholding period of 5–10 days should be enforced to enable excretion of 1080, so that residues will not occur in meat. High-quality baits reduce non-target impacts on birds. Current evidence suggests that populations of bird species and invertebrates are not adversely affected, and adverse

Table 7. Summary of key advantages and disadvantages of sodium fluoroacetate (compound 1080) for possum control (from Eason 2002a).

Advantages	Disadvantages
• Highly effective for achieving a rapid reduction in possum numbers	• Controversial, especially aerial operations
• Can achieve consistently high kills	
• The only poison available for aerial applications	• Secondary poisoning risk (especially dogs) from possum carcasses
• Biodegradable in the environment	• No effective antidote
• Inexpensive compared to most other poisons	• Can only be used by licensed operators in government agencies
• Kills other pest species	• Poor-quality bait causes bird deaths
• High-quality efficacy data exist to support both aerial- and ground-baiting techniques	• Generates bait shyness if target animal receives sub-lethal doses

effects of 1080 use are outweighed by ecosystem protection and the reduction of pest impacts on native species. However, further monitoring using current aerial baiting techniques that now include prefeeding is required to ensure that short-term negative effects on non-target species are minimised. Table 7 summarises the main advantages and disadvantages of 1080 use. Long-term exposure to sub-lethal doses can have harmful effects, and strict safety precautions are enforced to protect contractors and workers in the bait manufacturing industry. When its use is controversial, risk communicators must take care not to trivialise the toxicity of 1080, but to discuss the risk alongside measures taken to mitigate adverse effects (Eason 2002a).

The benefits of 1080 use in conservation, pest control, and disease control need to be weighed alongside the risks of using 1080 and alternative techniques for pest control. Considerable care must be taken when using 1080 to ensure that the risks of its use are outweighed by ecological benefits achieved.

Acknowledgements

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